

# Special Invited Article Concussion, Sports and Psychiatry

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**Abstract:** *Background:* Concussions (also known as mild traumatic brain injuries, MTBI) are common among adolescents, especially those who play sports. While often considered non-serious injuries, they can result in significant impairment in functioning. Neuropsychiatric symptoms, including cognitive impairment, behavioral dysfunction, and psychiatric disorders are among the most common sequelae.

*Method:* This article presents an overview of what is known at this point and makes recommendations regarding assessment and management.

*Results:* The knowledge base has expanded rapidly concerning risk factors, sequelae and neurobiology of MTBI. It is still underdeveloped regarding treatment, especially with regard to adolescents. Nonetheless, it is possible to extrapolate from the research on adults to develop a rational approach to management.

*Conclusions:* Key aspects of management include education and support for patients and families, teaching coping skills, and making academic accommodations. There is a limited role for psychopharmacologic approaches in conjunction with a comprehensive multidisciplinary approach. Most patients will recover but treatment must be individualized to address the patient's specific problems and concerns.

**Keywords:** Concussion, traumatic brain injury, adolescents, sports injuries.

## INTRODUCTION

Until fairly recently concussions, or mild traumatic brain injuries, were not considered significant enough to warrant medical attention or restrictions in subsequent level of activity. We now know that improper management of these injuries increases the risk for further injury, prolonged recovery, long term physical/neuropsychiatric consequences and the rare but sometimes fatal second impact syndrome (Meehan, 2009). Since many of the sequelae of concussion involve disruptions in cognitive and emotional functioning, it is important that mental health providers learn more about this under recognized condition, its diagnosis, and appropriate management.

This article will draw on the existing knowledge base to summarize what is known at this point. Although the knowledge base is expanding with regard to adolescents, most of the literature refers to adults, and except where noted, the studies cited in this article are of adults.

Concussion, also known as mild traumatic brain injury, is the most common form of traumatic brain injury (over 85%) and is often overlooked (Centers for Disease Control and Prevention, n. d.). Although concussion has been recognized for a long time, the role of sports in this injury and its significance in terms of long-term consequences have only recently been appreciated, both on the part of the scientific community and the general public. For example, there was

an article in The New York Times on May 10<sup>th</sup>, 2012 titled "Concussions May Be More Severe in Girls and Young Athletes (O'Connor, 2012)." The article was based on research published in *The American Journal of Sports Medicine* showing high school athletes performed worse than college athletes on verbal and visual memory following a concussion (Covassin, Elbin, Harris, Parker, & Kontos, 2012)." Numerous efforts have been undertaken to improve safety, particularly for high school athletes, and the National Football League (which recently settled a lawsuit brought on behalf of players who alleged their head injuries were due to play) has launched a major public relations campaign aimed at convincing parents that football can be played safely.

Adolescents, particularly high school athletes, are at high risk both for concussions as well as long-term sequelae known as post concussive syndrome. For example, four to five percent of high school football players will have a concussion during their playing career. These adolescent athletes' concussions are rarely reported. Players may not even realize that they have had one, and many continue to play despite post-concussive symptoms. A study published in the journal *Pediatrics* found that less than 50% had some knowledge of concussion, over 25% continued to play despite dizziness; over 60% played despite having a headache, and over 65% played despite loss of consciousness from a sports-related concussion (Meehan & Bachur, 2009).

The maxim, "When in doubt, sit them out" is based on solid evidence. Recently concussed athletes are at increased risk for repeat concussion. If they have a loss of consciousness, the risk is increased six fold. The greatest risk first is in

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the first 7-10 days after the initial injury, and there is often a longer recovery after repeat concussion (Centers for Disease Control and Prevention, n. d.).

## DEFINITIONS

A traumatic brain injury (TBI) is a traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force (Department of Veterans Affairs and Department of Defense, 2009). A mild traumatic brain injury, or concussion, is generally defined by a Glasgow Coma Scale score of 13-15 in the first 24 hours, together with a mental status change. Typically there are no imaging findings (Wilson & Yeates, 2009).

