Emergent Treatment of Athletes With Brain Injury

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The focus of this article is the early recognition and emergent treatment of severe or catastrophic traumatic brain injury. The pathophysiology and management of mild traumatic brain injury are reviewed extensively in other sections. Classification of head injuries can be based on anatomic location (epidural, subdural, intraparenchymal), mechanism of injury (coup, contrecoup, linear, rotational), distribution (focal or diffuse), and clinical presentation. Severe head injuries include epidural hematoma, subdural hematoma, intracerebral contusion/hemorrhage, traumatic subarachnoid hemorrhage, diffuse axonal injury, and malignant brain edema syndrome, either alone or in combinations. Protective equipment, regulations, and athletic training have improved dramatically as a result of the recognition of potentially devastating neurological injuries from competitive play. Physicians and other healthcare professionals have become integral members of organized sport and must advocate for the players' best interest. Once a neurological injury has been identified on field, it is imperative that prompt management and prevention of secondary injury occur. The goal of this article is to help provide a clear plan of action that is well thought out and rehearsed and that will lead to improved outcomes for the players, particularly those with severe or catastrophic brain injury.

KEY WORDS: Athletic injuries, Concussion, Critical care management, Emergency sideline management, Sports-related brain injury, Sports medicine, Traumatic brain injury

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The focus of this article is the early recognition and emergent treatment of severe or catastrophic traumatic brain injury (TBI). The pathophysiology and management of mild TBI are reviewed extensively in other sections. Classification of head injuries can be based on anatomic location (epidural, subdural, intraparenchymal), mechanism of injury (coup, contrecoup, linear, rotational), distribution (focal or diffuse), and clinical presentation.[1,2] Severe head injuries include epidural hematoma, subdural hematoma, intracerebral contusion/hemorrhage, traumatic subarachnoid hemorrhage, diffuse axonal injury, and malignant brain edema syndrome, either alone or in combinations.[3,4]

ABBREVIATIONS: BTF, Brain Trauma Foundation; **CBF,** cerebral blood flow; **ECP,** emergency care plan; **GCS,** Glasgow Coma Scale; **ICP,** intracranial pressure; **TBI,** traumatic brain injury

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According to the National Center for Catastrophic Sports Injury Research, a catastrophic injury can also be defined as either direct (those experienced as a result of participating in the skills of the sport) or indirect (resulting from systemic failure secondary to exertion while participating in a sport). Each can be further subclassified as fatal (the injury causes the death of the athlete), nonfatal (the injury causes a permanent neurological functional disability), or serious (although severely injured, the athlete has no permanent functional disability). An example of a serious injury is an intraparenchymal contusion causing no permanent neurological disability.[2,5]

HISTORY

During the 1905 season alone, 18 college and amateur football players reportedly died as a result of injuries suffered during competition.[6] Amid a growing movement to abolish the sport, President Theodore Roosevelt, himself an avid fan, held a meeting of the influential leaders of collegiate football (the highest level of competition at the time), encouraging them to redesign and regulate their sport to preserve the game.[7] As

a result of this gathering, 2 important processes were set in motion. First, the Intercollegiate Athletic Association and Intercollegiate Rules Committee were created, which, 4 years later, merged into the National Collegiate Athletic Association, the current governing body of collegiate athletics. More important, the game was redesigned by the newly generated rules. None were more notable than the forward pass, aimed at opening the field of play and decreasing the traditional rugby-style gang tackling that often led to head injuries.[6-8]

Although the first football helmet was worn in 1893 during the annual Army-Navy game, its use did not become mandatory until 1939 (by the National Collegiate Athletic Association) and 1940 (by the National Football League).[9] Despite the evolution from leather to plastic helmets and the addition of the facemask, the number of head injuries and spine injuries continued to mount throughout the 1950s and 1960s. The increased injury frequency and the promising research of Dr Richard C. Schneider, a neurosurgeon at the University of Michigan, prompted the initiation of a prospective injury data registry in 1967. The National Operating Committee on Standards for Athletic Equipment was also formed at that time. In 1975, Dr Joseph Torg of Temple University reported that although the helmet decreased the severity of head injuries, it encouraged players to strike opponents head first, which increased spinal axial load during impact, leading to increased numbers of paralyzing cervical spine injuries. Subsequently, both the National Football League and National Collegiate Athletic Association implemented rules aimed at preventing players from deliberately initiating contact with their helmet.[10] Additionally, continued research into head and spine injury biomechanics led to more sophisticated, individualized helmets aimed at optimal protection.[11] Consequently, a significant decline in the number of catastrophic sports-related head and neck injuries was noted in the latter part of the 20th century. A recent review estimated the number of direct sports-related catastrophic injuries to be approximately 0.60 per 100,000 participants per year among high school athletes.[12]

