# The Effects of Multiple Concussions in Youth & Adolescents

Cameron Krisko PA-S, B.Sc.

7669672

kriskocc@myumanitoba.ca

Supervisors: Dr. Ellis & Dr. Russell

A capstone project submitted to the Faculty of Graduate Studies of The University of Manitoba in partial fulfillment of the requirements for the degree of Master of Physician Assistant Studies

*Physician Assistant Studies, University of Manitoba, Winnipeg*

May 9, 2016

## Abstract

**Objective:** There were two goals of this study. 1) To determine if there was a difference between youth with a past history of a concussion and youth with no past history of a concussion in terms of number of symptoms and days to recovery. 2) To determine if there was a difference between youth with a past history of a concussion and youth with no past history of a concussion in terms of post concussion symptom score upon initial presentation as well as a subsequent diagnosis of post concussion syndrome.

**Methods:** A retrospective chart review was performed for all pediatric patients (between the ages seven and 19) referred to the Pan Am Concussion Program in Winnipeg, Manitoba, Canada between May 1, 2013 and May 1, 2015. Those patients referred for acute sport related concussions with complete medical records were included in this study. The institutional ethics review board at the University of Manitoba approved this study.

**Results:** A total of 306 participants met the inclusion criteria. The mean age of the participants presenting to the concussion clinic without a history of a concussion was 13.4 years and they were significantly younger than participants with a history of a concussion who had a mean age of 14.9 years ($p<0.0001$). Among those with no concussion history, 12.3% reported a loss of consciousness; where as 17.4% of those with a concussion history reported a loss of consciousness ($p=0.216$). For the participants with no past concussion history, the median number of recovery days was 22 days (IQR: 15-43) compared with 23 days (IQR: 16-39) for youth with a past concussion history ($p=0.41$). Those participants with no concussion history had significantly fewer concussion symptoms (median: 5.5; [IQR 1-10]) compared to those with a past

concussion history (median: 7.0; [IQR 2-14]; p=0.0366). The median post concussion symptom score was nine (IQR: 1-22) for those with no concussion history and 13 (IQR: 3-34) for those with a concussion history (p=0.0328). There was no significant difference in being subsequently diagnosed with post concussion syndrome between the two groups (no concussion history: 40.1%, concussion history: 41.7%, p=0.729).

**Conclusions:** Although there was a significantly higher median number of symptoms and higher post concussion symptom score in those with a past concussion history compared with those without, there was no statistical difference in the median recovery days or subsequent post concussion syndrome diagnosis. Therefore, having a history of a concussion does not appear to increase recovery time or increase the risk of developing post concussion syndrome.

## Table of Contents

## Introduction

### Background Information

A concussion, whether it is sport or non-sport related, is a form of traumatic brain injury that affects thousands of youth and adolescents across Canada every year (1). The 4th International Concussion Conference held in Zurich states that concussion is

> "A brain injury and is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces" (1).

A concussion may be caused either by direct contact to the head, face, neck or another part of the body with a subsequent force radiating to the head (1). A concussion will most often result in a quick onset of brief impairment of neurological function that resolves without intervention (1). The consequence of a concussion may be acute neurological changes, but the initial symptoms result from a temporary disturbance in brain function rather than a structural brain injury (1). The end product of a concussion is a spectrum of the number of symptoms, symptom severity, and length of recovery. This will vary from patient to patient (1).

### Pathophysiology

Multiple studies have shown that concussions are caused by a rapid rotational acceleration of the brain, however other mechanisms have been shown to cause concussions as well (2-3). In the majority of cases, the rotation is caused by direct contact with the head; however, contact can also be indirect (1-3). With direct contact, there does not appear to be any correlation with the location of contact and worsening outcomes. A specific example from American High School Football trainers noted that place of head

contact did not significantly predict the number of symptoms and duration of recovery (4).

Rotational acceleration of the brain causes a stressing of the neural elements, which leads to neuronal depolarization, local lactic acid build up and a decrease in blood flow to the brain (5-8). Neuronal depolarization leads to the opening of the sodium-potassium channels, which in turn results in a large movement of sodium into the cells and potassium out of the cells (5-8). This results in the depolarization of affected neurons, which in turn cause depolarization in downstream neurons (5-8). This spreading of depolarization is a similar mechanism to the way in which depression spreads in the brain (5-8). In order to operate the sodium-potassium pump, a large amount of energy or adenosine triphosphate is required (5,6,9,10). This increase in requirement of adenosine triphosphate results in an increase in glycolysis, ultimately resulting in a local lactate acid build up (5,6,9,10). Finally, during this sustained increase in energy demand there is also a decrease in blood flow delivery to the brain leading to a global metabolic energy crisis (7, 11). It is this mechanism that is attributed to the neurological dysfunction and symptoms of a concussion (7, 11).

## Epidemiology

The Center for Disease Prevention and Control estimate that up to 3.8 million traumatic brain injuries occur world wide each year and the large majority of these injuries are concussions (12). Specifically surrounding sport related concussions in youth; an observational study was conducted for American youth and American high school football players. In a total of 16,000 athletes polled, 10% of injuries sustained in these two groups were concussions (13). The incidence of concussions is most significant in

boys playing hockey, lacrosse or football. In girls, participating in soccer, lacrosse and field hockey leads to the highest incidence of a concussion (14-16). It is important to remember that youth concussions are not just sport related. Some non-sport mechanisms include motor vehicle accidents and falls (14-16). In terms of specific numbers, over 325,000 youth and adolescents worldwide are diagnosed with a concussion each year (17).

## Examination of Patient with a Head Injury

The first step in evaluating a child or adolescent presenting with a known head injury is taking a detailed history. This includes determining the mechanism of injury, whether there was a loss of consciousness or seizure activity, whether there is any neck pain, whether there are any neurologic symptoms, whether there are any symptoms suggesting a concussion, the timing of the symptoms and previous history of a head injury or a concussion (18-21).

Additional considerations surrounding the mechanism of injury include the force of impact and if there were any high-risk mechanisms (18-21). The higher the force of impact for both linear and rotational acceleration, the greater the likelihood of concussion diagnosis (22-26). However, there is no specific impact threshold that will invariably lead to a concussion diagnosis. A low impact force may result in a concussion in some cases and a high impact force may not result in a concussion in others (22-26). It is often important to correlate force with a high risk or low risk mechanism of injury. High-risk mechanisms of injury include a double hit and contact with a rotational force. An example of a double hit, which may increase severity of the injury, is when a player's head makes contact with an opponents shoulder and then with the boards or the ground

(24-26). Contact with a rotational component involves an individual who makes contact with another person, object or surface while their head is rotating (24-26). This mechanism also increases the severity of the concussion (24-26). Lastly, second impact syndrome is a rare but dangerous injury where a second concussion is sustained prior to the resolution of the first concussion and results in severe brain swelling that is often fatal. The second impact may occur later that day or days to weeks after the first concussion. A second hit of similar or greater impact can cause severe cognitive dysfunction, which is why physical rest is crucial in the context of an acute concussion (24-26).

