Dynamic Postural Control In Individuals With Multiple Concussions

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ABSTRACT

There are an estimated 1.6 – 3.8 million sports-related concussions which occur in athletics annually in the United States; however, the actual rate may be higher, as many studies suggest that nearly half of the concussions that occur may go unreported. Recent evidence suggests life-long behavioral and cognitive impairments in individuals with multiple, usually 3 or more, concussions; however to date there has been limited assessment of motor impairments in this population. The transitional movement of gait initiation, literally the act of starting to walk, has effectively identified impairments in postural control in a wide range of neurologically impaired populations (e.g., post-stroke, Parkinson’s, aging). Therefore, the purpose of this study was to identify impairments in postural motor control in individuals with a history of 3 or more concussions. Subjects performed 5 trials of gait initiation and we compared their performance to healthy subjects with no history of concussion. We hypothesized that subjects with a history of concussions will display significant differences in spatiotemporal measurements and postural control through gait initiation when compared to healthy subjects. There were no significant main effects for group for displacement of COP during S1, S2, or S3 of G1 (F=0.809, P=0.581). There were no significant effects for group for separation of COP-COM during the GI task (F=0.062, P=0.992). There was also no significant main effect for group for spatiotemporal measures of step length and velocity (F=0.819, p=0.458). The results of this study suggest that athletes with a history of 3 or more concussions do not display deficits in postural control during GI compared to age and sport matched controls.

INDEX WORDS: Concussion, Postural control, Gait initiation.
DYNAMIC POSTURAL CONTROL IN INDIVIDUALS WITH MULTIPLE CONCUSSIONS

by

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DYNAMIC POSTURAL CONTROL IN INDIVIDUALS WITH MULTIPLE CONCUSSIONS

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CHAPTER 1

INTRODUCTION

There are an estimated 1.6 – 3.8 million sports-related concussions which occur in athletics annually in the United States.\(^1\) The actual rate may be higher, as some reports suggest nearly half of the concussions that occur may go unreported.\(^2\) A concussion is a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces to the head.\(^3\) There has been much debate over the past decade over whether the pathology associated with concussion is transient. Once thought to be purely functional neuronal disturbance, most experts now agree that there may also be structural pathology associated with concussion.\(^4\) Further, there is a growing body of research that suggests the presence of cumulative and long term effects resulting from multiple concussions.

Immediately following a concussion, there is a period of 7-10 days where a metabolic and physiological cascade of events occur causing neuronal dysfunction which presents as a plethora of neurocognitive dysfunction, postural control deficits, and clinical symptoms.\(^5\) During this period, the brain is susceptible to repeat injury that may be more severe than the initial injury or in some cases fatal due to the brain’s inability to recover from the repeat trauma.\(^5\)\(^-\)\(^7\) Additionally, athletes who have suffered multiple concussions may have an increased risk of suffering further concussions, as findings suggest that athletes who suffer 1 or 2 concussions are 1.5 to 2.8 times more likely to suffer a repeat concussion during the same season respectively.\(^8\)\(^,\)\(^9\) Furthermore, athletes with a previous history of 3 or more concussions are 3 to 6 times more likely to suffer recurrent concussion than those without previous concussion history.\(^8\)\(^,\)\(^9\)

Following cumulative concussions, specifically 3 or more, individuals may experience slower recovery and long-term symptoms as demonstrated through neurocognitive testing and
Recent findings suggest that multiple recurrent concussions may be risk factors for development of later-life cognitive and neural impairments including: early onset of Alzheimer’s Disease, mild cognitive impairment, clinically diagnosed depression, motor system dysfunction, Chronic Traumatic Encephalopathy (CTE), and Amyotrophic Lateral Sclerosis. Specifically, Guskiewicz reported, using retrospective surveys of retired professional football players, that athletes with a history of three or more concussions are at a significantly higher risk of developing early onset of Alzheimer’s disease, late life clinically diagnosed depression and mild cognitive impairment. De Beaumont et al. conducted a series of studies looking at cortical silent period and neuropsychological testing where they reported that retired athletes with a history of three or more concussions displayed long-term cognitive and primary motor cortex dysfunction. Further, recent case studies have reported the discovery of CTE after autopsy of deceased professional football players, a lethal condition reported to develop in at least 17% of boxers who suffer repetitive concussions. McKee et al. recently reported epidemiological evidence that repetitive head trauma experienced in collision sports may be associated with the development of amyotrophic lateral sclerosis, a fatal progressive degeneration of motor neurons in the brain and spinal cord. While the majority of these studies focus on the long-term presence of clinical symptoms, neuropsychological degeneration, and cognitive dysfunction; deficits in motor and postural control are also commonly reported throughout the literature in association with concussion. Interestingly, motor related symptoms typically manifest earliest in individuals who suffer chronic TBI, but such prevalence has not been thoroughly investigated in relation to long term effects of multiple concussions.
Acute impairments in static postural control are speculated to be the result of a “sensory interaction problem” that commonly occurs when concussed individuals are unable to properly integrate sensory information correctly from the visual, vestibular, and somatosensory systems. The rapid deceleration of the head that occurs during a concussive impact produces strong shearing forces that can disrupt the axon, thus affecting its ability to transfer necessary information to the areas of the brain responsible for static balance. It has also been suggested that the imbalance of glucose due to the neurometabolic cascade during the early acute phase following concussion may partially explain the presence of initial postural control deficits, but the severity and duration of this neural deficit is still not completely understood. Multiple investigations have reported that acute deficits in static postural control tend to be most prevalent during the first three days post-concussive injury. Using commonly accepted measures of static postural control such as the Sensory Organization Test (SOT) and Balance Error Scoring System, static postural control typically returns to baseline within 3–5 days post-injury; often prior to cognitive and neuropsychological testing and self-report symptom resolution. Unlike static balance, dynamic postural control requires integration of several sensory and motor pathways by the central nervous system in order to coordinate posture and intentional movement required for locomotion. Research measuring dynamic postural control during gait has revealed postural stability deficits during dual-task gait as far as 1 month post-injury, well beyond symptom resolution. Several studies have reported that individuals who suffer concussion might adopt a conservative gait strategy, during the first 30 days post-injury, as compensation for decreased dynamic postural control, and this is accomplished by adopting a slower gait velocity, smaller stride length and smaller medio-lateral sway of center of mass (COM). While decreased gait velocity has been used to identify postural control impairment,
not all populations with postural control deficit will display significantly decreased gait velocity.\textsuperscript{24} Therefore, analysis of the transition from static to dynamic postural control following concussion has been proposed to be more sensitive for identifying postural control deficit than the previously investigated static balance testing and dynamic steady-gait locomotion analysis.\textsuperscript{24}

Gait initiation (GI) is a functional task that represents a transition from a stable static stance to dynamic locomotion, thus challenging the systems responsible for postural control as the COM transitions from a large to small base of support.\textsuperscript{30} During the initiation of gait, several preparatory actions take place known as anticipatory postural adjustments (APAs).\textsuperscript{31} Likely controlled by the premotor cortex, these initial APAs occur prior to any voluntary movement forward and largely determine initial step limb selection, step length and gait velocity.\textsuperscript{31} Prior to heel lift of the initial stepping limb, movement initiation (MI) actually begins as center of pressure (COP) and COM decouple by inhibition of the gastrocnemius/soleus complex followed by activation of the tibialis anterior and gluteus medius, thus accelerating COM towards the initial stance foot.\textsuperscript{32} The COP and COM during quiet standing are typically centered equally between the two feet, just anterior to the medial malleolus.\textsuperscript{37} The separation of COP-COM during GI is a valid tool in the analysis of postural control.\textsuperscript{24} When separation of COP-COM peaks during GI, the moment arm for the ground reaction forces is greatest, indicating an individual’s tolerance for dynamic unsteadiness.\textsuperscript{24,34} Studying the COP-COM interaction during dynamic locomotion activities such as GI may provide better insight into postural control than by looking at either variable individually.\textsuperscript{24} Further, many recent studies have suggested that analysis of COP-COM interaction during GI is a sensitive indicator of dynamic postural control in a wide range including Parkinson’s Disease, stroke, chronic ankle instability, aging, and
amputees.\textsuperscript{24,30-40} However, no studies were found that focus on potential postural control deficit in GI in individuals following cumulative concussion.

