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Measurement, characterization, and effects of head impacts in women's soccer

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Eric A. Nauman
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MEASUREMENT, CHARACTERIZATION, AND EFFECTS OF HEAD IMPACTS IN WOMEN'S SOCCER

A Thesis

Submitted to the Faculty

of

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Emily C. McCuen

In Partial Fulfillment of the

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of

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West Lafayette, Indiana

For my parents, Mike and Donna, for their continual love and support.

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ABBREVIATIONS

BOLD	Blood-Oxygen-Level Dependent
CBF	cerebral blood flow
CDC	Centers fro Disease Control and Prevention
CNS	central nervous system
cPAA	cumulative peak angular acceleration
CPP	cerebral perfusion pressue
cPTA	cumulative peak translational acceleration
CVR	cerebrovascular reactivity
fMRI	functional magnetic resonance imaging
MR	magnetic resonance
PAA	peak angular acceleration
PNS	peripheral nervous system
PTA	peak translational acceleration
NCAA	National Collegiate Athletic Association
NFL	National Football League
NHL	National Hockey League
RSME	root mean squared error
WWE	World Wrestling Entertainment

ABSTRACT

McCuen, Emily C. M.S.B.M.E., Purdue University, May 2015. Measurement, Characterization, and Effects of head impacts in Women's Soccer. Major Professor: Eric A. Nauman.

The potential for long term neurological deficits resulting from repetitive head trauma is a major concern for collision sport athletes [1] [2]. Research conducted on football played has found neurophysiologic changes in the absence of concussion in athletes as early as high school age [3] [4] [5]. Given that female soccer players show the highest rate of concussion for female athletes and a higher rate of concussion than their male counterparts, it is important to characterize the types of impacts female soccer athletes receive and assess female soccer athletes for neurophysiologic changes due to these impacts [6]. This work paired head impact sensors with functional MRI to assess the effects of head impacts experienced by female soccer players at both the high school and collegiate level. A total of 29 high school female soccer athletes were studied from two different high schools (HS1: n=12, HS2: n=17) and 14 collegiate Division I female soccer athletes from one university. Collegiate athletes sustained significantly higher cumulative loads in terms of peak translational and peak angular accelerations than high school athletes ($p<.001$). However, a subset of high school athletes sustained cumulative loading on par with collegiate athletes despite their season being 2-4 weeks shorter than the collegiate season. High school athletes experiencing high cumulative loads through a 10-12 week season exhibited a significant decline in cerebrovascular reactivity. Overall, the results indicate that the female soccer athletes sustain significant mechanical loading to the head throughout a season, capable of causing neurophyisiologic changes in the brain. These results

indicate that soccer athletes may also be at risk for chronic neurologic damage even in the absence of concussion.

1. INTRODUCTION

1.1 Motivation

In the United States it is estimated that between 1.6 and 3.8 million sports related concussions occur annually, with affiliated treatment costs of up to $60 billion per year [7], although recent work shows the number of concussions may be grossly underestimated [8] [9]. A study showed that that 5% of all sports related injuries are mTBI [10], with head injuries accounting for at least 11% of all injuries in soccer [11] [12]. While football has the highest percentage of sports-induced head trauma in males, soccer is the leading source of concussion in female athletes, with a rate of concussion higher than that of male soccer players [6] [13]. Most importantly, studies looking at all levels of soccer play have shown neurological deficits associated with soccer athletes, even in the absence of head injury diagnoses [11] [14] [15].

Current concussion management relies on resolution of neurocognitive symptoms, however, there is now evidence from studies using alternative techniques which suggest residual effects of mTBI may be present even after neurocognitive symptoms have resolved and athletes have returned to play [3] [16] [17]. Given that sub-concussive hits (defined as head impacts inducing no readily observable symptoms) have the ability to cause neural damage that could lead to long-term deficits if not given proper time to heal [1] [18], further research is required to characterize the effects of sub concussive impacts during play. Recent studies have begun to do this for football athletes, showing that at least 70% of high school football athletes exhibit changes in neurophysiology without easily diagnosable symptoms [8].

Football players sustain a broad range of head impacts with an average magnitude of about 22g, and some impacts exceed 250g [3]. Characterization of head impacts in soccer has only been studied in laboratory controlled studies. These studies show

typical head impacts in soccer to be in the range of 16 - 25 G for soccer balls travelling between 9 - 12 m/s [19] [20] [21] [22]. Further work is required to characterize head impacts in soccer during the uncontrolled settings of live practice and game play.

In addition to characterizing head impacts in soccer, the effects of these head impacts needs to be assessed. A viable metric for assessing the neurophysiological effects of head trauma in soccer is cerebrovascular reactivity (CVR) to CO_2, a compensatory mechanism where blood vessels dilate in response to hypercapnia to regulate blood flow. CVR is known to be a contributor to cerebrovascular sequelae of mild traumatic brain injury (mTBI) as animal and human studies have demonstrated CVR reductions following mTBI contributing to susceptibility to secondary injury [23] [24] [25]. Previous research has shown changes in CVR for high school football athletes throughout a competition season [5] [26]. This work will assess high school and collegiate female soccer athletes are assessed for similar changes.

1.2 Objectives

The purpose of this work was twofold. The first goal was to characterize the head impacts sustained by high school and collegiate female soccer athletes, in terms of magnitudes of individual impacts and cumulative loading throughout a season. This works to expand on previous studies in the interest of determining the actual distribution of head impacts sustained by athletes during a full season of practices and games. In order to accomplish this, a validation study was performed on the sensors used in this study to ensure accurate characterization of head impacts. The second goal was to determine if the magnitudes of hits taken or if the cumulative loading sustained throughout a season of soccer had an effect on cerebrovascular reactivity. It was hypothesized that high cumulative loading resulting from head impacts for athletes would lead to an impairment in cerebrovascular reactivity in female soccer athletes as has been found in studies looking at football athletes.

2. LITERATURE REVIEW

2.1 Clinical Neuroanatomy

The nervous system consists of the central nervous system (CNS), composed of the brain and spinal cord, and the peripheral nervous system (PNS), composed of all the nervous system components outside the CNS [27]. There are four major parts of the brain, the brain stem, cerebellum, diencephalon and cerebrum (Fig. 2.1). The brain stem is continuous with the spinal cord and consists of the medulla oblongta, the pons and the midbrain. Located superior to the brain stem, the diencephalon contains the thalamus, hypothalamus and epithalamus [27].

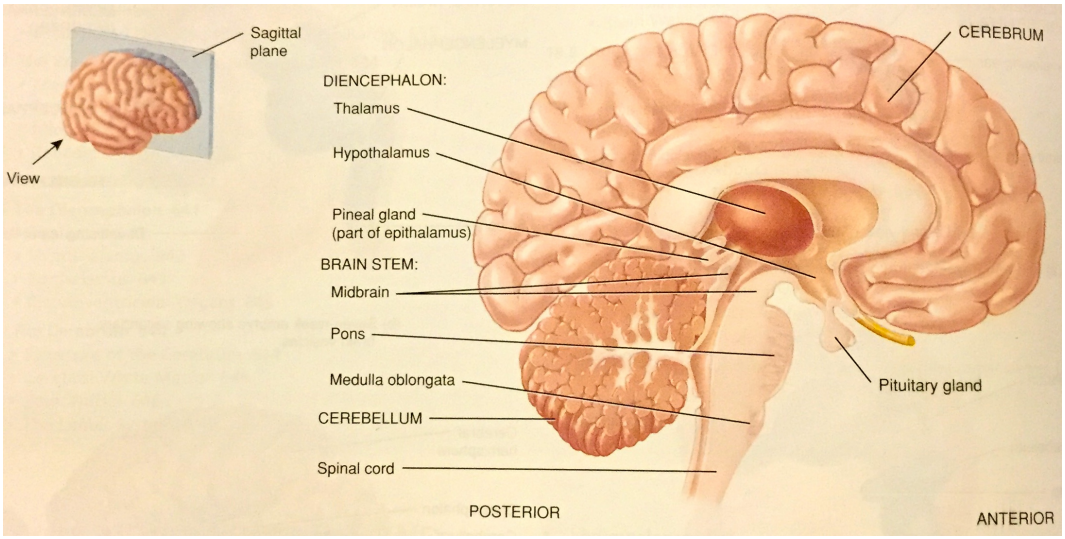


Fig. 2.1. The locations of the four major parts of the brain. Adapted from [27]

The cerebrum is divided into two hemispheres, separated by a groove called the longitudinal fissure [27]. The outer part of the hemispheres contain grey matter (or the cerebral cortex), holding the neuronal cell bodies. The surface contains folds

called gyri and grooves between the folds called sulci. Beneath this layer is white matter which holds the mylinated axons of the neuronal cell bodies [27].

There are four lobes that further divide the cerebral cortex, the frontal lobe, parietal lobe, occipital lobe and the temporal lobe (Fig. 2.2). The precentral gyrus, located in the frontal lobe, contains the primary motor area and the postcentral gyrus, located in the parietal lobe, contains the primary somatosensory area. The primary visual area is located on the occipital lobe, the primary auditory area and primary olfactory area are located on the temporal lobe [27].

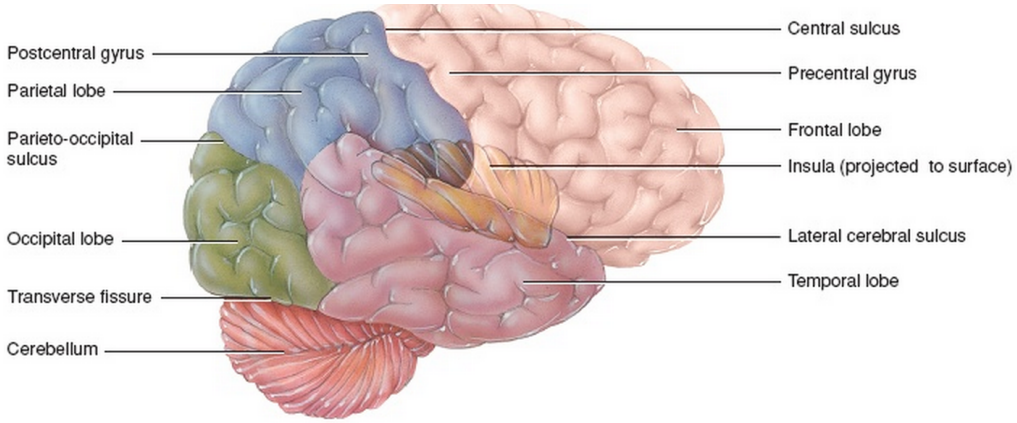


Fig. 2.2. A right lateral view of the cerebral cortex. Adapted from [27]

The brain is protected by the cranial meninges, consisting of three layers. The outermost layer is the dura mater, the middle layer is the arachnoid mater and the innermost layer is the pia mater. Extensions of the dura mater separate the two hemispheres of the cerebrum, the hemispheres of the cerebellum and the cerebrum from the cerebellum. Cerebrospinal fluid is a clear fluid circulating through cavities and around the brain and spinal cord that protects the brain from chemical and physical harm [27].

Blood flow enters the brain through the internal carotid and the vertebral arteries. It is drained out of the brain through the internal jugular veins. Blood from the internal carotid flows into the anterior and middle cerebral arteries, supplying blood

to the lateral and anterior portions of the cerebrum (Fig. 2.3). Blood from the ventral arteries flow into the basilar artery which leads to the posterior cerebral arteries, supplying blood flow to the posterior and inferior lateral parts of the cortex. The basilar artery also connects to arteries supplying blood flow to the pons and the cerebellum [27].

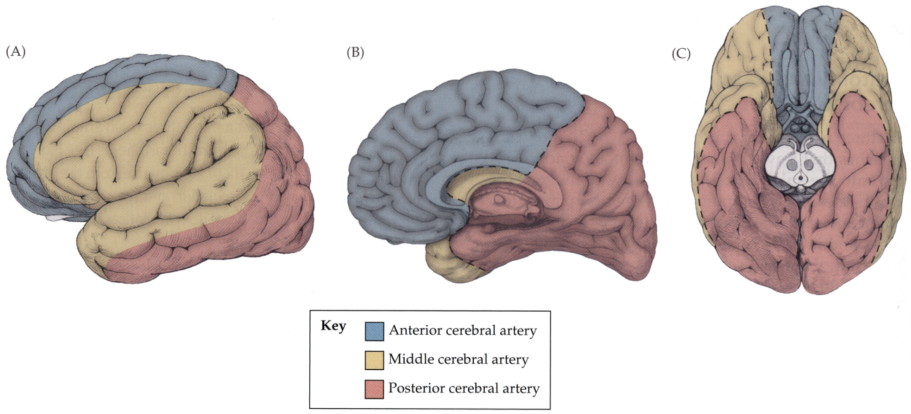


Fig. 2.3. The sections of the brain with blood supply from the anterior, middle and posterior cerebral arteries. Adapted from [28]

The cellular structure of the brain consists of neurons and glial cells. Neurons carry action potentials which provide motor and sensory information. There are three types of glial cells. Astrocytes help to create the blood brain barrier, restricting movement of substances between the blood and the interstitial fluid of the CNS [27]. Oligodendrocytes form and maintain myelin, the protective covering around CNS axons [29]. Finally, microglia function as phagocytes removing cellular debris and damaged nervous tissue [27].

2.2 Concussion Definition

At the first International Conference on Concussion in Sport, concussion was defined as “a complex pathophysiological process affecting the brain, induced by trau-

matic biomechanical forces" where "clinical symptoms largely reflect a functional disturbance rather than structural injury" resulting in "a set of clinical syndromes that may or may not involve loss of consciousness" [30]. This definition has largely remained consistent through the second, third, and fourth International Conference on Concussion in Sport, while acknowledging that symptoms may be prolonged or persistent [31] [32] [33]. The definition further describes the characteristics of concussion to be caused by a direct blow to the body or head, resulting in the rapid onset of short lived impairments that may result in neuropathological changes but largely reflect functional rather than structural changes. Concussions may result in a set of clinical symptoms that could include a loss of consciousness [33].

