

# Sideline and ringside evaluation for brain and spinal injuries

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✓Participation in contact and collision sports carries an inherent risk of injury to the athlete, with damage to the nervous system producing the most potential for significant morbidity and death. Neurological injuries suffered during athletic competition must be treated promptly and correctly to optimize outcome, and differentiation between minor and serious damage is the foundation of sideline/ringside management of the injury. In this article the authors present a guide to the sideline or ringside identification and management of head and spinal injuries.

**KEY WORDS** • brain injury • cervical spine injury • boxing • sports medicine

**P**ARTICIPATION in contact and collision sports involves an inherent risk of injury to the athlete, with injuries to the nervous system bearing the most potential for significant morbidity and death. In fact, head and neck injuries account for up to 70% of deaths from trauma and 20% of cases of permanent disability in athletes,<sup>52,82</sup> and have been described in virtually every sport. This association often necessitates involvement of the neurosurgical community in the field of sports medicine.

Differentiation between minor and serious damage is the foundation of sideline/ringside management of the injury. A seemingly minor blow to the head can result in a slowly developing SDH, whereas, paradoxically, a more severe impact can cause LOC but only a “minor” concussion. This article can be used as a guide to the sideline or ringside identification and management of head and spinal injuries.

## Head Injuries

It is estimated that 750,000 Americans per year suffer injuries while engaging in unorganized recreational activities, with 82,000 (10.9%) sustaining some form of head injury. The frequency of concussions related to organized sports has been estimated to be up to 300,000 annually in the US alone.<sup>19</sup> Severe sports-related head injuries include EDH, SDH, brain contusion and parenchymal hemorrhage, DAI, traumatic SAH, and cerebral edema. Mild TBI is a more common injury that poses difficulty with its management, particularly when consideration of the athlete’s return to competition is necessary.

*Abbreviations used in this paper:* AP = anteroposterior; CSF = cerebrospinal fluid; CT = computed tomography; DAI = diffuse axonal injury; EDH = epidural hematoma; ICP = intracranial pressure; LOC = loss of consciousness; MR = magnetic resonance; SAH = subarachnoid hemorrhage; SCI = spinal cord injury; SDH = subdural hematoma; TBI = traumatic brain injury; VB = vertebral body.

American football, ice hockey, and boxing are usually referenced when discussing sports-related head injury because of the frequent and obvious violent contact. Nevertheless, head injuries are often observed in sporting activities that are considered less violent. The US Consumer Product Safety Commission reported in 1990 that four of the top five sports that cause head injury requiring hospitalization are not the traditional “collision” games. These four were basketball, bicycling, baseball, and playground activities (football was the only collision sport to break the top five).<sup>27</sup> Although sports such as gymnastics and cheerleading have traditionally been responsible for the highest number of head injuries in the female athlete,<sup>49</sup> women are now participating in previously male-dominated sports such as boxing, and an increasing number of serious head injuries are being incurred by women in contact and collision sports.<sup>50</sup>

During the last century, our level of understanding of the types of cerebral insults, their causes, and their treatment advanced significantly. Recent research has better defined the epidemiological issues related to sports injuries involving the central nervous system, and has also led to classification and management paradigms that help guide decisions regarding athletes’ return to play.

## Epidural Hematoma

A common TBI in the athletic population, EDHs are found especially in sports in which the players do not wear helmets. These injuries have been described in baseball players and golfers struck on the head by a ball traveling at a high velocity.<sup>31,39,55,57</sup> Although the “lucid interval” associated with this injury only occurs in one third of athletes, an understanding of this clinical picture is crucial for all caregivers, especially athletic trainers, coaches, and team physicians.<sup>12</sup> A typical example of this situation would be a pole vaulter who strikes his or her head on the ground outside the landing pit. After being stunned for a brief period, the athlete may walk off the field fully alert.

Within 15 to 30 minutes they experience a sudden, excruciating headache accompanied by progressive neurological deterioration. This eventuality requires that an adequate observation period be planned for athletes who display potential for delayed hematoma formation and neurological deterioration. Early recognition and management are essential, and if treated early, complete neurological recovery can be expected, because EDHs are not usually associated with other brain injuries.

### *Subdural Hematoma*

The most common form of sports-related intracranial bleeding, SDHs account for the majority of lethal brain injuries.<sup>4,16</sup> It is important to understand that SDHs in athletes are not the same as those usually seen in the elderly population; the athlete usually does not have the large potential subdural space that an elderly patient possesses, so mass effect and increases in ICP occur more rapidly. In addition to injury from the mass effect of blood under the dura mater, there is often significant associated damage to the underlying brain (contusion or edema). Therefore, even with prompt treatment, the prognosis is less favorable than for an EDH, with mortality rates as high as 60%. An SDH can occur at any location in the brain, and presentation is usually within 72 hours of injury. Athletes who suffer an SDH may immediately become unconscious and/or experience focal neurological deficits, or the symptomatology may develop insidiously over days or even weeks.

### *Brain Contusion and Parenchymal Hemorrhage*

Brain contusions and parenchymal hemorrhages represent regions of primary neuronal and vascular injury. They contain edematous, punctate parenchymal hemorrhages that may extend into the white matter and the subdural and subarachnoid spaces and are usually the result of either direct trauma or an acceleration/deceleration injury. The latter causes the brain to strike the skull, most often resulting in damage to the inferior frontal and temporal lobes. The areas of the brain adjacent to the floor of the anterior or posterior cranial fossa, the sphenoid wing, the petrous ridge, the convexity of the skull, and the falx or tentorium are also vulnerable. Contusions can also be observed in the lateral midbrain, inferior cerebellum and adjacent tonsil, and in the midline superior cerebral cortex.

