[Primary Care]

CEU

Concussion in Sports

Heather J. McCrea, MD, PhD,* Kenneth Perrine, PhD,* Sumit Niogi, MD,* and Roger Härtl, MD*†

Context: Recently, concussion has become a topic of much discussion within sports. The goal of this review is to provide an overview of the literature concerning the definition of concussion, management of initial injury, return to play, and future health risks.

Evidence Acquisition: This article reviews the most recent findings on recognizing and managing sports-related concussion, which has become a significant health risk. We reviewed articles from the literature discussing concussion and its effects.

Results: Though concussion patients typically have negative head imaging, imaging is warranted in those with severe mechanism, significant loss of consciousness, focal neurologic deficit, or worsening symptoms. The existence of "second-impact syndrome," whereby a first minor head injury predisposes an athlete to later catastrophic injury, remains controversial; however, it is clear that concussion has significant effects on a patient and should be considered carefully in return-to-play decisions.

Conclusions: A comprehensive understanding of concussion and its related risks is important in making return-to-play decisions as well as health care and league policy.

Keywords: concussion; sports concussion; return to play; mild traumatic brain injury

Each year, approximately 1.4 million people are hospitalized with traumatic brain injury (TBI) in the United States, and an estimated 57 million people worldwide suffer from a TBI[8] (Table 1). Of these injuries, 20% occur secondary to physical activity and sports.[18] The Centers for Disease Control and Prevention estimates that 1.6 million and 3.8 million treated and untreated concussions related to sports occur each year in the United States, respectively.[16] Recently, concussion has become an important issue of discussion in athletics, as an understanding of the long-term effects of mild brain injury and concussion has begun to emerge. Repeated loss of consciousness for more than 30 minutes may increase risk of Alzheimer's disease, and amyloid proteins are seen as early as 2 hours after severe TBI.[4] More recently, as autopsies have been conducted on boxers with dementia pugilistica (punch-drunk syndrome) and deceased football players, it has become apparent that repeated concussion even without more severe injury may put some athletes at risk for cognitive and psychologic problems in the future.[4] Consequently, it is important for those involved in athletics to recognize concussion, assess patients for concussion, rule out more severe injury, and safely navigate return-to-play decisions for these players.

DEFINITION OF CONCUSSION AND MILD TBI

A definition of concussion is difficult because of the lack of objective clinical and radiographic findings. There are usually no findings on routine imaging, such as computed tomography (CT), which makes the definition of concussion less clear than definitions of other types of brain injury. Definitions have been offered by the Department of Defense, as have consensus statements from the Concussion in Sport Group from Vienna[1] and Prague.[13] The most recent and agreed-on definition of concussion is that set out in the Zurich consensus statement,[7] which refined the Vienna and Prague definitions. The Zurich statement defines concussion as "a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces." This definition does not require loss of consciousness to diagnose concussion. Other features frequently present in concussion are a brief period of retrograde amnesia (impaired recall of events just prior to the injury) and posttraumatic amnesia (impaired recall for the time between the injury or resumption of consciousness and the point at which new memories are stored and retrieved). For practical purposes, the Cantu Grading Scale has been helpful in assessing severity

From *Weill-Cornell College of Medicine, New York, New York.
†Address correspondence to Roger Härtl, MD, Department of Neurological Surgery, Weill-Cornell College of Medicine, 525 East 68th Street, Box 99, New York, NY 10065 (e-mail: roger@hartlmd.net).
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Table 1. Epidemiology of traumatic brain injury (TBI) in the United States.[a]

Approximately 1.4 million people in United States with TBI per year
Contributing factor in 30.5% of injury-related deaths
Concussion or other mild TBI accounts for 75% of TBI
20% of TBI secondary to physical activity and sports
1.365 million people treated and released by the emergency room each year for TBI
275 000 people hospitalized for TBI each year
52 000 deaths from TBI each year
Most common in ages: 0-4, 15-19, and > 65 years
Direct medical costs and indirect costs (eg, lost productivity) of TBI totaled an estimated $60 billion in the United States in 2000

[a]Adapted from http://www.cdc.gov/traumaticbraininjury/statistics.html.

of concussion and for making return-to-play decisions. Grade 1 includes posttraumatic amnesia less than 30 minutes and no loss of consciousness. Grade 2 is defined as loss of consciousness less than 5 minutes or amnesia 30 minutes to 24 hours, and grade 3 includes loss of consciousness greater than 5 minutes or amnesia greater than 24 hours.[2]

Mild TBI is a different yet potentially overlapping entity. As with concussion, various definitions have been offered by the American Congress of Rehabilitation Medicine, the Centers for Disease Control and Prevention, the World Health Organization, and the American Academy of Neurology. A synthesis of these definitions from the World Health Organization includes a TBI resulting in a score of 13 to 15 on the Glasgow Coma Scale (a 15-point scale combining motor response, eye opening, and verbal response), without other factors, such as acute substance abuse, other focal or systemic injuries, coexisting medical conditions, or penetrating craniocerebral injury. In addition, it includes one of the following: confusion or disorientation, less than 30 minutes of loss of consciousness, posttraumatic amnesia of less than 24 hours, or transient neurologic abnormalities.