Although the term concussion is used interchangeably with traumatic brain injury, perceptions and responses differ according to which term is used. Whether a brain injury is labeled a concussion or traumatic brain injury makes a difference not only in public perception, but in medical treatment. An article in the *Journal Pediatrics* titled, "My Child Doesn't Have a Brain Injury, He Only Has a Concussion" showed that if an injury was labeled concussion instead of traumatic brain injury it was strongly predictive of an earlier hospital discharge and return to school (Dematteo *et al.*, 2010). This difference in management is very concerning given the prognostic implications of inadequate management.

## PREVALENCE

There are an estimated 1.7 million traumatic brain injuries each year in the US. These are estimated to affect 5.3 million Americans or 2% of the US population (Centers for Disease Control and Prevention, n. d.). They may result in long-term functional impairment; lost productivity was estimated at \$60 billion in 2000. The highest risk age groups are those between 0 and 4 years old and 15 to 19 years old. (Faul, Xu, Wald, & Coronado, 2010). The majority of traumatic brain injuries are mild and are not treated in emergency departments or hospitals (Bazarian *et al.*, 2005). Motor vehicle accidents account for 20%, being struck by an object 19%, and assaults, 11%. Bicycle accidents and sports injuries are the leading causes of traumatic brain injuries among adolescents (Centers for Disease Control and Prevention, 2011).

## OUTCOMES

A variety of unfavorable outcomes are associated with incomplete recovery from concussion. These outcomes include post concussive symptoms and post concussive syndrome (PCS) which refers to continued physical and neurocognitive symptoms that may continue for months to years after a concussion. Second impact syndrome is a fortunately rare, but serious and sometimes fatal condition that involves a second head injury before recovery from a concussion (Bey & Ostick, 2009). The second injury may be minor, but can lead to death due to increased pressure in the brain. Children may be at increased risk. Chronic traumatic encephalopathy (CTE), originally termed *dementia pugilistica* because of the frequency of its occurrence in fighters, involves memory disturbances, behavioral and

personality changes, Parkinsonism, and speech and gait abnormalities that are distinct from other neurodegenerative conditions. CTE occurs in at least 17% of people with repetitive mTBI (Stern *et al.*, 2013).

## NEUROBIOLOGY

While the actual mechanism of action in mild TBI has not been elucidated, there are several theories explaining the neurobiology of the injury. One of the most common is that the trauma to the brain is due to rotational acceleration forces sustained by the brain as well as a neuro-metabolic cascade that occurs following the injury. The acceleration forces lead to axonal damage through stretching, twisting and compression of the nerve axons. It is believed that following the trauma to the axons the cells break and release first potassium, then glutamate, followed by more potassium. As energy expenditure occurs in the brain glycolysis occurs leading to lactic acid accumulation which damages the cell (Meehan & Bachur, 2009). Based on this hypothesis, decreasing energy expenditure in the brain, by body and brain rest (the first and likely the most effective treatment), prevents added glycolysis and speeds the healing process.

Standard imaging studies such as computerized tomography (CT scan) or magnetic resonance imaging (MRI) do not show any abnormal findings following a mild traumatic brain injury. However, experimental imaging techniques such as diffusion tensor imaging (DTI) have shown abnormalities and may aid diagnosis in the future. One study compared 10 adolescents with mild traumatic brain injury to matched controls and showed changes in the corpus callosum as measured by fractional anisotropy and radial diffusivity, which were correlated with the severity of post concussive symptoms (Wilde *et al.*, 2008).

## CATEGORIZATION

The severity of traumatic brain injuries is primarily assessed in terms of the presence or absence of loss of consciousness, posttraumatic amnesia, and ratings on the Glasgow coma scale (Teasdale & Jennett, 1974). Table 1 shows the comparative findings at each level of severity of TBI.

## CLINICAL PRESENTATION

Acute concussion symptoms include changes in cognitive status, dizziness or brief loss of consciousness, and may include other symptoms, such as forgetting things that happened before or after the trauma (anterograde or retrograde amnesia). In addition the adolescent may appear to be dazed, stunned or confused, move clumsily, answer questions slowly, and show behavior or personality change. Loss of consciousness occurs in only 4-10%, and seizures are present in less than 5% of concussions. Typical recovery from concussion occurs within 7-10 days of injury (Meehan, III & Bachur, 2009).

