**Long-Term Effects of Concussion on Motor Performance**
**Across the Lifespan**

**by**


**Douglas N. Martini**

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**Doctoral Committee:**

        **Associate Professor Steven P. Broglio, Chair**
        **Assistant Professor James T. Eckner**
        **Assistant Professor Deanna Gates**
        **Assistant Research Scientist Grant C. Goulet**
        **Associate Professor Jeffrey S. Kutcher**
        **Assistant Professor Sean K. Meehan**

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| | |
|---|---|
| $^1$H-MRS (MRS) | Proton Magnetic Resonance Spectroscopy |
| Acetyl-CoA | Acetyl Coenzyme A |
| AD | Alzheimer's Disease |
| ANOVA | Analysis of Variance |
| ApEn | Approximate Entropy |
| ATP | Adenosine Triphosphate |
| BA6 | Brodmann Area 6 |
| BESS | Balance Error Scoring System |
| BOLD | Blood-Oxygen Dependence Level |
| Ca+ | Calcium |
| Cho | Choline |
| Cm | Centimeters |
| CNS | Central Nervous System |
| COM | Center of Mass |
| COP | Center of Pressure |
| Cr | Creatine |
| CRI | Concussion Resolution Index |
| CSP | Cortical Silent Period |
| CTE | Chronic Traumatic Encephalopathy |
| DLPFC | Dorsal Lateral Prefrontal Cortex |
| DTI | Diffusion Tensor Imaging |
| EEG | Electroencephalography |
| ERN | Error-Related Negativity |
| ERP | Event Related Potential |
| fMRI | Functional Magnetic Resonance Imaging |
| GSC | Graded Symptom Checklist |
| GTs | Glial Tangles |
| ImPACT | Immediate Post Concussion |
| Kg | Kilograms |
| m | Meters |
| m/s | Meters/Second |
| MANOVA | Multivariate Analysis of Variance |
| MCI | Mild Cognitive Impairment |
| MRI | Magnetic Resonance Imaging |
| MVC | Maximum Voluntary Contraction |
| NAA | *N*-Acetylasparate |
| Na-K | Sodium-Potassium |

| | |
|---|---|
| NFTs | Neurofibrillary Tangles |
| Nm | Newton meters |
| NTs | Neuropil Threads |
| PCS | Post-Concussion Syndrome |
| PNS | Peripheral Nervous System |
| PPC | Posterior Parietal Cortex |
| RMSE | Root Mean Squared Error |
| RT | Reaction Time |
| RTP | Return to Play |
| ROI | Region of Interest |
| SAC | Standard Assessment of Concussion |
| SOT | Sensory Organization Test |
| TMS | Transcranial Magnetic Stimulation |
| yo | Years old |
| Yrs | Years |

# ABSTRACT

This dissertation aims to provide critical insight into the possible long-term effects of concussion on motor and cognitive performance, through a set of cross-sectional investigations. To date, the elite athlete population has garnered most of the research and public attention, while the largest athletic population, high school athletes, has been largely overlooked. The hypotheses state that individuals with a concussion history will have worse cognitive and motor performance and that this trend will be divergent with age. That is, the previously concussed individuals will exhibit worse performance, and will be divergently worse from the control group with age. With this in mind, the three investigations focus on cognitive and motor performance in three age groups (i.e. 20, 40, and 60 year olds), in those with and without an adolescent concussion history. The first investigation assessed cognition between concussion history and control groups, within age groups. Using a standard computer-based, clinical concussion assessment, processing speed, attention, learning, working memory accuracy and working memory speed were quantified for each concussion group by age. There were no differences between the concussion history and control groups, within age. The second investigation assessed gait spatio-temporal, kinematic, and toe clearance variables. Again, no significant concussion history group differences were observed in the multivariate assessment for the gait spatio-temporal, kinematic, and toe clearance variables. In addition, there appeared to be no pattern suggesting that a concussion history adversely affects gait, across age. The final investigation assessed skill acquisition, implicit

learning, and the internal timing mechanism between concussion history and control groups, within age. Again, there was no consistent pattern to suggest an adverse relationship between concussion history and motor performance, across age. Considering this set of observations, there does not appear to be a long-term, negative relationship between adolescent concussion history and cognition or motor performance in this population.

# Chapter 1: Introduction

**1.1 Statement of Problem**

Interest and research in sport concussion has grown exponentially over the previous decade due to the media and political spotlight. The findings of these investigations have led to an evolving understanding of the acute/chronic effects of a concussion(s). Traditional incidence rates of concussion were thought to approach 250,000 cases annually (Gerberich, Priest, Boen, Staub, & Maxwell, 1983), however, recent changes in the understanding of concussive injuries have resulted in an updated estimate of 1.6-3.8 million concussions resulting from sport and recreation participation annually (Langlois, Rutland-Brown, & Wald, 2006). The current definition of concussion is "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces" (Aubry et al., 2002). Common sequelae that incorporate clinical, pathological, and biomechanical injury constructs define the nature of a concussive head injury, an injury caused by a direct blow to the body (i.e. head, neck, torso) with "impulsive" force transmitted to the head, resulting in rapid onset of short lived impairment of function and neuropathological changes (Aubry et al., 2002). This denotes that a concussion reflects a functional disturbance opposed to a structural change in the cerebral tissue (Aubry et al., 2002). The underlying root of this dysfunction has been observed metabolically in both animals and humans via different methodologies. (Giza & Hovda, 2001, 2014; Jantzen, Anderson, Steinberg, & Kelso, 2004; Johnson et al., 2011; Vagnozzi et al., 2010).

With the onset of a concussive impact, there is an increased metabolic demand in combination with a simultaneous decrease in cerebral blood flow (Giza & Hovda, 2001, 2014). The injury induces hypermetabolism of glucose and the disparity between glucose supply and demand triggers cellular energy crisis (Giza & Hovda, 2001, 2014). This energy crisis makes the brain more susceptible to recurrent concussion due to cells' inability to cope with the energy demands of secondary injuries, possibly leading to cell termination (Giza & Hovda, 2001, 2014). The clinical impairments brought about by the metabolic injury appear to resolve around two weeks post-concussion, although there are multiple factors that can play a role in the duration and severity of symptoms.

The clinical presentation of this metabolic shift is frequently a delay in verbal and motor responses, confusion, memory deficits, motor performance deficits, and inability to focus attention (American Academy of, 1997). These effects are most commonly presented during the acute stage of concussion. There are two groups of concussion symptoms that can be present, early symptoms (minutes and hours) and late symptoms (days and weeks). The early symptoms commonly include headache, dizziness/vertigo, lack of awareness of surroundings, and nausea and/or vomiting (American Academy of, 1997). An abbreviated list of late symptoms includes a persistent low-grade headache, light-headedness, poor attention and concentration, anxiety and/or depressed mood, and memory dysfunction (American Academy of, 1997). For the purpose of this paper, the acute phase of a concussion will be defined as the time period with both clinical and metabolic symptoms present (typically 7-10 days post-concussion). The sub-acute phase will be defined as the time period after clinical recovery, yet continuous metabolic alterations are present (typically up to 30 days post-concussion). The chronic phase will be

defined as the time beyond one-year post concussion. Importantly, research has shown that metabolic alterations can exist following a series of head impacts without any clinical diagnosis of a concussion (Bazarian, Zhu, Blyth, Borrino, & Zhong, 2011; Breedlove et al., 2012; Talavage et al., 2010). This anomaly will be defined as sub-clinical impacts.

A multitude of investigations have evaluated different techniques to assess concussion, with the clinical exam remaining the gold standard for diagnosis. In assessing a concussion, it is important to test the individual's neurostatus, neurocognitive function, and motor performance. Tests that are commonly used to assess an individual's cognitive status include (but not limited to) the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT), the Standard Assessment of Concussion (SAC - neurostatus), the Concussion Resolution Index (CRI), and the CogState by AXON sports. Tests used to assess individuals' motor performance include the Sensory Organization Test (SOT) and the Balance Error Scoring System (BESS). A combination of neurocognitive function, neurostatus, and balance assessments can be used to support the clinical assessment for a successful diagnosis.

Other investigations have analyzed gait and have found significant differences between acutely concussed and matched controls (Catena, Van Donkelaar, & Chou, 2009; Catena, von Donkelaar, & Chou, 2007a, 2007b; Chou, Kaufman, Walker-Rabatin, Brey, & Basford, 2004; Parker, Osternig, Van Donkelaar, & Chou, 2006). These differences range from decreased gait velocity, increased stride time, more conservative sway in the sagittal and coronal planes (Catena et al., 2009; Catena et al., 2007a; Parker et al., 2006). Although gait analysis is not commonly used in the initial clinical concussion assessment, there are data indicating it has the requisite

sensitivity for clinical use. These investigations have analyzed temporal gait parameters, kinetics, and kinematics in concussed participants and reported resolution within 7-10 days, similar to other assessment tools (Catena et al., 2009; Catena et al., 2007a; Parker et al., 2006).

While the clinical declines following concussion are transient in nature, new evidence suggests there may be persistent effects, which are sub-clinical in nature. Using electroencephalography (EEG) and transcranial magnetic stimulation (TMS), investigators observed alterations in cognitive function in those with a concussion history (Broglio, Pontifex, O'Connor, & Hillman, 2009; De Beaumont, Brisson, Lassonde, & Jolicoeur, 2007; De Beaumont et al., 2009; Pontifex, O'Connor, Broglio, & Hillman, 2009). Additionally, there is a growing body of evidence suggesting that multiple concussions may be linked to post-concussion syndrome (PCS), memory and cognitive decrements, chronic traumatic encephalopathy (CTE), and motor performance alterations (Chou et al., 2004; Guskiewicz et al., 2005; Guskiewicz et al., 2007; Martini et al., 2011; McKee et al., 2009; McKee et al., 2012; Omalu et al., 2006; Omalu et al., 2005; Ryan & Warden, 2003; Sosnoff, Broglio, Shin, & Ferrara, 2011).

Investigations of retired professional athletes found those with a history of three or more concussions were five-times more likely to be diagnosed with Mild Cognitive Impairment (MCI) and three times more likely to report memory impairment than players with no concussion history (Guskiewicz et al., 2005; Guskiewicz et al., 2007). Case studies describe how previous concussions may lead to deposits of tau in neural tissue, perhaps leading imaginations to a cause and effect event (McKee et al., 2009; Omalu et al., 2005). Though the underpinnings of the increased cerebral disease rates are not fully understood, some are eager to claim that sustaining

multiple concussions and sub-clinical impacts will lead to an early on-set of dementia (Gavett, Stern, Cantu, Nowinski, & McKee, 2010; McKee et al., 2009).

To a lesser extent, motor deficits found in older adults reporting a history of concussion parallel the neurodegeneration reported in former professional athletes. Discrepancies in balance and gait have been observed in both young and middle-aged adults with a concussion(s) history (Chou et al., 2004; Martini et al., 2011; Sosnoff et al., 2011). Among these investigations, sub-clinical differences in motor performance have been measured from one to 30 years post-concussion (Chou et al., 2004; De Beaumont et al., 2009; Martini et al., 2011; Sosnoff et al., 2011).

There is a growing body of evidence suggesting that concussions can no longer be perceived as a transient injury without long-term consequences. However, most scientific literature is based on the professional athlete. Since the elite athlete is subject to a greater exposure to concussions, an understanding of limited exposure to concussion is warranted. The high school athlete who doesn't sustain concussion after adolescence is then the primary focus. Of the 7.7 million high school students participating in athletics each year, only a small percentage participates in collegiate athletics and an even smaller percentage of college athletes make it to the professional level. With incidence rates around 0.25 (per 1000 athletic exposures) for boys and girls high school athletics and over seven million athletes participating, the largest population of previously concussed athletes has been largely ignored (Lincoln et al., 2011; Marar, McIlvain, Fields, & Comstock, 2012).

Ultimately, this culminates in a substantial gap in the concussion history literature. There is a limited understanding of the possible long-term effects of sport-related concussion, suffered in adolescence and never again, on motor and cognitive performance. This lack of knowledge could potentially leave a substantial population vulnerable to future injuries as a result to previous concussion(s). The failure to address how the combination of age and adolescent concussion history affects activities of daily living is a large public health concern. Filling this gap in the literature will provide the first comprehensive insight as to how this populations' motor and cognitive performance is affected as they age.

Given the nature of the evidence for long-term effects of concussion, we hypothesize that individuals with an adolescent concussion history will exhibit an accelerated decline in cognitive and motor performance compared to individuals without a concussion history (Figure 1.1). This decline in cognitive and motor performance could stem from negative changes in brain functionality that would not exist in a concussion free population. The young, previously concussed population may be capable of compensating for the negative alteration in brain functionality, allowing for only subtle differences in performance, though with age, the compensatory mechanism might be incapable of overcoming the intrinsic decrease in brain functionality. The reduction could occur at an accelerated rate for individuals with a concussion history when compared to what is observed in the natural aging process (Broglio, Eckner, Paulson, & Kutcher, 2012). This idea rises from other many accepted behavioral and environmental factors that have a negative influence on neurocognitive performance (Etgen, Sander, Bickel, & Forstl, 2011; McAuley, Kramer, & Colcombe, 2004). An accelerated decline in cognition and motor performance could significantly decrease the quality of life for those with

a concussion history. In an attempt to elucidate further information for this hypothesis, additional

research clarifying the relationship between suffering a sport related concussion(s) during high

school late-life motor performance needs be completed.
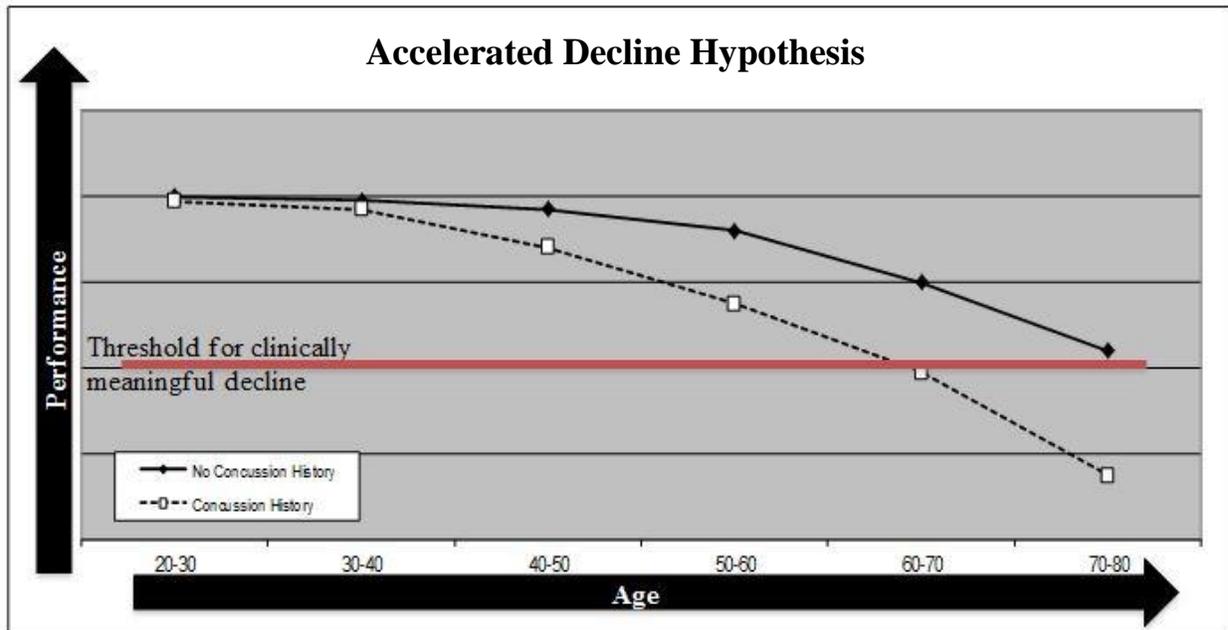


Figure 1.1: Adopted from Broglio, Eckner, Paulson, & Kutcher (2012)

## 1.2 Statement of Purpose and Hypotheses

The purpose of this investigation is to evaluate the long-term effects of sport concussion

sustained prior to college on motor performance, across a lifetime.

Hypothesis 1: Those with a concussion history will perform worse on standard clinical measures

of cognitive function and reaction time. Memory will decrease and reaction time will increase

across ages and diverge across ages and concussion history

Hypothesis 2: Those with a concussion history will adopt a more proactive gait strategy as

indicated by alterations in temporal gait parameters (e.g. velocity, step width, percentage of time

in double leg stance, etc.), gait kinematics (e.g. joint rotations), and obstacle clearance (e.g. toe

clearance). The proactive gait strategy will be an attempt to reduce the risk of falling. Further, these alterations will diverge across age groups and concussion history.

Hypothesis 3: Those with a concussion history will demonstrate a slower ability to implicitly learn a motor pattern and acquire the associated skill during an upper and lower extremity visuomotor task. These discrepancies will diverge across ages and concussion history. Those with a concussion history will present a more variable timing profile during both upper and lower extremity discrete, auditory timing tasks. These discrepancies will diverge across ages and concussion history.

# Chapter 2: Literature Review

The purpose of this literature review is to detail 1) the epidemiology and metabolic effects of a concussion, 2) concussion assessment and management, and 3) the long-term effects of concussion on cognitive, behavioral and motor performance. Providing background of each concussion stage is critical to understanding the persistent nature of concussion related changes and the basis of the Accelerated Decline Hypothesis.

## 2.1 Epidemiology

Incidence rates of sport concussion vary among participation level (i.e. college or high school), sex, and sport (i.e. football, basketball, soccer, etc.). The overall incidence rate in high school athletics, regardless of sport or sex, is 0.24 per 1000 athletic-exposures (Lincoln et al., 2011). An athletic exposure is defined as one athlete participating in a single practice or competition. High school males incur the highest incidence rates during football, ranging from 0.33 to 0.6 (per 1000 exposures) (Gessel, Fields, Collins, Dick, & Comstock, 2007; Lincoln et al., 2011; Schulz et al., 2004). Lacrosse (0.3/1000 exposures) and soccer (0.17-0.23/1000 exposures) round out the top three sports for male high school athletes (Gessel et al., 2007; Lincoln et al., 2011; Schulz et al., 2004). High school females incur concussions most frequently participating in soccer (0.13-0.35/1000 exposures), lacrosse (0.2/1000 exposures), and basketball (0.16-0.21/1000 exposures) (Gessel et al., 2007; Lincoln et al., 2011; Schulz et al., 2004). In general, collegiate athletes incur higher incidence rates than their high school counterparts.

The top three males sports for concussion incidence are football (0.37-0.61/1000 exposures), soccer (0.28-0.49/1000 exposures), and wrestling (0.25-0.49/1000 exposures) (Gessel et al., 2007; Hootman, Dick, & Agel, 2007). The top collegiate female sports report higher rates for soccer (0.41-0.63/1000 exposures), basketball (0.22-0.43/1000 exposures), and lacrosse (0.25/1000 exposures) than any other sports (Gessel et al., 2007; Hootman et al., 2007). Female athletes incur higher rates than males, though the reason for this is still not fully understood.

Player to player contact is responsible for roughly 76% of concussions, while contact with the playing surface is reported at just over 15% of concussions (W. P. Meehan, d'Hemecourt, & Comstock, 2010). Following a concussion, the most common symptoms are headache (86%) dizziness (67%), and confusion (59%) (Guskiewicz, Weaver, Padua, & Garrett, 2000). However, the two signs most associated with sustaining a concussion, loss of consciousness (9%) and amnesia (28%), occur infrequently (Guskiewicz et al., 2000). The lack of knowledge regarding the clinical signs and symptoms of a concussion might be responsible for the undiagnosed rates associated with concussions. Undiagnosed concussion rates are over 50% in high school football and approach 80% for college football (McCrea, Hammeke, Olsen, Leo, & Guskiewicz, 2004; Sefton, Pirog, Capitao, Harackiewicz, & Cordova, 2004). Collectively, these findings suggest a large number of high school athletes sustain at least one concussion during their brief career. Athletes that continue participating through college further increase their risk of sustaining a concussion. However, with significantly fewer collegiate athletes than high school athletes, the focus concussion research should address the 24% of the 7.7 million high school athletes statistically likely to sustain a concussion (Lincoln et al., 2011).

**2.2 Physiology**

A concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces (Aubry et al., 2002). For the purposes of this investigation, concussion will be defined by the following four phases: Sub-concussive/sub-clinical, consisting of metabolic alterations without the athlete presenting clinical signs and symptoms of a concussion. The acute phase consists of a concussed individual presenting both clinical and metabolic alterations following injury. Next, the sub-acute phase will be defined as clinical recovery with ongoing metabolic alterations. Finally, the chronic phase will be both clinical and metabolic recovery with persistent sub-clinical changes.

*2.2.1 Acute Phase*

The idea that brain tissue suffers metabolic dysfunction post-concussion was first presented by Hovda, Yoshino, Kawamata, Katayama, and Becker (1991). Through animal models, disruption of the neuronal membranes lasting 6-24 hours post-injury were observed (Giza & Hovda, 2001, 2014). Impact forces result in axonal stretching which forces the opening of the voltage-dependent potassium (K+) channels, which leads to a marked increase in extracellular K+ (Giza & Hovda, 2001, 2014). In an effort to restore homeostasis, energy requiring membrane pumps are activated and trigger an increase in glucose consumption (Giza & Hovda, 2001, 2014). The sodium-potassium (NA-K) pumps work in an attempt to restore neuronal membrane potential (Giza & Hovda, 2001, 2014). In order to keep the NA-K pump operational, glucose metabolism dramatically increases to synthesize the adenosine triphosphate (ATP) required to operate the NA-K pump (Giza & Hovda, 2001, 2014). Concurrent with the efflux of K+, an influx of calcium (Ca+) occurs (Giza & Hovda, 2001, 2014). In an attempt to control the amount of Ca+ in the cell, the Ca+ may be sequestered in the mitochondria, resulting in impaired oxidative

metabolism (Giza & Hovda, 2001, 2014). This ionic flux is thought to be correlated with the clinical symptoms associated with concussion (i.e. headache) (Giza & Hovda, 2014). Because of the post-injury ion imbalance, energy demand surpasses energy supply, resulting in increased glycolysis (Giza & Hovda, 2001, 2014). The energy crisis leads to increased vulnerability to a subsequent concussion (Giza & Hovda, 2014). Subsequently, lactic acid production is increased, altering the inter-cellular pH (Giza & Hovda, 2001, 2014). As cells die, they release signaling proteins that up-regulate this inflammatory process (O. I. Schmidt, Infanger, Heyde, Ertel, & Stahel, 2004). The time it takes for this cascade of events to transpire may explain why symptoms tend to increase between 6-24 hours post-concussion (Grady, Master, & Gioia, 2012). After the initial period of hyperglycolysis, cerebral glucose consumption diminishes by 24 hours post-injury and remains low for 2-4 weeks post-concussion (Giza & Hovda, 2001, 2014). These metabolic alterations commonly present as functional discrepancies in cognitive and motor function among the acutely concussed. Giza and Hovda suggest while emerging evidence of chronic axonal dysfunction is alarming, the connection between post-concussion axonal pathology and altered axon physiology are still unknown (Giza & Hovda, 2014).

Variant applications of magnetic resonance imaging (MRI) have also provided insight into neurological alterations in both the acutely concussed and sub-concussed. To be clear, sub-concussed is not synonymous with sub-acute. Sub-concussed athletes sustain one or more impacts, but do not result in symptoms or other clinically measurable changes in function. Sub-concussed athletes are not diagnosed with a concussion. For the purpose of this paper, sub-acute is defined as a concussed athlete who is clinically recovered from a concussion, but complete metabolic recovery has not yet occurred. Functional MRI (fMRI) uses blood-oxygen level

dependent (BOLD) contrast signals to assess which regions of the brain are active and how active these regions are during a particular task. The measure of oxygenated blood and deoxygenated blood is possible due to the difference in magnetism arising from the available iron hemoglobin. BOLD signals have been associated with aerobic metabolism, which is linked to neuronal activity. As brain activity increases, so does the demand for oxygenated blood in the areas needed to achieve success on the cognitive task. One major limitation of measuring BOLD signal is the inherent delay between onset of activity and demand of oxygenated blood. This makes fMRI observations weak in temporal resolution, but great for spatial resolution. That said, fMRI data suggests that persistent alterations in BOLD signals exist in both the concussed and sub-concussed athletes (Breedlove et al., 2012; Jantzen et al., 2004; Lovell et al., 2007; Slobounov et al., 2011; Talavage et al., 2010). Lower levels of oxygenated blood preclude a lower nutrient supply, which is consistent with the idea that there is a dearth of available glucose post-concussion.

Among the acutely concussed, fMRI observations of various regions of interest (ROIs) indicate both increases and decreases of BOLD levels. fMRI observations report an increase in BOLD levels in the areas involving working memory and sensorimotor coordination, in the acutely concussed, particularly as task difficulty increased (tested within 10 days of injury) (Jantzen et al., 2004). Interestingly, no behavioral changes were observed (Jantzen et al., 2004). A separate investigation assessed the acutely concussed (tested within a week of injury) and found similar activation abnormalities, though measured from different ROIs. Using a standard memory task, correlations between concussion and the posterior parietal cortex (PPC), as well as Brodmann area 6 (BA6) were reported from BOLD contrasts (Lovell et al., 2007). Interestingly, the

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previously concussed group had lower activation in the PPC and an increase in activity in BA6. Reporting higher symptom severity correlated with lower PPC activity (Lovell et al., 2007). Lastly, higher activity in BA6 correlated with length of recovery (Lovell et al., 2007). More recently, BOLD contrasts measured during a visuospatial task revealed further activity abnormalities. Both the concussed and control groups performed the behavioral task with similar speed and accuracy; however the concussed group had greater activation during the task (Slobounov et al., 2011). The ROIs with higher activation were the dorsal lateral prefrontal cortex (DLPFC), left parietal cortex, and right hippocampus. Unlike the previous fMRI investigations, a major limitation here concerns the time frame of data collection. The authors' state that fMRI data were collected within 30 days of injury, and that all subjects were asymptomatic by day 10 (Slobounov et al., 2011). Such variability in collection period could affect the activity levels measured from the recovering concussed participants.