The Centers for Disease Control and Prevention (CDC) similarly defined mTBI as a pathophysiologic process that affects the brain caused by biomechanical forces following a direct or indirect blow to the head. This is usually associated with normal structural findings, and a variety of physical, cognitive, emotional and/or sleep related symptoms. As with the definition for concussion, mTBI may or may not involve a loss of consciousness and the symptoms may be prolonged or persistent [34].

2.3 Injury Cascade

Following biomechanical injury to the brain, there is a dysfunction of brain metabolism [34]. This cascade begins as neurotransmitters are released and ionic fluxes occur, leading to neuronal depolarization and ionic shifts causing changes in cellular physiology [35]. A period of "hypermetabolism" occurs, creating an energy crises, followed by a period of depressed metabolism [35]. The cerebral pathophysiology is also effected in other ways including generation of lactic acid, decreased intracellular magnesium, production of free radicals, inflammatory response and altered neurotransmission [35].

Cerebral blood flow (CBF) is also decreased, possibly by up to 50% following injury, and can remain lowered for a period of time depending on the severity of injury [24] [35]. This blood flow is controlled by the cerebral perfusion pressure (CPP),

cerebrovascular resistance [23] [24]. Additionally, blood vessels dilate in response to changes in the partial pressure of CO_2 in order to regulate cerebral blood flow [24]. This compensatory mechanism, called cerebrovascular reactivity (CVR) has been shown to be decreased following mTBI [23] [24] [25]. A recent case study demonstrated preliminary evidence supporting the use of CVR in breath-hold fMRI as a biomarker for mTBI [36]. Other work has also shown decreased CVR in football players during a season of play [26].

2.4 Clinical Diagnosis and Treatment of Concussion

Diagnosis of mTBI is done by completing a systematic assessment of the injury looking at clinical symptoms, physical signs, cognitive impairment, neurobehavioral features, and sleep disturbance [33] [34]. This is done using a evaluation tool such as the Acute Concussion Evaluation (ACE), the Sport Concussion Assessment Tool (SCAT3), or the Child-SCAT3 form (Appendix A). Symptoms include somatic, cognitive or emotional symptoms, physical symptoms such as loss of consciousness, behavioral changes such as irritability, cognitive deficits and sleep disturbances [33].

Clinical management of a patient with a diagnosis of concussion includes monitoring the patient in office or at home, looking for symptoms that remain constant or worsen. Symptoms are expected to decrease or resolve over a period of 3-5 days [34]. If symptoms are not reducing over that period of time it might be appropriate for a doctor to refer a patient to an mTBI specialist who will evaluate the patient further and manage their return to normal activities. Diagnostic testing (either neuroimaging or neurophsychological) may also be prescribed to provide more information during the acute phase of concussion [34].

2.5 Current Return to Play

Following a period where the athlete is symptom free for at least 24 hours the athlete can return to play gradually advancing to higher levels of physical activity

following a symptom free period of at least 24 hours after each level. [37]. This step wise return to play is outlined in Table 2.1 [33].

Table 2.1.
Return to play levels. Adapted from [33]

Level of Rehabilitation	**Description of Possible Activities**	**Objective**
1. No activity	Physical and cognitive rest	Recovery
2. Light Aerobic Exercise	Walking, swimming, low intensity cycling	Increase HR
3. Sport specific exercise	Running drills, no head impacts	Add movement
4. Noncontact training drills	More complex training drills	Exercise, coordination and cognitive loading
5. Full contact practice	Participation in normal training activities	Restore confidence and assess functional skills by coaches
6. Return to Play	Normal Game play	

As stated above, neurocognitive testing is often used in addition to visible symptom checks. The most common computerized neurocognitive testing tool is the Immediate Post Concussion Assessment and Cognitive Testing (ImPACT) for making return to play decisions, utilized in the NFL, NHL and WWE [38]. Athletes will undergo baseline testing prior to athletic contact practices, followed by further testing upon injury until the athlete returns to baseline.

2.6 Concussion Epidemiology

While football has the highest rate of concussion overall, in female sports, soccer athletes suffer from the highest rate of concussion, higher than that of male soccer athletes [6] [13]. A study conducted by Gessel et al. showed that high school soccer girls had a rate of .36 concussions per 1000 athletic exposures, with .63 concussions per 1000 athletic exposures for collegiate athletes. Another study, by Covassin et al., showed the concussion rate for games was about 2.06 concussions per 1000 game exposures, and .13 for practices per 1000 practice exposures for collegiate female soccer athletes [13]. This is compared to football athletes with concussion rates of .47 concussions in 1000 athletic exposures for high school athletes, and .61 concussions in 1000 athletic exposures for collegiate athletes [6].

2.7 Subconcussive Impacts

The potential for chronic brain damage resulting from repetitive head trauma is a serious concern for collision sport athletes [18]. It has been shown that sub-concussive hits (defined as head impacts inducing no readily observable symptoms) have the ability to cause neural damage that could lead to long-term deficits if not given proper time to heal [1] [3] [18]. Despite the known importance of characterizing head trauma in collision sport athletes, little is known about the number and magnitude of head impacts experienced by female soccer players.

2.8 Chronic Traumatic Encephalopathy

Omalu et al. reported on a professional football player that had no known history of brain trauma outside the game of football. The athlete showed severe neurocognitive deficits, and an autopsy revealed mild pallor of the substantia nigra, mild neuronal dropout in frontal, parietal and temporal neocortex, as well as amyloid plaques and sparse neurofibrillary tangles [2]. This study was the first case showing

neurodegeneration in this football population, coining the term for this pathology "chronic traumatic encephalopathy" (CTE) [2].

The neuropathology found by Omalu was similar to the neuropathology of dementia pugilistic, a neurodegenerative disorder associated with head trauma in boxing [39]. It has been noted that CTE and dementia pugilistica appear to be the same pathology with dementia pugilistic being a more severe variant of the disease [1]. The characteristics of this pathology include astrocytic tangles, and neurofibrillary tangles [40]. Many other cases of CTE have been reported including cases of CTE in over 50 football players, boxers, hockey players, a soccer player, a professional wrestler, abuse victims, chronic head bangers, and a circus clown involved in "dwarf throwing" [40] [41]. Common symptoms reported in these case studies include mild to severe memory deficits, mild to severe parkinsonian symptoms, chronic headaches, depression, anxiety, and mood swings (15,40) [2] [40] [42] [43] [44].

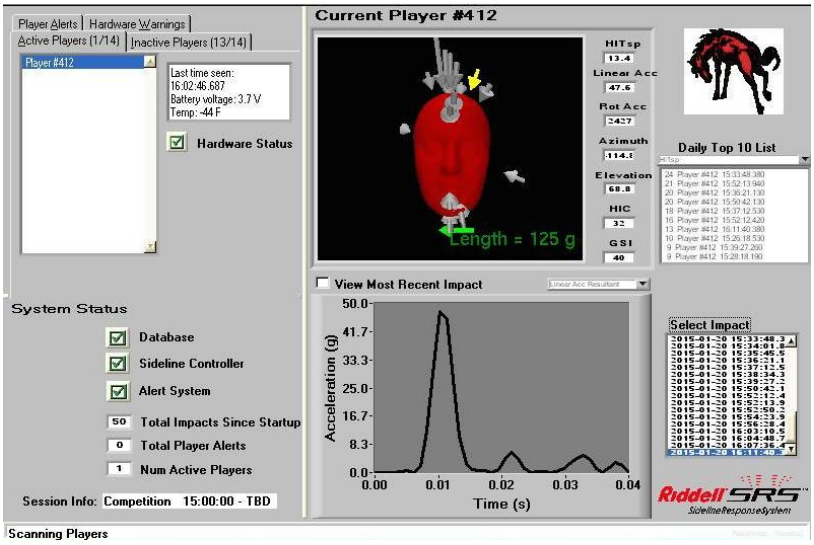
2.9 Head Collision Event Monitoring

2.9.1 Head Telemetry and HITS

The 6 accelerometer measurement device commonly used by researchers in studying head impacts in football is the Head Impact Telemetry (HIT) System (Simbex, Lebanon, NH) [3] [45] [46]. This device, placed within the helmet, is commercially available and uses 6 nonorthangonally mounted single-axis accelerometers oriented normal to the head (Fig. 2.4). The device wirelessly transmits data from the measurement device to a computer located at the sideline. The computer processes and displays the data in real time. The algorithm determines peak resultant linear acceleration and estimates peak rotational accelerations based on an assumed pivot point in the neck.

Many researchers have utilized this device to collect telemetry data. Guskiewicz et al. found that a wide range of hit magnitudes caused concussions, reporting a range of 65.1g - 168.71 g for collegiate football athletes. This study noted that less than

(a) The HITS system within a helmet, the red sections show where the sensors are located within the helmet padding.



(b) The output of the HITS system to a computer via wireless communication.

Fig. 2.4. A look at the HITS System.

half of 80g+ impacts led to a concussion [46]. Additionally, Breedlove et al. studied a group of high school players and found comparable hit magnitudes [3]. This device is most commonly used to study football, however one group did use the technology to look at head impacts in soccer. Firing the ball at an average of 40 m/hr, Naunheim et al. found helmeted soccer athletes were undergoing linear accelerations averaging 54.7 + 4.1 g [47]. However, this method of testing is not indicative of a typical soccer situation.

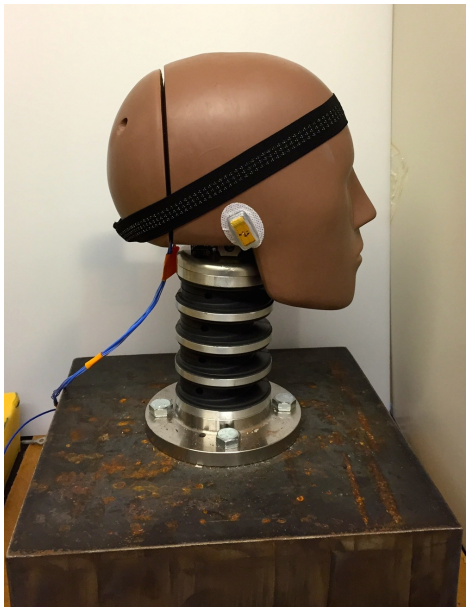
2.9.2 X2 xPatch

For non-helmeted sports, such as soccer, other sensors have been developed. The xPatch, developed by x2 Biosystems, measures about 3 x 1.5 x 1 cm and can be worn on a patch behind the athletes right or left ear, or can be placed inside a headband for the athlete to wear (Fig. 2.5). The xPatch uses a three axis accelerometer to track linear accelerations and a gyroscope to track angular velocities. Any impact over a 10g threshold is stored to the device and later downloaded to a computer using Impact Monitoring System software (X2 Biosystems, Seattle, WA). This software outputs the resultant peak translational and angular acceleration for each impact recorded.

2.10 Functional Magnetic Resonance Imaging

Functional Magnetic Resonance Imaging (fMRI) utilizes the magnetic differences between oxygenated and deoxygenated blood, where oxygenated brain regions produce a larger magnetic resonance (MR) signal than less oxygenated areas. During brain activation localized areas increase in blood flow causing the MR signal to increase, reflecting neuronal activity in those regions [48]. This type of imaging is called blood-oxygen-level-dependent imaging (BOLD). BOLD fMRI can be used with a breath hold task, to induce hypercapnia, to measure CVR to CO_2 [49]. Given that CVR is reduced in athletes with mTBI it is a useful measure of neurophysiologic changes without neurocognitive changes.

(a) xPatch Headband.



(b) xPatch Right Ear.

Fig. 2.5. Two methods of wearing the xPatch device during live play.

3. XPATCH SENSOR VALIDATION

3.1 Methods

Validation of the xPatch sensors (X2 Biosystems, Seattle, WA) was performed by placing a set of sensors (right ear patch, left ear patch, and headband) on a Hybrid 3 headform (Fig. 3.1) and applying impact loads using an instrumented mallet. The attachment for sensors worn as a patch involves a small adhesive sticker with the sensor stuck on one side and the other side firmly placed behind the ear. Sensors worn in a headband were placed in a sleeve within the headband and the sensors sat at the base of the skull on the back of the head. Loads were applied to five different locations on the head (front, left side, right side, back and top). A total of five sensors were tested, each in all three locations on the head (right ear patch, left ear patch and headband), with 50 impacts for each sensor in each location. The xPatch sensor outputs a value for the peak translational acceleration and for peak rotational acceleration. The software also shows the linear time graph of the raw accelerometer and gyroscope data (Fig. 3.2).

The translational acceleration was obtained from a 3 single axis accelerometers at the head center of mass and compared to the translational acceleration obtained by the xPatch sensors. Angular accelerations were obtained using the 3 accelerometers located at the center of the mass of the head and 6 additional single axis accelerometers and compared to the angular acceleration determined from the gyroscope within the xPatch sensor [50](Fig. 3.3, Eqns. 3.1, 3.2, 3.3). The impacts loaded to the head form ranged from 20 - 120 g (mean 39g, median 37g).

$$\dot{\omega_X} = \frac{(A_{1Z} - A_{PZ})}{2r_X} - \frac{(A_{3Y} - A_{PY})}{2r_Z} \tag{3.1}$$

$$\dot{\omega_Y} = \frac{(A_{3X} - A_{PX})}{2r_Z} - \frac{(A_{2Z} - A_{PZ})}{2r_Y} \tag{3.2}$$

$$\dot{\omega_Z} = \frac{(A_{2Y} - A_{PY})}{2r_Y} - \frac{(A_{1X} - A_{PX})}{2r_X} \tag{3.3}$$

3.2 Results

The xPatch sensor verification demonstrated a root mean square error for sensors worn on the right and left ear were less than 53% for both PTA and PAA (Table 3.1). Sensors worn as a headband measured PTA with 41.7% root mean squared error, but an error of 305.4% for PAA.

