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## Examining the Relationship Between State Anxiety and Vestibular and Ocular Motor Impairments and Symptoms in High School Athletes with Concussion

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Examining the Relationship Between State Anxiety and Vestibular and Ocular Motor  
Impairments and Symptoms in High School Athletes with Concussion

A thesis submitted in partial fulfillment  
of the requirements for the degree of  
Master of Science in Kinesiology

by

Mallory McElroy  
University of Arkansas  
Bachelor of Arts in Biology, 2012

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This thesis is approved for recommendation to the Graduate Council.

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## Abstract

Anxiety has been associated with vestibular and ocular motor impairment in the general population. However, there is limited research regarding the connection between the vestibular and ocular motor systems and anxiety following sport-related concussion (SRC). **OBJECTIVE:** The purpose of this study is to compare state anxiety between concussed adolescent male and female athletes with and without vestibular and ocular motor impairments and symptoms. **DESIGN:** Prospective, repeated measures **SUBJECTS:** Thirty adolescent athletes between the ages of 15-18 years, diagnosed with a SRC completed the STAI-State at initial and medical clearance clinical visit. These adolescent athletes were categorized into vestibular and ocular motor provocation (PROV) or no vestibular and ocular motor provocation (NO PROV) based on VOMS change scores at initial clinical visit. **MEASUREMENTS:** A computerized neurocognitive test (ImPACT); VOMS, STAI, PCSS. **PROCEDURES:** Participants completed at least 2 clinical visits (initial and medical clearance) to include ImPACT, VOMS, PCSS, and the STAI-State at initial clinical visit and STAI-State and STAI-Trait at medical clearance clinical visit. **RESULTS:** There was a significant between-subjects effect for provocation groups ( $F_{1,1} = 8.38, p = .007, \eta^2 = .23$ ) on state anxiety. State anxiety scores were higher between PROV ( $M=43.73, SD=14.64$ ) and NO PROV ( $M=31.68, SD=9.15$ ) groups. In addition, there was a significant within-subjects main effect for time ( $F_{1,1} = 6.948, p = .01, \eta^2 = .20$ ) for state anxiety. State anxiety at initial clinical visit ( $M=36.10, SD=12.67$ ) was higher when compared to state anxiety at medical clearance clinical visit ( $M=29.73, SD=9.84$ ). The interaction between provocation groups and time was not significant (Wilks'  $\lambda = .94, p = .20, \eta^2 = .06$ ). **CONCLUSIONS:** These findings suggest that state anxiety is elevated acutely following SRC and may present comorbidly with vestibular and ocular motor impairments. It is imperative for

clinicians to assess these symptoms and impairments acutely following SRC in order to begin quick and effective treatment. Effective treatment options help alleviate symptoms, such as anxiety and vestibular and ocular motor impairments, and provide quicker recovery times.

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*“Yeah, there was this one time we stayed up way past midnight...”*

*-Lenny, That Thing You Do (1996)*

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I love you all.

## Dedication

To my family:

Dad, Mom, Madison, Mariah, PawPaw, and Mawmaw

This was all because of you.

To myself:

Look at what you can do.

*“...A thousand may fall at your side, ten thousand at your right hand, but it will not come near you...For he will command his angels concerning you to guard you in all your ways...”Because he loves me”, says the Lord, “I will rescue him; I will protect him, for he acknowledges my name. He will call on me, and I will answer him; I will be with him in trouble, I will deliver him and honor him...””*

*Psalm 91*

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## Introduction

### Overview of the Problem

The assessment of the vestibular and ocular motor system following sport-related concussion (SRC) is emerging as a key component in the clinical evaluation of this injury, and the relationship of vestibular and ocular motor impairment to other co-morbid conditions is not well understood. Vestibular and ocular motor impairment is reported in 29% to 63% of individuals with SRC (Corwin et al., 2015; Guskiewicz, Ross, & Marshall, 2001; Kleffelgaard, Roe, Soberg, & Bergland, 2012; Mucha, et al., 2014). Vestibular and ocular motor impairment results in a constellation of symptoms and deficits including blurry vision, abnormal near point of convergence, dizziness, and balance problems (Ellis, Leddy, & Willer, 2015; Mucha, et al., 2014; Kapoor & Ciuffreda, 2002). Recently, clinicians and researchers have proposed a clinical treatment model that requires using objective (e.g., vestibular/ocular motor, neurocognitive assessment) and subjective (e.g., symptom reports) clinical data to better determine clinical profiles of SRC with a corresponding and targeted treatment approach (Collins et al., 2014; Collins et al., 2016). Vestibular and ocular are two of the six profiles proposed by a recent clinical consensus statement (Collins et al., 2016). These two profiles, though separate, have linkages in their neuropathology and neurocircuitry, making the differentiation between these profiles difficult.

The vestibular and ocular motor systems share several neuroanatomical components that function together to assist with spatial orientation, balance, and eye motion (Armstrong, McNair, & Taylor, 2008; Ellis, Leddy, & Willer, 2015; Khan & Chang, 2013). The two functional units of the vestibular system include the vestibulo-ocular system and the vestibulospinal system. The vestibulo-ocular reflex (VOR) ensures proper vision during head motion (Cullen & Sadeghi,

2008; Hain & Helminski, 2007; Oghalai & Brownell, 2012). This reflex creates a stable line of sight and is crucial to adapt to our environment and many activities of daily living. The vestibulospinal reflex maintains balance and postural stability (Hain & Helminski, 2007; Oghalai & Brownell, 2012). This reflex facilitates muscular compensation to allow for body movement and is crucial for daily activities. These connections are necessary for performing activities of daily living; however, these reflexes may be impaired following SRC. These systems share similar but not identical neural pathways, which warrants accurate assessment of these reflexes and the vestibular and ocular motor systems.

Several tests are available to evaluate potentially impaired vestibular and ocular motor systems. Options for evaluating vestibulo-ocular function include electro-nystagmography, video-nystagmography, and rotational tests (Fife et al., 2000). Electro-nystagmography and video-nystagmography includes caloric testing and evaluating eye movement tracking targets and in different head positions (Fife et al., 2000). These evaluations include smooth pursuit tracking, saccade testing, and dynamic visual acuity, among others. Rotation tests, including the rotary chair, evaluate how well the visual and vestibular systems cofunction (Fife et al., 2000). Evaluation of the vestibulospinal reflex can be completed via posturography, the Balance Error Scoring System (BESS) or the Sensory Organization Test (SOT) (Nashner, Black, & Wall, 1982; Norre, Forrez, & Beckers, 1987; Riemann, Guskiewicz, & Shields, 1999). These assessments require specialized equipment and clinical personnel, which are not cost or time effective for the acute and serial evaluation of SRC.

In an effort to address the shortcomings of the gold-standard vestibular/ocular motor assessments, the Vestibular/Ocular Motor Screening (VOMS) was developed by Mucha and colleagues (2014). The VOMS evaluates vestibular and ocular motor function and is comprised

of a series of vestibular and ocular motor components. The ocular motor components of the VOMS include smooth pursuits, horizontal and vertical saccades, and near point-convergence distance (NPC) and symptom provocation. The vestibular components of the VOMS include horizontal and vertical VOR and the visual motion sensitivity test, among others. This exam is freely available, takes approximately five to seven minutes to administer, and does not require specialty equipment. The internal consistency of VOMS items ranges from 0.92 to 0.97 in adolescent and collegiate populations (Kontos et al., 2016; Mucha et al., 2014). The false-positive rate for the VOMS is 11% and a combination of the VOR, VMS, and NPC components from this measure is reported to be 89% accurate in identifying SRC from healthy controls (Mucha et al., 2014). In addition, the VOMS is not related to other balance assessments (i.e., Balance Error Scoring System: BESS), which supports the unique contribution of the VOMS for the assessment of SRC. The clinical value of assessing the vestibular and ocular motor systems in individuals with SRC is becoming clearer as this area of clinical research continues to grow. As a result of these clinical advances, new clinical questions and considerations regarding the influence of co-morbidities on vestibular and ocular motor function have emerged.

### **Significance of the Problem**

Clinical observations and anecdotes highlight the role that anxiety, secondary to vestibular and ocular motor dysfunction, has on SRC recovery outcomes (Collins et al., 2014). Although not well established in the SRC literature, the link between vestibular/ocular motor dysfunction and anxiety is well documented in non-athlete samples and patients with vestibular and ocular motor dysfunction. Specifically, 46% of patients with vestibular vertigo reported having a history of generalized anxiety disorder, and were more than 3 times as likely to report a history of anxiety compared to the general population (Bigelow et al., 2016; Monzani, Casolari,

Guidetti, & Rigatelli, 2001). Anxiety has also been associated with other vestibular impairments, including dizziness, decreased balance efficacy, and instability in postural balance control (Hauk, Carpenter, & Frank, 2008; Odman & Maire, 2008; Ohno et al., 2004). In addition to vestibular impairments and symptoms, researchers have reported associations between anxiety and ocular motor impairments. Recently, researchers have reported that individuals with higher trait anxiety exhibited poorer gaze stability than those with lower trait anxiety (Laretzaki et al., 2011), and other researchers report that higher state anxiety is associated with quicker fixation (Quigley et al., 2012; Richards, Benson, & Hadwin, 2012). The purported relationship between anxiety and vestibular/ocular motor impairment is also supported by overlapping neuroanatomical correlates as the ascending pathways of the vestibular nuclei and the parabrachial nucleus of the vestibular and ocular motor systems and the brain regions associated with emotional regulation (i.e., amygdala in the temporal lobe) are located in close proximity in the brain (Balaban & Thayer, 2001; Furman, Balaban, & Jacob, 2001). However, to date, there has been limited research regarding the connection between the vestibular and ocular motor systems and anxiety following concussion.

The hypothesized relationship between vestibular/ocular motor dysfunction and anxiety is likely influenced by other factors such as sex. Previous research has documented that females are more likely to sustain a SRC (Gessel et al., 2007; Lincoln et al., 2011; Marar, McIlvain, Fields, & Comstock, 2012), exhibit greater symptoms (Covassin et al., 2013, 2012; Frommer et al., 2011) and experience longer recovery times (Berz, et al., 2013; Colvin et al., 2009; Henry et al., 2016) compared to males. Given the higher prevalence for anxiety in females compared to males among the general population (Gulliver, Griffiths, Mackinnon, Batterham, & Stanimirovic, 2015; Schaal et al., 2011; Storch, Storch, Killiany, & Roberti, 2005) and females'

higher propensity for exhibiting vestibular and ocular motor impairment (i.e., nearly three times more likely than males) (Monzani, Casolari, Guidetti, Rigatelli, 2001; Neuhauser, 2016; Neuhauser et al., 2005), future studies should investigate the interaction between anxiety and sex on vestibular/ocular motor function following SRC.

## **Purpose**

The purpose of this study is to compare state anxiety between concussed adolescent male and female athletes with and without vestibular and ocular motor impairments and symptoms.

**Specific aim.** To determine the effect of vestibular and ocular motor impairment and symptoms on state anxiety scores at initial and medical clearance clinical visits in concussed adolescent athletes.

***Hypothesis 1.*** State anxiety scores will be higher for concussed adolescent athletes with vestibular and ocular motor impairment and symptoms compared to concussed adolescent athletes without vestibular and ocular motor impairment and symptoms.

***Hypothesis 2.*** State anxiety scores will be higher for concussed adolescent athletes at initial clinical visit compared to medical clearance clinical visit.

***Hypothesis 3.*** There will be a significant interaction between vestibular and ocular motor impairment and symptoms and clinical visit time points on state anxiety scores in concussed adolescent athletes. Athletes with vestibular and ocular motor impairment and symptoms at initial clinical visit will have higher state anxiety scores.

*Exploratory question 1.* Is there an interaction between sex and vestibular and ocular motor impairment and symptoms on state anxiety scores in adolescent athletes with concussion?

### **Operational Definitions**

**Concussion.** Concussion is defined as a functional brain injury induced by biomechanical forces that results in neurological impairment and neuropathological changes (McCrory et al., 2017).

**VOMS provocation.** Provocation on the VOMS is defined as a change of total symptom score greater than or equal to two compared to baseline total symptom score (Yorke, Smith, Babcock, Alsalaheen, 2017).

**State Anxiety.** State anxiety is the transitory emotion of worry and apprehension due to anticipation of a self-perceived threat (C. D. Spielberger, 1966; Staab, 2014a).

**Trait Anxiety.** Trait anxiety is the inherent emotional reaction to self-perceived threat (C. D. Spielberger, 1966; Staab, 2014a).

### **Assumptions**

General assumptions for this study include concussion diagnoses and evaluation will be based on the same criteria and guidelines. Maximal effort will be assumed to be given by patients on all clinical measures. It will be assumed that all patients will answer honestly and truthfully to all clinical measures. It will be assumed that the sample will be representative of all high school athletes with a SRC. In addition, post-injury vestibular provocation will be used to create groups of patients, which will be assumed to precede the state anxiety measured, as anxiety may be secondary to the injury.



**Delimitations**

This study will include high school athletes, aged 15-18 years, who sustain a sport-related concussion and are evaluated and treated at the Inova Sports Medicine Concussion Program. The athletes will seek care at the specialty clinic within four days of sustaining a concussion. The athletes will not have a history of previous brain injury within the past three months, a diagnosed learning disability or hyperactivity disorder, a pre-existing psychological disorder, previous neurological disorder, previous brain surgery, or history of substance abuse.

## **Review of Literature**

### **Definition of Sport-Related Concussion**

For this study, concussion is defined as a functional brain injury induced by biomechanical forces that results in neurological impairment and neuropathological changes (McCrory et al., 2017). While this is the most widely accepted definition, similar definitions have circulated through the research and clinical communities. The American Academy of Neurology defines concussion as a biomechanically induced alteration of brain function that may or may not involve loss of consciousness (Giza et al., 2013). The National Athletic Trainers' Association Position Statement defines concussion using a combination of the definition used by McCrory and colleagues and the definition used by the American Academy of Neurology (Broglio et al., 2014). The American Academy of Pediatrics uses the definition set forth by McCrory and colleagues, as well (Halstead et al., 2010). The American Medical Society for Sports Medicine defines concussion as a traumatically induced disturbance to the brain and neuropathology and is a subset of mild traumatic brain injury (Harmon et al., 2013). These definitions provide comments to the impairments and the biomechanical mechanisms of the injury; however, due to the variability of each injury and its clinical presentations, these definitions fail to provide a clear and specific definition of what a concussion is exactly.

### **Prevalence of Sport-Related Concussion**

According to the Centers for Disease Control and Prevention, concussion has become a leading public health concern as the result of increased injury awareness and education shedding light on the injury's acute and chronic consequences (Centers for Disease Control and Prevention, 2011). Between 1.1 and 1.9 million sports- and recreation-related concussions occur annually in the United States (Bryan, Rowhani-Rahbar, Comstock, & Rivara, 2016), which has

increased for both adult and adolescent populations (Bakhos, Lockhart, Myers, & Linakis, 2010). Within the adolescent population, emergency department visits for concussion increased from 150,000 visits to approximately 250,000 visits each year (Bakhos et al., 2010; W. P. Meehan & Mannix, 2010), with the highest rate within the middle school and high school age groups (Centers for Disease Control and Prevention, 2011). While incidence rates have increased recently, the actual rates may be an underestimation due to many social, cultural, and other factors (Kroshus, Baugh, et al., 2015; Kroshus, Garnett, Hawrilenko, Baugh, & Calzo, 2015; Llewellyn, Burdette, Joyner, & Buckley, 2014; McCrea M, Hammeke T, Olsen G, Leo P, & Guskiewicz K, 2004; Meehan, Mannix, O'Brien, & Collins, 2013). As a result, approximately 50-70% go unreported or undetected (Faul & Coronado, 2015; Langlois, Rutland-Brown, & Wald, 2006; Llewellyn, Burdette, Joyner, & Buckley, 2014; McCrea M, Hammeke T, Olsen G, Leo P, & Guskiewicz K, 2004; W. P. Meehan, Mannix, O'Brien, & Collins, 2013). According to Bryan and colleagues (2016), between 511,590 and 1,240,972 concussions go unreported every year. Marar, McIlvain, Fields, and Comstock (2012) studied 20 sports over two years, with a total of 1936 (13.2%) concussions out of a total 14,635 injuries. These concussions were further stratified into 1,289 (66.6%) concussions occurring during competition and 647 (33.4%) occurring during practice (Marar, McIlvain, Fields, & Comstock, 2012). The researchers calculated a concussion rate of 2.5 concussions per 10,000 athletic exposures from a total of 7,780,064 athletic exposures (Marar et al., 2012). Football, girls' soccer, boys' wrestling, and girls' basketball have been identified as having the majority of reported concussions (Marar et al., 2012). The majority of sport-related concussions occur in the youth and high school athletic populations due to the large number of participants. However, when only assessing football concussion rates, Dompier et al (2015) concluded that football concussion rates were highest

among high school practices and college games when comparing youth, high school, and college football. Injury rates differ among epidemiological studies, presenting an important issue in concussion reporting (Powell & Barber-Foss, 1999; Schulz et al., 2004). Higher injury rates may be the result of increased concussion education, awareness, or diagnosis (McCrea et al., 2004).

### **Biomechanics of Sport-Related Concussion**

A concussion is caused by impacts upon the head or body, resulting in biomechanical outcomes. Head impacts involved in a concussion are either direct, an injurious blow that makes direct contact with the head, or indirect, an impact that sets the head in motion without directly striking it (Zhang, Yang, & King, 2001). These forces, which can be linear or rotational (Bailes & Cantu, 2001), are translated through the body to cause the brain to rapidly accelerate and then rapidly decelerate upon hitting the inside of the skull (Guskiewicz & Mihalik, 2011). Linear accelerations can occur when an individual is hit by a moving object or encounters a solid object during movement (Bailes & Cantu, 2001). This results in focal effects caused by an increase in intracranial pressure (Gennarelli, Thibault, & Ommaya, 1972; Gurdjian, Lissner, Evans, Patrick, & Hardy, 1961; Gurdjian, Webster, & Lissner, 1955; Hodgson et al., 1969; King, Yang, Zhang, Hardy, & Viano, 2003; Rowson et al., 2016; Thomas, Roberts, & Gurdjian, 1966; FJ Unterharnscheidt, 1971). Linear accelerations are common during sports, such as when a football player is hit head on by another player. Rotational accelerations cause diffuse injuries produced from shearing and tensile strain on the brain as a result of shear stress to the brain (Adams, Graham, Murray, & Scott, 1982; Gennarelli et al., 1982, 1972; Guskiewicz KM & Mihalik JP, 2011; Holbourn, 1943; Meaney & Smith, 2011; F Unterharnscheidt & Higgins, 1969). Rotational accelerations are common during sports, such as when an athlete is running forward and another athlete hits them from the side and causes a whiplash-type injury. These accelerations and

decelerations are the primary contributors to the biomechanical induction of concussion; however, the primary contributor and at what magnitude is largely debated.

It is unknown as to which acceleration type is the largest contributor to the incidence of concussion. Gurdjian and colleagues (1954, 1955, & 1961) stated linear acceleration to be the primary cause of concussion. They interpreted the intracranial pressure gradients caused by the linear acceleration to be the main factor in this injury. Ommaya et al (1966) also reported that rotational acceleration could not produce a concussion without the linear acceleration caused by direct impact. The importance of linear acceleration in the production of a concussion was also illustrated by Ono et al (1980) whose experiments with monkeys resulted in similar findings. However, Ommaya and Hirsch (1971) later discovered that rotational and linear acceleration were equally accountable for brain injury. In opposition, Holbourn (1943) was the first to indicate rotational acceleration to be the main contributing mechanism in head injury. Similar findings suggesting that rotational acceleration may produce concussions were found by Gennarelli and colleagues (1971, 1972, & 1982). These studies concluded that rotational acceleration was a larger contributor of concussion than linear acceleration. Though there is still controversy over this issue, it is accepted that both types of acceleration are contributors to concussive injuries. These concussive impacts to the head and the body can cause disruptions to the brain, which cause the typical concussion signs, symptoms, and impairments, such as balance and cognitive impairments.

