Classification of Sport-Related Head Trauma: A Spectrum of Mild to Severe Injury

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Objective: To identify the types of injuries the human brain incurs as a result of traumatic forces applied to the cranium. In athletic events and endeavors, the full spectrum of intracranial hemorrhages in various compartments, raised intracranial pressure, and diffuse nonhemorrhagic damage may be seen. In this review, we describe these serious injuries and the more common mild traumatic brain injury in their clinical presentations and relate concussion classification to the overall picture of traumatic brain injury.

Methods: Our cumulative experience with athletic injuries, both at the catastrophic and mild traumatic brain injury levels, has led us to a management paradigm that serves to guide us in the classification and treatment of these athletes.

Discussion: The occurrence of intracranial injuries in sports has now been well documented. Intracranial hematomas (epidural, subdural, and parenchymal) and cerebral contusions can result from head injury. Many patients sustain a diffuse brain injury, resulting in elevated intracranial pressures, without a blood clot or mass lesion. The classification of concussion and the use of concussion guidelines are not uniform. However, the major emphases are agreed upon: the close and careful scrutiny of the athlete, an expeditious but reliable neurologic examination, and proper on-field management. Return-to-play decisions are based on many factors that affect normal functioning, both on and off the playing field.

Conclusions: Sufficient knowledge now exists to allow us to carefully evaluate the injured athlete, to place him or her in the management scheme to minimize the potential for permanent cerebral dysfunction, and to know when the athlete can safely return to contact sport participation.

Key Words: diffuse brain injury, hemotoma

As the number of participants in recreational and organized sporting events continues to increase, we are faced with an expanding interest in understanding the effects of mild traumatic brain injury (MTBI). Concussion, as MTBI is commonly termed, is a concern in every sport that has head impact or collision as a possible consequence. Paramount to an appreciation for the seriousness and potential consequences of concussion is the concept that it exists as a pathophysiologic entity along a spectrum of injury, ranging from mild concussion to severe, diffuse injuries. Associated severe cognitive, memory, and motor deficits persist among survivors of severe, diffuse brain injury, and the mortality rate is greater than 50%. Acceleration-deceleration injury, also considered translational (linear) impact, usually results when the subject’s body and head are traveling at a particular speed and strike a solid object. Similarly, a head at rest may be struck by a moving object. The resultant injury causes linear, tensile, and compressive strains that disrupt the cerebral anatomy and cytoarchitecture. Rotational (angular) movements also take place, secondary to the fixation of the brain at the foramen magnum and craniospinal junction and the relative tethering of the midbrain as it passes through the tentorial hiatus. Energy directed to the head may cause transmission of force in a rotatory direction, often producing diffuse brain injury with shearing of the white-matter fiber tracts. As kinetic energy is applied to the human cranium, both acceleration-deceleration and rotational mechanisms occur. Acceleration-deceleration injury, also considered translational (linear) impact, usually results when the subject’s body and head are traveling at a particular speed and strike a solid object. Similarly, a head at rest may be struck by a moving object. The resultant injury causes linear, tensile, and compressive strains that disrupt the cerebral anatomy and cytoarchitecture. Rotational (angular) movements also take place, secondary to the fixation of the brain at the foramen magnum and craniospinal junction and the relative tethering of the midbrain as it passes through the tentorial hiatus. Energy directed to the head may cause transmission of force in a rotatory direction, often producing diffuse brain injury with shearing of the white-matter fiber tracts. As kinetic energy is applied to the human cranium, both acceleration-deceleration and rotational mechanisms occur. Acceleration-deceleration injury, also considered translational (linear) impact, usually results when the subject’s body and head are traveling at a particular speed and strike a solid object. Similarly, a head at rest may be struck by a moving object. The resultant injury causes linear, tensile, and compressive strains that disrupt the cerebral anatomy and cytoarchitecture. Rotational (angular) movements also take place, secondary to the fixation of the brain at the foramen magnum and craniospinal junction and the relative tethering of the midbrain as it passes through the tentorial hiatus. Energy directed to the head may cause transmission of force in a rotatory direction, often producing diffuse brain injury with shearing of the white-matter fiber tracts. As kinetic energy is applied to the human cranium, both acceleration-deceleration and rotational mechanisms occur. Acceleration-deceleration injury, also considered translational (linear) impact, usually results when the subject’s body and head are traveling at a particular speed and strike a solid object. Similarly, a head at rest may be struck by a moving object. The resultant injury causes linear, tensile, and compressive strains that disrupt the cerebral anatomy and cytoarchitecture. Rotational (angular) movements also take place, secondary to the fixation of the brain at the foramen magnum and craniospinal junction and the relative tethering of the midbrain as it passes through the tentorial hiatus. Energy directed to the head may cause transmission of force in a rotatory direction, often producing diffuse brain injury with shearing of the white-matter fiber tracts.
forces that cause loss of consciousness (LOC), providing the etiology for many athletic cerebral concussions.