## POST CONCUSSIVE SYNDROME

Psychiatrists should be particularly aware of post concussive syndrome and the neuropsychiatric complications. Post-concussion signs and symptoms can occur minutes to

**Table 1. Severity of TBI**

| Level of TBI          | Mild                              | Moderate          | Severe                        |
|-----------------------|-----------------------------------|-------------------|-------------------------------|
| GCS score             | 13-15 in 1 <sup>st</sup> 24 hours | ≥ 9-12            | 3-8 within 48 hours of injury |
| Posttraumatic amnesia | Not necessary for dx              | 1-24 hours        | ≥1 day                        |
| Loss of consciousness | < 30 min                          | 30 min – 24 hours | 1 to 24 hours                 |
| brain scan            | Normal                            | May be abnormal   | Abnormal                      |

weeks after the initial injury. Post concussive syndrome (PCS), is not clearly defined and includes a group of individuals with prolonged recovery, typically greater than 3 months duration. PCS encompasses a variety of physical, cognitive, and emotional and behavioral symptoms. Criteria for post concussive syndrome include 1) a history of head trauma, 2) neuropsychologically assessed cognitive deficits in either attention or memory, and 3) three or more symptoms lasting three months or more. In the DSM-5 terminology post concussive syndrome is categorized as a major or mild neurocognitive disorder due to traumatic brain injury (American Psychiatric Association, 2013).

**Cognitive deficits.** Cognitive symptoms are correlated with TBI severity and include “fogginess,” difficulty concentrating, memory deficits, and cognitive fatigue. The prevalence of cognitive deficits following TBI has been reported as ranging from 25-70%. Core features of cognitive deficits are inattention, difficulty learning new information, inability to process information, inability to problem solve, and executive dysfunction. It is particularly concerning that neurocognitive deficits have been demonstrated in high school football players with histories of head collisions even when there has been no apparent concussion (Talavage *et al.*, 2013).

**Disordered sleep.** Sleep alterations are common, including difficulty falling asleep, early morning awakenings, and fragmented sleep, and may involve either too much or too little sleep. These disturbances tend to be associated with mild TBI, depression and pain.

**Somatic symptoms.** These are also frequently present, including headaches (up to 78%); dizziness (up to 50%); nausea, and light/sound sensitivity.

**Mood and behavior symptoms.** Disruptions in mood and behavior may include irritability, feelings of sadness, and anxiety.

**Agitation and aggression.** Agitation and aggression are correlated with a pre-injury history of substance abuse and aggressive behavior, and frontal lobe injuries. Core features include verbal outbursts, profanity, destruction of property, and violent attacks.

#### **PSYCHIATRIC DISORDERS ASSOCIATED WITH POST-CONCUSSION SYNDROME**

Significant and prolonged neuropsychiatric complications have been reported in adults following undefined severities of traumatic brain injuries (Nicholl & LaFrance, Jr., 2009). The percentage of patient’s who develop psychi-

atric disorders following mild traumatic brain injury is about 10-40%. At 12 months follow up 22% of patients with mild TBI developed a psychiatric disorder that they had never experienced before. Comorbidity is common, with about 40% of TBI victims having 2 or more psychiatric disorders. Past psychiatric history is a significant predictor of psychiatric illness following TBI. With no past psychiatric history the adjusted relative risk is increased nearly threefold compared to no TBI.

The more common disorders in adolescents include symptoms resembling attention deficit hyperactivity disorder (termed “secondary attention deficit hyperactivity disorder”), disruptive behavior disorders, depressive disorders, anxiety disorders and personality changes (Taylor, 2012). Less commonly mania (sometimes with psychotic features) psychosis, or obsessive compulsive disorder can occur. Substance abuse and suicide are additional complications. Alzheimer’s disease can be a late complication.

Additionally, adolescents presenting with psychiatric symptoms following a head injury may have a psychiatric diagnosis that is related to psychological reactions to their concussion. The differential is quite broad but may include premorbid disorders described above, post-traumatic stress disorder, conversion disorder and factitious disorders (for example, youngsters may exaggerate their symptoms to avoid returning to school). Younger individuals, and those with prior psychiatric history may be at increased risk of post concussive syndrome (Covassin, *et al.*, 2012). Worsening of prior psychiatric symptoms following a head injury is also common.