Moderate and severe TBI represent the ends of the TBI spectrum. The Glasgow Coma Scale score at the time of injury is lower in moderate[8-12] and severe[3-9] TBI.[7] There is a longer duration of loss of consciousness (greater than 30 minutes) and a much longer period of posttraumatic amnesia that can extend from weeks in moderate TBI to months or even years in severe TBI. The period of retrograde amnesia is also more extensive. There is a much greater likelihood of focal neurologic deficit in both moderate and severe TBI, at the time of injury as well as in recovery. Similarly, positive neuroimaging is often found, which could include subdural or epidural hematoma, intracerebral hemorrhage, and contusions as well as diffuse axonal injury or encephalomalacia in later stages. There is also a stronger possibility of persistent disability in moderate TBI and a nearly inevitable disability to some extent in severe TBI.[10]

The set of clinical symptoms that accompany concussion can be classified into 4 categories: physical, cognitive, emotional, and sleep disturbance. The symptoms and signs can be assessed with a checklist such as the Pittsburgh Post-Concussion Scale. The most commonly recognized symptom is headache or a feeling of being "in a fog." However, more subtle issues can also suggest concussion, such as emotional lability, irritability, slowed reaction times, and drowsiness.

Resolution of the clinical and cognitive symptoms typically follows a sequential course; however, it is important to note that in a small percentage of patients, postconcussive symptoms may be prolonged. After 7 to 10 days, most concussed patients (80%-90%) are symptom free.[14] However, certain patient groups, including younger patients and those with previous concussive episodes, may require longer recovery times..

EVALUATION OF INJURY ON THE FIELD: ASSESSMENT OF COGNITIVE FUNCTION AND THE CERVICAL SPINE

After a patient with a suspected concussion is identified, standard medical procedures should first be applied, including that of ensuring an open airway and adequate circulation. Stabilizing the cervical spine should be conducted if C-spine injury is suspected or cannot be ruled out—for example, with clouded or loss of consciousness. In such cases, the patient should be transported on a longboard. Evaluation of the patient should be done by a health care provider. If one is not available on scene, the patient should be urgently referred to a physician off-site. Serial monitoring to detect any deterioration in mental status is critical over the first few to 24 hours following injury. The patient should not be left alone or allowed to sleep continuously during this time. Except in rare cases of adult athletes with comprehensive sideline teams to rule out concussive injury, a player with concern for concussion should not be allowed to return to play on the same day. Concussion symptoms may be immediate after injury, but symptoms can also appear in a delayed fashion several hours postinjury, so it is important to stay vigilant.

The sideline assessment of concussion is best accomplished with standardized instruments that are widely available. The Sport Concussion Assessment Tool–2 (SCAT-2)[14] has been adopted by nearly every professional sports team and many college and even high school teams. The SCAT-2 includes several independent tests imbedded within it. The Glasgow Coma Scale[7] is standard for assessing level of consciousness on a 3 to 15 rating scale based on best eye response, best verbal response, and best motor response. A crucial component of the sideline assessment is the Standardized Assessment of Concussion,[11] which includes orientation questions, verbal learning of 5 words over 3 trials, digit span backward, and

a delayed recall of the 5-word list. Cerebellar testing should be included, with at least a test of finger to nose requiring 5 accurate repetitions that can be supplemented with the Balance Error Scoring System,[5] which assesses maintenance of balance on both legs, single-leg stance (nondominant leg), and tandem stance. In addition to these independent tests, the SCAT-2 includes a Physical Signs Score, which documents the presence of loss of consciousness or confusion and the presence of balance/unsteadiness by simple observation. The Modified Maddocks Score[9] is used to assess orientation by asking the athlete about game events that day and the week before. Medical assessment includes history and neurologic examination, including an emphasis on mental status, cognitive function, balance, and gait. An understanding of whether symptoms have improved, worsened, or remained the same since injury is also important. Combining information from the history and physical examination with an understanding of symptom severity allows the health professional to determine what course of action to take. Transportation to the emergency department by ambulance is recommended if there is any suspected spinal cord involvement, focal neurologic deficit on segmental examination, worsening of condition suggestive of intracranial hematoma, indicators of a skull fracture (otorrhea/rhinorrhea, raccoon eyes, Battle's sign), bleeding from the nose or ear, or a seizure. If the patient is symptomatic with moderate to severe postconcussive symptoms but stable (ie, not declining and having none of the preceding factors), she or he may be transported to the emergency department by a parent or guardian. If the patient is not transported, the parent or guardian should be alerted and provided with the Parent's Fact Sheet, available from the Centers for Disease Control and Prevention (http://www.cdc.gov/concussion/HeadsUp/high_school.html). The fact sheet includes instructions not to leave the patient alone and to bring him or her to the emergency department if any of the following is present:

- a headache that worsens,
- drowsiness or inability to be woken up,
- inability to recognize people or places,
- repeated vomiting,
- worsening confusion/irritability,
- seizures,
- hemiparesis/hemisensory loss,
- unsteadiness, or
- slurred speech.

The parent/guardian is also encouraged to keep the patient at rest and, for at least 24 hours, have him or her avoid

- strenuous activity,
- alcohol,
- recreational drugs,
- sleeping medication (can mask a subdural hematoma), and
- aspirin or nonsteroidal anti-inflammatory drugs (can worsen bleeding).

Patients are also encouraged to abstain from driving and avoid exercise or play until medically cleared. Red flags suggesting that a patient may require neurosurgical intervention in the future include

- Glasgow Coma Scale score less than 15 at 2 hours after injury,
- suspected open skull fracture or basal skull fracture,
- vomiting even once,
- decrease in Glasgow Coma Scale score with time,
- severe headache,
- restlessness, and
- focal temporal blow.

Additional medium-risk factors include retrograde amnesia before impact of greater than 30 minutes or dangerous mechanism of injury.[19]

NEUROIMAGING

Imaging may be warranted in patients with prolonged loss of consciousness, severe mechanism, worsening symptoms over time, or focal neurologic deficit. In all of these cases, risk of an underlying structural lesion (subarachnoid hemorrhage or intraparenchymal, subdural, or epidural hematoma) in the brain is more likely than in the typical concussion patient and thus should be ruled out. CT imaging is a sensitive and readily available test to detect bone fractures and hemorrhage, and it should be used if the patient demonstrates deterioration in clinical examination (Figure 1). In stable patients, magnetic resonance imaging (MRI) can detect shear injury in the white matter, which would be missed on CT. In the future, functional MRI and other highly specialized imaging technologies (positron-emission tomography scan, magnetic resonance spectroscopy, and diffusion tensor imaging) may prove useful in evaluation of concussion, but at this stage, they are still experimental research tools.[14] In particular, diffusion tensor imaging (DTI), a special type of MRI scan, appears to be promising in detecting diffuse axonal injury.[17] Diffuse axonal injury arises from acceleration/deceleration forces that cause shearing injury in the white matter. These injuries may be missed on conventional MRI but may be readily detected with DTI, which better images white matter tracts (Figure 2).[17] A recent study demonstrated that injuries found on DTI correlated with cognitive reaction time, suggesting that DTI may be a useful component of assessing injury in the future and may help making return-to-play/activity decisions.[17]

Cervical spine imaging should be considered in cases with neck pain or clinical suspicion of involvement of the cervical spine. Clinical suspicion is usually raised by mechanism of injury or symptoms such as numbness, tingling, and weakness. Fine-cut CT scans are sensitive and have replaced regular X-rays for the assessment of spinal trauma. Dynamic flexion/extension lateral X-rays are still used if ligamentous injury to the cervical spine is suspected. MRI is also frequently used if ligamentous injury is suspected.

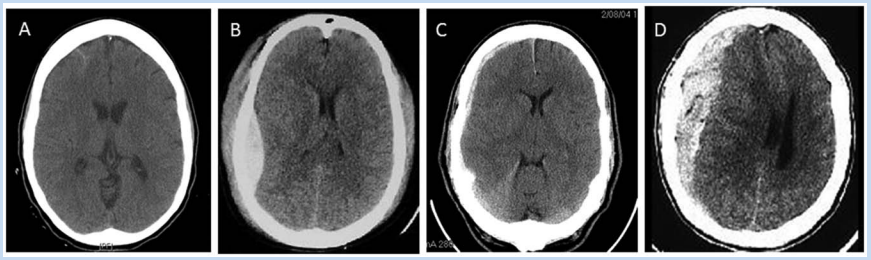

Figure 1. Examples of intracranial hemorrhage on head computed tomography. (A) Right frontal traumatic subarachnoid hemorrhage. (B) Right epidural hematoma. (C, D) Two images of right subdural hematomas demonstrating varying thickness of clot and degree of midline shift.