In terms of loss of consciousness, the patient's account of the event, eyewitness account and video from the event are all important to obtain the most accurate possible story (19-22). In the event that the patient recalls the incident, loss of consciousness for a significant period of time is unlikely. Loss of consciousness for more than one minute and seizures as a result of the concussion are rare in children and adolescents (19-22). In the event that this does take place, an urgent evaluation for a potential intracranial injury is important (19-22). To properly evaluate a potential intracranial injury, the children and adolescents must be placed in a high, moderate or low risk category (19-22). High-risk patients tend to appear well, but may have focal neurologic deficits, signs of a basilar skull fracture, seizure, persistent altered level of mental status and prolonged loss of consciousness (19-22). These patients should have emergent brain imaging to rule out intracranial abnormalities (19-22). Moderate risk patients have a headache with vomiting, a brief loss of consciousness and a high mechanism of injury (19-22). These patients should be monitored closely for changes in mental status and for worsening symptoms. If

the symptoms begin to worsen, brain imaging is indicated (19-22). Low risk patients have no loss of consciousness, normal mental status, no signs of a basilar skull fracture, no vomiting and a headache that is improving with treatment (19-22). Brain imaging should be avoided in these patients (19-22).

In patients with neck pain or extremity numbness and tingling, cervical spine immobilization must be instituted until the spine can be clinically or radiographically cleared (27). To clinically clear the neck, the following questions must be asked and answered (27). Is the patient awake? Does the patient have a previous disposition to neck injuries? Was there a high-risk mechanism? Is the patient having neck pain? Is there a distracting painful injury? Are there any neurological deficits? Is there pain on palpation of the neck? Is there pain on active motion of the neck? If the patient is awake and you can answer no to the rest of the above questions, the neck can be cleared and there is no need for imaging (27). In the event that there is a need for imaging, the following questions must be asked and process must be followed to radiographically clear the neck (27). Are there any abnormal neurological findings on examination? If there are, the collar must remain on and an MRI or CT scan is recommended (27). If the answer is no, anterior and lateral c spine x-rays may be performed (27). In the event that these x-rays are abnormal, a follow up CT scan should be performed and the spine service should be consulted (27). If the x-rays are normal, frequent reassessment of level of consciousness for the next 1-3 days is very important (27).

The next step involves a thorough physical exam. Children and adolescents that present with a concussion very often have a normal physical examination. The signs of a more serious head injury may include raccoon eyes, visual disturbances, altered eye

movements, loss of sensation on the forehead, loss of sensation on the lower face, tinnitus, hemotypanium, battle's sign and an impaired gag reflex (28-29). The neurological examination must always include testing of cranial nerves, motor function, sensation, reflexes, cerebellar function, gait, balance and cognitive functioning. A focused vestibular-ocular examination is also frequently performed.

## Diagnosis and Differential Diagnosis

Once a thorough history and physical exam have taken place, one must ask if the findings are consistent with a concussion. The diagnostic criteria for a concussion are as follows (1, 4). First, there must be a history of trauma that results in either linear or rotational acceleration of the brain. This trauma can either be a direct contact to the head or an indirect force that causes subsequent brain acceleration. Second, there must be an onset of signs and symptoms consistent with a change from the patient's baseline and these changes must take place soon after the contact. Third, exclusion of intracranial abnormalities (such as a subdural or epidural hematoma) must be carried out when it is clinically indicated. Finally, if the mechanism of injury is unclear, or if the mechanism of injury is inconsistent with a typical concussion, it is important to consider a differential diagnosis (1, 4). The differential diagnosis for a concussion is quite broad, but it is crucial to rule out life threatening injuries. The differential can include intracranial injuries, primary headaches, overtraining or psychiatric disorders (30).

When it comes to significant intracranial injuries, the difficulty is that symptoms of a concussion and an intracranial injury overlap. In this case, prolonged loss of consciousness, seizures, altered mental status that does not seem to be improving, an

abnormal neurological examination, a headache that is worsening and vomiting uncontrollably are all reasons for a physician to consider neuroimaging (30).

Primary headache, which is the most common symptom of a concussion, is an important differential diagnosis of concussion. Primary headaches can be divided into migraine headaches, cluster headaches and tension headaches (15, 16). When it comes to a primary headache, the causes may include cervical injuries, muscle inflammation or infection (15, 16). Concussions are included in the category of a secondary headache. Secondary conditions always have an underlying or known etiology (15, 16).

Overtraining with corresponding decrease in performance is a condition that often goes under diagnosed in youth and is a condition with very similar symptoms to that of a concussion (31-33). These symptoms include decreased sleep, difficulty concentrating, and decreased interest in training (31-33). The key factor in making this diagnosis versus that of a concussion is that these symptoms typically develop over a long period of time and that overtraining is not linked to head trauma (31-33).

Psychiatric disorders and psychiatric related symptoms can be very challenging to evaluate in the situation of a suspected concussion. Although many patients report emotional symptoms such as sadness, anxiety, and irritability following concussion, some can develop new psychiatric conditions or worsening symptoms of a previously known psychiatric disorder (34).

## Previous Literature

Concerning the literature surrounding youth with a history of previous concussions versus those with no history of concussions, it has been stated that those with a prior history may be at an increased risk for a greater number of symptoms, including

declining neuropsychological functioning, attention span, and mental speed (35). However, there does not appear to be any previous literature comparing recovery times for those youth with and without a history of concussions.

### Objective

The objective is to determine if children aged seven to 19 with a previous history of a concussion and who present to the Pan Am Concussion Program with an acute sport related concussion take longer to recover, have a greater number of symptoms, have a higher post concussion symptom score or have a greater chance of being diagnosed with post concussion syndrome than those with no previous history of a concussion.

## Methods

A retrospective chart review was performed for all pediatric patients (between the ages seven and 19) referred to the Pan Am Concussion Program in Winnipeg, Manitoba, Canada between May 1, 2013 and May 1, 2015. Those patients referred for an acute sport related concussion with complete medical information were included in this study. The institutional ethics review board at the University of Manitoba approved this study.