Studies have attempted to determine injury magnitude and threshold necessary to sustain a concussion. Pellman et al (2003) studied sport impacts reconstructed in crash dummies. This study reported linear acceleration at a minimum injury magnitude of 70-75g to be most common in producing concussion (Pellman, Powell, Viano, Casson, & Tucker, 2003). Also, the mean

injury threshold was indicated to be 98g (Pellman et al., 2003). However, Guskiewicz et al. (2007) reported a mean linear acceleration with a magnitude of 102.8g and mean rotational acceleration with a magnitude of 5311.6 rad/s<sup>2</sup> to be consistent with sustaining a concussion. This opposition highlights the biomechanical controversy within the concussion research community. Zhang, Yang, and King (2004) described a similar finding of 106g magnitudes for linear acceleration and 79000-rad/s<sup>2</sup> magnitudes for rotational acceleration. While this is biomechanically similar to other studies, there are still inconsistencies between them. Also, Broglio et al. (2010) reported rotational acceleration magnitudes at a minimum of 5582.3 rad/s<sup>2</sup> and linear acceleration magnitudes at a minimum of 96.1g necessary to sustain a concussion. The inconsistencies in magnitude and threshold levels highlight the complexity of the biomechanical processes involved in concussions. Concussions are highly individualized injuries and may be caused by various biomechanically induced traumas to the head or body, which emphasizes the possibilities of mechanism, threshold, and magnitude necessary to sustain a concussion.

### **Pathophysiology of Sport-Related Concussion**

The force of a concussion causes multiple neurochemical and neurometabolic changes within the brain (Giza & Hovda, 2014; Katayama, Becker, Tamura, & Hovda, 1990; Takahashi, Manaka, & Sano, 1981). Acutely after impact, potassium exits the cell into the extracellular space, while glutamate is released within the cells, and sodium and calcium are transported via pumps into the cells (Giza & Hovda, 2001, 2014; Katayama et al., 1990; Takahashi et al., 1981). This alteration in cell membrane permeability activates ion channels causing neuronal suppression (Barkhoudarian et al., 2011; Faden, Demediuk, Panter, & Vink, 1989; Giza & Hovda, 2001, 2014), which may be the reason behind post-injury symptoms and impairments (Kawamata, Katayama, Hovda, Yoshino, & Becker, 1992; Yoshino, Hovda, Kawamata,

Katayama, & Becker, 1991). Also, increased calcium travels into the cell mitochondria, which results in mitochondrial dysfunction (Barkhoudarian et al., 2011; Ellis, Leddy, et al., 2015; Giza & Hovda, 2014). In an attempt to restore homeostasis, the ionic fluxes trigger an intracellular energy crisis with an increased demand for glucose metabolism; however, with the mitochondrial dysfunction, glucose metabolism is unable to proceed (Barkhoudarian et al., 2011; Giza & Hovda, 2014). This occurs during reduced cerebral blood flow, which produces a discrepancy between energy supply and demand (Barkhoudarian et al., 2011; Giza & Hovda, 2001, 2014; Prins, Hales, Reger, Giza, & Hovda, 2010; Vagnozzi et al., 2010). Glucose metabolism is then impaired with hypoglycolysis for days following injury and may be associated with impairments in behavior (Yoshino et al., 1991).

Head impacts not only affect intracellular processes, but also the microstructures within the brain. Axonal dysfunction is caused by collapse of neurofilaments and microtubules, causing an increased potential for axon disconnection (Giza & Hovda, 2014; Maxwell, Povlishock, & Graham, 1997; Pettus & Povlishock, 1996; Povlishock & Pettus, 1996; Saatman et al., 2003). This occurs via phosphorylation of the neurofilament side-arms because of the calcium influx (Mata, Staple, & Fink, 1986; Maxwell, McCreath, Graham, & Gennarelli, 1995; Nakamura et al., 1990; Nixon, 1993; Sternberger & Sternberger, 1983). Axon disconnection can lead to neuron shrinkage and decreased functionality (Giza & Hovda, 2014; Singleton, Zhu, Stone, & Povlishock, 2002). While concussion is placed on the brain injury spectrum and results in neuropathological changes, it is a misconception that it is a structural injury and not a functional injury; however, this is incorrect. In 2002, the Concussion in Sport Group proposed a definition of concussion that reflected the view that concussion is a functional injury and is still accepted in the most recent consensus statement (Aubry et al., 2002; McCrory et al., 2017). Evidence of this

is in normal structural neuroimaging and in the signs, symptoms, and impairments that reflect a functional, not structural, injury.

### **Signs, Symptoms, and Impairments of Sport-Related Concussion**

Sport-related concussion is characterized by a heterogeneous array of signs, symptoms, and impairments (McCrory et al., 2017). Clinical presentations differ between each injury, making recognition of these signs, symptoms, and impairments a critical aspect of concussion management. Common concussion symptoms include, but are not limited to, headache, nausea, vomiting, dizziness, photophobia, phonophobia, fatigue, and mental foginess (McCrory et al., 2017). Common concussion signs include, but are not limited to, loss of consciousness retrograde or anterograde amnesia, disorientation, confusion, and memory difficulties (McCrory et al., 2017). Common concussion impairments include cognitive, emotional, vestibular, ocular motor, and balance difficulties (McCrory et al., 2017). Concussion's varied appearance demonstrates the difficulty in diagnosis of the injury. Common symptomatology is categorized into five domains including: clinical symptoms, physical signs, cognitive impairments, neurobehavioral features, and sleep disturbances (McCrory et al., 2017). An athlete that exhibits impairments in one or more of these domains should be suspected to have sustained a concussion (McCrory et al., 2017).

There are multiple symptom scales used in research and clinical practice to evaluate symptoms following SRC. The 21-item revised post-concussion scale (PCS) was the first published symptom scale and has been revised and modified into the 21-item post-concussion symptom scale (PCSS) as well as incorporated into computerized neurocognitive testing (Lovell et al., 2006; Lovell & Collins, 1998; Pardini et al., 2004). The graded symptom checklist (GSC) was also a modification of the PCS, while within the research it has been modified to incorporate



17 and 18 items (Lovell & Collins, 1998; McCrea et al., 2003; Patel et al., 2007). Recently, the GSC was categorized into four symptom domains including cognitive, somatic, emotional, and sleep problems (Patel et al., 2007). A modification of the GSC is the head injury scale (HIS), a 9-item and 16-item scale with three symptom clusters including somatic, neuropsychological, and cognitive (Piland et al., 2003). Another adaptation of the PCS became the 20-item McGill abbreviated concussion evaluation (McGill ACE) post-concussion symptoms scale (Leclerc, 2004). The symptoms in this scale were grouped into three categories including somatic, cognitive, and affective symptom clusters (Leclerc, 2004). The symptom factors generally include a cognitive, affective, and somatic cluster, which is not surprising given the prevalence of these types of symptoms following SRC. For this study, we will be using symptom reports from the PCSS, which was analyzed recently. Kontos et al. (2012) conducted a factor analysis on the Post-Concussion Symptom Scale with more than 1400 high school and collegiate athletes and reported potential symptom factors including: cognitive-fatigue-migraine, affective, somatic, and sleep factors. The cognitive-fatigue-migraine factor includes self-reported headache, difficulty concentrating, fatigue, and dizziness. The affective factor includes self-reported sadness and nervousness. The somatic factor includes self-reported nausea and numbness. The sleep factor includes trouble sleeping and sleeping less than usual. The separation of symptoms into symptom factors helps with making determinations to individualized management and treatment programs.

### **Assessment and Management of Sport-Related Concussion**

Unlike other common sports injuries, concussions lack a single diagnostic test to determine the presence of the injury. The lack of a definitive diagnostic tool coupled with the unique injury presentation warrants the use of multiple tools to diagnose a SRC (McCroory et al.,

2017). This approach utilizes multiple systematic assessment tools, which includes a clinical interview, symptom report (described in the previous section), and cognitive, vestibular, ocular motor, and balance testing to allow for individualized management, treatment, and recovery trajectories.

A clinical interview is imperative for establishing clinical diagnosis and assessing potential recovery profiles. The clinical interview occurs immediately following a suspected SRC, usually by an athletic trainer or other readily available clinicians and a thorough clinical interview aims to establish a clinician-athlete relationship and determine if a concussion occurred (Broglio et al., 2014; Collins et al., 2014). It usually includes determining mechanism of injury, including location, force, and direction of trauma. Injury details are used in the differential diagnosis of concussion and can inform and corroborate the athlete's removal from play status (Broglio et al., 2014; Collins et al., 2014). Medical and biopsychosocial history, including age, sex, and prior history of concussion, is also obtained during the clinical interview. This information helps the clinician determine possible recovery trajectories, such as prolonged recovery for adolescents and females (Collins et al., 2014; Guskiewicz, Weaver, Padua, & Garrett, 2000; Lincoln et al., 2011). This is an important initial step in the assessment of SRC, which is followed by other assessment tools, including those testing motor control and mental status.

The impairments in brain function following SRC can lead to impaired motor control and mental status, both typically assessed during the clinical interview. Motor control impairments can be observed by abnormal postural control (McCrea et al., 2003; Peterson et al., 2003; Guskiewicz, Ross, & Marshall, 2001) due to the miscommunication between sensory information (Kuo, 2005). Common balance tests include the Sensory Organization Test (SOT)

and the Balance Error Scoring System (BESS). The SOT has been validated in quantifying balance disruptions; however, the high cost and inconvenience of the test deters clinicians from using this measure (Broglio, Ferrara, Sapiar, & Kelly, 2008; Broglio et al., 2014). The BESS is also a validated measure of balance impairment; however, it is inexpensive and portable (McCrea et al., 2003, 2005). The BESS has been transformed into the modified BESS (mBESS), making it even more inexpensive and portable. Objective measures of mental status are valuable to the differential diagnosis of concussion because of the mental status deficits following SRC. The Standardized Assessment of Concussion (SAC) is a common assessment of mental status, which includes evaluation of orientation, memory, and concentration (McCrea, 2001). The SAC is sensitive to concussion and increases with a symptom report and motor control test (McCrea et al., 1997). However, sensitivity decreases 24 hours after the injury (McCrea et al., 2005), making it a valuable sideline assessment tool. The Sport Concussion Assessment Tool-5 (SCAT-5) is a shortened clinical interview, which includes symptom report, motor control and mental status testing, and is a common sideline assessment (Echemendia, et al., 2017). This tool was developed in order to combine separate assessment tools, including physical and cognitive evaluations, to provide medical professionals with a brief comprehensive standardized SRC evaluation tool (Echemendia et al., 2017). This tool incorporates a symptom assessment, Maddocks Questions, the Standardized Assessment of Concussion (SAC) and the Balance Error Scoring System (BESS). The SCAT-5 is used to evaluate SRC in individuals 13 years or older and is useful in concussion diagnosis and recovery tracking (Echemendia et al., 2017; McCrory et al., 2017). The SCAT-3 showed a ceiling effect on the word list of the SAC, leading to the introduction of a 10-word list instead of the initial 5-word list (Echemendia et al., 2017).

Computerized neurocognitive examinations are an objective measure of concussion-induced deficits. They provide clinicians with cognitive performance data, which can be useful during diagnosis and management (Collins et al., 2014). Domains tested by these exams include information processing, planning, memory, and switching mental set (Elleberg et al., 2009). Several computerized neurocognitive examinations that exist within the research and clinical community include the Automated Neuropsychological Assessment Metrics (ANAM), Cogstate Axon, Concussion Vital Signs, Headminder Concussion Resolution Index (CRI), and Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT). A commonly used neurocognitive exam is the ImPACT test, which provides cognitive data, as well as demographics and symptoms (Collins et al., 2014). ImPACT is a computerized test of verbal memory, visual memory, visual-motor processing speed, and reaction time aimed at examining neurocognitive performance (Lovell, Collins, Podell, Powell, & Maroon, 2000). Iverson, Lovell, & Collins (2003) found ImPACT to have a reliability coefficient of over 0.60, while Nelson et al. (2016) found modest test-retest reliability and moderate group level sensitivity less than 24 hours following SRC. Clinicians use baseline exams and reliable change indices on serial post-concussion exams to determine the progression of cognitive function back to pre-concussion levels (Clark & Guskiewicz, 2015). In military populations, individuals returned to their baseline neurocognitive performance using ANAM within four days of injury (Warden et al., 2001), while non-military populations testing using ImPACT returned to their baseline neurocognitive performance within five days of injury (Covassin et al., 2008). These similar recovery timelines illustrate the reliability in computerized neurocognitive examinations. However, these exams do not reflect the metabolic cascade that occurs following SRC (Elleberg et al., 2009), and can be affected by several factors, including motivation, fatigue,

and substance use (Gualtieri & Johnson, 2006). The use of computerized neurocognitive exams is useful in the multimodal assessment and management of SRC; however, these results should be used in conjunction with other assessment tools in order to provide comprehensive injury assessment and recovery details.

Vestibular and ocular motor function can be assessed using several gold standard techniques. These options include electro-nystagmography, video-nystagmography, and rotational tests (Fife et al., 2000). The objective of electro- and video-nystagmography is to record eye movement to test for nystagmus and eye tracking (Fife et al., 2000). These tests include caloric testing and evaluating eye movement tracking targets and in different head positions (Fife et al., 2000). Several domains tested are smooth pursuit tracking, saccade testing, dynamic visual acuity, and nystagmus, among others. Another assessment of the vestibulo-ocular system is rotation tests. The objective of this examination using a rotary chair is to evaluate the integration of the vestibular and ocular motor systems and their functions (Fife et al., 2000). Evaluation of the vestibulospinal reflex includes posturography, the Balance Error Scoring System (BESS) and/or the Sensory Organization Test (SOT) (Nashner, Black, & Wall, 1982; Norre, Forrez, & Beckers, 1987; Riemann, Guskiewicz, & Shields, 1999). While these are the gold standard of assessing vestibular and ocular motor function, there are several hindrances to using these assessment measures in the acute and serial evaluation of SRC.

The VOMS was developed in order to address the weaknesses of the gold standard vestibular and ocular motor function evaluations (Mucha et al., 2014). Vestibular function is assessed using tests of horizontal and vertical VOR and the visual motion sensitivity test. Ocular motor function is assessed using tests of smooth pursuit, horizontal and vertical saccades, and near point of convergence distance and symptoms. The VOMS is less time consuming and less

expensive than the previously mentioned vestibular and ocular motor function tests and it does not require special equipment. The internal consistency of VOMS items ranges from 0.92 to 0.97 in adolescent and collegiate populations (Kontos et al., 2016; Mucha et al., 2014). The false-positive rate for the VOMS is 11%, and a combination of VOR, VMS, and NPC components from this measure are reported to be 89% accurate in identifying SRC from healthy controls (Mucha et al., 2014). The VOMS is a unique and necessary tool in the multimodal assessment of SRC.

Injury management usually follows a strict guideline for safe return-to-play, directed usually by multimodal assessment results. The multimodal approach is beneficial in identifying those with increased risk of protracted recovery or certain deficits needing other specific treatments who do not respond to typical return-to-play protocols (Collins et al., 2016, 2014; Henry, Elbin, Collins, Marchetti, & Kontos, 2016). However, most concussions follow a step-wise progression of aerobic exertion (Broglio et al., 2014; Harmon et al., 2013; McCrory et al., 2013). An athlete may not begin the protocol until asymptomatic, and the advancement depends upon the athlete remaining asymptomatic through each step (Broglio et al., 2014; Harmon et al., 2013; McCrory et al., 2013). The protocol endorsed by the most recent Consensus Statement on Concussion in Sport from 2016 includes six stages, progressively increasing activity levels with no symptom provocation (McCrory et al., 2017). Stage one aims towards reintroducing daily activities, such as school. Stage two aims to increase heart rate by incorporating light aerobic exercise. Stage three includes adding sport-specific activity with the exception of head impact activities. Stage four reincorporates non-contact training drills in order to increase coordination and cognition. Stage five is after medical clearance, allowing the athlete to participate in full contact training in order to help the athlete regain sport-specific confidence and abilities. Stage

six is complete return to sport. This protocol consists of 24 hours between stages and the decline in test performance or return of symptoms informs the athlete and clinician to be repeated after 24 hours rest (McCroory et al., 2017). The progression through stages based on symptoms and tolerance is necessary to keep athletes safe.

Concussion should be managed through a teamwork of specialized professionals trained in concussion treatment and management (Collins et al., 2016). This approach allows for the safe, most effective, and targeted treatment plan appropriate for each injury (Reynolds, Collins, Mucha, & Troutman-Ensecki, 2014; Stewart, McQueen-Borden, Bell, Barr, & Juengling, 2012; Wilkins et al., 2014). This requires advocates from several specialties chosen through the unique clinical injury profile; however, it is suggested the central figure be the primary physician or medical provider (Stewart et al., 2012). Depending upon the symptoms and deficits, other specialists, such as athletic trainers, vestibular therapists, neuropsychologists, ocular therapists, and others should be included within the framework of the recovery team (Collins et al., 2016). These specialists bring new perspectives to the recovery profile to allow a multimodal and targeted approach.

### **Treatment of Sport-Related Concussion**

The presentation of specific clusters of symptoms has suggested the presence of specific concussion profiles. Although concussions can present similarly, they can be characterized into clinical profiles by symptom presentation and evaluation findings in order to guide concussion evaluation, management, and treatment (M. W. Collins et al., 2016, 2014; Ellis et al., 2015). Collins et al. (2014) described the clinical trajectories to include cognitive/fatigue, vestibular, ocular motor, post-traumatic migraine, affective, and cervical. The cognitive/fatigue profile includes symptoms relating to decreased energy levels, sleep disturbances, difficulty

concentrating, and non-specific headaches. Symptoms may increase as the day progresses and/or with cognitive activity. Neurocognitive test data may show deficits in processing speed, reaction time, and memory. Treatment for this profile involves limited physical and cognitive rest, behavioral regulation, decreased stress, and, in prolonged cases, cognitive and speech therapy. Symptoms categorized into the vestibular profile include dizziness, balance issues, foginess, nausea, and anxiety, with symptoms getting worse when in stimulating environments or with quick movements. Processing speed and reaction time deficits may result on neurocognitive tests. Vestibular therapy is the typical treatment for this profile. The ocular motor profile present with frontal headaches, eye pressure, visually-based academic difficulties, and distractibility. Vision therapy and/or vestibular therapy is recommended for this profile. The post-traumatic migraine profile involves unilateral pulsing headaches with nausea and photosensitivity and/or phonosensitivity. These symptoms may be increased with physical activity, stress, and dietary and sleep dysregulation. It is important to check for personal and/or family history of migraines. Memory deficits are common with neurocognitive data. It is recommended to start the return-to-play exertion protocol to increase cardiovascular activity and to regulate sleep, diet, and stress. Those in the affective profile typically have a global increase in anxiety, rumination, and sleep disturbances. It is important to check for personal and/or family history of anxiety, in order to guide assessment and treatment. The typical return-to-play exertion protocol is critical for treatment, as it has been shown to decrease arousal, even without injury. Regulating sleep, exercise, diet, and stress is also important in order to speed recovery due to more regulated autonomic functioning. Characteristic symptoms of the cervical profile include headache and neck pain that is uncharacteristic of other trajectories in regards to headache location and



neurocognitive and/or vestibular and ocular motor impairments. The use of range of motion exercises, posture re-education, and biofeedback is recommended to treat these headaches.

### **Risk Factors and Recovery Time following Sport-Related Concussion**

Concussion is a complex injury due to the highly variable nature of the injury, the sequelae post-injury, and the individuals to whom the injury occurs. This complexity influences the recovery timelines. Typically, concussions resolve within one to four weeks (Collins, Lovell, Iverson, Ide, & Maroon, 2006; Henry et al., 2016; McCrory et al., 2017); however, approximately 20 percent of athletes experience protracted recovery (Babcock et al., 2013; Barlow et al., 2010; Collins, Lovell, Iverson, Ide, & Maroon, 2006; Grubenhoff et al., 2014; Henry, Elbin, Collins, Marchetti, & Kontos, 2016; Iverson et al., 2006; Meehan, Mannix, Stracciolini, et al., 2013; Yeates et al., 1999). The exact timeline has varied throughout research, ranging from days to months. The recovery timeline has been shown to vary depending upon the assessment quality, management, and the population studied (Alsalaheen et al., 2010; Buckley, Munkasy, & Clouse, 2016; Collins et al., 2016; DiFazio, Silverberg, Kirkwood, Bernier, & Iverson, 2016; Reddy, Collins, Lovell, & Kontos, 2013). Research has shown injury resolution within seven to 21 days when immediate cognition and postural stability assessments, such as the BESS and SAC, are used (Broglia & Puetz, 2008; Michael McCrea et al., 2003, 2005; Michael McCrea, Iverson, Echemendia, Makdissi, & Raftery, 2013; Prichep, McCrea, Barr, Powell, & Chabot, 2013; Quatman-Yates et al., 2014). When only symptom reports are utilized, athletes recover between five to 14 days (Eisenberg, Meehan, & Mannix, 2014; Fazio, Lovell, Pardini, & Collins, 2007; B. C. Lau, Collins, & Lovell, 2012; B. Lau, Lovell, Collins, & Pardini, 2009; Makdissi, Cantu, Johnston, McCrory, & Meeuwisse, 2013; Michael McCrea et al., 2003, 2005, 2013; W. P. Meehan 3rd et al., 2013). Comprehensive assessments have shown to prolong

recovery to last up to 28 days likely due to assessing multiple components that can be affected following injury (Henry et al., 2016).

