



On-the-field evaluation of an athlete with a head or neck injury

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Care of the athlete is the responsibility of team physicians, athletic trainers, coaches and physical therapists. All of these involved individuals should understand the evaluation and management of neurologic injuries. Head and neck injuries are usually the result of either direct or indirect contact, and the consequences of these neurological injuries have a potentially high incidence of morbidity and mortality. It has been estimated that 70% of traumatic deaths and 20% of permanent disability in sports-related injuries are due to head and neck injuries [1,2]. Furthermore, attention has focused on the epidemic of concussions seen in sports and the resultant negative neuropsychologic sequelae seen in the scholar athlete [3].

Health care providers have investigated the mechanisms of injury and individual risk factors in an attempt to advance the safety of athletes. In addition, rules and regulations have been outlined by officiating bodies to facilitate the safety of the athlete. Ever-changing sport-specific rules and regulations as well as equipment modifications have been implemented in an effort to decrease the incidence of these injuries, with remarkably positive results [4,5].

The obligation is not only to treat the injured athlete, but also to prevent such injuries through proper conditioning, education and protective measures (Fig. 1). Health care providers and athletic officials must be made aware of the frequency and magnitude of these injuries so that they may provide optimal care for all athletes in all sports. The medical team caring for the athlete must be able to assess the injured athlete and make important decisions regarding his or her diagnosis and prognosis for further participation. An ostensibly minor head

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Fig. 1. Protective gear worn by football players. Note polypropylene helmet and protective neck collar.

trauma may leave an athlete with a developing subdural hematoma, whereas a more violent head trauma may cause loss of consciousness in an athlete, but only result in a minor concussion. The medical team must be able to quickly and accurately differentiate between these types of neurologic injuries and should always be prepared to treat the athlete acutely and appropriately.

This article addresses the mechanism of injury, epidemiology, pathophysiology, evaluation, and on-the-field management of athletic head and neck injuries. Current return-to-play guidelines are outlined and discussed as well. A greater understanding of the athlete with a head or neck injury will advance the care of the athlete today and will establish a foundation for further research and safety for the athlete of tomorrow.

Epidemiology

The epidemiology of neurologic injuries has been studied extensively. Overall, head injuries occur more commonly than spine injuries in athletes. Head injuries occur at an annual rate of ranging from 132 to 367 cases per 100,000 people per year, with sporting activities accounting for approximately 14% of these injuries [6]. It is estimated that 200,000 to 250,000 mild traumatic brain injuries occur annually in high school football [7,8]. Spine injuries are less common, with approximately 10,000 spinal cord injuries occurring yearly in the United States, 10% of which are related to athletic events [9].

Head and neck injuries have been described in almost every sport from football to golf, and have an obvious demographic and geographic distribution

[10–26]. Although football has the highest incidence of head injuries, spine injuries are actually more common in nonorganized sports such as diving, surfing, and skiing [9,27,28]. A Japanese spinal cord injury study revealed a rate of 1.95 injuries per million athletes per year, with the top causes being diving, skiing, football, sky sports, judo, and gymnastics [27]. Because data concerning injuries in recreational sports are more difficult to assimilate and analyze, most of the data available for athletic head and neck injuries are compiled from football and organized sports injuries.

The National Center for Catastrophic Sports Injury Research in Chapel Hill, North Carolina is the organization that compiles data on organized sports injuries. Head and neck injuries in American football have been extensively studied at this center and by many other authors [2–4,10,25,27,29–54]. Eighty-five percent of the annual death rate in football is the result of head and neck injuries, with subdural hematomas as the leading cause of these deaths [5]. It is estimated that 250,000 to 300,000 head injuries occur in the United States in football players each year, with a risk of 15% to 30% per player for each season of play [4,8,34,37,55]. Moreover, individuals who have sustained a concussion have a four-fold relative risk of a repeat head injury [7].

Studies have shown that repeat head injuries may have sustained neurological or psychological implications [3,56–58]. These investigations have highlighted the importance of adequate evaluation of minor head injury and appropriate early intervention to prevent deleterious long-term sequelae [57,59]. A disturbing statistic is that 30% to 70% of American football players who are knocked out during a game return to play that same day [7,37]. Similarly, only 8% of ice hockey players in another study saw a physician after sustaining an athletic brain injury [60]. Clearly, the management of head trauma is inadequate. The long-term sequelae of minor head trauma mandate that these treatment patterns and sport-specific governing guidelines must change in order to protect the athlete.