### Definitions and Outcomes

A sport related concussion is diagnosed when the injury takes places in the setting of organized or unorganized sport. For the purposes of this study, we defined an acute concussion as a diagnosis made by the treating neurosurgeon at the Pan Am Concussion Program within 30 days from the time of the injury (34). Loss of consciousness was defined as a patient who reported a loss of consciousness at the time of the injury.

The primary outcome was number of days to recovery, which was reported by the neurosurgeon. Secondary outcomes included being subsequently diagnosed with post-

concussion syndrome, the number of symptoms at initial presentation and total post-concussion symptom scale score. In concordance with previous pediatric concussion studies and the International Classification of Disease (version 10), we defined post concussion syndrome as having 3 or more symptoms for at least 30 days following the initial date of the injury (34). Recovery was defined by the patient being able to manage a full time return to school, having successfully finished the return to play protocol set forth by the International Consensus on Concussion in Sport and no longer meeting the neurosurgeon's criteria for vestibular ocular dysfunction (34).

## Clinical Assessments

Upon initial presentation to the Pan Am Concussion Clinic for medical examination, each patient completed a standardized data collection form that included demographic data, sport played at the time of concussion or activity/event that resulted in the concussion, past medical history, past concussion history and family history (34). At this initial visit, each patient also completed the Post Concussion Symptom Scale (PCSS) to determine the number of symptoms and symptom severity (34) (Figure 1). This scale allowed the post concussion symptom score to be calculated, along with the median number of symptoms. In addition, the patients indicated what sport they were playing or what activity they were participating in at the time of the injury. Every patient underwent a clinical history and physical examination by a single neurosurgeon. Patients were then seen in follow up at 1-4 week intervals depending on the severity of their symptoms, pace of their recovery, and availability of the physician rather than using a predesigned research protocol (34). At each follow up appointment, each patient completed the PCSS and underwent a follow up history and physical examination, as indicated by the

neurosurgeon. The majority of the patients were managed conservatively, which included physical and mental rest, followed by slowly reintroducing full time school and sporting and/or regular every day activities (34). For the patients who remained symptomatic at 1 month, they were considered for specialized interventions, which included referrals to experts in vestibular or cervical physiotherapy, exercise science, headache neurology, neuropsychology and neuro-ophthalmology (34). The neurosurgeon also screened these patients for post injury psychiatric outcomes and referrals to appropriate outlets, such as mobile crisis or adolescent psychiatry were considered on a case-by-case basis. There were no predetermined criteria for a psychiatry referral. Neuroimaging studies were ordered as clinically indicated. Finally, the diagnosis of post concussion syndrome was made by the neurosurgeon based on the definition above (34). When the patient was discharged from the concussion clinic, follow up with the patients primary care provider was recommended.

## Statistical Analysis

The distributions of baseline characteristics were described for those presenting with no previous history of a concussion and those presenting with a previous concussion history. If a continuous variable was not normal distributed, it was reported as a median with interquartile ranges or a mean with standard deviations for normally distributed data. Dichotomous/polytomous clinical characteristics of the two groups were compared using a chi-square test. Continuous, normally distributed clinical characteristics were compared using the unpaired t-test. If continuous data were not normally distributed, statistical significance was assessed using the rank-sum test. For all statistical tests, a 2-sided p value of less than 0.05 was deemed statistically significant (34).

## Results

Table 1 is an overview of the baseline characteristics from the study. There were a total of 306 participants in this study. The mean age of the participants presenting to the concussion clinic without a history of a concussion was significantly lower (13.4 years) than the mean of age of the participants with a history of a concussion (14.9 years; $p<0.0001$). Among those with no concussion history, 64.1% were males and of those with a past concussion history, 68.4% were males ($p=0.507$). Furthermore, of those with no concussion history, 12.3% reported a loss of consciousness compared with 17.4% of those with a concussion history who reported a loss of consciousness ($p=0.216$). Table 1 also describes both the symptoms at their initial presentation to the Pan Am Concussion Program as well as the particular sport or activity that resulted in the concussion.

**Table 1: Clinical baseline characteristics of pediatric patients at the Pan Am Concussion Clinic with no concussion history or a previous concussion history**

| Outcome (n=306) | No Concussion History (n=192) | Concussion History (n=114) | P Value |
|---|---|---|---|
| Mean age in years (SD) | 13.4 (2.4) | 14.9 (1.9) | <0.0001 |
| **Sex** | | | |
| Male | 125 (64.1) | 78 (68.4) | 0.507 |
| Female | 70 (35.9) | 37 (31.7) | 0.507 |
| **Loss of Consciousness** | 24 (12.3) | 20 (17.4) | 0.216 |
| **Symptoms** | | | |
| Headache | 182 (93.3) | 109 (94.8) | |
| Disorientation | 65 (33.3) | 47 (40.9) | |
| Confusion | 69 (35.4) | 43 (37.4) | |
| Dizziness | 145 (74.4) | 91 (79.3) | |
| Walking Imbalance | 80 (41.0) | 47 (40.9) | |
| Fogginess | 90 (46.2) | 73 (63.5) | |
| Difficulty Focusing | 39 (20.0) | 40 (34.8) | |
| Tinnitus | 86 (44.1) | 63 (54.8) | |
| Seeing Stars | 35 (17.9) | 27 (23.5) | |

| | | | |
|---|---|---|---|
| Blurred Vision | 66 (33.8) | 47 (40.9) | |
| Uncoordinated | 32 (16.4) | 24 (20.9) | |
| Sad | 24 (12.3) | 14 (12.2) | |
| Seizure | 1 (0.005) | 1 (0.009) | |
| Neck Pain | 76 (39.0) | 55 (47.8) | |
| Amnesia | 47 (24.1) | 30 (26.1) | |
| Nausea | 95 (48.7) | 52 (45.2) | |
| Vomiting | 17 (8.7) | 8 (7.0) | |
| Fatigue | 111 (56.9) | 73 (63.5) | |
| Light Sensation | 82 (42.1) | 68 (59.1) | |
| Sound Sensation | 78 (40.0) | 54 (47.0) | |
| Feeling Slow | 66 (33.8) | 50 (43.5) | |
| Irritability | 42 (21.5) | 29 (25.2) | |
| Feeling Nervous | 31 (15.9) | 18 (15.7) | |
| **Sport** | | | |
| Hockey | 87 (45.1) | 55 (48.2) | |
| Snowboarding | 4 (2.0) | 4 (3.5) | |
| Soccer | 29 (15.1) | 12 (10.5) | |
| Basketball | 12 (6.3) | 4 (3.5) | |
| Football | 20 (10.4) | 16 (14.0) | |
| Volleyball | 0 (0) | 5 (4.4) | |
| Ringette | 8 (4.2) | 4 (3.5) | |
| Rugby | 4 (2.0) | 1 (0.009) | |
| Other | 20 (10.4) | 14 (12.3) | |
| **Cleared to Return to Play** | 165 (84.6) | 90 (78.2) | 0.157 |

There were 30 participants who were lost to follow up; however, there were no differences in the baseline characteristics between those lost to follow up and those where recovery days could be calculated (Table 3, appendix).