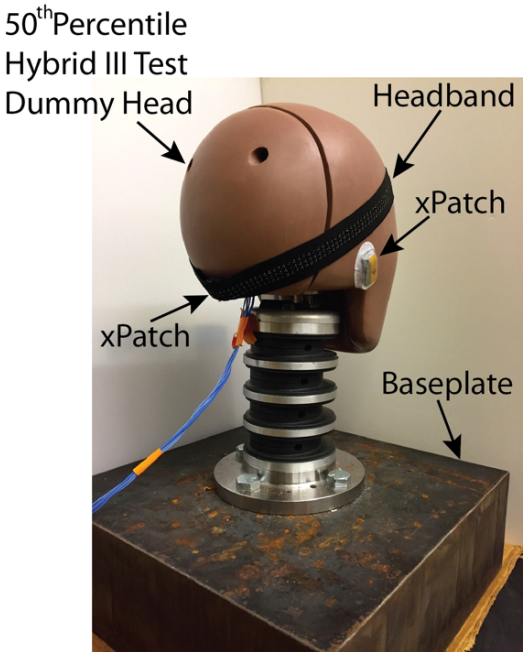


Fig. 3.1. The testing set up for the Hybrid III test dummy. Three xPatches were used simultaneously, one on the right ear, the headband and on the left ear (not shown). The baseplate was 12.25 x 12.25 x 4 and weighed 170 lbs.

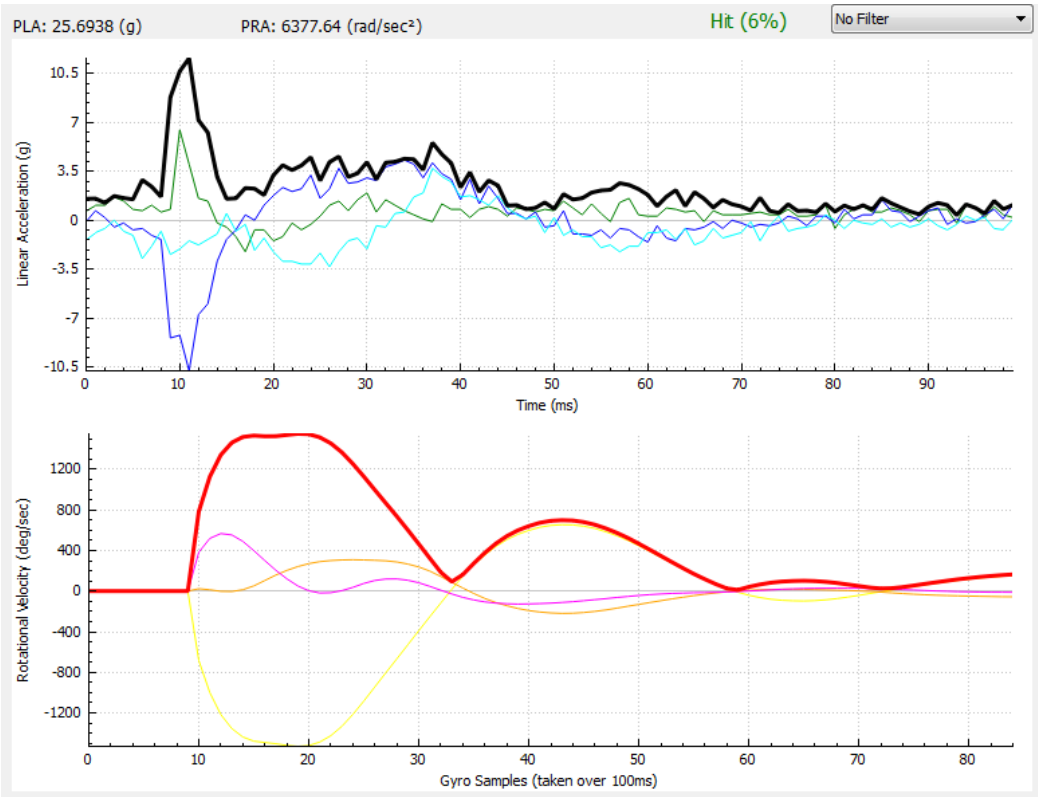


Fig. 3.2. The output from the xPatch sensor to the software for each impact. The time series plots show the raw linear acceleration and rotational velocity. The final peak acceleration values are listed at the top of the figure.

3.3 Discussion

Overall, sensors worn as a patch were relatively accurate when compared to the center of the mass of the head. In lab testing has shown that xPatch is one of the better sensors currently available for head impact monitoring. However, sensors worn as a headband were very unreliable as shown by the high RMS errors obtained in the validation study. The sensors within the headbands are only constrained by the headband on two sides, allowing rotation when the head is impacted in certain directions which could be the cause of the large errors. For this reason, data from athletes wearing the sensor on a headband were excluded from all analyses.

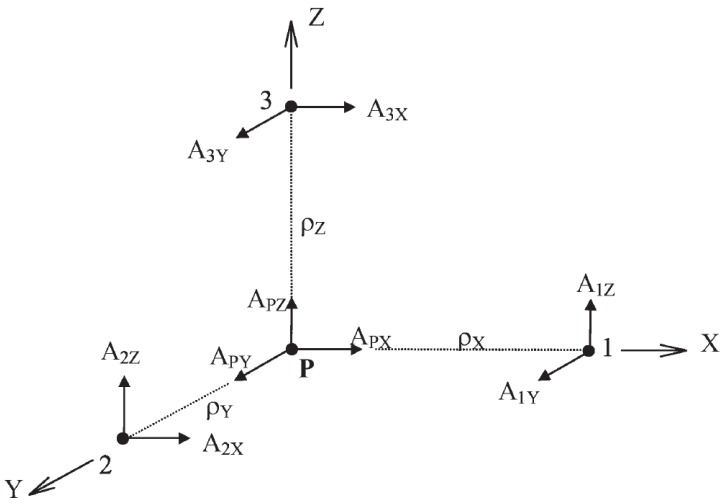
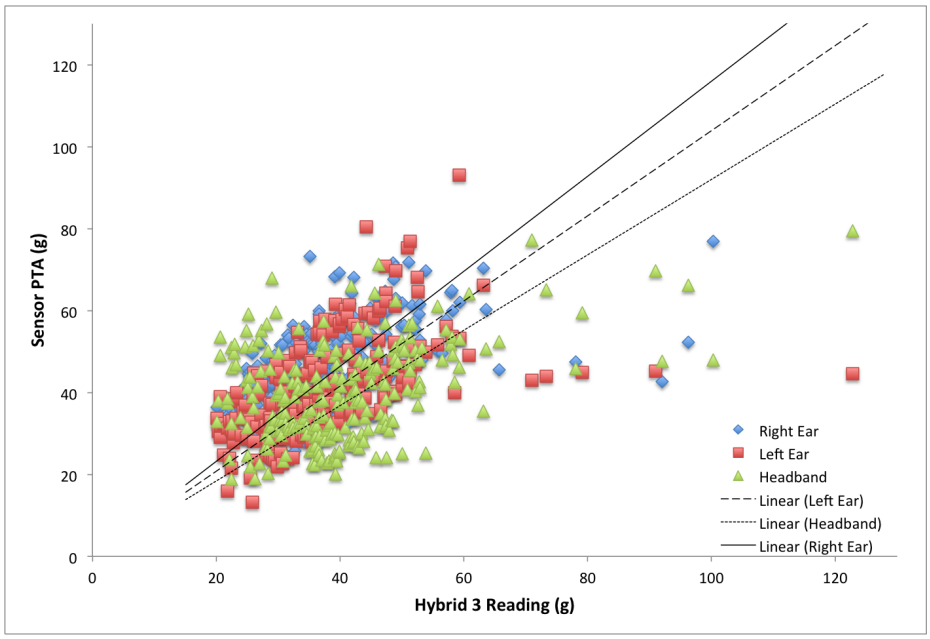


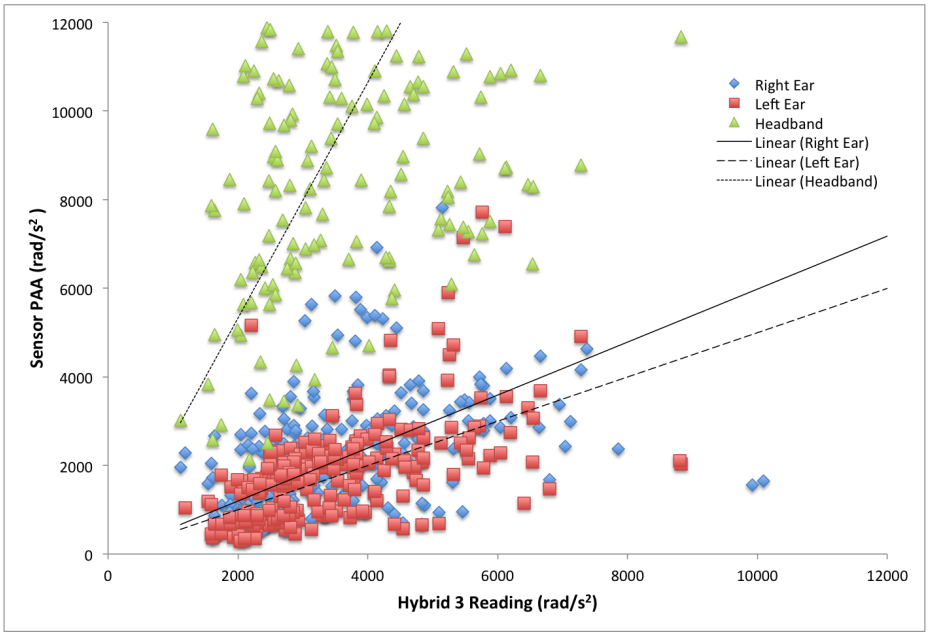
Fig. 3.3. The nine accelerometer package used to calculate angular acceleration. Adapted from [50]

Table 3.1.
RMSE for each of five sensors used to evaluate the accuracy of the xPatch systems.

Sensor Number	**Location**	**RMSE PTA**	**RMSE PAA**
1	Right Ear	45.85%	48.29%
2	Right Ear	33.97%	42.09%
3	Right Ear	47.16%	48.00%
4	Right Ear	36.49%	48.02%
5	Right Ear	33.18%	51.91%
Overall	**Right Ear**	**40.10%**	**48.10%**
1	Left Ear	34.31%	55.78%
2	Left Ear	25.98%	53.92%
3	Left Ear	17.97%	50.44%
4	Left Ear	34.97%	48.83%
5	Left Ear	34.32%	54.74%
Overall	**Left Ear**	**30.60%**	**52.70%**
1	Headband	35.56%	369.89%
2	Headband	30.33%	254.74%
3	Headband	52.43%	357.69%
4	Headband	35.25%	340.52%
5	Headband	49.10%	179.11%
Overall	**Headband**	**41.70%**	**305.40%**



(a) Peak Translational Accelerations.



(b) Peak Angular Accelerations.

Fig. 3.4. The acceleration data recorded for sensors worn on the right ear, left ear and headband compared to the acceleration data from the Hybrid III Test dummy. A perfect match between the sensor and test dummy would show a slope of one.

4. HIGH SCHOOL AND COLLEGIATE HIT COMPARISONS

4.1 Methods

4.1.1 Subjects

All research methods involving human subjects were approved by Purdue's Institutional Review Board prior to the initiation of the study. Participant written informed consent was obtained from those 18 years of age and over and both parental consent and participant assent were obtained from all subjects under the age of 18.

Data were collected from 29 female high school athletes (ages 14-18, mean: 15.7; 2014) and 24 female National Collegiate Athletic Association (NCAA) athletes (ages 17-22, mean: 18.8; 2013) across one competition season. Two high schools were represented: HS1 (n=12), HS2 (n=17) and one collegiate team (n=24). For the final analysis a subset of collegiate athletes were used, due to the exclusion of players wearing headbands. In the final analysis, complete datasets from a group of 14 female collegiate soccer athletes (ages 17-22, mean: 18.7; 2013) were analyzed.

4.1.2 X2 xPatch Sensors

For the purposes of this study, each practice and game was considered an athletic "session." The sensors were placed on the players head with an adhesive patch placed behind the player's right ear. The sensors continuously monitored and measured three axes of translational acceleration and three axes of angular velocity. Head impacts were recorded to the sensor when it measured translational accelerations greater than 10 g. The angular acceleration was calculated by numerically differentiating the angular velocity and the rigid body kinematics equations were used to determine the

accelerations of the head center of mass. Following each practice and game the data were downloaded using the Head Impact Monitoring System software (X2 Biosystems, Seattle, WA). Features within the software provided the peak translational acceleration (PTA) and peak angular acceleration (PAA) and the estimated location of the impact. The sensors also collected low acceleration events (10-20 g) for hard stops, cuts and hard kicks. To focus the analysis on head accelerations likely resulting from actual impacts to the head or the body, only those head acceleration events that surpassed 20 g were included.

4.1.3 Statistical Analysis

To compare data from high school players to collegiate athletes, t-tests were used on the average PTA, average PAA, cumulative PTA and cumulative PAA. For all statistical tests a significance level of $p < .05$ was used.