There are specific risk factors that accompany protracted recovery. These can be divided into primary risk factors, those that exist pre-injury, and secondary risk factors, those that occur post-injury (Collins et al., 2014; Elbin, Covassin, Gallion, & Kontos, 2015). Primary risk factors include age younger than 18 years, female sex, history of more than two concussions, history of migraines, diagnosed neurocognitive impairments, such as learning disorder or attention deficit hyperactivity disorder, history of vestibular dysfunction, and history of ocular motor dysfunction. Secondary risk factors include continuation of play after injury, on-field dizziness, difficulty concentrating, photosensitivity, phonosensitivity, post-traumatic amnesia, post-traumatic migraine, sleep disturbances, loss of consciousness, and neurocognitive impairment, including three or more reliable change index (RCI) changes on computerized neurocognitive tests. The length of recovery is dependent upon the individual and the unique injury profile; however, an athlete may have predisposed tendencies towards protracted recovery.

### **Vestibular System**

The vestibular system is an intricate arrangement of structures, neural pathways, and other systems to maintain balance, spatial orientation, and visual processing during motion (Khan & Chang, 2013). The following is an in-depth overview of the vestibular system, including the structures and neurocircuitry involved in the complex processes.

The peripheral vestibular system contains an intricate network of structures used to detect motion. These structures include a bony labyrinth, a membranous labyrinth, hair cells, utricle, saccule, semicircular ducts (Khan & Chang, 2013). The bony and membranous labyrinth sit within the otic capsule in the petrous part of the temporal bone within the inner ear. The bony

labyrinth is comprised of the cochlea, the vestibule, and the semicircular canals. These are filled with a perilymph-like fluid (Hain & Helminski, 2007). Within this fluid is the sensory epithelium and structures of the vestibular apparatus, which make up the membranous labyrinth. The membranous labyrinth contains endolymph, an intracellular fluid-like substance (Hain & Helminski, 2007). The vestibular apparatus housed in the membranous labyrinth is comprised of the utricle, saccule, and the lateral, superior, and posterior semicircular ducts. The utricle and saccule are located within the vestibule, while the semicircular ducts are located in the bony semicircular canals.

The hair cells of the vestibular system are embedded in a neuroepithelium membrane of the macula and crista ampullaris. There are two structural forms of hair cells, Type I and Type II. Type I hair cells are those with a round base encased in an afferent nerve fiber nerve calyx. Most hair cells are Type II hair cells and are those with a columnar structure and bouton synaptic connections to afferent nerve fibers. Both types of hair cells are rod-shaped sensory mechanoreceptors that include a single large kinocilium and 70-100 stereocilia on its apex (Oghalai & Brownell, 2012). The kinocilium is a non-motile cilium-like structure with a 9 + 2 doublet arrangement of microtubule pairs (Oghalai & Brownell, 2012). The stereocilia are organized in rows around the kinocilium, with the tallest stereocilia closest and shortest stereocilia farthest. The stereocilia are composed of actin-rich filaments coated in myosin isoforms. The stereocilia are connected tip to adjacent body. Head motion that causes the stereocilia to tilt toward the kinocilium causes the stereocilia to tilt and shift the “tip links” (Khan & Chang, 2013). Shifting of the “tip links” causes the transduction channels to be mechanically opened to cause an influx in potassium. This ion shift causes a depolarization cascade, including the opening of calcium channels, which promotes neurotransmitter release into synapses to cause

increased firing rate of afferent vestibular nerve fibers. Head motion that causes the stereocilia to tilt away from the kinocilium reduces the tip link tension, closing the potassium and calcium channels due to hyperpolarization of the hair cell. This reduces the firing rate of vestibular nerve fibers because of decreased neurotransmitter release (Mescher 2010). To help modulate sensitivity, hair cells have efferent vestibular nuclei connections (Mescher 2010). The anatomy of these hair cells is specific to type of motion and helps regulate the vestibular system.

The utricle and saccule sense spatial orientation of the head and respond to linear acceleration, gravitational forces, and head tilting. Both the utricle and saccule contain macula, a sensory neuroepithelium that detects motion. The macula contains a gelatinous membrane coating that contains otoliths or otoconia, which are calcium carbonate particles embedded into the membrane. The hair cells protrude through the otolithic membrane and gravity prevents motion of the stereocilia while the head is static (Hain & Helminski, 2007). Linear and tilting head motions cause inertial drag and shear force between otolithic membrane and macular surface, which results in hair cells bending (Oghalai & Brownell, 2012). The stereocilia of the hair cells in the macula are positioned according to the curvilinear striola (Tascioglu, 2005), a thinning area of the utricle and a thickened area of the saccule (Oghalai & Brownell, 2012). In the utricle, the stereocilia are oriented toward the striola. In the saccule, the stereocilia are oriented away from the striola. Varied orientation of the stereocilia allows the detection of various head motions, resulting in accurate information relayed to the central nervous system. The macula in the utricle detects horizontal head motion and the macula in the saccule detects vertical head motions (Barrett et al., 2012). However, when the motion stimulus remains stable over a timed threshold, the depolarized membrane potentials return to normal in order to adapt to present and future head motions (Oghalai & Brownell, 2012).

The semicircular ducts and canals are arranged at right angles to the next, allowing it to detect angular head acceleration or rotation. The posterior and superior ducts are oriented at a 45 degree angle to the sagittal plane. The lateral canals are oriented at a 30 degree angle to the axial plan (Lee et al., 2011). The contralateral semicircular ducts are paired as the right superior and left posterior, left superior and right posterior, and left horizontal and right horizontal ducts (Smouha & Wanna, 2009). This orientation allows rotational motion to be detected in all planes. At the end of each duct is the ampulla, a dilation of the duct opening into the utricle that contains the crista ampullaris. The crista ampullaris is similar to the macula because it is too coated by a gelatinous membrane with embedded hair cells called the cupula; however, the cupula is thicker and does not contain otoliths (Mescher, 2010). The kinocilia of the hair cells in the cupula are oriented in a similar fashion as the stereocilia of the utricle and saccule. The lateral ducts have kinocilia that are positioned toward the utricle and the superior and posterior ducts have kinocilia positioned toward the duct. Head rotation causes endolymph to displace the cupula, which bends the hair cells in an opposite direction of the rotation (Barret et al., 2012), ultimately causing a depolarization of the hair cells and increased afferent fiber firing. A constant rotational motion causes the depolarization to return to normal. Rotational deceleration causes the hair cells to bend in the same direction of the motion (Barret et al., 2012), which causes a hyperpolarization and reduced afferent fiber firing. The semicircular ducts are contralaterally paired, so that when endolymph moves in one duct, it inhibits hair cells of the paired duct.

The vestibular ganglion acts as an impulse recipient. It is located in the internal auditory meatus (Ropper & Samuels, 2009) and is composed of approximately 20,000 bipolar cell bodies. These cell bodies receive afferent nerve impulses from crista ampullaris and macular hair cells. The vestibular ganglion is comprised of superior and inferior fiber sections. The superior

division is connected via peripheral fibers to the crista ampullaris of the superior and lateral semicircular ducts and macula of the utricle. The inferior division is connected via peripheral fibers to the crista ampullaris of the posterior semicircular ducts and macula of the saccule.

Merging axons from the vestibular ganglion form the vestibular nerve. At the point it merges with the cochlear nerve it becomes the vestibulocochlear nerve. The vestibulocochlear nerve parallels through the internal auditory canal with the nervus intermedius, a facial nerve, and the labyrinth artery. These emerge at the pontomedullary junction within the brainstem where the vestibular nerve and cochlear nerve separate. Most of the vestibular nerve fibers connect to the ipsilateral vestibular nuclear complex in the pons, while other vestibular nerve fibers connect to the flocculo-nodular lobe of the cerebellum and adjacent vermian cortex (Ropper & Samuels, 2009).

The vestibular nuclear complex processes the vestibular nerve input. This is achieved by the Schwalbe, Bechterew, Deiter, and descending nuclei (Lee et al., 2011; Waxman, 2010), or simply the medial, superior, lateral, and inferior nuclei respectively (Tascioglu, 2005). These four nuclei are located beneath the fourth ventricle and continue from the medulla to the pons in two columns, the medial and lateral columns. The medial column consists of the medial vestibular nucleus and receives afferent impulses from the crista ampullaris in the lateral semicircular ducts. The vestibulo-ocular reflex and the vestibular spinal reflex are mediated by the ascending medial axonal nerve fibers. This allows for head and neck motion coordination. The lateral column consists of the lateral, superior, and inferior nuclei (Tascioglu, 2005). The lateral nuclei in the lateral column receive afferent input from the crista ampulla, macula, and vestibulocerebellum and projects efferent nerves to form the lateral vestibular tract within the ipsilateral spinal cord. This aids in maintaining posture and balance via the vestibular spinal

reflex. The superior nuclei in the lateral column receive afferent input from the superior and posterior semicircular ducts' crista ampullaris and projects to extraocular muscles to mediate the vestibulo-ocular reflex (Lee et al., 2011). The inferior nuclei in the lateral column receive afferent input from the utricle and saccule macula and projects efferent impulses to the other vestibular nuclei and to the cerebellum (Lee et al., 2011).

The cerebellum in regards to the vestibular system functions as a processing unit that allows for adaptations due to motion. Termed the vestibulocerebellum, it acts to balance vestibular performance and inputs through inhibition of the vestibular system when necessary (Hain & Helminski, 2007). It has ipsilateral efferent connections with bilateral vestibular nuclei and the ipsilateral fastigial nucleus. The axons from the fastigial nucleus send information through the juxarestiform body to the contralateral vestibular nuclei (Ropper & Samuels, 2009). The cerebellar flocculus regulates the VOR gain (Hain & Helminski, 2007), while the cerebellar nodulus regulates the VOR duration and processes afferent information from the macula (Hain & Helminski, 2007). The anterior superior vermal cortex helps regulate the vestibulospinal reflex.

### **The vestibulo-ocular reflex (VOR).**

Retinal images during movements of the head are stabilized through the vestibulo-ocular reflex (VOR). The semicircular ducts, the vestibular nuclei, and the extraocular muscles are involved in a three-neuron reflex arc that detects head motion and initiates eye movement in an opposite direction (Cullen & Sadeghi, 2008). During head rotation, the endolymph flows in the ampulla of the semicircular ducts and will repel the cupula in the opposite direction, causing depolarization and hyperpolarization of hair cells. The adjusted membrane potential causes an increased firing frequency of afferent fibers, sending impulses to the vestibular nuclei and

cerebellum. These impulses are sent to the oculomotor nuclei and results in opposing eye movements (Hain & Helminski, 2007).

### **The vestibulospinal reflex.**

Many structures and connections of the vestibular system are important in the coordination of the vestibulospinal reflex. Information from the macula, crista ampullaris, visual system, axial and limb muscles, cerebellum, and the lateral and medial vestibular spinal tracts have overlapping pathways. The primary pathway involves the process of the macula transmitting efferent vestibular signals to the lateral vestibular nucleus, which sends information ipsilaterally to neurons in the spinal cord on all spinal levels. Ipsilateral trunk and proximal limb extensors are activated through monosynaptic activation by these neurons, and contralateral proximal extensors are inhibited through disynaptic inhibition (Oghalai & Brownell, 2012). Also, head and neck motion is coordinated by the activation of cervical axial muscles. This occurs when the semicircular ducts detect angular head rotation and transmits impulses to the medial vestibular nucleus, which transfers bilaterally to cervical spinal cord motor neurons.

### **Ocular Motor System**

The ocular motor system consists of cranial nerves, ocular muscles, and neural regions to process external stimuli into eye movements. The orbital muscles are responsible for eyeball movement. Dr. James Carl (1997) describes these muscles necessary for all eye movements. The eyeball is grounded within the orbit by antagonist muscles attached to it, creating viscoelastic forces that resist eyeball motion. The eyeball is surrounded by protagonist muscles that provide a force for the eyeball to return back to center. The action between the protagonist and antagonist muscles creates each eye movement. There are six muscles organized into three pairs that constitute these protagonist and antagonist muscles. The medial, lateral, superior, and inferior



recti muscles allow the eyeball to rotate toward it. These muscles run from the orbital apex to the eyeball's front hemisphere. The inferior and superior oblique muscles allow the eyeball to rotate away from the muscles and run from the front of the orbit to the back hemisphere of the eyeball. The contraction of these muscle pairs allows for rotation about their specific axes because of the close proximity of the pairs' attachment points. The muscles and muscle pairs allow for all eye movement.

The cranial nerves serve as bridges between external stimuli and eye movement coordination. The previously described muscles are innervated by six cranial nerves, consisting of cranial nerves III, IV, and VI on the left and right sides of the eyeball. The brainstem houses the cranial nerve nuclei near the midbrain and they continue to their innervated muscle, carrying the afferent and efferent nerves. Cranial nerve III, named the oculomotor nerve, innervates the superior, inferior, and medial rectus muscles and the inferior oblique muscles, notably the pupillomotor and eyelid muscles. Disruption of the oculomotor nerve causes the eyeball to be positioned in a "down and out" position, eyelid drooping, or unreactive pupils. Disruption can be caused by diabetes or posterior communicating artery aneurysms. Cranial nerve IV, named the trochlear nerve, innervates the superior oblique muscle. Disruption of the trochlear nerve may cause diplopia, twisting of images, or separation of images. Disruption to the trochlear nerve is commonly due to head trauma. Cranial nerve VI, named the abducens nerve, innervates the lateral rectus muscle. Disruption to the abducens nerve causes crossed eyes, double vision, and diplopia. Disruptions to the abducens nerve are common after trauma and increased intracranial pressure. These nerves transform the stimuli into eye movements due to their innervations between the brain and extraocular muscles.

Higher order processing is necessary for translating stimuli into eye movement. The visual information is processed within the primary visual cortex within the occipital lobes and then sent to the brainstem and the cerebellum. The cranial nerve nuclei are housed within the brainstem and are housed in pairs. The organization of pairs is due to the collaborative efforts of different muscles to achieve a specific eye movement. The medial and lateral recti muscles are paired via the longitudinal fasciculus nerve fiber tract, as is the right inferior rectus and left superior oblique muscles. The nerve pairing is also advantageous for vestibular reflexes to avoid unnecessary actions, such as indirect processing pathways and unnecessary eye movements.

Common eye movements tested with concussion assessments include saccades, pursuits, and vergence. Horizontal saccadic eye movements are generated by frontal eye field activity, such that right side activation would result in left frontal eye field activity. Vertical saccadic eye movements are generated by bilateral activation of both right and left frontal eye fields. Cortical areas involved in horizontal saccades are the superior colliculi, which receives information from the contralateral frontal eye fields and basal ganglia, and the paramedian pontine reticular formation, which receive information from the superior colliculi. Cortical areas involved in vertical saccades are the rostral interstitial nucleus of the medial longitudinal fasciculus and the interstitial nucleus of Cajal. Pursuit movements are generated by negative feedback due to eye motion and speed disparities. These disparities are overcome through the network between the middle temporal visual area and the visual centers of the occipital lobe striate cortex.

Convergence is the adduction of both eyes in order to maintain binocular vision on nearing targets (Ventura, Balcer, & Galetta, 2014). The anatomical pathway involved in convergence is not well understood, but it is thought to include the cerebellum, cerebrum, and brainstem (Ventura et al., 2014). These eye movements are commonly assessed following concussion.

Oculomotor dysfunction following SRC may induce abnormal eye movements, resulting in common post-concussion signs, symptoms, and impairments. Heitger et al. (2004) studied patients within 10 days following mTBI and found impaired antisaccades and prolonged saccadic latency. This research was expanded to patients three to five months following mTBI and found abnormal antisaccades, memory-guided saccades, and self-paced saccade tests (Heitger, 2009). Abnormal saccadic function has been associated with impaired executive function, attention, and memory (Heitger et al., 2004; Heitger, Anderson, Jones, 2002; Heitger et al., 2009; Kraus et al., 2007; Drew et al., 2007). Smooth pursuit abnormalities, such as decreased target prediction and increased eye position errors, were found in patients with traumatic brain injury (Suh et al., 2006a, 2006b). According to Capo-Aponte (2012), 60% of patients with mTBI had pursuit abnormalities. These abnormalities have been associated with attention and executive function deficits (Suh et al., 2006b). Vergence dysfunction has been found in 47% - 64% of patients following concussion (Barnes, 2008; Brahm et al., 2009; Capo-Aponte, Urosevich, Temme, Tarbett, & Sanghera, 2012; Cohen, Groswasser, Barchadski, Appel, 1989; Suh et al., 2006a, 2006b).

The interaction between the vestibular and ocular motor systems is based on the brainstem organization. The eyes move in horizontal, vertical, and mixed directions and these movements are coordinated by the extraocular muscles and cranial nerves. The movements are also associated with movement within the semicircular canals of the vestibular system, with the brainstem most likely organizing the eye movements into canal-plan coordinates.

## **Anxiety**

Anxiety has become a hot topic in the mental health realm because of its devastating consequences, but the underlying neurocircuitry has remained ambiguous. Anxiety is the

emotion characterized by uncontrollable worry, tension, and apprehension about daily activities and problems, leading to physiological symptoms concerning autonomic arousal, chest and abdomen, brain and mind, muscle tension, and other non-specific symptoms (World Health Organization, 1993). This emotion can be divided into two types: state anxiety and trait anxiety. State anxiety is the transitory emotion of worry and apprehension due to anticipation of a self-perceived threat (Spielberger, 1966; Staab, 2014). Trait anxiety is the inherent emotional reaction to self-perceived threat (Spielberger, 1966; Staab, 2014). Anxiety encompasses generalized anxiety disorder (GAD), PTSD, social anxiety disorder, specific phobia, and panic disorder. Anxiety can be a prolonged state of worry or negative outlook on uncertain future events (Barlow, 2000), which can be prove advantageous in helping us identify potential threats. However, in excess, anxiety can develop into an anxiety disorder that persists and affects daily living. Studies to understand anxiety neural circuitry have identified several structures that may make up the anxiety circuits. The identified structures include thalamus, amygdala, medial prefrontal cortex, hippocampus, rostral anterior cingulate cortex, dorsolateral prefrontal cortex, dorsal anterior cingulate cortex, and insula (Shankman et al., 2014; Shin & Liberzon, 2010; Stein, Simmons, Feinstein, & Paulus, 2007). The exact neurocircuitry of anxiety still confounds researchers.

Anxiety has been identified as a side effect following injury. This may be due to lack of social support, fear of reinjury, or pain (Cassidy, 2006; Podlog, Dimmock, & Miller, 2011; Weinberg & Gould, 2007). Luis and Mittenberg (2002) compared anxiety in patients with mTBI and orthopaedic injuries and found that those who sustained an mTBI were 4.3 times more likely to be diagnosed with a new-onset anxiety disorder at 6 months following injury. Also, children who sustained an mTBI were found to be 2.24 times more likely to have increased anxiety

symptoms (Liu & Li, 2013). These findings identify anxiety as a common post-concussion symptom.