The primary outcome illustrated in Table 2 was recovery days. For the participants with no past concussion history, the median number of recovery days was found to be 22 days (IQR: 15-43) compared to participants with a past concussion history in which the median number of recovery days was found to be 23 days (IQR: 16-39)**.** This difference in medians was not statistically significant (p=0.41)**.** Next, the median post concussion symptom score upon initial presentation at Pan Am Concussion Program

for those with no concussion history was nine (IQR: 1-22) compared to those with a past concussion history in which the median post concussion symptom score was 13 (IQR: 3-34). This was statistically significant (p=0.0328). Furthermore, those participants with no concussion history had a statistically significant lower median number of symptoms, (5.5 symptoms; IQR: 1-10), upon initial presentation compared with those who had a past concussion history, (7.0 symptoms; IQR: 2-14; p=0.0366). Of those with no concussion history, 77 (40.1%) were diagnosed with post concussion syndrome and 48 (41.7%) of those with a past concussion history were diagnosed with post concussion syndrome. These proportions were not statistically significant (p=0.729). A graphical representation of the primary and secondary outcomes can be found in the appendix in figures 2-4.

**Table 2: Clinical outcomes of pediatric patients presenting to the concussion clinic with no history of a concussion or a history of a concussion**

| Outcome (n=306) | No Concussion History (n=192) | Concussion History (n=114) | P Value |
|---|---|---|---|
| Recovery Days (IQR) | 22 (15-43) | 23 (16-39) | 0.41 |
| Median PCSS (IQR) | 9 (1-22) | 13 (3-34) | 0.0328 |
| Median Number of Symptoms (IQR) | 5.5 (1-10) | 7.0 (2-14) | 0.0366 |
| Subsequent Diagnosis of PCS | 77 (40.1) | 48 (41.7) | 0.729 |
| Missing | 3 (75.0) | 1(25.0) | |

## Discussion

This study identifies several important findings. There was no significance in the median length of recovery between those youth with no past concussion history and those youth with a past concussion history. The implication of this is that the treatment and management of these two groups should be based on presenting signs and symptoms and

interventions should be targeted towards each individual patient and not based on concussion history.

Initial management in the setting of an acute concussion involves prevention of an additional head injury by immediately removing the youth from the sport. The individual should then seek attention from a licensed health care practitioner to determine if they have sustained a concussion. If a diagnosis is made, the individual must not return to competition until a full recovery takes place (36-38). Further to removal from competitive activities, youth diagnosed with a concussion should be removed from high-risk recreational activities that may lead to an additional head injury (36-38). During the recovery period, which varies for each individual, there may be an increase in the severity of symptoms and there is a greater chance of a recurrent concussion (36-38). Physical rest is an important part of the early stages of recovery. During the period of rest, activities of normal daily living are allowed, however, activities that result in an increased heart rate should be avoided (36-38). Throughout this period, it is important that parents and caregivers are aware that changes in sleep patterns, due to the lack of exercise, may take place (36-38). A decreased amount of sleep can cause the patient to become irritable, have difficulty concentrating or may result in a longer time to recovery (36-38). The last step in management is cognitive rest. This includes close monitoring of symptoms and academic adjustments such as reduced class time when appropriate (37). It is also important to note that a prolonged time out of the classroom may not be beneficial for the patient either. In fact, this may result in a negative academic situation (37). Thus, youth are divided into two categories when it comes to cognitive rest. First, those who develop symptoms related to cognitive efforts and second, those who do not develop these

symptoms. For those patients that are symptomatic, it is highly recommended that video games, screen time, test writing and any other activities that cause the symptoms be completely avoided (37). This may include absence from school until the symptoms begin to resolve (37). For the patients that are without symptoms, activities that require a great deal of concentration should be limited, however attendance at school can be an option for these individuals (37).

The next finding was that youth with a past concussion history had a statistically significant higher post concussion symptom score and higher number of symptoms compared to youth without a concussion history. This means that youth have more severe symptoms and a greater number of symptoms when they first present, if they have had previous concussions. This could have implications for parents, coaches, educators and health care practitioners, when it comes to management of a child with a history of concussions. And although the recovery time is not significantly different between the concussion history and no concussion history groups, the long-term effects of multiple worsening presentations of a concussion are unknown.

Some common symptoms of a concussion can include nausea, vomiting, headache, amnesia, difficulty focusing or concentrating, dizziness, photophobia, phonophobia, and changes in emotional state (1-4). In terms of the specific treatment of these symptoms, for concussion related headaches, Tylenol and non-steroidal anti-inflammatory drugs are recommended, but only in the first 2-3 days following the injury (39). Following this, the research has shown the use of these medications may cause worsening rebound headaches after the effect of the medication has worn off (39). For nausea resulting from a concussion, ondansetron can be considered in the first couple of

days (39). However, a side effect of this drug is headaches (39). It is important to note that metoclopramide should be avoided in youth due to negative side effects of drowsiness and syncope episodes (39). For those suffering from a sleep disturbance, an emphasis on a routine, including scheduled bed times and wake up times, is important (39). Pharmacotherapy is not recommended in this situation to aid in the process of sleeping (39). Finally, for dizziness, this symptom tends to resolve itself with adequate physical and cognitive rest (39).

Although a greater percentage of participants with a past concussion history went on to develop a diagnosis of post concussion syndrome this was not found to be statistically significant. Thus, history of concussion does not increase the risk of developing post concussion syndrome. However, approximately 40% of young athletes were diagnosed with post concussion syndrome. This is where the job of a health care practitioner becomes very important. It is important to inform parents that there is always a chance that their child will suffer a concussion and the potential long-term consequences that come along with it. At that point, ensuring they seek proper medical care and follow a proper return to play and learn protocol becomes very important. The return to learn and return to play protocol are both excellent resources for parents, administrators, coaches and any individual directly involved in the care of a particular child (38). Some children will require specific accommodations including a limited course load, shortened day or shortened periods, assistance with learning and delay of any major tests or exams (38). The return to learn or play protocol is not the same for every child. The key to success is a good communication between the child, the parent, the administrator and the physician (38).