The discrepancy in activity levels between investigations may be related to the ROIs and the time from injury at which data were collected. Lovell et al. (2007) collected fMRI data within a week and observed increased activation in BA6, located anterior to the central sulcus. Both Jantzen et al. (2004), within ten days of injury, and Slobounov et al. (2011), within a month of injury, observed increased activity in regions anterior to the central sulcus, as well as the parietal lobes. The only area among the investigations with a decrease in activity was the PPC, which may have been washed out in both the Jantzen et al. (2004) and Slobounov et al. (2011) investigations looking at the parietal regions observed.

*2.2.2 Sub-Clinical Impacts*

Acute concussion has garnered most of the spotlight in the literature, though sub-clinical impacts have started to gain attention. Attempting to investigate acute concussion through fMRI, a recent cohort observed sub-clinical alterations in high school football players. Significant differences were observed on clinical tests and fMRI data for the concussed when compared to controls. In-season, clinical testing depicted players who sustained the most sub-clinical impacts as clinically healthy, yet produced altered fMRI activation levels (Talavage et al., 2010). Using a N-back task, lower activation levels were observed in the DLPFC and cerebellum, areas associated with working memory (Talavage et al., 2010). By post-season testing, the activation in the sub-concussed, but clinically normal group was no longer significantly different from the clinically and functionally normal group (Talavage et al., 2010). However, post-season testing for the concussed group revealed a continuation of altered medial temporal gyri activation (Talavage et al., 2010). An investigation by a similar cohort extended these findings, observing greater impact counts for the sub-concussive group when compared to the concussed and control groups (Breedlove et al., 2012).

A separate MRI technique, diffusion tensor imaging (DTI), measures axonal pathways of the cortex. Instead of measuring oxygenated hemoglobin levels, DTI measures the diffusive properties of water in white matter (axons). Alterations in axonal diffusion are thought to be associated with the integrity of the axon(s). Since concussions have been described as a diffuse axonal injury, it is logical to believe that alterations in white matter persist post-injury (Maruta, Lee, Jacobs, & Ghajar, 2010). In line with Talavage and Breedlove, an investigation of high school football players report alterations in diffusion for both a sub-concussed and concussed group, compared to controls (Bazarian et al., 2011; Breedlove et al., 2012; Talavage et al., 2010).

High school football players are known to sustain over 650 impacts per seasons (Broglio et al., 2011), making it plausible there is a cumulative effect of sustaining impacts, not just concussions. Multiple variables play in to impact counts in football: level of play (i.e. college versus high school), position (i.e. linemen, running back, linebacker, etc.), and type of offense (running versus passing) (Broglio et al., 2011; Martini, Eckner, Kutcher, & Broglio, 2013; Schnebel, Gwin, Anderson, & Gatlin, 2007). In order to ensure athlete safety, proper management of a concussion is necessary, especially considering the possible cumulative effect of impacts with concussions. The first step in concussion management is concussion assessment.

*2.2.3 Sub-Acute Phase*
As previously discussed, the sub-acute phase of concussion recovery refers to clinical recovery, yet persistent, underlying metabolic alterations exist. This significance of the sub-acute phase is the potential for athletes to be returned to play prior to complete injury resolution. It is likely athletes are unknowingly returned during this phase, but the potential consequences for this are poorly understood. Through the use of proton magnetic resonance spectroscopy (^{1}H-MRS), investigators observed a significant decrease in the brain metabolite N-acetylasparate (NAA), choline (Cho), and creatine (Cr) ratios during sub-acute concussion (Johnson et al., 2011; Vagnozzi et al., 2010). NAA has a close relationship with ATP and has been suggested as an indirect biomarker of the brain's energy state (Johnson et al., 2011; Tavazzi et al., 2007; Vagnozzi et al., 2010; Vagnozzi et al., 2008; Vagnozzi et al., 2007). During periods of surplus ATP and acetyl coenzyme A (acetyl-CoA), NAA can be synthesized. The energetics model suggests enzymatic activity, converts L-glutamate and acetyl-CoA into NAA (C. Madhavarao, Chinopoulos, Chandrasekaran, & Namboodiri, 2003; C. N. Madhavarao & Namboodiri, 2006). Therefore, decreased levels of NAA may suggest decreased levels of ATP, acetyl-CoA, and the

variants of glutamate associated with NAA synthesis (Vagnozzi et al., 2007). A trio of separate

investigations observed a reduction in NAA/Cho and NAA/Cr ratios in the recently concussed

(Johnson et al., 2011; Vagnozzi et al., 2008; Vagnozzi et al., 2007). A significant reduction in

these compounds were measured on day three post injury, with partial recovery by day 22 and

full recovery by all participants by day 30 (Johnson et al., 2011; Vagnozzi et al., 2008; Vagnozzi

et al., 2007). Interestingly, Vagnozzi et al. (2010) reported clinical recovery by day 15 post-

injury, while ^{1}H-MRS scan indicated ongoing recovery out to day 30 post-injury. In addition

increased NAA ratios were observed with an increase in the number concussive injuries

sustained (Vagnozzi et al., 2008).


Research using other modalities has also evaluated the idea of continued metabolic alterations in

individuals who have suffered a concussion. Event related potentials (ERPs) are a way to

measure brain activity during a specific task (i.e. odd ball) through electroencephalography

(EEG). Instead of using a magnetic field (e.g. MRI, fMRI, and DTI) to induce/record molecular

changes, EEG uses electrodes placed on the scalp to record electrical activity from a group of

neurons. P3 amplitude and latency are generally the variables being measured in ERP

investigations. The "P" refers to the polarity of the wave and "3" (or 300) refers to the latency

post stimulus. P3 amplitude has been associated with the ability to allocate attentional resources

(Polich, Howard, & Starr, 1983; Reinvang, Nordby, & Nielsen, 2000). Multiple investigations

observed an inverse relationship between reported symptoms and P3 amplitude suppression

(Dupuis, Johnston, Lavoie, Lepore, & Lassonde, 2000; Lavoie, Dupuis, Johnston, Leclerc, &

Lassonde, 2004). Observations of electrical activity suggest there are factors stemming from

symptom severity and concussion history affecting neural activity. Evidence exists, suggesting in

the absence of symptoms, concussions may cause abnormal neurophysiological patterns when responding to attention demanding tasks (Gosselin, Theriault, Leclerc, Montplaisir, & Lassonde, 2006). However, more evidence exists for a reduction in P3 amplitude in the symptomatic (acute) stage of concussion (Dupuis et al., 2000; Lavoie et al., 2004). Athletes sustaining multiple concussions have greater P3 reduction than athletes with only one or no concussions (Theriault, De Beaumont, Gosselin, Filipinni, & Lassonde, 2009).

Together, these data suggests that even after the clinical recovery period, the neural activity is not fully recovered. Metabolic recovery appears to take an additional two to three weeks beyond the clinically observed recovery, possibly resulting in increased vulnerability to further injury. Importantly, direct metabolic measures have only occurred using animal models. Human observations made with $^1$H-MRS, fMRI, DTI, and EEG serve as a proxy for measuring neuro-metabolism. While these methods are not a direct measure of metabolism, each can be used to infer fluctuations of metabolic activity. Accounting for the volume of concussions, sub-clinical impacts, and evidence of metabolic alterations persisting beyond clinical recovery, sustaining further concussive and sub-clinical impacts before full recovery could predispose athletes to an accelerated decline of cognitive and motor performance as they age.

**2.3 Concussion Assessment and Management**
The diagnosis of an acute concussion is made through the physical exam by a medical professional and involves the assessment of a range of domains including: symptoms – somatic, cognitive, and/or emotional; physical signs; behavioral changes; cognitive impairment; motor impairment; and sleep disturbance (McCrory et al., 2009). Due to the complex nature of the injury it is crucial to follow a systematic sideline injury assessment. McCrory et al. (2009)

suggest the player should be medically evaluated on site, including an evaluation of player

disposition, first aid, concussion assessment, monitoring for deterioration, and preventing them

from returning to play on the day of the injury. The most recent Concussion Consensus

Statement (McCrory et al., 2013) provides a suggested return to play (RTP) protocol for proper

concussion management. The RTP decision will be covered in greater detailed following clinical

assessment.

There are three types of concussion assessment tools that are widely used in the field, symptom

self-reporting, balance, and neurological testing. Neuroimaging has been suggested as a possible

assessment tool, but is not practical for sideline assessment. Additionally, standard imaging is

only useful in determining further, structural damage to the brain. While advanced imaging

techniques have proven beneficial for research, they are not widely accepted for clinical use. The

following section will cover clinically observed changes in balance and cognitive function at the

time of injury (acute phase).

*2.4 Acute Sequelae*

   *2.4.1 Symptoms*

The most common method to evaluate for concussion is assessing for the symptoms. Figure 2.1

is the graded symptoms checklist (GSC), which is commonly used to assess the presence of

concussion symptoms (McCrea et al., 2003). The three most commonly reported concussion

symptoms are headache (86%), dizziness (67%), and confusion (59%) (Guskiewicz et al., 2000).

The two most common physical signs associate with a concussion, loss of consciousness and

amnesia, occur in only 9% and 28% of cases, respectively (Guskiewicz et al., 2000). In an

investigation to test the validity of self- reported symptoms, the GSC symptoms were grouped

into three categories of measurement: somatic, neurobehavioral, and cognitive (Piland, Motl, Guskiewicz, McCrea, & Ferrara, 2006). Using a factor analysis model Piland et al. (2006) created a 9- and 16-item GSC scale to test the correlation of the groupings. The 9-item scale had a better fit than the 16-item scale, groups were constructed as: somatic – headache, nausea, and balance problems; neurobehavioral – sleeping

**Graded Symptom Checklist (GSC)**

| Symptom | Time of injury | 2-3 Hours postinjury | 24 Hours postinjury | 48 Hours postinjury | 72 Hours postinjury |
|---|---|---|---|---|---|
| Blurred vision | | | | | |
| Dizziness | | | | | |
| Drowsiness | | | | | |
| Excess sleep | | | | | |
| Easily distracted | | | | | |
| Fatigue | | | | | |
| Feel "in a fog" | | | | | |
| Feel "slowed down" | | | | | |
| Headache | | | | | |
| Inappropriate emotions | | | | | |
| Irritability | | | | | |
| Loss of consciousness | | | | | |
| Loss or orientation | | | | | |
| Memory problems | | | | | |
| Nausea | | | | | |
| Nervousness | | | | | |
| Personality change | | | | | |
| Poor balance/coordination | | | | | |
| Poor concentration | | | | | |
| Ringing in ears | | | | | |
| Sadness | | | | | |
| Seeing stars | | | | | |
| Sensitivity to light | | | | | |
| Sensitivity to noise | | | | | |
| Sleep disturbance | | | | | |
| Vacant stare/glassy eyed | | | | | |
| Vomiting | | | | | |

NOTE: The GSC should be used not only for the initial evaluation but for each subsequent follow-up assessment until all signs and symptoms have cleared at rest and during physical exertion. In lieu of simply checking each symptom present, the ATC can ask the athlete to grade or score the severity of the symptom on a scale of 0-6, where 0=not present, 1=mild, 3=moderate, and 6=most severe.

**Figure 2.1:** Graded Symptoms Checklist (Guskiewicz, Bruce, Cantu, Ferrara, Kelly, McCrea, Putukian, & Valovich McLeod, 2004)

more than normal, drowsiness, and fatigue; and cognitive – feeling "slowed down", feeling "in a fog", and difficulty concentrating (Piland et al., 2006).

Individuals that suffered a concussion returned to symptom baseline level within five to seven days post-injury (McCrea et al., 2003). It is irresponsible to apply the GSC or its iterations as the lone tool in diagnosing a concussion. As a standalone assessment, self-reported symptoms are only up to 68% sensitive (Broglio, Macciocchi, & Ferrara, 2007). Indeed, many of the symptoms associated with a concussion may present from physical activity, nutrition, and/or environmental factors (i.e., headache, dizziness, nausea, etc.). Additionally, athletes can suppress and lie about symptoms during concussion assessment. The use of multiple assessments is the best approach in accurately (~90%) diagnosing a concussion (Broglio et al., 2007).

*2.4.2 Neurostatus*

The Standard Assessment of Concussion (SAC) is a sideline test of neurostatus that is quick and inexpensive in assessing if an athlete has sustained a concussion (McCrea, 2001; McCrea, Kelly, Kluge, Ackley, & Randolph, 1997). The largest limitation of the SAC is that the sensitivity to detecting a concussion declines following the initial 48 hours of the injury (Miller, Adamson, Pink, & Sweet, 2007). The components of the SAC consist of an immediate memory portion, an orientation portion, a concentration portion, and a delayed memory portion (McCrea, 2001; McCrea et al., 1997). McCrea et al. (2003) measured the acute effects of concussion and the continuous time course to recovery following a concussion using the SAC. McCrea et al. (2003) data suggests that collegiate football players require two days post-injury to fully recover from a sport related concussion, based on SAC data, suggesting a more rigorous neurocognitive test beyond 48 hours post-injury. The SAC is insensitive to sub-concussive impacts across a season, suggesting the results from the SAC remain un-confounded by sustaining multiple sub-concussive impacts (Miller et al., 2007). Accordingly, with a decline of one point from baseline score, the SAC is 94% sensitive (Barr & McCrea, 2001).

*2.4.3 Neurocognition*

Neurocognitive testing has become a common aspect of the concussion assessment battery. In the modern era, the majority of these tests are administered on computer platforms. The major limitation of the following test is they require a computer, making sideline assessment near impossible. There are a plethora of computer-based options, including the ImPACT, CRI, and CogState. This investigation will implement the Internet based CogSport by Axon. The CogState consists of four tasks, with each task involving a virtual deck of playing cards, facedown. The four tasks are: Detection Task - simple reaction time (RT); Identification Task – choice RT; One

Card Learning – continuous learning task; and One Back Task – N-back design. Each of these tasks is designed to assess different aspects of cognitive function including: the Identification task assesses visual attention; the One Card Learning assesses visual recognition memory and attention; and the One Back task assesses working memory and attention. Throughout the test the "K" key of the keyboard denotes a 'Yes' response and the 'D' key denotes a 'No' response. Each task produces outcome measures of accuracy and time. Built within the software is set of integrity checks, assuring that full participation is met. The CogState by AXON has shown good test-retest reliability (Collie, Maruff, Darby, & McStephen, 2003; J. T. Eckner, J. S. Kutcher, & J. K. Richardson, 2011). A greater detail of description can be found in previous literature (Collie, Maruff, Makdissi, et al., 2003; J. T. Eckner et al., 2011). With a decline of score on just two or more tasks, the sensitivity of the CogSport/Axon is 96.6% (Louey et al., 2014).

*2.4.4 Motor Control*

The last commonly evaluated domain for concussion assessment and management is motor control, with static balance serving as the most common motor control task. Static balance is not possible without the afferent signaling from the peripheral nervous system (PNS) to the central nervous system (CNS). The acquisition and conversion of stimuli from the muscles, tendons, joints, and deep tissues by the PNS that allows the motor components (i.e. cerebellum, basal ganglia, premotor, and supplementary motor cortices) to process afferent signal appropriately to an event (Lephart & Fu, 2000). Somatosensory inputs, primarily tactile and proprioceptive, are integrated with vestibular and visual signals in the brain stem to assist in the control of autonomic tasks such as postural equilibrium (Lephart & Fu, 2000). For the concern of this paper the focus of postural equilibrium or stability will encompass balance and gait (Lephart & Fu, 2000).

Voluntary movement is described as a goal directed movement that improves with practice. There are three known laws for voluntary movement and they are: 1) The brain creates an abstract depiction of the movement through the movement dependent cortical areas; 2) The amount of time it takes to compete the task depends on the complexity of the task itself; 3) Finally, there is a speed/accuracy tradeoff (Kandel, Schwartz, & Jessell, 2000). The abstract depiction is created though movement kinematics (joint angles) and movement dynamics (forces necessary to produce the aforementioned angles), and is known as the motor program (Kandel et al., 2000). The complexity of the task dictates a multitude of factors, including (but certainly not limited to) the number of cortices/central nervous system (CNS) areas necessary for the movement. Additionally, the number of options/choices/decisions available to produce the correct response is a factor in the speed/accuracy tradeoff. The latter factor is known as the "choice effect" (Kandel et al., 2000). This suggests that the more choices that are presented in directing a movement, the more time it will take to select the correct movement (Kandel et al., 2000).

Feed-forward mechanisms do not rely on direct sensory input throughout movement, but instead rely on experience and initial sensory observation to complete the movement goal. As previously mentioned, feedback mechanisms generally have longer movement times, as they must rely on afferent signals and integration before generating an efferent response (R. A. Schmidt & Lee, 2005). The movements using either feedback or feed-forward mechanisms will improve with time and practice.

The ability to improve is easily visualized at the complete system level, but there is an actual neural progression from early skill through automatic ability. In the very early stages of learning a task, there have been suggestions that the cerebellum is active with cerebellar cortex-dentate nucleus and cerebellar-cortical pathways active (Doyon et al., 2002; Kandel et al., 2000). As time and practice progresses, there is a shift to a striatal-cortical network, finally culminating in a striatal-supplementary motor area-task specific cortical network (Doyon et al., 2002). This progression has also been observed in a more recent investigation in an older, healthy population (Sean K Meehan, Randhawa, Wessel, & Boyd, 2011). Suggestions have been thusly made that the cerebellum and pre-supplementary motor area are responsible for learning a new skill, but that the actual storage of the acquired motor program occurs in the striatum, supplementary motor area, and movement relevant cortical areas (Doyon et al., 2002; Kandel et al., 2000). These early stages of motor learning are likely tied to a closed-loop, feedback controlled system. However, as the skill progresses, there is a shift to a feed-forward controlled system. The ability to anticipate relies on experience. This experience allows individuals to focus on the necessary sensory input as well as visualize the movement. Snyder, Batista, and Andersen (1997) showed that the posterior parietal cortex (PPC) was active during planned movements of the upper extremity, but when planning the movement was not an option, it remained quiet. Since the PPC is located next to the visual cortex and between that and the motor, Snyder et al. (1997) postulated that it may be involved in the visualization of a movement. The motor area associated with planning a movement is the supplementary motor area (Doyon et al., 2002; Kandel et al., 2000).

Greater depth is added to this idea though the work of Haxby et al. (1991) who showed that within the visual and adjacent cortices, the visual stimuli activated different areas based on the complexity of the stimuli. The presentation of facial recognition (the complex task) activated an occipital-temporal area, while a simpler spatial task (necessary for catching a ball) stimulus activated the lateral, superior parietal cortex (Haxby et al., 1991). So the initial cerebellar-cortical network active in early task acquisition is probable more complex than the just a cerebellar-pre-supplementary area connection. It likely also involves areas of sensory input that are pertinent to the movement. This increases the amount of time needed to complete the task, which will improve with practice. The aforementioned ability to ignore unattended stimuli, while focus on attended stimuli has been modeled by Hillyard, Vogel, and Luck (1998). Through a task that required individuals to indicate if the target stimuli appeared as red or green, Hillyard et al. (1998) showed (through measures of regional cerebral blood flow – rCBF) that there was an attenuation of activity (gain control) when the stimulus was present. One would expect the ability to attenuate focus on a target stimulus to improve with practice. As always, there is a top down control for every movement, whereby the spinal cord is moderated by the brainstem, which is moderated by the cortex and within each area, the medial descending (efferent/motor – lateral overall) pathways control the axial muscles, while the lateral pathways control the movement of the limbs. The ascending (afferent/sensory –medial overall) pathways run in parallel, allowing for quicker feedback via interneurons at each level of the CNS (Kandel et al., 2000). Lastly, the cerebellum is not just for skill acquisition, but also active in order to provide postural stability (with the vestibular nerves in brain stem) and smooth motion (with the basal ganglia) (Kandel et al., 2000).

2.4.4a Reaction Time

While most Neurocognitive computer tests measure reaction time (RT) and variants of, not every situation will allow for computer testing on the sideline. A cohort from the University of Michigan developed a tool for quick, sideline RT assessment. This task provides both a cognitive and motor aspect of motor control. The RT tool consists of a 1.3m long rod, with markings at .5cm increments, affixed to a weighted disk (James T Eckner, Kutcher, & Richardson, 2010; James T Eckner, Jeffrey S Kutcher, & James K Richardson, 2011). The tester holds the rod at the opposite end of the disk while the athlete watches for the disk to drop (James T Eckner et al., 2011). The RT is calculated by recording the distance the rod fell before being caught, with each athlete receiving eight attempts, which are averaged (James T Eckner et al., 2011). This RT tool has been shown to be both reliable and sensitive in measuring a decrease in RT post-concussion (J. T. Eckner, Kutcher, Broglio, & Richardson, 2013; James T Eckner et al., 2010; J. T. Eckner et al., 2011).

2.4.4b Balance

The Sensory Organization Test (SOT) is a device that assesses the stability of sensory function and balance performance (Dickin & Clark, 2007). The SOT manipulates visual and somatosensory inputs through six testing conditions, allowing for the subjects' center of pressure (COP) to be assessed (Dickin & Clark, 2007). The six conditions for the SOT create the following environments: eyes open with stable support surface and stable visual surround; eyes closed with stable support surface; eyes opened with a stable support surface and a sway-referenced surround; eyes open with a sway-referenced support and stable visual surround; eyes closed with a sway-referenced support; and eyes open with both support surface and visual surround sway-referenced (Dickin & Clark, 2007). Dickin and Clark (2007) reported that the

equilibrium score for each of the six conditions of the SOT are reliable. A change greater than 6.8 in the SOT composite score yields a sensitivity of about 62% in concussion diagnosis (Broglio et al., 2007).

The SOT was applied to concussion research in an attempt to assess postural control post-concussion (Guskiewicz, Riemann, Perrin, & Nashner, 1997). Guskiewicz et al. (1997) administered the SOT to a group of acutely concussed collegiate athletes indicating overall postural stability decreased until about three days post-injury, suggesting that an athlete who has suffered a concussion be held out of play at least three days post-injury (Guskiewicz et al., 1997; Guskiewicz, Ross, & Marshall, 2001). A separate investigation, using a longer testing period, found that individuals that suffered a concussion had significantly different balance ten days post-injury (Peterson, Ferrara, Mrazik, Piland, & Elliot, 2003). Sosnoff, Broglio, and Ferrara (2008) later observed an association between cognitive and motor decrements in the recently concussed, but not prior to injury. These findings suggest concussions increase the cognitive-motor association, insinuating coordinated cognitive-motor processes are functionally damaged (Sosnoff et al., 2008).

SOT evidence of postural alterations prompted the creation of a sideline assessment of balance following concussion. The Balance Error Scoring System (BESS) is a test of postural stability where three different stances – double leg stance, single leg stance, and tandem leg stance (one foot directly behind the other) – with eyes closed (Guskiewicz et al., 2001). The stances are performed on both a sturdy surface and unstable surface (i.e. foam mat) (Guskiewicz et al., 2001). Anytime an athlete skewed from the initial 30-degree angle of the hip, opened their eyes,

put their second foot down, or strayed from the test position the subject received a one point on the final score (Guskiewicz et al., 2001). Similar to the SOT, the acutely concussed return to baseline levels by post-injury day 3 (Guskiewicz et al., 2001). Based on the findings of this study, the BESS is thought to provide the same clinical information as the SOT, making it a practical, valid, and cost-effective method of objectively assessing postural stability in an athlete that might be suffering a concussion (Guskiewicz et al., 2001).

As previously discussed, the neural complexity to movement suggests that a linear approach to understanding balance may not capture the task complexity. For example, irregular oscillations occur within normal, quiet standing (Brouwer, Culham, Liston, & Grant, 1998; Després, Lamoureux, & Beuter, 2000), suggesting that a nonlinear approach to balance quantification may provide more clinical information. Approximate entropy (ApEn) is such an attempt to quantify this variance in a time series, not meant replace current COP measures (Cavanaugh, Guskiewicz, Giuliani, et al., 2005; Pincus, 1991). In the acutely concussed population, the ApEn values for COP medial-lateral sway decreased post injury, lower ApEn values indicating greater regularity in sway (Cavanaugh, Guskiewicz, Giuliani, et al., 2005; Cavanaugh et al., 2006). A greater regularity is theorized to suggest greater constraint is put on the postural system at some level (i.e. neural and/or mechanical) (Cavanaugh, Guskiewicz, Giuliani, et al., 2005; Cavanaugh et al., 2006). Interestingly, ApEn COP medial-lateral irregularity remained low beyond recovery of standard measures of postural stability (Cavanaugh, Guskiewicz, Giuliani, et al., 2005; Cavanaugh et al., 2006). While SOT measures show recovery starting at day three post-injury, ApEn COP medial-lateral irregularity persisted beyond the typical three day recovery (Cavanaugh, Guskiewicz, & Stergiou, 2005; Guskiewicz et al., 1997; Guskiewicz et al., 2001).