Research over the past decade suggests that individuals who suffer multiple concussions are at higher risk for developing long-term cognitive and neuropsychological deficits later in life, and motor related symptoms tend to appear earliest in individuals with chronic TBI.\textsuperscript{22} While postural control deficits immediately following concussion are frequently analyzed, few studies have been conducted looking into the presence of possible long-term postural control deficits following multiple concussions. Therefore, the purpose of this study was to examine the differences in dynamic postural control during GI between individuals with a history of 3 or more concussions and athletes with no prior history of concussion. We hypothesized that athletes with a concussion history and athletes without a concussion history will display differences in dynamic postural control, with cumulative concussion history predicting reduced COP displacement during S1, reduced COP-COM separation, and decreased spatiotemporal measurements compared to controls.
CHAPTER 2
METHODS

Participants

Twenty Division I athletes from a large Southeastern university participated in this investigation. (Appendix C; Table 1) The participants were divided into two groups based on concussion history. The concussion (CONC) group consisted of 10 current or former varsity intercollegiate student–athletes who have a previous history of three or more concussions. The CONC group consisted of 4 football, 3 women’s soccer, 1 baseball and 2 softball athletes. Participants were excluded from participating in the CONC group and from this study if they reported a history of chronic ankle instability, or self-reported a concussion or lower extremity injury in the previous 2 months. The control (CTRL) group consisted of 10 varsity intercollegiate student–athletes without a history of concussion who were matched to the participants in the CONC group based on sport, height ($\pm$ 5 cm), and gender. CTRL participants were also excluded from participation if they reported any prior history of chronic ankle instability or lower extremity injury in the previous 2 months. Participants were excluded from participation in either group if they reported a history of vestibular, metabolic or other neurological pathology that would otherwise negatively affect postural control. All subjects provided written informed consent prior to participation as approved by the university institutional review board.

Instruments

Participants reported to the biomechanics laboratory on the day of testing and performed GI trials along a 7-meter walkway surrounded by an 8-camera motion capture system (Vicon, Lake Forest, CA). Ground reaction forces were collected using three force platforms (Model
OR-6, AMTI, Watertown, MA) mounted flush with the surface of the walkway and oriented so that the laboratory coordinate system coincides with the left posterior corner of forceplate 3. (Appendix C; Figure 2) Forces and moments from the force platforms were sampled at 1,000 Hz and were subsequently used to calculate COP using Vicon Nexus software (Version 1.6, Vicon, Lake Forest, CA), while kinematic data was collected at 100 Hz through the 8-camera motion capture system. A 5-point MX calibration wand (Vicon, Lake Forest, CA) was used for 3-dimensional reconstruction. The kinematic cameras and force platform recordings were synchronized using the Vicon Nexus software (version 1.6).

Participants were barefoot and fitted with dark tight-fitting shirts and shorts during the testing session. Thirty-nine passive retroreflective markers were placed over landmarks according to the Plug-In-Gait marker system in order to construct a model to calculate the location of whole-body COM. The Plug-In-Gait system was composed of the placement of bilateral markers on the 2nd MTP head, heel, ankle, shank, knee, thigh, anterior superior iliac crest, posterior superior iliac crest, shoulder, upper arm, elbow, forearm, distal radius and ulna at the wrist, 2nd MCP, forehead, and posterior head. Single markers are placed on the jugular notch, inferior sternum, C7, T10, and right scapula. Subjects completed the unstable stances of the Balance Error Scoring System on a 45 cm2 x 13 cm thick Airex Pad (Alcan Airex AG, Sins, Switzerland).

Procedures

Participants completed a brief medical history questionnaire to confirm they met the inclusion criteria prior to participation in the study. (Appendix C; Figure 3) Participants began each trial of GI standing quietly in a relaxed position with the initial foot positioning self-selected on two separate force platforms and marked to maintain consistency. Following a
verbal cue, the participant initiated gait in the forward direction with the preferred leg and continued down the 7-meter walkway until they reached a visual landmark located approximately 2 meters past the end of the walkway. The participant then returned to the starting position and completed the remaining trials in succession following the same protocol. Participants were allowed 1 or 2 practice trials of gait initiation to ensure comfort with the tasks before beginning 5 trials for data collection.30

Subjects in both groups also completed the Balance Error Scoring System (BESS), Standardized Assessment of Concussion (SAC) and a 22 symptom graded symptoms checklist (GSC). The BESS test measures an individual’s ability to maintain balance for 20 seconds in 3 different positions on firm stable and foam unstable surfaces.42 The SAC test is a neurocognitive screening test that measures orientation, immediate memory, concentration, and delayed recall with a maximum score of 30.43 Finally, the GSC consisted of a 22-symptom subjective survey that requires individuals to subjectively report severity of concussion symptoms on a 0-6 point likert scale.44

Data Analysis

MI was identified as the first change (mean ± 2 SD’s) in the mean vertical ground reaction forces (VGRF) between the initial two force platforms during the frames of quiet standing.42 GI was divided into 3 segments (S1, S2, & S3) and 4 landmarks were identified (Appendix C; Figure 1).33 Landmark 1 was identified as the point when COP is the most lateral and posterior in the direction of the initial swing limb at the end of S1 and marks the beginning of the unloading phase as the heel of the initial swing limb lifts.33 Landmark 2 marks the end of S2 and was identified as the point when COP shifts from lateral to anterior motion when swing leg toe-off occurs.33 Landmark 3 was identified as the end of S3, when toe off occurs on the
initial stance limb. The final landmark, heel strike (HS⁻¹), was identified as the last sample prior to the end of single leg stance where peak separation of COP-COM occurs. Spatiotemporal measures of step length, velocity, and width were determined by analysis of displacement and velocity of the swing limb heel marker between MI and HS⁻¹. Step length (SL) was determined by calculating the anterior displacement of the swing limb heel marker from MI to HS⁻¹. Step velocity (SV) was calculated by dividing the step length by the step duration. Stance width (SW) was determined by measuring the distance between the left and right heel marker at MI.