Before returning to play, it is important that youth complete a course of non-contact exercise regimens, each with increasing intensity (1). In order to be eligible to begin this protocol, children must have a successful return to school, be symptom free and off any medications that were prescribed to help with the concussion, have a normal neurological exam and be back to their cognitive and balance baseline (1). The stages in this protocol include light aerobic exercise, more intensive training, sport specific exercises, non-contact participation, full practice and finally game participation (1).

Concussion prevention efforts have included education, enforcement and engineering strategies. The research has shown that education around concussions has led to an increase in retaining knowledge about signs and symptoms of a concussion and the seriousness of a concussion (40). Furthermore, the research has shown that altering the age at which contact begins in sports is correlated with a decrease in the amount of concussions (40). One particular hockey based study showed that decreasing the age of contact from 11 to nine years old increased the amount of concussions from four to 22 in one season (40). In addition, the American Academy of Pediatrics recommends no contact in youth sports until the age of 15 (40). Lastly, there has been some evidence showing that when there is an emphasis on fair play and sportsmanship in youth sport, this can result in a decrease in all injuries, including concussions (40). This also includes the use of proper technique in all facets of the game. As a result, coaches and referees play an invaluable role in the prevention of youth concussions (40).

## Limitations

The results of this research have some significant implications, however, it must be stated that there were some limitations. First, there was not a standardized protocol for each patient; rather patients were managed based on clinical judgment. Some patient were referred to specialists or sent for neuroimaging and therefore outcomes like neuroimaging could not be compared between the two groups. However, one physician provided care to all patients, which increased the continuity of care.

Secondly, there was no baseline assessment of patient's symptoms and signs prior to the concussion. The Post Concussion Symptom Scale asks people to rate their symptoms on a Likert scale and rating a headache may reflect a headache due to concussion or that the patient has long suffered from headaches. It is possible that the concussion may have worsened that symptom or perhaps the concussion had no effect on the symptom at all. However, part of the initial presentation includes a detailed patient history to determine if the patient has a history of common concussion symptoms. Also, the Post Concussion Symptom Scale is a subjective tool; one may rate their severe headache as a 4/6, while a second patient rates the same headache as 6/6. However, object criteria were included in the return to play decisions and not just symptom reporting.

Third, there were a number of factors that were not taken into consideration in this study that could potentially have an effect on recovery time and number of symptoms. Some of these include socio-economic status, parent/guardian support, living environment, and school support. While participants in this study were provided with

return to play and return to learn instructions, there was no formal check to determine if these guidelines were followed.

Finally, some patients were lost to follow up (~10%). However, as reported in the Appendix, Table 3, they were not systematically different than those who received medical clearance to return to sport.

### Future Research

Future research opportunities could focus more on the social determinants of health, such as socio-economic status, diet, living conditions and parent/guardian support to determine if this might change the number of recovery days or number of symptoms. In addition, a future study could follow the participants who have been diagnosed with post concussion syndrome two, five and 10 years post injury to see if the symptoms have resolved or changed, if any new symptoms have arose, if they are able to hold down a job, if they have a family and to find out if there are any day to day struggles directly attributed to their concussions during their years as a youth. Finally, as it was alluded to above, a comparison of the sexes, and determining which sex is more likely to suffer from a concussion could be a future area of research.

## Conclusion

Despite these limitations, the study showed that there was a statistically significant higher number of median symptoms and post concussion symptom score in those with a past concussion history compared to those without. In addition, there was no statistical difference in the median recovery days or subsequent post concussion syndrome diagnosis between those with no past concussion history and those with a past concussion history. The implications of these results further emphasize the importance of

awareness, safety, and preventative measures surrounding proper recovery and symptom management in the setting of concussions.

## Appendix:

**Please rate your symptoms based on how much you have felt in the last 24 hours.**

| | None | | | Moderate | | | Severe |
|---|---|---|---|---|---|---|---|
| Headache | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Nausea | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Vomiting | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Drowsiness | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Numbness or tingling | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Dizziness | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Balance problems | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Sleeping more than usual | 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 as if "in a fog" | 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 |
| Trouble falling asleep | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| More emotional than usual | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Irritability | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Sadness | 0 | 1 | 2 | 3 | 4 | 5 | 6 |
| Nervousness | 0 | 1 | 2 | 3 | 4 | 5 | 6 |

Medscape

Source: Med Sci Sports Exerc © 2015 American College of Sports Medicine

**Figure 1: Post Concussion Symptom Scale (41)**

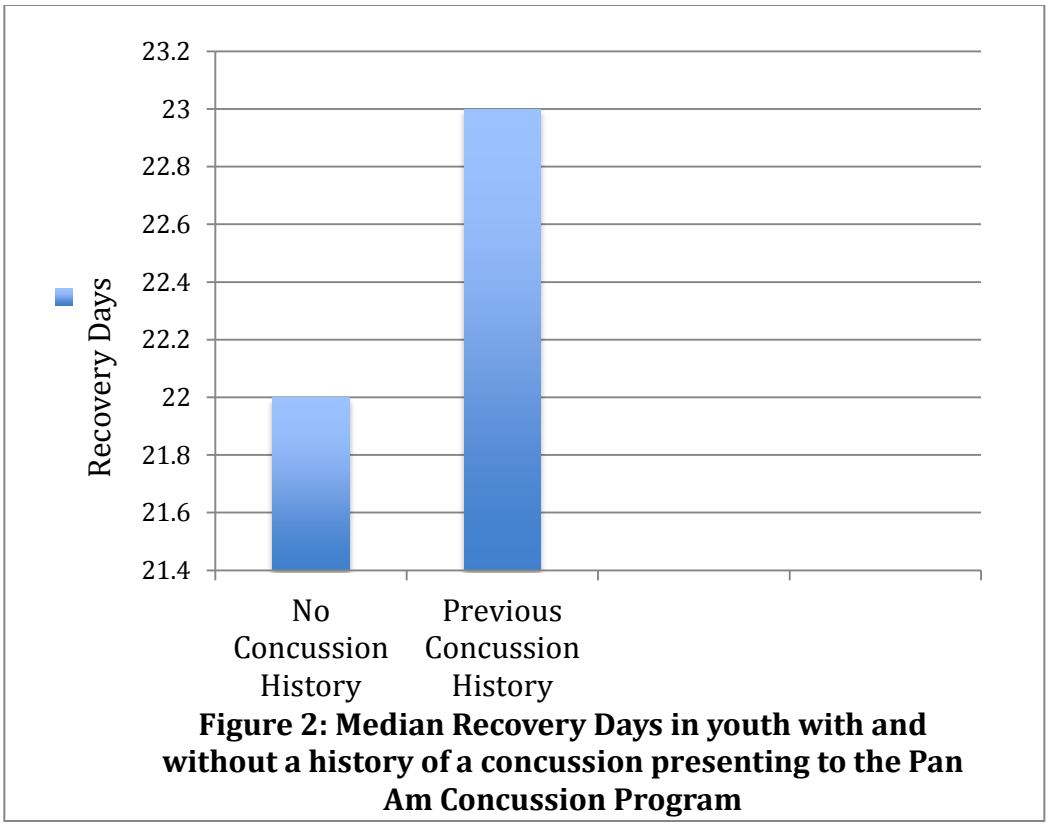


**Figure 2: Median Recovery Days in youth with and without a history of a concussion presenting to the Pan Am Concussion Program**