4.2 Results

The average PTA sustained by high school athletes was significantly lower than that of collegiate athletes when compared using a two sample t-test ($p<<.05$ for PTA, $p=.0526$ for PAA). In addition, the collegiate athletes received more than twice as many impacts per session than high school players (Table 4.1). High school athletes took an average of 2.05 hits per session at an average peak linear acceleration of 37.56g (95% CI [36.94; 38.18]), and an average peak angular acceleration of 7,523 rad/s2 (95% CI [7,390; 7,656]). In comparison, collegiate athletes recorded an average of 4.59 hits per session at an average peak linear acceleration compared to high school of 39.31g (95% CI [38.81; 39.81]), and an average peak angular acceleration of 7,713 rad/s2 (95% CI [7,606; 7,819]). Both average PTA and PAA were significantly higher for collegiate athletes as compared to high school athletes.

Given that the collegiate players took more impacts, their mean cumulative exposure to translational (cPTA) and angular accelerations (cPAA) throughout the season

was significantly higher than those of the high school players ($p<<.05$ for cPTA and cPAA) (Fig. **??**). High school teams sustained an average cumulative cPTA of 4,848g (95% CI [3,473; 6,222]), and an average cPAA of 947,940 rad/s2 (95% CI [661,516; 1,280,363]). These are both lower than the metrics of collegiate athletes, who sustained an average cPTA of 12,213g (95% CI [8,928; 15,498]), and an average cPAA of 2,368,700 rad/s2 (95% CI [1,711,176; 3,026,223]).

4.3 Discussion

The goal of this study was to quantify the translational and angular accelerations resulting from head impacts sustained over the course of a season by female athletes competing in soccer at the high school and collegiate levels. These data are notably absent from the literature despite the fact that soccer accounts for the greatest rate of sports-related concussions in women. While the number of head impacts per session was not significantly different between high school and collegiate athletes, there were small but significant increases in the average translational accelerations. More importantly, because the collegiate athletes participated in substantially more practices and games, their cumulative exposure was more than double that of the high school athletes.

These findings are particularly important given the fact that a nearly a two-fold increase in concussions has been documented in collegiate female soccer players as compared to those in high school [6]. Recent work suggests that some aspect of cumulative exposure is the primary risk factor in the development of pathological neurophysiological changes accrued throughout the season in contact sports [3] [8] [4] [26] [9]. Consequently, one would expect an increase in the number of sub-concussive blows as well as the number of large head impacts often thought to cause concussions. Regardless of which mechanism dominates the damage process, these data suggest that it is important to reduce the cumulative exposure.

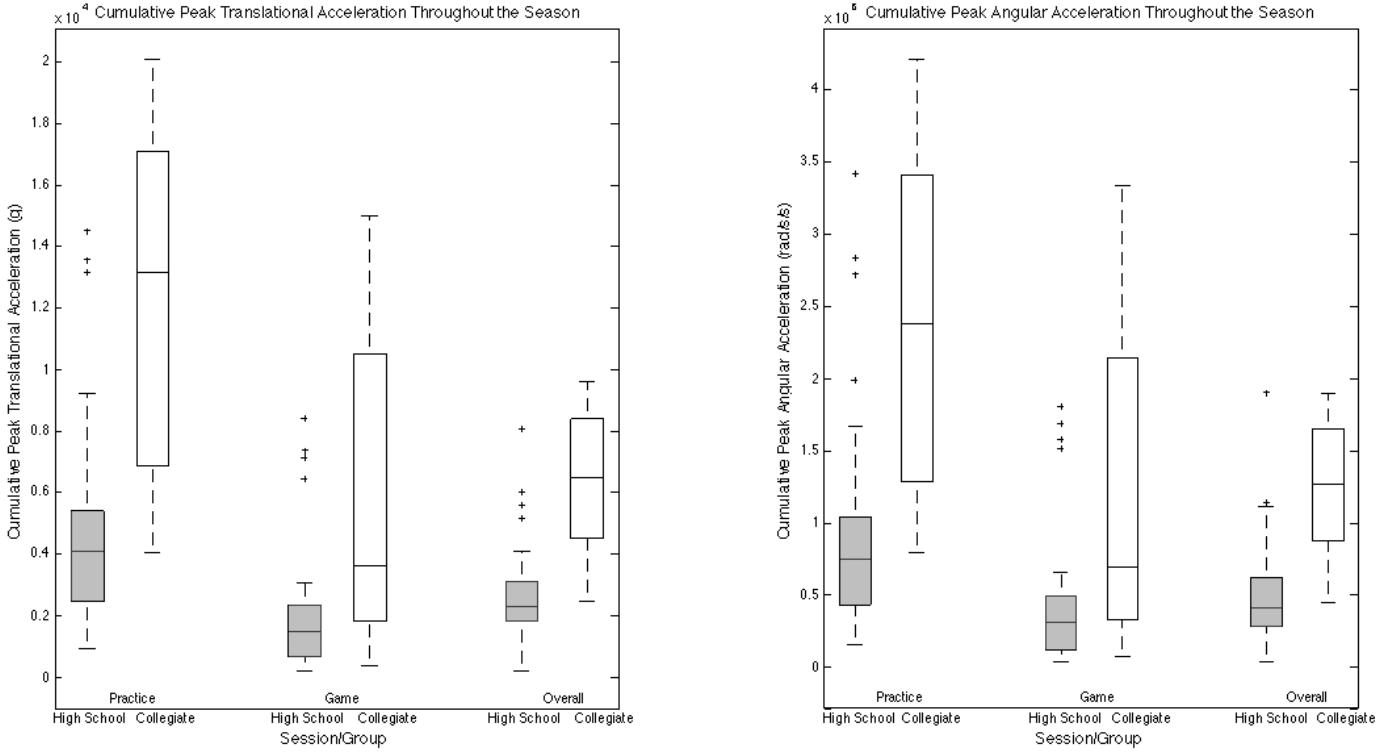


Fig. 4.1. The cumulative peak translational and peak angular accelerations for games and practices for high school and collegiate athletes. Shading indicates significant differences from the collegiate group ($p<0.001$). In all instances collegiate athletes sustained higher cumulative loading. Howeever, a (+) indicates an outlier, indicating there are several high school athletes who sustain higher cumulative loading.

Table 4.1.

Head impact metrics for female high school and collegiate soccer athletes. * indicates significantly different from Collegiate values.

	Impacts/Player/Session	PTA (g)	PAA (rad/s^2)	cPTA (g)	cPAA (rad/s^2)
Session Type		M edian; Mean [95% Confidence Interval]			
Practices					
High School	1.69	30.15; 37.7	6,371; 7,095	2,287; 2,752*	411,380; 517,460*
		[37.1; 38.3]	[6,969; 7,221]	[2,109; 3,398]	[372,946; 661,974]
Collegiate	3.52	30.7; 37.7	6,301; 7,297	6461.2; 6487.8	1,269,500; 1,248,200
		[37.15; 38.3]	[7,181; 7,413]	[5210; 7766]	[998,309; 1,498,091]
Games					
High School	2.85	30.85; 37.3*	6,792; 8,078	1,470; 2,095*	313,560; 453,480*
		[36.7; 37.9]	[7,939; 8,217]	[1,227; 2,964]	[433,858; 473,102]
Collegiate	6.98	31.9; 41.2	6,715; 8,213	3618.9; 5625.4	690410; 1120400
		[40.2; 42.2]	[8,005; 8,421]	[2791; 8460]	[528063; 1712737]
Overall					
High School	2.05	30.5; 37.56*	6,530; 7,523	4,063; 4,848*	745,690; 970,940*
		[36.9; 38.1]	[7,390; 7,655]	[3,473; 6,222]	[661,516; 1,280,363]
90th percentile		63.3	13,020	11,565	2,424,200
75th percentile		44.3	9,419	5,388	1,038,300
Collegiate	4.59	31.2; 39.3	6,429; 7,713	13,149; 12,213	2,374,000; 2,368,700
		[38.8; 39.8]	[7,606; 7,818]	[8,928; 15,498]	[1,711,176; 3,026,223]
90th percentile		71.2	14,421	19,529	3,892,900
75th percentile		47.3	10,153	17,075	3,406,600

5. CEREBROVASCULAR REACTIVITY CHANGES

5.1 Methods

5.1.1 Subjects

All research methods involving human subjects were approved by Purdue's Institutional Review Board prior to the initiation of the study. Participant written informed consent was obtained from those 18 years of age and over and both parental consent and participant assent were obtained from all subjects under the age of 18.

A subset of subjects from the high school and collegiate hit comparisons also underwent fMRI scanning throughout the season. Data were collected from 14 female high school soccer athletes (ages 15-17; mean 15.9; 2014), and 8 female collegiate athletes (ages 17-21; mean 18.5; 2013) across one competition season. Two high schools were represented: HS1 (Soccer: n=7), HS2 (Soccer: n=7), and one university (Soccer: n=8). High school soccer athletes underwent MRI scanning sessions before starting contact practices (Pre), within the first 6 weeks (In1) of the contact season, within the second 6 weeks (In2) of the contact season, 1-2 months after the end of the contact season (Post1), and 5-6 months after the end of the contact season (Post2). The contact season for HS1 was 12 weeks long while the contact season for HS2 was 10 weeks long. Collegiate soccer athletes underwent MRI scanning sessions before starting contact practices (Pre) and within 10 days after the end of the contact season (Post1). The collegiate contact season was 14 weeks long. It is important to note that the transitions from Pre to In marked the onset of contact as all athletes were physically active at the time of the Pre scan. Data were also collected from 8 control female athletes (ages 15-17; mean 16.1; 2014) who only participated in non-collision sports at the high school level (Basketball, Track and Field, Cross Country, Volley-

ball). Control athletes underwent two MRI scanning sessions (baseline, followup), 6 weeks apart, during comparable periods of comparable physical activity. Participants were not excluded from the study due to a history of concussion and no included participant was diagnosed with a concussion during the course of the study.

5.1.2 Imaging

A single blocked breath-hold fMRI run (4 breath holds, 20s duration, separated by paced breathing) was acquired in each session using a gradient-echo echo planar sequence (TR/TE = 1500/26 msec; 20cm FOV; 64 64 matrix; 34 slices; 3.8mm thickness; 117 volumes). PsychoPy software was used to cue the task with instructions presented via a fiber optic-goggle system (NordicNeuroLab). A respiration belt was used to monitor task compliance. A T1-weighted anatomical was acquired using a 3D spoiled gradient echo sequence (TR/TE 5.758ms/2.032ms, flip angle=73, 1mm isotropic resolution).

5.1.3 Data Processing and Statistical Analysis

Cumulative number of head impacts, cPTA, and cPAA measurements were obtained for each soccer athlete. Due to large variance in cumulative load among the high school cohort, high school athletes were further divided into two equal groups reflecting the top (Top 50%) and bottom (Bottom 50%) 50th percentiles for cumulative load. In order to do this, each athlete's cumulative peak translational acceleration and cumulative peak angular acceleration were summed after being normalized using the corresponding median. The Top 50% group was defined as those athletes sustaining a cumulative load above the group median while the Bottom 50% group was defined as those athletes sustaining a cumulative load below the group median. Grouping was done at each follow-up session to reflect changes in cumulative exposure as the season progressed. Of the fourteen athletes, 4 remained in the Top 50% group at every follow-up session and 4 remained in the Bottom 50% group at every follow-up

session. The remaining 6 athletes fluctuated between the two groups as the season progressed.

FMRI data were analyzed using a processing stream adapted from afni_proc.py including slice timing correction, motion correction, spatial smoothing, alignment to the structural scan, normalization to Talairach space, and conversion to percent signal change. Additionally, the FAST automated segmentation tool in FSL was used to create a gray matter (GM) mask for each subject. For each subject-session, the mean fMRI time series across all gray matter (GM) voxels was calculated. This series was regressed against the task time series calculated from the session-specific respiratory belt time series. The B-coefficient of the breath hold regressor was used as the CVR metric.

For all statistical tests, a significance level of $p < 0.05$ was used. To compare cumulative load sustained in a season between high school and collegiate soccer athletes, t-tests were conducted on number of head impacts per player, cumulative PTA, and cumulative PRA. After division, the Top 50% group and Bottom 50% group of high school athletes, at the end of the season, were also compared to collegiate athletes using a t-test.

For high school athletes, fMRI results from Top 50% group and Bottom 50% group were analyzed separately at each session. College soccer athletes were analyzed as one group. To find sessions deviating from Pre, the distribution of B-coefficients at each follow-up session (In1, In2, Post) was compared to the distribution of B-coefficients at Pre using a Wilcoxon signed rank test followed by false discovery rate correction. Finally, ΔB-weight (session - reference) was calculated to assess whether deviance from reference reflected subject-wise deviation of CVR.

5.2 Results

The cumulative load sustained by the cohort of athletes is detailed below (Table 5.1, Fig. 5.1). Overall, there was a significant difference between the cumulative load

sustained by collegiate soccer athletes and the cumulative load sustained by high school soccer athletes (head impacts per athlete: $p < 0.003$, cPTA: $p < 0.003$, cPAA: $p < 0.02$). There was also a significant difference in the cumulative load sustained by collegiate athletes and the cumulative load sustained by high school athletes in the Bottom 50% group with respect to all three cumulative loading measures (head impacts per athlete, cPTA, cPAA: $p<0.001$). In contrast, there was no significant difference in cumulative load sustained by collegiate athletes and cumulative load sustained by high school athletes in the Top 50% group for any cumulative loading metric.

Controls showed no significant change in CVR between baseline and follow-up. The ΔB-weight calculations showed a decrease in CVR in 5 of 8 controls at the follow-up session (Fig 5.2). Pairwise comparisons in the Bottom 50% group also showed no significant change from Pre at any follow-up session. The ΔB-weight calculations for the Bottom 50% group showed a decrease in CVR in 3 of 7 athletes at In1, 4 of 7 athletes at In2, 4 of 7 athletes at Post1, and 5 of 7 athletes at Post2. In contrast, pairwise comparisons in the Top 50% group showed significant CVR reductions from Pre at In1, In2, and Post1 ($pfdr < 0.05$) that were no longer present at Post2. The ΔB-weight calculations for the Top 50% group showed a decrease in CVR in 6 of 7 athletes at In1, 6 of 7 athletes at In2, 7 of 7 athletes at Post1, and 3 of 7 athletes at Post2. Collegiate athletes did not show a significant difference in CVR between Pre and Post1. The ΔB-weight calculations for collegiate athletes showed a decrease in CVR in 5 of 8 athletes at Post1.