Statistical Analysis

Descriptive statistics were calculated for height, weight, age, and concussion history for both groups. The mean of the five trials for each individual for each dependent variable were analyzed. Three separate multivariate analysis of variance (MANOVA) were conducted to test each dependent variables of interest: 1) Displacement of COP during S1, S2, and S3 of GI; 2) COP-COM separation at MI, landmark 1, landmark 2, and at HS⁻¹; and, 3) Spatiotemporal variables including step length and step velocity. Independent t-tests were conducted for all demographics, step width, BESS, SAC, and GSC. The alpha level was set at p<.05 á-priori. All statistical testing was completed using SPSS 17.0 for Windows (Chicago, Illinois).
CHAPTER 3
RESULTS

All participants were able to complete all trials of GI without incident or difficulty. There were no significant differences between groups for any demographic characteristics, with exception of concussion history, where the CONC group had significantly more concussions compared to the CTRL group \((t = 11.39, P < 0.001)\). There were no significant differences between groups for any demographics or stance width \((t = 0.860, P = 0.757)\), therefore none of the dependent variables were normalized to subject anthropometric characteristics. Further, there were no significant differences between groups for BESS \((t = 0.634, P = 0.534)\), SAC \((t = 0.831, P = 0.421)\) or GSC \((t = 0.708, P = 0.489)\). (Appendix C; Figure 4)

There were no significant main effects for group for COP displacement during S1, S2, or S3 of GI \((F = 0.809, P = 0.581, \text{Power}=0.217)\). (Appendix C; Table 2) Specifically, in the area of greatest interest, exploratory analysis found no differences between groups for COP displacement during S1 in either the A/P \((5.24 \pm 1.14\text{cm} \text{ and } 4.88 \pm 1.10\text{cm respectively, } P = 0.477)\) or M/L \((6.01 \pm 1.07\text{cm} \text{ and } 6.54 \pm 0.97\text{cm respectively, } P = 0.259)\). (Appendix C; Figure 5) Further, exploratory analysis did not identify any between group differences for either A/P or M/L COP displacement during S2 or S3.

There were no significant effects for group for COP-COM separation during the GI task \((F = 0.062, P = 0.992, \text{Power}=0.059)\). (Appendix C; Figure 6) The peak COP-COM separation occurred at the end of the terminal swing phase of gait and exploratory analysis revealed no differences between groups at this time \((30.74 \pm 3.16\text{cm} \text{ and } 30.19 \pm 2.67\text{cm respectively, } P = 0.676)\).
There was also no significant main effect for group for spatiotemporal measures of the initial step (F=0.819, P=0.458, Power=0.167). (Appendix C; Figure 7) Exploratory analysis also revealed no differences between groups for either initial step length (0.64 ± 0.04m and 0.61 ± 0.06m respectively, P=0.206) or initial step velocity (0.67 ± 0.06m/s and 0.65 ± 0.04m/s respectively, P=0.599).
CHAPTER 4
DISCUSSION

The purpose of this investigation was to determine if a history of three or more concussions in collegiate athletes identifies evidence of early impairments in postural control during the transitional motor task, gait initiation. The principle finding of this study was that there were no significant differences in postural control during GI between athletes who have never had a concussion and athletes who self-reported a previous history of 3 or more concussions. Impaired postural control and motor deficits are commonly reported acute post-concussion symptoms; however, few studies have investigated the potential long-term effects of concussions on postural and motor control. Rabidi suggested that motor related symptoms are generally the earliest to manifest in individuals who suffer chronic TBI. Furthermore, a recent investigation reported impaired motor cortex physiology resulting, potentially, from cumulative concussions sustained during collegiate athletes; however, our findings suggest that motor and postural control deficits may not be present during GI early in healthy collegiate athletes’ lives.

GI is a challenging task to the postural control systems because the body transitions from a relatively stable static position to unstable dynamic gait. Previous investigations have reported individuals presented decreased postural control during GI in populations including chronic ankle instability, aging, amputees, PD, and stroke. As the human body ages and encounters disease, the systems responsible for controlling balance are gradually impaired. In healthy individuals, the momentum necessary for forward initiation of gait is generated during the anticipatory postural adjustment, or S1 phase, of GI. Populations with postural control deficit may display decreased posterior displacement of S1, thus potentially decreasing the step length and velocity of the initial step due to the reduced ability to generate forward momentum.
Therefore, we hypothesized that athletes with a history of multiple concussions would demonstrate a decreased COP displacement during S1. However, both groups in the present study displayed “normal” posterior displacement of COP during S1 (4.88 – 5.24 cm), agreeing with Halliday who reported a mean COP A/P displacement of 4.70 ± 1.5 cm during S1 of GI in young healthy individuals.\(^{38}\) We further hypothesized that individuals with a history of multiple concussions would display decreased separation of COP-COM during GI, a conservative trend reported in PD patients who display the need to preserve stability by decreasing movement of COM outside of their base of support.\(^{33}\) Interestingly, participants in this study displayed maximum separations of COP-COM between 30 – 31 cm, on average, which is somewhat below previous reports for healthy young adults (36 cm).\(^{40}\) Parker reported one potential explanation for the neuropsychological and cognitive differences between athletes and non-athletes, stating that participation in contact sports subjects athletes to repetitive sub-concussive blows that could produce motor impairments when compared to non-athlete control subjects.\(^{25}\) Finally, the spatiotemporal measures observed in this study were not consistent with the conservative strategies we anticipated to see in participants with a previous history of concussions and did not differ from matched controls in both step length and velocity. Further, participants in this study demonstrated initial step lengths between 0.61 - 0.64 m, on average, which is consistent with those reported by Naugle for healthy young adults.\(^{46}\)

The results of this investigation did not find any significant differences in postural control amongst individuals with multiple self-reported concussions. These findings conflict with several recent investigations which have suggested potential motor impairments in individuals with multiple concussions. Specifically, De Beaumont reported that collegiate athletes displayed long-term primary motor cortex dysfunction more than 9 months after suffering a concussion and
that these deficits were more pronounced in individuals with a history of multiple concussions suggesting that this deficit was also cumulative.\textsuperscript{15} While our findings do not necessarily support those of De Beaumont, one potential explanation of this could be that the actual task of GI is not a function of the primary motor cortex, but rather from the premotor cortex. Chang reported, using a GI testing paradigm in stroke patients, that the premotor cortex is likely responsible for cued movement initiation preparation and limb selection.\textsuperscript{31} Specifically, the APA’s represented as the posterior and lateral displacement of the COP are controlled, in part, by the premotor cortex. Interestingly, Chang also reported that individuals with primary motor cortex lesions were still able to produce appropriate APA’s and suggested that the severity of primary motor cortex lesions was not the primary reason for impaired motor performance. Therefore, individuals with motor dysfunction may still be able to present with “normal” postural control during GI due to postural adjustments being controlled by the premotor cortex rather than the primary motor cortex.

Many investigations have been conducted to determine whether cumulative concussions increase the risk of further injury or long-term physical impairments, or if the effects of concussion merely result in a transient loss of function.\textsuperscript{13-21} Recent evidence has suggested that athletes who suffer multiple concussions may be at an increased risk of developing MCI, early onset of Alzheimer’s Disease, motor cortex dysfunction, CTE, and ALS.\textsuperscript{13-21} While, our analysis of postural control during this study did not identify pronounced cumulative dysfunction in the short-term, another potential explanation for this could be the compensation of the premotor cortex through recruitment of other brain regions. Chen, utilizing an fMRI testing paradigm to assess alterations in brain activity following concussion, suggested that individuals may use compensatory mechanisms through alternative cognitive resources to complete difficult tasks
following injury in order accommodate for deficit. Therefore, it is possible that the participants in our study were able to present with apparent normal postural control during GI due to accommodation by recruitment of functional reserves from other regions of the brain. By increasing the difficulty of the task at hand, such as including a secondary cognitive task to complete while initiating gait that may further stress or overload the functional reserves, future studies could perhaps be more successful at identifying deficits in postural control that accommodations may mask.