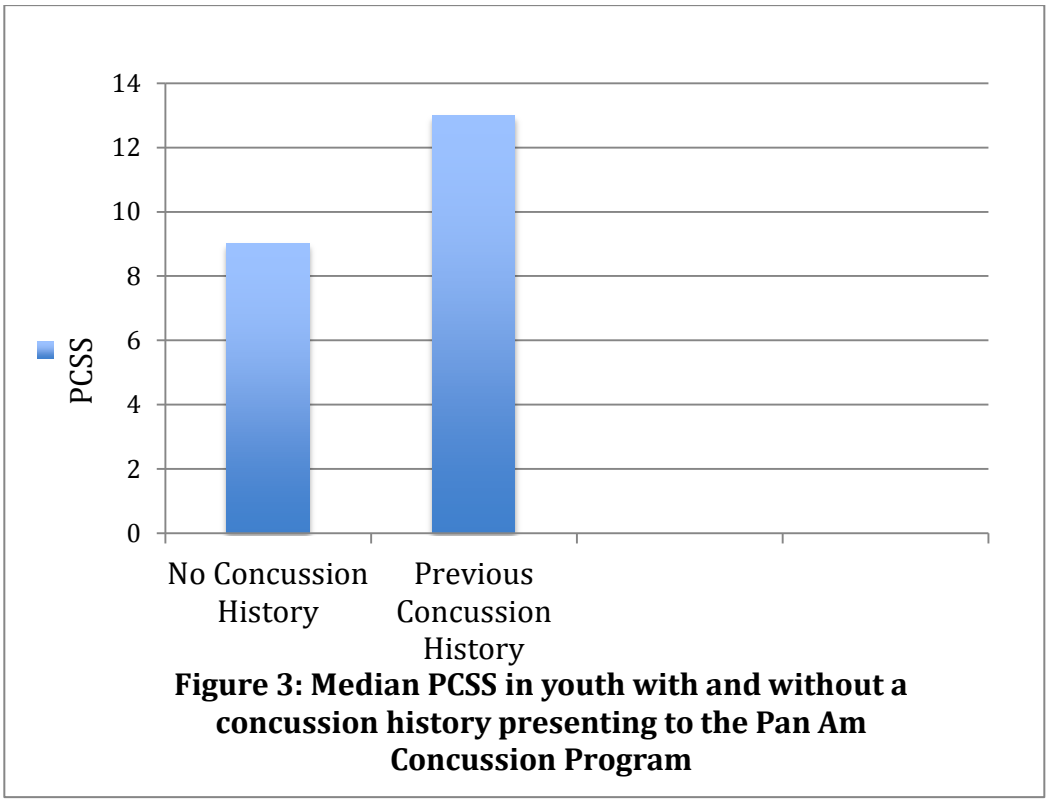


**Figure 3: Median PCSS in youth with and without a concussion history presenting to the Pan Am Concussion Program**

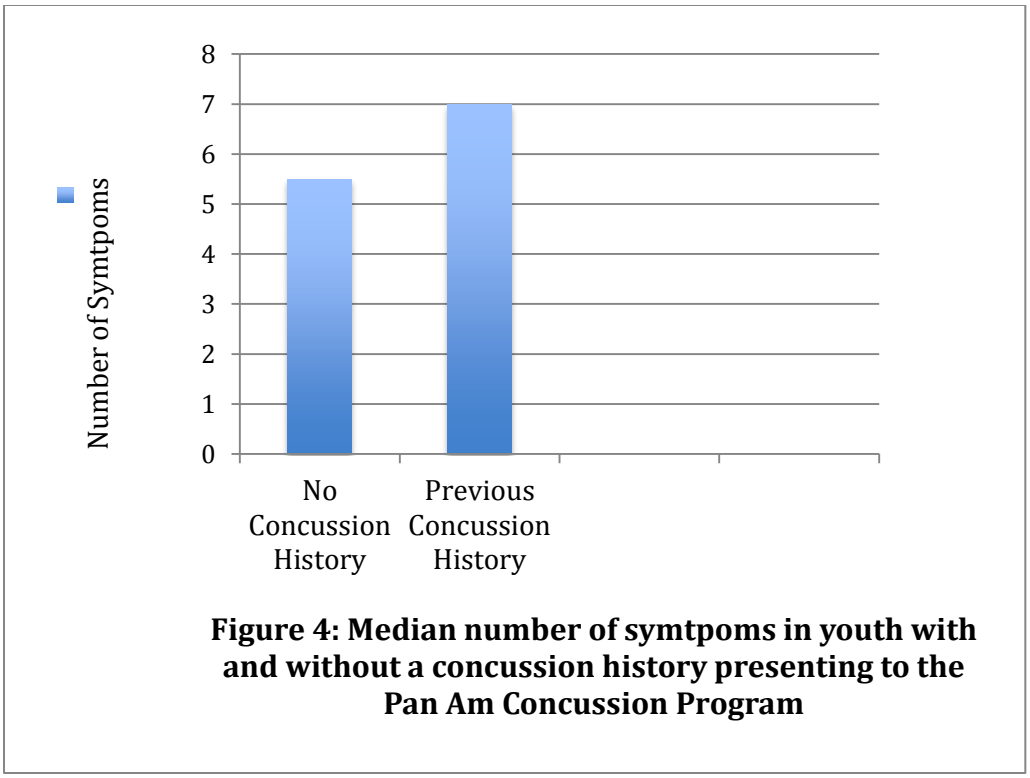


**Figure 4: Median number of symtpoms in youth with and without a concussion history presenting to the Pan Am Concussion Program**