<u>2.4.4c Gait</u>

As discussed, there are inherent changes in simple motor performance (i.e. RT and balance) post-concussion. More recently, a plethora of research has investigated how complex motor performance (i.e. gait, dual task gait, etc.) may be affected in the acute and sub-acute phases of concussion (Catena et al., 2007a, 2007b; Fait, McFadyen, Swaine, & Cantin, 2009; P. Fait, Swaine, Cantin, Leblond, & McFadyen, 2013; Parker, Osternig, Lee, Donkelaar, & Chou, 2005; Parker et al., 2006; Parker, Osternig, Van Donkelaar, & Chou, 2007). Gait alterations have been observed in simple gait paradigms (i.e. no obstacle and/or secondary task) and to a greater extent, in complex gait paradigms (with an obstacle and/or secondary task) (Catena et al., 2007a, 2007b; Fait et al., 2009; P. Fait et al., 2013; Parker et al., 2005; Parker et al., 2006, 2007).

Concussed individuals presented differences in gait velocity, step width, stride length, stride time, obstacle clearance, and center of mass (COM) motion in the coronal and sagittal planes, compared to matched controls (Catena et al., 2007a, 2007b; Fait et al., 2009; P. Fait et al., 2013; Parker et al., 2005; Parker et al., 2006, 2007). The greatest alterations were observed in the early stages (within 48 post-concussion) and during the complex gait paradigms (Catena et al., 2007a, 2007b; Parker et al., 2005; Parker et al., 2006, 2007). The recently concussed presented slower gait velocities, shorter stride lengths, greater stride times, and reduced COM and COP separation (Catena et al., 2007a, 2007b; Parker et al., 2005; Parker et al., 2006, 2007). These alterations were apparent in the simple gait paradigms, and significant in the complex gait paradigms. The COM motion in the coronal plane tended to be greater in all but one investigation (Catena et al., 2007a, 2007b; Parker et al., 2005; Parker et al., 2006, 2007). Interestingly, even after symptom resolution and return to baseline-level scores on neurocognitive tests, the concussed still presented gait alterations up to 30 days post-concussion (Catena et al., 2007a, 2007b; Fait et al.,

2009; P. Fait et al., 2013; Parker et al., 2005; Parker et al., 2006, 2007). In attempts to avoid/clear an obstacle, two different observations were made. Catena et al. (2007a)observed increased stride times, greater coronal and sagittal COM motion, and an increased distance between the trailing foot and the top of the obstacle.

Contrary to greater obstacle clearance, a separate cohort observed a smaller clearance when participants were instructed to navigate around an obstacle (Fait et al., 2009; P. Fait et al., 2013). Fait et al. (2009); (2013) also observed a preference for dominant side avoidance, in that there was a smaller clearance on the non-dominant side passing. Two different obstacle avoidance tasks result in two different results, suggesting there are different gait strategies in obstacle avoidance. In both observations there are arguments for a greater risk of falling. Greater obstacle clearance results in greater sagittal sway, shorter obstacle avoidance could result in an increased risk of contacting obstacles and increased risk for tripping. Interestingly, even after symptom resolution and return to baseline-level scores on neurocognitive tests, the concussed still presented gait alterations up to 30 days post-concussion (Catena et al., 2007a, 2007b; Fait et al., 2009; P. Fait et al., 2013; Parker et al., 2005; Parker et al., 2006, 2007). Understanding that subtle, persistent changes exist beyond symptom recovery suggests athletes may be vulnerable to further injury.

Additionally, there are observed difference between athlete and non-athlete concussions. This provides further evidence that sustaining multiple sub-clinical impacts and/or concussive injuries has a greater effect on neural operation and recovery. In each of these investigations, individuals with a concussion showed more balance instability in the dual task situations than the single task

and also adopted a more conservative gait strategy than their healthy counterparts (Catena et al., 2007a, 2007b; Parker et al., 2005; Parker et al., 2006, 2007). Parker and colleagues noted that the concussed subjects showed a slower gait on day two than they did on the final testing day (day 28) (Parker et al., 2005; Parker et al., 2006, 2007).

Lastly, a more recent investigation intended to provide the most effective gait task in concussion assessment (Catena et al., 2009). That is, what gait condition will properly separate the gait discrepancies between the concussed and control group. A question and answer cognitive task was effective up to six days post-concussion, but when an obstacle was introduced the cognitive task became effective again (Catena et al., 2009). Unfortunately, a major flaw of this investigation was the use of previously concussed in both the concussed and control groups. Previous findings suggest that history of concussion may be a factor in gait performance, as many as six years beyond the most recent concussion (Martini et al., 2011).

As discussed, there are a multitude of concussion assessment tools available for health professionals responsible for athlete care. Using a package of these tools is the most accurate way of diagnosing a concussion. Proper care for an athlete starts with proper assessment of possible concussion.

2.4.4d Dual Task

Current concussion research has separately focused on cognitive and motor testing as a means to assess recovery following concussion (Parker et al., 2007). Dual task conditions are used to better mimic real-world conditions where cognitive and motor tasks are combined. Commonly used motor tasks are balance and gait, coupled with spelling of a common five-letter word, continuous subtraction by a certain digit, and reciting the months of the year in reverse order

(Catena et al., 2007a, 2007b; Chou et al., 2004; Parker et al., 2005; Parker et al., 2006, 2007). It

is widely accepted that there is a certain amount of cognitive effort needed for each task and the

investment of less than the allotted effort will cause a deterioration of the performance

(Kahneman, 1973). This assumption was noted by Siu and Woollacott (2007), who observed a

subjects' ability to flexibly allocate attention between postural and cognitive tasks. Each

participant completed four different conditions; visual spatial memory tasked with the postural

task, performance of the two tasks with instructions to focus on the postural task; performance of

the two tasks with instructions to focus on the visual memory spatial memory task, and baseline

(Siu & Woollacott, 2007). The authors found that postural stability has a higher priority under

dual task contexts (Siu & Woollacott, 2007). "Tie-breaking" rules are consistently applied in

cases of simultaneous stimuli (Kahneman, 1973). Attentional demands associated with postural

control vary with postural task difficulty and secondary task difficulty (Woollacott & Shumway-

Cook, 2002). That is, age related differences in postural stability significantly increase with task

difficulty, including the Brooks' spatial memory task (Maylor & Wing, 1996). Concussed

individuals are less able to constrain their output task demands, which is similar to the concept

that investment of less than the allocated effort per task will ultimately result in the deterioration

of the task (Kahneman, 1973; Sosnoff et al., 2011).


2.4.4e Age-Related Declines in Motor Performance
Similar to the acute effects of concussion, normal aging will also influence motor control

performance in a negative manner. Falls are the leading cause of injury and death in those 65 and

older (National Safety Council, 2014). As individuals age there is a natural decline in both

cognition and motor performance. This decline has been measured in both balance and gait

(Bover & Woolacott, 1986; Hahn & Chou, 2004; Laughton et al., 2003; Morse, Tylko, & Dixon,

1987; Winter, 1995; Wollacott, 1993). Both intrinsic and extrinsic factors play a role in falls in

the elderly. The physiological breakdown of brain function that occurs with age plays a major

role in the risk of falling (Lockhart, Smith, & Woldstad, 2005). Additionally, inherent changes in

muscle strength, coinciding with advancing age plays a key role in the ability to recover from a

stumble or slip (Larsson, 1982; Rice & Cunningham, 2001). The combination of decreased

sensitivity of the proprioceptive, vestibular, and vision systems in detecting imbalance, followed

by insufficient muscle strength to recover lead to the fall (Lockhart et al., 2005). Alterations in

gait parameters are a sign of adopting a conservative gait strategy in healthy, elderly population

in order to decrease the risk of falling (Hahn & Chou, 2004; Maki, 1997). Conservative gait in

the elderly has been measured as increased toe clearance (typically observed at in the second half

of the swing phase), shorter stride lengths, greater step widths, reduced hip extension, and

reduced ankle plantarflexion (H.-C. Chen, J. A. Ashton-Miller, N. B. Alexander, & A. B.

Schultz, 1991; Kerrigan, Todd, Della Croce, Lipsitz, & Collins, 1998; Sparrow, Shinkfield,

Chow, & Begg, 1996).


*2.5 Return to Play*
The most recent consensus statement stresses that same day RTP should not occur, instead a

graduated protocol should begin once the athlete is asymptomatic (McCrory et al., 2013). The

RTP protocol (Figure 2.2) outlines a step-wise progression beginning with physical and mental

rest until symptom resolution, continuing through added physical and mental stresses every 24

hours, unless symptoms return (McCrory et al., 2013). If symptoms return at any stage of the

RTP process, the athlete returns to the first stage, physical and mental rest (McCrory et al.,

2013). However, without any setbacks, the protocol gets the athlete back to full participation by

the seventh day post symptom resolution (McCrory et al., 2013). While it is recommended that

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there are 24 hour windows between

each stage, it is recognized that some

athletes may progress more quickly

than the protocol dictates. The pace

of RTP depends on the individual

and the severity of the injury at

assessment.

| Rehabilitation stage | Functional exercise at each stage of rehabilitation | Objective of each stage |
|---|---|---|
| 1. No activity | Symptom limited physical and cognitive rest | Recovery |
| 2. Light aerobic exercise | Walking, swimming or stationary cycling keeping intensity <70% maximum permitted heart rate No resistance training | Increase HR |
| 3. Sport-specific exercise | Skating drills in ice hockey, running drills in soccer. No head impact activities | Add movement |
| 4. Non-contact training drills | Progression to more complex training drills, eg, passing drills in football and ice hockey May start progressive resistance training | Exercise, coordination and cognitive load |
| 5. Full-contact practice | Following medical clearance participate in normal training activities | Restore confidence and assess functional skills by coaching staff |
| 6. Return to play | Normal game play | |

In most cases, adhering to these RTP

guidelines allows the athlete to return

upon clinical recovery, generally

**Figure 2.2**: RTP Guidelines from the 4th International Consensus Statement on Concussion in Sport (McCrory et al., 2013)

within two weeks. Yet, as previously

stated, metabolic and subtle gait alterations can persist up to 30 days following a concussion.

Though still unproven, persistent metabolic and gait alterations could be inherently linked. The

neural pathways responsible for gait motor control could be incapable of overcoming the

metabolic shortcomings originating from the concussion.

Interestingly, the acutely concussed youth exhibit gait patterns characteristic of a healthy elderly

population. Both groups exhibit a decreased walking velocity and shorter stride lengths, thought

to be associated to a more conservative walking strategy (Parker et al., 2005; Parker et al., 2006)

(H.-C. Chen et al., 1991; Kerrigan et al., 1998; Sparrow et al., 1996). Though these observed

differences in the concussed dissipate by day 30, more subtle cognitive and motor alterations

may persist. These gait alterations may be the subtle onset of an accelerated decline in motor

performance. The young brain may be capable of compensating for the physiological breakdown of brain function beyond the 30-day mark. However, with a natural, age-related decrease in of proprioceptive, vestibular, and visual sensitivity, the compensating mechanisms may become unsustainable. Typically, healthy elderly increase obstacle toe clearance, which increases the stride time (H.-C. Chen et al., 1991; Sparrow et al., 1996). By increasing stride time, the elderly increases the amount of time with the COM outside of the base of support. Evidence suggests that instability in the coronal plane is a major risk factor for hip fractures in the elderly (Greenspan et al., 1998). The combination of concussion history and age may accelerate the natural cognitive and motor performance decline.

**2.6 Chronic Sequelae**
There are a plethora of studies reporting on the short-term effects of concussion on balance and gait, yet there is a dearth of knowledge that analyzes the effects on gait and stability long-term. Instead, the focus turns to the cognitive and behavioral deficits associated with brain damage thought to be associated with concussion (Broglio et al., 2009; De Beaumont et al., 2007; De Beaumont et al., 2009; Gaetz, Goodman, & Weinberg, 2000; Gosselin et al., 2006; Guskiewicz et al., 2005; Guskiewicz et al., 2007; Omalu et al., 2006; Omalu et al., 2005). Metabolic, cognitive, and motor performance deficit observations up to a month post-concussion bolster the theory that long-term consequences are a serious concern. The next logical step in understanding concussions is investigating potential chronic changes.

*2.6.1 Post-Concussion Syndrome*
Post-concussion syndrome (PCS) has been acknowledged since at least World War I, though it has had a variety of names (Jones, Fear, & Wessely, 2007). While most (80-90%) concussive

injuries last 7-10 days (McCrory et al., 2013), a small number of concussed athletes have

persistent symptoms. PCS is defined as concussion symptoms persisting beyond three months of

injury, however no truly universal definition exists for the syndrome (Center for Disease Control,

2011). The International Classification of Disease-10 has been most frequently used in

classification of PCS (Mittenberg, Canyock, Condit, & Patton, 2001; Mittenberg & Strauman,

2000). Persistence of cognitive, emotional, and somatic symptoms are noted in the limited cases

of unresolved concussion (Gaetz et al., 2000; William Drew Gouvier, Barbara Cubic, Glenn

Jones, Phillip Brantley, & Quinton Cutlip, 1992; Smith-Seemiller, Fow, Kant, & Franzen, 2003).

The most common symptoms associated with PCS were headache and irritability (Gaetz et al.,

2000). History of concussion, anxiety/stress, and/or other injuries could be factors in determining

the duration of PCS symptom presence (William Drew Gouvier et al., 1992). If the standard RTP

guidelines (McCrory et al., 2013) were not followed, it is easy to understand that the symptoms

could be exacerbated, prolonging symptom recovery. PCS (along with other lifestyle habits)

could be the predecessor to chronic traumatic encephalopathy (CTE) or a CTE-like dementia,

though more research needs to be done to determine if this is accurate.

*2.6.2 Chronic Traumatic Encephalopathy*
The current understanding of CTE suggests that neural degradation occurs as a result of

sustaining multiple sub-clinical/concussive impacts and/or concussions. Professional athletes

(i.e. football, boxing, etc) are especially vulnerable due to the mass accumulation of both

concussive and sub-clinical impacts. The concept of CTE has been around since Martland's

(Martland, 1928) motor control observations in professional boxers. The term "punch drunk"

was coined for the motor alterations found in boxers. CTE is a progressive neurological

deterioration associated with tau-immunoreactive deposits in the superficial cortical layers

(McKee et al., 2009). The slow neurological deterioration eventually progresses into full

dementia (McKee et al., 2009). Microscopic characteristics of CTE include neurofibrillary

tangles (NFTs), neuropil threads (NTs), glial tangles (GTs), and β-amyloid plaques (Gavett,

Stern, & McKee, 2011; McKee et al., 2009; Omalu et al., 2005). The neurological deterioration

results in Alzheimer's and Parkinsonian-like signs and symptoms (Baugh et al., 2012; Gavett et

al., 2011; McKee et al., 2009; McKee et al., 2012; Omalu et al., 2006; Omalu et al., 2005; Stern

et al., 2011). The bulk of the findings are from case studies and/or from a niche inside of the

professional athlete population. Additionally, many of the athletes that have been reported on

have had a list of confounding lifestyle choices (i.e. drug abuse) and personal and family medical

histories. Well controlled studies evaluating the epidemiology or pathological development of

CTE have not been completed, suggesting that any claim be taken lightly until more robust

investigations are conducted. However, concerns still exist when considering possible long-term

effects of sustaining multiple concussions. While the evidence is not as strong as the acute

effects, there is a growing body of literature that suggests negative alterations may be occurring

to individuals with a history of concussions.


*2.6.3 Behavioral Alterations*
The increased risks of depression, memory impairment, MCI, and AD have been identified as

possible chronic consequences of concussions, specifically recurrent concussions (Guskiewicz et

al., 2005; Guskiewicz et al., 2007). In an attempt to quantify the likelihood of MCI and neural

degeneration, retired NFL athletes were surveyed, along with an informant (spouse or close

relative of former athlete) (Guskiewicz et al., 2005). For a percentage of athletes (those over 50)

a second questionnaire was used to assess MCI according to the American Academy of

Neurology (Guskiewicz et al., 2005). 61% of the athletes surveyed sustained at least one

concussion, while 24% sustained three or more concussions throughout their playing career (Guskiewicz et al., 2005). Interestingly, those with three or more concussions were five times more likely to suffer from MCI (Guskiewicz et al., 2005). A similar cohort built off the previous investigation to assess the likelihood of depression in retired athletes. Participants received a general health questionnaire that consisted of the Short Form 36 Measurement Model for Functional Assessment of Health and Well-Being (SF-36) (Guskiewicz et al., 2007). 36% of respondents reported at least one concussion, while 24% reported three or more concussions (Guskiewicz et al., 2007). Individuals with three or more concussions were three times more likely to be diagnosed with depression, whereas those with one or two concussions were only one and a half times more likely to be diagnosed with depression (Guskiewicz et al., 2007). Similar to the CTE data, there are concerns of possible links a history of concussions and late-life cognitive impairment. Though, this population is also specific to the elite athlete, as well as the standard limitations (i.e. reporting bias, self-reported health history, etc) of survey data.

*2.6.4 Neurocognitive Alteration*
Beyond microscopic (tau immunostaining) evidence of chronic effects of a concussion, there is large-scale evidence of electrophysiological alterations in those with a history of concussion. There are EEG and transcranial magnetic stimulation (TMS) evidence that those who suffer a concussion have altered brain activity beyond the initial recovery phase. These electrophysiological alterations have been observed from 30 months to 30 years post-concussion (Broglio et al., 2009; De Beaumont et al., 2007; De Beaumont et al., 2009; Pontifex et al., 2009). Separate investigations observed college-aged individuals whose most recent concussion was 2.9 years prior to testing (Broglio et al., 2009; Pontifex et al., 2009). Pontifex et al. (2009) found that not only did the concussions group perform worse on a flanker task; they also had a reduction in

error-related negativity (ERN). ERN is linked to the brain's activity in response to performing an incorrect task response (i.e. flanker task) (Pontifex et al., 2009). Broglio et al. (2009) observed P3b suppression and latency, as well as N2 suppression in those who suffered a concussion 3.4 years prior to testing. N2 is thought to be involved with response inhibition and attentional orienting (Broglio et al., 2009). Suppression of the P3 components (P3a and P3b) have also been recorded in older adults whose most recent concussion was beyond 30 years prior to testing (De Beaumont et al., 2009). The P3a component is thought to be involved in engaging focal attention, while the P3b is linked to allocating attentional resources.

Using a TMS paradigm, individuals who suffered a concussion(s) (60 months post-concussion) had a prolonged cortical silent period (CSP), and the latency was exacerbated with more injuries (De Beaumont et al., 2007). De Beaumont et al. (2007); (2009) provide evidence that the degree and number of previous injuries have chronic changes in electrophysiological activity. Though, how these apparent attention decrements affect quality of life is not clear. More than anything, these data provide motivation to investigate a broader spectrum of the possible effects of sustaining a concussion(s).

These findings are similar to the sub-acute phase of concussion. The major difference between the two stages of concussion being that years have passed between the most recent concussion and the time of testing, opposed to weeks. These data provide the first empirical evidence that there is a tangible difference between individuals with and without a concussion history. How these differences alter daily life for individuals with a concussion history is still unknown.

Though, effort is being made to find alterations in performance of daily activity (i.e. gait and balance).

*2.6.5 Motor Performance Alterations*
In recent years, there has been a tremendous focus on how concussions may affect cognitive performance years after injury. There is a dearth of knowledge however, about chronic motor performance differences and concussions and/or sub-clinical impacts. Separate investigations have observed alterations in both balance and gait from one year to over six years post-concussion (Chou et al., 2004; Martini et al., 2011; Sosnoff et al., 2011). In the only study that has evaluated balance, a group of college-aged individuals, 44 months post-concussion, had greater anterior-posterior sway irregularity and less medial-lateral sway irregularity with increased task difficulty (Sosnoff et al., 2011). The non-linear measure Approximate Entropy (ApEn) was used to estimate postural sway predictability (regular versus irregular) with lower ApEn scores are thought to represent a more predicable sway that results from fewer control processes, making the individual more susceptible to falls (Sosnoff et al., 2011). Similar observations have been reported in an older population suggesting these changes may be associated with increased risk of falls (Holtzer, Verghese, Xue, & Lipton, 2006).

Within gait, middle-aged individuals with a history of concussion (1year post-concussion) demonstrated a slower velocity, shorter stride length, and an increase in the medial-lateral COM motion (Chou et al., 2004). The observed slower velocity and shorter stride length is thought to be responsible for the reduction in whole body anterior-posterior COM motion in the previously concussed. Similar gait alterations were observed in a previously concussed young adult population. The college-aged group of previously concussed (6.3 years post-concussion) spent a

greater percentage of time in double leg stance compared to matched controls. Additionally, as

task difficulty increased, so did the percentage of time spent in double leg stance (Martini et al.,

2011). Similar to the electrophysiological data discussed above, alterations in motor performance

are being observed in older and younger populations following concussion resolution. These

changes do not appear to be measurable using standard clinical tools of injury recovery, but they

do exist and appear to get worse with severity and number of concussions.

**2.7 Summary of Literature Review**
The current literature suggests that in the retired, elite athlete population there are noticeable

differences in behavior and cortical tissue integrity. What is unknown is if and how these same

issues affect those sustaining concussion(s) during their adolescence; yet do not continue to

participate in collegiate or professional level sports where an increased exposure to concussion(s)

could be a difference between the elite athlete long-term outcomes and the general population.

The failure to address how the combination of age and adolescent concussion history affects

activities of daily living is a large public health concern. There are over 7.7 million high school

students participating in athletics each year, which dwarfs the numbers at the collegiate and

professional levels. In order to provide answers and care for the 5.3 million suffering chronic

concussion issues there needs to be further research within the largest subset of former athletes

(Langlois et al., 2006).

Both cognitive and motor alterations have been observed beyond clinical recovery from

concussion (Broglio et al., 2009; Chou et al., 2004; De Beaumont et al., 2007; De Beaumont et

al., 2009; Martini et al., 2011; Sosnoff et al., 2011). Perhaps most concerning is that these

observations are made in college students, not just elite athletes. How the cognitive and motor

alterations progress as the previously concussed population ages remains unknown. The observed cognitive and motor alterations could decline at an accelerated rate when compared to individuals without a concussion history, leading to increased risk of gait instability at a younger age than the norm for motor performance and aging.

The goal of the following investigation is to fill gaps in the existing literature on how a history of adolescent-only concussions affects individuals in their 20s, 40s, and 60s. This data could help provide answers to patients suffering from chronic concussion issues and lay the foundation for rehabilitation protocols to improve the cognitive and motor performance of individuals suffering from the long-term effects of concussion. In addition, the findings from these proposed projects will be the first to elucidate further insight into the Accelerated Decline Hypothesis (Broglio et al., 2012). The clinical assessment project will provide more information for the cognitive and motor control components of the Accelerated Decline Hypothesis. The gait analysis will be more motor driven, providing greater depth to previous investigations observations of long-term effects of concussion on motor performance. The discrete temporal task will provide a motor control analysis, via a separate pathway than the clinical assessment project. The clinical assessment is visually driven, while the discrete temporal is auditory. Lastly, the continuous tracking task will provide more depth for both the cognitive and motor control aspects of the Accelerated Decline Hypothesis. The continuous tracking task is visually driven, with feedback, but differs from the clinical assessment in difficulty. The continuous tracking requires a constant graded response, instead of a series of singular ballistic responses required to complete the clinical assessment.

# Chapter 3: Projects

This chapter is designed to provide the specific aims, hypotheses, methodologies, results, and discussion for each of the proposed investigations. To attain demographic information for each proposed project, a questionnaire covering health, education, fall, sport, and concussion history was used. Information gathered from the questionnaire will provide classification of possible confounding issues between groups (e.g. symptoms felt at testing; contact sport versus non-contact sport participation; number of previous concussions). These possible confounding issues could allow for secondary analysis of the data collected. Groups were further divided by number of pervious concussions, and Pearson's Correlations were used to analyze concussion history (i.e. one concussion versus two or more concussions) within age groups. Additionally, groups could be divided by contact versus non-contact sport participation history. The full questionnaire can be found in Appendix A.

<u>Demographics</u>

All participants (N=100) were recruited from the Southeastern Michigan area through the University of Michigan clinical studies website (umichclinicalstudies.org), The Michigan Daily newspaper, The University Record newspaper, and email. The aforementioned demographic data can be found in Table 3.1. There were no significant differences between groups, within age for any of the descriptive statistics. The 40yo and 60yo concussed groups were predominantly male, though this is likely due to the disproportionately large ratio of male to female sport participation prior to the enactment of Title IX, which was implemented in 1972.

In addition to the exclusionary criterion set in the consent form, individuals with a history of diagnosed depression (n =11) were excluded from all analyses due to the known impairments of depression on motor performance, bringing the total participants to 89 (Caligiuri & Ellwanger, 2000; Gross, Crane, & Fredrickson, 2012; Michalak et al., 2009; Sabbe, Hulstijn, van Hoof, Tuynman-Qua, & Zitman, 1999; Sabbe, Hulstijn, Van Hoof, & Zitman, 1996). Aside from the primary demographic data in Table 3.i, Fall Fear/Risk (Table B.i) and Headache (Table B.ii) tables can be found in Appendix B.