5.3 Discussion

High school soccer athletes, as a whole, as well as soccer athletes in the Bottom 50% group experienced cumulative loads that were significantly lower than those of collegiate soccer athletes. High school soccer athletes in the Top 50% group, on the other hand, experienced cumulative loads on par with those of collegiate soccer

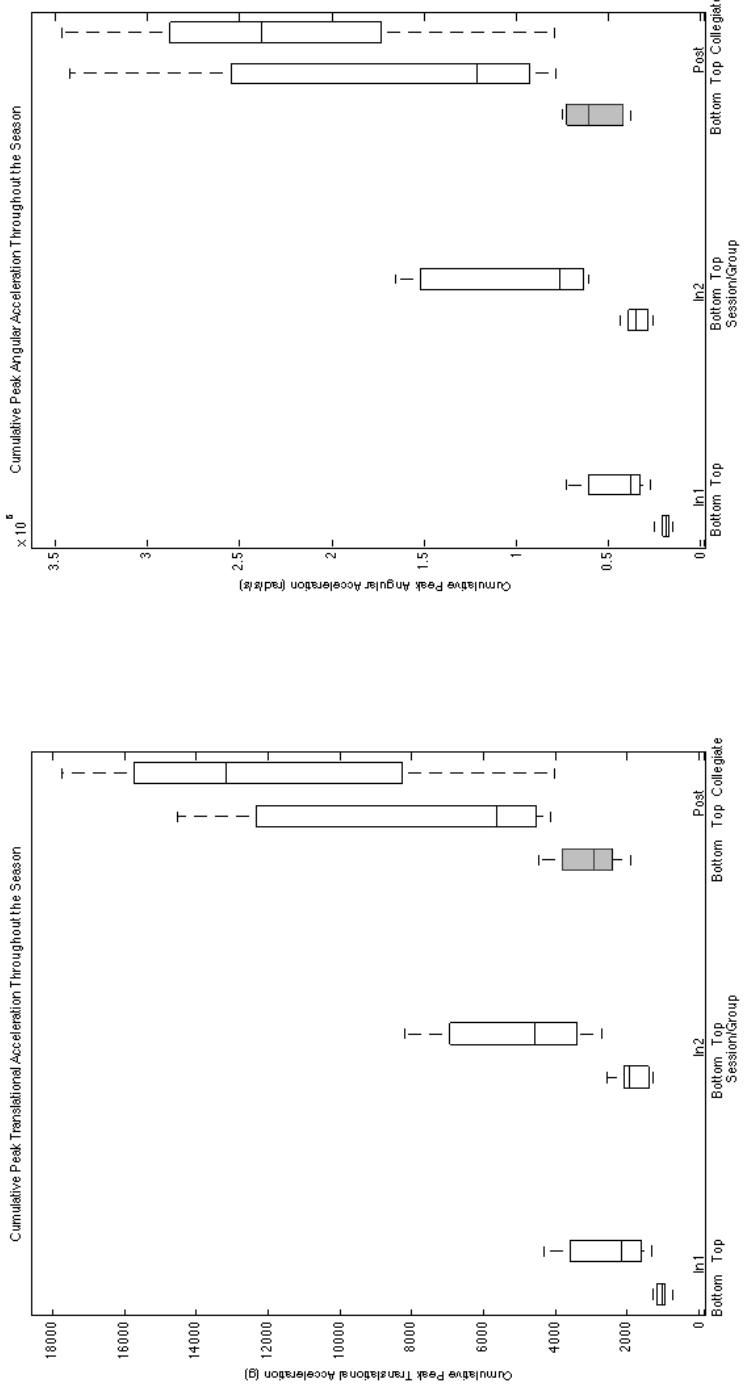


Fig. 5.1. The cumulative peak translational and peak angular accelerations throughout the season. Shading indicates significant differences from the collegiate and Top 50% groups ($p<0.001$).

Table 5.1.
The average and cumulative translational and angular accelerations sustained by the different groups throughout one season of play.

	Total Hits	Impacts/ Player/ Season	PTA(g)	PAA(rad/s^2)	cPTA (g)	cPAA (rad/s^2)
			M ean [95% Confidence Interval]			
High School						
Top 50%	1402	200	39.5	8,251.3	7,920	1,685,514
			[35.3; 43.7]	[7224.8; 9277.8]	[3,819; 12,022]	[726,750; 2,644,279]
Bottom 50%	589	84	36.3	6862.7	3,075	579,116
			[31.5; 41.1]	[5978.6; 7746.9]	[2,236; 3,915]	[431,538; 726,694]
Overall	1991	142	37.9	7557.0	5,498	1,132,315
			[35.0; 40.8]	[6847.6; 8266.4]	[3,204; 7,791]	[603,975; 1,660,655]
Collegiate						
Overall	2586	323	37.6	7237.5	12,000.60	2,274,604.80
			[34.2; 41.0]	[6337.2; 8137.67]	[7,900.1; 16,101.0]	[1,526,728.1; 3,022,481.6]

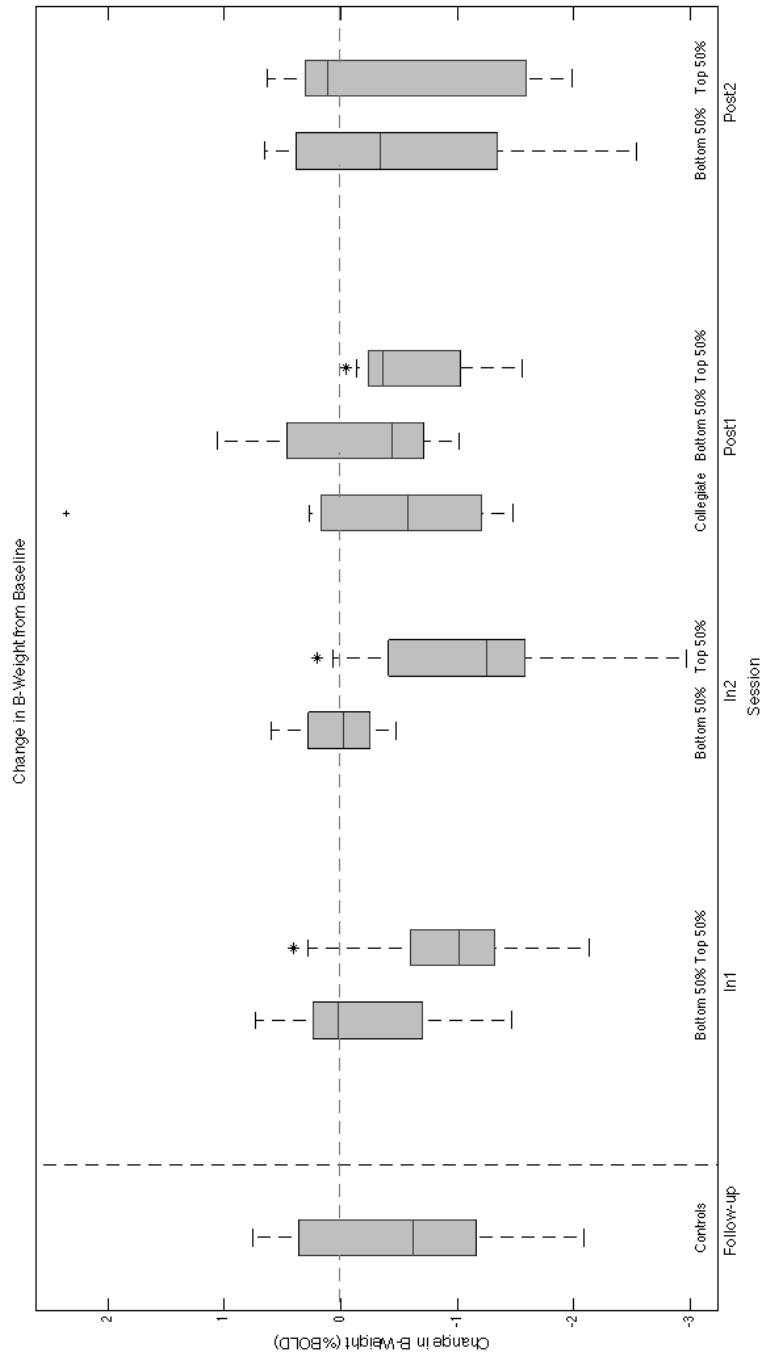


Fig. 5.2. Distributions showing change in GM B-weight female non-collision sport athletes as well as soccer athletes in the top 50 percentile for cumulative hit exposure. Sessions deviating significantly (pfdr < 0.05: Wilcoxon signed rank test) from pre-season are indicated by asterisks above the distribution.

athletes. Although there were no significant differences in cumulative load per player between collegiate athletes and high school athletes in the Top 50% group, it is important to note that high school athletes experienced cumulative loading over a 10-12 week season while collegiate athletes experienced cumulative loading over a 14 week season.

Other considerations include the method of division for the Top and Bottom 50% of high school athletes. The high school athletes were sorted based on a combination of cumulative loading from peak translational and peak angular accelerations. The Top and Bottom 50% could have been sorted based on cPTA or cPAA individually, however this only changed one player in one session so the results would stay largely the same.

As expected, control athletes showed no significant change between reference and follow-up sessions. Similarly, soccer athletes in the Bottom 50% group did not experience significant CVR changes, relative to pre-season, at any follow-up session. In contrast, female soccer athletes in the Top 50% group experienced significant CVR decreases, relative to pre-season, at the onset of the contact season that persisted 1 month after the contact season but recovered by 5 months after the contact season. Collegiate soccer athletes did not show a significant CVR change from Pre to Post1.

There was no significant change in CVR over the season for non-collision sport control athletes as well as high school soccer athletes in the Bottom 50% group. The Top 50% group sustained loads on par with those of collegiate athletes over a period of 10-12 weeks and showed a significant decline in CVR. In contrast, collegiate soccer athletes did not show significant changes in CVR after loading over a period of 14 weeks. This discrepancy may demonstrate the damaging effects of high loading over a short period of time. However, the lack of changes in collegiate athletes could also be indicative physical changes associated with older, more experienced athletes increasing their tolerance for mechanical loading of the brain. Along with an increase in tolerance, collegiate players do not experience much of an off-season compared to

high school athletes, indicating that baseline scans are done when the players are already physically active and involved in training.

This work is some of the first to show neurophysiologic changes in female soccer athletes attributable to repetitive head trauma, and suggests that reducing cumulative PTA and PAA exposure over a season may help prevent chronic neurological deficits in athletes who participate in collision sports.

6. CONCLUSIONS

The impacts sustained by high school and collegiate female soccer athletes were tracked throughout a season of play using the xPatch. The data indicated that these athletes are sustaining high cumulative loading due to repetitive head impacts. It was shown that collegiate athletes generally sustain impacts of higher magnitudes and take more hits than high school athletes, leading to a higher cumulative loading. However, a subset of high school athletes sustain loading on par with collegiate athletes, during a season that is 2-4 weeks shorter than that of the collegiate athletes. The subset undergoing high cumulative loading, Top 50%, also shows significant decreases in cerebrovascular reactivity indicating neurophysiologic changes in the absence of concussion. The collegiate athletes did not show changes in cerebrovascular reactivity despite undergoing cumulative loading on par with the Top 50% possibly due to the shorter season, a shorter recovery time compared to high school athletes, or better technique and preparation that comes with experience.

To eliminate neurophysiologic effects, the cumulative loading sustained throughout a season of play should be decreased. This can be obtained reducing the number of days the athletes participate in heading drills and therefore reducing the number of impacts sustained by the athletes. A second method of decreasing cumulative loading would be to decrease the average magnitude per hit. This can be done by decreasing the rigidity of ball, for example utilize a "training" ball, for use during technical heading drills. Protective equipment could also be introduced. It should be noted, however, that some form of assessment such as MRI-based imaging methods or detailed neurocognitive batteries will be required to determine whether these measures go far enough toward reducing the overall risk of neurological damage.

6.1 Limitations and Future Work

More players should be tracked and scanned throughout a season of play to ensure the effects seen here are widespread and consistent throughout the soccer population. A larger study could be used to verify the results seen here, after which predictive models should be used to determine the threshold as which damage occurs and at which point the athletes do not recover from the damage. Following verification of the neurophysiologic effects of heading, changes, such as the ones suggested above, should be implemented. The effectiveness of any form of intervention should also be examined to determine if there is a decrease in cumulative loading sustained and also to determine if the neurophysiologic changes are still present. The end goal is to ensure all soccer athletes can participate safely without seeing the any short or long term effects on neurophysiologic health.