Importantly, these types of injuries often demonstrate progression over time with respect to the size and number of contusions and the amount of hemorrhage within them. This progression usually occurs during the first 24 to 48 hours, with one fourth of cases demonstrating delayed hemorrhage in areas that were previously free of blood. Additionally, initial CT findings can be normal or minimally abnormal because the partial volumes between the dense microhemorrhages and the hypodense associated edema can render contusions isoattenuating relative to the surrounding brain.

### *Diffuse Axonal Injury*

Diffuse axonal injury plays a significant role in sports-related head injury. It occurs in nearly half of athletes who have suffered a severe head injury and is partially respon-

sible for one third of all head injury–related fatalities.<sup>29</sup> Radiographically, DAI typically consists of several focal white matter lesions in a characteristic distribution.

The pathophysiological features of DAI were first described in 1943. It is the result of the shearing of multiple axons secondary to rotational forces on the brain, usually from lateral rotation of the head. These forces exert more effect on areas of the brain in which tissue density is greatest, such as at the gray–white junction. A DAI was classically believed to represent a primary injury (one occurring at the instant of the trauma). However, it is apparent that the axoplasmic membrane alteration, transport impairment, and retraction ball formation may represent secondary (or delayed) components of the disease process. Although the initial trauma may not completely tear the axon, it can still produce focal alteration of the membrane, resulting in subsequent impairment of axoplasmic transport. This results in axoplasmic swelling and rupture. A retraction ball forms, which is a pathological hallmark of shearing injury, followed by wallerian degeneration.

Although areas of the brain with different tissue densities have a predilection for DAI, the exact location depends on the plane of rotation and is independent of the distance from the center of rotation. The magnitude of injury depends on the distance from the center of rotation, the arc of rotation, and the duration and intensity of the force.

### *Traumatic SAH*

Sports-related head trauma can result in SAH. Some degree of SAH is usually present in any serious head injury. Although this usually results in meningeal irritation from blood between the pia mater and the arachnoid layer, the condition is usually not life threatening and immediate treatment is not required for a good outcome.<sup>58</sup> In large amounts, subarachnoid blood may lead to vasospasm. Also, SAH may result in the development of a communicating hydrocephalus, which can present clinically with a slower than expected recovery or late clinical deterioration.

### *Second-Impact Syndrome*

In 1984, the death of a college football player was reported that was thought to have resulted from a seemingly minor blow to the head following a more severe blow that had occurred days earlier. At the time it was hypothesized that this fatality was the result of “a repeat blow to an already compliance-compromised brain which precipitated a catastrophic increase in intracranial pressure, perhaps through a loss of vasomotor tone.”<sup>63</sup> The term “vascular congestion syndrome” was coined in 1991 following the death of a 17-year-old high school football player due to an uncontrollable increase in ICP.<sup>36</sup> Both of these deaths are thought to have been the result of what is now known as “second-impact syndrome.” This syndrome is defined as a fatal, uncontrollable increase in ICP caused by diffuse brain swelling that occurs after a blow to the head is sustained, before recovery from a previous blow to the head.<sup>37</sup> Significant controversy exists about the validity of this condition, due to problems with documentation of the initial event, persistent symptoms, and severity of the second impact.<sup>47</sup>

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The pathophysiological mechanism of second-impact syndrome is thought to involve a loss of autoregulation of the brain's blood supply, edema, and uncontrolled intracranial hypertension. This loss of autoregulation leads to vascular engorgement within the cranium, which in turn markedly increases ICP and leads to a syndrome of uncal herniation, cerebellar herniation, or both. Research in animals has shown that vascular engorgement in the brain after a mild head injury may be difficult if not impossible to control in this "double impact" setting. The usual time from second impact to uncontrollable edema is rapid (2–5 minutes).<sup>4</sup> There have been more than 20 reported cases of this condition,<sup>15</sup> which is most often seen in boxing, football, and ice hockey, and in most cases involves adolescent males or young adults.

Typically, the athlete experiences some degree of post-concussion symptoms after the first head injury. These may include visual, motor, or sensory changes and difficulty with cognitive and memory processes. Before these symptoms resolve, which may take days or weeks, the athlete returns to competition and receives a second blow to the head. The second impact may be minor, perhaps only involving a blow to the chest that jerks the athlete's head and indirectly imparts accelerative forces to the brain. Affected athletes may seem stunned but usually do not lose consciousness and often complete the play. They usually remain on their feet for 15 seconds to 1 minute or so, but seem dazed, similar to a Grade I concussion without LOC. Often, affected athletes remain on the playing field or walk off under their own power. Within seconds to minutes of the second impact, the athlete who is conscious yet stunned suddenly collapses to the ground and becomes comatose, with rapidly dilating pupils, loss of eye movement, and evidence of respiratory failure.

This phenomenon is associated with a 50% mortality and nearly a 100% morbidity rate.<sup>4</sup> It is important to understand this condition when making return-to-play decisions about an athlete who has suffered a head injury. Any athlete who is still symptomatic from a previous head injury should not be allowed to return to full practice or participation in a contact or collision sport.

### *Concussion and Mild TBI*

Concussions are by far the most common type of sports-related head injury; they account for approximately three fourths of all head injuries in this population. The recognition in the early 1980s that mild TBI exists as an important clinical entity began to pave the way for an increased appreciation of concussion in sports. In the 1990s, there was an increased focus on defining and categorizing mild TBI in athletes. There was more evidence to suggest that concussion may be more common and serious than previously believed,<sup>33,37,46,59</sup> and that the long-term sequelae of repetitive head trauma may be far from benign. In contrast to the attitudes in earlier times, when sustaining a concussion was considered an acceptable occurrence for a contact athlete, new evidence provided proof that cerebral dysfunction often persists.<sup>8,10,42,46</sup> The concept of mild TBI or concussion has evolved in recent years, aided in great part by the application of formal neuropsychological and cognitive studies and by studies of patients involved in vehicular accidents and other significant trauma.