Proctor and Cantu outlined the sports that are most hazardous to the head and spine [61]. Most of these sports are contact sports or are considered high-energy sports. It should be noted that injuries in these sports have an obvious geographic and demographic distribution. Other authors, with different regional demographics, list sports such as diving and surfing as the most hazardous sports, with an injury prevalence of 69.1% compared with football at 6.1% [6]. Sports with the highest rates of head and neck injuries include [61]:

1. Auto racing
2. Boxing
3. Cycling
4. Diving
5. Equestrian sports
6. Football
7. Gymnastics
8. Hang gliding
9. Ice hockey

10. Lacrosse
11. Martial arts
12. Motorcycling
13. Parachuting
14. Rugby
15. Roller blading
16. Skiing
17. Sky diving
18. Soccer (goalie)
19. Track (pole vaulting)
20. Trampolining
21. Wrestling

The National Center for Catastrophic Sports Injury Research lists football, gymnastics, ice hockey, and wrestling as the sports with the highest risk for head and spine injury [62]. Because many more youths play football than the other three sports combined, the absolute number of head and spine injuries seen in football is the highest.

Pathophysiology and mechanism of athletic head injuries

Head injuries in athletes can be classified by anatomic location (epidural, subdural, intracerebral), mechanism of injury (coup, contrecoup), or clinical presentation. Severe closed head injuries include diffuse axonal injury (DAI), intracerebral hemorrhage, contusion, subdural hematoma, and epidural hematoma. These injuries are relatively rare in athletes, but may have devastating consequences if not recognized early and managed appropriately. Although there may be a lucid interval, severe head injuries usually present with a Glasgow Coma Scale score of less than nine and continue with protracted periods of loss of consciousness or coma [63].

Severe diffuse axonal injury without a mass lesion occurs in almost half of athletes with severe head injury, and accounts for one third of all head-injury related deaths [64]. The pathophysiology of DAI is shearing of multiple axons caused by rotational forces. In severe cases, focal lesions in the corpus callosum occur and are frequently associated with intraventricular hemorrhage. Also, focal lesions occur in the brainstem and pervasive axonal damage can be seen throughout the white matter [63]. These severe injuries are difficult to diagnose with neurodiagnostic imaging.

Intracerebral, epidural and subdural hematomas

Traumatic intracerebral hematoma (Fig. 2) or brain contusion may occur without antecedent neurologic deficit or loss of consciousness. Athletes may

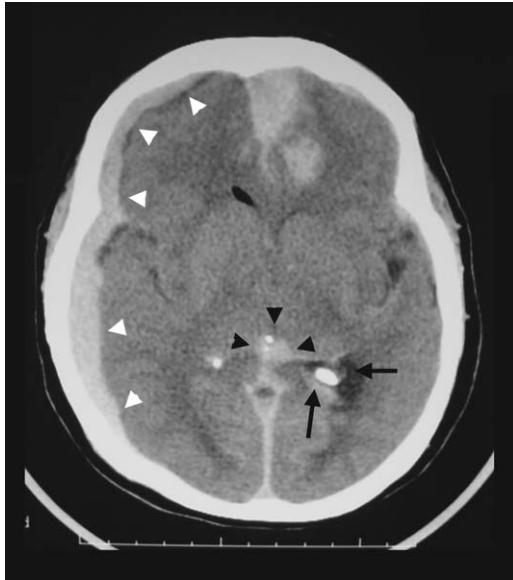


Fig. 2. CT Scan of severe head injury. Note intracerebral hematoma (*black arrowheads*). Note blood in left ventricle (*black arrows*). Subdural hematoma also present (*white arrowheads*).

present with persistent headache or confusion as well as retrograde amnesia. If the athlete's mental status changes do not return to baseline, further investigation and usually computed tomography (CT) imaging is indicated to evaluate for intracerebral pathology. These injuries have the potential of developing a mass effect and require close observation and occasional surgical evacuation.