Limitations

We attempted to control for major limitations, however some factors were present that may have affected the validity of this investigation. One limitation was that the inclusion criteria required the reliance on the subjective reporting of diagnosed concussions from the participants in both groups. While the recruitment of subjects for both groups was aided by the athletic training staff with a working knowledge of the concussion history of each student-athlete they oversee, it is entirely possible that many of the athletes in either group may have misreported the number of clinically diagnosed concussions. Secondly, the inherently small subject sample in this study limits the power of our findings due to strict inclusion criteria, which limited the number of potential subjects. Lastly, availability of matched controls was also a limitation, as there was no way to recruit ideal control subjects until after the CONC group had been recruited.

Conclusion

This is the first study, to our knowledge, to biomechanically assess postural control during GI of athletes with a history of multiple concussions outside of the acute or subacute recovery phases. The results of this study suggest that athletes with a self-reported history of 3 or more concussions do not display any significant postural control deficits during GI compared
to age and sport matched controls. Previous investigators have suggested that multiple concussions may create potential persistent and long-term neurological and cognitive impairments in athletes; however, potential association between long-term postural control deficit and cumulative concussion history remains unclear. Future research on postural control in athletes with multiple concussions should focus on increasing the difficulty of the task, perhaps by adding secondary tasks to provide greater challenge to the systems that control postural adjustments.
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APPENDIX A

Research Hypothesis

- H₀¹ = Athletes with a concussion history will have no differences in separation of COP-COM compared to athletes without concussion history.
- H₁⁻ = Athletes with concussion history will have reduced separation of COP-COM than athletes without concussion history.
- H₀₂ = Athletes with concussion history will have no differences in displacement of COP during gait initiation compared to athletes without concussion history.
- H₂⁻ = Athletes with concussion history will have reduced COP displacement in the S1 phase of gait initiation than athletes without concussion history.
- H₀₃ = Athletes with concussion history have no differences in spatiotemporal measurements compared to athletes without concussion history.
- H₃⁻ = Athletes with concussion history will have decreased spatiotemporal measurements compared to athletes without concussion history.

Delimitations

This study included only Division-I collegiate student athletes from a single institution. Of these athletes, only athletes with a history of three or more concussions (CONC) and athletes with no previous history of concussion (CTRL) were included for participation.

Assumptions

The first assumption of this study was that all tests and measurements had proper set-up and calibration necessary for accurate data collection. Secondly, we assumed that participants performed these tests to their maximum ability. Another assumption for this study was that the tests that were administered would show differences between normal and abnormal postural
control systems. Lastly, it must be assumed that the CRTL group was matched properly to CONC group.
APPENDIX B

REVIEW OF LITERATURE

Historical Background

Concussion has been defined as a complex pathophysiological process, induced by traumatic biomechanical forces, which affects the brain.\textsuperscript{1} Even though concussion is well recognized amongst clinicians, the basis of the pathophysiological process is still debated today. During the 10\textsuperscript{th} century, a Persian physician named Rhazes made the first modern definition of concussion. Rhazes described concussion as an abnormal physiological state, rather than a severe brain injury. During the 16\textsuperscript{th} century, Jean Louis Petit suggested that concussion could be due to a vascular issue. Of the theories developed since the 16\textsuperscript{th} century, the only hypothesis that has yet to be disproven is that concussions are the result of complex movement of the brain, resulting in a pathophysiological process.\textsuperscript{2} While the debate on the complexity of concussion surges on today, studies in the recent years have lead to a much greater understanding of the processes that affect normal brain function.

Neurometabolic Cascade

In 2001, Giza\textsuperscript{3} described the pathophysiological process of concussion in the form of a neurometabolic cascade that results following a concussive blow to the brain. Initially, the biomechanical force to the brain causes neuronal depolarization. The depolarization of the neurons releases excitatory amino acids, such as glutamate, which bind to NMDA. This results in a massive efflux of potassium (\(K^+\)) from the neuron cell, possibly causing loss of consciousness. To offset the efflux of \(K^+\), sodium-potassium pumps are increased to force \(K^+\) back into the cell, resulting in ATP use. Hyperglycolysis, an anaerobic pathway that produces ATP, is then initiated to produce ATP to fuel the Na/K pumps. Hyperglycolysis creates lactate
accumulation, which causes a calcium (Ca$^{++}$) influx of the neuron. Such an influx causes the pH of the mitochondria to change dramatically which then decreases the mitochondrion’s ability to produce ATP. With a global shortage of ATP, the cells give up and structural cell death occurs, resulting in axonal pathology. This cascade of events following concussion creates a period of elevated risk for further injury. While the cascade was developed using animal subjects, it is a testament to the advances that have been made in concussion research in the past 10 years. Yet, unless athletic trainers better understand and utilize this explanation, the number of recurrent concussions that occur will not decrease.

Epidemiology of Concussion

An estimated 1.6 to 3.8 million concussions occur each year in athletics. In fact, sports and recreation are amongst the leading causes of traumatic brain injury. Several studies have been done to outline concussion prevalence and incidence in high school, collegiate and professional sports. The average incidence rate of concussion is between 5 – 8%. In 2007, Shankar et al. found that 11% of all injuries in football were concussion, translating to one of every 19 high school football players obtaining a concussion per year. Seven years earlier, Guskiewicz et al. found concussion that 5.1% of high school and collegiate football players encountered concussion during a single season. Both Shankar and Guskiewicz suggest similar incidence rates of concussion in football players, though an extended period of time separates their data collection. Interestingly enough, both Powell and Covassin et al. found similar concussion rates (5.5% and 5.9%, respectively), however these studies collected data for many sports, rather than just football. Gessel et al. suggested that while 8.9% of all high school injuries were concussion, there may be a higher rate of concussion in collegiate athletics, however more injuries that are sustained in high school athletics are concussions. One rather
liberal study found that over 70% of football players had experienced at least one concussion during a single season.\textsuperscript{10} However, this study used a very open definition of concussion and thus displays many significant limitations.

Several other factors play a role in the incidence of concussion in sport. Some of these factors include scenario (practice vs. competition) and gender (males vs. females). Shankar et al. found that of all athletic injuries reported as concussions, 12\% occurred during competition and 9.2\% occurred during football practices.\textsuperscript{5} Covassin et al. discovered a concussion incidence rate of 4.2\% at practice and 7.8\% at games.\textsuperscript{8} Guskeiwicz et al reported that athletes are 8.15 times more likely to suffer a concussion during a game rather than in a practice.\textsuperscript{11} This clearly shows that more concussions occur during competition than in practice. Gender types have also been a debated topic in concussion research. Gessel et al. found the concussion incidence rate to be 9.4\% in males and 15.1\% in females, while Covassin et al. found similar results with rates of 6.4\% for males and 9.5\% of females.\textsuperscript{8,9} These differences may be explained by biomechanical and structural differences between males and females and the demands of their sport preference.\textsuperscript{9} It is clear that concussions are prevalent in athletics at any level, but the debate on how to identify, assess, and manage them continues even today.

**Concussion Assessment**

Immediately following a concussive incident, an athlete may elicit a wide range of signs and symptoms, neurocognitive deficit, and postural instability. There are many factors contributing to the occurrence of these concussion effects, and each concussion must be assessed and managed on an individual basis. We are all quite aware of the dangers that may result with improper management, thus several concussion-grading scales have been developed in order to aid athletic trainers and clinicians in diagnosis and management.\textsuperscript{12} However, there is no
universal agreement on grading. In 2001, Robert Cantu described the many different grading scales of concussion in detail. The majority of the current grading scales focus depth and duration of loss of consciousness, amnesia, and post-concussive symptoms as major determinants of concussion severity. 