**Depression.** Depression is the most common mood disorder in TBI with a prevalence of 25-50%, with about 25% meeting criteria for major depressive disorder. Rates are higher in mild TBI vs. severe TBI, and in trauma with TBI compared to trauma without TBI. Depression correlates with injuries in the left dorsolateral-frontal and/or basal ganglia regions (Jorge *et al.*, 2004). Depression is often comorbid with anxiety disorders and aggression. Guskiewicz and colleagues studied retired NFL football players, and found a threefold increased prevalence of depression if they sustained three or more concussions (2007). The presence of depression interferes with rehabilitation and increases the number and severity of other post concussive symptoms.

**Anxiety.** The prevalence of anxiety has been variously reported as being from 10-77%. It typically consists of feelings of apprehension or dread with or without autonomic

signs and symptoms, and is correlated with lesions to the right hemisphere in anxious depression.

**Substance use disorders.** SUDs are a major risk factor for experiencing TBI, often related to assaults, motor vehicle accidents, and so on. Substance use usually decreases shortly after TBI, but then returns, and is increased by one year after TBI.

**Personality changes.** Changes include decreased social perception, decreased control and self-regulation, problems with initiating and planning behavior, emotional change (e.g. apathy, silliness and lability), and difficulties with learning from experience. The most common personality disorders post TBI are avoidant, borderline, and paranoid. About 10% of persons will develop apathy, which correlates with damage to the mesial frontal lobe and its subcortical structures. The core features of this syndrome are lack of motivation, lack of initiative, and absence of dysphoria.

**Suicide.** Suicide is also higher among TBI victims. In a large population-based study in Denmark, Teasdale and Engberg (2001) reported a suicide rate of 0.59% among victims of concussion, threefold higher than in the general population. Interestingly, risk was lower for those under age 20 and over age 60.

**Mania.** Mania is relatively rare, with a prevalence of 1-10%. It correlates with lesions to temporal lobe and right-orbitofrontal cortex. Core features include episodes of irritability and/or elated mood, increased energy, and impulsivity. It may include psychosis.

**Acute stress disorder and post-traumatic stress disorder.** Acute stress disorder can occur in the immediate aftermath of the traumatic injury, and can lead to posttraumatic stress disorder later on. Harvey and Bryant (2000) studied survivors of motor vehicle accidents with mild traumatic brain injury. They found acute stress disorder in 14%; about 70% of these went on to develop PTSD within two years. Posttraumatic stress disorder is more common in mild than in more severe TBI. It includes the typical symptoms of nightmares, hyperarousal, avoidance, re-experiencing trauma, hypervigilance, and dissociative symptoms (e.g. emotional numbing).

**Psychosis.** A variety of symptoms may be present, including transient delusional misidentification, brief confabulation, and delusions and hallucinations. Psychotic symptoms are most common after moderate to severe brain injury. They usually occur in association with abnormalities on MRI/CT and EEG. Negative symptoms are uncommon. Of note, TBI may increase risk for schizophrenia.

## ASSESSMENT

### Initial Assessment

The initial response should be to assess for immediate signs and symptoms. Generally, first responders such as athletic trainers or emergency medical technicians (EMT) will do the initial assessment, looking for signs and symptoms of mild TBI. One useful assessment tool for this initial assessment in athletes is the Sport Concussion Assessment Tool (SCAT 3 <http://bjsm.bmj.com/content/47/5/259.full.pdf>). The assessment looks at signs such as loss of consciousness

(which occur in approximately 4-10% of concussions), balance problems, the Glasgow Coma Scale, a coordination exam, and a cognitive assessment (Meehan, d'Hemecourt, & Comstock 2010).

The initial assessment should include a clear description of the injury, a review of post concussive symptoms, risk factors for protracted recovery and red flags for urgent referral to emergency care (deteriorating condition, significant neurological findings, and so on). The review of post concussive symptoms generally falls within four categories. These categories include 1) cognitive symptoms; 2) sleep changes often with too much sleep at first and sometimes followed by difficulty with falling asleep, staying asleep or sleep continuity disturbances; 3) somatic symptoms such as headaches, dizziness, nausea, visual difficulties, and light and sound sensitivity; and 4) mood and anxiety symptoms such as irritability, depression and nervousness (Romain, 2012).