Vestibular and oculomotor dysfunction are comorbidly linked with anxiety. Anxiety is common in vestibular and ocular motor disorders (Eagger et al., 1992; Ellis et al., 2015; Jacob & Furman, 2001; Matheson et al., 1999; Shefer, Gordon, Avraham, & Mintz, 2010). Vestibular vertigo is associated with a diagnosis of anxiety and panic disorder more than three times greater than the general population (Bigelow, Semenov, du Lac, Hoffman, & Agrawal, 2016). Between 24% and 49% of patients were also able to meet diagnostic criteria for psychiatric disorders when presenting with vertigo and dizziness (Grunfeld, Gresty, Bronstein, & Jahanshahi, 2003; Lahmann et al., 2015). Anxiety is also correlated with diminished visual filtering efficiency and visual working memory performance (Berggren, Curtis, & Derakshan, 2017; Moriya & Sugiura, 2012; Qi, Ding, & Li, 2014; Stout, Shackman, & Larson, 2013). The vestibular-ocular reflex (VOR) varies between anxious and non-anxious patients, with a decreased VOR time constant and increased VOR gain in anxious patients (J. M. Furman, Redfern, & Jacob, 2006; Matta & Enticott, 2004; Naranjo et al., 2016, 2017a; Swinson et al., 1993). However, the comorbid link within the literature between vestibular and oculomotor dysfunction and anxiety following SRC is scarce.

Though anxiety has been categorized as a post-concussion clinical profile (Collins et al., 2014) and post-concussion symptom factor (Kontos et al., 2012), the role of anxiety following SRC has not been studied in depth and has produced inconsistent results. Sing and colleagues (2016) reported increased anxiety at three days and one week following SRC and Meier and colleagues (2015) reported increased anxiety for the first two assessments following SRC; however, Putukian and colleagues (2015) reported no significant interaction between anxiety and

time of assessment following SRC. The few studies that looked at anxiety throughout recovery produced varying results, while few studies reported risk factors. Yang et al. (2015) reported an increased likelihood of state anxiety following SRC; however, it was correlated with pre-injury depression. Another risk factor for anxiety following SRC is concussion history, published by Meehan and colleagues (2016). The few studies regarding anxiety and the anxiety measures used following SRC are inconsistent and lead to unclear results for the research community and clinicians.

## Methods

### Research Design

A prospective, repeated measures design was used to compare state anxiety between concussed male and female adolescent athletes with and without vestibular and ocular motor symptoms and impairment.

### Participants

Participants included adolescent athletes (15-18 years of age) that were seeking care for a SRC at a specialty concussion clinic. Inclusion criteria consisted of the following: 1) enrollment in a patient research registry, 2) completion of an initial clinical visit within 21 days of the injury and 3) completion of a medical clearance clinical visit. Participants were excluded if they were not between the ages of 15 and 18 years. Participants with a history of prior concussion, attention-deficit hyperactivity disorder (ADHD)/learning disabilities (LD), history of treatment for headache or migraine, or pre-existing psychological disorders or neurological disorders were included in the study.

### Measures/Instrumentation

**Definition of Concussion.** All concussions were diagnosed via a clinical evaluation completed by a comprehensive concussion team under the supervision of a physician (i.e., primary care sports medicine or neuropsychologist). For the purposes of this study, the following diagnostic criteria were implemented for the diagnosis of concussion: 1) presence of a clear mechanism of injury; and 2) one or more on-field signs of concussion (e.g., loss of consciousness, post-traumatic amnesia, disorientation, confusion, disrupted balance) and/or at least one concussion symptom (e.g., headache, dizziness, nausea, mental foginess) with onset

immediately or up to 72 hours after the mechanism of injury and/or 3) neurocognitive composite scores outside reliable change indices and/or 4) VOMS symptom scores over clinical cutoffs.

**Demographics and Clinical Concussion Assessment.** Demographic data including age, sex, history of ADHD, LD, anxiety, depression, headache, migraine, seizure, and concussion history, as well as VOMS scores, STAI-State and STAI-Trait scores, computerized neurocognitive test scores, and total symptom scores were obtained from each patient clinical evaluation and is part of the normal standard of care for SRC. Each clinical assessment is described below.

**Clinical Interview.** The clinical interview is a thorough consultation between the trained clinical neuropsychologist and the patient. Specific areas of the interview include injury details (e.g., mechanism of injury, direct or indirect hit, on-field symptoms), current physical symptoms, cognitive symptoms, emotional changes, sleep difficulties, and nutrition and hydration habits, and patient health history (e.g. patient and family history of headache, anxiety, carsickness). The clinical interview is followed by a vestibular and ocular motor assessment, a computerized neurocognitive assessment, and a 22-item symptom report.

**Vestibular and ocular motor assessment.** The VOMS is comprised of eight components including: smooth pursuits, horizontal and vertical saccades, horizontal and vertical VOR, visual motion sensitivity (VMS), near point of convergence (NPC), and NPC distance (cm). Prior to completing any VOMS component, patients verbally rated their baseline (i.e., pretest) symptoms of headache, dizziness, nausea, and foginess on a Likert scale ranging from 0 (none) to 10 (severe). Patients then completed the VOMS and rated their symptoms again following each component. The NPC distance is average distance (cm) across three trials. The VOMS has high internal consistency (Cronbach  $\alpha = .92$ ) and is reported to differentiate between



concussed and healthy individuals (Kontos et al., 2016; Mucha et al., 2014). For youth athletes at baseline, the VOMS is reported to have a high internal consistency (Cronbach  $\alpha = .97$ ) and a low false-positive rate published by Moran et al. (2018). Symptoms were not provoked following the VOMS in healthy adolescents according to Yorke, Smith, Babcock, and Alsalaheen (2017). However, female sex and history of motion sickness has been reported to be a risk factor for VOMS provocation (Kontos, Sufrinko, Elbin, Puskar, & Collins, 2016).

There are several scoring methods for the VOMS. In the majority of the literature, the total symptom score is used for each VOMS component (Anzalone et al., 2016; Kontos et al., 2016; Mucha et al., 2014; Russell-Giller et al., 2017; Sufrinko et al., 2016; Sufrinko et al., 2017); However, VOMS change scores have also been used in the literature (Yorke et al., 2017). The current study used the change scoring method similar to Yorke et al., 2017 to better isolate vestibular/ocular motor provocation and control for pretest VOMS symptoms. To calculate VOMS change scores, total symptom scores were calculated by summing the individual symptom scores for headache, dizziness, nausea, and fogginess, and then subtracted from pretest symptom score. A negative change score (i.e., the total symptom score is less than the pretest VOMS score) was coded as a zero and assumed not to provoke the athlete. Provocation was categorized by any change score  $\geq 2$  and NPC distance  $\geq 5$ cm (Mucha et al., 2014). Participants exhibiting VOMS change scores over these cutoffs were categorized into a PROV group, and participants exhibiting VOMS change scores under these cutoffs were categorized into a NO PROV group based on vestibular/ocular motor presentation at their initial clinical visit.

**Computerized neurocognitive assessment.** The Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) is part of the normal standard of care at the clinical and used to assess neurocognitive function and concussion symptoms. This computerized

neurocognitive test is comprised of three sections: demographic information, the Post-Concussion Symptom Scale (PCSS), and neurocognitive testing. ImPACT produces composite scores for verbal memory, visual memory, processing speed, and reaction time and has acceptable validity and reliability over eight days with correlation coefficients ranging from 0.62 to 0.88 (Iverson, Lovell, & Collins, 2005).

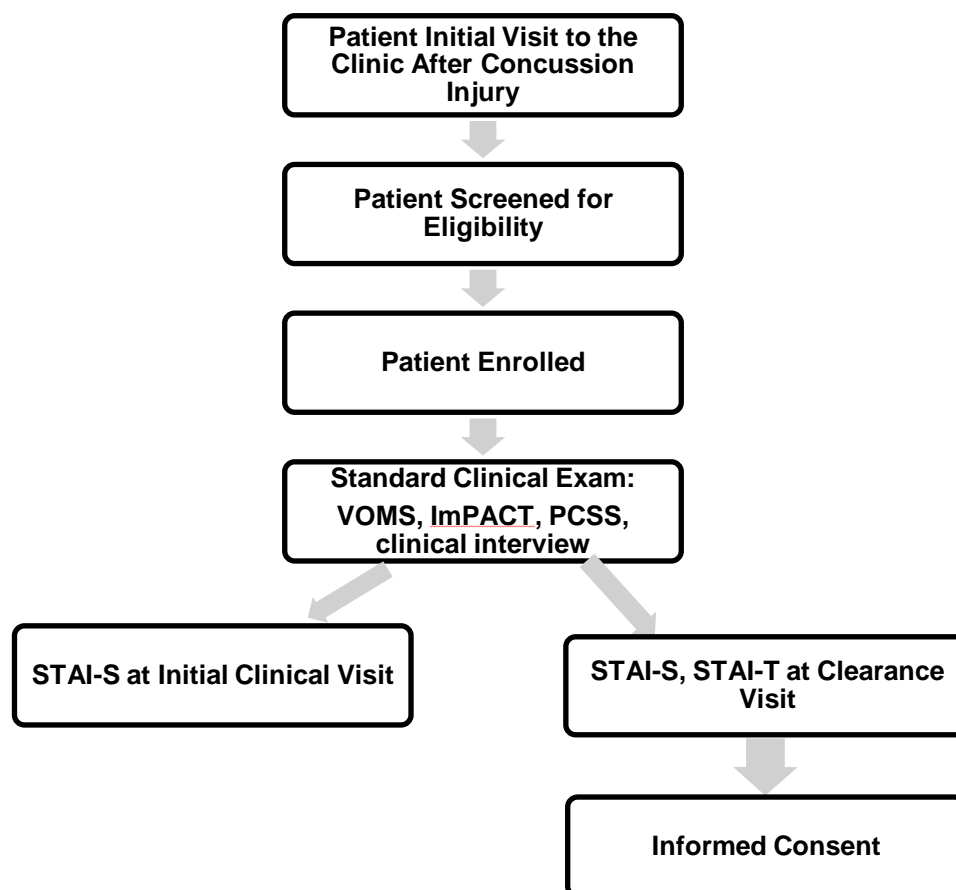
**Concussion symptoms.** ImPACT also includes a symptom inventory called Post-Concussion Symptom Scale (PCSS). The PCSS is a 22-item symptom inventory, with symptoms self-rated on a 7-point Likert scale from 0 (none) to 6 (severe). The PCSS provides a total symptom score as an outcome. Schatz and colleagues (2006) reported ImPACT and PCSS combined sensitivity of 81.9% and specificity of 89.4%.

**State and trait anxiety.** The STAI-Y is a clinical state and trait anxiety measure to diagnose anxiety (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). The STAI-Y is a 40-item assessment with 20 items for state anxiety and 20 items for trait anxiety that includes a two-factor model (e.g., anxiety-present and anxiety-absent items). The patients rated state items on a 4-point scale from 1 (Not at all) to 4 (Very Much So) and trait items on a 4-point scale from 1 (Almost Never) to 4 (Almost Always). Ten items on the state scale and nine items on the trait scale are reverse scored, and items on the state scale are summed to produce a total state anxiety score ranging from 20-80 and items on the trait scale are summed to produce a total trait anxiety score ranging from 20-80. Higher scores indicate higher levels of anxiety for both state and trait anxiety. According to the literature, anxiety levels have been classified into three categories based on state and trait STAI scores, with low scores less than 30, moderate scores between and including 30 and 44, and severe scores greater than or equal to 45 (Spielberger et al., 1983). However, several studies have used STAI scores as a continuous variable and used mean scores

to identify anxiety level (Gunning et al., 2010; Kennedy & Rogers, 2000; Leddy, Lambert, & Ogles, 1994; Schoenhuber & Gentilini, 1988). In the current study anxiety scores were analyzed as continuous variables for both state and trait subscales. The state and trait STAI-Y has shown internal consistency coefficients ranging from .86 to .95 in adolescent and adult samples (Spielberger et al., 1983).

## **Procedures**

The current study was a medical records review of patient data obtained from a clinical patient research registry conducted at the Inova Sports Medicine Concussion Program between September 1, 2017 and April 1, 2018. The patient research registry is currently approved by the Inova Institutional Review Board. The standard clinical visit for SRC at this clinic includes a neurocognitive (ImPACT), symptom assessment (PCSS), vestibular/ocular motor assessment (VOMS), and an anxiety measure (STAI-State and STAI-Trait), followed by an in-person clinical interview assessing patient health history (e.g., personal and family history of headache, anxiety, carsickness), current physical symptoms, cognitive symptoms, emotional changes, sleep difficulties, and nutrition and hydration habits, and injury-related information (e.g., mechanism of injury, location of impact, on-field symptoms). The clinical interview was conducted by neuropsychologist trained in the management of SRC. Data were obtained from patients' first clinical visit that occurred within 30 days following injury and also at each medical clearance visit. In an effort to control for possible confounding effect of SRC on the patient trait anxiety scores, the T-Anxiety scale of the STAI was only administered at the medical clearance visit. The order of procedures is presented in Figure 1.



*Figure 1.* Order of procedures for current study.

### Data Analysis

**Inspection of data for accuracy and completeness.** Two researchers inspected fifteen percent of the total number of cases, chosen at random, to verify accuracy and completeness.

**Examination of normality.** Given the parametric tests that were utilized in testing the study hypotheses, each variable were examined for normality using frequencies, skewness, and histograms. A power analysis was completed to determine the minimum sample size for the study.

**A priori power analysis.** *A priori* power analyses was conducted for each of the statistical analyses used for the three hypotheses to determine sample sizes required to achieve adequate statistical power. The effect sizes for interactions were used to calculate sample size using G-Power (Faul, Erdfelder, Buchner, & Lang, 2009) statistical software. Specifically, the low, median, and high effect sizes were used to calculate subsequent sample size using  $p = .05$ . The results of this post-hoc power analysis can be found in Table 1.

Table 1.

*Calculated Sample Sizes from A priori Power Analyses for Hypotheses 1-3.*

Analysis	Effect Size	N
Hypothesis 1-3	.10	1302
	.25	210
	.40	84

**Data analysis for H1 - State anxiety scores will be higher for concussed adolescent athletes with vestibular and ocular motor impairment and symptoms compared to concussed adolescent athletes without vestibular and ocular motor impairment and symptoms.** Hypothesis 1 was evaluated by the results of the between-subjects main effect of a repeated measures analysis of variance (ANOVA). The independent variables were vestibular/ocular motor impairment (yes, no) and time (initial clinical visit, medical clearance clinical visit) and the dependent variable was the total state anxiety score.

**Data analysis for H2 - State anxiety scores will be higher for concussed adolescent athletes at initial clinical visit compared to medical clearance clinical visit.** Hypothesis 2 was evaluated by the results of the within-subjects main effect of a repeated measures ANOVA. The

independent variable was time (initial clinical visit, medical clearance clinical visit) and the dependent variable was the total state anxiety score.

**Data analysis for H3 - There will be a significant interaction between vestibular and ocular motor impairment and symptoms and clinical visit time points on state anxiety scores in concussed adolescent athletes. Athletes with vestibular and ocular motor impairment and symptoms at initial clinical visit will have higher state anxiety scores.**

Hypothesis 3 was evaluated by the interaction between main effects of the repeated measures ANOVA. The independent variables were vestibular/ocular motor impairment (yes, no) and time (initial clinical visit, medical clearance clinical visit). The dependent variable was the total state anxiety score.

**Data Analysis for EQ1 – Is there an interaction between sex and vestibular and ocular motor impairment and symptoms on state anxiety scores in adolescent athletes with concussion?** Exploratory question 1 was evaluated by the interaction between sex and vestibular provocation from the repeated measures ANOVA. The independent variables were vestibular/ocular motor impairment (yes, no) and sex (male, female). The dependent variable was the total state anxiety score.

## Results

### Demographic Results

A sample of 123 adolescent athletes agreed to participate in the clinical research registry; however, only 30 (24%) were included in the current study based on exclusion criteria (See Figure 2). The final sample was comprised of 30 adolescent athletes seeking care for SRC at a specialty concussion clinic who were between the ages of 15-18 years, were seen within 30 days of the injury, and completed the STAI-State at the initial and medical clearance clinical visit. There were 15 males ( $M=16.13$ ,  $SD=0.99$  years) and 15 females ( $M=16.20$ ,  $SD=0.94$  years). The average number of days from injury to the initial clinical visit was approximately 10 days ( $SD=7.22$ , Range=0-24 days) and SRC recovery for the entire sample was approximately 30 days ( $SD=17.20$ , Range=11-75 days).

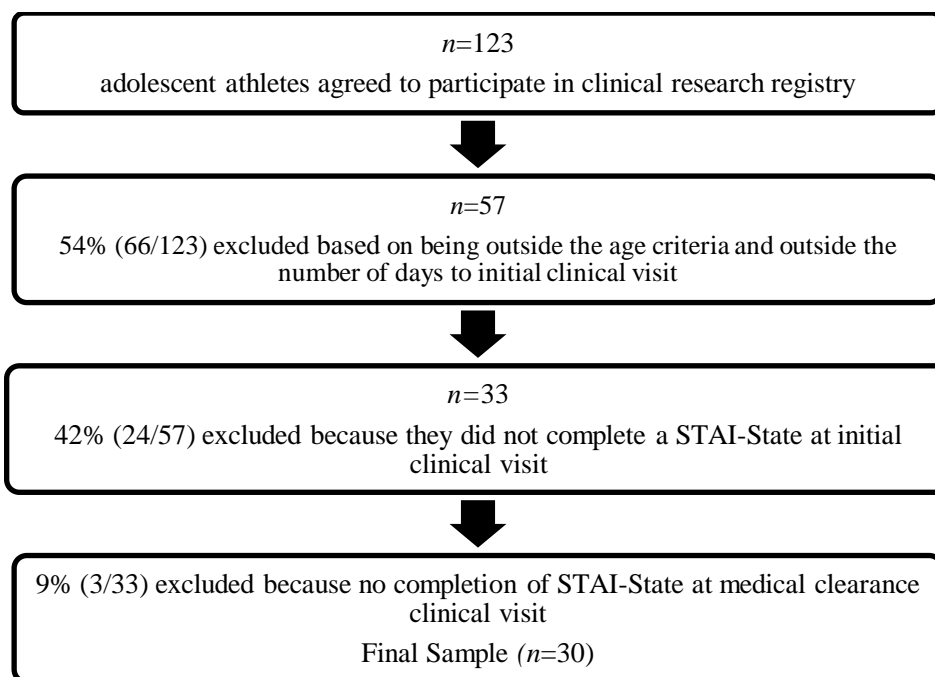


Figure 2. Sample determination based on exclusionary criteria.

At the initial clinical visit there were 37% (11/30) of participants that demonstrated vestibular provocation and 63% (19/30) that were not provoked on the VOMS. In addition, there

were 10% (3/30) of participants that exhibited abnormal NPC distance (e.g.,  $\geq 5\text{cm}$ ) and 90% (27/30) of participants that exhibited normal NPC distance. In addition, 17% (5/30) of participants received vestibular therapy and 83% (25/30) of participants did not receive vestibular therapy. Based on these clinical assessments there were 11 participants in the PROV group and 19 participants in the NO PROV group.

The PROV and NO PROV groups were compared on demographic variables to ensure group equivalency. The groups did not differ on age ( $t(28)=-.73, p=.47$ ), sex ( $\chi^2[1,1]=1.29, p=.26$ ), ADHD ( $\chi^2[1,1]=2.92, p=.09$ ), LD ( $\chi^2[1,1]=1.79, p=.18$ ), depression ( $\chi^2[1,1]=0.35, p=.55$ ), seizure ( $\chi^2[1,1]=0.60, p=.44$ ), migraine ( $\chi^2[1,1]=0.15, p=.70$ ), headache ( $\chi^2[1,1]=0.34, p=.56$ ), or carsickness ( $\chi^2[1,1]=0.07, p=.79$ ). However, there were differences between the groups for concussion history ( $\chi^2[1,1]=5.48, p=.02$ ) and self-reported anxiety ( $\chi^2[1,1]=4.75, p=.03$ ). Specifically, there were more participants that endorsed concussion history in the NO PROV group (17/29) compared to the PROV group (7/29) and more participants that endorsed self-reported anxiety in the NO PROV group (16/29) compared to the PROV group (6/29). In addition, the groups did not differ from days from injury to initial clinical visit ( $t(28)=-.54, p=.59$ ) or SRC recovery time (i.e., days from injury until medical clearance) ( $t(28)=0.65, p=.52$ ). The groups did not differ on trait anxiety (over clinical cutoff (6/26), under clinical cutoff (20/26)) ( $\chi^2[1,1]=0.73, p=.39$ ). The means, standard deviations, frequencies, and percentages for demographic variables are presented in Table 2.