The American Academy of Neurology (AAN) and Colorado Medical Society (CMS) grading systems place emphasis on loss of consciousness as the main determinant of concussion severity. Regardless of duration of symptoms and amnesia, a concussion can only be classified as a grade III concussion if there is the presence of loss of consciousness. Several studies have been done to determine the outcomes of concussion to determine if loss of consciousness is determinant of severity. Guskiewicz et al. found that only 6.3% of concussions result in loss of consciousness. Results from the same study indicated that over 24% of athletes who reported concussion experienced amnesia. McCrea et al. found 6.4% of concussions resulting in loss of consciousness and 19.1% of concussions result in some form of amnesia. Erlanger et al. found that loss of consciousness was not associated with the number of symptoms nor the overall duration of symptoms. While this does not justify removing loss of consciousness from grading scales, it does raise questions as to why it is so frequently utilized in severity assignment in concussion grading scales. Not to downplay the importance of loss of consciousness clinically, however it has been argued that a concussion that produces post-concussive symptoms lasting months without the presence of loss of consciousness should not be graded less than a concussion resulting in loss of consciousness that is resolved in a few days.

Utilizing such understanding, the Cantu-revised guidelines were recommended by Erlanger et al. to be the most appropriate grading scale. According to the Cantu-revised guidelines, a mild concussion may be classified as grade 1 if post-concussive symptoms and
amnesia last less than 30 minutes. A concussion may be classified as grade 2 (moderate) if loss of consciousness is present but less than 1 minute in duration, and amnesia and post-concussive symptoms last longer than 30 minutes but less than 24 hours. A concussion may be classified as grade 3 (severe) if loss of consciousness lasts longer than 1 minute, amnesia lasting longer than 24 hours, and symptoms lasting longer than 7 days. While determining the presence of loss of consciousness is relatively simple, the ability to identify amnesia and the post-concussive symptoms requires much more attention to detail.

Athletes who suffer concussion will encounter many different symptoms, depending on the location and severity of the concussive injury. However, several studies have reported common symptoms that may be overlooked if not associated with some form of head injury. Headache is the most commonly reported symptom according to several studies. Other common signs and symptoms also include dizziness, confusion, fatigue, blurred vision, and nausea. Few studies have been done to suggest the presence of a single symptom that will dictate the outcome of the injury, and this remains a hot topic for debate. Iverson et al. focused his study on whether presence of “fogginess” would provide worse outcomes in symptom duration and he found that athletes who present with fogginess one week after suffering concussion might have adverse effects.

Guskewicz et al. suggests that when gauging the progress of symptoms from injury event to return-to-play, a graded symptoms checklist may be a useful tool. These checklists list between 17-22 symptoms commonly associated with concussion that the athlete subjectively grades on a likert scale. It is also suggested that baselines of athletes be taken before the start of the academic season to ensure proper comparison between pre and post concussion. Aside
from physical symptoms, much research has been done describing the neurocognitive deficits and disorientation that also occur following concussion.

In 1995, Maddocks et al. explained a new outlook on the assessment of orientation in concussed athletes. Many athletes will display disorientation following concussion and until Maddocks et al. introduced several new questions (Now coined the “Maddocks questions”), assessment was limited to only to past orientation questions rather than recent ones. By introducing questions that ask for athletes to recall recently learned items, Maddocks et al. was able to establish better methods in identification of disorientation following concussion and suggest the presence of retrograde amnesia. Rather than ask only questions requiring the athlete to state their name, date of birth, and age, the Maddocks questions asked athletes to recall where they were playing, what quarter it was, who scored the last goal, and who they were playing. Maddocks et al. found that these questions were more sensitive to identifying cognitive disorientation than previously reported.16

Expanding on this principal six years later, Cantu explains the phenomenon of amnesia and how it can be classified as either retrograde or anterograde. Retrograde amnesia refers to a decreased ability to recall events that have occurred immediately before a concussive injury. These can be tested using the “Maddocks” type questions that test general orientation to their surrounding environment. Anterograde amnesia refers to a decreased ability to form a new memory following a concussive injury. Athletes with anterograde amnesia may also present with a decreased attention span and inaccurate perception. By asking athletes to perform immediate and delayed recall of words, repeat numbers forwards and reverse, and state the months of the year in reverse, clinicians may identify the presence of anterograde amnesia.13
Using such principles, McCrea et al. developed the Standard Assessment of Concussion (SAC).\textsuperscript{18} The SAC test is a mental screening test that may be administered anywhere a concussion may occur. The SAC test has been proven reliable and valid for identifying mental status change following concussion occurrence, even without the presence of loss of consciousness or posttraumatic amnesia.\textsuperscript{18,19} The SAC test takes approximately 5 minutes to complete and measures four domains including orientation, immediate memory, concentration, and delayed recall. An athlete can score a maximum of 30 points on the test. There are also alternate forms of the test to decrease the possibility of a practice effect when administration is repeated several times.\textsuperscript{18} As recommended with the graded symptom checklists, the SAC test is best utilized immediately following a concussion when a baseline measurement is present for comparison.\textsuperscript{12} The majority of neurocognitive deficits return to baseline within 48 hours following concussion as demonstrated in a study utilizing SAC\textsuperscript{19}, however other effects of concussion may remain present for much longer durations.

Postural stability and balance are other factors often negatively affected by concussion. Several tests have been developed to aid in the assessment and identification of postural instability following concussions. Perhaps the most commonly utilized means for assessment of postural stability following concussion is the Balance Error Scoring System (BESS). Originally described by Reimann et al., the BESS test measures an athlete’s ability to maintain static postural stability through 6 different positions for 20 seconds. The positions include a dual leg stance, tandem leg stance, and a single leg stance, each on both a firm and unstable airex foam surface. Once the patient is in position, they must close their eyes with their hands on their iliac crests. Errors are recorded if the patient lifts their hands of their iliac crests, opens their eyes, falls out of position, flexes or abducts the hip more than 30 degrees, lifts the forefoot or heel, and
if the patient is out of position for more than 5 seconds.\textsuperscript{20} The BESS test is a recommended tool for athletic trainers in the assessment and management of concussions.\textsuperscript{12} However, while the BESS test has shown reliability and validity\textsuperscript{20}, it also suffers some severe limitations.

Onate et al. explained one of such limitations, which suggest that the environment that BESS is tested in will affect the error totals. For example, if an athlete is baseline tested in a controlled athletic training room environment, then their post-injury evaluation should be done in a similar environment. The study showed that unconcussed athletes scored lower on repeated trials of BESS testing when the environment changed from a controlled locker room to an uncontrolled sideline or field. The results of this study recommended that when using BESS testing, special modifications should be made to allow testing both baseline and follow-up in a controlled environment to maximize efficacy.\textsuperscript{21}

In a study focusing on exercises affects on postural control, Fox et al. suggested a significant limitation to the BESS test. Athletes completed a total of 14 trials of BESS and yielded extremely low total errors. They suggested that postural control seems to return to baseline between 8-13 minutes following a exhausting exercise, however they also point out the possibility of a learning effect with the BESS test.\textsuperscript{22} Valovich et al. found that in high school students who were exposed to repeated administration of the BESS and SAC tests, the BESS test elicited a practice effect. However after 3 weeks of disuse, the skills necessary to repeat the earlier success of BESS testing had faded. This study also suggests that while it is quite apparent that there is a practice effect with BESS, there is not an apparent practice effect related to SAC testing when utilizing different forms.\textsuperscript{23}