Although the focus of this article is a discussion of the early recognition and management of sports-related severe catastrophic head injuries, it should be noted that clinicians must have a thorough understanding of the more common mild TBI. Not only does this allow a physician to quickly recognize the more severe injury but also may thwart delayed catastrophic neurological injury. Although a concussion may not present acutely as a catastrophic sports injury, its long-term sequelae are becoming an increasingly evident concern. Dr Harrison Martland first described the concept of repetitive traumatic head blows leading to permanent neurological dysfunction in 1928. After describing specific neuropathological changes in the injured brain, Martland identified cognitive impairment, loss of memory, slurred speech, balance disturbances, and abnormal movements that he originally called the punch-drunk syndrome, which has now evolved into the spectrum known as chronic traumatic encephalopathy.[13,14] As contact sports entered the 21st century, the focus shifted from recognizing a single injury to preventing, treating, and determining the long-term effects of mild TBI, including chronic traumatic encephalopathy.[13] Amid the growing public awareness and documented clinical evidence, all types of athletics are now implementing rule changes that redefine the way the sport is played to protect its participants.[15]

CONTINUUM OF INJURY

Sports-related TBI exists on a clinical spectrum with various degrees and combinations of biomechanical forces that produce distinct pathological and clinical patterns of injury. Head injury occurs after an athlete engages in a collision with either an object or another player. The impact primarily exposes the brain to either a translational (linear) force or a rotational (angular) force that produce a variety of clinical sequelae.

TBI can be divided into local or diffuse trauma, which is dependent on the extent and biomechanics of the injury (Table 1). Focal injuries are often the result of a direct blow causing mechanical disruption of vessels or other brain substance. A linear and direct impact force occurs when an object such as a well-hit baseball strikes a fielder, potentially leading to an epidural hematoma. Similarly, acceleration-deceleration injury occurs when an athlete has his or her momentum stopped suddenly by a solid object, causing the brain to strike the inside of the skull.

Diffuse injuries are commonly attributed to rotational (angular) forces applied to white matter tracts, resulting in shearing of multiple axons within the central nervous system. Angular forces exert more diffuse effects on areas of the brain in which tissue density is greatest such as at the gray matter–white matter junction.[4,16] Depending on severity, a variety of clinical presentations may occur. For example, an athlete presenting with a mild injury may exhibit minimal signs of central nervous system disruption, and standard imaging will likely be normal, leading to a diagnosis of a concussion or mild TBI. A more severe, diffuse injury may cause punctate hemorrhages, leading to a diagnosis of diffuse axonal injury. Clinically, this can cause significant disruption of consciousness and function, which might not return to the original functional baseline.[16] Infrequently, repetitive injury with short recovery periods between episodes can produce acute and diffuse cerebral edema, associated with malignant increases in intracranial pressure (ICP) referred to as second impact syndrome.[17] Whereas 1 biomechanical force is thought to predominate, local and diffuse injuries generally coexist.

TBI can also be divided into the initial biomechanical force of impact and its associated pattern of parenchymal injury, or primary injury, and the resultant secondary injury caused by the initiation of a cytotoxic biochemical cascade. Prevention is the only protection against primary injury. Much of the research in collision sports is aimed at analyzing the forces of impact and engineering protective gear intended to minimize its effects. Secondary injury is a progressive insult of inflammatory

TABLE 1. Classification of Traumatic Brain Injury[a]

	Injury Type	Pathophysiology/Biomechanics	Clinical Sequelae
Focal	Epidural hematoma	Direct blow (ie baseball)	May exhibit lucid interval
		Often nonhelmeted players	
		Often associated with skull fracture and middle meningeal artery injury	Arterial bleeding source, so can rapidly deteriorate
		Usually no underlying brain injury	Good neurological prognosis
	Subdural hematoma	Tearing of bridging veins	No. 1 cause of sports-related fatalities
		As opposed to elderly population, less subdural space, so expansion	Continued progression of symptoms; focal findings on examination common
		Leads to rapid increase in ICP	Usually underlying brain injury
	Intracerebral hemorrhage (contusion, bruise)	Primary neuronal or parenchymal vessel injury caused by direct blow or acceleration/deceleration injury	Potential to progress or blossom within first 24-48 h
		Common at frontal/temporal poles	
	Traumatic subarachnoid hemorrhage	Injury of surface vessels	Similar to brain contusion
		Cortical location, underlying parenchymal injury	Common in severe TBI
			Rarely, vasospasm as consequence
Diffuse	Concussion	Mild TBI	Loss of consciousness not a requirement
			Most common clinical complaints: headache, dizziness, memory loss
	Malignant cerebral edema (brain swelling)	Most common in younger athletes	Classically associated with second impact syndrome
		Diffuse cerebral swelling	Rapid progression to fatal ICP secondary to hyperemia and vascular engorgement
	Diffuse axonal injury	First-degree injury; rotational forces causing axonal shearing	Often lower GCS score and permanent neurological deficits
		Force greatest at areas of highest tissue density (gray-white junction)	