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APPENDICES

A. CONCUSSION EVALUATION FORMS

ACUTE CONCUSSION EVALUATION (ACE)
PHYSICIAN/CLINICIAN OFFICE VERSION

Gerard Gioia, PhD[1] & Micky Collins, PhD[2]
[1]Children's National Medical Center
[2]University of Pittsburgh Medical Center

Patient Name:_____
DOB: _____ Age:_____
Date:_____ ID/MR#_____

A. Injury Characteristics **Date/Time of Injury**_____**Reporter: __Patient __Parent __Spouse __Other**_____

1. Injury Description _____

1a. Is there evidence of a forcible blow to the head (direct or indirect)? __Yes __No __Unknown
1b. Is there evidence of intracranial injury or skull fracture? __Yes __No __Unknown
1c. Location of Impact: __Frontal __Lft Temporal __Rt Temporal __Lft Parietal __Rt Parietal __Occipital __Neck __Indirect Force
2. Cause: __MVC __Pedestrian-MVC __Fall __Assault __Sports (*specify)*_____Other_____
3. Amnesia Before (Retrograde) Are there any events just BEFORE the injury that you/ person has no memory of (even brief)? __ Yes __No Duration
4. Amnesia After (Anterograde) Are there any events just AFTER the injury that you/ person has no memory of (even brief)? __ Yes __No Duration
5. Loss of Consciousness: Did you/ person lose consciousness? __ Yes __No Duration
6. EARLY SIGNS: __Appears dazed or stunned __Is confused about events __Answers questions slowly __Repeats Questions __Forgetful (recent info)
7. Seizures: Were seizures observed? No__ Yes___ Detail_____

B. Symptom Check List* Since the injury, has the person experienced any of these symptoms any more than usual today or in the past day?
Indicate presence of each symptom (0=No, 1=Yes). ***Lovell & Collins, 1998 JHTR***

PHYSICAL (10)		COGNITIVE (4)		SLEEP (4)	
Headache	0 1	Feeling mentally foggy	0 1	Drowsiness	0 1
Nausea	0 1	Feeling slowed down	0 1	Sleeping less than usual	0 1 N/A
Vomiting	0 1	Difficulty concentrating	0 1	Sleeping more than usual	0 1 N/A
Balance problems	0 1	Difficulty remembering	0 1	Trouble falling asleep	0 1 N/A
Dizziness	0 1	**COGNITIVE Total (0-4)** _____		**SLEEP Total (0-4)** _____	
Visual problems	0 1	**EMOTIONAL (4)**			
Fatigue	0 1	Irritability	0 1		
Sensitivity to light	0 1	Sadness	0 1		
Sensitivity to noise	0 1	More emotional	0 1		
Numbness/Tingling	0 1	Nervousness	0 1		
PHYSICAL Total (0-10) _____		**EMOTIONAL Total (0-4)** _____			
(Add Physical, Cognitive, Emotion, Sleep totals) Total Symptom Score (0-22)		_____			

Exertion: Do these symptoms worsen with:
Physical Activity __Yes __No __N/A
Cognitive Activity __Yes __No __N/A

Overall Rating: How different is the person acting compared to his/her usual self? (circle)
Normal 0 1 2 3 4 5 6 Very Different

C. Risk Factors for Protracted Recovery *(check all that apply)*

Concussion History? Y ___ N___	√	Headache History? Y ___ N___	√	Developmental History	√	Psychiatric History
Previous # 1 2 3 4 5 6+		Prior treatment for headache		Learning disabilities		Anxiety
Longest symptom duration Days__ Weeks__ Months__ Years__		History of migraine headache __ Personal __ Family_____		Attention-Deficit/ Hyperactivity Disorder		Depression
						Sleep disorder
If multiple concussions, less force caused reinjury? Yes__ No__				Other developmental disorder_____		Other psychiatric disorder _____

List other comorbid medical disorders or medication usage (e.g., hypothyroid, seizures)_____

D. RED FLAGS for acute emergency management: Refer to the emergency department with sudden onset of any of the following:

* Headaches that worsen	* Looks very drowsy/ can't be awakened	* Can't recognize people or places	* Neck pain
* Seizures	* Repeated vomiting	* Increasing confusion or irritability	* Unusual behavioral change
* Focal neurologic signs	* Slurred speech	* Weakness or numbness in arms/legs	* Change in state of consciousness

E. Diagnosis (ICD): __Concussion w/o LOC 850.0 __Concussion w/ LOC 850.1 __Concussion (Unspecified) 850.9 __Other (854) _____
__No diagnosis

F. Follow-Up Action Plan **Complete *ACE Care Plan* and provide copy to patient/family.**
___ **No Follow-Up Needed**
___ **Physician/Clinician Office Monitoring**: Date of next follow-up _____
___ **Referral:**
___ Neuropsychological Testing
___ Physician: Neurosurgery____ Neurology____ Sports Medicine____ Physiatrist____ Psychiatrist____ Other_____
___ Emergency Department

ACE Completed by:_____

This form is part of the "Heads Up: Brain Injury in Your Practice" tool kit developed by the Centers for Disease Control and Prevention (CDC).

A concussion (or mild traumatic brain injury (MTBI)) is a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces secondary to direct or indirect forces to the head. Disturbance of brain function is related to neurometabolic dysfunction, rather than structural injury, and is typically associated with normal structural neuroimaging findings (i.e., CT scan, MRI). Concussion may or may not involve a loss of consciousness (LOC). Concussion results in a constellation of physical, cognitive, emotional, and sleep-related symptoms. Symptoms may last from several minutes to days, weeks, months or even longer in some cases.

ACE Instructions

The ACE is intended to provide an evidence-based clinical protocol to conduct an initial evaluation and diagnosis of patients (both children and adults) with known or suspected MTBI. The research evidence documenting the importance of these components in the evaluation of an MTBI is provided in the reference list.

A. Injury Characteristics:

1. Obtain **description of the injury** – how injury occurred, type of force, location on the head or body (if force transmitted to head). Different biomechanics of injury may result in differential symptom patterns (e.g., occipital blow may result in visual changes, balance difficulties).
2. Indicate the **cause of injury**. Greater forces associated with the trauma are likely to result in more severe presentation of symptoms.

3/4. **Amnesia:** Amnesia is defined as the failure to form new memories. Determine whether amnesia has occurred and attempt to determine length of time of memory dysfunction – before (retrograde) and after (anterograde) injury. Even seconds to minutes of memory loss can be predictive of outcome. Recent research has indicated that amnesia may be up to 4-10 times more predictive of symptoms and cognitive deficits following concussion than is LOC (less than 1 minute).[1]

5. **Loss of consciousness (LOC)** – If occurs, determine length of LOC.
6. **Early signs.** If present, ask the individuals who know the patient (parent, spouse, friend, etc) about specific signs of the concussion that may have been observed. These signs are typically observed early after the injury.
7. Inquire whether **seizures** were observed or not.

B. Symptom Checklist: [2]

1. Ask patient (and/or parent, if child) to report presence of the four categories of symptoms since injury. It is important to assess all listed symptoms as different parts of the brain control different functions. One or all symptoms may be present depending upon mechanisms of injury.[3] Record "1" for Yes or "0" for No for their presence or absence, respectively.
2. For all symptoms, indicate presence of symptoms as experienced within the past 24 hours. Since symptoms can be present premorbidly/at baseline (e.g., inattention, headaches, sleep, sadness), it is important to assess change from their usual presentation.
3. **Scoring**: Sum total number of symptoms present per area, and sum all four areas into Total Symptom Score (score range 0-22). (Note: most sleep symptoms are only applicable after a night has passed since the injury. Drowsiness may be present on the day of injury.) If symptoms are new and present, there is no lower limit symptom score. Any score > 0 indicates positive symptom history.
4. **Exertion:** Inquire whether any symptoms worsen with physical (e.g., running, climbing stairs, bike riding) and/or cognitive (e.g., academic studies, multi-tasking at work, reading or other tasks requiring focused concentration) exertion. Clinicians should be aware that symptoms will typically worsen or re-emerge with exertion, indicating incomplete recovery. Over-exertion may protract recovery.
5. **Overall Rating:** Determine how different the person is acting from their usual self. Circle "0" (Normal) to "6" (Very Different).

C. Risk Factors for Protracted Recovery: Assess the following risk factors as possible complicating factors in the recovery process.

1. **Concussion history:** Assess the number and date(s) of prior concussions, the duration of symptoms for each injury, and whether less biomechanical force resulted in re-injury. Research indicates that cognitive and symptom effects of concussion may be cumulative, especially if there is minimal duration of time between injuries and less biomechanical force results in subsequent concussion (which may indicate incomplete recovery from initial trauma).[4-8]
2. **Headache history:** Assess personal and/or family history of diagnosis/treatment for headaches. Research indicates headache (migraine in particular) can result in protracted recovery from concussion.[8-11]
3. **Developmental history**: Assess history of learning disabilities, Attention-Deficit/Hyperactivity Disorder or other developmental disorders. Research indicates that there is the possibility of a longer period of recovery with these conditions.[12]
4. **Psychiatric history**: Assess for history of depression/mood disorder, anxiety, and/or sleep disorder.[13-16]

D. Red Flags: The patient should be carefully observed over the first 24-48 hours for these serious signs. Red flags are to be assessed as possible signs of deteriorating neurological functioning. Any positive report should prompt strong consideration of referral for emergency medical evaluation (e.g. CT Scan to rule out intracranial bleed or other structural pathology).[17]

E. Diagnosis: The following ICD diagnostic codes may be applicable.

850.0 (Concussion, with no loss of consciousness) – Positive injury description with evidence of forcible direct/ indirect blow to the head (A1a); plus evidence of active symptoms (B) of any type and number related to the trauma (Total Symptom Score >0); no evidence of LOC (A5), skull fracture or intracranial injury (A1b).

850.1 (Concussion, with brief loss of consciousness < 1 hour) – Positive injury description with evidence of forcible direct/ indirect blow to the head (A1a); plus evidence of active symptoms (B) of any type and number related to the trauma (Total Symptom Score >0); positive evidence of LOC (A5), skull fracture or intracranial injury (A1b).

850.9 (Concussion, unspecified) – Positive injury description with evidence of forcible direct/ indirect blow to the head (A1a); plus evidence of active symptoms (B) of any type and number related to the trauma (Total Symptom Score >0); unclear/unknown injury details; unclear evidence of LOC (A5), no skull fracture or intracranial injury.

Other Diagnoses – If the patient presents with a positive injury description and associated symptoms, but additional evidence of intracranial injury (A 1b) such as from neuroimaging, a moderate TBI and the diagnostic category of 854 (Intracranial injury) should be considered.

F. Follow-Up Action Plan: Develop a follow-up plan of action for symptomatic patients. The physician/clinician may decide to (1) monitor the patient in the office or (2) refer them to a specialist. Serial evaluation of the concussion is critical as symptoms may resolve, worsen, or ebb and flow depending upon many factors (e.g., cognitive/physical exertion, comorbidities). Referral to a specialist can be particularly valuable to help manage certain aspects of the patient's condition. (Physician/Clinician should also complete the ACE Care Plan included in this tool kit.)

1. **Physician/Clinician serial monitoring –** Particularly appropriate if number and severity of symptoms are steadily decreasing over time and/or fully resolve within 3-5 days. If steady reduction is not evident, referral to a specialist is warranted.
2. **Referral to a specialist –** Appropriate if symptom reduction is not evident in 3-5 days, or sooner if symptom profile is concerning in type/severity.
   - Neuropsychological Testing can provide valuable information to help assess a patient's brain function and impairment and assist with treatment planning, such as return to play decisions.
   - Physician Evaluation is particularly relevant for medical evaluation and management of concussion. It is also critical for evaluating and managing focal neurologic, sensory, vestibular, and motor concerns. It may be useful for medication management (e.g., headaches, sleep disturbance, depression) if post-concussive problems persist.

SCAT3™

Sport Concussion Assessment Tool – 3rd Edition

For use by medical professionals only

Name | Date/Time of Injury: | Date of Assessment: | Examiner:

What is the SCAT3?[1]

The SCAT3 is a standardized tool for evaluating injured athletes for concussion and can be used in athletes aged from 13 years and older. It supersedes the original SCAT and the SCAT2 published in 2005 and 2009, respectively[2]. For younger persons, ages 12 and under, please use the Child SCAT3. The SCAT3 is designed for use by medical professionals. If you are not qualified, please use the Sport Concussion Recognition Tool[1]. Preseason baseline testing with the SCAT3 can be helpful for interpreting post-injury test scores.

Specific instructions for use of the SCAT3 are provided on page 3. If you are not familiar with the SCAT3, please read through these instructions carefully. This tool may be freely copied in its current form for distribution to individuals, teams, groups and organizations. Any revision or any reproduction in a digital form requires approval by the Concussion in Sport Group.

NOTE: The diagnosis of a concussion is a clinical judgment, ideally made by a medical professional. The SCAT3 should not be used solely to make, or exclude, the diagnosis of concussion in the absence of clinical judgement. An athlete may have a concussion even if their SCAT3 is "normal".

What is a concussion?

A concussion is a disturbance in brain function caused by a direct or indirect force to the head. It results in a variety of non-specific signs and/or symptoms (some examples listed below) and most often does not involve loss of consciousness. Concussion should be suspected in the presence of **any one or more** of the following:

- Symptoms (e.g., headache), or
- Physical signs (e.g., unsteadiness), or
- Impaired brain function (e.g. confusion) or
- Abnormal behaviour (e.g., change in personality).

SIDELINE ASSESSMENT

Indications for Emergency Management

NOTE: A hit to the head can sometimes be associated with a more serious brain injury. Any of the following warrants consideration of activating emergency procedures and urgent transportation to the nearest hospital:

- Glasgow Coma score less than 15
- Deteriorating mental status
- Potential spinal injury
- Progressive, worsening symptoms or new neurologic signs

Potential signs of concussion?

If any of the following signs are observed after a direct or indirect blow to the head, the athlete should stop participation, be evaluated by a medical professional and **should not be permitted to return to sport the same day** if a concussion is suspected.

Any loss of consciousness?	☐ Y	☐ N
"If so, how long?" _____		
Balance or motor incoordination (stumbles, slow/laboured movements, etc.)?	☐ Y	☐ N
Disorientation or confusion (inability to respond appropriately to questions)?	☐ Y	☐ N
Loss of memory:	☐ Y	☐ N
"If so, how long?" _____		
"Before or after the injury?" _____		
Blank or vacant look:	☐ Y	☐ N
Visible facial injury in combination with any of the above:	☐ Y	☐ N

1 Glasgow coma scale (GCS)

Best eye response (E)	
No eye opening	1
Eye opening in response to pain	2
Eye opening to speech	3
Eyes opening spontaneously	4
Best verbal response (V)	
No verbal response	1
Incomprehensible sounds	2
Inappropriate words	3
Confused	4
Oriented	5
Best motor response (M)	
No motor response	1
Extension to pain	2
Abnormal flexion to pain	3
Flexion/Withdrawal to pain	4
Localizes to pain	5
Obeys commands	6
Glasgow Coma score (E + V + M)	of 15

GCS should be recorded for all athletes in case of subsequent deterioration.