*Falling Assessment*

The Falls Efficacy Scale was implemented in the questionnaire to measure the fear of falling and the Falls Risk Assessment tool was implemented to assess the risk of falling (Nandy et al., 2004; Tinetti, Richman, & Powell, 1990). There were no significant ($p$'s $> 0.05$) main effects for age group, concussion history group, or age by concussion history interaction for the Falls Efficacy Scale. There was no significant ($p$'s $> 0.05$) main effect for concussion history group or an age by

concussion history interaction, yet there was a significant ($p < 0.05$) main effect for age group on the Falls Risk Assessment tool. The 60yo concussed group reported the highest average score on the Falls Efficacy Scale, at 10.75 out of a maximum of 100. The average score for community living elderly who are not afraid of falling was 26.42 (Tinetti et al., 1990). Only one of the participants per 60yo concussion groups reported a fear of falling. Additionally, the 60yo control (6%) and 60yo concussed (13%) groups reported falling per year at smaller percentages than the rates observed in community dwelling (30%) (O'Loughlin, Robitaille, Boivin, & Suissa, 1993). This suggests the 60yo concussion groups that participated in the following investigations appear to have less risk of falling than a community dwelling population. The Falls Risk Assessment Tool positively predicts (.57 predictive value) a fall in the next six months when three or more of the risk factors are present (Nandy et al., 2004). The Falls Risk Assessment Data from this sample shows that only the 60yo concussed sample producing three of the risk factors. However, one participant is responsible for two of the three predictive values. This suggests that none of the concussion groups in the following investigation are at risk of falling.

*Symptom and Migraine Report*

There were no significant ($p's > 0.05$) main effects for age group, concussion history group, or age by concussion history interaction for either the Post-Concussion Symptom Scale or the Migraine Report. There appears to be a trend of the previously concussed reporting more symptoms, though never significantly more. The lack of observed significance between groups, within age, is likely due to the standard deviation values associated with the previously concussed age groups, particularly in the 20yo and 60yo concussed groups. Previous literature provides evidence for there being a link to long-term concussions and symptoms reporting (B. L.

Brooks et al., 2013; Collins et al., 1999). Based on the findings of this investigation, it appears

there are no significant differences between groups, within age, for symptom reporting. The 20yo

concussion group reported the highest rates of suffering migraine headaches; however they are

not significantly different from the 20yo control group. The most common persistent symptom of

concussion is headache (Gaetz et al., 2000). Both history of concussion and anxiety/stress are

thought to be factors associated with determining the duration of persistent symptoms (W. D.

Gouvier, B. Cubic, G. Jones, P. Brantley, & Q. Cutlip, 1992). Interestingly, only 19% of the

20yo control group and 11% of the 20yo concussed group reported anxiety on the graded

symptom checklist. Participants were instructed to complete the graded symptom checklist based

on how they were feeling at the moment of filling out the questionnaire. Consequently, the

graded symptom checklist might not represent symptom traits, but rather symptoms states.

**Table 3.1 Participant Demographics**

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | Control (n = 27) | Concussed (n = 19) | Control (n = 15) | Concussed (n = 4) | Control (n = 16) | Concussed (n = 8) |
| Sex (% male) | 44 | 47 | 47 | 75 | 44 | 88 |
| Age (yrs) | 23.41(8.09) | 20.26(1.63) | 45.13(3.38) | 47.25(3.50) | 63.50(3.52) | 63.75(5.04) |
| Height (cm) | 171.94(8.94) | 173.19(9.74) | 171.33(11.67) | 180.98(10.24) | 169.62(10.02) | 174.94(6.29) |
| Weight (Kg) | 68.59(11.94) | 68.74(14.75) | 79.80(11.14) | 94.69(20.06) | 78.35(14.17) | 78.36(8.38) |
| Previous # Concussions | . | 1.63(.68) | . | 2.75(1.71) | . | 2.63(1.92) |
| Time Since (yrs) | . | 5.64(3.20) | . | 23.71(13.22) | . | 49.49(5.53) |

**3.1 AXON by CogState**
<u>AIM 1</u>

To determine the long-term effect of sport concussion on cognition and reaction time across age, as measured by a clinical neurocognitive concussion assessment.

> <u>Hypothesis 1A:</u> Individuals with a history of concussion will exhibit poorer cognitive based scores (e.g. attention, learning, etc.) than matched controls, particularly with increased age.

> <u>Hypothesis 1B:</u> Individuals with a history of concussion will exhibit longer reaction times than matched controls, particularly with increased age.

<u>Significance of Aim</u>

The disconnection hypothesis suggests that cognitive decline in normal aging is a result of functional degradation between systems of the brain. Since concussion is defined as a functional injury, testing the long-term functional ability of concussion groups, across age, could predict greater motor control deficits in the previously concussed group (O'Sullivan et al., 2001). Provided the EEG and TMS data, concussion history appears to have a chronic effect on cognitive processes associated with attention and decision making (Broglio et al., 2009; De Beaumont et al., 2007; De Beaumont et al., 2009; Pontifex et al., 2009). The AXON by CogState provides variants of reaction time and memory components that influence performance on more complex tasks (e.g. obstacle crossing and continuous tracking).

<u>Participants</u>

Data from 85 participants were included in these analyses. Participants were categorized into one of six groups based the individual's self-report of a previous diagnosis of concussion and age

(18-30, 40-50, 60-70 years). Participants were excluded if their most recent concussion occurred after they were 18yo. There was no attempt to grade injury severity because of the known bias in injury recall and the abundant number and inconsistency of grading scales (Aubry et al., 2002).

Testing Protocol

Each participant completed the participant questionnaire detailing education, health, and concussion history. Functional cognitive performance was evaluated using the Axon concussion assessment. Axon is a computerized cognitive test that consists of four test modules: detection (simple reaction time), identification (choice reaction time), one card learning (working memory), and one back speed/accuracy (attention and working memory). The test is based on rapidly tapping a computer key in response to images of playing cards displayed on the computer monitor. Greater detail of the AXON by CogState test has been previously reported (Collie, Maruff, Makdissi, et al., 2003; James T Eckner et al., 2011).

Measures

The AXON by CogState provides five output scores: processing speed, attention, learning, working memory speed, and working memory accuracy. Higher output scores indicate better performance.

Statistical Analysis

A multivariate ANOVA was used to assess group, concussion history by age, for the test score differences. Means and standard deviations were calculated for each AXON output variables and one-way ANOVAs were used to compare means between concussion history and control groups,

within age. Any age related difference was attributed to the known natural differences of motor performance across age. Where appropriate, *post-hoc* Bonferroni multiple comparisons corrections were implemented. Effect sizes were calculated to assess the performance differences within group. Effect sizes are interpreted as weak ($<.5$), moderate ($.50-.79$), or strong ($\geq.8$) as described by Cohen (1977). *Post hoc* and *a priori* power was calculated using the sample sizes and Cohen's d effect sizes. The α-level was set a priori at $p \leq .05$. Statistical analysis was completed using the Statistical Package for Social Sciences (SPSS) software version 21 (SPSS, Inc., Chicago, IL, USA).

Results
Means and standard deviations can be found on Table C.i, in Appendix C. There were no significant main effects for age group, concussion history group, or age by concussion history interaction (p's $> 0.05$).

*Processing Speed*

Within age means and standard deviations of concussion history and control groups yielded effect sizes of .30 between the 20yo groups, .07 between the 40yo groups, and .38 between the 60yo groups. *Post hoc* power analysis yielded a value of .16 for the 20yo groups, .05 for the 40yo groups, and .12 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 1100 participants per group would be needed to reach a power of .80 for any future investigation.

*Attention*

Within age means and standard deviations of concussion history and control groups yielded effect sizes of .11 for the 20yo groups, .52 for the 40yo groups, and .31 for the 60yo groups. *Post*

*hoc* power analysis yielded a value of .07 for the 20yo groups, .14 for the 40yo groups, and .10

for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated

an average of 500 participants per group would be needed to reach a power of .80 for any future

investigation.

*Learning*

Within age means and standard deviations of concussion history and control groups yielded

effect sizes of .22 for the 20yo groups, .60 for the 40yo groups, and .31 for the 60yo groups. *Post*

*hoc* power analysis yielded a value of .11 for the 20yo groups, .17 for the 40yo groups, and .1 for

the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an

average of 180 participants per group would be needed to reach a power of .80 for any future

investigation.

*Working Memory Accuracy*

Within age means and standard deviations of concussion history and control groups yielded

effect sizes of .21 for the 20yo groups, 1.13 for the 40yo groups, and .41 for the 60yo groups.

*Post hoc* power analysis yielded a value of .11 for the 20yo groups, .47 for the 40yo groups, and

.13 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes

indicated an average of 160 participants per group would be needed to reach a power of .80 for

any future investigation.

*Working Memory Speed*

Within age means and standard deviations contrasts of concussion history and control groups

yielded effect sizes of .53 for the 20yo groups, 1.16 for the 40yo groups, and .64 for the 60yo

groups. *Post hoc* power analysis yielded a value of .40 for the 20yo groups, .49 for the 40yo

groups, and .25 for the 60yo groups. *Post hoc* sample size and power estimates with these effect

sizes indicated an average of 40 participants per group would be needed to reach a power of .80

for any future investigation.

Using the sample sizes, the smallest effect sizes that could be observed per group, assuming a

power of .80, are .86 between the 20yo groups, 1.67 between the 40yo groups, and 1.39 between

the 60yo groups. These hypothetical effect sizes would be associated with significant concussion

history group differences, within age. There were a few moderate and large effect sizes in the

current observations, particularly with the two older populations; however, the effects sizes

exceeded .80 once. Together, these values suggest that adolescent concussion has little effect on

cognitive and motor performance in this population.

Discussion

The purpose of this investigation was to determine if sustaining a concussion during adolescence

has a significant effect on cognition and reaction time, as measured by a clinical neurocognitive

assessment. Previous literature suggests that there are electrophysiological effects, associated

with attention and decision making processes, observed between three and 30 years post-

concussion recovery (Broglio et al., 2009; De Beaumont et al., 2007; De Beaumont et al., 2009;

Pontifex et al., 2009). Axon by CogState provides a quintet of clinical outcome measures and

these findings indicate no significant, long-term effects associated with concussion on an

individual's processing speed, attention, learning, working memory accuracy, or working

memory speed.

These findings mimic previous investigations, which report no significant long-term differences, as measured by clinical assessment tools in college athletes (Broglio, Ferrara, Piland, & Anderson, 2006; Collie, McCrory, & Makdissi, 2006). In addition, most acutely concussed individuals return to their baseline scores on clinical measures in seven to ten days post-concussion (McCrory et al., 2013). Should long term cognitive impairment result from concussion, these and previous null findings could result from clinical neurocognitive assessments not being sensitive enough to measure the subtle changes in the previously concussed. Neurocognitive assessments were designed to be used in the clinical diagnosis process of a concussion, with a goal of seeing a concussed athlete return to a baseline score. While being used to track the concussion recovery process of an athlete, neurocognitive scores generally return to baseline within two weeks of injury (McCrory et al., 2013). An alternate rationale would be that there are no significant long-term effects of a concussion, sustained during adolescence, on full system cognition, at any age, as measured by clinical neurocognitive concussion assessment.

Conclusion
This investigation is the first to use a standard clinical neurocognitive concussion assessment, AXON by CogState, to determine if there are significant long-term clinical effects of a concussion on cognition and reaction time, across age. Based on the current findings, there are no apparent long-term effects of sustaining a sport concussion, during adolescence, on processing speed, attention, learning, working memory accuracy, or working memory speed. There were no significant findings, and one effect size exceeding a Cohen's d value of 1. Therefore, the Accelerated Decline Hypothesis is not supported from the findings of this investigation (Broglio et al., 2012).

**3.2 Gait Performance**

<u>AIM 2</u>

To determine the long-term effect of sport concussion on gait performance across age, as measured by a spatio-temporal, kinematic, and toe clearance variables.

> <u>Hypothesis 2A</u>: Individuals with a concussion history will exhibit slower gait velocities, greater step width, smaller stride lengths, and more percentage of cycle time in double stance compared to controls. These discrepancies will diverge across ages and concussion history.

> <u>Hypothesis 2B</u>: Individuals with a concussion history will exhibit reduced gait hip extension and reduced ankle plantarflexion compared to controls. These discrepancies will diverge across ages and concussion history.

> <u>Hypothesis 2C</u>: These alterations in gait performance will lead to an observable proactive strategy in reducing the risk of falling in the concussed group at an advanced age. Individuals with a concussion history will exhibit a greater obstacle toe clearance than controls. These discrepancies will diverge across ages and concussion history.

<u>Significance of Aim</u>

The disconnection hypothesis suggests that cognitive decline in normal aging is a result of functional degradation between systems of the brain (O'Sullivan et al., 2001). Since concussion is defined as a functional injury, testing the long-term functional ability of both groups, across age, could detect greater motor control deficits in the previously concussed group. Both balance and gait alterations have been observed in the young, previously concussed population (Martini

et al., 2011; Sosnoff et al., 2011). If these altered motor performances change with age remains unknown.

Older adults have been shown to use a greater lead toe clearance in obstacle avoidance, which is associated with increasing chances of successful obstacle crossing (H.-C. Chen et al., 1991; T. W. Lu, H. L. Chen, & S. C. Chen, 2006; Patla & Goodale, 1996; Sparrow et al., 1996). In addition to greater toe clearance, the older population altered fewer joints during obstacle crossing, perceived as an attempt to limit the difficulty of the obstacle crossing task (T. W. Lu et al., 2006). Kinematically, the most consistent and greatest difference between healthy young and old occurs at the hip and ankle joints (Kerrigan et al., 1998; T. W. Lu et al., 2006). Trips and falls are associated with activities (e.g. steps and obstacle crossing) that require hip extension and ankle plantarflexion (Kerrigan et al., 1998). Increasing the motor control demands of gait by placing an obstacle is thought to increase the risk of falling (Austin, Garrett, & Bohannon, 1999). Imbalance in the elderly population is an important factor in predicting falls and hip fracture (Greenspan et al., 1998).

Participants

Data from 61 participants were included in these analyses. Data from the remaining 28 participants were lost to equipment and data collection errors. For example, the capture volume was large, and constant fine-tuning of camera focus still didn't completely clear the trials of error. In addition, time became an issue with data collection, not permitting a full three quality trials for each condition. This is particularly true with gait conditions 1 and 2, the unobstructed walks. Participants were categorized into one of six groups based the individual's self-report of a

previous diagnosis of concussion and age (18-30, 40-50, 60-70 years). Participants were

excluded if they sustained a concussion after they were 18 years old. No attempt to grade injury

severity was made because of the known bias in injury recall and the abundant number and

inconsistency of grading scales (Aubry et al., 2002).

Testing Protocol

During participation, each participant completed the questionnaire detailing education, health,

and concussion history. The gait assessment was completed using motion capture analysis of

four walking conditions. Each condition was 10m long and completed at a self-selected pace.

Condition 1 was an unobstructed walk; condition 2 was an unobstructed walk while completing

Brooks' Mental Task (described below); condition 3 was an obstructed walk with the obstacle at

20% of the participant's average leg length; and condition 4 was an obstructed (20% of leg

length) walk while completing the Brooks' Mental Task. The obstacle was completely visible to every participant for both conditions 3 and 4. Participants were asked to complete 5 trials of each condition and instructed to walk as naturally as possible while performing the Brooks'
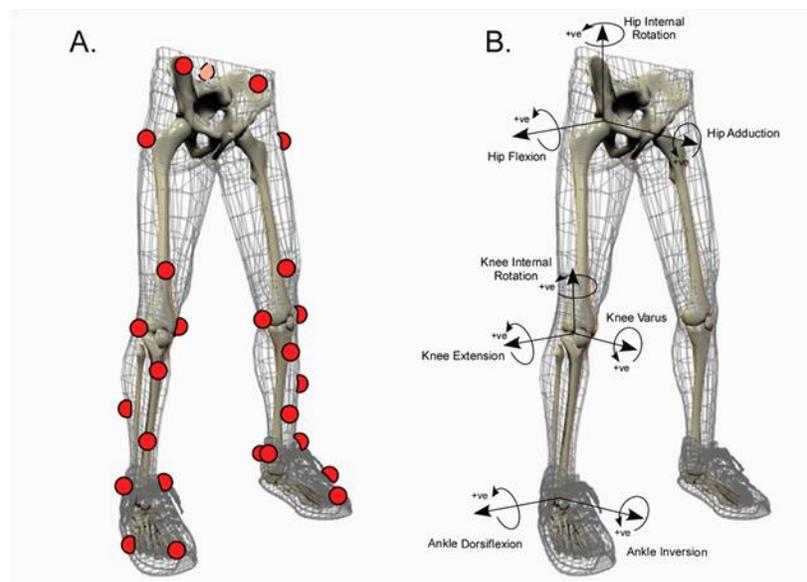


Figure 3.1: Marker set for movement analysis (**A**) and model for skeleton kinematics and kinetics (**B**) of the right leg. Adopted from Borotikar et al., (2008).

Mental Task to the best of their abilities. Kinematic data processing: The three-dimensional

biomechanical data were collected for the hip, and ankle joints using a Vicon system (Vicon Corp, Boulder, CO, USA) sampling at 240Hz synchronized with analog data sampling at 1200Hz. The joint rotations were defined based on 32 precisely located reflective markers (anterior and posterior iliac spines, iliac crest, greater trochanter, lateral and distal thigh, medial and lateral femoral epicondyles, tibial tuberosity, lateral and distal shank, medial and lateral malleoli, calcaneus, base of the fifth metatarsal, and top of the first toe) for each limb (Figure 3.1). An initial static trial of each participant aligned with the laboratory coordinate system was recorded. From which a kinematic model of seven skeletal segments (pelvis, bilateral thigh, shank, and foot segments) and 24 degrees of freedom was created using Visual 3D version 5.0 software (C-Motion, Rockville, MD, USA) (Lepley, 2014; McLean, Lipfert, & van den Bogert, 2004). Rotations were calculated using the Cardan rotation sequence and was expressed relative to each subject's relative neutral (static) position (Cole, Nigg, Ronsky, & Yeadon, 1993; Lepley, 2014; McLean et al., 2007).

Toe clearance was measured as the distance (cm) between the marker on the top of the first toe and the top of the obstacle crossbar. The obstacle was comprised of two uprights, a lower cross support, and an upper crossbar. The obstacle was marked with 15 permanently placed reflective markers (three on each upright, nine on the crossbar - three at each distal end and three in the middle). The position of the top of the crossbar was calculated from the average position of the nine crossbar markers.

The Brooks' Mental Task requires the participant to remember and recall the order and location of eight sequential numbers in a 4x4 grid that are presented via an audio recording (L. R. Brooks,

1967). An example sequence played for the participant would be: "In the starting square put a 1, in the square to the right put a 2, in the square below put a 3, in the next square beneath put a 4, in the square to the right put a 5." The participant was instructed to recall the number sequence and location while walking for conditions 2 and 4. Additionally, the Brooks' Mental Task will be completed during a seated task condition and treated as a baseline.

<u>Statistical Analysis:</u> Means and standard deviations were calculated for the spatio-temporal (e.g. velocity, stride length, etc.), kinematic (e.g. hip extension angle), toe clearance, and Brooks' Mental Task variables observed under each walking condition. To determine the long-term effect of concussion on gait performance, multivariate ANOVAs were used to assess group (concussion history by age) for gait variable differences. One-way ANOVAs were used to compare with an age concussion history group differences. Bonferroni post-hoc corrections were implemented as necessary. Any age related difference was attributed to the known natural differences of motor performance across age. Effect sizes were calculated to assess the within age differences between concussion group performance. Effect sizes are interpreted as weak (<.5), moderate (.50-.79), or strong (≥.8) as described by Cohen (1977). Post hoc and *a priori* power was calculated using the sample sizes and Cohen's d effect sizes. The α-level was set a priori at $p \leq .05$. Statistical analysis was completed using the Statistical Package for Social Sciences (SPSS) software version 21 (SPSS, Inc., Chicago, IL, USA).

<u>Results</u>

**Temporal and Spatial Gait Parameters**

Means and standard deviations can be found in Tables D.i through D.iv, in Appendix D.

Condition 1 yielded significant (p's < 0.05) main effects for age group and age by concussion history interaction, but not for concussion history group (p > 0.05). Condition 2 yielded no significant (p's > 0.05) main effects for age group, concussion history group, or age by concussion history interaction. Condition 3 yielded significant main effects for age group, concussion history group, and age by concussion history interaction (p's < 0.05). Condition 4 yielded significant main effects for age group and concussion history group (p's < 0.05), but not an age by concussion history interaction (p > 0.05).

*Gait Velocity*

There were no significant (p's > 0.05) main effects for age group, concussion history group, or age by concussion history interaction for gait velocity in conditions 1 or 2. Conditions 3 and 4 yielded a significant age group difference for velocity (p's < .004). Condition 1 means and standard deviations of concussion history and control groups yielded effect sizes of .21 for the 20yo groups, .14 for the 40yo groups, and .85 for the 60yo groups. *Post hoc* power analysis yielded a value of .07 for the 20yo groups, .05 for the 40yo groups, and .29 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 400 participants per group would be needed to reach a power of .80.

Condition 2 within age means and standard deviations of the concussion history and control groups yielded effect sizes of .41 for the 20yo groups and .00 for the 60yo groups. *Post hoc* power analysis yielded a value of .10 for the 20yo groups and .05 for the 60yo groups. *Post hoc*

sample size and power estimates with these effect sizes indicated an average of 1000 participants per group would be needed to reach a power of .80.

Condition 3 means and standard of the concussion history and control groups yielded effect sizes of .12 for the 20yo groups, .03 for the 40yo groups, and 1.53 for the 60yo groups. *Post hoc* power analysis yielded a value of .06 for the 20yo groups, .05 for the 40yo groups, and .88 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 6000 participants per group would be needed to reach a power of .80.

Condition 4 means and standard deviations of the concussion history and control groups yielded effect sizes of .10 for the 20yo groups, 1.07 for the 40yo groups, and .63 for the 60yo groups. *Post hoc* power analysis yielded a value of .06 for the 20yo groups, .43 for the 40yo groups, and .24 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 550 participants per group would be needed to reach a power of .80.

*Step Width*

There were no significant (p's > 0.05) main effects for age group, concussion history group, or age by concussion history interaction for step width in conditions 1, 2 or 4. Condition 3 yielded a significant age group difference for step width (p < .004). Condition 1 means and standard deviations of concussion history and control groups yielded effect sizes of .15 for the 20yo groups, .70 for the 40yo groups, and .14 for the 60yo groups. *Post hoc* power analysis yielded a value of .06 for the 20yo groups, .12 for the 40yo groups, and .06 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 510 participants per group would be needed to reach a power of .80.

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Condition 2 means and standard deviations of the concussion history and control groups yielded effect sizes of .21 for the 20yo groups and 1.31 for the 60yo groups. *Post hoc* power analysis yielded a value of .06 for the 20yo groups and .29 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 184 participants per group would be needed to reach a power of .80.

Condition 3 means and standard deviations of the concussion history and control groups yielded effect sizes of .13 for the 20yo groups, .21 for the 40yo groups, and .25 for the 60yo groups. *Post hoc* power analysis yielded a value of .07 for the 20yo groups, .06 for the 40yo groups, and .08 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 510 participants per group would be needed to reach a power of .80.

Condition 4 means and standard deviations of the concussion history and control groups yielded effect sizes of .33 for the 20yo groups, .83 for the 40yo groups, and .37 for the 60yo groups. *Post hoc* power analysis yielded a value of .19 for the 20yo groups, .28 for the 40yo groups, and .11 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 95 participants per group would be needed to reach a power of .80.

*Stride Length*

There were no significant (p's > 0.05) main effects for age group, concussion history group, or age by concussion history interaction for stride length in conditions 1 or 2. Conditions 3 and yielded significant differences for both age group and concussion history group for stride length (p's < .004). Condition 1 means and standard deviations of the concussion history and control

groups yielded effect sizes of .45 for the 20yo groups, 1.87 for the 40yo groups, and .96 for the 60yo groups. *Post hoc* power analysis yielded a value of .15 for the 20yo groups, .51 for the 40yo groups, and .35 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 35 participants per group would be needed to reach a power of .80.

Condition 2 means and standard deviations of the concussion history and control groups yielded effect sizes of .63 for the 20yo groups and .67 for the 60yo groups. *Post hoc* power analysis yielded a value of .17 for the 20yo groups and .11 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 24 participants per group would be needed to reach a power of .80.

No within age group significant differences (p's > 0.05) were observed between control and concussion history groups. Condition 3 means and standard deviations of the concussion history and control groups yielded effect sizes of .15 for the 20yo groups, 1.45 for the 40yo groups, and 1.27 for the 60yo groups. *Post hoc* power analysis yielded a value of .07 for the 20yo groups, .67 for the 40yo groups, and .74 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 240 participants per group would be needed to reach a power of .80.

No within age group significant differences (p's > 0.05) were observed between control and concussion history groups. Condition 4 means and standard deviations of the history and control groups yielded effect sizes of .36 for the 20yo groups, 1.66 for the 40yo groups, and .71 for the

60yo groups. *Post hoc* power analysis yielded a value of .22 for the 20yo groups, .79 for the 40yo groups, and .29 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 54 participants per group would be needed to reach a power of .80.

*Percent of Time Spent in Double Stance*

There were no significant (p's > 0.05) main effects for age group, concussion history group, or age by concussion history interaction for percent of time in double stance in conditions 1, 2 or 4. Condition 3 yielded a significant age group difference for percent of time in double stance (p < .004). Condition 1 means and standard deviations of the concussion history and control groups yielded effect sizes of .52 for the 20yo groups, .86 for the 40yo groups, and .34 for the 60yo groups. *Post hoc* power analysis yielded a value of .19 for the 20yo groups, .15 for the 40yo groups, and .09 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 73 participants per group would be needed to reach a power of .80.