[a]GCS, Glasgow Coma Scale; ICP, intracranial pressure; TBI, traumatic brain injury.

modulators set in motion by the primary injury, with the potential to cause further permanent neurological dysfunction or even death. TBI treatment focuses on preventing and attenuating any of these secondary insults.[16-18]

SPECIFIC ATHLETIC CRANIAL INJURIES

According to the Centers for Disease Control and Prevention, an estimated 1.6 to 3.8 million sports-related TBIs occur annually, and the majority are considered mild and do not receive emergency room or hospital treatment.[19] Epidemiological data obtained by Bailes and Cantu[16] have demonstrated that <0.5% of competitive high school and collegiate traumas are major head injuries. Football accounted for the overwhelming majority of such injuries (>90%), and fatalities occurred at a rate of 0.11 per 100,000 participants. With this in mind, various procedures have been generated to promote early recognition of potentially life-threatening intracranial lesions. For instance, the Canadian CT Head Rule (Table 2) and New Orleans Criteria (Table 3) were created as screening tools for cranial pathology in those sustaining mild head injuries.[20,21] Prospective validation studies have since been performed and have demonstrated a 100% sensitivity and 60% to 76% specificity for the Canadian CT Head Rule, which has more stringent criteria, and an 82% to 100% sensitivity and 12% to 26% specificity for the New Orleans Criteria in identifying lesions that require neurosurgical intervention.[22,23] On the other hand, recognizing that brain computed tomography (CT) contributes little to concussion evaluation, the 4th International Consensus Statement on Concussion in Sport recommends imaging only when there is suspicion of an intracerebral or structural lesion (eg, a focal neurological deficit, worsening symptoms, or prolonged state of altered sensorium).[15]

Skull Fractures

Acute structural alterations of the skull or intracranial contents are generally considered to be season-ending injuries. These criteria include not only intracranial hemorrhage but also basilar and cranial vault fractures. The physician should be suspicious of skull fractures in the setting of a significant soft-tissue swelling or ecchymosis (around the eyes, raccoon's sign; behind the ear over the mastoid process, Battle's sign), visible or palpable bony deformity, tender bony prominences, and clinical evidence of cerebrospinal fluid leakage. Amnesia, altered consciousness, worsening headaches, and focal deficits also increase the likelihood of an underlying skull fracture. Athletes with occipital bone fractures frequently complain of suboccipital and neck pain. In these circumstances, play should be suspended until CT brain and bone imaging can be performed to exclude fractures and intracranial hemorrhage as the culprit.

The majority of simple depressed or linear fractures can be managed conservatively. Most basilar skull fractures represent extensions of cranial vault fractures but may pose potential threat to the neighboring neurovasculature, the hypothalamic-pituitary axis, and adjacent cranial nerves.[24] Depressed skull fractures should be surgically elevated when they are compound in nature, when there is radiographic or clinical evidence of injury to the underlying brain parenchyma, when there are significant concerns for cosmesis, and when bone depression extends beneath or below the adjacent inner table level.[24]

TABLE 2. Canadian CT Head Rules[a]

CT of the Head Indicated for Minor Head Injury With Any 1 of the Following
GCS of 13-15 after witnessed loss of consciousness, amnesia, or confusion
Medium-risk criteria
Retrograde amnesia >30 min
Dangerous mechanism of injury
High-risk criteria
GCS score <15 for >2 h
Suspected open or depressed skull fracture
Suspected basilar skull fracture
Emesis ≥2 times
>65 y of age

[a]CT, computed tomography; GCS, Glasgow Coma Scale.

TABLE 3. New Orleans CT Head Criteria[a]

CT of the Head Indicated for Minor Head Injury in Those With a GCS Score of 15 and Any 1 of the Below Criteria
Age > 60 y
Headache
Vomiting
Drug or alcohol intoxication
Seizures
Trauma above the clavicle
Persistent anterograde amnesia

[a]CT, computed tomography; GCS, Glasgow Coma Scale.