2 Maddocks Score[3]

"I am going to ask you a few questions, please listen carefully and give your best effort."

Modified Maddocks questions (1 point for each correct answer)

What venue are we at today?	0	1
Which half is it now?	0	1
Who scored last in this match?	0	1
What team did you play last week/game?	0	1
Did your team win the last game?	0	1
Maddocks score		of 5

Maddocks score is validated for sideline diagnosis of concussion only and is not used for serial testing.

Notes: Mechanism of Injury ("tell me what happened"?):

Any athlete with a suspected concussion should be REMOVED FROM PLAY, medically assessed, monitored for deterioration (i.e., should not be left alone) and should not drive a motor vehicle until cleared to do so by a medical professional. No athlete diagnosed with concussion should be returned to sports participation on the day of Injury.

BACKGROUND

Name: _____ Date: _____

Examiner: _____

Sport/team/school: _____ Date/time of injury: _____

Age: _____ Gender: ☐ M ☐ F

Years of education completed: _____

Dominant hand: ☐ right ☐ left ☐ neither

How many concussions do you think you have had in the past? _____

When was the most recent concussion? _____

How long was your recovery from the most recent concussion? _____

Have you ever been hospitalized or had medical imaging done for a head injury? ☐ Y ☐ N

Have you ever been diagnosed with headaches or migraines? ☐ Y ☐ N

Do you have a learning disability, dyslexia, ADD/ADHD? ☐ Y ☐ N

Have you ever been diagnosed with depression, anxiety or other psychiatric disorder? ☐ Y ☐ N

Has anyone in your family ever been diagnosed with any of these problems? ☐ Y ☐ N

Are you on any medications? If yes, please list: ☐ Y ☐ N

_____

SCAT3 to be done in resting state. Best done 10 or more minutes post excercise.

SYMPTOM EVALUATION

3 How do you feel?

"You should score yourself on the following symptoms, based on how you feel now".

	none	mild		moderate		severe	
Headache	0	1	2	3	4	5	6
"Pressure in head"	0	1	2	3	4	5	6
Neck Pain	0	1	2	3	4	5	6
Nausea or vomiting	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Blurred vision	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling like "in a fog"	0	1	2	3	4	5	6
"Don't feel right"	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Fatigue or low energy	0	1	2	3	4	5	6
Confusion	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
Trouble falling asleep	0	1	2	3	4	5	6
More emotional	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervous or Anxious	0	1	2	3	4	5	6

Total number of symptoms (Maximum possible 22) _____

Symptom severity score (Maximum possible 132) _____

Do the symptoms get worse with physical activity? ☐ Y ☐ N

Do the symptoms get worse with mental activity? ☐ Y ☐ N

☐ self rated ☐ self rated and clinician monitored

☐ clinician interview ☐ self rated with parent input

Overall rating: If you know the athlete well prior to the injury, how different is the athlete acting compared to his/her usual self?

Please circle one response:

no different | very different | unsure | N/A

Scoring on the SCAT3 should not be used as a stand-alone method to diagnose concussion, measure recovery or make decisions about an athlete's readiness to return to competition after concussion. Since signs and symptoms may evolve over time, it is important to consider repeat evaluation in the acute assessment of concussion.

COGNITIVE & PHYSICAL EVALUATION

4 Cognitive assessment

Standardized Assessment of Concussion (SAC)[4]

Orientation (1 point for each correct answer)

What month is it?	0	1
What is the date today?	0	1
What is the day of the week?	0	1
What year is it?	0	1
What time is it right now? (within 1 hour)	0	1
Orientation score		of 5

Immediate memory

List	Trial 1		Trial 2		Trial 3		Alternative word list		
elbow	0	1	0	1	0	1	candle	baby	finger
apple	0	1	0	1	0	1	paper	monkey	penny
carpet	0	1	0	1	0	1	sugar	perfume	blanket
saddle	0	1	0	1	0	1	sandwich	sunset	lemon
bubble	0	1	0	1	0	1	wagon	iron	insect
Total									

Immediate memory score total of 15

Concentration: Digits Backward

List	Trial 1		Alternative digit list		
4-9-3	0	1	6-2-9	5-2-6	4-1-5
3-8-1-4	0	1	3-2-7-9	1-7-9-5	4-9-6-8
6-2-9-7-1	0	1	1-5-2-8-6	3-8-5-2-7	6-1-8-4-3
7-1-8-4-6-2	0	1	5-3-9-1-4-8	8-3-1-9-6-4	7-2-4-8-5-6
Total of 4					

Concentration: Month in Reverse Order (1 pt. for entire sequence correct)

Dec-Nov-Oct-Sept-Aug-Jul-Jun-May-Apr-Mar-Feb-Jan	0	1
Concentration score		of 5

5 Neck Examination:

Range of motion Tenderness Upper and lower limb sensation & strength

Findings: _____

6 Balance examination

Do one or both of the following tests.

Footwear (shoes, barefoot, braces, tape, etc.) _____

Modified Balance Error Scoring System (BESS) testing[5]

Which foot was tested (i.e. which is the **non-dominant** foot) ☐ Left ☐ Right

Testing surface (hard floor, field, etc.) _____

Condition

Double leg stance:	Errors
Single leg stance (non-dominant foot):	Errors
Tandem stance (non-dominant foot at back):	Errors

And/Or

Tandem gait[6,7]

Time (best of 4 trials): _____ seconds

7 Coordination examination

Upper limb coordination

Which arm was tested: ☐ Left ☐ Right

Coordination score of 1

8 SAC Delayed Recall[4]

Delayed recall score of 5

INSTRUCTIONS

Words in *Italics* throughout the SCAT3 are the instructions given to the athlete by the tester.

Symptom Scale

"You should score yourself on the following symptoms, based on how you feel now".

To be completed by the athlete. In situations where the symptom scale is being completed after exercise, it should still be done in a resting state, at least 10 minutes post exercise.
For total number of symptoms, maximum possible is 22.
For Symptom severity score, add all scores in table, maximum possible is 22 x 6 = 132.

SAC[4]

Immediate Memory

"I am going to test your memory. I will read you a list of words and when I am done, repeat back as many words as you can remember, in any order."

Trials 2 & 3:

"I am going to repeat the same list again. Repeat back as many words as you can remember in any order, even if you said the word before."

Complete all 3 trials regardless of score on trial 1 & 2. Read the words at a rate of one per second. **Score 1 pt. for each correct response.** Total score equals sum across all 3 trials. Do not inform the athlete that delayed recall will be tested.

Concentration

Digits backward

"I am going to read you a string of numbers and when I am done, you repeat them back to me backwards, in reverse order of how I read them to you. For example, if I say 7-1-9, you would say 9-1-7."

If correct, go to next string length. If incorrect, read trial 2. **One point possible for each string length**. Stop after incorrect on both trials. The digits should be read at the rate of one per second.

Months in reverse order

"Now tell me the months of the year in reverse order. Start with the last month and go backward. So you'll say December, November … Go ahead"

1 pt. for entire sequence correct

Delayed Recall

The delayed recall should be performed after completion of the Balance and Coordination Examination.

"Do you remember that list of words I read a few times earlier? Tell me as many words from the list as you can remember in any order."

Score 1 pt. for each correct response

Balance Examination

Modified Balance Error Scoring System (BESS) testing[5]

This balance testing is based on a modified version of the Balance Error Scoring System (BESS)[5]. A stopwatch or watch with a second hand is required for this testing.

"I am now going to test your balance. Please take your shoes off, roll up your pant legs above ankle (if applicable), and remove any ankle taping (if applicable). This test will consist of three twenty second tests with different stances."

(a) Double leg stance:

"The first stance is standing with your feet together with your hands on your hips and with your eyes closed. You should try to maintain stability in that position for 20 seconds. I will be counting the number of times you move out of this position. I will start timing when you are set and have closed your eyes."

(b) Single leg stance:

"If you were to kick a ball, which foot would you use? [This will be the dominant foot] Now stand on your non-dominant foot. The dominant leg should be held in approximately 30 degrees of hip flexion and 45 degrees of knee flexion. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes and return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."

(c) Tandem stance:

"Now stand heel-to-toe with your non-dominant foot in back. Your weight should be evenly distributed across both feet. Again, you should try to maintain stability for 20 seconds with your hands on your hips and your eyes closed. I will be counting the number of times you move out of this position. If you stumble out of this position, open your eyes and return to the start position and continue balancing. I will start timing when you are set and have closed your eyes."

Balance testing – types of errors

1. Hands lifted off iliac crest
2. Opening eyes
3. Step, stumble, or fall
4. Moving hip into > 30 degrees abduction
5. Lifting forefoot or heel
6. Remaining out of test position > 5 sec

Each of the 20-second trials is scored by counting the errors, or deviations from the proper stance, accumulated by the athlete. The examiner will begin counting errors only after the individual has assumed the proper start position. **The modified BESS is calculated by adding one error point for each error during the three 20-second tests. The maximum total number of errors for any single condition is 10.** If a athlete commits multiple errors simultaneously, only one error is recorded but the athlete should quickly return to the testing position, and counting should resume once subject is set. Subjects that are unable to maintain the testing procedure for a minimum of **five seconds** at the start are assigned the highest possible score, ten, for that testing condition.

OPTION: For further assessment, the same 3 stances can be performed on a surface of medium density foam (e.g., approximately 50 cm x 40 cm x 6 cm).

Tandem Gait[6,7]

Participants are instructed to stand with their feet together behind a starting line (the test is best done with footwear removed). Then, they walk in a forward direction as quickly and as accurately as possible along a 38mm wide (sports tape), 3 meter line with an alternate foot heel-to-toe gait ensuring that they approximate their heel and toe on each step. Once they cross the end of the 3m line, they turn 180 degrees and return to the starting point using the same gait. A total of 4 trials are done and the best time is retained. Athletes should complete the test in 14 seconds. Athletes fail the test if they step off the line, have a separation between their heel and toe, or if they touch or grab the examiner or an object. In this case, the time is not recorded and the trial repeated, if appropriate.

Coordination Examination

Upper limb coordination
Finger-to-nose (FTN) task:

"I am going to test your coordination now. Please sit comfortably on the chair with your eyes open and your arm (either right or left) outstretched (shoulder flexed to 90 degrees and elbow and fingers extended), pointing in front of you. When I give a start signal, I would like you to perform five successive finger to nose repetitions using your index finger to touch the tip of the nose, and then return to the starting position, as quickly and as accurately as possible."

Scoring: 5 correct repetitions in < 4 seconds = 1
Note for testers: Athletes fail the test if they do not touch their nose, do not fully extend their elbow or do not perform five repetitions. **Failure should be scored as 0.**

References & Footnotes

1. This tool has been developed by a group of international experts at the 4th International Consensus meeting on Concussion in Sport held in Zurich, Switzerland in November 2012. The full details of the conference outcomes and the authors of the tool are published in The BJSM Injury Prevention and Health Protection, 2013, Volume 47, Issue 5. The outcome paper will also be simultaneously co-published in other leading biomedical journals with the copyright held by the Concussion in Sport Group, to allow unrestricted distribution, providing no alterations are made.

2. McCrory P et al., Consensus Statement on Concussion in Sport – the 3rd International Conference on Concussion in Sport held in Zurich, November 2008. British Journal of Sports Medicine 2009; 43: i76-89.

3. Maddocks, DL; Dicker, GD; Saling, MM. The assessment of orientation following concussion in athletes. Clinical Journal of Sport Medicine. 1995; 5(1): 32–3.

4. McCrea M. Standardized mental status testing of acute concussion. Clinical Journal of Sport Medicine. 2001; 11: 176–181.

5. Guskiewicz KM. Assessment of postural stability following sport-related concussion. Current Sports Medicine Reports. 2003; 2: 24–30.

6. Schneiders, A.G., Sullivan, S.J., Gray, A., Hammond-Tooke, G. & McCrory, P. Normative values for 16-37 year old subjects for three clinical measures of motor performance used in the assessment of sports concussions. Journal of Science and Medicine in Sport. 2010; 13(2): 196–201.

7. Schneiders, A.G., Sullivan, S.J., Kvarnstrom. J.K., Olsson, M., Yden. T. & Marshall, S.W. The effect of footwear and sports-surface on dynamic neurological screening in sport-related concussion. Journal of Science and Medicine in Sport. 2010; 13(4): 382–386

ATHLETE INFORMATION

Any athlete suspected of having a concussion should be removed from play, and then seek medical evaluation.

Signs to watch for

Problems could arise over the first 24–48 hours. The athlete should not be left alone and must go to a hospital at once if they:

- Have a headache that gets worse
- Are very drowsy or can't be awakened
- Can't recognize people or places
- Have repeated vomiting
- Behave unusually or seem confused; are very irritable
- Have seizures (arms and legs jerk uncontrollably)
- Have weak or numb arms or legs
- Are unsteady on their feet; have slurred speech

Remember, it is better to be safe.
Consult your doctor after a suspected concussion.

Return to play

Athletes should not be returned to play the same day of injury.
When returning athletes to play, they should be **medically cleared and then follow a stepwise supervised program,** with stages of progression.

For example:

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

There should be at least 24 hours (or longer) for each stage and if symptoms recur the athlete should rest until they resolve once again and then resume the program at the previous asymptomatic stage. Resistance training should only be added in the later stages.