There are many characteristics of the population with sports-related mild TBI and nuances in their condition that make diagnosis and treatment difficult. One such difficulty is that athletes are the only group of patients who routinely and often fervently ask to be returned to play, thus invariably subjecting themselves to multiple future instances of head impact.<sup>4</sup> Many of these impacts will result in at least subclinical head injury. Although a single episode of mild TBI seems to be well tolerated overall in the majority of athletes, long-term deficiencies in mental status have been thought to be associated with two or more episodes of concussion.<sup>23,30</sup> Advances in the fields of diagnostic neuroradiology, neurobiology, neuropsychology, and sports medicine now provide the neurosurgeon with more accurate and objective methods with which to analyze this population of patients.

There has been no universal agreement on the definition and grading of concussion, and attempts at classification have tended to focus on the presence or absence of a period of LOC and amnesia. However, concussion may present with any combination of the following signs and symptoms: a feeling of being stunned or seeing bright lights, a very brief LOC (lasting for seconds), lightheadedness, vertigo, loss of balance, headaches, cognitive and memory dysfunction, tinnitus, blurred vision, difficulty concentrating, lethargy, fatigue, personality changes, inability to perform daily activities, sleep disturbance, and motor or sensory symptoms. Numerous classification systems exist for grading the severity of concussion. Although there is little evidence-based support for any of the systems, because most have been developed based on clinical experience, three are in widespread use. These are the Cantu,<sup>14</sup> Colorado Medical Society,<sup>26</sup> and American Society of Neurology<sup>3</sup> systems, which also provide associated return-to-play guidelines.

### *On-Field Management*

Athletes who suffer catastrophic injuries to the head or spinal cord are usually easy to identify, as are those in whom an immediate neurological deficit develops. More challenging is the diagnosis of an injury with minimal initial symptomatology. The Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete was formed in 1998 and subsequently developed guidelines for the management of catastrophic injuries in athletes.<sup>2</sup> There are five categories of on-field management: 1) preparation for any neurological injury; 2) suspicion and recognition; 3) stabilization and safety; 4) immediate treatment and possible secondary treatment; and 5) evaluation for return to play.

It is mandatory that a backboard, cervical collar, and cardiopulmonary resuscitation equipment are on site and easily accessible during an athletic contest. Specific equipment for protective gear removal (for example, football face mask) should also be readily available. If a head or neck injury is suspected, an athlete should immediately be assessed for level of consciousness while still on the field. After the initial evaluation, as in any patient with head trauma, an athlete with a head injury should be assumed to have suffered an associated cervical injury, and spinal stabilization is essential to limit any further damage. If an athlete is wearing protective gear that includes a face mask, it should be removed.

The helmet itself or shoulder pads do not usually require removal on the field,<sup>84</sup> although several situations have been identified that would require removal of the helmet and chin strap. These include the following: 1) the player has a loose-fitting helmet that does not hold the head securely, so that if the helmet is immobilized the head will still be mobile; 2) the airway cannot be controlled or ventilation provided even after removal of the face mask; 3) the face mask cannot be removed after a reasonable period of time; and 4) the helmet prevents immobilization for transportation of the player in an appropriate position.<sup>3</sup> If necessary, helmet removal should be performed with concomitant occipital support or simultaneous removal of the shoulder pads. If left in place following helmet removal, the shoulder pads may cause cervical hyperextension. Obviously, if the helmet is removed, cervical immobilization must be maintained during the procedure.

In a neurologically intact athlete who has a normal mental status and in whom cervical spine involvement has been excluded, the player may be assisted to a sitting position and, if stable in this position, to a standing position. If able to stand, the athlete can then be walked off the field for further evaluation. Unconscious athletes need to be stabilized before any neurological appraisal is conducted. Initial evaluation should begin with assessment of the airway, breathing, and circulation (the basic cardiopulmonary life support system). Cardiopulmonary support can most often be accomplished by face-mask removal for airway access. The front of the shoulder pads can also be opened to allow compression and/or defibrillation.<sup>3</sup> When sudden unconsciousness occurs without preceding craniocervical trauma, a cardiac origin should be considered. An athlete with prolonged alteration of consciousness, worsening symptoms, or focal neurological deficit should be immediately transferred to a facility offering neurosurgical capabilities. Transport should be performed based on the assumption of a concomitant SCI, and spinal stabilization is mandatory.

#### *Ringside Management*

The role of the physician involved in the ringside management of injuries incurred by athletes competing in boxing or mixed martial arts competitions is to monitor participants for signs and symptoms of serious injury and provide immediate injury assessment as well as to give emergency care and supervise the rapid and orderly transfer to a hospital, if necessary. The practitioner will frequently be called on to evaluate the participants following concussion or brief LOC caused by a blow to the head. During this evaluation, he or she must consider the unique nature of these matches, and in most cases cannot pull the athlete from competition for observation at the first sign of insult to the neurological system without terminating the match.

The US Amateur Boxing Association, which has one of the safest records for its athletes, suggests that the physician enter the ring when requested by the referee to evaluate and aid the athlete who has been dropped or is suspected of suffering a serious injury, or following a standing eight count. The physician may, at his or her own discretion, indicate between rounds to the referee that he or she

wants to examine a boxer. The referee will then signal "stop" at the beginning of the next round and escort the boxer to ringside for the physician's evaluation. This is preferred to the physician attempting to examine the athlete between rounds during the limited time the boxer has to rest and be given instructions from his or her corner. The ringside physician can suspend the bout at any time by mounting the ring apron or by asking the timekeeper to ring the bell.<sup>83</sup> This should be done if the physician thinks that a participant is in any danger of serious physical injury, and this decision should take precedence over all other considerations.