Vascular Injuries

Carotid injury, although uncommon in sports, may result from direct compressive forces, from neck hyperextension with extreme lateral rotation, or secondary to fractures, dislocations, or subluxations of adjacent bone (ie, jaw). The most common injury site is the high cervical internal carotid artery region as the artery nears the skull base, well above the cervical bifurcation into internal and external branches. The petrous and cavernous internal carotid artery segments may be injured secondary to basilar skull fractures. Carotid injuries with intravascular consequences (stroke) are characterized by the delayed onset (1-24 hours in 57%-73%) of cortical dysfunction (aphasia, hemiparesis, hemisensory abnormalities, or visual deficits) secondary to ischemia resulting from vessel occlusion by an intimal flap, arterial thrombosis, or thromboembolism. Some patients may present

with ipsilateral headaches, carotidynia, and incomplete Horner syndrome from injury of the sympathetic nerves on the outside layers of the internal carotid artery.[25] Traumatic carotid-cavernous fistulas, rarely associated with athletic injuries, represent direct high-flow shunts between the cavernous carotid artery and cavernous sinus. Presenting signs and symptoms include impaired vision, diplopia, epistaxis, proptosis, chemosis, ocular bruit, and restricted extraocular movement.[25-27]

Vertebral artery injury is also a phenomenon associated with athletic competition. In contrast to carotid injury, the symptoms are more likely to be immediate in onset (neck pain, occipital headaches, and ischemia-related brainstem or cerebellar dysfunction). Injury may occur during hyperextension, which places the vertebral artery under stretch, and direct trauma to the artery by compressive forces or fractures, subluxations, and dislocations of the cervical spine. The most common level of injury is high in the neck (occiput-C2), near the point where the vertebral artery begins to bend and enters the skull.

Screening for a vascular injury can be performed with CT angiography or magnetic resonance angiography, but the player may require conventional angiography for definitive diagnosis or treatment. Management should be tailored to the patient's clinical presentation and may include thrombolytic agents, anticoagulation, or endovascular intervention to provide the best outcome.[28,29]

Intracranial Hematomas

Intracranial hemorrhages are the most common cause of sports-related fatalities. Subdural hematomas are the most frequent hematomas reported in football, whereas the incidence of epidural hematomas is higher in unhelmeted sports. Improvements in helmet design and regulations and other preventive measures have contributed to a downward trend in these serious lesions. A 54-year retrospective analysis by Cantu and Mueller[30] found that brain injuries accounted for 69% of football-related deaths and that subdural hematomas represented 86% of these lesions.[4] Surgical intervention in athletes experiencing intracranial hemorrhage should be managed in accordance with the Brain Trauma Foundation (BTF) recommendations, as outlined in Table 4.[31]

TABLE 4. Surgical Indications for Intracranial Hemorrhage[a]

Subdural hematoma
10-mm thickness and/or 5-mm midline shift
Deterioration of GCS score by ≥2 points in an individual whose GCS score is ≤8 despite degree of midline shift or clot thickness
Pupil asymmetry or fixed and dilated pupils and/or ICP >20 mm Hg
Epidural hematoma
15-mm thickness, 5-mm midline shift, or 30-cm^3 volume
Intraparenchymal hematoma
≥ 50-cm^3 volume
Frontal or temporal contusions ≥ 20 cm^3 with 5-mm midline shift and/or cisternal effacement
Parenchymal clots with progressive neurological deterioration referable to the lesion, refractory intracranial hypertension, or other signs of mass effect
Acute subdural hematoma
Neurological dysfunction or deterioration referable to the mass lesion
Effacement or obliteration of the fourth ventricle and basal cisterns
Obstructive hydrocephalus

[a]GCS, Glasgow Coma Scale; ICP, intracranial pressure.

Epidural Hematoma

This injury is most likely to occur in unhelmeted sports or in individuals struck in an unhelmeted region of the skull. Epidural hematomas may affect both the supratentorial and infratentorial compartments but are most common in the temporal region secondary to fracture of the squamous portion of the temporal bone, which causes laceration of the middle meningeal artery in most cases. It may occur in other regions secondary to venous pooling from the diploic spaces of fractured bones and worsen as a result of rundown from injured soft tissue. Classically, the affected individual has an initial loss of consciousness from impact and then regains consciousness before subsequently deteriorating. Other patients may remain unconscious from a rapidly expanding hematoma during brisk arterial bleeding and will have anisocoria from oculomotor nerve compression against the tentorium. These lesions must be rapidly identified and treated because the patients tend to do well since the brain is often spared of any direct injury. Surgical evacuation is indicated in the following circumstances: a clot ≥30 cm^3 in volume or 15 mm in thickness, ≥5 mm of midline shift, the presence of focal deficit, and a Glasgow Coma Scale (GCS) score <8.