Table 2.

*Means, standard deviations, frequencies, and percentages for demographic variables and clinical measures of concussion among participants in the PROV (n=11) and NO PROV (n=19) groups at initial clinical visit.*

	PROV		NO PROV		<i>p</i>
	<i>n</i>	(%)	<i>n</i>	(%)	
<b>Demographic Variables</b>					
ADHD	8	73	18	95	.09
LD	10	91	19	100	.18
Depression	9	82	17	89	.55
Seizure	11	100	18	95	.44
Migraine	8	73	15	79	.70
Headache	7	64	10	53	.56
Carsickness	7	64	13	68	.79
Concussion History *	8	73	18	95	.02
Anxiety History*	6	55	17	89	.03
Sex (% Female)	7	64	8	42	.26
	<b>M</b>	<b>SD</b>	<b>M</b>	<b>SD</b>	<b><i>p</i></b>
Age	16.00	1.00	16.26	.93	.47
Recovery Time (days)	32.27	21.09	28.00	14.91	.52
Days from Injury to Clinical Visit	8.55	7.37	10.05	7.28	.59

\* $p \leq .05$

The PROV and NO PROV groups were also compared on clinical measures of concussion (VOMS, ImPACT, and symptoms) to determine possible injury severity differences between the groups at the initial visit. As expected, there were significant differences between

PROV and NO PROV groups on VOMS components that included the pre-test component ( $t(28)=2.65, p=.01$ ) smooth pursuit ( $t(28)=3.01, p=.01$ ), horizontal saccades ( $t(28)=3.60, p=.001$ ), vertical saccades ( $t(28)=3.64, p=.001$ ), horizontal VOR ( $t(28)=4.15, p=.000$ ), vertical VOR ( $t(28)=3.89, p=.001$ ), visual motion sensitivity ( $t(28)=4.48, p=.000$ ), and NPC symptoms ( $t(28)=3.30, p=.003$ ). The PROV group reported higher VOMS component symptom scores when compared to NO PROV group at initial clinical visit. (See Table 3). There were no significant differences between PROV and NO PROV groups for the neurocognitive composite scores of verbal memory ( $t(28)=-0.79, p=.44$ ), visual memory ( $t(28)=-1.84, p=.08$ ), visual motor speed ( $t(28)=-1.55, p=.13$ ), or reaction time ( $t(28)=1.77, p=.09$ ). In addition, there were no significant differences in total symptom score on the PCSS between groups ( $t(28)=1.88, p=.07$ ). Means and standard deviations for neurocognitive scores and total PCSS scores are presented in Table 4. At the medical clearance clinical visit, there were no significant differences due to lack of vestibular provocation on the VOMS.

Table 3.

*Means and standard deviations for VOMS change scores between PROV (n=11) and NO PROV (n=19) groups and total sample (N=30) at initial clinical visit.*

VOMS Components	PROV		NO PROV		TOTAL	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Pretest Symptoms	8.91*	5.30	4.47	3.85	6.10	4.86
Smooth Pursuits	9.45*	5.16	4.47	3.85	6.30	4.94
Horizontal Saccades	10.27*	4.75	4.58	3.82	6.67	4.96
Vertical Saccades	10.73*	5.22	4.68	3.84	6.90	5.23
Near Point Convergence (Sx)	10.55*	6.07	4.47	4.03	6.70	5.63
Horizontal VOR	11.645*	5.14	4.58	4.07	7.17	5.60
Vertical VOR	11.731*	6.03	4.47	4.17	7.13	6.00
VMS	12.731*	5.46	4.84	4.13	7.73	5.98

\* $p \leq .05$

Table 4.

*Means and standard deviations for computerized neurocognitive composite scores for PROV (n=11) and NO PROV (n=19) groups at initial clinical visit.*

Concussion Clinical Outcomes	PROV		NO PROV		<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Verbal Memory	82.64	10.46	86.00	11.66	.44
Visual Memory	67.82	16.20	77.53	12.46	.08
Visual Motor Speed	35.84	10.21	40.84	7.41	.13
Reaction Time	0.65	0.12	0.58	0.08	.59
Total PCSS Symptoms	33.91	17.98	22.26	15.44	.07

\* $p \leq .05$

## Evaluation of Hypotheses

**Hypothesis 1. State anxiety scores will be higher for concussed adolescent athletes with vestibular and ocular motor impairment and symptoms compared to concussed adolescent athletes without vestibular and ocular motor impairment and symptoms.** The results of a 2 Group (PROV, NO PROV) X 2 Time (initial clinical visit, medical clearance clinical visit) repeated-measures ANOVA supported a significant between-subjects effect for provocation groups ( $F_{1,1} = 8.38, p = .007, \eta^2 = .23$ ) on state anxiety. State anxiety scores were higher between PROV ( $M=43.73, SD=14.64$ ) and NO PROV ( $M=31.68, SD=9.15$ ) groups. These findings support Hypothesis 1, which hypothesized that state anxiety scores would be higher for concussed adolescent athletes with vestibular and ocular motor provocation compared to concussed adolescent athletes without vestibular and ocular motor provocation.

**Hypothesis 2. State anxiety scores will be higher for concussed adolescent athletes at initial clinical visit compared to medical clearance clinical visit.** There was a significant within-subjects main effect for time ( $F_{1,1} = 6.95, p = .01, \eta^2 = .20$ ) for state anxiety. These findings indicate state anxiety at initial clinical visit ( $M=36.10, SD=12.67$ ) was higher when compared to state anxiety at medical clearance clinical visit ( $M=29.73, SD=9.84$ ). These findings support Hypothesis 2, which hypothesized that state anxiety scores will be higher for concussed adolescent athletes at initial clinical visit compared to medical clearance clinical visit.

**Hypothesis 3. There will be a significant interaction between vestibular and ocular motor impairment and symptoms and clinical visit time points on state anxiety scores in concussed adolescent athletes. Athletes with vestibular and ocular motor impairment and symptoms at initial clinical visit will have higher state anxiety scores.** The interaction between provocation groups and time was not significant (Wilks'  $\lambda = .94, p = .20, \eta^2 = .06$ ).

State anxiety did not differ between PROV and NO PROV groups at either time point (see table 4). These findings do not support Hypothesis 3, which hypothesized that there will be a significant interaction between vestibular and ocular motor impairment and symptoms and clinical visit time points on state anxiety scores in concussed adolescent athletes. Athletes with vestibular and ocular motor impairment and symptoms at initial clinical visit did not have higher state anxiety scores. The means and standard deviations for state anxiety scores across clinical time points and for each provocation group is presented in Table 5.

Table 5.

*Means and standard deviations for state anxiety scores at initial and medical clearance clinical visits for athletes in the PROV (n=11) and NO PROV (n=19) groups.*

	Initial Clinical Visit				Medical Clearance Clinical Visit			
	PROV		NO PROV		PROV		NO PROV	
	M	SD	M	SD	M	SD	M	SD
State Anxiety Score	43.73	14.64	31.68	9.15	32.73	11.77	28.00	8.39

**Analysis of Exploratory Question 1. Is there an interaction between sex and vestibular and ocular motor impairment and symptoms on state anxiety scores in adolescent athletes with concussion?** There were no significant differences in state anxiety ( $t(28)=-.04, p=.95$ ) between males ( $M=36.00, SD=14.41$ ) and females ( $M=36.20, SD=11.18$ ). There was no significant vestibular and ocular motor impairment by sex interaction (Wilks'  $\lambda = .90, p = .10, \eta^2 = .10$ ). Means and standard deviations for state anxiety scores for males and females with and without vestibular and ocular motor impairment are shown in Table 6.

Table 6.

*Means and standard deviations for state anxiety scores at initial visit for males in the PROV group (n=4), females in the PROV group (n=7), males in the NO PROV group (n=11), females in the NO PROV group (n=8) and at medical clearance visit for males in the PROV group (n=4), females in the PROV group (n=7), males in the NO PROV group (n=11), females in the NO PROV group (n=8).*

	Initial Clinical Visit								Medical Clearance Clinical Visit							
	PROV				NO PROV				PROV				NO PROV			
	Males		Females		Males		Females		Males		Females		Males		Females	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
State Anxiety Score	52.25	11.18	38.86	14.78	30.09	10.45	33.88	7.04	34.00	15.75	32.00	10.26	29.82	9.54	25.50	6.21

## Discussion

### General Discussion of Results

This study examined the relationship between state anxiety and vestibular and ocular motor impairments and symptoms in concussed adolescent athletes with concussion. There were expected significant differences in VOMS component symptom scores between PROV and NO PROV groups, which allowed the current study to investigate differences between groups. The PROV and NO PROV groups were not significantly different on demographic variables and neurocognitive composite scores, which controlled for confounding variables. The primary findings from this study were that concussed adolescent athletes with vestibular and ocular motor impairments and symptoms had higher state anxiety scores and state anxiety scores were higher at initial clinical visit than at medical clearance visit. However, this study does not suggest a significant interaction between vestibular and ocular motor impairment and symptoms and clinical time point on state anxiety scores. It is unknown whether the lack of vestibular provocation at the medical clearance visit caused the lack of interaction or if there are other mediators between anxiety and vestibular and ocular motor impairment and symptoms. A larger provocation sample at medical clearance clinical visit would be necessary to test this hypothesis.

### Discussion of Hypotheses

**Discussion of Hypotheses 1 and 2.** Due to the main findings in this study, Hypotheses 1 and 2 were supported. Athletes with vestibular and ocular motor impairments had higher state anxiety scores. State anxiety scores were higher at the initial clinical visit. Non-concussed individuals with anxiety report symptoms similar to vestibular and ocular motor dysfunction, including vertigo, dizziness, poor gaze stability, and quicker fixation (Bigelow et al., 2016; Hauk et al., 2008; Laretzaki et al., 2011; Monzani et al., 2001; Quigley et al., 2012). This symptom

overlap highlights a hypothesized relationship between these two disorders in the general population and could potentially suggest a comorbid relationship following SRC. Also, previous research has reported increased anxiety and vestibular and ocular motor impairments acutely following injury (Broglia et al., 2009; Kontos et al., Master et al., 2016; Meier et al., 2015; Sing et al., 2016; Yang et al., 2015). This study's findings are in agreement with the previous literature such that anxiety is a prominent psychological consequence following injury and vestibular and ocular motor impairment and is prevalent during the acute phase of SRC (Bigelow et al., 2016; Leddy, Lambert, & Ogles, 1994; Monzani et al., 2001). However, this relationship is only hypothesized due to clinical observations and anecdotes in concussed samples (Collins et al., 2014).

**Discussion of Hypothesis 3.** Hypothesis 3 was not supported. Though it was hypothesized that state anxiety scores would be increased at initial clinical visit for athletes with vestibular and ocular motor impairment and symptoms, the findings did not support this. Vestibular and ocular motor impairment at initial clinical visit did not predict state anxiety scores over time, and, therefore, may not suggest a relationship between these variables. Possible reasons for this lack of interaction include the differences between groups on concussion history and the lack of vestibular and ocular motor impairment at medical clearance clinical visit. The relationship between state anxiety and vestibular and ocular motor impairment has not been studied following SRC, and a larger sample size with greater vestibular and ocular motor impairment would be necessary to evaluate this further.

**Exploratory Question 1.** The exploratory question was not supported through the findings of this current study. This is in disagreement with literature regarding the sex differences seen in anxiety, vestibular and ocular motor deficits, and SRC outcomes. There could



be several possible explanations for the lack of differences between sex, anxiety, and vestibular and ocular motor impairments and symptoms. Previous research suggests females endorse more symptoms, including anxiety and vestibular and ocular motor impairments and symptoms, in the general population (Covassin et al., 2013; Gulliver et al., 2015; Monzani et al., 2001); however, there is limited research regarding the connection between the anxiety and vestibular and ocular motor impairments and symptoms following SRC. The athletes participating in this study were recruited from a specialty concussion clinic, which may present as a more anxious population based on a potential assumption that those seeking specialty care have higher anxiety. Though differences in state anxiety based on sex were not supported by this study, future research is needed on this subject to further understand these differences in this population.

### **Implications**

State anxiety was increased at the initial clinical visit and was higher in adolescent athletes with vestibular and ocular motor impairment following SRC. This emphasizes the assessment of both anxiety and vestibular and ocular motor impairments following SRC during the acute phase. The acute assessment of both of these symptoms and impairments, as recommended, could inform the correct identification into clinical profiles, which aims to provide the most effective treatment options, and ultimately quicker recovery time. With the comorbid relationship of state anxiety with vestibular and ocular motor impairment, failing to assess one could lead to not identifying the other. This overlap may highlight the potential for anxiety treatment opportunities to help alleviate symptoms and impairments for individuals in the vestibular and ocular motor profiles, but a greater understanding of the interaction between these would be necessary to uncover this possible treatment approach. In addition, this study provides preliminary evidence that these profiles may not be directly related. Vestibular and

ocular motor impairment did not predict state anxiety scores over time even though both between- and within-subjects effects were significant, which may be due to other potential mediators or lack of vestibular and ocular motor impairment at the medical clearance clinical visit. This is another reason for using the recommended multimodal approach to concussion assessment and management. There may not be an interaction between vestibular and ocular motor impairment and clinical visit time point on state anxiety, but there is considerable overlap between these symptoms and impairments that warrants the acute and multimodal assessment and management approach.

### **Limitations**

There were several limitations included within this study. There are normative values for state anxiety in high school adolescents; however, there are no normative data for concussed high school adolescents. The lack of norms for this population may have caused increased type II error in this sample. The constraints of the inclusion and exclusion criteria made the sample size too small; however, this was addressed by limiting the sample to the age specification and completion of the anxiety measure and testing the sample for significant differences for the remaining criteria. The sample size did not meet the specification recommended by the *a priori* power analysis, which decreases the significance of the results. The participants of this study were recruited from a specialty concussion clinic, which limits the generalizability of the results. Both anxiety and vestibular impairment were self-reported measures. It is assumed all athletes reported honestly and accurately on these measures.

### **Future Research**

Future research should continue to examine the clinical concussion profiles and the overlap between them, including the symptoms and impairments and the neurocircuitry and

neuropathology, especially the association between anxiety and vestibular and ocular motor impairments. A larger sample size is necessary to provide substantial evidence for this hypothesized association, including the sex differences that were not supported in this study. Also, a larger sample size and other recruitment strategies in several other locations, other than specialty clinics, may also provide a larger range of anxiety levels and vestibular and ocular motor impairments.

It would also be beneficial to track state anxiety throughout recovery in order to investigate how state anxiety changes over time and how it is affected by recovery time. Anxiety is a symptom of post-concussion syndrome, so it would be interesting to examine the relationship between state anxiety in prolonged recovery times. The psychological deficits of concussion reach beyond anxiety, including depression, stress, and potentially increased trait anxiety. The investigation into the association between these factors could prove beneficial for psychological outcomes following SRC.

## **Conclusions**

The results of this study supported the hypotheses that state anxiety scores were higher in concussed adolescent athletes with vestibular and ocular motor impairment following SRC and at the initial clinical visit, but no interaction was found. In addition, there were no sex differences observed in this sample, failing to support exploratory questions. The results of this current study suggest clinicians should examine all aspects of concussion, including anxiety and vestibular and ocular motor impairments and symptoms, in order to make clinical treatment decisions that could provide more effective treatment options, quicker recovery times, and better SRC outcomes.

## References

- Adams, J. H., Graham, D. I., Murray, L. S., & Scott, G. (1982). Diffuse axonal injury due to nonmissile head injury in humans: an analysis of 45 cases. *Annals Of Neurology*, *12*(6), 557–563. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=7159059&site=ehost-live&scope=site>
- Alsalaheen, B. A., Mucha, A., Morris, L. O., Whitney, S. L., Furman, J. M., Camiolo-Reddy, C. E., ... Sparto, P. J. (2010). Vestibular rehabilitation for dizziness and balance disorders after concussion. *Journal Of Neurologic Physical Therapy: JNPT*, *34*(2), 87–93. <https://doi.org/10.1097/NPT.0b013e3181dde568>
- Anzalone, A. J., Blueitt, D., Case, T., McGuffin, T., Pollard, K., Garrison, J. C., ... Oliver, J. M. (2017). A Positive Vestibular/Ocular Motor Screening (VOMS) Is Associated With Increased Recovery Time After Sports-Related Concussion in Youth and Adolescent Athletes. *The American Journal of Sports Medicine*, *45*(2), 474–479. <https://doi.org/10.1177/0363546516668624>
- Armstrong, B., McNair, P., & Taylor, D. (2008). Head and neck position sense. *Sports Medicine*, *38*(2), 101–117.
- Aubry, M., Cantu, R., Dvorak, J., Graf-Baumann, T., Johnston, K., Kelly, J., ... Schamasch, P. (2002). Summary and agreement statement of the first International Conference on Concussion in Sport, Vienna 2001. *British Journal of Sports Medicine*, *36*(1), 6–7.
- Babcock, L., Byczkowski, T., Wade, S. L., Ho, M., Mookerjee, S., & Bazarian, J. J. (2013). Predicting postconcussion syndrome after mild traumatic brain injury in children and adolescents who present to the emergency department. *JAMA Pediatrics*, *167*(2), 156–161.
- Bailes, J. E., & Cantu, R. C. (2001). Head injury in athletes. *Neurosurgery*, *48*(1), 26–46.
- Bakhos, L. L., Lockhart, G. R., Myers, R., & Linakis, J. G. (2010). Emergency department visits for concussion in young child athletes. *Pediatrics*, *126*(3), e550–e556. <https://doi.org/10.1542/peds.2009-3101>
- Balaban, C. D., & Thayer, J. F. (2001). Neurological bases for balance–anxiety links. *Journal of Anxiety Disorders*, *15*(1–2), 53–79.
- Bandak, F., Ling, G., Bandak, A., & De Lanerolle, N. (2015). Injury biomechanics, neuropathology, and simplified physics of explosive blast and impact mild traumatic brain injury. *Handbook of Clinical Neurology*, *127*, 89–104. <https://doi.org/10.1016/b978-0-444-52892-6.00006-4>
- Barkhoudarian, G., Hovda, D. A., & Giza, C. C. (2011). The molecular pathophysiology of concussive brain injury. *Clinics In Sports Medicine*, *30*(1), 33. <https://doi.org/10.1016/j.csm.2010.09.001>

- Barlow, D. H. (2000). Unraveling the mysteries of anxiety and its disorders from the perspective of emotion theory. *American Psychologist*, 55(11), 1247.
- Barlow, K. M., Crawford, S., Stevenson, A., Sandhu, S. S., Belanger, F., & Dewey, D. (2010). Epidemiology of postconcussion syndrome in pediatric mild traumatic brain injury. *Pediatrics*, 126(2), e374–e381.
- Barnes, G. R. (2008). Cognitive Processes Involved in Smooth Pursuit Eye Movements. *Brain and Cognition*, 68(3), 309–326. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=eric&AN=EJ819428&site=ehost-live&scope=site>
- Barrett, K. E., Barman, S. M., Boitano, S., & Brooks, H. (2009). Ganong's review of medical physiology. 23. NY: McGraw-Hill Medical.
- Berggren, N., Curtis, H. M., & Derakshan, N. (2017a). Interactions of emotion and anxiety on visual working memory performance. *Psychonomic Bulletin & Review*, 24(4), 1274–1281. <https://doi.org/10.3758/s13423-016-1213-4>
- Berggren, N., Curtis, H. M., & Derakshan, N. (2017b). Interactions of emotion and anxiety on visual working memory performance. *Psychonomic Bulletin & Review*, 24(4), 1274–1281. <https://doi.org/10.3758/s13423-016-1213-4>
- Berz, K., Divine, J., Foss, K. B., Heyl, R., Ford, K. R., & Myer, G. D. (2013). Sex-specific differences in the severity of symptoms and recovery rate following sports-related concussion in young athletes. *The Physician and Sportsmedicine*, 41(2), 58–63.
- Bigelow, R. T., Semenov, Y. R., du Lac, S., Hoffman, H. J., & Agrawal, Y. (2016). Vestibular vertigo and comorbid cognitive and psychiatric impairment: the 2008 National Health Interview Survey. *Journal of Neurology, Neurosurgery & Psychiatry*, 87(4), 367–372. <https://doi.org/10.1136/jnnp-2015-310319>
- Brahm, K. D., Wilgenburg, H. M., Kirby, J., Ingalla, S., Chang, C.-Y., & Goodrich, G. L. (2009). Visual impairment and dysfunction in combat-injured servicemembers with traumatic brain injury. *Optometry And Vision Science: Official Publication Of The American Academy Of Optometry*, 86(7), 817–825. <https://doi.org/10.1097/OPX.0b013e3181adff2d>
- Broglio, S. P., Cantu, R. C., Gioia, G. A., Guskiewicz, K. M., Kutcher, J., Palm, M., & Valovich McLeod, T. C. (2014). National Athletic Trainers' Association position statement: management of sport concussion. *Journal Of Athletic Training*, 49(2), 245–265. <https://doi.org/10.4085/1062-6050-49.1.07>
- Broglio, S. P., & Puetz, T. W. (2008). The effect of sport concussion on neurocognitive function, self-report symptoms and postural control: a meta-analysis. *Sports Medicine (Auckland, N.Z.)*, 38(1), 53–67. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=18081367&site=ehost-live&scope=site>