In addition, forces applied to the head in contact sports have also been described as impact or impulsive loading. Resulting from a rapidly applied energy input, impact loading is a direct blow to the cranium occurring over less than 200 milliseconds. This can cause skull deformation and energy shock-wave propagation through the skull and brain, resulting in underlying cerebral injury. In contrast, when the head is placed in motion and suddenly accelerated or decelerated as a result of either an impact to another part of the body or as a secondary response to a direct impact, impulsive loading occurs. This mechanism causes compressive, shear, and tensile stresses to the brain, leading to more diffuse or remote injuries. It is more often seen with rotational energy inputs and is less effectively prevented by protective headgear, which is better able to dissipate impact-loading energy input.

Presence of the normal state of consciousness signifies that the person is awake and alert with the ability to interact with the environment. A normal level of consciousness depends on a complex interaction of cortical, subcortical, and brain stem nuclei. Alteration of the state of consciousness occurs when the integrity of this neurophysiologic functional unit has been interrupted. The reticular activating system extending through the brain stem must interact with the hypothalamus and cerebral hemispheres in a normal feedback-loop mechanism for consciousness to be maintained. Any alteration of this circuitry and feedback, therefore, produces a change in the state of consciousness. The major focus of categorizing concussions in sports in the past has centered on the occurrence of LOC. We now appreciate that other and ongoing expressions of cerebral dysfunction, such as memory and cognitive dysfunction, are important and likely predictive. This is especially true when considering only the very brief period of LOC, which may not be as indicative of a serious athletic injury.

In addition to the biomechanics, recent experimenters have elucidated the biomechanical abnormalities that represent concussion. Activation of the glycolytic process as a result of cellular requirements to maintain ionic gradients is believed to be present in concussion. Large increases occur in extracellular potassium concentrations through voltage-gated potassium channels. Neurotransmitters, in particular glutamate, appear to play a significant role in opening ionic channels after even MTBI. A quantitative rise in glucose utilization, in an effort to correct ionic perturbations in transmembrane potentials, is seen in newer concussion models. Metabolic dysfunction after concussive injury produces a period of neuronal vulnerability in which there is both higher demand for glucose to correct ionic movements and a paradoxical reduction in cerebral blood flow, the latter influenced by calcium movements. The rise in glycolytic energy requirements has been shown both experimentally and clinically to be present within the first several days after concussive brain injury.

Experimental concussion models have demonstrated transient cerebral ischemia, edema, widespread neuronal depolarization from release of acetylcholine, and the shearing of neurons and nerve fibers (particularly in the brain stem) as potential explanations for alterations of mental status after closed head injury. While no gross neuropathologic changes are consistently demonstrated in experimental concussion, disruption of multiple neurons and their connections, along with scattered capillary damage in the brain stem reticular formation, has been reported.
Epidural Hematoma

Epidural hematoma is an accumulation of blood between the dura and skull. The dura becomes detached and dissects to the point of dural attachment to the overlying cranium. Hemorrhage occurs beneath the skull and outside the dura, resulting in the classic computed tomography (CT) appearance of a biconvex or lenticular shape of the hematoma (Figure 3). Epidural hematoma is caused by head impact, usually of the acceleration-deceleration type, and can result in inward deformity, leading to dural detachment from the inner table of the skull. Most patients with an epidural hematoma have a skull fracture, which leads to laceration of the middle meningeal artery or vein. In addition, bleeding can occur from the actual bone fragments or the diploic space, leading to collection of blood in the epidural location.

Characteristic of epidural hematoma is an isolated injury to the skull, dura, and dural vessels that leads to hematoma formation. In most of these acceleration-deceleration injuries, the skull has sustained the major impact forces and absorbed the resultant kinetic energy. The heavy force delivered to the cranium and transmitted to the brain often disrupts the state of consciousness. In contrast with other injuries, such as subdural hematoma, in which the brain often sustains a primary and major injury, epidural hematoma is often not associated with primary brain injury.