Treatment typically is limited to a craniotomy and evacuation of epidural clot; however, in the setting of a moribund presentation, ICP monitoring with or without hemicraniectomy may be considered.[24,32,33]

Subdural Hematoma

This entity may present either acutely or chronically. Generally, acute subdural hematomas occur as a result of tears of bridging veins, but they may also result from accumulation of blood from cortical lacerations and rupture of intraparenchymal hemorrhage into the subdural space. These injuries are typically associated with a poor initial neurological examination as a result of concomitant intrinsic brain injury and are reported to have mortality rates as high as 60%.[3] On the other end of the spectrum, patients who had either small initial subdural hematomas or previously unrecognized subdural hematomas may present later with persistent headaches, altered mentation, blurred vision, nausea, focal weakness, and seizures. The subdural hematomas should be evacuated if the patient meets the following indications: clot ≥1 cm in thickness, ≥5 mm of midline shift, a decline in GCS score by ≥2 points, or anisocoria.

Chronic subdural hematomas can generally be managed via burr hole drainage, but the surgeon should be prepared to perform a craniotomy if clot membrane creates resistance to adequate

decompression. Acute subdural hematomas require craniotomy owing to the viscous nature of acute blood products, and craniectomy with ICP monitoring should be considered in those with an initial GCS score <9.[34]

Intracerebral Hemorrhage

Brain contusions commonly occur as a result of sudden acceleration and deceleration of the orbitofrontal lobes and temporal poles along the skull base, but they may also reflect direct trauma (ie, depressed skull fragments). Serial CT scans demonstrating stability are essential to avoid missing a potentially life-threatening lesion because contusions can coalesce or necrotic brain may progress the hemorrhage. The majority of hemorrhages stabilize in the initial 48 hours after injury. Surgery should be considered in the following situations: neurological deterioration referable to the contusion; contusion volume of ≥ 50 cm^3, frontal and temporal contusion volume ≥ 20 cm^3 in those with ≥ 5 mm of midline shift or effaced basal cisterns, and GCS score of 6 to 8.[3,35]

Diffuse Axonal Injury

This injury pattern is thought to account for the persistent comatose state in patients without radiographic evidence of hemorrhage or ischemia. It has been reported to affect as many as half of severe brain injuries and one-third of brain injury–related fatalities.[3] The spectrum ranges from mild to moderate to severe. It is characterized by the presence of punctuate hemorrhages within the white matter tracts that are best demonstrated on magnetic resonance imaging and is caused by rotational acceleration and deceleration forces. A landmark study of primates described various clinical grades that have been seen in humans as well that range from mild to moderate to severe. The mild subtype describes those who have been comatose 6 to 24 hours and may awaken with impaired memory and functional disturbances. Moderate injuries are characterized by comatose state for >24 hours and the presence of memory, behavioral, and cognitive deficits once consciousness is regained. Finally, severe cases are associated with comatose states that may persist for months and the presence of severe functional impairments once the injured patient awakens.[36] Patients may require ICP monitoring because of poor neurological status and may require other neurosurgical interventions to treat associated mass lesions in the acute setting.

Malignant Intracranial Hypertension

Posttraumatic cerebral edema develops as a result of impaired autoregulation of cerebral perfusion pressures. Consequently, the brain loses its ability to adjust vascular tone to maintain strict cerebral blood flow (CBF). Cerebral perfusion pressure then becomes dependent on systemic blood pressures that may fluctuate greatly in the acute management setting. This predisposes the brain to either hyperemia or hypoperfusion. This very mechanism is thought to account for the phenomenon known as second impact syndrome, which has been described as occurring when an athlete who suffered a previous mild head injury rapidly deteriorates after sustaining a recurrent injury during the recovery period. The player deteriorates rapidly within minutes, and mortality and morbidity are exceedingly high.[3] Neuroimaging often demonstrates global loss of cerebral sulcations, basal cistern effacement, and small ventricles as a result of diffuse cerebral edema.

Management is more challenging in this population because the injury pattern is global and patients are refractory to medical management. A multimodal, stepwise escalation of care should be executed to promote the best outcome. After diagnosis, ICP monitoring will be necessary to guide care in those with a GCS score <9 or patients who have deteriorated.[33] Rescue maneuvers such as hyperventilation, sedation, and hyperosmolar therapy can be used to temporize patients. Generally, hyperosmolar agents will have a treatment effect between 2 and 6 hours.[37] Early decompressive surgery should be considered in those who are refractory to medical management because it has been shown to improve ICPs and outcomes.[38,39] Hypothermia therapy has been shown to improve mortality risk and Glasgow Outcome Scale scores in patients who fail conventional treatment when used for >48 hours.[40] Alternatively, patients without hemodynamic compromise and under appropriate critical care monitoring may respond to barbiturate therapy. Responders have shown a 92% likelihood of survival compared with nonresponders (17%).[40,41]