Epidural hematomas (Fig. 3) usually result when skull fractures tear the middle meningeal artery or vein. Classically, arterial bleeding is thought to cause rapid neurologic symptoms and damage, whereas a venous bleed has a more insidious onset. Athletes sustaining an epidural hematoma may experience a loss of consciousness followed by a lucid period. After this lucid interval, the athlete will have a gradual decline in mental status, with focal neurologic findings such as weakness, ipsilateral dilated pupil, and decerebrate posturing. This triad of symptoms, however, is only present in one third of athletes with epidural hematomas [65].

Subdural hematomas (Fig. 4) occur more frequently than epidural hematomas and are the leading cause of catastrophic death in football players [5]. These occur when the bridging veins between the brain and the dura are torn. These subdural hematomas are different from those seen in the elderly population, in which there is a potential subdural space due to brain atrophy. This potential space is not present in the young athlete, and an athlete with a subdural hematoma

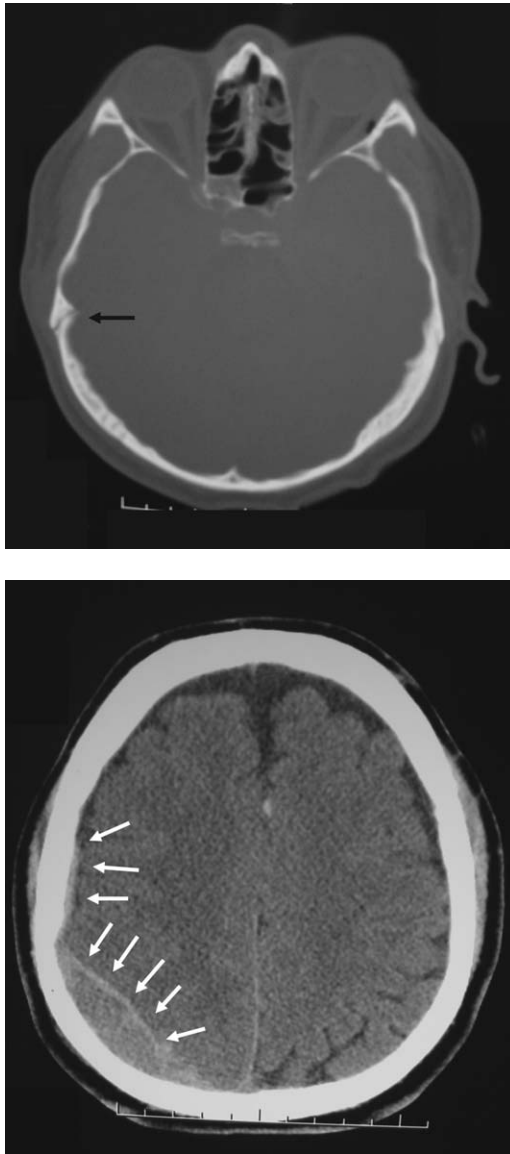


Fig. 3. CT Scan of skull fracture (*black arrow*), with resultant epidural hematoma (*white arrows*).

often has an associated brain contusion and hemispheric swelling. As such, the neurologic findings with a subdural hematoma may correlate more consistently with the associated injuries than with the subdural hematoma itself [65]. The athlete with an acute subdural hematoma is usually unconscious and may have had the aforementioned lucid interval. There are usually focal neurologic



Fig. 4. CT Scan of subdural hematoma (*black arrowheads*). Note mass effect compromising right ventricle.

findings, and these athletes should have emergent evaluation and possible surgical intervention.

Coup and contrecoup injuries

The mechanism for closed head injury is usually a sudden acceleration-deceleration force, or less commonly a force that sharply deforms the skull. The coup lesion is a lesion that results at the site of impact, usually with a resting but moveable head. A contrecoup injury usually occurs when a moving head strikes a relatively stationary object and produces an injury in the area opposite the site of impact. When a skull fracture is present, the displacement of the bone fragments can cause additional injury. Both the coup and contrecoup mechanisms are capable of producing shear forces across the neurons, which may lead to loss of consciousness.

The forces generated by these mechanisms of injury can be divided into compressive, distraction, and shearing. Compressive forces are tolerated fairly well, due to the shock-absorbing contribution of cerebrospinal fluid (CSF). Shearing forces, however, are tolerated poorly. Shearing forces are concentrated where rotational gliding is hindered. These areas of hindered motion are distinguished by sparse CSF between the skull and the brain, irregular surface matching between the brain and skull, or dural attachments to the skull.