During the match, the ringside physician monitors the participants for signs of excess fatigue or injury, and the athlete's ability to defend him- or herself is constantly assessed. When athletes are leaning on the ropes or cage, rapid determinations must be made as to whether the boxer is using them to maneuver or whether these structures are holding him or her upright.

As with any fallen athlete, when a ringside physician enters the ring to assess a boxer with altered mental status, it must first be ascertained whether the athlete has an adequate airway and pulse. The mouthpiece, if in place, should be removed and emesis and/or signs of aspiration should be guarded against. Next the physician should establish the athlete's level of consciousness and look for spontaneous movement of the extremities and for signs of seizure.<sup>40</sup> Evaluation of pupillary responses, although of low yield during the 1st minute of unconsciousness, may provide early signs of the presence of increases in ICP. If the boxer remains unconscious for more than 1 to 2 minutes, he or she should be expeditiously transferred by stretcher to an ambulance and then to a prearranged referral hospital.

The athlete should remain lying down until fully reactive. If an athlete is responding to simple commands within 1 to 2 minutes and is moving all extremities voluntarily, more time may be taken to assess him or her for recovery before transport needs are determined. If the athlete's recovery is slowed beyond 2 minutes or is regressive, the individual should be transported to a designated hospital.<sup>40</sup> Gentle restraint is used for a confused athlete who tries to get immediately to his or her feet. Once reactive, the boxer can be permitted to sit, then stand up, then walk to his or her corner. A detailed neurological examination is then performed to establish a baseline for future reference, and the athlete is further observed. Facilities should be available for continued close observation under the direct supervision of the ringside physician. If recovery progresses satisfactorily, with no evidence to suggest a progressive intracranial injury process, the boxer can be released to the care and further observation of his or her coach, family, or other responsible adults. The athlete and/or guardian should be given information about head injury.<sup>83</sup> If no responsible party is available, serious consideration should be given to admission to a designated hospital for 12 to 24 hours of observation. Likewise, an athlete who suffers amnesia, confusion, nausea, headache, dizziness, or speech or gait disturbances should be transferred to a designated hospital for evaluation.

#### *Neuroimaging Guidelines*

Specific guidelines on when to perform brain imaging

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in a head-injured athlete do not exist. Because of this, the physician needs to individualize when to perform imaging on a patient-by-patient basis. Patients who exhibit focal neurological deficits, persistent alterations in mental status, Glasgow Coma Scale scores of 13 or less, and the possibility of a skull fracture are common examples of cases in which an athlete would require at least a CT scan.<sup>66</sup> In cases that are not as clear-cut, the duration of LOC or amnesia have been used to aid in this decision.<sup>58</sup> If there is any doubt, CT scanning is a rapid and efficacious diagnostic modality.

### Spine Injuries

Although a spectrum of soft-tissue, bone, and nervous system injuries can occur to the spine of an athlete, and often result in significant disability and time lost from competition, SCI is perhaps the most feared consequence of athletic activities, and no other sports injury is potentially more catastrophic. Each year approximately 10,000 cases of SCI are reported in the US, and sporting events are the fourth most frequent cause of these injuries.<sup>5</sup> Sports-related SCIs also occur at a younger mean age (24 years) and are the second most frequent cause of SCI in the first three decades of life.<sup>25,53</sup> Several organized sports have been identified as placing the participant at high risk for SCI. These include football, ice hockey, rugby, skiing, snowboarding, and equestrian sports.<sup>41,60,64,67</sup>

A structural distortion of the cervical spinal column associated with actual or potential damage to the spinal cord is classified as a catastrophic cervical spine injury. Because this condition is fortunately rare, few physicians have extensive experience in the emergency care of these injuries. Improper handling of the patient on the field or during transport can worsen or precipitate spinal cord dysfunction. Failure to manage a catastrophic neck injury appropriately can result in compromise of the athlete's cardiac, respiratory, and neurological status. Improved understanding of these injuries can facilitate early diagnosis and effective on-field management.

#### *Mechanisms of Spine Injuries*

Cervical spine injury can be divided into several categories, including unstable fractures and dislocations, transient quadriplegia, and acute central disc herniation,<sup>6</sup> which produce neurological symptoms and signs that involve the extremities in a bilateral distribution. Sports-related cervical spine injuries have been divided into three groups, which provide useful information when making return-to-play decisions.<sup>5,45,85</sup> Type 1 injuries are those in which the athlete sustains a permanent SCI. This includes both immediate, complete paralysis and incomplete SCI syndromes. The incomplete injuries are basically of four types: Brown-Séquard syndrome, anterior spinal syndrome, central cord syndrome, and mixed types. Mixed types include the finding of crossed motor and sensory deficits, with upper extremities more prominently involved, which is considered to be a central cord/Brown-Séquard variant. In addition, there are a few individuals in whom the neurological deficit may be relatively minor but is associated with demonstrable spinal cord damage on imaging studies. For example, a high-intensity lesion within the spinal cord seen on MR imaging documents a spinal cord contusion.

Type 2 injuries occur in individuals with normal results on radiographic studies. These deficits resolve completely within minutes to hours, and eventually the athlete has normal results on neurological examination. An example of a Type 2 injury is the "burning hands syndrome," a variant of central cord syndrome characterized by burning dysesthesia of the hands and associated weakness in the hands and arm.<sup>44</sup> Most of these patients have normal results on radiographic studies, and their symptoms completely resolve within approximately 24 hours. Type 3 injuries comprise players with radiographic abnormalities but without neurological deficits. This category includes fractures, fractures with dislocations, ligamentous and soft-tissue injuries, and herniated intervertebral discs.