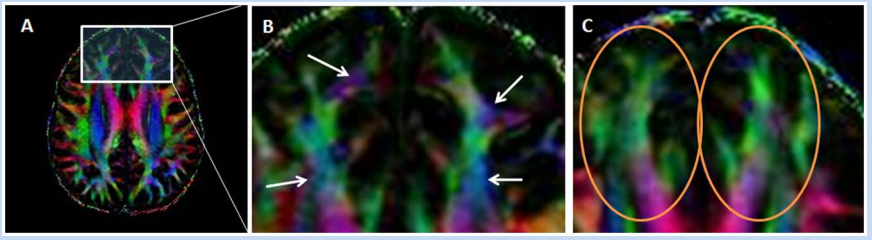

Figure 2. (A, B) Diffusion tensor imaging of a patient with concussion. B is blowup of frontal region seen in A. Note heterogeneous color suggesting discontinuity of tracts (arrows). Fibers should normally all run anterior-posterior (green fibers) as seen in the area circled in a control patient (C).

SECOND-IMPACT SYNDROME, REPEAT INJURY, AND DEATH AFTER ATHLETIC HEAD TRAUMA

A recent study of sudden death in young athletes aged less than 21 years found that trauma-related injuries accounted for 14% of deaths. The estimated rate of death from trauma was 0.11 per 100 000 participants, with an average of 9 deaths per year.[20] Of these, 89% of trauma-related deaths were from trauma to the head and/or neck. In football players and boxers, this rate was even higher, with head and/or neck injuries accounting for 93% and 100% of deaths from trauma, respectively. During the 30-year period from 1980 to 2009, 138 football players died of subdural hematoma. Of these, 12% had a reported history of concussion within the 4 weeks of death.[20] However, it is not clear whether this history of concussion had any relationship to their subsequent injuries and death. College football players with a history of 3 or more concussions are 3 times more likely to suffer a concussion than those with no history of concussion. In same-season repeat concussions in college football, 91.7% occur within 10 days of the first injury, suggesting a possible increased susceptibility to injury following a concussion.[6] Additionally, patients with repeat concussion also appeared to have a longer duration of symptoms.[6]

Case reports exist of patients who suffer an initial minor TBI and, after return to play and a second minor blow, quickly deteriorate and die. In many of these cases, an acute space-occupying mass, such as a hematoma, was not detected, and death was thought to result from severe and rapid brain edema.[21] This series of events and the potential for rapid deterioration after a second injury, known as "second-impact syndrome," remains controversial.[21] It is known that in severe TBI loss of autoregulation (ability to vasoconstrict or dilate intracranial blood vessels to maintain intracerebral blood flow in a safe range despite systemic variation) in association with rapid catecholamine release can lead to severe brain edema.[12] Proponents of second-impact syndrome believe that after an initial minor TBI, children and young adults may be at increased risk of developing disturbances in autoregulation and catecholamine release, with subsequent brain edema and potentially death, even with a minor second injury. Cantu and Gean[3] recently published a series of cases of patients with presumed second-impact syndrome. In these cases, patients underwent an initial head injury with persistent symptoms and then suffered acute deterioration after a presumed second impact that occurred at an interval ranging from later in the same game to up to 4 weeks later. All patients in their series were found to have small subdural hematomas < 1 cm, with severe edema out of proportion to what would be expected for the size of the subdural hematoma; patients also did poorly with permanent neurologic deficit or death.[3] The authors concluded that subdural hematoma and edema are likely secondary to shear forces and dysautoregulation/second-impact syndrome.[3]

Mori et al similarly described cases in the literature of patients with acute cerebral edema associated with thin subdural hematomas.[15] However, in both series, it is not entirely clear that the trauma seen on imaging was not (1) the direct result of the first hit, in the case of patients who deteriorated in the same game or within the first few days, or (2) the result of the second hit, alone in the case of patients with injury several weeks later. There is no clear scientific evidence that an initial concussion predisposes these individuals to deterioration after less significant repeat head trauma.[12] Indeed, many of the cases of suggested second-impact syndrome may be the result of a single traumatic injury severe enough to cause intracranial hematoma or cerebral edema.[12] In a recent study of supposed deaths from second-impact syndrome, all 17 athletes were actually found to have died from subdural hematoma.[20] Consequently, this syndrome remains controversial. Although it is clear that seemingly innocuous head injury can have catastrophic results and small subdural hematoma and significant cerebral edema are often found in these cases, the existence of a second-impact syndrome or an increased susceptibility to catastrophic cerebral edema when still suffering from concussive symptoms remains controversial. Regardless, consideration of concussion in return-to-play decisions makes sense, as athletes still suffering from concussion may be at increased risk of injury due to delayed response times and as, after an initial TBI, patients are at increased risk of future concussion.[6,12,21]

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