- Broglia, S. P., Schnebel, B., Sosnoff, J. J., Shin, S., Feng, X., He, X., & Zimmerman, J. (2010). The biomechanical properties of concussions in high school football. *Medicine and Science in Sports and Exercise*, 42(11), 2064.
- Broshek, D. K., De Marco, A. P., & Freeman, J. R. (2015). A review of post-concussion syndrome and psychological factors associated with concussion. *Brain Injury*, 29(2), 228–237. <https://doi.org/10.3109/02699052.2014.974674>
- Bryan, M. A., Rowhani-Rahbar, A., Comstock, R. D., & Rivara, F. (2016). Sports-and recreation-related concussions in US youth. *Pediatrics*, 138(1), e20154635.
- Buckley, T. A., Munkasy, B. A., & Clouse, B. P. (2016). Acute Cognitive and Physical Rest May Not Improve Concussion Recovery Time. *The Journal Of Head Trauma Rehabilitation*, 31(4), 233–241. <https://doi.org/10.1097/HTR.0000000000000165>
- Cannon, C R, & Jahrsdoerfer, R. A. (1983). Temporal bone fractures. Review of 90 cases. *Archives Of Otolaryngology (Chicago, Ill.: 1960)*, 109(5), 285–288. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=6847478&site=ehost-live&scope=site>
- Cannon, C Ron, & Jahrsdoerfer, R. A. (1983). Temporal bone fractures: review of 90 cases. *Archives of Otolaryngology*, 109(5), 285–288.
- Capó-Aponte, J. E., Urosevich, T. G., Temme, L. A., Tarbett, A. K., & Sanghera, N. K. (2012). Visual dysfunctions and symptoms during the subacute stage of blast-induced mild traumatic brain injury. *Military Medicine*, 177(7), 804–813. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=22808887&site=ehost-live&scope=site>
- Carl, J. R. (1997). Practical anatomy and physiology of the ocular motor system. *Handbook of Balance Function Testing*, 57.
- Centers for Disease Control and Prevention. (2011). Nonfatal traumatic brain injuries related to sports and recreation activities among persons aged ≤ 19 years---United States, 2001--2009. *MMWR: Morbidity and Mortality Weekly Report*, 60(39), 1337–1342.
- Chew, B. G., Spearman, C. M., Quigley, M. R., & Wilberger, J. E. (2012). The prognostic significance of traumatic brainstem injury detected on T2-weighted MRI. *Journal Of Neurosurgery*, 117(4), 722–728. <https://doi.org/10.3171/2012.6.JNS111736>
- Clark, M., & Guskiewicz, K. (2016). Sport-Related Traumatic Brain Injury. *Translational Research in Traumatic Brain Injury*. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=cmedm&AN=26583180&site=ehost-live&scope=site>
- Cohen, M., Groswasser, Z., Barchadski, R., & Appel, A. (1989). Convergence insufficiency in brain-injured patients. *Brain Injury*, 3(2), 187–191. Retrieved from <http://0->

search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=cmedm&AN=2471568&site=ehost-live&scope=site

- Collins, M., Lovell, M. R., Iverson, G. L., Ide, T., & Maroon, J. (2006). Examining concussion rates and return to play in high school football players wearing newer helmet technology: a three-year prospective cohort study. *Neurosurgery*, *58*(2), 275–286. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=16462481&site=ehost-live&scope=site>
- Collins, M. W., Kontos, A. P., Okonkwo, D. O., Almquist, J., Bailes, J., Barisa, M., ... Zafonte, R. (2016). Statements of Agreement From the Targeted Evaluation and Active Management (TEAM) Approaches to Treating Concussion Meeting Held in Pittsburgh, October 15-16, 2015. *Neurosurgery*, *79*(6), 912–929. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=27741219&site=ehost-live&scope=site>
- Collins, M. W., Kontos, A. P., Reynolds, E., Murawski, C. D., & Fu, F. H. (2014). A comprehensive, targeted approach to the clinical care of athletes following sport-related concussion. *Knee Surgery, Sports Traumatology, Arthroscopy: Official Journal Of The ESSKA*, *22*(2), 235–246. <https://doi.org/10.1007/s00167-013-2791-6>
- Colvin AC, Mullen J, Lovell MR, West RV, Collins MW, & Groh M. (2009). The role of concussion history and gender in recovery from soccer-related concussion. *American Journal of Sports Medicine*, *37*(9), 1699–1704. <https://doi.org/10.1177/0363546509332497>
- Corwin, D. J., Wiebe, D. J., Zonfrillo, M. R., Grady, M. F., Robinson, R. L., Goodman, A. M., & Master, C. L. (2015). Vestibular deficits following youth concussion. *The Journal of Pediatrics*, *166*(5), 1221–1225.
- Covassin, T., Stearne, D., & Elbin III, R. (2008). Concussion history and postconcussion neurocognitive performance and symptoms in collegiate athletes. *Journal of Athletic Training*, *43*(2), 119–124.
- Cullen, K., & Sadeghi, S. (2008). Vestibular system. *Scholarpedia*, *3*(1), 3013.
- DiFazio, M., Silverberg, N. D., Kirkwood, M. W., Bernier, R., & Iverson, G. L. (2016). Prolonged Activity Restriction After Concussion: Are We Worsening Outcomes? *Clinical Pediatrics*, *55*(5), 443–451. <https://doi.org/10.1177/0009922815589914>
- Dompier, T. P., Kerr, Z. Y., Marshall, S. W., Hainline, B., Snook, E. M., Hayden, R., & Simon, J. E. (2015). Incidence of concussion during practice and games in youth, high school, and collegiate American football players. *JAMA Pediatrics*, *169*(7), 659–665.
- Drew, A. S., Langan, J., Halterman, C., Osternig, L. R., Chou, L.-S., & van Donkelaar, P. (2007). Attentional disengagement dysfunction following mTBI assessed with the gap saccade task. *Neuroscience Letters*, *417*(1), 61–65. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=cmedm&AN=2471568&site=ehost-live&scope=site>

search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=cmedm&AN=17363165&site=ehost-live&scope=site

- Eagger, S., Luxon, L., Davies, R., Coelho, A., & Ron, M. (1992). Psychiatric morbidity in patients with peripheral vestibular disorder: a clinical and neuro-otological study. *Journal of Neurology, Neurosurgery & Psychiatry*, *55*(5), 383–387.
- Echemendia, R. J., Broglio, S. P., Davis, G. A., Guskiewicz, K. M., Hayden, K. A., Leddy, J. J., ... Schneider, K. J. (2017). What tests and measures should be added to the SCAT3 and related tests to improve their reliability, sensitivity and/or specificity in sideline concussion diagnosis? A systematic review. *Br J Sports Med*, *51*(11), 895–901.
- Echemendia, R. J., Meeuwisse, W., McCrory, P., Davis, G. A., Putukian, M., Leddy, J., ... Herring, S. (2017). The Sport Concussion Assessment Tool 5th Edition (SCAT5): Background and rationale. *British Journal Of Sports Medicine*, *51*(11), 848–850. <https://doi.org/10.1136/bjsports-2017-097506>
- Eisenberg, M. A., Meehan, W. P., 3rd, & Mannix, R. (2014). Duration and course of post-concussive symptoms. *Pediatrics*, *133*(6), 999–1006. <https://doi.org/10.1542/peds.2014-0158>
- Elbin, R., Covassin, T., Gallion, C., & Kontos, A. P. (2015). Factors influencing risk and recovery from sport-related concussion: reviewing the evidence. *Perspect Neurophysiol Neurogenic Speech Lang Disord*, *25*, 4–16.
- Elleberg, D., Henry, L. C., Macciocchi, S. N., Guskiewicz, K. M., & Broglio, S. P. (2009). Advances in sport concussion assessment: from behavioral to brain imaging measures. *Journal of Neurotrauma*, *26*(12), 2365–2382.
- Ellis, M. J., Leddy, J. J., & Willer, B. (2015). Physiological, vestibulo-ocular and cervicogenic post-concussion disorders: an evidence-based classification system with directions for treatment. *Brain Injury*, *29*(2), 238–248. <https://doi.org/10.3109/02699052.2014.965207>
- Faden, A. I., Demediuk, P., Panter, S. S., & Vink, R. (1989). The role of excitatory amino acids and NMDA receptors in traumatic brain injury. *Science (New York, N.Y.)*, *244*(4906), 798–800. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=2567056&site=ehost-live&scope=site>
- Faul, M., & Coronado, V. (2015). Epidemiology of traumatic brain injury. *Handbook Of Clinical Neurology*, *127*, 3–13. <https://doi.org/10.1016/B978-0-444-52892-6.00001-5>
- Fazio, V. C., Lovell, M. R., Pardini, J. E., & Collins, M. W. (2007). The relation between post concussion symptoms and neurocognitive performance in concussed athletes. *Neurorehabilitation*, *22*(3), 207–216. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=17917171&site=ehost-live&scope=site>



- Fife, T. D., & Kalra, D. (2015). Persistent vertigo and dizziness after mild traumatic brain injury. *Annals Of The New York Academy Of Sciences*, 1343, 97–105. <https://doi.org/10.1111/nyas.12678>
- Fife, T., Tusa, R., Furman, J., Zee, D., Frohman, E., Baloh, R., ... Eviatar, L. (2000). Assessment: Vestibular testing techniques in adults and children Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology*, 55(10), 1431–1441.
- Fino, P. C., Peterka, R. J., Hullar, T. E., Murchison, C., Horak, F. B., Chesnutt, J. C., & King, L. A. (2017). Assessment and rehabilitation of central sensory impairments for balance in mTBI using auditory biofeedback: a randomized clinical trial. *BMC Neurology*, 17(1). <https://doi.org/10.1186/s12883-017-0812-7>
- Frommer, L. J., Gurka, K. K., Cross, K. M., Ingersoll, C. D., Comstock, R. D., & Saliba, S. A. (2011). Sex differences in concussion symptoms of high school athletes. *Journal of Athletic Training*, 46(1), 76–84.
- Furman, J., Balaban, C., & Jacob, R. (2001). Interface between vestibular dysfunction and anxiety: more than just psychogenicity. *Otology & Neurotology*, 22(3), 426–427.
- Furman, J. M., Redfern, M. S., & Jacob, R. G. (2006). Vestibulo-ocular function in anxiety disorders. *Journal of Vestibular Research: Equilibrium & Orientation*, 16(4–5), 209–215.
- Gennarelli, T. A., Thibault, L. E., Adams, J. H., Graham, D. I., Thompson, C. J., & Marcincin, R. P. (1982). Diffuse axonal injury and traumatic coma in the primate. *Annals Of Neurology*, 12(6), 564–574. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=7159060&site=ehost-live&scope=site>
- Gennarelli, T. A., Thibault, L. E., & Ommaya, A. K. (1972). Pathophysiologic Responses to Rotational and Translational Accelerations of the Head. <https://doi.org/10.4271/720970>
- Gessel, L. M., Fields, S. K., Collins, C. L., Dick, R. W., & Comstock, R. D. (2007). Concussions among United States high school and collegiate athletes. *Journal of Athletic Training*, 42(4), 495.
- Giza, C. C., & Hovda, D. A. (2001). The Neurometabolic Cascade of Concussion. *Journal Of Athletic Training*, 36(3), 228–235. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=12937489&site=ehost-live&scope=site>
- Giza, C. C., & Hovda, D. A. (2014). The new neurometabolic cascade of concussion. *Neurosurgery*, 75 Suppl 4, S24–S33. <https://doi.org/10.1227/NEU.0000000000000505>
- Grubenhoff, J. A., Deakyne, S. J., Brou, L., Bajaj, L., Comstock, R. D., & Kirkwood, M. W. (2014). Acute concussion symptom severity and delayed symptom resolution. *Pediatrics*, 134(1), 54–62.

- Grunfeld, E. A., Gresty, M. A., Bronstein, A. M., & Jahanshahi, M. (2003). Screening for depression among neuro-otology patients with and without identifiable vestibular lesions. *International Journal Of Audiology*, *42*(3), 161–165. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=12705781&site=ehost-live&scope=site>
- Gulliver, A., Griffiths, K. M., Mackinnon, A., Batterham, P. J., & Stanimirovic, R. (2015). The mental health of Australian elite athletes. *Journal Of Science And Medicine In Sport*, *18*(3), 255–261. <https://doi.org/10.1016/j.jsams.2014.04.006>
- Gunning, M., Denison, F., Stockley, C., Ho, S., Sandhu, H., & Reynolds, R. (2010). Assessing maternal anxiety in pregnancy with the State- Trait Anxiety Inventory (STAI): issues of validity, location and participation. *Journal of Reproductive and Infant Psychology*, *28*(3), 266–273.
- Gurdjian, E. S., LISSNER, H. R., EVANS, F. G., PATRICK, L. M., & HARDY, W. G. (1961). Intracranial pressure and acceleration accompanying head impacts in human cadavers. *Surgery, Gynecology & Obstetrics*, *113*, 185–190. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=13709837&site=ehost-live&scope=site>
- Gurdjian, E. S., Webster, J. E., & Lissner, H. R. (1955). Observations on the mechanism of brain concussion, contusion, and laceration. *Surgery, Gynecology & Obstetrics*, *101*(6), 680–690.
- Guskiewicz, K. M., Weaver, N. L., Padua, D. A., & Garrett, W. E. (2000). Epidemiology of concussion in collegiate and high school football players. *The American Journal of Sports Medicine*, *28*(5), 643–650.
- Guskiewicz KM, & Mihalik JP. (2011). Biomechanics of sport concussion: quest for the elusive injury threshold. *Exercise & Sport Sciences Reviews*, *39*(1), 4–11. <https://doi.org/10.1097/JES.0b013e318201f53e>
- Hain, T. C., & Helminski, J. O. (2014). Anatomy and Physiology of the Normal Vestibular System. In *Vestibular rehabilitation*. Philadelphia, PA: FA Davis.
- Harmon, K. G., Drezner, J., Gammons, M., Guskiewicz, K., Halstead, M., Herring, S., ... Roberts, W. (2013). American Medical Society for Sports Medicine position statement: concussion in sport. *Clinical Journal Of Sport Medicine: Official Journal Of The Canadian Academy Of Sport Medicine*, *23*(1), 1–18. <https://doi.org/10.1097/JSM.0b013e31827f5f93>
- Hauck, L. J., Carpenter, M. G., & Frank, J. S. (2008). Task-specific measures of balance efficacy, anxiety, and stability and their relationship to clinical balance performance. *Gait & Posture*, *27*(4), 676–682.
- Heinmiller, L., & Gunton, K. B. (2016). A review of the current practice in diagnosis and management of visual complaints associated with concussion and postconcussion

- syndrome. *Current Opinion In Ophthalmology*, 27(5), 407–412.  
<https://doi.org/10.1097/ICU.0000000000000296>
- Heitger, M H, Anderson, T. J., & Jones, R. D. (2002). Saccade sequences as markers for cerebral dysfunction following mild closed head injury. *Progress In Brain Research*, 140, 433–448. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=cmedm&AN=12508607&site=ehost-live&scope=site>
- Heitger, Marcus H, Anderson, T. J., Jones, R. D., Dalrymple-Alford, J. C., Frampton, C. M., & Ardagh, M. W. (2004). Eye movement and visuomotor arm movement deficits following mild closed head injury. *Brain: A Journal Of Neurology*, 127(Pt 3), 575–590. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=cmedm&AN=14736751&site=ehost-live&scope=site>
- Heitger, Marcus H., Jones, R. D., Macleod, A. D., Snell, D. L., Frampton, C. M., & Anderson, T. J. (2009). Impaired eye movements in post-concussion syndrome indicate suboptimal brain function beyond the influence of depression, malingering or intellectual ability. *Brain: A Journal of Neurology*, 132(10), 2850–2870. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=a9h&AN=45305380&site=ehost-live&scope=site>
- Henry, L. C., Elbin, R. J., Collins, M. W., Marchetti, G., & Kontos, A. P. (2016). Examining Recovery Trajectories After Sport-Related Concussion With a Multimodal Clinical Assessment Approach: *Neurosurgery*, 78(2), 232–241.  
<https://doi.org/10.1227/NEU.0000000000001041>
- Hodgson, V. R., Thomas, L. M., Gurdjian, E. S., Fernando, O. U., Greenberg, S. W., & Chason, J. L. (1969). Advances in Understanding of Experimental Concussion Mechanisms1. SAE International. <https://doi.org/10.4271/690796>
- Hoffer, M. E., Balough, B. J., & Gottshall, K. R. (2007). Posttraumatic balance disorders. *The International Tinnitus Journal*, 13(1), 69–72. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=17691667&site=ehost-live&scope=site>
- Holbourn, A. (1943). Mechanics of head injuries. *The Lancet*, 242(6267), 438–441.
- Iverson, G. L., Brooks, B. L., Collins, M. W., & Lovell, M. R. (2006). Tracking neuropsychological recovery following concussion in sport. *Brain Injury*, 20(3), 245–252. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=16537266&site=ehost-live&scope=site>
- Iverson, G. L., Lovell, M. R., & Collins, M. W. (2003). Interpreting change on ImPACT following sport concussion. *The Clinical Neuropsychologist*, 17(4), 460–467.