Spinal cord injury can also be divided into the upper (occiput, atlas, and axis) and lower (C3-T1) cervical spine. A thorough understanding of the normal anatomy and unique motion of the spine at various segments is mandatory when treating these injuries.

Unstable fracture and/or dislocation is the most frequent cause of catastrophic cervical spine trauma. The most common primary injury vector is axial loading with flexion in football and hockey.<sup>68,80</sup> Eighty percent of injuries to the cervical spine result from the accelerating head and body striking a stationary object or another player.<sup>9,24</sup> The cervical spine is compressed between the instantly decelerated head and the mass of the continuing body when an axial force is applied to the vertex of the helmet. In neutral alignment, the cervical spinal column is slightly extended as a result of its normal lordotic posture, and it is believed that compressive forces can be effectively dissipated by the paravertebral musculature and vertebral ligaments. This buffering cervical lordosis is eliminated when the cervical spinal column is straightened and large amounts of energy are transferred directly along the spine's longitudinal axis.<sup>80</sup> Under high enough loads, the cervical spine can respond to this compressive force by buckling.

Two major patterns of spinal column injury result from the compression injury vector. Compressive-flexion injury is the most common variant that results from the combination of axial loading and flexion. It results in shortening of the anterior column because of compressive failure of the VB and lengthening of the posterior column because of tensile failure of the spinal ligaments.<sup>77</sup> If a cervical vertebra is subjected to a relatively pure compression force, both the anterior and posterior column shorten, resulting in a vertical compression (burst) fracture. The VB essentially explodes, during which event it is possible that disc material extrudes through the fractured endplate, and retropulsion of osseous material into the spinal canal results in cord damage.<sup>1</sup> Alternatively, there may be significant SCI without major disruption of the spinal column's integrity. This type of injury is the result of transient spinal column distortion with energy transfer to the spinal cord.

Catastrophic cervical trauma caused by the primary disruptive vector (flexion) generally results from either a direct blow to the occipital region or rapid deceleration of the torso. The flexion-distraction injury most likely to result in spinal cord dysfunction is a bilateral facet dislocation.<sup>61,89</sup> Unilateral facet dislocation, which is associated with SCI in up to 25% of cases, can occur with the addition of axial rotation to the distractive force.<sup>22</sup> It should be

recognized that unstable cervical fractures and/or dislocations do not always result in upper motor neuron dysfunction. A unilateral facet dislocation can cause a monoradiculopathy due to foraminal compression of a nerve root on the side of the dislocated articular process. In other cases, major osseous or ligamentous damage will produce no neurological impairment. An SCI in these scenarios is potential rather than actual, based on the amount of loss of structural integrity of the vertebral column.<sup>6</sup>

#### *Upper Cervical Spine Injury*

In sports-related injuries, the upper cervical spine is considered to be the occiput, atlas (C-1), and axis (C-2). The major function of the atlantooccipital joint is motion in the sagittal plane, which accounts for 40% of normal flexion and extension of the spine and 5° to 10° of lateral bending. The midline atlantodens articulation is stabilized by the transverse atlantal ligament, which prevents forward translation of the atlas. This specialized osseoligamentous anatomy allows the atlas to rotate in a highly unconstrained manner. The atlantoaxial complex is responsible for 40 to 60% of all cervical rotation.<sup>28</sup> This rotation is limited by the alar ligaments extending from the odontoid process to the inner borders of the occipital condyles. The apical ligaments attach the odontoid centrally to the anterior foramen magnum. Atlantoaxial joint strength is provided by the transverse ligament and the lateral joint capsules.<sup>29</sup>

Spinal cord damage due to fractures or dislocations involving the upper cervical spine is rare because there is proportionately greater space available within the upper spinal canal compared with the lower cervical segments. Injuries that destabilize the atlantoaxial complex (fracture of the odontoid or rupture of the transverse atlantal ligament) are most likely to result in spinal cord dysfunction. Flexion is the most frequent cause of injury at the atlantoaxial joint. Odontoid fractures can also result from extension injuries. Unilateral rotary dislocations are usually the result of rotational forces. Cord compression is unusual with a burst fracture of the atlas or traumatic spondylolisthesis of the axis because these osseous injuries further expand the dimensions of the spinal canal. If AP radiographs are obtained and there is spreading of the lateral masses of more than 7 mm, the transverse ligament is probably torn. Bilateral pedicle fractures of the axis may occur from extension of the occiput on the cervical spine. Importantly, although these injuries can result in instability, they usually do not cause neurological deficits secondary to the anatomically wide spinal canal, which is also present at this level.<sup>29</sup> If an upper cervical cord injury does occur, diaphragmatic paralysis with acute respiratory insufficiency can occur along with quadriplegia, because the phrenic nerve arises from three cervical nerve roots (C3–5).

#### *Lower Cervical Spine Injury*

The lower cervical spine is composed of the C3–7 vertebrae. This area accounts for the remaining arcs of neck flexion, extension, lateral bending, and rotation, and has several important anatomical differences when compared with the upper cervical spine. The spinal canal is not as

wide at this level and the facet joints are oriented at a 45° angle. Because of this angulation, axial rotation is somewhat limited. The facet articulations also restrain forward vertebral translation.