Davis et al. provided an in depth literature review of all the current management, diagnostic tools, and imaging techniques currently utilized in the management of sport-related
concussion. Unfortunately, the majority of the tools described are primarily used as research tools, and thus, are not clinically available to be incorporated in concussion management programs. However, functional MRI does offer insights into pathophysiological and functional deficits following concussions. Relevant correlations have been made between fMRI abnormalities and scores from cognitive testing. However, the price of fMRI may be the only factor affecting its involvement in further research. Not every high school student can afford to have a functional MRI done for each concussion. In fact, Davis et al. suggest that while there are many diagnostic tools that are currently being utilized in research to study the pathophysiological effects of concussion, many of them are not clinically applicable. Furthermore, clinical balance testing is the only tool that has not remained experimental. 24

The NATA position statement on athletic concussion provides recommendations on utilizations of the aforementioned concussion assessment tools. The recommendations state that proper concussion assessment is made using multiple tools in combination for best results and outcomes. Regardless of the scale used, a concussion should be graded for consistency and each concussion graded individually. The position statement also recommends the use of baseline testing with each of the assessment tools utilized for comparison rather than comparing to normative values. The NATA position statement also warns athletic trainers about athletes with learning disabilities or previous history of concussion. It states that athletes who have a previous history of concussion may perform lower on baseline scores than normal.12 This suggestion may be true, as a gathering amount of research suggests that athletes who have sustained a concussion in the past have an increased risk for developing further concussions.6 However, unless athletes report their concussions, it is unclear how many concussions an athlete may have had in their lifetime.
Concussion Reporting and Awareness

Reporting of concussions is an issue amongst athletic trainers, athletes and coaches. McCrea et al. stated that 15.3% of high school football players reported sustaining a concussion during a single football season. Of the concussions that they sustained, only 47.3% reported the event to medical personnel or coaches. This suggests that over half of the concussions that actually occur are not reported. Of those who did not report their concussions, 66.4% stated that the reason they did not report their concussions because they felt it wasn’t important or serious enough to warrant reporting. One-third of athletes who sustained a concussion did not recognized their symptoms were related to concussions. Sefton et al. reported that 73% of athletes who did not report their concussion, did not do so because they didn’t feel it was important enough. Kaut et al. reported that 19.5% of incoming freshman in college athletics reported a history of at least one concussion. Of those surveyed, only 43.7% indicated that they had knowledge in the area of head injury.

Sye et al. surveyed 477 high school rugby players and found that 27% of those surveyed felt as though someone with a suspected concussion should return to play in an important game, regardless of symptom status. Valovich et al. suggests that over ¼ of the athletic population has a history of concussion before reaching their senior year of high school. Regardless of the reasoning, concussions are underreported in sports at all levels. Interestingly enough, if the reporting rates of concussion increase, so should the concussion injury rate. As long as athletes continue to display such ignorance and misunderstanding about the consequences related to concussion, clinicians will miss half the concussions that occur. Many efforts have been made to increase awareness of concussion in athletic populations.
In 2005, the Center for Disease Control (CDC) released a package entitled “Heads Up”, which was developed to promote concussion awareness to coaches, athletes, and athletic trainers. Several studies have aimed at determining the effectiveness of such concussion awareness programs. Valovich et al. stated that youth coaches who participated in coaching education programs were more likely to recognize signs and symptoms of concussions. However, the availability of such programs in the rural and less developed areas obviously creates an issue for some places. Their study also suggested that 32% of coaches believed an athlete who suffered from a grade I concussion could return to play. While it is unlikely that coaches can differentiate between concussion grading scales, this study concludes that coaching education may increase their ability to recognize some common signs and symptoms, thus reducing the risky return to play decisions being made. Guilmette et al. also looked into coaches awareness, but in high school football. They found that only 31% of coaches in the New England region of the United States who responded to the survey had received the “Heads Up” concussion awareness kit, however 80% claimed receiving concussion information from coaching associations, conferences and the media.

Another major concern is the parent’s knowledge and understanding of concussion. Sullivan et al. surveyed 200 parents of youth rugby players. Though this group only consisted of parents from a New Zealand youth rugby league, they reported that those who participated in the survey displayed a good lay knowledge of concussive injury. Sullivan et al. concluded that parents contribute significantly in the identification and adherence to management programs of sport related concussion. Concussion awareness programs may not be utilized fully, however it is apparent that those who are taking advantage of such resources are providing more support to the difficult task of identifying concussions. With each concussion that occurs and goes
unnoticed, the risk of repeated concussion and the cumulative effects that result from repeated concussions also increases.

Cumulative Effects of Concussion

In 2003, Guskiewicz et al. completed the NCAA Concussion Study, which focused on the cumulative effects of repeated or recurrent concussions. Their findings suggest that after an athlete suffers a concussion, there is a 7 to 10 day period of increased susceptibility for further concussion. Their results also suggest that athletes who have a history of concussion are likely to have future concussions. In fact, their study found that 1 of every 15 players who suffers a concussion will encounter a concussion during the same season, the majority of which occurring within the 7 to 10 day period of increased susceptibility. Athletes with a history of 3 concussions have a 3-fold greater risk of future concussions. Lastly, this study found that athletes with a history of previous concussion have been associated with longer recovery following later concussions.\textsuperscript{11} The theory of cumulative effects of concussion has developed a lot of press lately in the professional sports world, however Guskiewicz et al. were not the only group who has looked into the possibility of cumulative effects following multiple concussions.

Zemper et al. reported that athletes who had a previous history of concussion were 5.8 times more likely to sustain a repeated concussion. These results suggest that 1 of every 6 players with a previous concussion history will suffer a repeat concussion during a season. However, only 1 of every 35 athletes without a history of previous concussion will suffer a concussion during a single season.\textsuperscript{33} Covassin et al. completed a repeated measures design looking at ImPACT testing in athletes with a concussion history. They concluded that athletes with multiple concussions took longer to return to baseline, suggesting that athletes with a history of multiple concussions recover slower from these repeat concussions.\textsuperscript{34}
Athletes with a previous history of concussion may also have longer duration of symptoms following concussion. Bruce et al. found that athletes with a previous history of concussion reported more symptoms than athletes without a concussion history at baseline. They also reported that one week following a concussive injury, athletes with a previous history of concussion reported more symptoms than athletes without a concussion history, suggesting that athletes with a previous history of concussion may have longer effects following repeat concussions. Iverson et al. also suggested that athletes with multiple concussions may suffer cumulative effects from concussions. In yet another study that utilized ImPACT for baseline and post-concussion follow-up, they reported that athletes with a previous history of concussion had increased memory problems. They also suggested that athletes with a previous history of concussion might have an increased susceptibility to concussions of greater severity in the future. In other words, athletes may suffer more severe repeat concussions as a result of previous concussive injuries. While athletes who have multiple concussions may take longer to recover, some research suggests that even while asymptomatic, there may still be evidence of brain dysfunction.

**Concussion Recovery**

As a part of the NCAA Concussion Study, McCrea et al. looked into the recovery patterns of athletes following concussions. Of those who suffered a concussion, 91% reported baseline symptom scores seven days after injury. Obviously, there is a major issue in the reporting of concussion symptoms. Athletes may lie about their symptoms to return to participation faster, thus making concussion management all that much more challenging. However, as more research is beginning to suggest, athletes who are “asymptomatic” may not be completely healed anyway. Both Broglio et al. and McClincy et al. suggested that cognitive
deficits were evident using ImPACT, even after athletes who suffered a concussion were no longer reporting symptoms. In a study using EEG wavelet information quality measures, Slobounov et al. found that even when clinical symptoms and neurological deficits were absent 7 days after injury, brain dysfunction was still detected.

