

# Mild Traumatic Brain Injuries: Evaluation and Care

*The most common head injury in athletics is concussion. DCs should be alert for its symptoms.*

by **Bill Moreau, DC, DACBSP**

People who have had mild traumatic brain injury (TBI) can often present to a DC's office. It is not just the athletic population that sustains concussions. DCs need to be aware of the presenting symptoms and guidelines of care for people who present for care with a mild TBI. Head injuries are typically classified into the basic categories of severe or minor. Concussions are classified as minor TBI, and severe TBI includes injuries like cerebral contusion and intracranial hemorrhages. This article will examine the DC's role in the evaluation and care of mild TBI.

## **What is a mild TBI?**

The severity of TBI is defined by its acute injury characteristics, not by the severity of symptoms. Mild TBI is characterized by the following:

1. Head trauma may be due to contact forces or to acceleration/deceleration trauma. This includes motor-vehicle collisions and assaults.
2. The duration of unconsciousness is brief, usually seconds to minutes, and in some cases there is no loss of consciousness (LOC) but simply a brief period of dazed consciousness.
3. When the patient was evaluated in the emergency room or at the scene, the Glasgow Coma Scale (GCS) must have been 13 to 15. The GCS is scored between 3 and 15, 3 being the worst, and 15 the best. Only a score of 15 probably represents true mild TBI. A score of 13 or 14 is due to confusion or disorientation and will usually be associated with a longer period of amnesia.
4. Whether the patient is briefly unconscious or not, confusion with amnesia is present for minutes to a few hours.
5. When evaluated, the patient has no focal signs of neurologic injury, although in the immediate post-traumatic period, patients may be pale, diaphoretic, nauseated, and ataxic.
6. Neuroimaging studies are negative.

The most common mild TBI is called a concussion. Do not confuse the word "mild" with "insignificant." All concussions are significant. The study of concussion is still undergoing transition. A concussion is defined as an immediate and transient impairment of neural function secondary to trauma. The altered neuronal function includes such symptoms as alteration of consciousness, disturbance of vision, equilibrium and other similar symptoms. When a person states he has had his "bell rung," he has most likely sustained a concussion. A person does not need to be rendered unconscious to have sustained a

concussion. Remember to add this assessment to motor-vehicle-collision patients who cannot recall the events associated with the car accident when the loss of recall is related to head or neck trauma from the accident. The International Classification of Diseases, Adapted code number for cerebral concussion is 851.0.

All DCs need to be alert for concussion symptoms, especially in the athlete.

The most common head injury in athletics is concussion. More than 250,000 concussions take place annually in football alone.<sup>1</sup> The annual incidence of concussion for a single football player is typically identified in the literature as ranging from 3.6% to 47%.<sup>2</sup> I recently studied the reporting instance of concussion in high school football players and found the incidence rate to be 65%.<sup>2</sup> This study demonstrated a concussion occurrence rate that was much higher than the typically reported incidence rate.

Another interesting fact about the mechanisms of injury was that concussion may be caused either by a direct blow to the head or by an impact elsewhere on the body, resulting in an impulse force transmitted to the head. This is how passengers in a motor vehicle accident can sustain a TBI without actually striking their head. When there is trauma sufficient to result in diffuse axonal injury (DAI), it seems reasonable to anticipate there would be concomitant suboccipital and cervical spine problems that would be amendable to chiropractic methods.

When the brain absorbs energy that exceeds the ability of the body to dissipate the force of the energy successfully, tissue damage typically occurs. The neuropathology mechanisms of how TBI occurs are still being evaluated. The acute clinical symptoms of TBI usually reflect a functional disturbance secondary to structural damage to DAI. The shearing forces generated in the brain by sudden deceleration most commonly cause DAI. The magnitude of DAI changes are proportional to the deceleration force. Therefore "more force means more injury." The duration of unconsciousness and duration of post-traumatic amnesia are probably the best indicators of the severity of the DAI. These findings are definite indicators of a loss of neurologic function. This loss of function will then result in characteristic features that identify TBI. Identifying the symptoms of concussion and the length of time these symptoms have been present will then allow the practitioner to apply a grade to the concussion. The acute symptoms of concussion have been examined in many studies. The only validated symptoms are amnesia, loss of consciousness, headache, dizziness, blurred vision, attention deficit, and nausea.