Another important distinguishing feature of this clinical entity is a lucid interval. The lucid interval occurs when a substantial blow has been transmitted to the cranium and causes the person to lose consciousness. The patient may subsequently appear asymptomatic and have a normal neurologic examination. The problem arises when an injury to the skull or dural vessels or both leads to a slow accumulation of blood in the epidural space. This hematoma outside the brain may remain relatively asymptomatic until it reaches a critically large size and compresses the underlying brain. The compression can be transmitted to the brain stem and rapidly progress to neurologic dysfunction, brain herniation, and possibly death. Any patient or athlete who has sustained a significant head impact should be observed in the awake state and not allowed to retire for sleep until the longer lasting effects of the head impact are known. Any patient with a significant LOC (minutes) or neurologic abnormality should have a more thorough medical evaluation, including CT scanning. The clinical manifestations of epidural hematoma depend on the type and amount of energy transferred, the time course of the hematoma formation, and the presence of simultaneous brain injuries. Often, the size of the hematoma determines the clinical effects. In addition to a lucid interval, patients with an epidural hematoma may present with no LOC, persistent unconsciousness, or any variation of these features.

Subdural Hematoma

This form of intracranial hemorrhage has been divided into acute subdural hematoma, which presents within 48 to 72 hours after injury, and chronic subdural hematoma, which occurs in a later time frame with more variable clinical manifestations. An acute subdural hematoma is the most common major head injury and is associated with severe neurologic disability and death in many patients. Acute subdural hematoma results from bleeding within the subdural space as a result of stretching and tearing of the subdural veins. These veins drain from the cerebral surface and connect to the dura or dural sinuses. In addition, the bone irregularities of the middle cranial fossa, sphenoid bone, and frontal fossa form a rough surface over which inferior cortical surface contusions can form, resulting in hemorrhage in the subdural space.

A subdural hematoma may occur as an isolated collection of blood within the subdural space or as a more complicated hematoma associated with brain parenchymal injury. Many patients with complicated acute subdural hematomas sustain diffuse irreversible brain damage and do not improve after evacuation of the hematoma, the latter representing an epiphenomenon in the injury process. The outcome of subdural hematoma is thus often influenced by the extent of the concomitant parenchymal brain injury more than the subdural hematoma collection per se.

The clinical presentation of any patient, including an athlete, with acute subdural hematoma can vary and includes those who are awake and alert with no focal neurologic deficits, but typically patients with any sizable acute subdural hematoma have a significant neurologic deficit (Figure 4). This may consist of alteration of consciousness, often to a state of coma or major focal neurologic deficit. Skull fracture is much less commonly associated with subdural hematoma than with epidural hematoma. Football players with 2 mild concussions without LOC, separated by 7 and 10 days, sustained acute subdural hematomas. A chronic subdural hematoma is defined as a hematoma present 3 weeks or more after a traumatic injury. The pathogenesis of chronic subdural hematoma involves an injury that results in bleeding into the subdural space. The initial hemorrhage may be a small amount that fails to generate significant brain compression. However, bleeding or oozing of blood into the subdural space may continue. After 1 week, a chron-
ic subdural hematoma involves infiltration of fibroblasts to organize into an outer membrane. Subsequently, an inner membrane may form, and this encapsulated hematoma may become a dynamic osmotic membrane that interacts with the production and absorption of cerebrospinal fluid. Effusion of protein may occur, setting up an active process within the membrane.25

The diagnosis of chronic subdural hematoma is often difficult because of the protean clinical manifestations. Patients may have clinical symptoms suggestive of increased intracranial pressure, mental disturbance such as personality change or even dementia, symptoms with focal transient neurologic deficits similar to transient ischemic attacks, a meningeal syndrome with nuchal rigidity and photophobia, a clinical course with a slow progression of neurologic signs reminiscent of cerebral neoplasm, or a progressive and severe headache syndrome.26 Although not common in athletes, chronic subdural hematoma must always be the differential diagnosis, especially in those presenting with a remote history of head impact. The diagnosis is confirmed by CT scanning demonstrating the extra-axial low-density fluid collection in the subdural space.