**Table 3: Comparison of baseline characteristics of the youth lost to follow up and those who were not lost to follow**

| Outcome (n=306) | No Concussion History (n=192) | Concussion History (n=114) | Lost to Follow Up |
|---|---|---|---|
| Mean age in years (+/- SD) | 13.4 +/- 2.4 | 14.9 +/- 1.9 | 14.5 +/- 2.2 |
| **Sex** | | | |
| Male | 125 (64.1) | 78 (68.4) | 14 (46.7) |
| Female | 70 (35.9) | 37 (31.7) | 16 (53.3) |
| Loss of Consciousness | 24 (12.3) | 20 (17.4) | 4 (13.3) |
| **Symptoms** | | | |
| Headache | 182 (93.3) | 109 (94.8) | 29 (96.7) |
| Disorientation | 65 (33.3) | 47 (40.9) | 16 (53.3) |
| Confusion | 69 (35.4) | 43 (37.4) | 14 (46.7) |
| Dizziness | 145 (74.4) | 91 (79.3) | 25 (83.3) |
| Walking Imbalance | 80 (41.0) | 47 (40.9) | 14 (46.7) |
| Fogginess | 90 (46.2) | 73 (63.5) | 25 (83.3) |
| Difficulty | 39 (20.0) | 40 (34.8) | 13 (43.3) |

| | | | |
|---|---|---|---|
| Focusing | | | |
| Tinnitus | 86 (44.1) | 63 (54.8) | 20 (66.7) |
| Seeing Stars | 35 (17.9) | 27 (23.5) | 4 (13.3) |
| Blurred Vision | 66 (33.8) | 47 (40.9) | 17 (56.7) |
| Uncoordinated | 32 (16.4) | 24 (20.9) | 8 (26.7) |
| Sad | 24 (12.3) | 14 (12.2) | 7 (23.3) |
| Seizure | 1 (0.005) | 1 (0.009) | 0 |
| Neck Pain | 76 (39.0) | 55 (47.8) | 15 (50.0) |
| Amnesia | 47 (24.1) | 30 (26.1) | 5 (16.7) |
| Nausea | 95 (48.7) | 52 (45.2) | 15 (50.0) |
| Vomiting | 17 (8.7) | 8 (7.0) | 1 (3.3) |
| Fatigue | 111 (56.9) | 73 (63.5) | 24 (80.0) |
| Light Sensation | 82 (42.1) | 68 (59.1) | 18 (60.0) |
| Sound Sensation | 78 (40.0) | 54 (47.0) | 18 (60.0) |
| Feeling Slow | 66 (33.8) | 50 (43.5) | 13 (43.3) |
| Irritability | 42 (21.5) | 29 (25.2) | 11 (36.7) |
| Feeling Nervous | 31 (15.9) | 18 (15.7) | 5 (16.7) |
| **Sport** | | | |
| Hockey | 87 (45.1) | 55 (48.2) | 10 (33.3) |
| Snowboarding | 4 (2.0) | 4 (3.5) | 0 |
| Soccer | 29 (15.1) | 12 (10.5) | 4 (13.3) |
| Basketball | 12 (6.3) | 4 (3.5) | 2 (13.3) |
| Football | 20 (10.4) | 16 (14.0) | 6 (20.0) |
| Volleyball | 0 (0) | 5 (4.4) | 1 (3.3) |
| Ringette | 8 (4.2) | 4 (3.5) | 0 |
| Rugby | 4 (2.0) | 1 (0.009) | 2 (6.7) |
| Other | 20 (10.4) | 14 (12.3) | 5 (16.7) |
| Cleared to Return to Play | 165 (84.6) | 90 (78.2) | |

**Acknowledgements:**

I would like to give a very special thank you to Dr. Michael Ellis and Dr. Kelly Russell for their guidance and support throughout the entire process. This project would simply have not been possible without them. I would also like to thank all of the staff at the Pan Am Concussion Clinic for their cooperation and support. Finally, I would like to thank all of the participants who consented to this study.

**References:**


1. McCrory P, Meeuwisse W, Aubry M, Cantu B, Dvorak J, Echemendia RJ, et al. Consensus Statement on Concussion in Sport—the 4th International Conference on Concussion in Sport Held in Zurich, November 2012. Clin J Sport Med [Internet]. 2013;23(2). Available from: http://journals.lww.com/cjsportsmed/Fulltext/2013/03000/Consensus_Statement_on_Concussion_in_Sport_the_4th.1.aspx
2. Lovell M. The Management of Sports-Related Concussion: Current Status and Future Trends. Clinics in Sports Medicine. 2009. p. 95–111.
3. Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness. Correlation of experimental and clinical observations of blunt head injuries. Brain. 1974;97(4):633–54.
4. Gennarelli TA, Adams JH, Graham DI. Acceleration induced head injury in the monkey.I. The model, its mechanical and physiological correlates. Acta Neuropathol Suppl [Internet]. 1981;7:23–5. Available from: http://www.ncbi.nlm.nih.gov/pubmed/6939241
5. Giza CC, Hovda DA. The new neurometabolic cascade of concussion. Neurosurgery. 2014;75:S24–33.
6. Katayama Y, Becker DP, Tamura T, Hovda D a. Massive increases in extracellular potassium and the indiscriminate release of glutamate following concussive brain injury. J Neurosurg [Internet]. 1990;73(6):889–900. Available from: http://thejns.org/doi/abs/10.3171/jns.1990.73.6.0889
7. Yoshino a, Hovda D a, Kawamata T, Katayama Y, Becker DP. Dynamic

changes in local cerebral glucose utilization following cerebral conclusion in rats: evidence of a hyper- and subsequent hypometabolic state. Brain Res. 1991;561:106–19.

8. Yuan XQ, Prough DS, Smith TL, Dewitt DS. The effects of traumatic brain injury on regional cerebral blood flow in rats. J Neurotrauma. 1988;5(4):289–301.
9. Kerr Y. Z, Collins L. C, Mihalik P. J, Marshall W. S, Guskiewicz M. K, Comstock Dawn R. Impact Locations and Concussion Outcomes in High School Football Player-to-Player Collisions. Pediatrics [Internet]. 2014;134(3):5. Available from: http://spot.lib.auburn.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=cin20&AN=2013045414&site=ehost-live\nhttp://spot.lib.auburn.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=cin20&AN=2012712802&site=ehost-live
10. Zohar O, Schreiber S, Getslev V, Schwartz JP, Mullins PG, Pick CG. Closed-head minimal traumatic brain injury produces long-term cognitive deficits in mice. Neuroscience. 2003;118(4):949–55.
11. Johnston KM, McCrory P, Mohtadi NG, Meeuwisse W. Evidence-Based review of sport-related concussion: clinical science. Clin J Sport Med. 2001;11(3):150–9.
12. Shaw N a. Neurophysiology of concussion. Prog Neurobiol. 2002;67(4):281–344.
13. Marar M, McIlvain NM, Fields SK, Comstock RD. Epidemiology of Concussions

Among United States High School Athletes in 20 Sports. Am J Sports Med. 2012;40(4):747–55.