If the athlete is symptomatic for more than 10 days, then consultation by a medical practitioner who is expert in the management of concussion, is recommended.

Medical clearance should be given before return to play.

Scoring Summary:

Test Domain	Score		
	Date:	Date:	Date:
Number of Symptoms of 22			
Symptom Severity Score of 132			
Orientation of 5			
Immediate Memory of 15			
Concentration of 5			
Delayed Recall of 5			
SAC Total			
BESS (total errors)			
Tandem Gait (seconds)			
Coordination of 1			

Notes:

CONCUSSION INJURY ADVICE

(To be given to the **person monitoring** the concussed athlete)

This patient has received an injury to the head. A careful medical examination has been carried out and no sign of any serious complications has been found. Recovery time is variable across individuals and the patient will need monitoring for a further period by a responsible adult. Your treating physician will provide guidance as to this timeframe.

If you notice any change in behaviour, vomiting, dizziness, worsening headache, double vision or excessive drowsiness, please contact your doctor or the nearest hospital emergency department immediately.

Other important points:

- Rest (physically and mentally), including training or playing sports until symptoms resolve and you are medically cleared
- No alcohol
- No prescription or non-prescription drugs without medical supervision. Specifically:
  - No sleeping tablets
  - Do not use aspirin, anti-inflammatory medication or sedating pain killers
- Do not drive until medically cleared
- Do not train or play sport until medically cleared

Clinic phone number

Patient's name

Date/time of injury

Date/time of medical review

Treating physician

Contact details or stamp

B. IMPACT

Table B.1.: The average and cumulative translational and angular accelerations for each player, as well as the number of flags received on ImPACT. Acceleration values for Post1 and Post2 are the same, showing acceleration data for one full season.

Player ID	**PTA (g)**	**PAA (rad/s2)**	**cPTA (g)**	**cPAA (rad/s2)**	**Flagged Impact**	**Δ B**
In1						
Bottom 50%						
3610	28.88	6013.92	721.90	150348.06	0	-1.474
5608	32.18	4468.07	1222.92	169786.81	1	0.729
5604	38.12	7638.45	914.89	183322.88	2	-0.866
5611	38.72	7171.27	1006.84	186452.93	1	-0.222
3608	28.90	5819.48	1011.65	203681.76	1	0.278
3603	28.65	5643.83	1060.00	208821.56	0	0.0866
5602	40.66	8074.70	1260.41	250315.74	0	0.019
Top 50%						
3602	36.02	7492.30	1296.83	269722.82	1	-0.825
5613	38.56	7359.74	1735.03	331188.31	1	0.273
5601	39.91	6193.87	2155.12	334468.78	0	-1.329
3609	33.21	8007.76	1560.72	376364.80	1	-2.129
3604	38.06	6885.13	3044.69	550810.74	1	-1.018
3605	35.78	5955.18	3756.81	625294.13	0	-1.307

Continued...

Player ID	PTA (g)	PAA (rad/s2)	cPTA (g)	cPAA (rad/s2)	Flagged Impact	Δ B
5617	43.55	7384.08	4311.09	731023.62	0	-0.523
In2						
Bottom 50%						
3608	28.49	5700.78	1282.11	256535.17	1	-0.268
5608	33.50	4925.21	1942.80	285662.43	1	-0.031
3610	31.10	6768.24	1337.10	291034.37	0	-0.219
3603	29.28	6493.08	1580.87	350626.51	2	0.591
5604	40.88	7811.80	1962.26	374966.63	1	-0.477
5613	38.63	7199.25	2124.42	395958.54	0	0.311
5611	39.38	6724.11	2559.88	437067.41	0	0.154
Top 50%						
3602	38.80	8714.03	2715.68	609981.90	1	-0.452
5601	38.62	6971.09	3437.11	620427.13	0	-0.405
5602	43.23	8713.27	3371.69	679634.85	0	-1.663
5617	43.46	7282.87	4562.94	764701.80	0	0.0604
3605	35.29	6441.61	6069.37	1107956.75	1	-1.255
3609	36.44	8349.06	7215.38	1653113.89	0	-1.356
3604	45.54	9197.13	8197.58	1655483.81	0	-2.965
Post1						
Bottom 50%						
5608	32.12	5064.72	2409.2	379853.87	0	0.301
3608	29.93	6257.86	1915.79	400502.99	1	-0.714
5604	41.18	7909.67	2470.67	474580.34	0	-0.709
5613	36.62	7573.97	2929.98	605917.51	0	-0.446
3610	32.42	6877.1	3404.53	722095.48	0	0.505
5601	37.81	6972.75	3932.06	725165.79	0	1.055

Continued...

Player ID	PTA (g)	PAA (rad/s2)	cPTA (g)	cPAA (rad/s2)	Flagged Impact	Δ B
5617	44.19	7383.12	4463.62	745694.68	0	-1.019
Top 50%						
5611	40.42	6976.72	4526.73	781392.89	0	-0.322
3602	39.4	8677.76	4136.53	911164.68	0	-1.031
3603	35.24	7644.51	4510.68	978497.28	0	-0.230
5602	40.69	8768.6	5614.85	1210066.65	0	-0.140
3605	36.7	7108.68	8623.53	1670540.85	0	-0.361
3604	48.54	10151.67	13542.99	2832314.56	0	-1.008
3609	35.77	8431.17	14487.35	3414624.14	0	-1.563
Collegiate						
2006	35.62	6992.62	4025.20	790166.50	1	-1.427
2017	38.20	7725.17	6073.63	1228301.70	0	-1.001
2001	36.51	7783.63	10442.30	2226117.84	0	-0.414
2009	41.75	7709.36	12651.32	2335937.53	0	2.345
2010	34.46	5784.46	14368.47	2412119.67	1	-0.755
2015	41.23	7823.47	13645.77	2589568.83	1	0.069
2012	30.88	5496.95	17722.83	3155251.87	0	-1.475
2003	42.37	8584.06	17074.91	3459374.72	0	0.266
Post2						
Bottom 50%						
5608	32.12	5064.72	2409.2	379853.87	0	-0.329
3608	29.93	6257.86	1915.79	400502.99	1	-1.583
5604	41.18	7909.67	2470.67	474580.34	0	-0.342
5613	36.62	7573.97	2929.98	605917.51	0	-2.540
3610	32.42	6877.1	3404.53	722095.48	1	0.652
5601	37.81	6972.75	3932.06	725165.79	0	0.611

Continued...

Player ID	**PTA (g)**	**PAA (rad/s2)**	**cPTA (g)**	**cPAA (rad/s2)**	**Flagged**	**Impact**	**Δ B**
5617	44.19	7383.12	4463.62	745694.68	0		-0.631
Top 50%							
5611	40.42	6976.72	4526.73	781392.89	1		0.333
3602	39.4	8677.76	4136.53	911164.68	1		-0.514
3603	35.24	7644.51	4510.68	978497.28	0		0.103
5602	40.69	8768.6	5614.85	1210066.65	1		0.620
3605	36.7	7108.68	8623.53	1670540.85	1		0.212
3604	48.54	10151.67	13542.99	2832314.56	0		-1.959
3609	35.77	8431.17	14487.35	3414624.14	2		-1.983

C. ADDITIONAL SENSOR PLOTS

The validation data for the sensors looked at the accelerations for the sensor compared to the accelerations for the head form in terms of PTA and PAA. However the PTA compared to the PAA for the sensors themselves could offer some insights into how the sensors work. For that reason the plot of sensor PTA versus sensor PAA is included here (Fig. C.1).

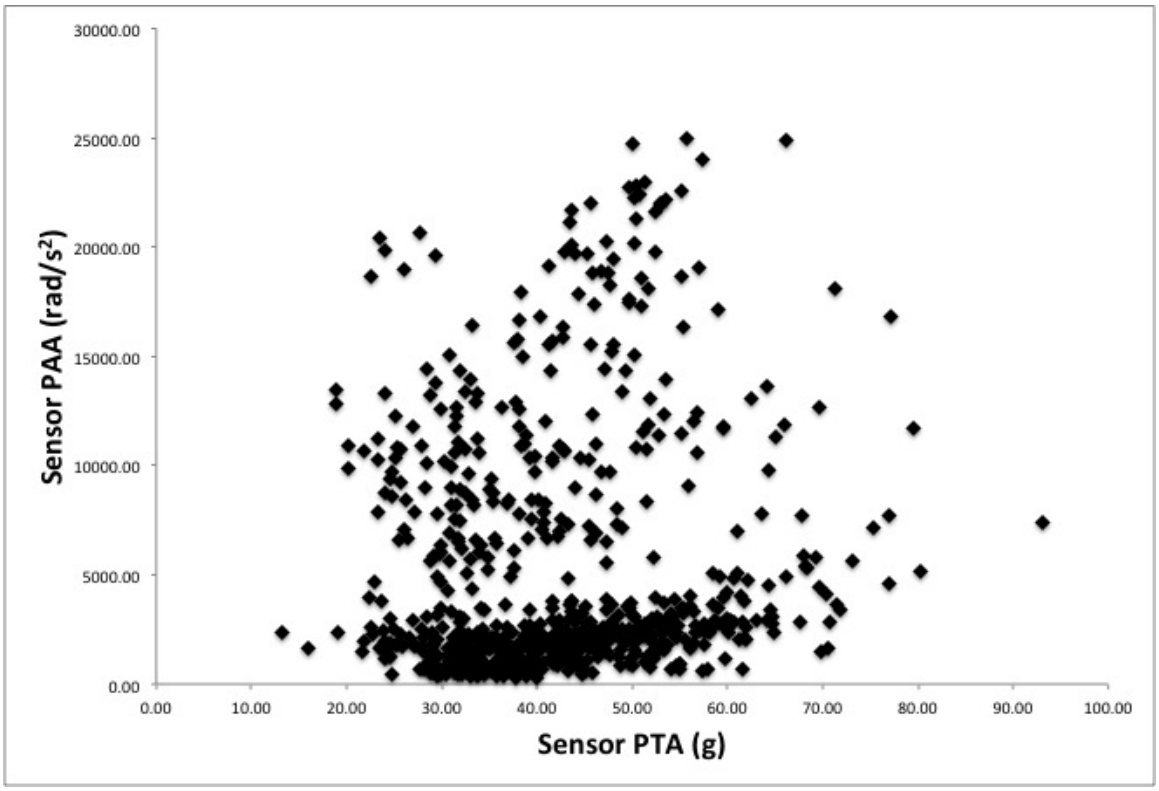

Fig. C.1. The PTA compared to the PAA for the x2 sensors tested on the Hybrid 3 head form.

D. ANALYSIS OF TECHNIQUE

The cumulative peak translational acceleration compared to the cumulative peak angular accelerations for the high school and collegiate populations might indicate a difference in technique. The players able to sustain higher PTA with lower PAA could have better technique or neck strength in order to be able to manage the loads better. The cumulative loading for the two populations is shown in the figure below (Fig. D.1).

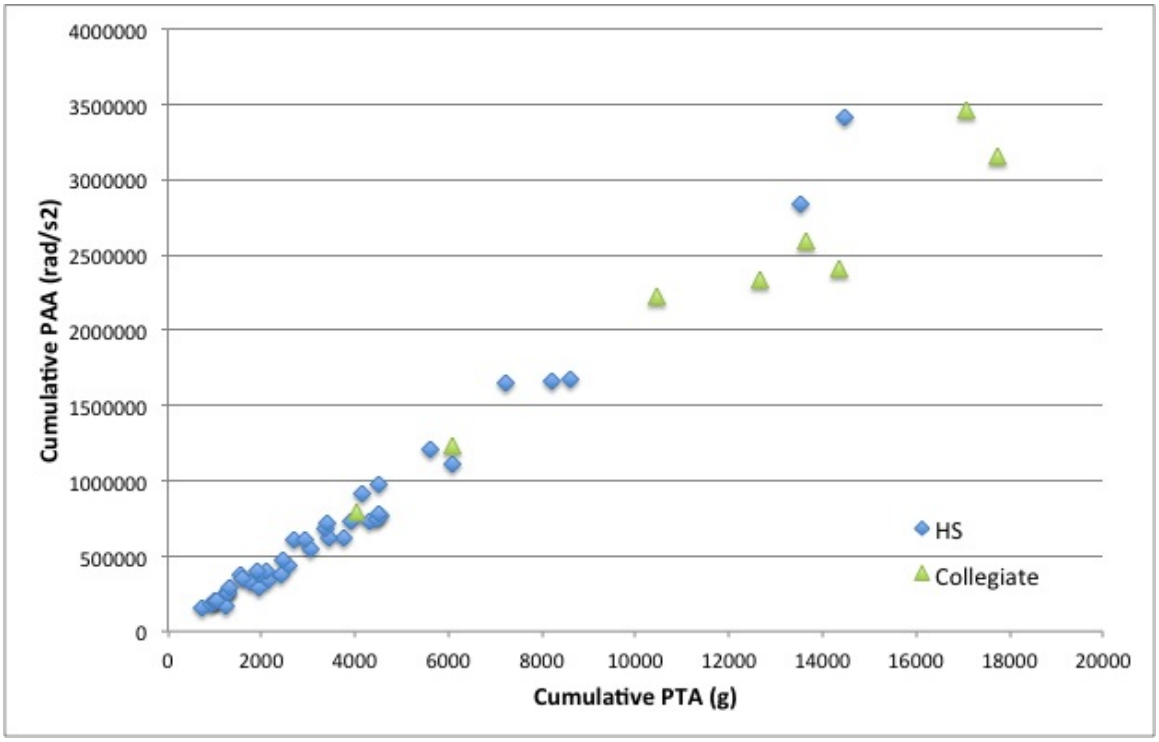

Fig. D.1. The cumulative PTA versus the cumulative PAA for the high school and collegiate populations. The collegiate players show lower cumulative PAA with high cumulative PTA.