Intracerebral Hemorrhage

A cerebral contusion is a heterogeneous zone of brain damage that consists of hemorrhage, cerebral infarction, necrosis, and edema. Cerebral contusion is a frequent sequela of head injury and in some studies represents the most common traumatic lesion of the brain visualized on radiographic evaluation. Contusions occur most often as a result of acceleration-deceleration mechanisms from the inward deformation of the skull at the impact site. This results in transient compression of the brain against the skull and the focal area of parenchymal injury. This energy is conducted to the underlying brain, resulting in cerebral contusion, the degree of which depends on the energy transmitted, the area of contact, the involved area of the cranium, and other factors (Figure 5).27

Contusions can vary from small, localized areas of injury to large, extensive areas of involvement. A cerebral contusion injury can evolve over hours and days after the injury. Multiple small areas of contusions may coalesce into a large area resembling a lesion, more accurately termed intraparenchymal hemorrhage. Injuries remote from the site of cranial impact may also occur. The direct, or coup, lesion results from injury at the impact site, and the remote, or contrecoup, lesion occurs as the opposite side of the brain rebounds against the skull or because of vacuum phenomena existing within the parenchyma at that location. The contrecoup lesion results in a hemorrhagic lesion in the cerebral tissue directly opposite the impact site, typically at the inferior surfaces of the frontal and temporal lobes. Contusions are often multiple and are frequently associated with other extra-axial and intra-axial hemorrhagic lesions.

The clinical course of patients with cerebral contusion varies greatly, depending on the location, number, and extent of the hemorrhagic contusion lesions. The patient may present with essentially normal function or may experience any type of neurologic deterioration, including coma. Frequently, behavioral or mental status changes exist due to involvement of the fron-
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Figure 6. CT scan showing right temporal lobe contusions. These contusions tend
to enlarge and lead to brain swelling.

tal or temporal lobes. The diagnosis of cerebral contusion is
firmly established by CT scanning, which is also useful for
following patients as the lesions evolve throughout their clinical
course (Figure 6).

Intracerebral hematoma is a parenchymatous hemorrhage
that is often similar in pathophysiology and radiographic ap-
parence to a cerebral contusion. An intracerebral hematoma
represents a localized collection of blood within the brain. The
distinction between a hemorrhagic contusion and intraparen-
chymal hematoma depends upon the latter being recognized
as a confluent area of homogeneous bleeding within the brain.
Intracerebral hematomas usually present with a focal neuro-
logic deficit but may progress to further neurologic deteriora-
ration, including coma and death resulting from brain hernia-
tion syndromes. This diagnosis is readily made by CT
scanning, which shows a hyperdense, localized collection of
blood. Intracerebral hematomas have been, along with sub-
dural hematomas, the most common cause of sport-related le-
thal brain injuries.

Another entity, delayed traumatic intracerebral hematoma,
is a clot that forms hours to days after the initial trauma. Al-
though most frequently seen in the older population, it must
always be borne in mind when evaluating and attending to any
patient who has sustained a significant head impact. The ath-
lete is also at risk because these hematomas are seen more
commonly when there has been rotational head trauma. De-
layed traumatic hematomas are believed to be due to later
bleeding into an already contused region of the brain, to vas-
cular injury, or to the development of a coagulopathy.

Finally, there are severe brain injuries in which the patients
do not sustain any form of hematoma or mass effect but in-
stead have a global or diffuse brain insult. This often results
in widespread cerebral edema and elevated intracranial pres-
sure (ICP), similar to the pathophysiologic response seen in
the second-impact syndrome. In the absence of an operable,
space-occupying lesion, the patient is often treated for in-
creased ICP with online ICP monitoring. This is performed by
placing a catheter in the brain parenchyma or ventricles, pro-
viding a constant measure of ICP. The cerebral perfusion pres-
sure is defined as the mean systemic blood pressure minus the
ICP and should be kept at 50 to 70 mm Hg or higher.

Cerebral Concussion

Concussion, or MTBI, is the most common form of head
injury seen in athletes. The Centers for Disease Control recently
proclaimed that approximately 300,000 cases of MTBI
occur annually in the United States from athletic activities.
Powell and Barber-Foss estimated 62,000 cases of concussion
in American high school sports annually.