EMERGENCY SIDELINE MANAGEMENT

The initial survey of any sports-related injury should first address and secure the ABCs: airway, breathing, and circulation. The airway must be assessed for both patency and the athlete's ability to protect himself or herself from aspiration of oral secretions, blood, and gastric contents. Individuals at high aspiration risk are those who demonstrate altered levels of consciousness or evidence of high cervical cord injury (quadriparesis/quadriplegia or belly breathing). It is important to remember that patients with impaired respiration may demonstrate spontaneous breathing, but tidal volumes may be inadequate for acceptable ventilation levels, thereby predisposing the nervous system to secondary injury. First responders should determine whether the injured athlete is breathing at a rate that may exhaust the accessory muscles of breathing, ultimately leading to hypoventilation. Athletes who need an artificial airway should have their cervical spine maintained in neutrality during this process. Intubation should be preceded by a chin lift instead of a neck extension. Nasotracheal intubations should be considered in those who are still spontaneously breathing to minimize the manipulation of a potentially unstable cervical spine.[42,43]

In the setting of an awake and cooperative patient, knowledge of a player's neuropsychological baseline is essential for both short- and long-term management after sports-related head injury. Various examination batteries have been created to aid in sideline assessment, which rely on intact consciousness, attentiveness, and memory. The Standardized Assessment of Concussion, generated by McCrea et al and later adopted by Bailes and Cantu,[16] and the Sports Concussion Assessment Tool V.3

(SCAT3), which is now the global standard, are useful tools for recognizing those who may have underlying structural injuries. In addition, any athlete removed from play must be monitored for signs of neurological deterioration, which may be indicative of worsening intracranial hemorrhage or cerebral edema. Although various factors such as fatigue, gender, age, illness, and education have a significant impact on the screening results, any deviation from that player's baseline should be considered a warning of intracranial pathology that may warrant emergent intervention.[44,45]

TRANSPORT AND MANAGEMENT OF PROTECTIVE EQUIPMENT

Multisystem trauma may exist and limit the neurological examination, or the trauma may be isolated to the brain, brainstem, spinal cord, nerve roots, or peripheral nerves.[42] Until clear, the athlete should be managed as if he/she harbors an unstable spine and spinal cord injury. The athlete should be placed on a rigid spine board while maintaining in-line stabilization of the spinal column. This maneuver can be achieved via either a log roll or 6-person lift. The log roll can be performed safely with 4 people: One manages the head to maintain cervical neutrality and provides the count; another cradles the shoulders and hips during the roll; a third ensures in-line movement of the lower extremities; and the fourth slides the board beneath the player during the uniform motion. The 6-person lift requires a scoop stretcher to transport the player to the spine board. Once the athlete is on the spine board, the head, torso, pelvis, and legs should be strapped to the board to maintain immobilization. The head is secured by placing towels, blocks, or commercial immobilizers on each side before taping. Currently, it is recommended that players wearing helmets and pads retain these devices during placement of cervical orthoses to prevent unnecessary cervical motion in attempts to remove the equipment. Once the spine has been immobilized and the athlete has been taken to the emergency department, the helmet and pads can be removed as a unit. Table 5 summarizes exceptions to this practice.[42,43]

We recommend the following protocol when transporting injured players:

1. All unconscious or obtunded patients should be managed under the presumption that the cervical spine is unstable and should have their spines immobilized.
2. Anyone with complaints of neck pain, paresthesias, or focal deficits should also have the cervical spine immobilized.
3. At least 4 individuals should participate in the log roll: 1 person secures the head and neck; another rolls the shoulder and hips; another maintains alignment of the legs with spinal axis; and the other carefully places the spine board beneath the player once sufficiently rolled. The individual securing the head and neck is responsible for the count.
4. The patient is then carefully secured to the spine boards, and spine immobilizers can be used at this time.[46,47]

TABLE 5. National Athletic Trainers' Association Guidelines: Indications for Helmet Removal[a]

Facemask cannot be removed in a reasonable amount of time
Airway cannot be established even with the facemask removed
Life-threatening hemorrhage under the helmet that can be controlled only by helmet removal
Helmet and strap do not hold head securely, so immobilizing the helmet does not adequately immobilize the spine
Helmet prevents immobilization for transportation in an appropriate position
Patient is unstable (physician's decision)

[a]Early helmet removal, that is, before standard stabilization and clearance methods have been completed.