In another study by Slobounov et al, all participants were clinically asymptomatic ten days after first and second concussions. However, they found that athletes who suffered second concussions were slower to recover visual-kinesthetic integration using a complex virtual reality postural stability program. They also suggested that while all athletes were asymptomatic, they still presented residual abnormalities that were not detected by neuropsychological testing. Both studies concluded resolution of symptoms may not indicate brain injury resolution.

While the majority of assessment tools are extremely sensitive to detecting deficits during the acute stages following concussions, they are not sensitive enough. Slobounov et al. suggests that integration of computerized balance testing in addition to typical neuropsychological testing will offer further the best outcome of concussion management. While not every athletic trainer has access to such complex technology, the utilization of postural stability assessment as a gauge of concussion has gained much interest.

Postural Stability and Concussion

Postural stability is defined as the ability maintain a desired postural orientation. Immediately following concussion there is a 3 to 5 day period in which athletes appear to suffer postural stability deficit. Guskiewicz et al. describes how this deficit may be a result of a “sensory interaction problem” that occurs when concussed athletes are unable to exchange sensory information correctly from the visual, vestibular, and somatosensory systems. However, the neural deficits causing postural instability are still not completely understood. Guskiewicz
explains that postural instability may be due to the rapid deceleration of the head that occurs during a concussive impact. This biomechanical force is so strong that shearing forces can disrupt the axon, thus affecting its ability to transfer necessary information to the areas of the brain responsible for balance. It has also been suggested that the imbalance of glucose due to the neurometabolic cascade during the initial acute phase following concussion may explain the presence of postural stability deficit.42

Postural stability is maintained by combining information received from the vestibular, visual and somatosensory systems in otherwise healthy individuals. It is possible however, that inhibition of one of these systems may also play a role in postural instability of athletes following a concussion.20,42 In a healthy individual, visual and somatosensory information may provide enough information to adequately maintain postural stability.42 A breakdown in visual systems, or simply closing your eyes in otherwise healthy individuals, would only cause an increase in postural sway.43 However, in a concussed individual, the possible breakdown in transmission of crucial information creates a reliance on the other systems. Guskiewicz et al. describes how concussions may cause damage to peripheral receptors or the brain centers that integrate vestibular information with visual and somatosensory. Any combination of peripheral or central damage can contribute to the presence of postural instability.42,43 Guskiewicz et al. also describe how the presence of decreased attention and concentration following concussive injury may also contribute to postural instability. While their study did suggest that postural instability was seen in the majority of athletes that they observed, they also warn us that concussions never present the same. Different concussions may affect different parts of the brain and thus should be analyzed independently each and every time.42
Several studies have taken new approaches at assessing deficit in postural stability in concussed athletes. Cavanaugh et al. explained the theory of approximate entropy as a method of determining postural stability in athletes following concussion. Approximate entropy (ApEn) is essentially the randomness of one’s center of pressure oscillations while they stand static. ApEn values decrease in athletes following a concussion, thus suggesting less randomness of oscillations of the body’s center of pressure. The main findings of this study suggest that even though previous research shows that postural stability returns to baseline within 3-5 days post-concussion, the ApEn values did not. This phenomenon of decreased randomness of the center of pressure oscillations may be explained by changes in postural control following concussion. Another possible explanation states that a concussed athlete attempts to establish muscular control over postural sway, thus reducing the ApEn values. Though ApEn is not a measure of postural stability, it does provide an alternative measurement that may compliment other postural stability methods. However, because ApEn appears to be more sensitive, Cavanaugh et al. reported that athletes might return to baseline in common postural control methods well before doing so in ApEn.41

Using a variable known as virtual time to contact (VTC), Slobounov et al. discuss how they could estimate postural stability based on a stability boundary. Their findings suggest that VTC measures can detect postural abnormalities that cannot be observed in traditional balance testing, such as BESS testing. They concluded that postural abnormalities exist beyond symptom resolution and that current clinical testing may not detect such abnormalities.44 This is yet another example of the advances in postural stability research outside of the clinical balance standards that are nearly universally accepted. Researchers have not settled with simple static
stability in athletes following concussion. Several studies have looked into the affects of concussion on stability during gait and locomotion.

Gait Stability and Concussion

While static postural control measurements tend to return to baseline rather quickly, some methods of concussion assessment have been more sensitive in their return to pre-concussion level. Parker et al. looked into gait stability following concussion, and explained that speed of information processing and reaction time have shown longer recovery time after an athlete suffers a concussion. Complex tasks such as walking under special conditions appear to fall into this category. Parker et al. hypothesized that tasks such as dual-task walking may require longer recovery time when compared to traditional static balance testing. They reported that athletes who suffered from a concussion walked significantly slower in the first 48 hours following injury. They also found that the stride length was longer in single-task walking. This would suggest that athletes following concussion tend to approach gait more conservatively than those without concussion. While monitoring center of pressure (COP) and center of mass (COM) during gait, they found that athletes who had a concussion displayed less separation of COP and COM displacement. Parker et al. concluded that athletes displayed gait stability deficit up to 4 weeks following concussion and that the period of increased vulnerability may be much longer than earlier discussed.45

Cantena et al. also looked into the possibility of gait instability following concussion. Their research methods required young adults to walk down a hallway and presented them with various obstacles or tasks requiring their attention. They found that following concussion, overall gait velocity was slower and stride length was longer during question/answer and obstacle tasks when compared to controls. They concluded that concussed individuals not only
take a conservative approach in gait following concussion, but that dynamic attention tests were more sensitive to revealing gait stability deficits. They also suggested that attention gait stability testing may provide better assessments following concussion. Recent research has suggested that athletes who suffer concussion will display longer deficit in gait stability than previously believed. This may suggest that the period of increased susceptibility may be longer than previously believed. Much more research is needed in this area to support the theory that long term effects of concussion may exist and the current return to play guidelines may not protect athletes fully.

Long Term Effects of Concussion

Concussions may result in long-term effects that manifest themselves later on in life. Guskiewicz et al. developed a pair of studies in 2005 and 2007, which were both controversial and groundbreaking. Though both studies consisted of some hefty limitations, insight into the possibility that athletes may suffer from long-term effects from concussions they suffered while they were younger suggested that cumulative effects of multiple concussions may not be limited to short term deficits. By surveying retired NFL football players, they were able to make suggestions about concussion recovery that had not yet been hypothesized. Guskiewicz et al. surveyed all living retired members of the NFL players association. The survey included a general health questionnaire (SF-36) that also included information about concussion history and prevalence. They reported that over 60% of those who responded to the survey had at least 1 concussion in the NFL. They also reported that 24% of responding players suffered from at least 3 or more concussions during their career in the NFL. At least 54% of those who had experienced concussion had experienced loss of consciousness during one of those concussions,
and 52% had experienced memory loss or amnesia following one of their concussions.\textsuperscript{47,48} The survey also looked into the respondents self perceived long-term effects of concussion. They reported that over 17% claimed that they suffered from thinking and memory deficit. Also, Guskiewicz et al. reported that 1.3% of those who responded to the survey had been clinically diagnosed with Alzheimer’s disease.\textsuperscript{47}

Depression was another item that was analyzed as a possible long-term effect following concussion. In 2007, the study by Guskiewicz et al. found that over 11% of retired athletes surveyed suffered from clinically diagnosed depression. Also, they reported that over 11% reported feeling sad, stressed or nervous often.\textsuperscript{48} Ultimately, both studies conclude that multiple concussions may create long term effects that manifest themselves later in life. They suggested that a single concussion did not provide any additional risk for development of depression, but found that the more concussions you suffer, the more likely you are display some form of long term effects. Between both studies, they reported that athletes who had three or more concussions were 3 times more likely to be diagnosed with depression, five times more likely to become diagnosed with mild cognitive impairment (MCI), and three times more likely to have memory problems compared with athletes who have not had a concussion.\textsuperscript{47,48} More research is needed in order for athletic trainers and clinicians to fully comprehend the cumulative and long term effects of concussion.