Condition 2 means and standard deviations of the concussion history and control groups yielded effect sizes of .47 for the 20yo groups and .31 for the 60yo groups. *Post hoc* power analysis yielded a value of .12 for the 20yo groups and .06 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 119 participants per group would be needed to reach a power of .80.

Condition 3 means and standard deviations of the concussion history and control groups yielded effect sizes of .08 for the 20yo groups, .88 for the 40yo groups, and .82 for the 60yo groups. *Post*

*hoc* power analysis yielded a value of .06 for the 20yo groups, .31 for the 40yo groups, and .39 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 830 participants per group would be needed to reach a power of .80.

Condition 4 standard deviations of the concussion history and control groups yielded effect sizes of .46 for the 20yo groups, .25 for the 40yo groups, and .59 for the 60yo groups. *Post hoc* power analysis yielded a value of .33 for the 20yo groups, .07 for the 40yo groups, and .22 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 125 participants per group would be needed to reach a power of .80.

**Gait Kinematics**

Means and standard deviations can be found in Tables D.v through D.viii, in Appendix D. Condition 1 yielded no significant main effects for age group, for concussion history group, or an age by concussion history interaction ($p$'s $> 0.05$). Condition 2 yielded significant ($p$'s $< 0.05$) main effects for age group and concussion history group, but not an age by concussion history interaction ($p > 0.05$). Condition 3 lead limb yielded a significant ($p < 0.05$) main effect for concussion history group, but not for age group or for the age by concussion history interaction ($p$'s $> 0.05$). Condition 4 lead limb yielded a significant main effect for age group ($p < 0.05$), but not for concussion history group or for the age by concussion history interaction ($p$'s $> 0.05$). Condition 3 trail limb yielded no significant main effects for age group, concussion history group, or for the age by concussion history interaction ($p$'s $> 0.05$). Condition 4 trail limb yielded a significant ($p < 0.05$) main effect for age group, but not for concussion history group or for the age by concussion history interaction ($p$'s $> 0.05$).

*Ankle Plantarflexion*

There were no significant (p's > 0.05) main effects for age group, concussion history group, or age by concussion history interaction for ankle plantarflexion in conditions 1, 3 or 4. Condition 2 yielded a significant age group difference for ankle plantarflexion (p < .008). Condition 1 means and standard deviations of the concussion history and control groups yielded effect sizes of .71 for the 20yo groups, .10 for the 40yo groups, and .41 for the 60yo groups. *Post hoc* power analysis yielded a value of .25 for the 20yo groups, .05 for the 40yo groups, and .09 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 570 participants per group would be needed to reach a power of .80.

Condition 2 one-way ANOVAs of the concussion history and control groups yielded effect sizes of .42 for the 20yo groups and .58 for the 60yo groups. *Post hoc* power analysis yielded a value of .10 for the 20yo groups and .10 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 69 participants per group would be needed to reach a power of .80.

Condition 3 lead limb one-way ANOVAs of the concussion history and control groups yielded effect sizes of .33 for the 20yo groups, .70 for the 40yo groups, and .25 for the 60yo groups. *Post hoc* power analysis yielded a value of .16 for the 20yo groups, .20 for the 40yo groups, and .08 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 144 participants per group would be needed to reach a power of .80.

Condition 4 lead limb one-way ANOVAs of the concussion history and control groups yielded effect sizes of .48 for the 20yo groups, .06 for the 40yo groups, and .06 for the 60yo groups. *Post hoc* power analysis yielded a value of .35 for the 20yo groups, .05 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 2930 participants per group would be needed to reach a power of .80.

Condition 3 trail limb one-way ANOVAs of the concussion history and control groups yielded effect sizes of .68 for the 20yo groups, .32 for the 40yo groups, and .03 for the 60yo groups. *Post hoc* power analysis yielded a value of .51 for the 20yo groups, .08 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 5875 participants per group would be needed to reach a power of .80.

Condition 4 trail limb one-way ANOVAs of the concussion history and control groups yielded effect sizes of .36 for the 20yo groups, .95 for the 40yo groups, and .48 for the 60yo groups. *Post hoc* power analysis yielded a value of .22 for the 20yo groups, .35 for the 40yo groups, and .16 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 70 participants per group would be needed to reach a power of .80.

*Hip Extension*

There were no significant (p's > 0.05) main effects for age group, concussion history group, or age by concussion history interaction for hip extension in conditions 1, 3 or 4. Condition 2 yielded a significant concussion history group difference for hip extension (p < .008). Condition 1 means and standard deviations of the concussion history and control groups yielded effect sizes

of .67 for the 20yo groups, .89 for the 40yo groups, and .16 for the 60yo groups. *Post hoc* power analysis yielded a value of .27 for the 20yo groups, .16 for the 40yo groups, and .06 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 224 participants per group would be needed to reach a power of .80.

No within age group significant differences (p's > 0.05) were observed between control and concussion history groups. Condition 2 means and standard deviations of the concussion history and control groups yielded effect sizes of .83 for the 20yo groups and 1.63 for the 60yo groups. *Post hoc* power analysis yielded a value of .27 for the 20yo groups and .41 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 16 participants per group would be needed to reach a power of .80.

Condition 3 lead limb means and standard deviations of the concussion history and control groups yielded effect sizes of .49 for the 20yo groups, .69 for the 40yo groups, and .02 for the 60yo groups. *Post hoc* power analysis yielded a value of .30 for the 20yo groups, .20 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 13115 participants per group would be needed to reach a power of .80.

Condition 4 lead limb means and standard deviations of the concussion history and control groups yielded effect sizes of .65 for the 20yo groups, .55 for the 40yo groups, and .50 for the 60yo groups. *Post hoc* power analysis yielded a value of .57 for the 20yo groups, .15 for the 40yo groups, and .17 for the 60yo groups. *Post hoc* sample size and power estimates with these

effect sizes indicated an average of 52 participants per group would be needed to reach a power of .80.

Condition 3 trail limb means and standard deviations of the concussion history and control groups yielded effect sizes of .54 for the 20yo groups, .83 for the 40yo groups, and .17 for the 60yo groups. *Post hoc* power analysis yielded a value of .35 for the 20yo groups, .27 for the 40yo groups, and .06 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 208 participants per group would be needed to reach a power of .80.

Condition 4 trail limb means and standard deviations of the concussion history and control groups yielded effect sizes of .53 for the 20yo groups, .50 for the 40yo groups, and .34 for the 60yo groups. *Post hoc* power analysis yielded a value of .41 for the 20yo groups, .13 for the 40yo groups, and .10 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 86 participants per group would be needed to reach a power of .80.

**Toe Clearance**

Means and standard deviations can be found in Figures D.i through D.iv, in Appendix D. Condition 3 yielded a significant ($p < 0.05$) main effect for concussion history group, but not for age group or for the age by concussion history interaction (p's $> 0.05$). Condition 4 yielded no significant main effects for age group, concussion history group, or for the age by concussion history interaction (p's $> 0.05$).

There were no significant (p's > 0.008) main effects for age group, concussion history group, or an age by concussion history interaction for either limb's toe clearance in condition 3. Condition 3 lead limb means and standard deviations of the concussion history and control groups yielded effect sizes of 1.42 for the 20yo groups, .91 for the 40yo groups, and .26 for the 60yo groups. *Post hoc* power analysis yielded a value of .98 for the 20yo groups, .10 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 88 participants per group would be needed to reach a power of .80.

Condition 4 lead limb means and standard deviations of the concussion history and control groups yielded effect sizes of .83 for the 20yo groups, .32 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* power analysis yielded a value of .78 for the 20yo groups, .10 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 2153 participants per group would be needed to reach a power of .80.

Condition 3 trail limb means and standard deviations of the concussion history and control groups yielded effect sizes of .68 for the 20yo groups, .11 for the 40yo groups, and .39 for the 60yo groups. *Post hoc* power analysis yielded a value of .51 for the 20yo groups, .05 for the 40yo groups, and .16 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 480 participants per group would be needed to reach a power of .80.

Condition 4 trail limb means and standard deviations of the concussion history and control groups yielded effect sizes of .63 for the 20yo groups, .09 for the 40yo groups, and .13 for the 60yo groups. *Post hoc* power analysis yielded a value of .55 for the 20yo groups, .05 for the 40yo groups, and .06 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 970 participants per group would be needed to reach a power of .80.

Using the current sample sizes, the smallest effect sizes, with statistical significance, that could be observed per group, assuming a power of .80, are 1.19 between the 20yo groups, 2.22 between the 40yo groups, and 1.38 between the 60yo groups for condition 1. Condition 2 minimum effect sizes, under the same assumption are 1.43 between the 20yo groups, 10.03 between the 40yo groups, and 2.00 between the 60yo groups. Condition 3 minimum effect sizes, under the same assumption are .94 between the 20yo groups, 1.53 between the 40yo groups, and 1.32 between the 60yo groups. Condition 4 minimum effect sizes, under the same assumption are .89 between the 20yo groups, 1.56 between the 40yo groups, and 1.43 between the 60yo groups. The observed effect sizes rarely approached the minimum effect size for any parameter in any of the four conditions, let alone these estimated effect sizes associated with significant within age, concussion history group differences. This suggests that there is no substantive effect of concussion history on gait spatio-temporal, joint kinematics, or obstacle toe clearance in this previously concussed population when compared to a control population.

Discussion
The purpose of this investigation was to determine if sustaining a concussion during adolescence negatively affects gait spatio-temporal parameters, gait mechanics, and toe clearance in a

divergent patter, across ages. The spatio-temporal gait data shows a trend for the 60yo concussed group walking with greater velocity than the 60yo control group in condition 3, the obstructed walk condition. An additional trend to walking with greater velocity, the 60yo concussed group also walks with greater stride length in condition 4, the obstructed walk with mental task. The trend of quicker gait velocity in the 60yo concussed is likely being driven by the group's increased stride length, as compared to the 60yo control group. Increasing stride length is a known strategy to increased gait velocity, and this trend has been observed in an elderly population comparing slow and fast gait trials (Kerrigan et al., 1998; Neumann, 2002). Based on the gait velocity and stride length observations, the 60yo concussed group does not appear to be negatively affected by their concussion history. While the investigation does appear to be underpowered, the effect sizes between the previously concussed and control groups, within age, don't suggest a consistent pattern of more conservative performance in the previously concussed.

Aside from condition 1 values for percent of time in double stance, the 20yo previously concussed group has a trend of equal or greater time in double stance percentages, albeit with small effect sizes, and no significance. This mimics previous findings, with the interpretation presented that the 20yo concussed group was sub-consciously adopting a conservative gait strategy to prevent falls and additional injury (Martini et al., 2011). However, the trend for greater percentage of time in double stance is not observed in the 60yo concussed group, when compared to the 60yo control group. The inconsistency in observed percentage of time in double stance could indicate that any previously adopted gait strategy in the 20yo concussed group becomes irrelevant with age, or that there is no sub-conscious proactive strategy being adopted for any of the previously concussed groups at any age.

A previous investigation postulated that reducing ankle plantarflexion might be a proactive strategy in maintaining balance during gait, though there was not significantly reduced ankle plantaflexion between the concussion groups, within age (Kerrigan et al., 1998). A plethora of literature suggests that reduced hip extension during gait is prevalent in the elderly, with a greater reduction observed in an elderly population with recurrent falls (DiBenedetto et al., 2005; Kerrigan, Lee, Collins, Riley, & Lipsitz, 2001; Kerrigan et al., 2000; Kerrigan, Xenopoulos-Oddsson, Sullivan, Lelas, & Riley, 2003). The hypothesis that the previously concussed group would exhibit reduced hip extension and reduced ankle plantar flexion is not supported by the data. Neither the ankle plantarflexion angle nor the hip extension angle variables significantly differed between concussion groups, within age. Considering the null findings in the spatio-temporal data, the subsequent null findings in the kinematic data are not surprising. This is understood from the intimate relationship between spatio-temporal results and joint angles. For instance, any increase or decrease in gait velocity will have a direct effect on joint angles. The observations of the ankle plantarflexion and hip extension angles suggest that the previously concussed group does not alter joint angle as a means of adopting proactive strategy, in an attempt to reduce the risk of falls.

The toe clearance provided no significant differences either. Greater toe clearance has been described as a way to increase successful obstacle crossing (H. C. Chen, J. A. Ashton-Miller, N. B. Alexander, & A. B. Schultz, 1991; T.-W. Lu, H.-L. Chen, & S.-C. Chen, 2006; Patla, Rietdyk, Martin, & Prentice, 1996; Sparrow et al., 1996). A general trend of within age greater toe clearance of an obstacle that is completely visible was not observed. There is not a divergent increase between the previously concussed and control across age.

The observations from the Brooks' Mental Task scores and time to complete the Brooks' Mental Task were non-significant between concussion groups, within age. These results mimic a previous investigation utilizing the Brook's Mental Task, which observed no significant group differences between a 20yo previously concussed and a 20yo control group (Martini et al., 2011). Together with the results from the AXON investigation and previous literature, the results from the Brooks' task provide further evidence that there are no functional level cognitive differences in the previously concussed group (Broglio et al., 2006; Dretsch, Silverberg, & Iverson, 2015; Guskiewicz, Marshall, Broglio, Cantu, & Kirkendall, 2002).

All of the participants for this study were screened for pre-existing condition that would affect their gait pattern. Though, there were not any attempts to quantify current physical activity levels in any of the concussion groups, across age. Changes in gait performance could be masked if the previously concussed groups are currently more physically active than the control groups (Carter, Kannus, & Khan, 2001; Judge, Underwood, & Gennosa, 1993; Krebs, Jette, & Assmann, 1998). A future investigation could collect physical activity information to use as a covariate when comparing elderly, previously concussed populations. Collectively, these findings demonstrate subtle positive and negative differences in some gait variables, yet there is no consistent pattern of a divergent decrease of functional gait. This point builds on emerging literature, demonstrating both significant and non-significant differences in neurocognitive and motor performance between individuals with a concussion history and those without (Broglio et al., 2006; Chou et al., 2004; Collie, Makdissi, Maruff, Bennell, & McCrory, 2006; Dretsch et al., 2015; Iverson, Brooks, Lovell, & Collins, 2006; Martini et al., 2011; Sosnoff et al., 2011;

Thornton, Cox, Whitfield, & Fouladi, 2008; Wall et al., 2006). In addition to these observations,

and the data from survey on falling (Table B.i, Appendix B), these differences do not appear to

manifest in a clinically meaningful manner that compromises gait across age.


Conclusion
Previous investigations have reported subtle differences in cognitive and gait performance

between concussion groups (Broglio et al., 2006; Chou et al., 2004; Collins, Lovell, Iverson, Ide,

& Maroon, 2006; Iverson et al., 2006; Martini et al., 2011), however there does not appear to be

any negative relationship in gait or cognitive performance as the previously concussed age. As

the previously concussed age, there appears to be no evidence in their gait that would increase

their risk of falls, as compared to individuals without a concussion history. While not directly

tested, the results from this investigation do not support the Accelerated Decline Hypothesis

(Broglio et al., 2012).

**3.3 Motor Control Performance**

AIM 3: To determine the long-term effect of sport concussion on motor control across age, as measured by a continuous tracking task and a discrete temporal task.

Hypothesis 3A: Individuals with a concussion history will exhibit higher root mean squared errors on a continuous tracking task than controls. This difference will diverge with age and concussion history. Increased root mean squared errors are associated with worse performance.

Hypothesis 3B: Individuals with a concussion history will exhibit greater temporal variability to a known, auditory stimulus than controls. This difference will diverge with age and concussion history.

Significance of Aim: The disconnection hypothesis suggests that cognitive decline in normal aging is a result of functional degradation between systems of the brain (O'Sullivan et al., 2001). Since concussion is defined as a functional injury, testing the long-term functional ability of both groups, across age, could predict greater motor control deficits in the previously concussed group. Implicit motor learning reductions have been observed in young adults with a concussion history (13 months past most recent) (De Beaumont, Tremblay, Poirier, Lassonde, & Theoret, 2012). The continuous tracking task tests the implicit motor learning ability, an important factor in motor skill acquisition (Vidoni & Boyd, 2007; Wulf & Schmidt, 1997). This information could lead to rehabilitation techniques that could assist individuals with a concussion history in preventing instability and falls with age.

Participants: Data from 82 participants were included in these analyses. Participants were categorized into one of six groups based the individual's self-report of a previous diagnosis of

concussion and age (18-30, 40-50, 60-70 years). Participants were excluded if their most recent concussion occurred after 18 years old. There was no attempt to grade injury severity because of the known bias in injury recall and the abundant number and inconsistency of grading scales. Written informed consent was collected from each subject prior to testing.

Testing Protocol: Each participant completed the participant questionnaire detailing education, health, and concussion history. Both the continuous tracking and discrete temporal tasks were designed and administered using LabView 2011 software (National Instruments, Austin, Texas, USA). The continuous tracking task was completed with both the dominant hand and foot (without shoe) while seated. While seated, the heel of the foot was placed on the foot-tapping device (Figure 2, bottom) with the bulb secured between the top and bottom boards. The bulb was placed in the participant's hand for the hand trials. A pressure sensor (Omega.com; model PX209-015G5V; range of 0-15 psi with a 5V output) was connected to a rubber air bladder located under the pedal. The participant was asked to press as hard as possible on the pedal (i.e. maximum voluntary contraction or MVC) for 15seconds. The required force level (between 25 and 75% of MVC) was displayed on a computer monitor (Figure 3.2, top), which also served as feedback to the participant's efforts. Three trials were completed with both the hand and foot. Each trial lasted one and a half minutes, with three passes of the screen (30-seconds per pass). Each pass of the screen is broken into continuous, 10-second intervals. The first and third intervals are completely random within and across passes and trials. The second interval is identical within and across passes and trials. This allows for differentiating between skill acquisition and pattern recognition. Rest was permitted, as requested. Root mean square (RMS) values were calculated for each trial.

The discrete temporal task was completed with both the dominant hand and foot (without shoe) while seated. A pacing tone was played over speakers at either a one Hertz (one tone per second) or half a Hertz (one tone every two seconds) pace for a total of one minute. Participants were instructed to react to the designated pace by squeezing/depressing the air bladder as soon as the pace is heard. In six trials the pace was present for the full duration of the one-minute trial. For an additional six trials, the pacing mechanism ceased after the first 10 seconds, requiring the subject to continue the pace for the last 50 seconds. The un-paced trials allow detection of the consistency of the internal timing mechanism. The participant



Figure 3.2: Foot-tapping device. LabView based visuomotor task (top). Participant's heel touches back lip (bottom).

completed 12 trials (one set of paces and one set of un-paced) with the hand and 12 trials with the foot. Again, rest was permitted as requested.

Statistical Analysis: For the continuous tracking task, RMS values from the three trials were analyzed using two-way ANOVAs (concussion history by age). One-way ANOVAs were used to compare with an age concussion history group differences. Bonferroni post-hoc corrections were implemented as indicated. Effect sizes were calculated to assess the within group performance differences. Effect sizes are interpreted as weak ($<.5$), moderate ($.50-.79$), or strong ($\geq.8$) as described by Cohen (1977). *Post hoc* and *a priori* power was calculated using the sample sizes and Cohen's d effect sizes. The $\alpha$-level was set a priori at $p \leq .05$. Statistical analysis was

completed using the Statistical Package for Social Sciences (SPSS) software version 21 (SPSS, Inc., Chicago, IL, USA).

For the discrete timing task, mean and maximum values from the 24 trials were analyzed using two-way ANOVAs (concussion history by age). One-way ANOVAs were used to compare with an age concussion history group differences. Bonferroni post-hoc corrections were implemented as indicated. In addition, coefficient of variation was calculated to compare group variability. Effect sizes were calculated to assess the within group performance differences. Effect sizes are interpreted as weak (<.5), moderate (.50-.79), or strong (≥.8) as described by Cohen (1977). *Post hoc* and *a priori* power was calculated using the sample sizes and Cohen's d effect sizes. The α-level was set a priori at $p \leq .05$. Statistical analysis was completed using the Statistical Package for Social Sciences (SPSS) software version 21 (SPSS, Inc., Chicago, IL, USA).

Results

**Visuomotor Tracking**

 Means and standard deviations can be found on Figure E.i through E.iv, in Appendix E. The hand condition of the continuous tracking task yielded a significant (p's < 0.05) main effect for age group and an age by concussion history interaction, but not for concussion history group (p > 0.05). The foot condition of the continuous tracking task yielded a significant (p's < 0.05) main effect for concussion history group and an age by concussion history interaction, but not for age group (p > 0.05).

*Hand Performance, Skill Acquisition*

The skill acquisition portion of the continuous tracking task yielded a significant (p's < 0.05) main effect for age group and an age by concussion history interaction, but not for concussion history group (p > 0.05). There were no significant (p's > 0.05) differences between concussion history and control groups, within age. Means and standard deviations of the concussion history and control groups yielded effect sizes of .21 for the 20yo groups, .06 for the 40yo groups, and .04 for the 60yo groups. *Post hoc* power analysis yielded a value of .10 for the 20yo groups, .05 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 4840 participants per group would be needed to reach a power of .80.

*Hand Performance, Implicit Learning Recognition*

The implicit learning portion of the continuous tracking task yielded a significant (p's < 0.05) main effect for age group and an age by concussion history interaction, but not for concussion history group (p > 0.05). There were no significant (p's > 0.05) differences between concussion history and control groups, within age. Means and standard deviations of the concussion history and control groups yielded effect sizes of .24 for the 20yo groups, .09 for the 40yo groups, and .20 for the 60yo groups. *Post hoc* power analysis yielded a value of .12 for the 20yo groups, .05 for the 40yo groups, and .07 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 870 participants per group would be needed to reach a power of .80.

*Foot Performance, Skill Acquisition*

The skill acquisition portion of the continuous tracking task yielded a significant (p's < 0.05) main effect for age group and an age by concussion history interaction, but not for concussion history group (p > 0.05). There was one significant difference between concussion history and control groups, within age, for the 60yo group (*p < .001*). Means and standard deviations of the concussion history and control groups yielded effect sizes of .06 for the 20yo groups, .12 for the 40yo groups, and .40 for the 60yo groups. *Post hoc* power analysis yielded a value of .05 for the 20yo groups, .06 for the 40yo groups, and .13 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 1850 participants per group would be needed to reach a power of .80.

*Foot Performance, Implicit Learning*

The implicit learning portion of the continuous tracking task yielded a significant (p's < 0.05) main effect for age group and an age by concussion history interaction, but not for concussion history group (p > 0.05). There were no significant (p's > 0.05) differences between concussion history and control groups, within age. Means and standard deviations of the concussion history and control groups yielded effect sizes of .02 for the 20yo groups, .07 for the 40yo groups, and .42 for the 60yo groups. *Post hoc* power analysis yielded a value of .05 for the 20yo groups, .05 for the 40yo groups, and .13 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 14,000 participants per group would be needed to reach a power of .80.

Considering these results and the sample sizes, the minimum effect sizes to produce significant differences, while assuming a power a .80, are .87 between the 20yo groups, 1.69 between the

40yo groups, and 1.39 between the 60yo groups. The observed effect sizes never approached the minimum effect size for any parameter in any of the hand or foot conditions, suggesting that there is no substantive effect of concussion history on implicit learning or skill acquisition.

**Discrete Auditory Timing**

Means and standard deviations can be found on Tables E.i through E.iv, in Appendix E. The 1Hz condition of the hand yielded significant (p's > 0.05) main effects for age group, concussion history group, and age by concussion history interaction. The .5Hz condition of the hand yielded a significant interaction age by concussion history interaction (p > 0.05), but no significant main effects for age group or concussion history group. The 1Hz condition of the foot yielded significant (p's < 0.05) main effects for age group and an age by concussion history interaction, but not a concussion history group main effect (p > 0.05). The .5Hz condition of the foot yielded a significant interaction age by concussion history interaction (p > 0.05), but no significant (p's > 0.05) main effects for age group or concussion history group.

*Hand Performance Paced*

There were significant differences between the 40yo (*p < .001*) and 60yo (*p < .001*) concussion history and control groups, within age, at the 1Hz pacing tone condition. Means and standard deviations of the 1Hz pacing tone yielded effect sizes of .02 for the 20yo groups, .23 for the 40yo groups, and .17 for the 60yo groups. *Post hoc* power analysis yielded a value of .05 for the 20yo groups, .07 for the 40yo groups, and .06 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 13000 participants per group would be needed to reach a power of .80.

There were significant differences between the 20yo (*p < .001*) and 40yo (*p < .001*) concussion history and control groups, within age, at the .5Hz pacing tone condition. Means and standard deviations of the .5Hz pacing tone yielded effect sizes of .17 for the 20yo groups, .28 for the 40yo groups, and .07 for the 60yo groups. *Post hoc* power analysis yielded a value of .08 for the 20yo groups, .08 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 1315 participants per group would be needed to reach a power of .80.

*Hand Performance Un-Paced*

There were significant differences between the 20yo (*p < .001*), 40yo (*p < .001*), and 60yo (*p < .001*) concussion history and control groups, within age, at the un-paced, 1Hz pacing tone condition. Means and standard deviations of the un-paced, 1Hz pacing tone yielded effect sizes of .13 for the 20yo groups, .49 for the 40yo groups, and .18 for the 60yo groups. *Post hoc* power analysis yielded a value of .07 for the 20yo groups, .13 for the 40yo groups, and .07 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 495 participants per group would be needed to reach a power of .80.