The Centers for Disease Control (CDC) has put together a number of resources for training schools, coaches, families, health care providers and students on the diagnosis and immediate management of concussion. They have created multiple "Heads Up Concussion" programs as well as information for physicians such as the Acute Concussion Evaluation (ACE) protocol (<http://www.cdc.gov/concussion/headsup/pdf/ACE-a.pdf>). The evaluation includes the characteristics of the injury, the types and severity of the symptoms, signs of deteriorating neurological function, and risk factors that can lead to protracted recovery.

Guidelines for return to play have also been developed (McCrory *et al.*, 2013). These stipulate that athletes should not return in the current game or practice, and should return only after monitoring and medical evaluation using a step-wise process. They should first be symptom free at rest, then symptom free with physical/cognitive exertion, and finally have intact neurocognitive functioning.

It is extremely important to perform a comprehensive clinical evaluation, including performing a physical/neurological exam, and obtain information regarding the developmental history, premorbid functioning and any past neuropsychiatric history. Additionally, information regarding family history (especially neurological and psychiatric concerns), prior concussions and past treatment responses are all very important in determining an appropriate care plan.

### Neuropsychological Testing

Commonly neuropsychological testing is used as a part of the initial and follow-up diagnostic assessment and at this point is considered the standard of care (McCrory *et al.*, 2013). Many brief computerized neuropsychological tests are available for use. Neuropsychological testing generally includes a clinical examination, pencil and paper testing, and computerized neuropsychological tests, such as the ImPACT, Headminder, Automated Neuropsychological Assessment Metrics (ANAM), and CogState.

Some pediatric offices and schools offer the testing on site. One of the more popular tests is the ImPACT test, which is a computerized test that can be given to student athletes to obtain a baseline and then again after an injury to

determine if there are any changes in cognitive function (Iverson, Gagnon, & Griesbach, 2012). If there is no baseline test, the ImPACT test can be compared to age and gender norms. Additionally, if there are questionable results on the computerized test or concerns of lingering cognitive symptoms not demonstrated on the ImPACT test, referral for a complete neuropsychological battery may be appropriate. Neuropsychological testing is only one piece to the diagnosis of mild traumatic brain injury and should not be used in place of clinical and physical examination.

In the acute phase, concussion is generally evaluated and monitored by a primary care physician. Referral to a hospital emergency department is indicated if there is any deterioration of neurological function. Neuroimaging is not indicated for simple concussions, but should be obtained if there is a focal neurological deficit or suspicion of intracranial bleeding. The CDC recommends referral to a TBI specialist if there is no symptom resolution over 3-5 days (other recommendations are available at Heads Up Facts for Physicians [http://www.cdc.gov/concussion/headsup/pdf/Facts\\_for\\_Physicians\\_booklet-a.pdf](http://www.cdc.gov/concussion/headsup/pdf/Facts_for_Physicians_booklet-a.pdf)). The treatment of post concussive syndrome generally requires a team based approach to manage the complexity of managing return to sports, school, work, and so on. Other specialists who may be involved, depending on the complexity of the situation, include neuropsychologists, physiatrists, neurologists, psychiatrists, and others.

## MANAGEMENT

Treatment with appropriate education, reassurance and rest may lead to prompt resolution of symptoms (Iverson *et al.*, 2012). Typical recovery occurs in 7-10 days; 80% of athletes recover spontaneously within three weeks of trauma (Meehan & Bachur, 2009).

### Rest

The cornerstone of the initial approach is rest (for both the brain and body). Body rest means not only abstinence from competitive sports but any activities requiring physical exertion (running, bicycling, weight lifting and so on). Sleep hygiene is important to ensure rest as well as to deal with sleep disturbances associated with TBI. Patients should be told to: 1) only go to bed when tired; 2) lie in bed no more than 20 minutes; 3) relax each night before bed; 4) wake up at the same time every morning; 5) avoid taking naps; and 6) avoid any caffeine after lunch. Mental rest is more difficult to define, but refers to activities requiring heavy concentration or focus, memory, reading or writing. This means no homework or classwork, as well as other activities commonly engaged in by adolescents, such as playing video games, or texting (Centers for Disease Control, n. d.; Meehan, 2011). Parents need to be actively involved in helping assure adequate rest. Common activities to consider during this time of brain and body rest include relaxing art projects or simple cooking.