EMERGENCY CARE PLAN, PREHOSPITAL CARE, AND CRITICAL CARE MANAGEMENT

Emergency Care Plan

The Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete and the National Athletic Trainers' Association have published guidelines educating all medical personnel and coaches about emergency planning for the initial management of the head- or neck-injured athlete. Every institution participating in organized sports must adopt an emergency care plan (ECP). ECPs ensure that appropriate measures have been created to promote rapid and efficient treatment of an athlete at risk for a potentially catastrophic sports injury. Furthermore, ECPs provide responsible medical personnel with a standard of care, both as an organization and individually, intended to arm them with the appropriate knowledge and skills needed to recognize, react, properly stabilize, and transport any patient with a suspected catastrophic head or spinal cord injury.[18,48]

Preparation includes ensuring that all proper equipment is available and functioning, ensuring that treating personnel are appropriately certified in administrating emergency medical services, and conducting rehearsals to ensure that rapid recognition and stabilization can be executed flawlessly in the event of a catastrophic injury. For high-risk events such as high-velocity or collision sports, emphasis should be placed on having an onsite ambulance and emergency medical services personnel. Access to an emergency medical facility should be incorporated into the ECP with consideration given to both location and the tertiary trauma services available at the facility. The ECP should be reviewed with proper medical facility administrators and clinicians to ensure the availability of necessary treatment measures and that advance notice of athletic events is given to ensure preparedness. Finally, an ECP should be reviewed and updated by all medical personnel, institutional administration, coaches, and any other organizations or individuals potentially involved in its seamless execution on no less than an annual basis.[48]

In the initial on-field management of an injured athlete, it is important to quickly determine a need for higher level of care and,

depending on the severity of injury, to alert the proper response team. On-field personnel should understand the mode of transportation and choice of destination, factors that ultimately determine the timing of treatment and likely make a significant difference in the morbidity and mortality of TBI patients. Air transport by helicopter has shown a decrease in mortality compared with ground transportation, possibly because of the higher level of training of the response team and the destination being a level I trauma center.[49] Although the triage of trauma patients varies from region to region, in general, an athlete's GCS score guides the appropriateness of triage and transportation. Patients who have a GCS score of 14 to 15 with no focal neurological deficit can be transported by ground to the local emergency room. Athletes with a moderate TBI (GCS score, 9-13) or severe TBI (GCS score $\leq$ 8) should be transported to the nearest high-volume, preferably level I, trauma center with specialized neurocritical and neurosurgical physicians experienced in the acute management of TBI.[49,50]

Prehospital Management

There is a fine balance between the time consumed for appropriate prehospital resuscitation/stabilization and the time delay this introduces in those who might require urgent surgical intervention. For example, surgical evacuation of subdural hematomas in <4 hours is associated with a significantly lower mortality rate than intervention after 4 hours (<30% and >90%, respectively).[51,52] However, prehospital hypoxia and hypotension significantly worsen outcomes in patients with TBI compared with the general population.[18,53-56]

Initiation of appropriate medical treatment in an athlete who has experienced a TBI is essential to improving overall outcome and minimizing morbidity and mortality associated with secondary brain injury.[18,37] Under normal physiological conditions, the brain uses aerobic metabolism to provide enough energy to meet the high demands of neuronal activity. Through autoregulation of blood vessel tone, normal brain parenchyma has the ability to maintain a constant CBF despite a range of systemic blood pressures. Local alterations in CBF are meant to match metabolic demands. In severe TBI, however, these mechanisms are disrupted, and the brain becomes dependent on systemic pressures to maintain CBF and to meet metabolic needs.[53] As a result, the clinician must maintain the cerebral milieu during a brain-injured state by addressing mass lesions, hypotension, hypoxemia, and electrolyte disturbances.

Hypoxia (PaO_2 < 60 mm Hg) has been demonstrated in several trials to be independently associated with worse outcomes in severe TBI.[54,55] Athletes sustaining a head injury should be monitored for hypoxemia (oxygen saturation $\leq$ 90%), and all patients should receive supplemental oxygen to maintain appropriate saturation levels. The BTF currently recommends endotracheal intubation (in lieu of supplemental oxygenation) as the mainstay of treatment in an unresponsive patient (GCS score $\leq$ 8), patients incapable of airway protection, and hypoxic patients.[37] Carbon dioxide, a potent cerebral vasodilator, should be kept at low-normal levels ($PaCO_2$, 30-35 mm Hg). Currently, the BTF guidelines cite Level II evidence against prophylactic hyperventilation to a $PaCO_2$ <25 mm Hg. Furthermore, Level III evidence states that hyperventilation should be used only as a temporizing measure for elevated ICP, that hyperventilation should be avoided within the first 24 hours of injury, and that, when hyperventilation is used, jugular venous oxygen saturation or brain tissue oxygen tension is recommended to monitor oxygen delivery.[31] Hyperemia and malignant ICPs can occur in the setting of hypoventilation secondary to concomitant vasodilatation and increased CBF. Conversely, hyperventilation and low $PaCO_2$ can lead to cerebral vasoconstriction, thereby reducing CBF and complicating ischemia-prone regions of the central nervous system.