There were significant differences between the 20yo (*p < .001*), 40yo (*p < .001*), and 60yo (*p < .001*) concussion history and control groups, within age, at the un-paced, 5Hz pacing tone condition. Means and standard deviations of the .5Hz pacing tone yielded effect sizes of .18 for the 20yo groups, .26 for the 40yo groups, and .54 for the 60yo groups. *Post hoc* power analysis yielded a value of .09 for the 20yo groups, .07 for the 40yo groups, and .19 for the 60yo groups.

*Post hoc* sample size and power estimates with these effect sizes indicated an average of 740 participants per group would be needed to reach a power of .80.

*Foot Performance Paced*

There were significant differences between the 20yo ($p < .001$) concussion history and control groups in the 1Hz pacing tone condition. Means and standard deviations of the 1Hz pacing tone yielded effect sizes of .20 for the 20yo groups, .06 for the 40yo groups, and .12 for the 60yo groups. *Post hoc* power analysis yielded a value of .10 for the 20yo group, .05 for the 40yo groups, and .06 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 1950 participants per group would be needed to reach a power of .80.

There were significant differences between the 40yo ($p < .001$) concussion history and control groups in the .5Hz pacing tone condition. Means and standard deviations of the .5Hz pacing tone yielded effect sizes of .05 for the 20yo groups, .24 for the 40yo groups, and .17 for the 60yo groups. *Post hoc* power analysis yielded a value of .05 for the 20yo groups, .07 for the 40yo groups, and .06 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 2350 participants per group would be needed to reach a power of .80.

*Foot Performance Un-Paced*

There were significant differences between the 20yo ($p < .001$) and 40yo ($p < .001$) concussion history and control groups, within age, at the un-paced, 1Hz pacing tone condition. Means and

standard deviations of the un-paced, 1Hz pacing tone yielded effect sizes of .13 for the 20yo groups, .45 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* power analysis yielded a value of .07 for the 20yo groups, .12 for the 40yo groups, and .05 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 2430 participants per group would be needed to reach a power of .80.

There were significant differences between the 40yo (*p < .001*) and 60yo (*p < .001*) concussion history and control groups, within age, at the un-paced, .5Hz pacing tone condition. Means and standard deviations of the .5Hz pacing tone yielded effect sizes of .11 for the 20yo groups, .32 for the 40yo groups, and .69 for the 60yo groups. *Post hoc* power analysis yielded a value of .06 for the 20yo groups, .08 for the 40yo groups, and .40 for the 60yo groups. *Post hoc* sample size and power estimates with these effect sizes indicated an average of 490 participants per group would be needed to reach a power of .80.

Considering these results and the sample sizes, the minimum effect sizes to produce significant differences, while assuming a power a .80, are .87 between the 20yo groups, 1.69 between the 40yo groups, and 1.39 between the 60yo groups. The observed effect sizes never approached the minimum effect size for any parameter in any of the hand or foot conditions, suggesting that there is no substantive effect of concussion history on discrete auditory timing.

Discussion
The purpose of this investigation was to determine the long-term effect of sustaining a sport concussion, during adolescence, on upper and lower extremity motor control. The data from this project is the first to apply a continuous tracking or a discrete timing task to a previously

concussed population. Discrete timing has previously been implemented to differentiate those with and without Alzheimer's disease and similarities between CTE and Alzheimer's disease suggest that similar assessments may differentiate those with and without a concussion history (i.e. the discrete timing task) (Bangert & Balota, 2012; Baugh et al., 2012; Gavett et al., 2011; McKee et al., 2009; McKee et al., 2012). Based on the results from the continuous tracking and discrete timing, it appears as though there are no functional significant long-term effects on pattern recognition, skill acquisition or internal timing, as measured by a continuous tracking task or discrete auditory timing task. While some significant differences were present between concussion history and control groups, across age, these significant differences were neither consistent nor did they provide any substantial effect sizes.

These results mimic the previous two projects, as well as emerging literature (Broglio et al., 2006; Collie, Makdissi, et al., 2006; Dretsch et al., 2015; Iverson et al., 2006; Thornton et al., 2008; Wall et al., 2006). While significant differences exist between concussion history and control groups, there is no consistency suggesting that the previously concussed group suffers cognitive or motor performance declines with age. In addition, a pilot investigation evaluating concussed and non-concussed participant response times with the upper extremity observed large effect sizes, but no significance. Those data showed slightly greater response times for the 20yo previously concussed, but did so with smaller sample sizes. The lighted board only produced a response time (includes movement and reaction times), while the discrete timing task provides a truer reaction time. Had there been a significant difference between concussion groups, within age, the continuous tracking task could have indicated that a concussion history has an effect on implicit learning. Research in a stroke population used a similar paradigm, and saw

improvements in motor learning with practice on the continuous tracking task (S. K. Meehan, Linsdell, Handy, & Boyd, 2011).

Conclusion
As the previous investigations have reported, we found subtle differences between concussion groups, across age. However, there does not appear to be any negative relationship between adolescent concussion history and motor control as the previously concussed age. The Accelerated Decline Hypothesis is not supported by the findings of these visuomotor and discrete auditory timing investigations (Broglio et al., 2012). Given the current results, there appears to be no functional downfall to sustaining a concussion during adolescence on pattern learning, skill acquisition, or internal timing.

**Additional Statistical Analysis**

In addition to assessing the mean concussion group differences, within age, for the discrete

timing task, Coefficient of variation (Tables E.iii and E.iv, Appendix E) was assessed to look at

the group variability. The coefficient of variation is the ratio of the standard deviation to the

mean (i.e. $\sigma/\mu$). Importantly, as the mean approaches zero, the ratio will become very large. So

any group's mean that represents an anticipatory RT (e.g. RT approaching zero), will have large

coefficient of variation values. In addition, the coefficient of variation is designed for ratio

scales, not interval scales, so the interpretations for reaction time should be taken lightly. There

were no significant within age concussion group differences for any of the coefficients of

variation (p's > .05). Unlike the observations made comparing the discrete timing means of the

concussions groups, there appears to be a pattern in the un-paced conditions. The 60yo

concussed group has a much larger mean coefficient of variation than the 60yo control group, in

particular for the 1Hz paced trials of the hand and foot. This suggests that a history of concussion

might affect internal timing variability. Previous literature suggests that when compared to

healthy older adults, adults with the early signs of Alzheimer's disease had greater coefficients of

variation in a continuous tapping task that used an auditory pacer (Bangert & Balota, 2012). The

increased variability was considered as part of the interpretation that breakdowns in attention

disrupt error correction, particularly with perceived internal timing expectancies and the

demands of the environment (e.g. the timing task) (Bangert & Balota, 2012). Importantly, there

were significant differences between the healthy elderly and the early stages of Alzheimer's

disease elderly, an observation that does not occur between the 60yo control and 60yo concussed

samples. None of the effect sizes comparing the 60yo concussed groups exceed .69, with most in

the small range of Cohen's d values, suggesting that whatever effect a concussion history has on

internal timing is moderate.

Given the known relationship between number of previous concussions (e.g. one concussion

versus three or more) and cognitive and motor performance, we estimated the correlation

between cognitive and motor control variables and previous number of injuries in the previously

concussed groups (Collins et al., 1999; De Beaumont et al., 2007; Moser, Schatz, & Jordan,

2005; Theriault, De Beaumont, Tremblay, Lassonde, & Jolicoeur, 2011). The samples from the

current investigations only allowed for comparing individuals with one concussion history to

individuals with two or more. Point-Biserial correlations and 95% confidence intervals were

conducted between variables from gait (e.g. stance length), discrete timing, RMS, cognition (e.g.

AXON scores) assessments and number of previous concussions. The maximum self-reported

concussions were three for the 20yo, five for the 40yo, and six for the 60yo age groups. There

were 26 variables that presented a significant correlation between previous number of

concussions and each variable from the three projects. The table of correlation values and

concussion history group means can be found in Appendix F. the 20yo previously concussed

broke into nine with one concussion and eleven with two or more. The 40yo previously

concussed group had one with one concussion and three with two or more. The 60yo previously

concussed group had three with one concussion and five with two or more.


There were no significant correlations between previous number of concussions and any of the

axon variables. There was a significant correlation for gait velocity ($r = -.65$; $p < .05$) for the

20yo previously concussed group in gait condition 1, normal walk. The 60yo previously

concussed group presented significant correlation in step width ($r = .59$; $p < .05$) in gait condition

3, the obstructed walk; as well as gait velocity ($r = -.64$; $p < .05$), percent of time in double

stance ($r = .67$; $p < .05$), and stride length ($r = -.76$; $p < .01$) in gait condition 4, the obstructed

walk with mental task. The 60yo previously concussed group presented significant correlations in gait condition 1 maximum ankle plantar flexion ($r = -.96$; $p < .01$) and in lead limb maximum hip extension ($r = -.61$; $p < .05$) in condition 3. The 60yo previously concussed group also presented a significant correlation in lead limb toe clearance ($r = -.57$; $p < .05$) in condition 3. For the continuous tracking task, the 40yo previously concussed group presented significant correlations for waveform 2, pattern learning, in the hand ($r = .53$; $p < .01$), and waveforms 1 and 3, skill acquisition, in both the hand ($r = -.53$; $p < .01$) and foot($r = .40$; $p < .01$). In addition, the 60yo previously concussed group presented significant correlations for hand ($r = .24$; $p < .05$) and foot ($r = .30$; $p < .05$) of waveform 2, as well as the hand ($r = .30$; $p < .01$) and foot ($r = .26$; $p < .01$) of waveforms 1 and 3. Each of the previously concussed groups presented significant correlations between number of previous concussions and RT from the discrete timing task. The 20yo previously concussed group presented significant correlations for the paced 1Hz hand ($r = .22$; $p < .01$) and foot($r = -.06$; $p < .01$) conditions, as well as the un-paced 1Hz hand ($r = .11$; $p < .01$), foot ($r = .12$; $p < .01$), and .5hz hand ($r = .09$; $p < .01$) conditions. The 40yo previously concussed group presented significant correlations for the paced .5Hz hand ($r = -.33$; $p < .01$) condition, as well as the un-paced 1Hz hand ($r = .11$; $p < .01$) and foot ($r = -.20$; $p < .01$) conditions. Lastly, the 60yo previously concussed presented significant correlations for the 1Hz hand ($r = -.14$; $p < .01$) and foot ($r = .08$; $p < .01$) conditions, as well as for the un-paced 1Hz hand ($r = .06$; $p < .05$) and foot ($r = .26$; $p < .01$) conditions.

Based on the point-biserial correlational analysis, there appears to be a trend for poorer performance for the 60yo population with a history of two or more concussions, versus a single concussion. This observation has been previously reported in younger populations (Gaetz et al.,

2000; Iverson, Gaetz, Lovell, & Collins, 2004). While these relationships are mostly moderate to small in nature, there may be evidence that a future investigation looks at an elderly population, split by number of previous concussions. Based on the available literature, previously concussed groups should be broken into one or two previous concussions and three or more (De Beaumont et al., 2007; Dretsch et al., 2015; Gaetz et al., 2000; Iverson et al., 2004; Theriault et al., 2011). Though, at this time, there is no known "threshold" to indicate how many previous concussions lead to clinically relevant decreases in cognition and motor performance. Unfortunately the previously concussed groups of these investigations are too small to further evaluate the relationship between previous number of concussions and clinically relevant long-term cognition and motor performance.

# Chapter 4: Summary and Future Directions

**4.1 Summary**

The primary goal of this investigation was to examine motor performance in previously concussed individuals, across age. With approximately 7.7 million high school students participating in athletics each year, the novel findings of this investigation are critical to providing a greater understanding on the possible long-term effects of sustaining a concussion during adolescence and never again. The Accelerated Decline Hypotheses (Figure 4.1) suggested that as the previously concussed age, there would be a divergent pattern between concussion groups on cognition and motor performance, across age (Broglio et al., 2012). However, the data observed in the three projects suggests that there is no apparent difference, let alone a pattern in cognition and motor performance as the previously concussed age. This expands on previous findings, suggesting that there are not differences in younger populations (Broglio et al., 2006; Collie, McCrory, et al., 2006; Thornton et al., 2008).

Based on these findings, there appears to be no significant risk of a divergent decrease in functional cognition or functional motor performance from sustaining a concussion only during adolescences, across age.

This project does not contradict the evidence suggesting that prolonged exposure to multiple concussive incidents leads to behavioral (e.g. depression and MCI) and/or a progressive neuro-degenerative disease, CTE (Baugh et al., 2012; Gavett et al., 2011; Guskiewicz et al., 2005; Guskiewicz et al., 2007; McKee et al., 2009; McKee et al., 2012; Omalu et al., 2005). The differing results may be attributed to two different populations being investigated, the current being individuals with a concussion history prior to being 19 years old, while the former investigates collegiate and professional level athletes with extensive exposure to head impacts in addition to concussions. The number of years of sport participation increases with each new level of participation, also increasing exposure to head impacts. The population for this investigation did not continue sport participation after high school, while collegiate athletes add an additional three to four years of participation, and professional athletes add more years beyond college. In addition, the CTE investigations primarily involve football athletes, who accumulate many sub-clinical head impacts aside from concussions (Gavett et al., 2011; McKee et al., 2009; McKee et al., 2012; Stern et al., 2011). The evidence from CTE and sub-clinical impact accumulation, suggests there might be a head impact exposure factor that is not an issue in the observations of the three projects presented here (Bazarian et al., 2011; Breedlove et al., 2012; Talavage et al., 2010). This is likely due to the difference in head impact exposure between high school athletes and professional athletes. Further research is required to be certain that a history of multiple concussions and sub-clinical head impacts do not have a negative effect on functional cognitive and/or functional motor performance across age, particularly in the population of this investigation.
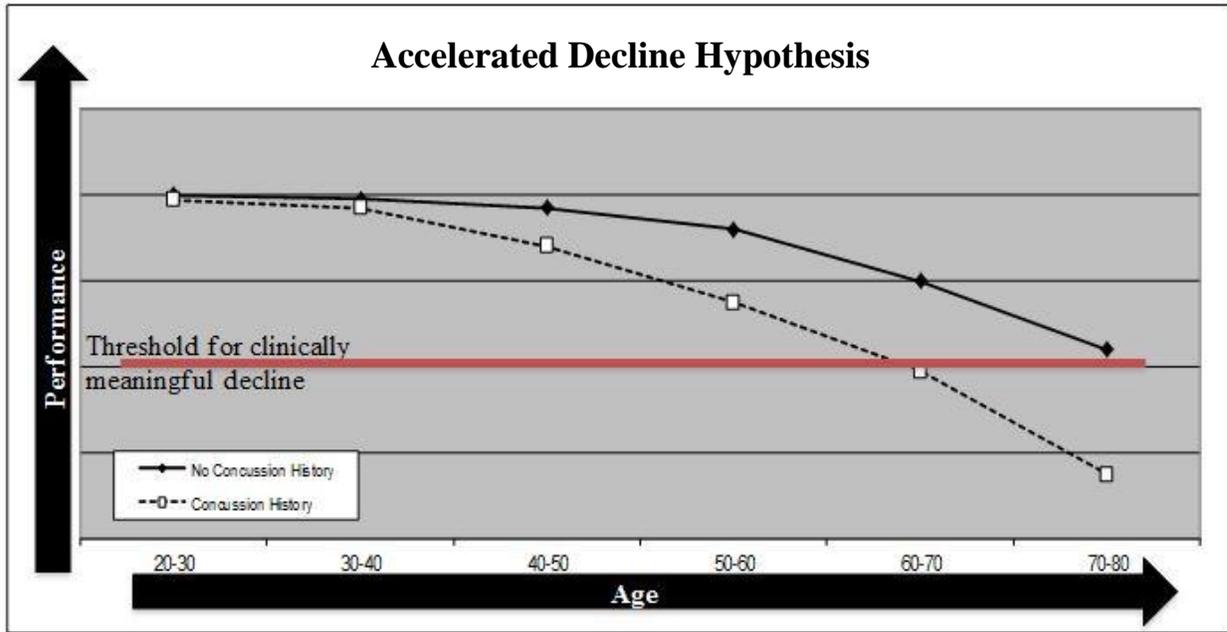
Figure 4.1: Adopted from Broglio, Eckner, Paulson, & Kutcher (2012)

## 4.2 Future Directions and Limitations

While the investigations of this dissertation provide advances in the understanding of the

possible long-term effects of concussion on cognition and motor performance, there are

limitations to this project and future research is necessary to elucidate a greater understanding of

the risks of concussion on late life cognition and motor performance.

The primary limitation being the lack of power associated with the group size. Pilot testing failed

to identify a methodological quirk that affected gait data collection. However, an *a priori*

calculation of power using the observed effect sizes indicate that substantial group sizes would

be needed to observe a power of .80 for a majority of the outcome measures. This indicates that

there is unlikely to be a clinically meaningful significant pattern of differences in upper and

lower extremity motor control, in the adolescent-only concussion history population. Due to the

inability to access medical records, all concussions are self-diagnosed from memory. While this

is not an ideal way to aggregate the previously concussed group, it has been previously reported

in the literature and attaining the medical history from the 40yo and 60yo age groups with proper

concussion diagnosis would be impractical (Broglio et al., 2006; Collie, McCrory, et al., 2006;

Martini et al., 2011). Not accounting for physical activity levels prevents us from accounting for

a possible confounding factor, affecting the performance of the concussion groups. Lastly, as

with any cross-sectional design, there may be a sample bias of the previously concussed

population. However, we employ a rigorous screening protocol to limit the number of

confounding factors (e.g. injury history, neurocognitive history, education history, etc.) that

might skew the data.


A future investigation could examine the center of mass sway in the coronal plane during

obstacle crossing, as there have been reports that both healthy elderly populations and previously

concussed individuals present greater motion in the coronal plane (Austin et al., 1999; Chou,

Kaufman, Hahn, & Brey, 2003; Chou et al., 2004). Additionally, riskier obstacle/step approach

has been observed in the elderly, and could be an additional variable to be observed in a sample

with a concussion history (Begg & Sparrow, 2000; H. C. Chen et al., 1991; Chou et al., 2003). In

addition, the implementation of electromyography, along with a survey of physical activity could

be used to account for any strength or activity differences between the concussion groups, across

age. If differences were observed, therapeutic techniques could be applied to improve muscle

strength and flexibility, improving fall-related gait mechanics (Christiansen, 2008; DiBenedetto

et al., 2005; Judge, Ounpuu, & Davis, 1996; Judge et al., 1993; Kerrigan et al., 2003).

Another investigation could be aimed at examining the effects of sustaining multiple sub-clinical impacts on cognition and motor performance, across age. This is based on the evidence that sustaining multiple (three or more) clinically diagnosed concussions and sub-clinical impacts have a more consistent long-term effect on neurophysiology and cognition (Bazarian et al., 2011; Breedlove et al., 2012; De Beaumont et al., 2007; Talavage et al., 2010; Theriault et al., 2011). A longitudinal investigation measuring reaction time in individuals before participation in contact sports, then at time intervals during participation, could elucidate a connection between reaction time and sport related concussion incidence rates. This is based on the evidence that slower reaction times increase the risk of lower extremity injuries (e.g. strains and sprains) and concussions in military personnel (Nordstrom, Edin, Lindstrom, & Nordstrom, 2013; Swanik, Covassin, Stearne, & Schatz, 2007; Wilkerson, 2012). Lastly, an investigation focused on cognition and motor performance in the elderly who sustained a concussion during adulthood. Given the natural decline in cognition and motor performance with age, and an increasing trend of physical activity in adults, there could be a potential subset of the United States population that is at risk of sustaining a significant decline in cognitive and motor performance.

# APPENDICES

## Appendix A: Participant Questionnaire

<div align="center">

**PARTICIPANT QUESTIONNAIRE**

</div>

Id#_____
Date_____

Age _____          Height (in) _____          Weight (lbs)_____

**SCREENING**
Please answer the following as honestly and accurately as possible:

    a.   Do you have or are you being treated for any condition that may affect your balance or ability to walk? This includes medications.
              Yes          No

    b.   Are you capable of stepping over an object approximately 12in (30cm) tall?
              Yes          No

    c.   Do you have or are you being treated for any condition that may affect your ability to reach with your arm? This includes medications.
              Yes          No

    d.   Do you have normal vision with or without contacts or glass?
              Yes          No

    e.   Have you been diagnosed with a learning disability?
              Yes          No

    f.   Are you currently being treated for or experiencing concussion related symptoms?
              Yes          No

**ACADEMIC HISTORY**
How many years of education have you completed:_____
        (elementary= 8yrs; high school=12yrs; Bachelor's = 16yrs, Master's = 18yrs, PhD = 22yrs):

What is your highest level of education? (eg BA, Masters, PhD): _____

Have you repeated a year of school?            Yes          No     If yes,

which?_____

Have you ever skipped a year of school?      Yes          No   If yes, which?_____

Have you ever received academic assistance?      Yes          No

Do you have a learning disability (eg dyslexia)? _____

Do you have diagnosed attention deficit disorder? _____

Are you taking any medication for learning disorders?     Yes          No

        If yes, which? _____

## MEDICAL HISTORY
Have you ever had or currently have:

| | YES | NO |
|---|---|---|
| Depression | | |
| Mild Cognitive Impairment | | |
| Alzheimer's Disease | | |
| Bipolar Disorder | | |
| Schizophrenia | | |
| Other Cognitive Disorder | | |

If 'Yes' to any of these, please  explain (include diagnosis date)_____

_____

Are you taking medication for this illness?:   Yes      No

If yes, what_____

Have you ever been under general anesthesia:          Yes      No      How many times? _____

Accidents involving the head (NOT concussions):   Yes      No      How many times? _____

Do you take stimulants (eg caffeine):          Yes          No
          If so, at what frequency:          Rarely          Sometimes          On a regular basis

Have you been diagnosed with migraines?   Yes          No

Have you had one or more headaches in the past 3 months?   Yes          No

          If yes, have you had a headache associated with the following:

                    -limited your ability to work, study, or do what you wanted to do?          Yes          No

                    -light bothered you (more than when you don't have a headache)?          Yes          No

                    -you got nauseous or sick to your stomach?          Yes          No


## RISK OF FALLING ASSESSMENT
Have you fallen within the previous year?          Yes          No

Are you taking 4 or more prescribed medications?          Yes          No

Do you have a history of stroke or Parkinson's disease?          Yes          No

Do you have any balance problems?          Yes          No

Can you rise from a chair without using your arms?          Yes          No

Are you afraid of falling?          Yes          No

Has a fear of falling made you avoid any activities?          Yes          No

| How confident are you that you can… | Extreme Confidence | | | | | | | | | No Confidence at All |
|---|---|---|---|---|---|---|---|---|---|---|
| Take a bath or shower? | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Reach into cabinets or closets? | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Prepare meals not requiring carrying heavy or hot objects | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Walk around the house | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Get in and out of bed | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Answer the door or telephone | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Get in and out of a chair | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Get dressed and undressed | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Personal grooming (eg washing hair) | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Get on and off the toilet without falling | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

Based on **how you feel right now**, indicate each of the following by marking '0' for not at all, '3' as moderate, and '6' as severe.

| | NONE | | | MODERATE | | | SEVERE |
|---|---|---|---|---|---|---|---|
| Headache | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| "Pressure in Your head" | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Neck Pain | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Nause or 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 |
| Feeling "in a fog" | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| "Don't feel right" | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Difficulty Concentrating | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Difficulty Remembering | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Fatigue or 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 than Usual | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Sadness | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Nervous or Anxious | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

**CONCUSSION HISTORY**

Did you participate in a sport(s) during prior to the age of 19 (18 or younger)?     Yes                 No

If yes, what sport(s)_____

Have you ever been told by a medical professional (eg Doctor, Athletic Trainer, EMT, nurse) that you had a concussion?  The injury may or may not have been sport related (eg auto accident)

_____   Yes, how many times? _____                 No_____

| | Approximate Date (month/year) | Approximate age at time of injury | Did you lose consciousness at the time of injury (ie knocked out/blacked out) | Did/do you have difficulty remembering things before or after the injury | How many days did you experience symptoms related to the injury? |
|---|---|---|---|---|---|
| Injury #1 | | | | | |
| Injury #2 | | | | | |
| Injury #3 | | | | | |
| Injury #4 | | | | | |
| Injury #5 | | | | | |

Please use the following definition of concussion to answer the questions below.
**Definition of concussion**: *A concussion is a blow to your head that causes a variety of symptoms that may last for a short period of time, such as a few plays or minutes of a game, or a longer period of time. These symptoms may include any of the following:*

- Headache
- Difficulty concentrating or focusing
- Feeling slowed down
- Dizziness or balance problems
- Nausea
- Fatigue / lack of energy
- Feeling like you're in a fog
- Irritable
- Drowsiness
- Forgetting things (before or
- after the injury)
- Sensitivity to light
- Loss of balance
- Sensitivity to noise
- Blurred vision

**IMPORTANT**: A) you can have a concussion **without** being "knocked out" or unconscious

B) getting your "bell rung" and "clearing the cobwebs" **is** a concussion

Following a blow to the head, have you ever experienced any of the symptoms listed above or had a concussion that was not evaluated by a medical professional (eg Doctor, Athletic Trainer, EMT)

_____ Yes, how many times? _____ No_____

| | Approximate Date (month/year) | Approximate age at time of injury | Did you lose consciousness at the time of injury (ie knocked out/blacked out) | Did/do you have difficulty remembering things before or after the injury | How many days did you experience symptoms related to the injury? |
|---|---|---|---|---|---|
| Injury #1 | | | | | |
| Injury #2 | | | | | |
| Injury #3 | | | | | |
| Injury #4 | | | | | |
| Injury #5 | | | | | |

## Appendix B: Falling and Headache Demographics

Table B.i Fear of Falling and Fall Risk Assessment

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | Control (n = 27) | Concussed (n = 19) | Control (n = 15) | Concussed (n = 4) | Control (n = 16) | Concussed (n = 9) |
| FallEfficacyScore | 9.93(0.39) | 10.32(1.00) | 10(0.00) | 10(0.00) | 10(0.00) | 10.75(2.12) |
| Have you fallen within the previous year (%) | 11 | 16 | 0 | 25 | 6 | 13 |
| Are you taking 4 or more prescribed medications (%) | 0 | 0 | 0 | 0 | 19 | 25 |
| Do you have a history of stroke or Parkinson's (%) | 0 | 0 | 13 | 0 | 0 | 0 |
| Do you have any balance problems (%) | 0 | 0 | 0 | 0 | 0 | 13 |
| Can you rise from a chair without using your arms (%) | 93 | 100 | 100 | 100 | 100 | 100 |
| Are you afraid of falling (%) | 0 | 0 | 0 | 0 | 6 | 13 |
| Has a fear of falling made you avoid any activities (%) | 0 | 0 | 0 | 0 | 0 | 0 |

Mean(SD). No significant within age, concussion group differences (p's > .05). Abbreviations: FallEfficacyScore, Falls Efficacy Scale score.