### Education and Support

Reassurance and education may be particularly helpful shortly after injury. One study with children using early education and reassurance demonstrated fewer symptoms at 3

months following the intervention, which consisted of an information booklet (Ponsford *et al.*, 2001).

### Accommodations

Schools are required to develop and implement plans to accommodate to students' disabilities, and TBI will often require changes in the school environment, attendance policies, homework expectations, testing demands, the curriculum, and the methodology of teaching.

### Cognitive Behavioral Therapy

Cognitive behavioral therapy (CBT) and relaxation techniques can be helpful in dealing with the stress associated with TBI. One study looked at adults who had experienced a traumatic event within two weeks, and who had mild traumatic brain injury and acute stress disorder. They were treated with 16 sessions of either CBT or supportive therapy. The group who received CBT showed significantly less chance of developing post-traumatic stress disorder at six months (8% vs. 58%) (Bryant *et al.*, 2003). Relaxation exercises may include deep breathing, progressive muscle relaxation, calming visualization, and meditation.

### Ongoing Treatment

A stepwise, gradual return to normal activities can be implemented, once return of functioning to pre-injury levels occurs. All of the guidelines recommend a gradual return to playing sports after a period of rest, followed by increasing levels of activity, beginning with aerobic exercise (low), sports specific (moderate), non-contact (heavy), and eventually progressing to full contact and game play. Patients need to be monitored for return of symptoms upon resumption of activities.

If symptoms and deficits persist, the next management approach is generally more targeted treatments aimed at specific deficits or problems. Interventions that can help to improve cognitive functioning include teaching strategies to increase attention, memory, concentration, and retention, adapt study skills, and improve organization and planning.

Two comprehensive approaches are rehabilitation psychology and cognitive rehabilitation therapy. Rehabilitation psychology is a specialty field of psychology that deals with helping people with disabilities maximize their function (Riggar & Maki, 2004), often in the context of a comprehensive interdisciplinary program. Techniques used include assistance with coping, pain management, relaxation training, education, and cognitive behavioral therapy. Specific approaches for anxiety and depression in the context of disabilities have been developed.

Cognitive rehabilitation therapy focuses on helping brain-injured or otherwise cognitively impaired individuals to restore normal functioning, or to compensate for cognitive deficits. In addition to teaching problem solving skills, it teaches patients to become aware of and monitor the effectiveness of these skills. It may include occupational or speech therapy. Studies have shown improvement in neuropsychological testing, cognitive functioning, post-concussive symptoms and attention with this approach. However, it is unclear if effects are due to practicing, if the results are due

to lack of control groups, or if the treatment really leads to improved functioning. An Institute of Medicine report (2011) noted deficiencies in the evidence base for this treatment, but recommended that its use continue while it is studied systematically.

Symptoms associated with balance are typically treated with vestibular therapy, which may include exercises to address balance problems, dizziness, nausea, blurred vision and headaches. Visual concerns, such as problems with convergence, accommodative, and oculomotor dysfunction are common and can be treated with optometric vision therapy (Ciuffreda *et al.*, 2008). Deconditioning as a result of physical inactivity can be treated with physical therapy, including conditioning exercises.

Occupational therapists evaluate for gross and fine motor deficits, sensory processing or adaptive behavior differences, and teach strategies for activities of daily living, and identify solutions to barriers. Speech therapists may be involved in the assessment and treatment of speech and/or language disorders related to cognitive impairment. These may include difficulties with attention, lexical access, complex lexical-semantic manipulation, response monitoring, and organization, all of which affect social communication (Duff, Proctor, & Haley, 2002).

### Pharmacological Treatment

There currently are no medications that are FDA approved for the treatment of neuropsychiatric consequences of traumatic brain injury. The evidence base is limited for existing medications, especially with respect to children and adolescents and mild TBI; most studies have been done with adults and/or individuals with moderate or severe traumatic brain injury, and few are randomized controlled studies. A review by Warden *et al.*, (2006) noted support for the use of methylphenidate but cautioned, "There is insufficient evidence to support any standards or guidelines for the treatment of affective disorders, mania or psychosis in the TBI population."