- Iverson, G. L., Lovell, M. R., & Collins, M. W. (2005). Validity of ImPACT for measuring processing speed following sports-related concussion. *Journal of Clinical and Experimental Neuropsychology*, 27(6), 683–689.
- Jacob, R. G., & Furman, J. M. (2001). Psychiatric consequences of vestibular dysfunction. *Current Opinion in Neurology*, 14(1), 41–46.
- Kapoor, N., & Ciuffreda, K. J. (2002). Vision disturbances following traumatic brain injury. *Current Treatment Options in Neurology*, 4(4), 271–280.
- Katayama, Y., Becker, D. P., Tamura, T., & Hovda, D. A. (1990). Massive increases in extracellular potassium and the indiscriminate release of glutamate following concussive brain injury. *Journal Of Neurosurgery*, 73(6), 889–900. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=1977896&site=ehost-live&scope=site>
- Kawamata, T., Katayama, Y., Hovda, D. A., Yoshino, A., & Becker, D. P. (1992). Administration of excitatory amino acid antagonists via microdialysis attenuates the increase in glucose utilization seen following concussive brain injury. *Journal Of Cerebral Blood Flow And Metabolism: Official Journal Of The International Society Of Cerebral Blood Flow And Metabolism*, 12(1), 12–24. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=1345756&site=ehost-live&scope=site>
- Kennedy, P., & Rogers, B. A. (2000). Anxiety and depression after spinal cord injury: a longitudinal analysis. *Archives of Physical Medicine and Rehabilitation*, 81(7), 932–937.
- Khan, S., & Chang, R. (2013). Anatomy of the vestibular system: a review. *NeuroRehabilitation*, 32(3), 437–443.
- King, A. I., Yang, K. H., Zhang, L., Hardy, W., & Viano, D. C. (2003). Is head injury caused by linear or angular acceleration (pp. 1–12). Presented at the IRCOBI conference.
- Kleffelgaard, I., Roe, C., Soberg, H. L., & Bergland, A. (2012a). Associations among self-reported balance problems, post-concussion symptoms and performance-based tests: a longitudinal follow-up study. *Disability And Rehabilitation*, 34(9), 788–794. <https://doi.org/10.3109/09638288.2011.619624>
- Kleffelgaard, I., Roe, C., Soberg, H. L., & Bergland, A. (2012b). Associations among self-reported balance problems, post-concussion symptoms and performance-based tests: a longitudinal follow-up study. *Disability And Rehabilitation*, 34(9), 788–794. <https://doi.org/10.3109/09638288.2011.619624>
- Kontos, A. P., Elbin, R. J., Schatz, P., Covassin, T., Henry, L., Pardini, J., & Collins, M. W. (2012). A revised factor structure for the post-concussion symptom scale: baseline and postconcussion factors. *The American Journal Of Sports Medicine*, 40(10), 2375–2384. Retrieved from <http://0->

search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=22904209  
&site=ehost-live&scope=site

- Kontos, A. P., Sufrinko, A., Elbin, R. J., Puskar, A., & Collins, M. W. (2016). Reliability and Associated Risk Factors for Performance on the Vestibular/Ocular Motor Screening (VOMS) Tool in Healthy Collegiate Athletes. *The American Journal Of Sports Medicine*, 44(6), 1400–1406. <https://doi.org/10.1177/0363546516632754>
- Kraus, M. F., Little, D. M., Donnell, A. J., Reilly, J. L., Simonian, N., & Sweeney, J. A. (2007). Oculomotor function in chronic traumatic brain injury. *Cognitive And Behavioral Neurology: Official Journal Of The Society For Behavioral And Cognitive Neurology*, 20(3), 170–178. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=cmedm&AN=17846516&site=ehost-live&scope=site>
- Kroshus, E., Baugh, C. M., Daneshvar, D. H., Stamm, J. M., Laursen, R. M., & Austin, S. B. (2015). Pressure on Sports Medicine Clinicians to Prematurely Return Collegiate Athletes to Play After Concussion. *Journal of Athletic Training (Allen Press)*, 50(9), 944–951. <https://doi.org/10.4085/1062-6050-50.6.03>
- Kroshus, E., Garnett, B., Hawrilenko, M., Baugh, C. M., & Calzo, J. P. (2015a). Concussion under-reporting and pressure from coaches, teammates, fans, and parents. *Social Science & Medicine*, 134, 66–75. <https://doi.org/10.1016/j.socscimed.2015.04.011>
- Kroshus, E., Garnett, B., Hawrilenko, M., Baugh, C. M., & Calzo, J. P. (2015b). Concussion under-reporting and pressure from coaches, teammates, fans, and parents. *Social Science & Medicine*, 134, 66–75.
- Lahmann, C., Henningsen, P., Brandt, T., Strupp, M., Jahn, K., Dieterich, M., ... Schmid, G. (2015). Psychiatric comorbidity and psychosocial impairment among patients with vertigo and dizziness. *Journal Of Neurology, Neurosurgery, And Psychiatry*, 86(3), 302–308. <https://doi.org/10.1136/jnnp-2014-307601>
- Lange, R. T., Iverson, G. L., & Rose, A. (2011). Depression strongly influences postconcussion symptom reporting following mild traumatic brain injury. *The Journal Of Head Trauma Rehabilitation*, 26(2), 127–137. <https://doi.org/10.1097/HTR.0b013e3181e4622a>
- Langlois, J. A., Rutland-Brown, W., & Wald, M. M. (2006). The epidemiology and impact of traumatic brain injury: a brief overview. *The Journal Of Head Trauma Rehabilitation*, 21(5), 375–378. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=16983222&site=ehost-live&scope=site>
- Laretzaki, G., Plainis, S., Vrettos, I., Chrisoulakis, A., Pallikaris, I., & Bitsios, P. (2011). Threat and trait anxiety affect stability of gaze fixation. *Biological Psychology*, 86(3), 330–336.

- Lau, B. C., Collins, M. W., & Lovell, M. R. (2012). Cutoff scores in neurocognitive testing and symptom clusters that predict protracted recovery from concussions in high school athletes. *Neurosurgery*, *70*(2), 371–379. <https://doi.org/10.1227/NEU.0b013e31823150f0>
- Lau, B., Lovell, M. R., Collins, M. W., & Pardini, J. (2009). Neurocognitive and symptom predictors of recovery in high school athletes. *Clinical Journal of Sport Medicine*, *19*(3), 216–221.
- Leddy JJ, Kozlowski K, Donnelly JP, Pendergast DR, Epstein LH, & Willer B. (2010). A preliminary study of subsymptom threshold exercise training for refractory post-concussion syndrome. *Clinical Journal of Sport Medicine*, *20*(1), 21–27. <https://doi.org/10.1097/JSM.0b013e3181c6c22c>
- Leddy, M. H., Lambert, M. J., & Ogles, B. M. (1994). Psychological consequences of athletic injury among high-level competitors. *Research Quarterly for Exercise and Sport*, *65*(4), 347–354.
- Lee, S., Abdel Razek, O., & Dorfman, B. (2011). Vestibular system anatomy. Retrieved from: *Emedicine. Medscape. Com/Article/883956-Overview Aw2aab6c10*. Accessed August, 30.
- Lincoln, A. E., Caswell, S. V., Almquist, J. L., Dunn, R. E., Norris, J. B., & Hinton, R. Y. (2011). Trends in concussion incidence in high school sports: a prospective 11-year study. *The American Journal of Sports Medicine*, *39*(5), 958–963.
- Liu, J., & Li, L. (2013). Parent-reported mild head injury history and behavioural performance in children at 6 years. *Brain Injury*, *27*(11), 1263–1270.
- Llewellyn, T., Burdette, G. T., Joyner, A. B., & Buckley, T. A. (2014). Concussion Reporting Rates at the Conclusion of an Intercollegiate Athletic Career. *Clinical Journal of Sport Medicine*, *24*(1), 76–79. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=ccm&AN=107928234&site=ehost-live&scope=site>
- Lovell, M., Collins, M., Podell, K., Powell, J., & Maroon, J. (2000). ImPACT: Immediate post-concussion assessment and cognitive testing. *Pittsburgh, PA: NeuroHealth Systems, LLC*.
- Lovell, M. R., & Collins, M. W. (1998). Neuropsychological assessment of the college football player. *The Journal of Head Trauma Rehabilitation*, *13*(2), 9–26.
- Makdissi, M., Cantu, R. C., Johnston, K. M., McCrory, P., & Meeuwisse, W. H. (2013). The difficult concussion patient: what is the best approach to investigation and management of persistent (>10 days) postconcussive symptoms? *British Journal Of Sports Medicine*, *47*(5), 308–313. <https://doi.org/10.1136/bjsports-2013-092255>
- Marar, M., McIlvain, N. M., Fields, S. K., & Comstock, R. D. (2012). Epidemiology of concussions among United States high school athletes in 20 sports. *The American Journal Of Sports Medicine*, *40*(4), 747–755. <https://doi.org/10.1177/0363546511435626>

- Mata, M., Staple, J., & Fink, D. J. (1986). Changes in intra-axonal calcium distribution following nerve crush. *Journal Of Neurobiology*, *17*(5), 449–467. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=3772363&site=ehost-live&scope=site>
- Matheson, A. J., Darlington, C. L., & Smith, P. F. (1999). Dizziness in the elderly and age-related degeneration of the vestibular system. *New Zealand Journal of Psychology*, *28*(1), 10.
- Matta, F. V., & Enticott, J. C. (2004). The effects of state of alertness on the vestibulo-ocular reflex in normal subjects using the vestibular rotational chair. *Journal Of Vestibular Research: Equilibrium & Orientation*, *14*(5), 387–391. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=15598993&site=ehost-live&scope=site>
- Max, J. E., Schachar, R. J., Landis, J., Bigler, E. D., Wilde, E. A., Saunders, A. E., ... Levin, H. S. (2013). Psychiatric disorders in children and adolescents in the first six months after mild traumatic brain injury. *The Journal Of Neuropsychiatry And Clinical Neurosciences*, *25*(3), 187–197. <https://doi.org/10.1176/appi.neuropsych.12010011>
- Maxwell, W. L., McCreath, B. J., Graham, D. I., & Gennarelli, T. A. (1995). Cytochemical evidence for redistribution of membrane pump calcium-ATPase and ecto-Ca-ATPase activity, and calcium influx in myelinated nerve fibres of the optic nerve after stretch injury. *Journal Of Neurocytology*, *24*(12), 925–942. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=8719820&site=ehost-live&scope=site>
- Maxwell, W. L., Povlishock, J. T., & Graham, D. L. (1997). A mechanistic analysis of nondisruptive axonal injury: a review. *Journal Of Neurotrauma*, *14*(7), 419–440. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=9257661&site=ehost-live&scope=site>
- McClincy, M. P., Lovell, M. R., Pardini, J., Collins, M. W., & Spore, M. K. (2006). Recovery from sports concussion in high school and collegiate athletes. *Brain Injury*, *20*(1), 33–39.
- McCrea M, Hammeke T, Olsen G, Leo P, & Guskiewicz K. (2004). Unreported concussion in high school football players: implications for prevention. *Clinical Journal of Sport Medicine*, *14*(1), 13–17. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=ccm&AN=106676606&site=ehost-live&scope=site>
- McCrea, M, Kelly, J. P., Kluge, J., Ackley, B., & Randolph, C. (1997). Standardized assessment of concussion in football players. *Neurology*, *48*(3), 586–588.
- McCrea, Michael, Barr, W. B., Guskiewicz, K., Randolph, C., Marshall, S. W., Cantu, R., ... Kelly, J. P. (2005). Standard regression-based methods for measuring recovery after sport-related concussion. *Journal Of The International Neuropsychological Society*:

- JINS*, 11(1), 58–69. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=15686609&site=ehost-live&scope=site>
- McCrea, Michael, Guskiewicz, K. M., Marshall, S. W., Barr, W., Randolph, C., Cantu, R. C., ... Kelly, J. P. (2003). Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. *Jama*, 290(19), 2556–2563.
- McCrea, Michael, Iverson, G. L., Echemendia, R. J., Makdissi, M., & Raftery, M. (2013). Day of injury assessment of sport-related concussion. *British Journal Of Sports Medicine*, 47(5), 272–284. <https://doi.org/10.1136/bjsports-2013-092145>
- McCrory, P., Meeuwisse, W., Dvořák, J., Aubry, M., Bailes, J., Broglio, S., ... Vos, P. E. (2017). Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016. *British Journal Of Sports Medicine*, 51(11), 838–847. <https://doi.org/10.1136/bjsports-2017-097699>
- McCrory, P., Meeuwisse, W. H., Aubry, M., Cantu, R. C., Dvořák, J., Echemendia, R. J., ... Turner, M. (2013). Consensus statement on concussion in sport: the 4th International Conference on Concussion in Sport, Zurich, November 2012. *Journal Of Athletic Training*, 48(4), 554–575. <https://doi.org/10.4085/1062-6050-48.4.05>
- Meaney, D. F., & Smith, D. H. (2011). Biomechanics of Concussion. *Clinics in Sports Medicine*, 30(1), 19–31. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=s3h&AN=55741480&site=ehost-live&scope=site>
- Meehan, W. P., 3rd, Mannix, R. C., Stracciolini, A., Elbin, R. J., & Collins, M. W. (2013). Symptom severity predicts prolonged recovery after sport-related concussion, but age and amnesia do not. *The Journal Of Pediatrics*, 163(3), 721–725. <https://doi.org/10.1016/j.jpeds.2013.03.012>
- Meehan, W. P. I., Taylor, A. M., Berkner, P., Sandstrom, N. J., Peluso, M. W., Kurtz, M. M., ... Mannix, R. (2016). Division III collision sports are not associated with neurobehavioral quality of life. *Journal of Neurotrauma*, 33(2), 254–259. <https://doi.org/10.1089/neu.2015.3930>
- Meehan, W. P., & Mannix, R. (2010). Pediatric concussions in United States emergency departments in the years 2002 to 2006. *The Journal of Pediatrics*, 157(6), 889–893.
- Meehan, W. P., Mannix, R. C., O'Brien, M. J. ., & Collins, M. W. (2013). The Prevalence of Undiagnosed Concussions in Athletes. *Clinical Journal of Sport Medicine*, 23(5), 339–342. <https://doi.org/10.1097/JSM.0b013e318291d3b3>
- Meier, T. B., Bellgowan, P. S. F., Singh, R., Kuplicki, R., Polanski, D. W., & Mayer, A. R. (2015). Recovery of cerebral blood flow following sports-related concussion. *JAMA Neurology*, 72(5), 530–538. <https://doi.org/10.1001/jamaneurol.2014.4778>



- Mescher, A. L. (2013). *Junqueira's basic histology: text and atlas*. McGraw-hill.
- Moore, R. D., Sauve, W., & Ellemberg, D. (2016). Neurophysiological correlates of persistent psycho-affective alterations in athletes with a history of concussion. *Brain Imaging and Behavior*, *10*(4), 1108–1116. <https://doi.org/10.1007/s11682-015-9473-6>
- Moriya, J., & Sugiura, Y. (2012). High visual working memory capacity in trait social anxiety. *Plos One*, *7*(4), e34244–e34244. <https://doi.org/10.1371/journal.pone.0034244>
- Mrazik, M., Brooks, B. L., Jubinville, A., Meeuwisse, W. H., & Emery, C. A. (2016). Psychosocial Outcomes of Sport Concussions in Youth Hockey Players. *Archives of Clinical Neuropsychology*, *31*(4), 297–304. <https://doi.org/10.1093/arclin/acw013>
- Mucha, A., Collins, M. W., Elbin, R. J., Furman, J. M., Troutman-Enseki, C., DeWolf, R. M., ... Kontos, A. P. (2014). A Brief Vestibular/Ocular Motor Screening (VOMS) assessment to evaluate concussions: preliminary findings. *The American Journal Of Sports Medicine*, *42*(10), 2479–2486. <https://doi.org/10.1177/0363546514543775>
- Nakamura, Y., Takeda, M., Angelides, K. J., Tanaka, T., Tada, K., & Nishimura, T. (1990). Effect of phosphorylation on 68 KDa neurofilament subunit protein assembly by the cyclic AMP dependent protein kinase in vitro. *Biochemical And Biophysical Research Communications*, *169*(2), 744–750. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=2357230&site=ehost-live&scope=site>
- Naranjo, E. N., Cleworth, T. W., Allum, J. H. J., Inglis, J. T., Lea, J., Westerberg, B. D., & Carpenter, M. G. (2016). Vestibulo-spinal and vestibulo-ocular reflexes are modulated when standing with increased postural threat. *Journal Of Neurophysiology*, *115*(2), 833–842. <https://doi.org/10.1152/jn.00626.2015>
- Naranjo, E. N., Cleworth, T. W., Allum, J. H. J., Inglis, J. T., Lea, J., Westerberg, B. D., & Carpenter, M. G. (2017a). Threat effects on human oculo-motor function. *Neuroscience*, *359*(Supplement C), 289–298. <https://doi.org/10.1016/j.neuroscience.2017.07.024>
- Naranjo, E. N., Cleworth, T. W., Allum, J. H. J., Inglis, J. T., Lea, J., Westerberg, B. D., & Carpenter, M. G. (2017b). Threat effects on human oculo-motor function. *Neuroscience*, *359*, 289–298. <https://doi.org/10.1016/j.neuroscience.2017.07.024>
- Nashner, L. M., Black, F. O., & Wall, C. (1982). Adaptation to altered support and visual conditions during stance: patients with vestibular deficits. *Journal of Neuroscience*, *2*(5), 536–544.
- Nelson, L. D., Guskiewicz, K. M., Marshall, S. W., Hammeke, T., Barr, W., Randolph, C., & McCrea, M. A. (2016). Multiple Self-Reported Concussions Are More Prevalent in Athletes With ADHD and Learning Disability. *Clinical Journal Of Sport Medicine: Official Journal Of The Canadian Academy Of Sport Medicine*, *26*(2), 120–127. <https://doi.org/10.1097/JSM.0000000000000207>

- Nelson, L. D., LaRoche, A. A., Pfaller, A. Y., Lerner, E. B., Hammeke, T. A., Randolph, C., ... McCrea, M. A. (2016). Prospective, Head-to-Head Study of Three Computerized Neurocognitive Assessment Tools (CNTs): Reliability and Validity for the Assessment of Sport-Related Concussion. *Journal Of The International Neuropsychological Society: JINS*, 22(1), 24–37. <https://doi.org/10.1017/S1355617715001101>
- Nelson, L. D., Tarima, S., LaRoche, A. A., Hammeke, T. A., Barr, W. B., Guskiewicz, K., ... McCrea, M. A. (2016). Preinjury somatization symptoms contribute to clinical recovery after sport-related concussion. *Neurology*, 86(20), 1856–1863. <https://doi.org/10.1212/WNL.0000000000002679>
- Neuhauser, H. K. (2016). The epidemiology of dizziness and vertigo. *Handbook Of Clinical Neurology*, 137, 67–82. <https://doi.org/10.1016/B978-0-444-63437-5.00005-4>
- Neuhauser, H. K., von Brevern, M., Radtke, A., Lezius, F., Feldmann, M., Ziese, T., & Lempert, T. (2005). Epidemiology of vestibular vertigo: a neurotologic survey of the general population. *Neurology*, 65(6), 898–904. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=16186531&site=ehost-live&scope=site>
- Nixon, R. A. (1993). The regulation of neurofilament protein dynamics by phosphorylation: clues to neurofibrillary pathobiology. *Brain Pathology (Zurich, Switzerland)*, 3(1), 29–38. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=7505700&site=ehost-live&scope=site>
- Norre, M., Forrez, G., & Beckers, A. (1987). Vestibular dysfunction causing instability in aged patients. *Acta Oto-Laryngologica*, 104(1–2), 50–55.
- Oghalai, J. S., & Brownell, W. E. (2006). Chapter 44. Anatomy & Physiology of the Ear. In *Archives of Facial Plastic Surgery* (3e ed., Vol. 8).
- Ohno, H., Wada, M., Saitoh, J., Sunaga, N., & Nagai, M. (2004). The effect of anxiety on postural control in humans depends on visual information processing. *Neuroscience Letters*, 364(1), 37–39.
- Ommaya, A K, & Hirsch, A. E. (1971). Tolerances for cerebral concussion from head impact and whiplash in primates. *Journal Of Biomechanics*, 4(1), 13–21. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=5001829&site=ehost-live&scope=site>
- Ommaya, Ayub K, Hirsch, A., Flamm, E., & Mahone, R. (1966). Cerebral concussion in the monkey: an experimental model. *Science*, 153(3732), 211–212.
- Pardini, D., Stump, J., Lovell, M., Collins, M., Moritz, K., & Fu, F. (2004). 032 The Post-concussion Symptom Scale (pcss): A Factor Analysis. *British Journal of Sports Medicine*, 38(5), 661–662.