14. Bailes JE, Cantu RC. Head injury in athletes. Neurosurgery. 2001;48(1):26–46.
15. Meehan 3rd WP, d'Hemecourt P, Collins CL, Comstock RD. Assessment and management of sport-related concussions in United States high schools. Am J Sport Med [Internet]. 2011;39(11):2304–10. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21969181
16. Meehan WP, d'Hemecourt P, Comstock RD. High school concussions in the 2008-2009 academic year: mechanism, symptoms, and management. Am J Sports Med [Internet]. 2010;38(12):2405–9. Available from: http://ajs. Bakhos LL, Lockhart GR, Myers R, Linakis JG. Emergency department visits for concussion in young child athletes. Pediatrics. 2010;126(3):e550–6. sagepub.com/content/early/2010/08/17/0363546510376737
17. Bakhos LL, Lockhart GR, Myers R, Linakis JG. Emergency department visits for concussion in young child athletes. Pediatrics. 2010;126(3):e550–6
18. Grindel SH, Lovell MR, Collins MW. The assessment of sport-related concussion: the evidence behind neuropsychological testing and management. Clin J Sport Med. 2001;11(3):134–43.
19. Lovell MR, Collins MW, Iverson GL, Field M, Maroon JC, Cantu R, et al. Recovery from mild concussion in high school athletes. J Neurosurg Pediatr [Internet]. 2003;98(2):296–301. Available from: http://www.ncbi.nlm.nih.gov/pubmed/12593614
20. Asplund C a, McKeag DB, Olsen CH. Sport-related concussion: factors

associated with prolonged return to play. Clin J Sport Med. 2004;14(6):339-43.

21. McCrea M, Guskiewicz K, Randolph C, Barr WB, Hammeke TA, Marshall SW, et al. Incidence, clinical course, and predictors of prolonged recovery time following sport-related concussion in high school and college athletes. J Int Neuropsychol Soc [Internet]. 2013;19:22–33. Available from: http://www.journals.cambridge.org/abstract_S1355617712000872
22. Mihalik JP, Guskiewicz KM, Marshall SW, Blackburn JT, Cantu RC, Greenwald RM. Head impact biomechanics in youth hockey: Comparisons across playing position, event types, and impact locations. Ann Biomed Eng. 2012;40(1):141–9.
23. Darling SR, Schaubel DE, Baker JG, Leddy JJ, Bisson LJ, Willer B. Intentional versus unintentional contact as a mechanism of injury in youth ice hockey. Br J Sports Med. 2011;45(6):492–7.
24. Broglio SP, Eckner JT, Kutcher JS. Field-based measures of head impacts in high school football athletes. Curr Opin Pediatr [Internet]. 2012;24(6):702–8. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3643806&tool=pmcentrez&rendertype=abstract
25. Broglio SP, Schnebel B, Sosnoff JJ, Shin S, Fend X, He X, et al. Biomechanical properties of concussions in high school football. Med Sci Sport Exerc [Internet]. 2010;42(11):2064–71. Available from: http://www.ncbi.nlm.nih.gov/pubmed/20351593

26. Greenwald RM, Gwin JT, Chu JJ, Crisco JJ. Head impact severity measures for evaluating mild traumatic brain injury risk exposure. Neurosurgery. 2008;62(4):789–98.
27. Chung S, Mikrogianakis A, Wales PW, Dirks P, Shroff M, Singhal A, et al. Trauma association of Canada Pediatric Subcommittee National Pediatric Cervical Spine Evaluation Pathway: consensus guidelines. J Trauma [Internet]. 2011;70(4):873–84. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21610393
28. Dietrich AM, Ginn-Pease ME, Bartkowski HM, King DR. Pediatric cervical spine fractures: Predominately subtle presentation. J Pediatr Surg. 1991;26(8):995–1000.
29. Finch GD, Barnes MJ. Major Cervical Spine Injuries in Children and Adolescents. J Pediatr Orthop [Internet]. 1998;18(6):811. Available from: http://journals.lww.com/pedorthopaedics/Fulltext/1998/11000/Major_Cervical_Spine_Injuries_in_Children_and.22.aspx
30. Powell JW, Barber-Foss KD. Traumatic brain injury in high school athletes. JAMA. 1999;282(10):958–63.
31. Coris EE, Ramirez AM, Van Durme DJ. Heat Illness in Athletes. Sport Med. 2004;34(1):9–16.
32. Scher CD, Stein MB, Asmundson GJG, Mccreary DR, Forde DR. The Childhood Trauma Questionnaire in a community sample: Psychometric properties and normative data. J Trauma Stress. 2001;14(4):843–57.
33. Yeung RR. Review the Acute Effects of Exercise on Mood State. J Psychosom

Res. 1996;40(2):123–41.

34. Ellis MJ, Ritchie LJ, Koltek M, Hosain S, Cordingley D, Chu S, et al. Psychiatric outcomes after pediatric sports-related concussion. J Neurosurg Pediatr [Internet]. 2015;16(6):709–18. Available from: http://thejns.org/doi/10.3171/2015.5.PEDS15220
35. Eisenberg M a, Andrea J, Meehan W, Mannix R. Time interval between concussions and symptom duration. Pediatrics [Internet]. 2013;132(1):8–17. Available from: http://www.ncbi.nlm.nih.gov/pubmed/23753087
36. Gurley JM, Hujsak BD, Kelly JL. Vestibular rehabilitation following mild traumatic brain injury. NeuroRehabilitation. 2013;32(3):519–28.
37. Halstead ME, McAvoy K, Devore CD, Carl R, Lee M, Logan K. Returning to learning following a concussion. Pediatrics [Internet]. 2013;132(5):948–57. Available from: http://pediatrics.aappublications.org/content/132/5/948.abstract
38. Hunt T, Asplund C. Concussion Assessment and Management. Clinics in Sports Medicine. 2010. p. 5–17.
39. Meehan WP. Medical Therapies for Concussion. Clinics in Sports Medicine. 2011. p. 115–24.
40. Halstead ME, Walter KD. American Academy of Pediatrics. Clinical report--sport-related concussion in children and adolescents. Pediatrics [Internet]. 2010;126(3):597–615. Available from: http://pediatrics.aappublications.org/content/126/3/597.abstract
41. Joyce AS, Labella CR, Carl RL, Lai JS, Zelko FA. The postconcussion symptom

scale: Utility of a three-factor structure. Med Sci Sports Exerc. 2015;47(6):1119–23.