Second Impact Syndrome

There is a 7-10 day period of increased susceptibility of further concussion.\textsuperscript{3,11} However, in some cases, the concussions that occur during that period may not just become more severe, but deadly. An unusual phenomenon known as second impact syndrome (SIS) has been become increasingly more prevalent in high contact sports. Mostly common in athletes from the age of
14 to 16, SIS is the result of a second head injury that occurs when athletes return to activity too soon while still symptomatic following an initial concussion.\textsuperscript{49,50,51,53,54} Even though athletes may return to play weeks later, the second blow to the head starts a physiological process that often has catastrophic results.\textsuperscript{49,52}

The second impact required to initiate such a deadly process does not need to equal that of the initial blow. On the contrary, second impact syndrome may result from minor impacts to head or even other structures such as the chest or back, which may transfer forces and accelerate the brain within the skull. Once an athlete receives the impact necessary to initiate SIS, the athlete may present similarly to a minor concussion and usually will not lose consciousness. In the minutes following the necessary impact, commonly the athlete may collapse with presence of rapid dilation of the pupils, respiratory failure and loss of eye movement.\textsuperscript{49}

A pathophysiological explanation of such occurrences has been offered, suggesting that SIS is the result of a substantial loss of autoregulation of the blood supplied to the brain. This is followed by vascular enlargement within the brain that ultimately leads to herniation and total brainstem failure. The entire process takes only 2 to 5 minutes from second impact to brain herniation. This may be immediately followed by coma and respiratory failure that in many cases results in death. Second impact syndrome has proven to not exist exclusively to football, but to all contact sports. Anyone who suffers a concussion should at least be held out until symptom resolution, however as pointed out earlier, this may not be conservative enough. Cantu suggests that education for parents and coaches stands as the best defense towards such catastrophic outcomes.\textsuperscript{49}
Conclusion

Athletes who suffer concussion will display physical, neurocognitive, and postural differences. As they recover, these symptoms will decline and in order to ensure that athletes make a safe return to activity, a combination of several assessment methods and consistent return to play guidelines should be utilized. In most cases, athletes will make full recoveries and return to their sport without issue. As athletic trainers, it is our responsibility to ensure the safety and well being of our athletes. We do so by holding athletes out of participation until they are symptom free during exertion and within baseline levels of neurocognitive and postural stability measurements. However, a growing body of literature suggests that current guidelines and assessment tools do not provide sensitive enough analysis of symptom recovery to truly suggest that someone is “asymptomatic”. As more developments in our field continue to emerge, concussion management may become more conservative. Developments in postural stability may provide athletic trainers with an assessment tool necessary to understand the long term, cumulative, and possibly life threatening effects of sport-related concussion. Regardless of the success or failure of such efforts, the purpose is to determine if alternatives exists.
References


APPENDIX C

TABLES AND FIGURES

**TABLE 1. Subject demographics and concussion history**

<table>
<thead>
<tr>
<th>Group</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Concussion History</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONC</td>
<td>20.60 ± 1.17</td>
<td>176.91 ± 12.26</td>
<td>83.82 ± 24.87</td>
<td>3.50 ± 0.97</td>
</tr>
<tr>
<td>CTRL</td>
<td>20.50 ± 1.58</td>
<td>175.91 ± 11.29</td>
<td>83.57 ± 22.92</td>
<td>--</td>
</tr>
</tbody>
</table>

There were no significant differences between groups for age, height, or weight. There was a significant difference between groups for concussion history (p<0.001). The mean is provided along with the standard deviation.
**TABLE 2. COP Displacement during GI**

<table>
<thead>
<tr>
<th>Group</th>
<th>$S1\ A/P\ (cm)$</th>
<th>$S1\ M/L\ (cm)$</th>
<th>$S2\ A/P\ (cm)$</th>
<th>$S2\ M/L\ (cm)$</th>
<th>$S3\ A/P\ (cm)$</th>
<th>$S3\ M/L\ (cm)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>CONC</td>
<td>5.24 ± 1.14</td>
<td>6.01 ± 1.07</td>
<td>1.31 ± 1.17</td>
<td>21.07 ± 3.03</td>
<td>16.72 ± 3.05</td>
<td>1.27 ± 0.64</td>
</tr>
<tr>
<td>CTRL</td>
<td>4.88 ± 1.10</td>
<td>6.54 ± 0.97</td>
<td>0.47 ± 1.12</td>
<td>21.62 ± 3.21</td>
<td>16.15 ± 2.50</td>
<td>1.47 ± 1.29</td>
</tr>
</tbody>
</table>

COP displacement during S1, S2, and S3 of GI. There were no significant differences between groups for any sections of GI. The mean is provided along with the standard deviation.
FIGURE 1 – Overhead view of displacement of COP and COM during gait initiation with the right foot as the initial stepping foot.
FIGURE 2 – Layout of biomechanics lab with force platforms and camera set-up for data collection during testing.
**FIGURE 3 – Subject History Questionnaire**

Subject Initials: _____________________ (First MI Last)  
Subject ID #__________  
Date of Testing: ____/____/_____

**A. Demographic Data**

1. Date of Birth: ____ /____/ ______   
2. Sport: ____________________   
3. Age: __________ 
4. Gender: _______________ 
5. Height: ____________    
6. Weight: _______________ 
7. Year in School:  
   Freshman  
   Sophomore  
   Junior  
   Senior  
   Grad Student 

**B. Injury History**

1. Have you ever been diagnosed with a concussion? YES NO  
   If YES, how many?: _________________________________________________________  
   If YES, when did your last one occur?: ________________________________________  
   If YES, did you ever blackout or lose consciousness?: ___________________________  

2. Have you ever suffered an injury to either foot, ankle, leg or knee? YES NO  
   If YES, please describe: ___________________________________________________  

3. Have you ever had surgery on either foot, ankle, leg, or knee? YES NO  
   If YES, please describe: ___________________________________________________  

4. Do you have balance disorders? YES NO  
   If YES, please describe: ___________________________________________________  

5. Do you have diagnosed with a metabolic disorder? YES NO  
   If YES, please describe: ___________________________________________________  

6. Do you have been diagnosed with a neurological disorder? YES NO  
   If YES, please describe: ___________________________________________________  

7. Do you have been diagnosed with a vestibular disorder? YES NO  
   If YES, please describe: ___________________________________________________  

8. Are you currently taking any medications? YES NO  
   If YES, please describe: ___________________________________________________
FIGURE 4 – BESS, SAC, and GSC Total Scores between groups. There were no significant differences between groups for BESS, SAC, and GSC.
**FIGURE 5** – Displacement of COP during the S1 phase of GI in the A/P and M/L direction. There were no significant differences between groups.
FIGURE 6 – COP-COM Displacement at MI, L1, L2, and HS between CONC and CTRL groups. There were no significant differences between groups at any landmarks.
FIGURE 7 – Spatiotemporal measures of step length/velocity between CONC and CTRL groups. There were no significant differences between groups.