Some symptoms of a more severe brain injury may be present. The evaluation and workup of TBI patients should include more than an evaluation of their thinking skills. Physical evaluation is also very important. The physical examination list for TBI includes

- vital signs;

- mental status evaluation;
- orientation—time, place, person, and situation;
- concentration—recite the months of the year backward;
- memory—current events and previous events;
- provocation of symptoms with exercise;
- eye examination including pupils (anisocoria) and reactions to light;
- coordination—finger to nose, tandem gait, Romberg;
- superficial sensation; and
- ortho/neuro examination of the spine as indicated.

Anisocoria is an unequal size of the pupils. Many benign and life-threatening etiologies are associated with anisocoria. The trick is to determine if the anisocoria is a pathologic finding or not. If the anisocoria is pathologic, this is a real problem and it may be the harbinger of an emergency situation. Simple or physiologic anisocoria occurs in approximately 20% of the population. To determine if the anisocoria is pathologic, you will need to observe the pupils' reaction to light. This quickly narrows the differential diagnosis. A pupil that responds sluggishly to light is likely to be abnormal. An abnormal light reflex implies denervation, damage to the pupillary sphincter, or local ocular disease. In the absence of eye pain, a history of direct eye trauma, redness, or decreased acuity, the presence of acute injury becomes less likely. In these patients, pay special attention to the presence of ptosis or oculomotor palsies. If you perform preparticipation examinations, make sure you note benign anisocoria on the examination form.

### **TBI and Children**

Special attention needs to be provided to children who sustain TBI. Because of the lack of research on children and mild TBI, there are no hard guidelines to apply clinically. It is difficult to evaluate and provide treatment plans across the early age groups and developmental phases for children with TBI. Doctors are left to make deductions from guidelines developed for adult populations or to call upon their clinical experience to make decisions. One thing known is that children do not tolerate TBI as well as adults. Young people should typically be evaluated in an emergency room setting if they have had a trauma with an associated loss of consciousness.

The evaluation of children should include a careful review of the mechanism of injury. With unexplained injury, be alert for possible abuse. Many children will vomit once or twice after head trauma. Common emergency room recommendations suggest that if the vomiting is persistent, the child must be evaluated. I would suggest that if the child has a history of head trauma and vomiting he should be sent to the emergency room for evaluation. Infants (age 2 and less) with signs and symptoms of any TBI need to be evaluated in the ER.

Instructions should be provided to the acute mild TBI patient on when to contact you or when they should report to an emergency room. Following is an example.

- 1) Avoid strenuous activity for 24 hours after the injury.
- 2) Do not take medications without consulting your physician.
- 3) Eat a light diet such as soup, toast, or crackers for 24 hours after the injury.
- 4) Avoid alcohol.
- 5) Do not take aspirin, sleeping pills, or ibuprofen.

Patients should also contact you or go to the ER if they

- become sleepy or are difficult to awaken;
- vomit;
- have trouble with balance;
- experience odd eye movement, have difficulty focusing, or show unequal pupil size;
- have persisting or increasing headache;
- experience restlessness, irritability, or personality changes;
- have convulsions or seizures;
- experience new swelling at the area of the head injured;
- have increased neck stiffness;
- experience numbness;
- experience ringing in the ears;
- experience shortness of breath;
- experience confusion; or
- experience visual problems.

The clinician also needs to be aware of how to track the resolution of a patient's symptoms related to mild TBI. Baseline testing of neuropsychological function is an important method of performing this. It is recognized that preinjury testing can be useful in addressing the recovery from a TBI. Several software programs have been offered as a tool for evaluating an individual's recovery from a TBI. One of the problems with these approaches is that preinjury evaluation is important, and these types of evaluations are typically associated only with the athletic population.