Sparse CSF helps explain the mechanism of coup and contrecoup injuries. When an accelerating head strikes a relatively stable object, the layer of CSF is disproportionately thinner at the interface furthest from the point of impact and thickest at the site of impact. Thus, a brain injury occurs opposite the site (contrecoup) of impact. When the head is stationary, the CSF is evenly distributed and therefore more force is seen at the site of initial impact, and a contrecoup injury is usually not seen. Of course, the exact mechanism of injury will dictate the type of injury seen and can include both coup and contrecoup aspects. Irregular surface matching explains why major brain contusions occur most commonly in the frontal and temporal lobes. When a rotational force is applied to the head, the frontal lobes and temporal lobes cannot rotate because of the lack of relative sphericity in the front of the head.

Concussions

Concussions represent the most common type of sports-related head injury, accounting for an estimated 75% of the total number of injuries occurring to the head [55]. A concussion is usually defined as a clinical syndrome characterized by transient post-traumatic impairment of neural function, such as alteration of consciousness, and disturbance of vision or equilibrium, due to brain stem involvement.

The classic clinical hallmarks of concussion are confusion and amnesia. A concussion may also present with inappropriate verbal responses, incoherent speech, emotional lability, lack of focus or eye contact, impaired motor coordination, or inability to concentrate. These deficits may be immediate or have delayed onset. There are no imaging or physiologic measurements that can be used to document the severity of a concussion. The spectrum of symptoms can range from very short and transient periods of neurologic dysfunction, such as confusion, to a severe, coma inducing global brain insult.

Table 1
Classification schemes for concussions

	Mild: grade I	Moderate: grade II	Severe: grade III
Cantu [92]	No loss of consciousness and post-traumatic amnesia < 30min	Loss of consciousness < 5 minutes or post-traumatic amnesia > 30 minutes but < 24 hours	Loss of consciousness > 5 minutes or post-traumatic amnesia > 24 hours
Colorado Consortium [93]	No loss of consciousness; confusion without amnesia	No loss of consciousness; confusion with amnesia	Loss of consciousness
American Academy of Neurology [94]	Transient confusion; no loss of consciousness; symptoms or abnormalities resolve in less than 15 minutes	Transient confusion; no loss of consciousness; symptoms or abnormalities last more than 15 minutes	Any loss of consciousness

Table 2

Cantu's management and return-to-competition guidelines based on severity of injury and history of previous injury

Concussion grade	First concussion ^a	Second concussion	Third concussion
Grade I: Mild	May return when asymptomatic for 1 week	May return in 2 weeks if asymptomatic for entire week prior to return	Terminate season; may return to play next season if asymptomatic
Grade II: Moderate	May return in 2 weeks when asymptomatic for 1 week	May return in 1 month if asymptomatic for 1 week; consider termination of season	Terminate season; may return to play next season if asymptomatic
Grade III: Severe	May return in 1 month if asymptomatic for 1 week	Terminate season; May return to play next season if asymptomatic	Consider no further contact sports

^a CT/MRI recommended if signs or symptoms persist; terminate season if CT/MRI scans reveal any abnormalities

From Cantu RC. Head and spine injuries in the young athlete. Clin Sports Med 1988;7(3):466; with permission.

Multiple classifications have attempted to define cerebral concussions (Table 1). These are based on clinical presentation of the athlete and duration of the symptoms. Cantu's stratified severity scale has been well accepted, and can be used easily to outline initial and long-term management of the athlete (Table 2) [61]. This classification system, however, has come under new criticism by investigators that have showed no correlation between loss of consciousness and future neuropsychologic impairment [66].

Second-impact syndrome

Second-impact syndrome is a well-described condition of massive cerebral edema and impending death in athletes following a second head injury [42,67]. The majority of these athletes have suffered recent head insults and usually have residual symptoms when returning to play [68]. The second blow may be minor and may not even render the player unconscious. During the next 15 seconds to several minutes, the athlete precipitously collapses, becomes semicomatose, loses eye movement, and dilates his pupils rapidly. Respiratory failure may ensue shortly. It is believed that the athlete has vascular sensitivity from the first injury and the subsequent injury eventually leads to vascular hypertension and eventual herniation of the uncus below the tentorium or the cerebellar tonsils through the foramen magnum [69,70]. The usual time frame for this disastrous event is usually from 2 to 5 minutes.