Table B.ii Symptom and Migraine Report

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | Control (n = 27) | Concussed (n = 19) | Control (n = 15) | Concussed (n = 4) | Control (n = 16) | Concussed (n = 9) |
| PCS total score | 2.33(4.40) | 5.47(6.54) | 1.93(2.25) | 1.00(2.00) | 1.44(3.12) | 4.00(6.63) |
| diagnosed with migraines (%) | 7 | 0 | 7 | 50 | 6 | 0 |
| 1+ headache in past 3 months (%) | 63 | 74 | 33 | 50 | 13 | 13 |
| 1+ headache affected work (%) | 19 | 32 | 13 | 50 | 0 | 0 |
| 1+ headache, bothered by light (%) | 30 | 42 | 13 | 25 | 6 | 0 |
| 1+ headache, nausea (%) | 4 | 16 | 0 | 25 | 0 | 0 |

Mean(SD). No significant within age, concussion group differences (p's > .05). Abbreviations: GSC, Graded Symptom Checklist

**Appendix C: AXON by CogState Group Means**

Table C.i AXON Output Scores

| AXON Score | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | Control (n = 26) | Concussed (n = 19) | Control (n = 15) | Concussed (n = 4) | Control (n = 13) | Concussed (n = 7) |
| Processing Speed | 97.97(4.86) | 96.04(8.22) | 97.07(5.40) | 97.45(6.55) | 98.26(4.75) | 96.54(4.66) |
| Attention | 103.49(4.63) | 104.04(5.33) | 102.56(4.40) | 100.53(2.87) | 102.55(2.57) | 101.77(2.71) |
| Learning | 103.18(7.19) | 104.52(4.98) | 106.16(7.91) | 101.53(9.40) | 103.95(7.58) | 105.99(5.19) |
| Working Memory Speed | 101.75(5.90) | 100.42(7.49) | 99.49(4.34) | 94.38(6.51) | 96.1(0(3.46) | 97.16(1.96) |
| Working Memory Accuracy | 106.16(7.55) | 102.74(4.93) | 106.38(8.74) | 97.48(4.37) | 102.78(11.17) | 108.86(7.05) |

Mean(SD). No significant within age, concussion group differences (p's > .05).

**Appendix D: Gait Analysis Group Means**

Table D.i Spatio-temporal Gait Variables, Condition 1 (normal walk)

|  | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
|  | Control (n = 9) | Concussed (n = 10) | Control (n =3) | Concussed (n = 4) | Control (n = 9) | Concussed (n = 6) |
| velocity (m/s) | 1.31(.18) | 1.28(.12) | 1.18(.07) | 1.20(.18) | 1.16(.08) | 1.28(.24) |
| Percent of Time in Double Stance | 0.23(.03) | 0.21(.04) | 0.26(.05) | 0.30(.05) | 0.24(.01) | 0.25(.07) |
| Step width (m) | 0.11(.03) | 0.12(.03) | 0.14(.02) | 0.12(.03) | 0.10(.03) | 0.11(.01) |
| Stride Length (m) | 1.38(.11) | 1.32(.19) | 1.30(.07) | 1.43(.09) | 1.25(.09) | 1.36(.17) |

Means(SD). No significant within age, concussion group differences (p's > .05).

Table D.ii Spatio-temporal Gait Variables, Condition 2 (normal walk, mental task)

|  | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
|  | Control (n = 8) | Concussed (n = 6) | Control (n = 2) | Concussed (n = 1) | Control (n = 4) | Concussed (n = 4) |
| velocity (m/s) | 0.97(.16) | 0.91(.21) | 0.74(.43) | 1.01(.) | 0.99(.11) | 0.99(.23) |
| Percent of Time in Double Stance | 0.32(.05) | 0.34(.07) | 0.42(.19) | .33(.) | 0.31(.03) | 0.33(.07) |
| Step width (m) | 0.12(.03) | 0.12(.04) | 0.13(.02) | .08(.) | 0.12(.02) | 0.10(.03) |
| Stride Length (m) | 1.23(.10) | 1.29(.11) | 1.09(.09) | 1.48(.) | 1.25(.13) | 1.31(.10) |

Means(SD). No significant within age, concussion group differences (p's > .05).

Table D.iii Spatio-temporal Gait Variables, Condition 3 (obstructed walk)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | Control (n = 17) | Concussed (n = 13) | Control (n =11) | Concussed (n = 4) | Control (n = 9) | Concussed (n = 7) |
| velocity (m/s) | 1.13(.12) | 1.15(.10) | 1.014(.11) | 1.01(.10) | 0.92(.11)* | 1.10(.13) |
| Percent of Time in Double Stance | 0.22(.03) | 0.22(.04) | 0.24(.04) | 0.27(.04) | 0.28(.06) | .24(.05) |
| Step width (m) | 0.12(.03) | 0.12(.04) | 0.15(.03) | 0.14(.03) | 0.11(.03) | 0.10(.03) |
| Stride Length (m) | 1.43(.10) | 1.45(.11) | 1.32(.11) | 1.46(.03) | 1.26(.12) | 1.41(.12) |

Mean(SD). No significant within age, concussion group differences (p's > .05).

Table D.iv Spatio-temporal Gait Variables, Condition 4 (obstructed walk, mental task)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | Control (n = 18) | Concussed (n = 15) | Control (n =10) | Concussed (n = 4) | Control (n = 8) | Concussed (n =6) |
| velocity (m/s) | 0.90(.12) | 0.89(.15) | 0.73(.10) | 0.84(.16) | 0.74(.12) | 0.83(.18) |
| Percent of Time in Double Stance | 0.29(.06) | 0.32(.08) | 0.35(.08) | 0.37(.08) | 0.34(.04) | 0.32(.06) |
| Step width (m) | 0.12(.02) | 0.13(.03) | 0.13(.03) | 0.11(.03) | 0.11(.03) | 0.10(.04) |
| Stride Length (m) | 1.35(.11) | 1.39(.14) | 1.29(.12) | 1.47(.08) | 1.24(.08) | 1.31(.13) |

Means(SD). No significant within age, concussion group differences (p's > .05).

Table D.v Kinematic Gait Variables, Condition 1 (normal walk)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | Control (n = 9) | Concussed (n = 10) | Control (n = 3) | Concussed (n = 4) | Control (n = 9) | Concussed (n = 6) |
| MaxAnPlFlex | -35(9) | -29(8) | -31(7) | -30(10) | -26(7) | -23(7) |
| MaxHipExt | -12(8) | -17(9) | -16(16) | -26(11) | -22(14) | -21(6) |

Means(SD). No significant within age, concussion group differences (p's > .05).

Table D.vi Kinematic Gait Variables, Condition 2 (normal walk, mental task)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | Control (n = 8) | Concussed (n = 6) | Control (n = 2) | Concussed (n = 1) | Control (n = 4) | Concussed (n = 4) |
| MaxAnPlFlex | -37(11) | -33(7) | -33(4) | -44(.) | -19(2) | -16(13) |
| MaxHipExt | -14(7) | -19(6) | -14(1) | -39(.) | -12(7) | -20(4) |

Means(SD). No significant within age, concussion group differences (p's > .05).

Table D.vii Kinematic Gait Variables, Condition 3 (obstructed walk)

|  |  | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | Control (n = 17) | Concussed (n = 13) | Control (n =11) | Concussed (n = 4) | Control (n = 9) | Concussed (n = 7) |
| Lead Limb | MaxAnPlFlex | -29(7) | -29(9) | -26(8) | -32(9) | -25(8) | -27(10) |
|  | MaxHipExt | -11(7) | -15(7) | -16(12) | -24(12) | -19(13) | -19(4) |
| Trail Limb | MaxAnPlFlex | -40(7) | -35(8) | -37(10) | -34(7) | -32(10) | -33(7) |
|  | MaxHipExt | -13(7) | -17(9) | -16(13) | -26(12) | -18(13) | -20(4) |

Means(SD). No significant within age, concussion group differences (p's > .05).

Table D.viii Kinematic Gait Variables, Condition 4 (obstructed walk, mental task)

|  |  | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | Control (n = 18) | Concussed (n = 15) | Control (n =10) | Concussed (n = 4) | Control (n = 8) | Concussed (n =6) |
| Lead Limb | MaxAnPlFlex | -30(8) | -27(7) | -30(5) | -30(11) | -22(7) | -21(11) |
|  | MaxHipExt | -12(7) | -16(6) | -16(15) | -24(12) | -15(13) | -20(3) |
| Trail Limb | MaxAnPlFlex | -36(7) | -33(8) | -32(10) | -40(7) | -28(12) | -23(12) |
|  | MaxHipExt | -13(8) | -17(8) | -18(13) | -25(14) | -16(11) | -19(5) |

Means(SD). No significant within age, concussion group differences (p's > .05).
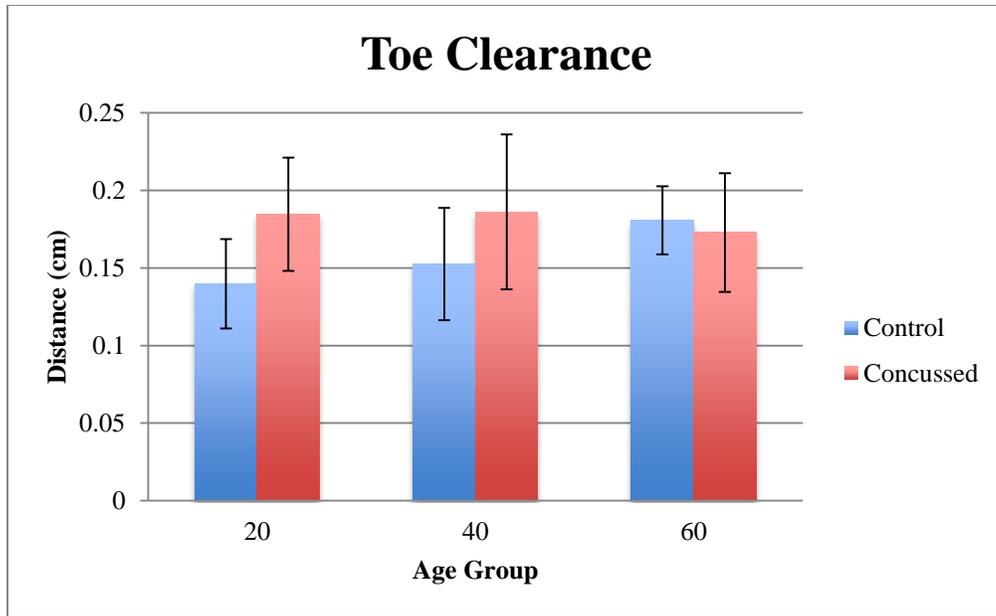
Figure D.i Lead Limb Toe Clearance, Condition 3 (obstructed walk condition). No Significant within age, concussion group difference (p's > .05). Error bars represent SD.
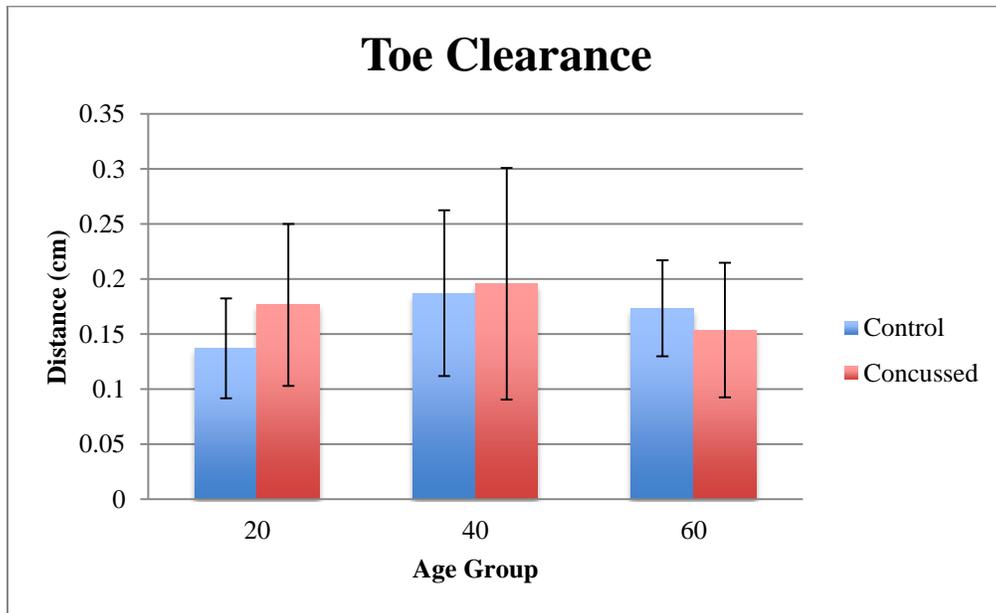


Figure D.ii Trail Limb Toe Clearance, Condition 3 (obstructed walk condition). No Significant within age, concussion group difference (p's > .05). Error bars represent SD.
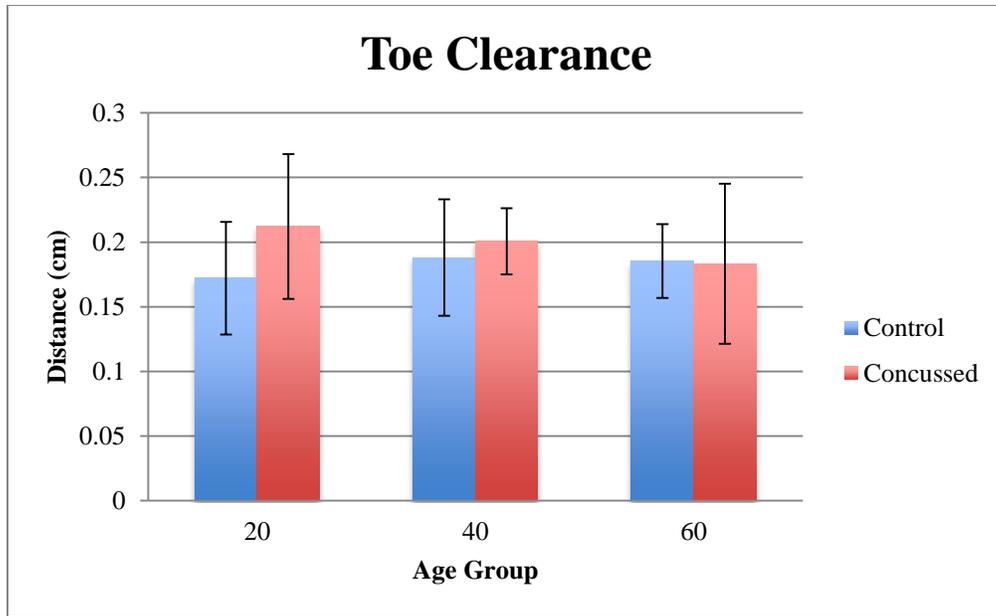
Figure D.iii Lead Limb Toe Clearance, Condition 4 (obstructed, mental task walk condition). No significant within age, concussion group differences (p's > .05). Error bars represent SD.
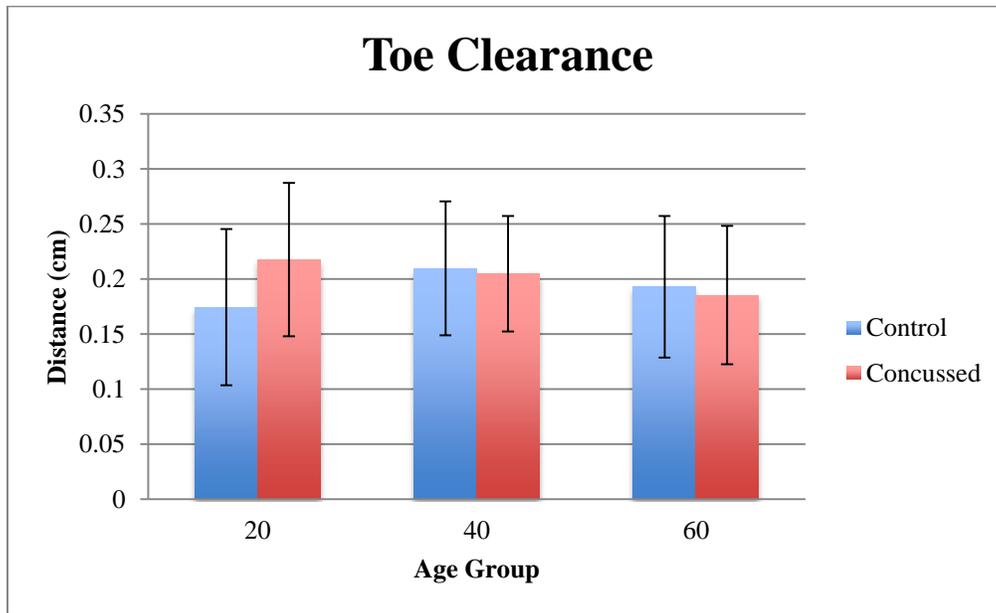


Figure D.iv Trail Limb Toe Clearance, Condition 4 (obstructed, mental task walk condition). No significant within age, concussion group differences (p's > .05). Error bars represent SD.
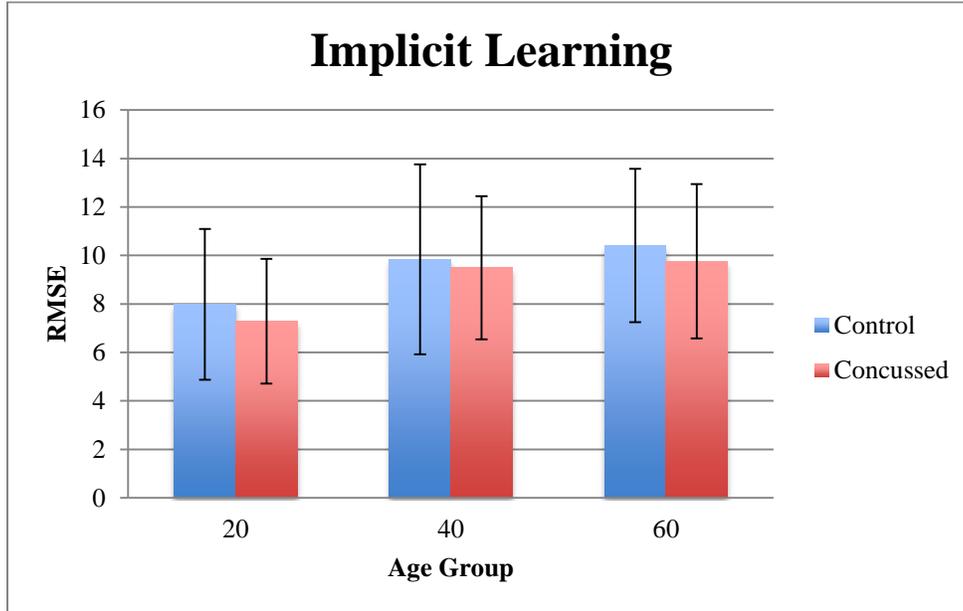
Figure E.i Hand Continuous Tracking, Implicit Learning (Waveform 2). No significant within age, concussion group differences (p's > .05). Error bars represent SD.
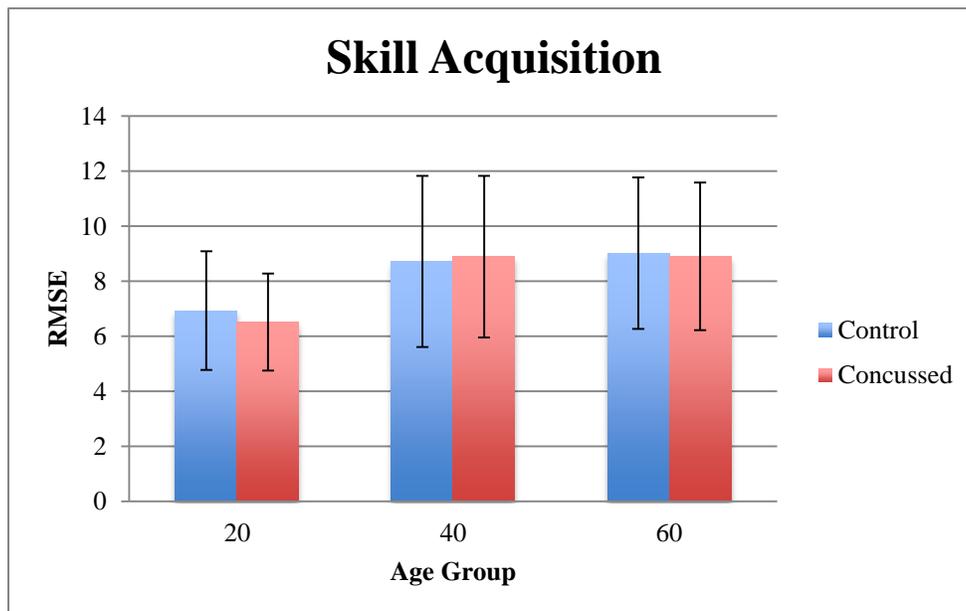


Figure E.ii Hand Continuous Tracking, Skill Acquisition (Waveforms 1 and 3). No significant within age, concussion group differences (p's > .05). Error bars represent SD.
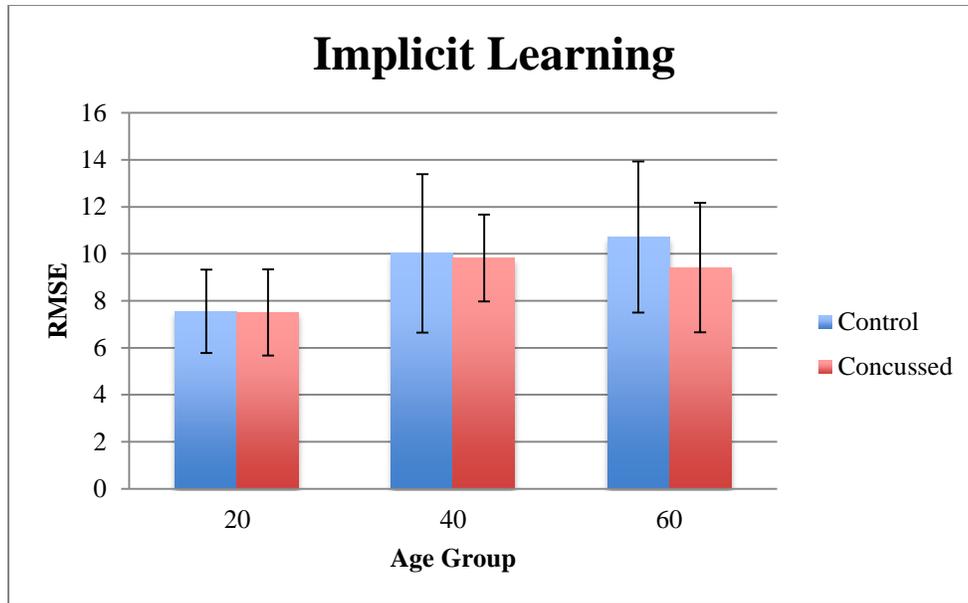
Figure E.iii Foot Continuous Tracking, Implicit Learning (Waveform 2). No significant within age, concussion group differences (p's > .05). Error bars represent SD.
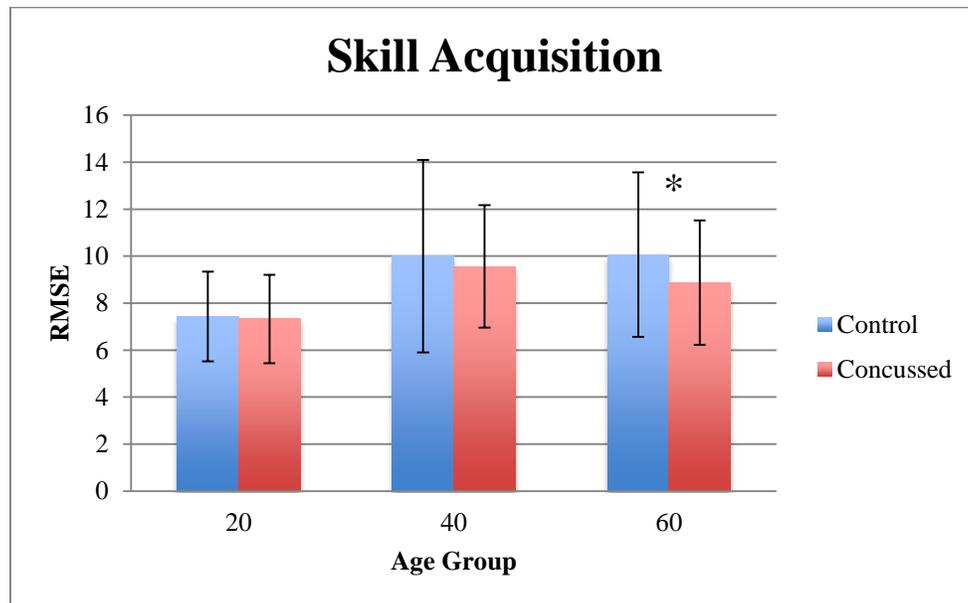


Figure E.iv Foot Continuous Tracking, Skill Acquisition (Waveforms 1 and 3). *Significant difference within age, concussion groups (p < .001). Error bars represent SD.