Each motion segment can be separated into an anterior and a posterior column. Stability of a cervical segment is derived mainly from the anterior spinal elements. Compression of the spinal column is primarily resisted by the VBs and intervertebral disc, whereas shearing forces are opposed primarily by paraspinal musculature and ligamentous support. Instability of the lower cervical spine has been defined radiographically as translational displacement of two adjacent vertebrae more than 3.5 mm or angulation of more than 11° between adjacent vertebrae.<sup>87</sup>

The majority of fractures and dislocations occur in the lower cervical region. Lower cervical spine injuries are defined by the forces acting on the area (that is, flexion, extension, lateral rotation, and axial loading). Dislocated joints are usually the result of a flexion mechanism with either distraction or rotation. The ligamentous structures are the primary restraints on distraction of the spine.<sup>28</sup> Compression of the posterior structures as well as damage to the anterior ones is usually the result of extension or whiplash injuries. This mechanism of injury often results in tearing of the anterior longitudinal ligament and fractures of the posterior elements.<sup>29</sup>

Compressive forces usually result in VB fractures. These are commonly seen in so-called spear-tackler's spine, which consists of four characteristics: reversal of cervical lordosis, radiographic evidence of previous healed minor VB fractures, spinal canal stenosis, and the habitual use of spear-tackling techniques.<sup>78</sup> Players in this population often have a flexed posture of the head and a loss of the protective cervical lordosis. Large axial loads can result in protrusion of disc material or fractured bone into the spinal canal. This is the most common mechanism for sports-related quadriplegia.<sup>69,71</sup> The C3–4 level is most frequently involved in cases of quadriplegia caused by cervical dislocations.<sup>34,79</sup>

#### *Central Cord and Burning Hands Syndromes*

Injury to the lower cervical spinal cord can result in a spectrum of neurological dysfunction. Incomplete SCI can occur, with partial preservation of sensory or motor function. Central cord syndrome is the most common manifestation of this, followed in frequency by the anterior cord syndrome.

Burning hands syndrome is considered to be a variant of central cord syndrome. It is characterized by burning dysesthesia in both upper extremities and is probably the result of vascular insufficiency affecting the medial aspect of the somatotopically arranged spinothalamic tracts.<sup>44,88</sup> The lower extremities may occasionally be involved and weakness may sometimes be evident. Cervical spine fracture or soft-tissue injury is seen radiographically in 50% of the patients with this syndrome. This condition should initially be managed as an SCI in any athlete who exhibits it.<sup>38</sup>

#### *Cervical Cord Neurapraxia and Transient Quadriplegia*

Neurapraxia of the cervical spinal cord resulting in transient quadriplegia has been estimated to occur in seven of 10,000 football players.<sup>73</sup> This alarming injury is charac-

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terized by a temporary loss of motor or sensory function and is thought to be the result of a physiological conduction block without true anatomical disruption of neuronal tissue. The affected athlete may report pain, tingling, or loss of sensation bilaterally in the upper and/or lower extremities. A spectrum of muscle weakness is possible, ranging from mild quadriparesis to complete quadriplegia. The athlete has a full, pain-free range of cervical motion and does not report neck pain. Hemiparesis or hemisensory loss is also possible.

This condition is thought to result from a pincer-type mechanism of compression of the spinal cord between the posteroinferior portion of one VB and the lamina of the vertebra below.<sup>56</sup> The condition can also occur during hyperflexion, but usually with extension movements accompanied by infolding of the ligamentum flavum, which can result in a 30% or more reduction of the AP diameter of the spinal canal.<sup>51</sup> The spinal cord axons become unresponsive to stimulation for a variable period of time, essentially creating a postconcussive effect.<sup>90</sup>

Cervical cord neurapraxia is described by the neurological deficit, the duration of symptoms, and the anatomical distribution. A continuum of neurological deficits that range from sensory only or sensory disturbance with motor weakness to episodes of complete paralysis may occur. These may be described as paresthesia, paresis, and plegia. An injury is defined as Grade 1 if the symptoms of cervical cord neurapraxia do not persist for more than 15 minutes. Grade 2 injuries are defined as lasting from 15 minutes to 24 hours, and Grade 3 injuries persist for 24 to 48 hours. All four extremities may be involved; this is considered a “quad” pattern. Upper- and lower-extremity patterns may also be observed.<sup>72</sup>

By definition, this condition is transient; complete resolution generally occurs within 15 minutes, but may take up to 48 hours. Administration of steroid drugs in this population in accordance with the protocol of Bracken, et al.,<sup>11</sup> is controversial. There have been no controlled studies in which the administration of steroid agents has reportedly altered the natural history of the entity in athletes who have suffered cervical cord neurapraxia.<sup>17</sup>

In players who return to play football, the rate of recurrence has been reported to be as high as 56%.<sup>72</sup> Considerable controversy exists regarding whether the presence of cervical stenosis makes an athlete more prone to permanent neurological injury or transient quadriparesis. The AP diameter of the spinal canal (measured from the posterior aspect of the VB to the most anterior point on the spinal laminar line), as determined using lateral cervical spine radiographs, is considered normal if the distance between C-3 and C-7 is more than 15 mm. Cervical stenosis is considered to be present if the canal diameter is less than 13 mm. This measurement has significant variability, however, due to variations in landmarks used for measurement, changes in target distances for making the radiographs, patient positioning, differences in the triangular cross-sectional shape of the spinal canal, and magnification of the canal because of a patient's large body habitus.

In an effort to eliminate the variability in measurements of the spinal canal AP diameter, Torg and Pavlov<sup>75</sup> designed a ratio method for determining the presence of cervical stenosis, comparing the sagittal diameter of the spinal canal to the sagittal midbody diameter of the VB at the

same level. A ratio of 1:1 was considered normal, and less than 0.8 was indicative of significant cervical stenosis. This ratio was found to mislabel as stenotic the spinal canals in many athletes with adequately sized canals but large VBs. This observation as well as an unprecedented ability to image the vertebral column, intervertebral discs, spinal canal, CSF, and spinal cord directly, has made MR imaging and not bone landmarks the method currently preferred for assessing “functional spinal stenosis.” The CSF signal around the spinal cord (termed the “functional reserve”) can be assessed using MR imaging, and the visualization of the CSF signal, its attenuation in areas of stenosis, and changes on dynamic sagittal flexion–extension MR imaging studies are of paramount importance in the diagnosis of this condition. In cases involving an absent CSF pattern on axial and, particularly, sagittal MR images, functional stenosis is diagnosed.