Marmarou et al[55] reviewed 295 cases in the Trauma Coma Database and found that systolic hypotension (systolic blood pressure $\leq$ 80 mm Hg) predicted worse neurological outcomes. Furthermore, prolonged periods of hypotension were associated with worse outcomes. Chestnut et al[57] analyzed a larger number of patients from the Trauma Coma Database and observed a 150% increase in mortality in patients with systolic blood pressure $\leq$ 90 mm Hg. When fluid resuscitation is needed, administration of intravenous isotonic fluids is traditionally used for volume resuscitation because of its availability and low cost, although there may be a role for hypertonic saline (see below).

In the general trauma literature, several critics advocate foregoing volume resuscitation while in transport, citing that gaining intravenous access can delay arrival to the emergency center compared with the short duration of the trip and that dilution of coagulation factors is more detrimental than a "permissive hypotension."[58] However, after TBI, hypotension is perhaps more detrimental because decreases in CBF have been associated with increased ICP and worse outcomes.[56,59] Hypertonic saline has been increasingly advocated for initial resuscitation because it expands the intravascular volume by 4 to 10 times the volume infused while simultaneously decreasing cerebral edema through osmotic pressure.[60] Recent studies have also shown that platelet transfusion for resuscitation of TBI patients may be associated with improved outcomes.[61]

If the athlete's neurological function becomes increasingly altered with a declining GCS score, a strong suspicion for an expanding intracranial lesion and increased ICP should arise. The development of irregular pupils is a cardinal sign of uncal herniation and impending transtentorial shift with resultant brainstem compression. Although the definitive treatment of an expanding intracranial lesion is surgical intervention, temporizing measures can be used before tertiary care can be instituted. After the establishment of adequate airway protection, oxygenation, and volume resuscitation, hyperosmotic medications such as hypertonic saline or mannitol will aid by acutely lowering the ICP.[62] However, transport to a trauma center with neurosurgical staff equipped to definitively diagnose and treat ICP should remain the priority.

Critical Care/Hospital Course

On arrival to the emergency center, rapid evaluation and continued resuscitation measures should be used. Patients who are hemodynamically stable need to receive an emergent CT scan of the brain and cervical spine, with additional studies appropriate to their injury profile. If the CT scan demonstrates any acute intracranial abnormality potentially requiring neurosurgical intervention, emergent action, which may be local or require transport to a trauma center, is mandated. Early surgical intervention has consistently been demonstrated to decrease morbidity and mortality in TBI.[51,52]

After stabilization and any surgical interventions are performed, any athlete who continues to experience signs of severely altered sensorium or CT evidence of intracranial injury needs to be monitored closely in an intensive care unit, preferably a specialized neurosurgical/neurological intensive care unit. The development of subspecialized neurointensivists intimately involved with the management of patients with TBI has been associated with a decrease in hospital-related mortality, improved disposition, and more organized, documented plans of care.[63] Neurocritical care doctors, either neurosurgeons or other specialists, collaborate to work as a team analyzing the primary injury and minimizing secondary injury. Diligent monitoring and stabilization of the hemodynamic status, ventilation/oxygenation, blood glucose, nutritional status, and deep vein thrombosis prevention have dramatically improved morbidity and mortality.[41]

ICP monitoring has become standard of care in the United States, according to the BTF guidelines, and any patient with an abnormal CT scan and a GCS score ≤ 8 is a candidate for ICP monitoring.[33] Multimodal intracranial monitoring of the injured patient, using brain tissue oxygenation, microdialysis, CBF, and continuous EEG monitoring, is increasingly applied in neurocritical care units. Traditional monitoring was based on trends from specific time points, but the newer multimodal monitoring allows a clinician to assess the injured patient continuously and to detect subtle changes in physiological activity. Multimodal monitoring subjects the patient to further invasive procedures, and there currently is a paucity of evidence to support the routine use of these additional measures. Ongoing TBI research is aimed at determining the appropriate combination of multimodal monitoring and demonstrating benefit from correcting cerebral metabolism.[64]

CONCLUSION

The majority of neurological injuries suffered in sports are in the mild spectrum. However, severe injuries to the brain and spinal cord do occur, and we must be prepared for the worst to adequately protect our athletes. Having a clear plan of action that is well thought out and rehearsed and understanding the types of injuries seen in athletic competition will lead to improved outcomes for the players.

A podcast associated with this article can be accessed online (http://links.lww.com/NEU/A667).

Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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