Nonetheless, many different medications have been tried, and there are some expert consensus guidelines (Comper, Bisschop, Carnide, & Tricco, 2005; Lombardi, 2008; Warden *et al.*, 2006). In addition, the Veterans' Administration and Department of Defense have issued a 109 page document of clinical guidelines (2009).

Because of the limited evidence base for pharmacotherapy, the risks involved, and the fact that many symptoms will resolve spontaneously, caution is warranted in the use of medications. The keys to treatment include first waiting for symptoms to go away on their own, then utilizing medication at low doses with slow titrations watching carefully for side effects, especially seizures, extrapyramidal symptoms and anticholinergic effects on cognition (Fleminger, 2008). Drugs can also worsen agitation and confusion. The clinician should discontinue any medications that don't work, and should watch for drug-drug interactions.

**Cognitive deficits.** The treatment of cognitive deficits generally includes stimulants such as methylphenidate and cognitive enhancers such as donepezil or amantadine (Tenovuo, 2006). Questions have continued regarding the

effectiveness of psychostimulants for TBI in children and adolescents since an early study showed no apparent benefit (Williams, Ris, Ayyangar, Schefft, & Berch, 1998) but they are commonly used and some studies have demonstrated improvements in attention, working memory, and processing speed with methylphenidate at a dose of 0.25-0.30 mg/kg BID. It is generally considered the first line pharmacotherapy for attention difficulties.

Donepezil at a dose of 5-10mg daily increases attention and memory and is a first line drug for memory problems. Amantadine is a reasonable option for improving cognition and agitation and can be considered as a first line drug for executive dysfunction. There have been a few small studies using amantadine in adolescents with all types of brain injury (Beers, Skold, Dixon, & Adelson, 2005; Green, Hornyak, & Hurvitz, 2004; Patrick *et al.*, 2006). One small randomized but non-blinded study compared amantadine to usual care in a group of children and adolescents with various degrees of TBI (Beers *et al.*, 2005). Amantadine was found to be safe; behavior improved in the group as a whole, and cognitive performance improved in a subgroup who had had their injury within 24 months. However, it is likely this study was underpowered to detect differences. Bromocriptine 2.5mg also improves executive function (Patrick *et al.*, 2006).

**Depression.** In the treatment of adult depression following MTBI, sertraline appears to have the best evidence and may be effective in doses as low as 25mg (Warden *et al.*, 2006); this drug is also generally well tolerated in adolescents. SSRIs may lower the seizure threshold in this population, however. Methylphenidate has also been shown to improve mood (Lee, 2005 and Gualtieri, 1998) and may be an option in the presence of attentional difficulties together with depression. Data are very limited on SNRIs, and bupropion should be used very cautiously due to increased seizure risk with this drug. Tricyclic antidepressants (TCAs) should be avoided in those with cognitive dysfunction due to anticholinergic effects.

**Disordered sleep.** Melatonin at a dose of 1-10mg qhs can be effective when combined with good sleep hygiene techniques (Clayton, 2012). Alternatives include trazodone (25-200mg qhs) and mirtazapine (7.5-15mg qhs). Anticholinergics generally should be avoided, however in clinical practice tricyclic antidepressants are frequently prescribed. Close monitoring of worsening cognitive functioning is warranted when these medications are used.

**Mania.** First line treatment is valproate. Lithium is a second line drug because it lowers seizure threshold. Cases series data support the use of lithium 900mg/day, valproate up to 750mg-1000mg/day, clonidine 150-600 µg/day, thioridazine 50mg/day with amitriptyline 100mg/day, and ECT. Valproate and carbamazepine are useful as they do not worsen cognition.

**Aggression.** A 2006 Cochrane review of treatment of MTBI in adults, although noting the inadequacy of the evidence, reported that the best evidence was for beta-blockers, e.g., propranolol at a maximum dose of 420-520mg/day, and pindolol at a maximum dose 40-100mg/day (Fleminger, Greenwood & Oliver, 2006). Atypical antipsychotics, specifically ziprasidone at a starting dose of 5mg/day, have been

studied for use in aggression and/or agitation in children and adolescents and found to be safe and effective (Scott, Green, McCarthy, & Conrad, 2009). Evidence is very limited, however (this study was a case series). Other alternatives include olanzapine (5-10mg/day), risperidone (0.25-0.5mg /day), quetiapine starting at 25mg BID, ziprasidone, and aripiprazole starting at 5mg/day.