The appropriate use of neuropsychological testing, grading scales, and return-to-play recommendations are outside the scope of this article. Interested practitioners are encouraged to pursue additional training by obtaining the Certified Chiropractic Sports Physician or Diplomate in Neurology certification. Additionally, areas requiring further research need to be identified, and future trends will need to be monitored to follow the evolution of care for mild TBI. CP



#### **Case Study**

As the team doctor for a high school football team, I noticed that one of the players, a 17-year-old male, was resting on a knee behind a group of his teammates during a practice. When I asked him if he was all right, he looked up with a glazed look in his eyes and slowly responded, "I think so." He was in a mental fog, and further questioning quickly demonstrated that he was unable to promptly answer simple questions. For instance, he knew his mother had blond hair, but he could not recall her name. He was also unable to recall

his home phone number. When he was asked any question about recent information that was just provided to him, he was unable to remember anything that he was just told. The player had sustained a head injury, and he was not processing either current or past memory. The coaching staff was alerted that the player most likely had sustained a significant concussion. No one knew that he had been injured. His mental fog was not clearing, and he was transported to the local emergency room.

Forty-five minutes after arriving at the emergency room, he still could not recall how he got there. He mentioned several times that he felt like he was dreaming. A CT scan of his head was ordered, and fortunately this study did not show signs of intracranial bleeding or injury. Three hours later, he started to clear his thoughts and he was released to go home with his parents.

The brain can be compared to a computer regarding the types of processing that take place. The computer has a hard drive that stores old information, and random-access memory (RAM) also processes current activity. The brain also processes old memory while it maintains current memory processing. The player's mental hard drive and RAM were "crashing," in the lexicon of computer terminology. The player sustained a significant traumatic brain injury (TBI) called a concussion.

One week later, the player presented for care and evaluation. He was back in school, but he had persisting dizziness with head movements, headache, and difficulty concentrating. Research indicates athletes with persistent foggy experience many other post-concussion symptoms compared to the athletes with no reported foggy. In addition, the athletes with persistent foggy had significantly slower reaction times, reduced memory performance, and slower processing speed, which result in the symptoms that the player was having.

Patients like this one who have sustained mild TBI often report a group of physical, cognitive, and behavioral symptoms referred to as post-concussion symptoms (PCS). The most commonly reported post-concussion symptoms are headache, dizziness, decreased concentration, memory problems, irritability, fatigue, visual disturbances, sensitivity to noise, judgment problems, depression, and anxiety. Although these symptoms often resolve within 1 month, in some individuals PCS can persist from months to years following injury and may even be permanent and cause disability. Findings suggest that organic brain primary damage may account for the occurrence of PCS.

The player's primary complaint was associated with nonvertigo dizziness. A multitude of conditions can cause vertigo or dizziness. There is evidence that vertigo can be produced by cervical disturbance, by either the proprioceptors of the cervical spine or through the vascular supply by the vertebral artery. A functional examination of the upper cervical spine is important in diagnosing and treating "cervical vertigo," because nonresolved joint dysfunctions of the upper cervical spine are a common cause of long-lasting dizziness. An important distinction is made when differentiating vertigo from nonvertigo.

Vertigo is the true rotational movement of self or the surroundings. Nonvertigo includes light-headedness, unsteadiness, motion intolerance, imbalance, floating, or a tilting sensation. Knowing this is helpful, because true vertigo is often due to inner ear disease, whereas nonvertigo symptoms may be due to central nervous system, cardiovascular, or systemic diseases.

The player's physical examination revealed upper cervical spine joint dysfunction or subluxation complex. Over the next few weeks, he was cared for with traditional manual chiropractic adjustments and soft-tissue work—which included myofascial release—to the involved areas. He

one time per week for 2 weeks, and one followup at the end of care.

He demonstrated slow and steady improvement of his complaints. It was also important that the player be held out of all contact sports until well after his symptoms resolved to avoid the further potential for a more serious head injury.

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### **References**

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