The classical cerebral concussion is defined as a posttrau-
static state that results in LOC, with the patient regaining full
return of consciousness within 24 hours. Clinically, concussion
has often been referred to as physiologic without anatomical
disruption in the brain. Newer research has shown both anat-
omic and biochemical markers of definitive injury secondary
to MTBI. Concussion has also been defined as an immediate
and transient impairment of neural function, such as alteration
of consciousness, disturbance of vision or equilibrium, and
other similar symptoms. We now appreciate that concussion
symptoms are related to either cerebral cortical dysfunction,
such as confusion, disorientation, memory, or information-pro-
cessing abnormalities, and others, or to brain stem abnormal-
ties. Examples of the latter include the unconscious state, vi-
sual or auditory symptoms, nausea and vomiting, ataxia, and
incoordination. Some of these patient complaints may also re-
late to injury to and dysfunction of the inner ear or vestibular
mechanism. Sleep disturbances are common and often con-
tribute greatly to the symptom complex.

We have gained a greater appreciation for the importance
of having accurate methods for classifying MTBI. This rec-
ognition has led to the development of several different clas-
sification schemes, unfortunately leading to some uncertainty
at times over nomenclature. One must keep in mind that all
scales thus far published were formulated based on experien-
tial theory and not on scientifically proven research. No pro-
spective, randomized clinical trial has established any particular
classification system as superior or proven. Rather, based on years of experience and observation, these
guidelines have been employed as our best and most accurate
means of currently categorizing athletic MTBI. The National
Athletic Trainers’ Association has not adapted or endorsed any
one grading scale or set of return-to-play guidelines, nor have
most organizations. The best grading scale and return-to-play
guidelines will be based on scientific evidence, but most im-
portantly, a systematic evaluation and management scheme
must be followed.

During the last 2 decades, no fewer than 8 classification
systems have been proposed for management of sport-related
concussions. While disagreement and differences existed regarding terminology and symptom priority, these efforts have led to the recognition of the importance of standardization in the care of the concussed athlete. In recent years, 3 concussion classification schemes have been accepted in widespread application. In 1986, Cantu proposed a concussion grading scale in which the persistence of memory disturbance, termed amnesia, is given greater emphasis. The Colorado guidelines and the American Academy of Neurology (AAN) practice parameters similarly place any athlete with any period of LOC, no matter how brief, within the most severe grade of concussion (Table).

CONCUSSION CLASSIFICATION

A mild concussion (grade 1) is most commonly seen in athletes. The Colorado guidelines define a grade 1 concussion as no LOC, with confusion being the hallmark sign. The Cantu definition of a mild concussion is one without LOC, with confusion alone or a brief (less than 30 minutes) period of amnesia. The AAN practice parameters classify a grade 1 concussion as having no LOC and mental status abnormalities that resolve in less than 15 minutes. This type of concussion is not infrequent in football games, occurring in at least 1 player in nearly every game. If thoroughly searched for, a grade 1 concussion is often found. It may be stated by nonmedical personnel that the player was “dinged.” The athlete, who is awake and alert, may be able to function unnoticed during the course of the athletic contest. If significant disorientation, confusion, memory disturbance, dizziness, headache, or any neurologic abnormality persists after the 15-minute observation period, the athlete has more than a mild concussion. We should remember that concussion may be present and significant without the person’s sustaining a LOC. Ommaya and Gennarelli showed in their animal model that 3 of 6 grades of concussion did not involve LOC. They postulated that, unless shearing forces reached the reticular activating system within the midbrain and brain stem, cortical and subcortical structures could be affected to produce amnesia and confusion but not LOC.

The Colorado guidelines classify a moderate or grade 2 concussion as associated with the development of amnesia either initially or during the period of observation; there is no LOC. The athlete is removed from competition and not allowed to return. Cantu defined the moderate concussion as less than 5 minutes of unconsciousness or posttraumatic amnesia for longer than 30 minutes but less than 24 hours’ duration, while the AAN system specified no LOC but only mental status changes lasting longer than 15 minutes.

A severe or grade 3 concussion is defined as a player having LOC by the Colorado and AAN guidelines. Cantu termed a severe or grade 3 concussion as one having greater than a 5-minute period of unconsciousness or 24 hours of posttraumatic amnesia. This patient may require emergent transport to the nearest hospital facility with CT scanning and, if indicated, consideration should be given for neurosurgical consultation. The possibility of concomitant cervical spine injury must always be considered in an unconscious patient and transport performed with cervical immobilization and maintenance of an adequate airway.

EVALUATION AND MANAGEMENT

The goals in evaluating the potentially head-injured athlete are threefold: (1) the fact that a head injury has potentially occurred must be recognized; (2) the athletes requiring transport to a medical facility for further workup and treatment must be accurately identified; and (3) a decision must be made regarding when the athlete may return to competition. Obviously, detection of a potentially life-threatening or neurologically devastating injury is of paramount importance. In light of the possibility of second-impact syndrome, those athletes with mild head injuries must be cautiously observed and returned to play only when they are absolutely symptom free.