**Psychosis.** Data are limited to case reports. Most authors have recommended atypical antipsychotics as described in the section on treatment of aggression, and typical agents (e.g. haloperidol) may not be as beneficial (Guerreiro, Navarro, Silva, Carvalho, & Gois, 2009; Fujii & Fujii, 2012).

## CASE EXAMPLE

The following case illustrates a typical approach to management in an outpatient psychiatric practice.

A 14 year old female presented to my office with her mother two months after a concussion during a sporting event. Her mother's chief concern was about her daughter's significant anxiety following the concussion. The adolescent had developed difficulty with headaches, focus, concentration and fatigue. She had been assessed and treated by a family practice sports medicine physician specializing in concussion. She initially had a low neuropsychological test score on the ImPACT. Due to her symptoms she was held out of school and she recovered nicely with brain and body rest over the next 2 months. She returned to school for the last few days of the school year. She had been given academic accommodations allowing for minimal academic pressure upon her return and the goal of her return was mainly to help her reintegrate socially into the school environment. However, upon returning to school she experienced significant anxiety, to the point that she was unable to remain through the school day. She was then referred for psychiatric treatment.

She presented with complaints of continued cognitive concerns even though she had returned to her supposed baseline on the ImPACT test. I worked with her and her mother starting with education about post-concussion syndrome and recovery, and explaining that it can take a long time to regain full cognitive functioning. We also discussed anxiety and began cognitive behavioral therapy. I found that she was easily overwhelmed with lists of tasks to do and struggled to prioritize, leading to her feeling stuck not knowing what to do and anxious. I worked on coping skills and accommodations at home to decrease her stress. Over the course of a few sessions she was able to utilize her coping skills and felt more aware and in control of her anxiety. She also began to feel that her focus and attention were returning to baseline. At this point I presented her with more complex tasks in session and at home and she was able to complete them without anxiety. She reported no further post concussive symptoms or anxiety. She was discharged to enjoy her summer.

## CONCLUSIONS

The recognition of the frequency of concussions in young athletes and their significant impact on cognitive and emotional functioning is a relatively recent development. The

risk of post-concussion syndrome, with its associated cognitive, emotional, and behavioral problems has also only recently been acknowledged. Psychiatrists, psychologists, and other mental health professionals may see patients who present with various psychiatric symptoms and disorders, whose complex symptomatology is due to a mild traumatic brain injury. It has been the author's experience that children and adolescents who see a psychiatrist following a concussion often present with symptoms meeting full criteria for mood, anxiety or neurodevelopmental disorders. These patients can be challenging to treat and it can be difficult to identify the part PCS is playing in their psychopathology. Although these adolescents often resist a referral for mental health treatment, without appropriate management these problems may become chronic.

Knowledge about risk factors and diagnosis of concussion and post concussive syndrome is fairly good; the level of evidence for management is primarily at the level of expert consensus. While the state of our knowledge is still incomplete regarding these conditions, at this point it can be said those individuals with no prior head injuries or past history or family history of any psychiatric disorders prior to their injury often demonstrate complete recovery in a relatively short time with appropriate management. Individuals with premorbid mental health conditions and/or repeated head injuries often can have a prolonged recovery and chronic symptoms. PCS is a complex disorder and treatment generally requires a multidisciplinary approach. Even in such cases significant improvement is possible with appropriate treatment.

Many questions are unanswered at this time. Yet clinicians are faced with making decisions about youngsters in the here and now. Two strong recommendations are consistent across all guidelines 1) physical and mental rest, and 2) cessation of involvement in sports until all symptoms are gone (and all medications for the purposes of PCS have been discontinued). Given the long road to recovery for some adolescents, return to school while still symptomatic is a matter of clinical judgment and the risk of being unable to meet the demands of the academic environment has to be balanced against the negative consequences of social isolation.

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## DISCLOSURES

The author reports no conflicts of interest.

## CONFLICT OF INTEREST

The authors confirm that this article content has no conflicts of interest.

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