Table E.i Discrete Auditory RT, Hand

| | | | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|---|
| | | | Control (n = 25) | Concussed (n = 19) | Control (n = 14) | Concussed (n = 4) | Control (n = 13) | Concussed (n = 7) |
| 1 Hz | Paced RT (ms) | | 0.98(.12) | 0.98(.12) | 0.98(.14)* | 1.00(.14) | 0.96(.11)* | 0.94(.10) |
| | Un-Paced RT (ms) | | 0.13(.18)* | 0.15(.18) | 0.08(.18)* | 0.00(.13) | 0.18(.22)* | 0.14(.18) |
| .5 Hz | Paced RT (ms) | | 1.92(.33)* | 1.98(.33) | 1.97(.33)* | 1.87(.37) | 1.96(.23) | 1.94(.20) |
| | Un-Paced RT (ms) | | 0.13(.20)* | 0.17(.21) | 0.12(.23)* | 0.17(.17) | 0.20(.21)* | 0.09(.19) |

Means(SD). *Significant difference within age, between concussion groups ($p < .001$).

Table E.ii Discrete Auditory RT, Foot

| | | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|
| | | Control (n = 25) | Concussed (n = 19) | Control (n = 14) | Concussed (n = 4) | Control (n = 13) | Concussed (n = 7) |
| 1 Hz | Paced RT (ms) | 0.98(.10)* | 0.96(.11) | 1.00(.15) | 0.99(.17) | 0.96(.12) | 0.98(.09) |
| | Un-Paced RT (ms) | 0.06(.21)* | 0.08(.23) | 0.01(.14)* | -0.06(.13) | 0.02(.30) | 0.03(.15) |
| .5 Hz | Paced RT (ms) | 1.93(.26) | 1.94(.27) | 2.01(.30)* | 1.94(.25) | 1.93(.28) | 1.97(.19) |
| | Un-Paced RT (ms) | 0.10(.25) | 0.13(.25) | 0.08(.32)* | -0.01(.25) | 0.17(.27)* | 0.00(.24) |

Means(SD). *Significant difference within age, between concussion groups ($p < .001$).

Table E.iii Discrete Auditory Coefficient of Variation, Hand

| | | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|
| | | Control (n = 25) | Concussed (n = 19) | Control (n = 14) | Concussed (n = 4) | Control (n = 13) | Concussed (n = 7) |
| 1 Hz | Paced RT | 0.11(.05) | 0.11(.04) | 0.11(.04) | 0.13(.08) | 0.09(.05) | 0.10(.02) |
| | Un-Paced RT | -2.26(14.00) | -1.37(7.85) | 3.24(9.59) | 4.32(12.67) | 1.22(4.97) | -326.07(866.07) |
| .5 Hz | Paced RT | 0.14(.08) | 0.14(.06) | 0.13(.05) | 0.14(.05) | 0.10(.03) | 0.10(.02) |
| | Un-Paced RT | 0.75(5.02) | -2.65(20.13) | -15.02(42.06 | 2.41(3.59) | -1.55(4.67) | 1.11(5.61) |

Means(SD). No significant within age, concussion group differences (p's > .05).

Table E.iv Discrete Auditory Coefficient of Variation, Foot

| | | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|
| | | Control (n = 25) | Concussed (n = 19) | Control (n = 14) | Concussed (n = 4) | Control (n = 13) | Concussed (n = 7) |
| 1 Hz | Paced RT | 0.09(.03) | 0.10(.04) | 0.12(.05) | 0.15(.09) | 0.10(.03) | 0.08(.02) |
| | Un-Paced RT | 1.93(7.45) | 0.97(4.84) | -1.05(2.65) | -3.64(3.22) | 2.64(8.53) | 102.42(268.87) |
| .5 Hz | Paced RT | 0.11(.07) | 0.12(.06) | 0.12(.05) | 0.12(.02) | 0.11(.08) | 0.09(.03) |
| | Un-Paced RT | 0.10(4.00) | -46.26(194.70) | 3.63(9.37) | -3.04(3.34) | -0.44(3.58) | 3.13(12.99) |

Means(SD). No significant within age, concussion group differences (p's > .05).

**Appendix F: Concussion History Correlations and Group Means**

Table F.i AXON by CogState Concussion History Point-biserial Correlations

| Age Group | Processing Speed | Attention | Learning | Working Memory Speed | Working Memory Accuracy |
|---|---|---|---|---|---|
| 20 Year Olds | 0.16 | -0.02 | -0.05 | 0.14 | 0.05 |
| 40 Year Olds | -0.79 | -0.88 | 0.04 | -0.90 | -0.02 |
| 60 Year Olds | -0.61 | -0.26 | 0.55 | 0.63 | 0.39 |

Point-biserial Correlation Coefficient, r. No significant within age, concussion group differences (p's > .05).

Table F.ii AXON by CogState Concussion History Group Means

|  | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
|  | 1 Concussion (n = 9) | 2+ Concussions (n = 10) | 1 Concussion (n = 1) | 2+ Concussions (n = 3) | 1 Concussion (n = 2) | 2+ Concussions (n = 5) |
| Processing Speed | 94.71(9.48) | 97.24(7.20) | 105.20(.) | 94.87(4.93) | 100.70(2.26) | 94.88(4.37) |
| Attention | 104.14(5.55) | 103.95(5.42) | 104.30(.) | 99.27(1.69) | 102.80(.28) | 101.36(3.20) |
| Learning | 104.79(4.25) | 104.28(5.78) | 100.90(.) | 101.73(11.50) | 101.80(5.94) | 107.66(4.39) |
| Working Memory Speed | 99.37(7.77) | 101.36(7.51) | 103.20(.) | 91.43(3.41) | 95.35(.64) | 97.88(1.84) |
| Working Memory Accuracy | 102.51(6.74) | 102.95(2.87) | 97.60(.) | 97.43(5.35) | 104.85(2.62) | 110.46(7.85) |

Means(SD).

Table F.iii Spatio-Temporal Gait Variables Concussion History Point-biserial Correlations, Condition 1 (normal walk)

| | Velocity (m/s) | Percent Time in Double Stance | Step width (m) | Stride Length (m) |
|---|---|---|---|---|
| 20 Year Olds | -0.65* | 0.44 | -0.05 | -0.40 |
| 40 Year Olds | -0.58 | 0.57 | 0.31 | -0.81 |
| 60 Year Olds | 0.69 | -0.62 | 0.74 | 0.71 |

Point-biserial Correlation Coefficient, r. *Significant within age, concussion group differences ($p < .05$).

Table F.iv Spatio-Temporal Gait Variables Concussion History Group Means, Condition 1 (normal walk)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | 1 concussion (n = 3) | 2+ concussions (n = 7) | 1 concussion (n = 1) | 2+ concussions (n = 3) | 1 concussion (n = 3) | 2+ concussions (n = 3) |
| Velocity (m/s) | 1.39(.12) | 1.23(.08) | 1.35(.) | 1.15(.18) | 1.16(.16) | 1.46(.26) |
| Percent Time in Double Stance | 0.19(.04) | 0.22(.04) | 0.25(.) | 0.31(.05) | 0.29(.05) | 0.21(.05) |
| Step width (m) | 0.12(.03) | 0.12(.04) | 0.11(.) | 0.13(.04) | 0.10(.01) | 0.12(.00) |
| Stride Length (m) | 1.42(.11) | 1.27(.19) | 1.53(.) | 1.39(.06) | 1.27(.12) | 1.49(.17) |

Means(SD).

Table F.v Spatio-Temporal Gait Variables Concussion History Point-biserial Correlations, Condition 2 (normal walk, mental task)

| | Velocity (m/s) | Percent Time in Double Stance | Step width (m) | Stride Length (m) |
|---|---|---|---|---|
| 20 Year Olds | -0.65 | 0.60 | 0.82 | 0.47 |
| 40 Year Olds | N/A | N/A | N/A | N/A |
| 60 Year Olds | -0.56 | 0.80 | 0.01 | 0.34 |

Point-biserial Correlation Coefficient, r. No Significant within age, concussion group differences (p's > .05).

Table F.vi Spatio-Temporal Gait Variables Concussion History Group Means, Condition 2 (normal walk, mental task)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | 1 concussion (n = 4) | 2+ concussions (n = 2) | 1 concussion (n = 0) | 2+ concussions (n = 1) | 1 concussion (n = 1) | 2+ concussions (n = 3) |
| Velocity (m/s) | 1.00(.19) | 0.75(.18) | N/A | 1.01(.) | 1.13(.) | 0.91(.27) |
| Percent Time in Double Stance | 0.32(.07) | 0.39(.04) | N/A | .33(.) | 0.26(.) | 0.36(.07) |
| Step width (m) | 0.10(.03) | 0.16(.01) | N/A | .08(.) | 0.10(.) | 0.10(.04) |
| Stride Length (m) | 1.25(.11) | 1.35(.12) | N/A | 1.48(.) | 1.27(.) | 1.33(.13) |

Means(SD).

Table F.vii Spatio-Temporal Gait Variables Concussion History Point-biserial Correlations, Condition 3 (obstructed walk)

| | Velocity (m/s) | Percent Time in Double Stance | Step width (m) | Stride Length (m) |
|---|---|---|---|---|
| 20 Year Olds | -0.02 | 0.36 | 0.01 | -0.15 |
| 40 Year Olds | -0.25 | 0.14 | -0.69 | -0.18 |
| 60 Year Olds | 0.36 | -0.37 | 0.59* | 0.30 |

Point-biserial Correlation Coefficient, r. *Significant within age, concussion group differences ($p < .05$).

Table F.viii Spatio-Temporal Gait Variables Concussion History Group Means, Condition 3 (obstructed walk)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | 1 concussion (n = 4) | 2+ concussions (n = 9) | 1 concussion (n = 1) | 2+ concussions (n = 3) | 1 concussion (n = 3) | 2+ concussions (n = 4) |
| Velocity (m/s) | 1.15(.07) | 1.14(.12) | 1.05(.) | 1.00(.12) | 1.05(.07) | 1.14(.16) |
| Percent Time in Double Stance | 0.21(.02) | 0.24(.04) | 0.26(.) | 0.27(.05) | 0.26(.04) | 0.23(.06) |
| Step width (m) | 0.12(.03) | 0.12(.04) | 0.17(.) | 0.13(.03) | 0.08(.02) | 0.12(.03) |
| Stride Length (m) | 1.47(.06) | 1.43(.13) | 1.47(.) | 1.46(.03) | 1.37(.09) | 1.44(.14) |

Means(SD).

Table F.ix Spatio-Temporal Gait Variables Concussion History Point-biserial Correlations, Condition 4 (obstructed walk, mental task)

| | Velocity (m/s) | Percent Time in Double Stance | Step width (m) | Stride Length (m) |
|---|---|---|---|---|
| 20 Year Olds | 0.10 | -0.03 | -0.19 | 0.29 |
| 40 Year Olds | 0.25 | -0.09 | 0.28 | -0.03 |
| 60 Year Olds | -0.64* | 0.67* | -0.56 | -0.76** |

Point-biserial Correlation Coefficient, r. *Significant within age, concussion group differences ($p < .05$). **Significant within age, concussion group differences ($p < .01$).

Table F.x Spatio-Temporal Gait Variables Concussion History Group Means, Condition 4 (obstructed walk, mental task)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | 1 concussion (n = 6) | 2+ concussions (n = 9) | 1 concussion (n = 1) | 2+ concussions (n = 3) | 1 concussion (n = 2) | 2+ concussions (n = 4) |
| Velocity (m/s) | 0.87(.12) | 0.90(.17) | 0.79(.) | 0.87(.20) | 1.01(.20) | 0.77(.13) |
| Percent Time in Double Stance | 0.32(.11) | 0.32(.05) | 0.38(.) | 0.37(.10) | 0.25(.05) | 0.34(.05) |
| Step width (m) | 0.14(.03) | 0.12(.03) | 0.10(.02) | 0.11(.04) | 0.13(.02) | 0.09(.03) |
| Stride Length (m) | 1.35(.11) | 1.43(.16) | 1.46(.) | 1.46(.09) | 1.47(.02) | 1.25(.10) |

Means(SD).

Table F.xi Kinematic Gait Variables Concussion History Point-biserial Correlations, Condition 1 (normal walk)

|  | Maximum Ankle Plantarflexion | Maximum Hip Extension |
|---|---|---|
| 20 Year Olds | 0.03 | 0.01 |
| 40 Year Olds | 0.36 | -0.68 |
| 60 Year Olds | -.95* | -0.54 |

Point-biserial Correlation Coefficient, r. *Significant within age, concussion group differences (p < .01).

Table F.xii Kinematic Gait Variables Concussion History Group Means, Condition 1 (normal walk)

|  | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
|  | 1 concussion (n = 3) | 2+ concussions (n = 7) | 1 concussion (n = 1) | 2+ concussions (n = 3) | 1 concussion (n = 3) | 2+ concussions (n = 3) |
| Maximum Ankle Plantarflexion | -30(5) | -30(9) | -31(9) | -26(8) | -18(0) | -31(3) |
| Maximum Hip Extension | -17(3) | -17(10) | -17(8) | -30(10) | -19(8) | -25(3) |

Means(SD). Joint angles are represented in degrees.

Table F.xiii Kinematic Gait Variables Concussion History Point-biserial Correlations, Condition 2 (normal walk, mental task)

| Age Group | Maximum Ankle Plantarflexion | Maximum Hip Extension |
|---|---|---|
| 20 Year Olds | -0.21 | -0.69 |
| 40 Year Olds | N/A | N/A |
| 60 Year Olds | 0.09 | -0.72 |

Point-biserial Correlation Coefficient, r. No significant within age, concussion group differences (p's > .05).

Table F.xiv Kinematic Gait Variables Concussion History Group Means, Condition 2 (normal walk, mental task)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | 1 concussion (n = 4) | 2+ concussions (n = 2) | 1 concussion (n = 0) | 2+ concussions (n = 1) | 1 concussion (n = 1) | 2+ concussions (n = 3) |
| Maximum Ankle Plantarflexion | -32(5) | -34(9) | N/A | -44(.) | -20(.) | -16(14) |
| Maximum Hip Extension | -15(3) | -23(7) | N/A | -40(.) | -16(.) | -23(4) |

Means(SD). Joint angles are represented in degrees.

Table F.xv Kinematic Gait Variables Concussion History Point-biserial Correlations, Condition 3 (obstructed walk)

|  | Age Group | Maximum Ankle Plantarflexion | Maximum Hip Extension |
|---|---|---|---|
| Lead Limb | 20 Year Olds | 0.02 | -0.03 |
|  | 40 Year Olds | 0.42 | -0.61 |
|  | 60 Year Olds | -0.05 | -.61* |
| Trail Limb | 20 Year Olds | 0.06 | -0.13 |
|  | 40 Year Olds | 0.70 | -0.68 |
|  | 60 Year Olds | 0.13 | -0.35 |

Point-biserial Correlation Coefficient, r. *Significant within age, concussion group differences ($p < .05$).

Table F.xvi Kinematic Gait Variables Concussion History Group Means, Condition 3 (obstructed walk)

|  |  | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|
|  |  | 1 concussion (n = 4) | 2+ concussions (n = 9) | 1 concussion (n = 1) | 2+ concussions (n = 3) | 1 concussion (n = 3) | 2+ concussions (n = 4) |
| Lead Limb | Maximum Ankle Plantarflexion | -29(8) | -29(10) | -37(.) | -30(10) | -26(6) | -27(13) |
|  | Maximum Hip Extension | -15(7) | -15(8) | -13(.) | -28(12) | -16(5) | -21(2) |
| Trail Limb | Maximum Ankle Plantarflexion | -35(8) | -34(9) | -42(.) | -32(6) | -34(7) | -32(8) |
|  | Maximum Hip Extension | -16(8) | -18(9) | -14(.) | -31(11) | -18(6) | -21(2) |

Means(SD). Joint angles are represented in degrees.

Table F.xvii Kinematic Gait Variables Concussion History Point-biserial Correlations, Condition 4 (obstructed walk, mental task)

|  | Age Group | Maximum Ankle Plantarflexion | Maximum Hip Extension |
|---|---|---|---|
| Lead Limb | 20 Year Olds | -0.23 | -0.13 |
|  | 40 Year Olds | 0.11 | -0.73 |
|  | 60 Year Olds | 0.23 | 0.06 |
| Trail Limb | 20 Year Olds | 0.08 | -0.10 |
|  | 40 Year Olds | -0.21 | -0.70 |
|  | 60 Year Olds | 0.24 | 0.26 |

Point-biserial Correlation Coefficient, r. No significant within age, concussion group differences (p's > .05).

Table F.xviii Kinematic Gait Variables Concussion History Group Means, Condition 4 (obstructed walk, mental task)

|  |  | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|
|  |  | 1 concussion (n = 6) | 2+ concussions (n = 9) | 1 concussion (n = 1) | 2+ concussions (n = 3) | 1 concussion (n = 2) | 2+ concussions (n = 4) |
| Lead Limb | Maximum Ankle Plantarflexion | -25(3) | -28(9) | -32(.) | -29(14) | -25(8) | -20(12) |
|  | Maximum Hip Extension | -16(4) | -17(8) | -12(.) | -30(11) | -20(5) | -20(3) |
| Trail Limb | Maximum Ankle Plantarflexion | -34(5) | -33(10) | -38(.) | -41(8) | -28(3) | -21(14) |
|  | Maximum Hip Extension | -17(5) | -18(11) | -12(.) | -32(13) | -20(4) | -18(6) |

Means(SD). Joint angles are represented in degrees.

Table F.xix Toe Clearance Gait Variables Concussion History Point-biserial Correlations, Condition 3 (obstructed walk)

| Age Group | Lead Limb | Trail Limb |
|---|---|---|
| 20 Year Olds | -0.05 | 0.16 |
| 40 Year Olds | 0.76 | 0.46 |
| 60 Year Olds | -0.57* | -0.21 |

Point-biserial Correlation Coefficient, r. *Significant within age, concussion group differences (p < .05).

Table F.xx Toe Clearance Variables Concussion History Group Means, Condition, Condition 3 (obstructed walk)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | 1 concussion (n = 4) | 2+ concussions (n = 9) | 1 concussion (n = 1) | 2+ concussions (n = 3) | 1 concussion (n = 3) | 2+ concussions (n = 4) |
| Lead Limb | 0.19(.03) | 0.18(.04) | 0.13(.) | 0.21(.04) | 0.20(.02) | 0.15(.04) |
| Trail Limb | 0.16(.03) | 0.19(.09) | 0.12(.) | 0.22(.11) | 0.17(.05) | 0.14(.07) |

Means(SD). Toe clearance is measured in centimeters.

Table F.xxi Toe Clearance Gait Variables Concussion History Point-biserial Correlations, Condition 4 (obstructed walk, mental task)

| Age Group | Lead Limb | Trail Limb |
|---|---|---|
| 20 Year Olds | -0.14 | 0.04 |
| 40 Year Olds | 0.41 | 0.16 |
| 60 Year Olds | -0.47 | -0.37 |

Point-biserial Correlation Coefficient, r. No significant within age, concussion group differences (p's > .05).

Table F.xxii Toe Clearance Variables Concussion History Group Means, Condition 4 (obstructed walk, mental task)

| | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|
| | 1 concussion (n = 6) | 2+ concussions (n = 9) | 1 concussion (n = 1) | 2+ concussions (n = 3) | 1 concussion (n = 2) | 2+ concussions (n = 4) |
| Lead Limb | 0.22(.06) | 0.21(.06) | 0.19(.) | 0.21(.02) | 0.23(.06) | 0.17(.17) |
| Trail Limb | 0.21(.06) | 0.22(.08) | 0.19(.) | 0.21(.06) | 0.22(.03) | 0.17(.17) |

Means(SD). Toe clearance is measured in centimeters.

Table F.xxiii Continuous Tracking RMSEs Concussion History Point-biserial Correlations

| Age Group | Implicit Learning | | Skill Acquisition | |
|---|---|---|---|---|
| | Hand RMSEs | Foot RMSEs | Hand RMSEs | Foot RMSEs |
| 20 Year Olds | 0.00 | 0.01 | -0.03 | -0.02 |
| 40 Year Olds | 0.53** | -0.23 | 0.53** | -0.40** |
| 60 Year Olds | 0.24* | 0.30* | 0.30** | 0.26** |

Point-biserial Correlation Coefficient, r. *Significant within age, concussion group differences (p < .05). **Significant within age, concussion group differences (p < .01).

Table F.xxiv Continuous Tracking RMSEs Concussion History Group Means

| | | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|
| | | 1 concussion (n = 9) | 2+concussions (n = 10) | 1 concussion (n = 1) | 2+concussions (n = 3) | 1 concussion (n = 3) | 2+concussions (n = 4) |
| Implicit Learning | Hand RMSEs | 7.29(2.04) | 7.29(3.01) | 6.81(1.08) | 10.39(2.83) | 8.88(1.78) | 10.39(3.79) |
| | Foot RMSEs | 7.49(2.11) | 7.52(1.52) | 10.95(1.73) | 10.03(1.69) | 8.46(1.45) | 10.13(3.27) |
| Skill Acquisition | Hand RMSEs | 6.56(1.76) | 6.46(1.76) | 6.23(.91) | 9.79(2.84) | 7.97(1.30) | 9.57(3.18) |
| | Foot RMSEs | 7.37(2.13) | 7.28(1.61) | 11.88(2.68) | 9.77(1.95) | 8.09(1.41) | 9.46(3.16) |

Means(SD).

Table F.xxv Continuous Tracking RMSEs Concussion History Point-biserial Correlations

| | | 1Hz | | .5Hz | |
|---|---|---|---|---|---|
| | Age Group | Hand RTs | Foot RTs | Hand RTs | Foot RTs |
| Paced | 20 Year Olds | 0.22** | -0.06** | -0.04 | 0.04 |
| | 40 Year Olds | 0.08 | 0.01 | -0.33** | -0.06 |
| | 60 Year Olds | -.14** | 0.08** | 0.05 | -0.06 |
| Un-Paced | 20 Year Olds | 0.11** | 0.09** | 0.12** | 0.04 |
| | 40 Year Olds | 0.11** | -0.20** | 0.1 | 0.03 |
| | 60 Year Olds | 0.06* | 0.26** | 0.01 | -0.06 |

Point-biserial Correlation Coefficient, r. *Significant within age, concussion group differences ($p < .05$). **Significant within age, concussion group differences ($p < .01$).

Table F.xxvi Discrete Auditory Paced Timing RTs Concussion History Group Means

| | | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|
| | | 1 concussion (n = 9) | 2+concussions (n = 10) | 1 concussion (n = 1) | 2+concussions (n = 3) | 1 concussion (n = 3) | 2+concussions (n = 4) |
| 1Hz | Hand RTs | 0.96(.09) | 1.00(.10) | 1.00(.07) | 1.02(.13) | 0.96(.09) | 0.94(.08) |
| | Foot RTs | 0.97(.11) | 0.95(.11) | 0.98(.07) | 0.99(.19) | 0.97(.08) | 0.98(.10) |
| .5Hz | Hand RTs | 2.01(.20) | 1.99(.26) | 2.09(.19) | 1.81(.37) | 1.94(.18) | 1.96(.16) |
| | Foot RTs | 1.93(.25) | 1.95(.27) | 1.96(.20) | 1.93(.26) | 1.98(.19) | 1.96(.19) |

Means(SD). Reaction times are measured in ms.

Table F.xxvii Discrete Auditory Un-Paced Timing RTs Concussion History Group Means

| | | 20 Year Olds | | 40 Year Olds | | 60 Year Olds | |
|---|---|---|---|---|---|---|---|
| | | 1 concussion (n = 9) | 2+concussions (n = 10) | 1 concussion (n = 1) | 2+concussions (n = 3) | 1 concussion (n = 3) | 2+concussions (n = 4) |
| 1Hz | Hand RTs | 0.14(.20) | 0.18(.15) | -0.03(.13) | 0.01(.14) | 0.14(.23) | 0.16(.16) |
| | Foot RTs | 0.06(.22) | 0.10(.23) | -0.01(.09) | -0.07(.14) | -0.01(.18) | 0.07(.11) |
| .5Hz | Hand RTs | 0.15(.24) | 0.20(.15) | 0.15(.15) | 0.19(.18) | 0.09(.24) | 0.10(.15) |
| | Foot RTs | 0.12(.27) | 0.14(.22) | -0.03(.16) | -0.01(.28) | 0.02(.30) | -0.01(.18) |

Means(SD). Reaction times are measured in ms.

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