- Patel, A. V., Mihalik, J. P., Notebaert, A. J., Guskiewicz, K. M., & Prentice, W. E. (2007). Neuropsychological performance, postural stability, and symptoms after dehydration. *Journal of Athletic Training, 42*(1), 66.
- Pettus, E. H., & Povlishock, J. T. (1996). Characterization of a distinct set of intra-axonal ultrastructural changes associated with traumatically induced alteration in axolemmal permeability. *Brain Research, 722*(1–2), 1–11. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=8813344&site=ehost-live&scope=site>
- Piland, S. G., Motl, R. W., Ferrara, M. S., & Peterson, C. L. (2003). Evidence for the factorial and construct validity of a self-report concussion symptoms scale. *Journal of Athletic Training, 38*(2), 104.
- Ponsford, J., Cameron, P., Fitzgerald, M., Grant, M., Mikocka-Walus, A., & Schönberger, M. (2012). Predictors of postconcussive symptoms 3 months after mild traumatic brain injury. *Neuropsychology, 26*(3), 304–313. <https://doi.org/10.1037/a0027888>
- Povlishock, J. T., & Pettus, E. H. (1996). Traumatically induced axonal damage: evidence for enduring changes in axolemmal permeability with associated cytoskeletal change. *Acta Neurochirurgica. Supplement, 66*, 81–86. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=8780803&site=ehost-live&scope=site>
- Powell, J. W., & Barber-Foss, K. D. (1999). Traumatic brain injury in high school athletes. *Jama, 282*(10), 958–963.
- Prichep, L. S., McCrea, M., Barr, W., Powell, M., & Chabot, R. J. (2013). Time course of clinical and electrophysiological recovery after sport-related concussion. *The Journal Of Head Trauma Rehabilitation, 28*(4), 266–273. <https://doi.org/10.1097/HTR.0b013e318247b54e>
- Prins, M. L., Hales, A., Reger, M., Giza, C. C., & Hovda, D. A. (2010). Repeat traumatic brain injury in the juvenile rat is associated with increased axonal injury and cognitive impairments. *Developmental Neuroscience, 32*(5–6), 510–518. <https://doi.org/10.1159/000316800>
- Putukian, M., Echemendia, R., Dettwiler-Danspeckgruber, A., Duliba, T., Bruce, J., Furtado, J. L., & Murugavel, M. (2015). Prospective Clinical Assessment Using Sideline Concussion Assessment Tool-2 Testing in the Evaluation of Sport-Related Concussion in College Athletes. *Clinical Journal of Sport Medicine, 25*(1), 36–42. <https://doi.org/10.1097/JSM.000000000000102>
- Qi, S., Ding, C., & Li, H. (2014). Neural correlates of inefficient filtering of emotionally neutral distractors from working memory in trait anxiety. *Cognitive, Affective & Behavioral Neuroscience, 14*(1), 253–265. <https://doi.org/10.3758/s13415-013-0203-5>

- Quatman-Yates, C., Hugentobler, J., Ammon, R., Mwase, N., Kurowski, B., & Myer, G. D. (2014). The utility of the balance error scoring system for mild brain injury assessments in children and adolescents. *The Physician And Sportsmedicine*, *42*(3), 32–38. <https://doi.org/10.3810/psm.2014.09.2073>
- Reddy, C. C., Collins, M., Lovell, M., & Kontos, A. P. (2013). Efficacy of amantadine treatment on symptoms and neurocognitive performance among adolescents following sports-related concussion. *The Journal Of Head Trauma Rehabilitation*, *28*(4), 260–265. <https://doi.org/10.1097/HTR.0b013e318257fbc6>
- Reynolds, E., Collins, M. W., Mucha, A., & Troutman-Ensecki, C. (2014). Establishing a clinical service for the management of sports-related concussions. *Neurosurgery*, *75* Suppl 4, S71–S81. <https://doi.org/10.1227/NEU.0000000000000471>
- Richards, H. J., Benson, V., & Hadwin, J. A. (2012). The attentional processes underlying impaired inhibition of threat in anxiety: The remote distractor effect. *Cognition & Emotion*, *26*(5), 934–942.
- Riemann, B. L., Guskiewicz, K. M., & Shields, E. W. (1999). Relationship between clinical and forceplate measures of postural stability. *Journal of Sport Rehabilitation*, *8*(2), 71–82.
- Ropper, A., & Samuels, M. (2009). Chapter 15. Deafness, Dizziness, and Disorders of Equilibrium. *Adams and Victor's Principles of Neurology*, *9e*. Retrieved August, 17, 2012.
- Rowson, S., Bland, M. L., Campoletano, E. T., Press, J. N., Rowson, B., Smith, J. A., ... Duma, S. M. (2016). Biomechanical Perspectives on Concussion in Sport: *Sports Medicine and Arthroscopy Review*, *24*(3), 100–107. <https://doi.org/10.1097/JSA.0000000000000121>
- Saatman, K. E., Abai, B., Grosvenor, A., Vorwerk, C. K., Smith, D. H., & Meaney, D. F. (2003). Traumatic axonal injury results in biphasic calpain activation and retrograde transport impairment in mice. *Journal Of Cerebral Blood Flow And Metabolism: Official Journal Of The International Society Of Cerebral Blood Flow And Metabolism*, *23*(1), 34–42. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=12500089&site=ehost-live&scope=site>
- Schaal, K., Tafflet, M., Nassif, H., Thibault, V., Pichard, C., Alcotte, M., ... Toussaint, J.-F. (2011). Psychological balance in high level athletes: gender-based differences and sport-specific patterns. *Plos One*, *6*(5), e19007–e19007. <https://doi.org/10.1371/journal.pone.0019007>
- Schatz, P., Pardini, J. E., Lovell, M. R., Collins, M. W., & Podell, K. (2006). Sensitivity and specificity of the ImPACT Test Battery for concussion in athletes. *Archives of Clinical Neuropsychology*, *21*(1), 91–99.
- Schoenhuber, R., & Gentilini, M. (1988). Anxiety and depression after mild head injury: a case control study. *Journal of Neurology, Neurosurgery & Psychiatry*, *51*(5), 722–724.

- Schulz, M. R., Marshall, S. W., Mueller, F. O., Yang, J., Weaver, N. L., Kalsbeek, W. D., & Bowling, J. M. (2004). Incidence and risk factors for concussion in high school athletes, North Carolina, 1996–1999. *American Journal of Epidemiology*, *160*(10), 937–944.
- Shankman, S. A., Gorka, S. M., Nelson, B. D., Fitzgerald, D. A., Phan, K. L., & O'daly, O. (2014). Anterior insula responds to temporally unpredictable aversiveness: an fMRI study. *Neuroreport*, *25*(8), 596.
- Shefer, S., Gordon, C. R., Avraham, K. B., & Mintz, M. (2010). Progressive vestibular mutation leads to elevated anxiety. *Brain Research*, *1317*, 157–164.
- Shin, L. M., & Liberzon, I. (2010). The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology*, *35*(1), 169.
- Singleton, R. H., Zhu, J., Stone, J. R., & Povlishock, J. T. (2002). Traumatically induced axotomy adjacent to the soma does not result in acute neuronal death. *The Journal Of Neuroscience: The Official Journal Of The Society For Neuroscience*, *22*(3), 791–802. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=11826109&site=ehost-live&scope=site>
- Smouha, E., & Wanna, G. (2009). Vertigo. In *Encyclopedia of Neuroscience* (pp. 91–97). Springer.
- Spielberger, C. D. (1966). The effects of anxiety on complex learning and academic achievement. *Anxiety and Behavior*, 361–398.
- Spielberger, C., Gorsuch, R., Lushene, R., Vagg, P., & Jacobs, G. (1983). Manual for the state-trait anxiety inventory (Palo Alto, CA, Consulting Psychologists Press). *Inc.*
- Staab, J. P. (2014a). The influence of anxiety on ocular motor control and gaze: *Current Opinion in Neurology*, *27*(1), 118–124. <https://doi.org/10.1097/WCO.0000000000000055>
- Staab, J. P. (2014b). The influence of anxiety on ocular motor control and gaze: *Current Opinion in Neurology*, *27*(1), 118–124. <https://doi.org/10.1097/WCO.0000000000000055>
- Stein, M. B., Simmons, A. N., Feinstein, J. S., & Paulus, M. P. (2007). Increased amygdala and insula activation during emotion processing in anxiety-prone subjects. *American Journal of Psychiatry*, *164*(2), 318–327.
- Sternberger, L. A., & Sternberger, N. H. (1983). Monoclonal antibodies distinguish phosphorylated and nonphosphorylated forms of neurofilaments in situ. *Proceedings Of The National Academy Of Sciences Of The United States Of America*, *80*(19), 6126–6130. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=6577472&site=ehost-live&scope=site>

- Stewart, G. W., McQueen-Borden, E., Bell, R. A., Barr, T., & Juengling, J. (2012). Comprehensive assessment and management of athletes with sport concussion. *International Journal Of Sports Physical Therapy*, 7(4), 433–447. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=22893863&site=ehost-live&scope=site>
- Storch, E. A., Storch, J. B., Killiany, E. M., & Roberti, J. W. (2005). Self-Reported Psychopathology in Athletes: A Comparison of Intercollegiate Student-Athletes and Non-Athletes. *Journal of Sport Behavior*, 28(1), 86. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=f5h&AN=16091312&site=ehost-live&scope=site>
- Stout, D. M., Shackman, A. J., & Larson, C. L. (2013). Failure to filter: anxious individuals show inefficient gating of threat from working memory. *Frontiers In Human Neuroscience*, 7, 58–58. <https://doi.org/10.3389/fnhum.2013.00058>
- Suh, M., Basu, S., Kolster, R., Sarkar, R., McCandliss, B., & Ghajar, J. (2006). Increased oculomotor deficits during target blanking as an indicator of mild traumatic brain injury. *Neuroscience Letters*, 410(3), 203–207. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=cmedm&AN=17055156&site=ehost-live&scope=site>
- Suh, M., Kolster, R., Sarkar, R., McCandliss, B., & Ghajar, J. (2006). Deficits in predictive smooth pursuit after mild traumatic brain injury. *Neuroscience Letters*, 401(1/2), 108–113. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=a9h&AN=20875026&site=ehost-live&scope=site>
- Swinson, R. P., Cox, B. J., Rutka, J., Mai, M., Kerr, S., & Kuch, K. (1993). Otoneurological functioning in panic disorder patients with prominent dizziness. *Comprehensive Psychiatry*, 34(2), 127–129. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=8485981&site=ehost-live&scope=site>
- Takahashi, H., Manaka, S., & Sano, K. (1981). Changes in extracellular potassium concentration in cortex and brain stem during the acute phase of experimental closed head injury. *Journal Of Neurosurgery*, 55(5), 708–717. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=7310492&site=ehost-live&scope=site>
- Thomas, L. M., Roberts, V. L., & Gurdjian, E. S. (1966). Experimental intracranial pressure gradients in the human skull. *Journal Of Neurology, Neurosurgery, And Psychiatry*, 29(5), 404–411. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=5926463&site=ehost-live&scope=site>

- Unterharnscheidt, F., & Higgins, L. S. (1969). Traumatic lesions of brain and spinal cord due to nondeforming angular acceleration of the head. *Texas Reports On Biology And Medicine*, 27(1), 127–166. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=4976573&site=ehost-live&scope=site>
- Unterharnscheidt, F. J. (1971). *Translational versus rotational acceleration-animal experiments with measured input* (No. 0148–7191). SAE Technical Paper.
- Vagnozzi, R., Signoretti, S., Cristofori, L., Alessandrini, F., Floris, R., Isgrò, E., ... Lazzarino, G. (2010). Assessment of metabolic brain damage and recovery following mild traumatic brain injury: a multicentre, proton magnetic resonance spectroscopic study in concussed patients. *Brain: A Journal Of Neurology*, 133(11), 3232–3242. <https://doi.org/10.1093/brain/awq200>
- Ventura, R. E., Balcer, L. J., & Galetta, S. L. (2014). The neuro-ophthalmology of head trauma. *The Lancet. Neurology*, 13(10), 1006–1016. [https://doi.org/10.1016/S1474-4422\(14\)70111-5](https://doi.org/10.1016/S1474-4422(14)70111-5)
- Ventura, R. E., Balcer, L. J., & Galetta, S. L. (2015). The Concussion Toolbox: The Role of Vision in the Assessment of Concussion. *Seminars In Neurology*, 35(5), 599–606. <https://doi.org/10.1055/s-0035-1563567>
- Warden, D., Bleiberg, J., Cameron, K., Ecklund, J., Walter, J., Sparling, M., ... Arciero, R. (2001). Persistent prolongation of simple reaction time in sports concussion. *Neurology*, 57(3), 524–526.
- Waxman, S. G. (2010). Chapter 17. The Vestibular System. In *Clinical neuroanatomy* (Vol. 26e). McGraw Hill.
- Weinberg, R. S., & Gould, D. (2007). *Foundations of sport and exercise psychology*. Champaign, IL: Human Kinetics.
- Wilkins, S. A., Shannon, C. N., Brown, S. T., Vance, E. H., Ferguson, D., Gran, K., ... Johnston, J. M., Jr. (2014). Establishment of a multidisciplinary concussion program: impact of standardization on patient care and resource utilization. *Journal Of Neurosurgery. Pediatrics*, 13(1), 82–89. <https://doi.org/10.3171/2013.10.PEDS13241>
- Wood, R. L., McCabe, M., & Dawkins, J. (2011). The role of anxiety sensitivity in symptom perception after minor head injury: an exploratory study. *Brain Injury*, 25(13–14), 1296–1299. <https://doi.org/10.3109/02699052.2011.624569>
- World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders: diagnostic criteria for research* (Vol. 2). World Health Organization.
- Yang, J., Peek-Asa, C., Covassin, T., & Torner, J. C. (2015). Post-concussion symptoms of depression and anxiety in division I collegiate athletes. *Developmental Neuropsychology*, 40(1), 18–23. <https://doi.org/10.1080/87565641.2014.973499>

- Yeates, K. O., Luria, J., Bartkowski, H., Rusin, J., Martin, L., & Bigler, E. D. (1999). Postconcussive symptoms in children with mild closed head injuries. *The Journal Of Head Trauma Rehabilitation*, 14(4), 337–350. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=10407207&site=ehost-live&scope=site>
- Yorke, A. M., Smith, L., Babcock, M., & Alsalaheen, B. (2017a). Validity and Reliability of the Vestibular/Ocular Motor Screening and Associations With Common Concussion Screening Tools. *Sports Health*, 9(2), 174–180. <https://doi.org/10.1177/1941738116678411>
- Yorke, A. M., Smith, L., Babcock, M., & Alsalaheen, B. (2017b). Validity and Reliability of the Vestibular/Ocular Motor Screening and Associations With Common Concussion Screening Tools. *Sports Health: A Multidisciplinary Approach*, 9(2), 174–180. <https://doi.org/10.1177/1941738116678411>
- Yoshino, A., Hovda, D. A., Kawamata, T., Katayama, Y., & Becker, D. P. (1991). Dynamic changes in local cerebral glucose utilization following cerebral conclusion in rats: evidence of a hyper- and subsequent hypometabolic state. *Brain Research*, 561(1), 106–119. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=1797338&site=ehost-live&scope=site>
- Zhang, L., Yang, K. H., & King, A. I. (2001). Biomechanics of neurotrauma. *Neurological Research*, 23(2–3), 144–156.
- Zhang, L., Yang, K. H., & King, A. I. (2004). A proposed injury threshold for mild traumatic brain injury. *Journal Of Biomechanical Engineering*, 126(2), 226–236. Retrieved from <http://0-search.ebscohost.com.library.uark.edu/login.aspx?direct=true&db=mdc&AN=15179853&site=ehost-live&scope=site>



## Supplemental Analyses

### Controlling for Concussion History, Anxiety History, and Total PCSS Symptom Score

The data were explored further using a repeated measures analysis of covariance (ANCOVA), controlling for concussion history, anxiety history, and total PCSS symptom score, to test for statistical significance between PROV and NO PROV groups.

**Hypothesis 1.** The results of a 2 Group (PROV, NO PROV) X 2 Time (initial clinical visit, medical clearance clinical visit) repeated-measures ANCOVA, controlling for concussion history, supported a significant between-subjects effect for vestibular provocation groups ( $F_{1,1}=4.12, p=.05, \eta^2=.14$ ) on state anxiety. When controlling for anxiety history, it supported a significant between-subjects effect for provocation groups ( $F_{1,1}=5.57, p=.03, \eta^2=.17$ ) on state anxiety. When controlling for total PCSS symptom score, it supported a significant between-subjects effect for vestibular provocation groups ( $F_{1,1}=4.56, p=.04, \eta^2=.15$ ) on state anxiety. State anxiety scores were higher for PROV ( $M=43.73, SD=14.64$ ) and NO PROV ( $M=31.68, SD=9.15$ ) groups.

**Hypothesis 2.** There was not a significant within-subjects main effect for time ( $F_{1,1} = 0.01, p = .93, \eta^2 = .00$ ) for state anxiety when controlling for concussion history. There was not a significant within-subjects main effect for time ( $F_{1,1} = 1.48, p = .24, \eta^2 = .05$ ) for state anxiety when controlling for anxiety history. There was not a significant within-subjects main effect for time ( $F_{1,1} = 1.63, p = .21, \eta^2 = .06$ ) for state anxiety when controlling for total PCSS symptom score.

**Hypothesis 3.** The interaction between provocation groups and time was not significant (Wilks'  $\lambda = 1.00, p = .93, \eta^2 = .000$ ) when controlling for concussion history. The interaction between provocation groups and time was not significant (Wilks'  $\lambda = .95,$

$p = .24$ ,  $\eta^2 = .05$ ) when controlling for anxiety history. The interaction between provocation groups and time was not significant (Wilks'  $\lambda = .98$ ,  $p = .42$ ,  $\eta^2 = .03$ ) when controlling for total PCSS symptom score. State anxiety did not differ between PROV and NO PROV groups at either time point. The means and standard deviations did not change from previous statistical analyses.

### **Results of ANOVA Using Only Vestibular Components**

The data were explored further using only the vestibular components (e.g., horizontal and vertical VOR and visual motion sensitivity) of the VOMS. The PROV and NO PROV group means and standard deviations did not change.

The results of a 2 Group (PROV, NO PROV) X 2 Time (initial clinical visit, medical clearance clinical visit) repeated-measures ANOVA, using only the vestibular components (e.g., horizontal and vertical saccades and visual motion sensitivity) of the VOMS supported a significant within-subjects main effect for time ( $F_{1,1} = 6.95$ ,  $p = .01$ ,  $\eta^2 = .20$ ) for state anxiety, a significant between-subjects main effect for vestibular provocation groups ( $F_{1,1} = 8.38$ ,  $p = .01$ ,  $\eta^2 = .23$ ) on state anxiety; however, the interaction between vestibular provocation groups and time was not significant (Wilks'  $\lambda = .94$ ,  $p = .20$ ,  $\eta^2 = .06$ ). The data were explored further using only the vestibular components (e.g., horizontal and vertical VOR and visual motion sensitivity) of the VOMS, controlling for concussion history, anxiety history, and total PCSS symptom score.

## **Results of ANCOVA Using Only Vestibular Components, Controlling for Concussion History, Anxiety History, and Total PCSS Symptom Score**

The data were explored further using only the vestibular components (e.g., horizontal and vertical VOR and visual motion sensitivity) of the VOMS, controlling for concussion history, anxiety history, and total PCSS symptom score.

The results of a 2 Group (PROV, NO PROV) X 2 Time (initial clinical visit, medical clearance clinical visit) repeated-measures ANCOVA, controlling for concussion history, supported a significant between-subjects main effect for vestibular provocation groups ( $F_{1,1} = 4.12$ ,  $p = .05$ ,  $\eta^2 = .14$ ) on state anxiety; however, the within-subjects main effect for time on state anxiety was not significant ( $F_{1,1} = .01$ ,  $p = .93$ ,  $\eta^2 = .00$ ) and the interaction between vestibular provocation groups and time was not significant (Wilks'  $\lambda = .95$ ,  $p = .24$ ,  $\eta^2 = .05$ ).

The results of a 2 Group (PROV, NO PROV) X 2 Time (initial clinical visit, medical clearance clinical visit) repeated-measures ANCOVA, controlling for anxiety history, supported a significant between-subjects main effect for vestibular provocation groups ( $F_{1,1} = 5.57$ ,  $p = .03$ ,  $\eta^2 = .17$ ) on state anxiety; however, the within-subjects main effect for time on state anxiety was not significant ( $F_{1,1} = 1.48$ ,  $p = .24$ ,  $\eta^2 = .05$ ) and the interaction between vestibular provocation groups and time was not significant (Wilks'  $\lambda = .98$ ,  $p = .47$ ,  $\eta^2 = .02$ ).

The results of a 2 Group (PROV, NO PROV) X 2 Time (initial clinical visit, medical clearance clinical visit) repeated-measures ANCOVA, controlling for total PCSS symptom score, supported a significant between-subjects main effect for vestibular provocation groups ( $F_{1,1} = 4.56$ ,  $p = .04$ ,  $\eta^2 = .15$ ) on state anxiety; however, the within-subjects main effect for time on state anxiety was not significant ( $F_{1,1} = 1.63$ ,  $p = .21$ ,  $\eta^2 = .06$ ) and the interaction between vestibular provocation groups and time was not significant (Wilks'  $\lambda = .98$ ,  $p = .42$ ,  $\eta^2 = .03$ ).

These results indicate that state anxiety scores were significantly different between vestibular provocation groups when using only the vestibular components of the VOMS; however, state anxiety scores did not significantly differ between initial and medical clearance clinical visit and that there was no significant interaction between vestibular provocation group and time on state anxiety scores and when controlling for concussion history, anxiety history, or total PCSS symptom score.