Any athlete who receives a blow to the head or any significant acceleration-deceleration-type force to the head should be presumed to have a possible head injury and should be thoroughly evaluated. The athlete should be evaluated for level of consciousness, steadiness of gait, orientation, and posttraumatic amnesia. Those players with grade 1 concussions generally should be observed for 20 to 30 minutes. If the sen-
<table>
<thead>
<tr>
<th>Classification</th>
<th>Grade</th>
<th>Signs/Symptoms*</th>
<th>First Concussion</th>
<th>Second Concussion</th>
<th>Third Concussion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colorado Medical Society Guidelines</td>
<td>1</td>
<td>Confusion without amnesia; no loss of consciousness</td>
<td>May return to play if without symptoms for at least 20 minutes</td>
<td>Terminate contest or practice; may return to play if without symptoms for at least 1 week</td>
<td>Terminate season or may return to play in 3 months if without symptoms</td>
</tr>
<tr>
<td>(mild)</td>
<td>2</td>
<td>Confusion with amnesia; no loss of consciousness</td>
<td>Terminate contest or practice; may return to play if without symptoms for at least 1 week</td>
<td>Consider terminating season; may return to play in 1 month if without symptoms</td>
<td>Terminate season; may return to play next season if without symptoms</td>
</tr>
<tr>
<td>3 (severe)</td>
<td></td>
<td>Loss of consciousness</td>
<td>Terminate contest or practice and transport to hospital; may return to play in 1 month, after 2 consecutive weeks without symptoms</td>
<td>Terminate season; may return to play next season if without symptoms</td>
<td>Terminate season; strongly discourage return to contact or collision sports</td>
</tr>
<tr>
<td>Cantu Grading System</td>
<td>1</td>
<td>No loss of consciousness; posttraumatic amnesia less than 30 minutes in duration</td>
<td>May return to play if asymptomatic for 1 week; terminate season if CT or MRI abnormality</td>
<td>Return to play in 2 weeks if asymptomatic at the time for 1 week</td>
<td>Terminate season; may return to play next season if asymptomatic</td>
</tr>
<tr>
<td>(mild)</td>
<td>2</td>
<td>Loss of consciousness less than 5 minutes in duration or posttraumatic amnesia longer than 30 minutes but less than 24 hours in duration</td>
<td>Return to play after asymptomatic for 2 weeks; terminate season if CT or MRI abnormality</td>
<td>Minimum of 1 month; may return to play then if asymptomatic for 1 week; consider terminating season</td>
<td>Terminate season; may return to play next season if asymptomatic</td>
</tr>
<tr>
<td>3 (severe)</td>
<td></td>
<td>Loss of consciousness for more than 5 minutes or posttraumatic amnesia for more than 24 hours</td>
<td>Minimum of 1 month; may then return to play if asymptomatic for 1 week§</td>
<td>Terminate season; may return to play next season if asymptomatic</td>
<td>Consider no further contact sports</td>
</tr>
<tr>
<td>American Academy of Neurology</td>
<td>1</td>
<td>Transient confusion; no loss of consciousness; symptoms or abnormalities resolve in less than 15 minutes</td>
<td>Remove from contest; may return to play if abnormalities or symptoms clear within 15 minutes</td>
<td>Terminate contest; may return to play after 1 week without symptoms at rest and with exercise</td>
<td>Terminate contest; may return to play after at least 2 asymptomatic weeks at rest and with exertion; terminate season if any CT or MRI abnormality</td>
</tr>
<tr>
<td>Guidelines</td>
<td>2</td>
<td>Transient confusion; no loss of consciousness; symptoms or abnormalities last more than 15 minutes</td>
<td>Terminate contest; may return to play after 1 full asymptomatic week at rest and with exertion§</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mild)</td>
<td>3</td>
<td>Any loss of consciousness, either brief (seconds) or prolonged (minutes)</td>
<td>Terminate contest; transport to hospital if unconscious or neurologic abnormality; if concussion brief (seconds) may return in 1 week if no symptoms at rest and with exertion; if concussion prolonged (minutes) return in 2 weeks, no symptoms</td>
<td>Terminate contest; may return to play after minimum of 1 month asymptomatic; terminate season if any CT or MRI abnormalities</td>
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