Developmental or acquired cervical stenosis seems to be a predisposition to cervical cord neurapraxia.<sup>70,74</sup> It had been previously argued by Torg and colleagues<sup>74,76</sup> that young patients who suffered an episode of cervical cord neurapraxia were not predisposed to permanent neurological injury. This assumption has recently been called into question now that a player who had experienced a cervical cord neurapraxia subsequently sustained a quadriplegic injury.<sup>13</sup>

### *Traumatic Intervertebral Disc Herniation*

Acute herniation of an intervertebral disc can occur during participation in sports and in the athletic population. Extrusion of disc material into the central spinal canal can result in acute cord compression and a transient or permanent SCI. Clinically, the athlete may present with acute paralysis of all four extremities and a loss of pain and temperature sensation. A traumatic central disc herniation is also typically accompanied by the sudden onset of posterior neck pain and/or paraspinal muscle spasm as well as true radicular arm pain or referred pain to the periscapular area.<sup>6</sup>

### *Stingers, Burners, Transient Brachial Plexopathy, or Nerve Root Neurapraxia*

This condition, which is known by various names, is one of the most common occurrences in collision sports, and it is not the result of an SCI. It was first described in 1965 by Chrisman, et al.<sup>20</sup> Because the mechanism was thought to be direct force applied to the shoulder with the neck flexed laterally away from the point of contact, the condition has also been referred to as “cervical pinch syndrome.”<sup>32</sup> This is a transient neurological event characterized by pain and paresthesia in a single upper extremity following a blow to the head or shoulder. The symptoms most often involve the C-5 and C-6 spinal roots. The affected athlete can experience burning, tingling, or numbness in a circumferential or dermatomal distribution. The symptoms may radiate to the hand or remain localized in the neck. These athletes often maintain a slightly flexed cervical spine posture to reduce pressure on the affected nerve root at the neural foramen or hold and/or elevate the affected limb in an attempt to decrease tension on the upper cervical nerve roots.

Weakness in shoulder abduction, external rotation, and arm flexion are reliable indicators of the injury.<sup>86</sup> If weakness is a component, it usually involves the C5–6 neurotome. The radiating arm pain tends to resolve first (within minutes), followed by a return of motor function (within 24–48 hours). Although the condition is usually self-limiting and permanent sensorimotor deficits are rare, a variable degree of muscle weakness can last up to 6 weeks in a small percentage of cases.

This injury is usually the result of downward displacement of the shoulder, with concomitant lateral flexion of the neck toward the contralateral shoulder. This is thought to result in a traction injury to the brachial plexus. The condition may also be associated with ipsilateral head rotation with axial loading, resulting in neural foramen narrowing and compression and/or impaction of the exiting nerve root within the foramen.<sup>21,62</sup> Direct blunt trauma at the Erb point, which is located superficially in the supraclavicular region, has also been reported to cause stingers.<sup>43</sup> This can occur when an opponent's shoulder or helmet is driven into the affected athlete's shoulder pad directly over this area.

This injury has been graded using the Seddon criteria. A Grade 1 injury is essentially a neurapraxia, defined as transient motor or sensory deficit without structural axonal disruption. This type of injury usually resolves completely, and full recovery can be expected within 2 weeks. Grade 2 injuries are equivalent to axonotmesis, which involves axonal disruption with an intact outer supporting epineurium. This results in a neurological deficit that lasts for at least 2 weeks, and axonal injury may be demonstrated on electromyographic studies obtained 2 to 3 weeks postinjury. Grade 3 injuries are considered neurotmesis, or total destruction of the axon and all supporting tissue. These injuries persist for at least 1 year with little clinical improvement.

Cervical canal stenosis has been implicated as a risk factor for stingers.<sup>35</sup> The dimensions of the spinal cord remain relatively constant in the subaxial cervical spine,<sup>54</sup> with an average midsagittal cord diameter in the range of 8 to 9 mm. In contrast, the size of the vertebral canal in the lower cervical region shows significant individual variation. Determining the functional reserve (the amount of CSF surrounding the spinal cord, as defined earlier) can be accomplished using MR imaging, and is currently the preferred method for assessing functional spinal stenosis.

Stingers with prolonged neurological symptoms are the most common reason for cervical spine evaluations performed in high school and college athletes in an emergency room.<sup>17,18,48</sup> The athlete usually demonstrates a full, pain-free arc of neck motion with no midline palpation tenderness on examination. If tenderness is present or unilateral neurological symptoms persist, a paracentral disc herniation with associated nerve root compression should be considered. This is usually accompanied by the sudden onset of posterior neck pain and spasm. Monoradiculopathy characterized by radiating pain, paresthesias, and/or weakness in the upper extremity also occurs due to compression and inflammation of the cervical root.

#### *On-Field and Ringside Management*

The immediate treatment of the player who has suffered

an SCI should follow standard trauma protocols that address airway, breathing, and circulation function. The initial objective in this primary survey is to assess the athlete for immediately life-threatening conditions and to prevent further injury. During this primary survey, appropriate resuscitation procedures are instituted and the emergency medical system is activated immediately after recognizing a life-threatening problem or serious spinal injury.<sup>7</sup>

Following the primary survey, one of three clinical scenarios will become apparent: 1) actual or impending cardiopulmonary collapse; 2) altered mental status but no compromise of the cardiovascular or respiratory system; or 3) a normal level of consciousness and normal cardiopulmonary function.

If the athlete is experiencing cardiopulmonary collapse, the use of advanced cardiac life support principles is essential. If the athlete is lying prone, he or she must be carefully "logrolled" into a supine position on a rigid backboard if one is available. Any face mask should be removed rapidly to provide adequate airway access. As mentioned earlier, removal of the helmet, headgear, and/or shoulder pads is not routinely indicated unless the equipment interferes with resuscitation of the patient. If still in place, the mouthpiece should be taken out while manual stabilization of the neck in a neutral position is maintained. Airway evaluation should be performed with the understanding that obstruction can be caused by a foreign body, facial fractures, or direct injury to the trachea or larynx. A depressed level of consciousness can also contribute to the inability to maintain an airway.

If breathing is of insufficient depth or rate, assisted ventilation is required. On the field, this usually is performed using a bag-valve device and face mask. Hypoxia should be rapidly corrected by providing adequate ventilation, with protection of the vertebral column at all times. In patients with a patent airway, respiratory collapse could be due to an upper cervical SCI, and may result in paralysis of the diaphragm and accessory breathing muscles. Indications for definitive airway control by endotracheal intubation include apnea, inability to maintain oxygenation with face-mask supplementation, and protection from aspiration. Circulation must also be addressed during the primary survey. Neurogenic shock due to SCI could result in diminished amplitude of the peripheral pulses in combination with bradycardia. If the femoral or carotid pulses are not palpable, cardiopulmonary resuscitation is required. If this is the case, the front of the shoulder pads can be opened to allow for chest compressions and/or defibrillation.

If the athlete is found to have an altered mental status without cardiopulmonary compromise, a brief neurological examination can be performed. The prevention of further injury to the spinal cord is of primary importance, and once initial resuscitation and evaluation are performed, the focus should be placed on immobilization. The helmet and shoulder pads should remain in place unless removal is required to gain access to the airway. Neutral axial alignment and occipital support must be maintained. If a player is unconscious, he or she should be logrolled into a supine position and the mouthpiece removed.

If, after completion of the primary survey, the athlete is found to have a normal mental status without cardiopulmonary compromise, a neurological assessment should be

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performed. If the individual exhibits symptoms or signs referable to spinal cord damage, a catastrophic cervical cord trauma should be assumed. If results of the neurological assessment are normal but the athlete exhibits cervicothoracic pain, focal spinal tenderness, or restricted neck motion, an unstable spinal column injury with potential cord compromise is assumed.

Removal of the player from the field should be performed with strict attention given to immobilization of the spine. A rigid backboard with cervical collar or bolsters on the sides of the head should be used. It is important to remember that the athlete's helmet may cause unintended cervical flexion on a rigid backboard. Once the athlete arrives at the hospital, if these are still in place, the helmet/headgear or shoulder pads should be removed before a radiographic examination is performed.

Athletes who suffer a burner should be immediately removed from competition until symptoms have fully resolved. Management of this injury in the participant who receives it is often dependent on the presence of residual symptoms. The entity is usually considered an isolated benign injury. On-field evaluation should include palpation of the cervical spine to determine any points of tenderness or deformity. Evaluation of sensation and muscle strength should be performed using the unaffected limb as a point of reference if necessary. Weakness in the muscles innervated by the upper trunk of the brachial plexus is often observed. These include the deltoid (C-5), biceps (C5-6), supraspinatus (C5-6), and infraspinatus (C5-6) muscles.<sup>81</sup> The shoulder of the affected limb should also be evaluated, with particular attention given to the clavicle, acromioclavicular joint, and supraclavicular and glenohumeral regions. Percussion of the Erb point can be performed in an attempt to elicit radiating symptoms.

Obviously, the athlete should be evaluated for other serious injuries such as cervical spine fractures and dislocations. It is unusual to find lower brachial trunk injury patterns involving the C-7 or C-8 nerve roots. It is also uncommon to see persistent sensory deficits involving either the lower or upper extremities. This condition is always unilateral and has never been reported to involve the lower extremities. If bilateral upper-extremity deficits are present, SCI should be at the top of the list for differential diagnosis. Localized neck stiffness or tenderness with apprehension to active cervical movement should alert the examiner to a potentially serious injury and the subsequent initiation of full spinal precautions, including backboard immobilization and transport for advanced neuroimaging.

If the player does not report neck pain, decreased range of motion, or residual symptoms, he or she can usually return to competition. If symptoms do not resolve or there is persistent pain, prompt assessment of the brachial plexus by using MR imaging is recommended. If the symptoms persist for more than 2 weeks, electromyography can be performed to establish the distribution and degree of injury.<sup>29</sup> Residual muscle weakness, cervical anomalies, or abnormal results on electromyographic studies are exclusion criteria for return to play.<sup>17</sup>

By definition, stingers or burners are transient phenomena, and they usually do not require formal treatment. The athlete should be followed closely with repeated neurological examinations, because although the condition usu-

ally resolves in minutes, motor weakness can develop hours or days postinjury.<sup>48,86</sup> Repeated stingers may result in long-term muscle weakness with persistent paresthesias.<sup>65</sup> Options available for participants to decrease the risk of future occurrences include changing their field positions or modifying their playing technique.

### Conclusions

Neurological injuries suffered during athletic competition must be treated promptly and correctly to optimize outcome. Fortunately, catastrophic injuries are rare. Because of this, however, very few physicians have significant experience in the management of these injuries. A solid understanding of the treatment principles discussed in this article will improve the preparedness of a physician to deal with these situations and provide the athlete with the best chance of recovery.

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