Because this catastrophic condition has a 100% morbidity and 50% mortality, prevention is mandatory [70]. No athlete should be allowed to return to sports

unless he or she has been asymptomatic, both during exertion and at rest, preferably for an entire week.

Pathophysiology and mechanism of cervical spine injury

Because the mechanisms of neck trauma and head trauma are intimately intertwined, one must always be suspicious of both injuries in an athlete with neurologic findings, altered level of consciousness, or change in mental status. Knowledge of normal anatomy and motion of the cervical spine is important in understanding neck injuries. The cervical spine is comprised of seven articulating vertebrae joined by multiple ligaments, joint capsules, and muscles. The normal lordosis of the cervical spine is stabilized by the anterior longitudinal ligament (ALL), posterior longitudinal ligament (PLL), intertransverse, interspinous, supraspinous, capsular ligaments, and the ligamentum flavum.

Eighty percent of cervical spine injuries occur by the accelerating head and body striking a stationary object [71,72]. The discussion of spine and spinal cord injury is usually separated into upper cervical injuries (occiput, atlas, and axis), lower cervical injuries (C3–C7 and T1), stingers, and transient quadriplegia. It is important to consider the unique motion of the spine at each segment, as it helps understanding of the mechanism of injury at that level.

Upper cervical spine

Forty percent of the normal flexion and extension of the spine occurs at the occipitocervical level. Abnormal forces at this level may disrupt the strong ligamentous attachments to the atlanto-occipital joints. Impaction fractures of the occipital condyles can occur, but these are exceedingly rare.

Forty percent of the rotation in the cervical spine occurs at the atlantoaxial joint. The rotation at this joint is restricted by the alar ligaments, which extend from the odontoid process to the inner border of the occipital condyles. The odontoid is attached centrally to the anterior foramen magnum by the apical ligament. The transverse ligament and the lateral joint capsules are responsible for atlantoaxial joint strength.

The most common mechanism of injury at the atlantoaxial joint is flexion, which may fracture the odontoid or may tear the transverse ligament and result in a rare atlantoaxial dislocation. Extension injuries do occur and may result in odontoid fractures, but these are rare as well. Rotation forces may produce a rare unilateral rotary dislocation. Direct axial loading may produce a centrifugal force against the lateral masses of the atlas, which fractures through its thinner parts of the ring posteriorly and anteriorly. Spreading of lateral masses of greater than 7 mm on an anteroposterior radiograph indicates tearing of the transverse ligament. Extension of the occiput on the cervical spine may cause rare bilateral pedicle fractures of the axis. Only a small percentage of injuries at the atlantoaxial level produce neurologic deficit because of the anatomically wide spinal canal at these levels.

Lower cervical spine

The spinal canal is less spacious from the third to the seventh cervical vertebrae. The facet joints are oriented at a 45° angle, thereby limiting axial rotation, but allowing cervical flexion and extension. The motions of the spine are coupled such that axial rotation is associated with lateral bending.

Acute cervical spine injury may produce instability if the bones or ligamentous structures are stressed beyond physiologic loads [73]. Instability of the lower cervical spine has been defined as translatory displacement of two adjacent vertebrae greater than 3.5 mm or angulation of greater than 11° between adjacent vertebrae [74].

Fractures may produce instability, whether by compression of the vertebral body anteriorly or by disruption of the posterior elements. Mechanisms of injury to the lower cervical spine are defined by the forces acting upon the spine. These forces include flexion, extension, lateral rotation and axial loading. Flexion injuries may result in jumped facets and are usually associated with either distraction or rotation. Torg identified the C3-C4 level as the most common dislocation in football injuries resulting in quadriplegia [75,76]. Extension or whiplash injuries usually disrupt the anterior structures and compress the posterior structures. The anterior longitudinal ligament may rupture and fractures may occur in the posterior elements.

Compression injuries, such as those seen with spear-tackler's spine, usually lead to vertebral body fractures. This is most likely due to the flexed posture of the head and loss of protective cervical lordosis during these injuries (Fig. 5).

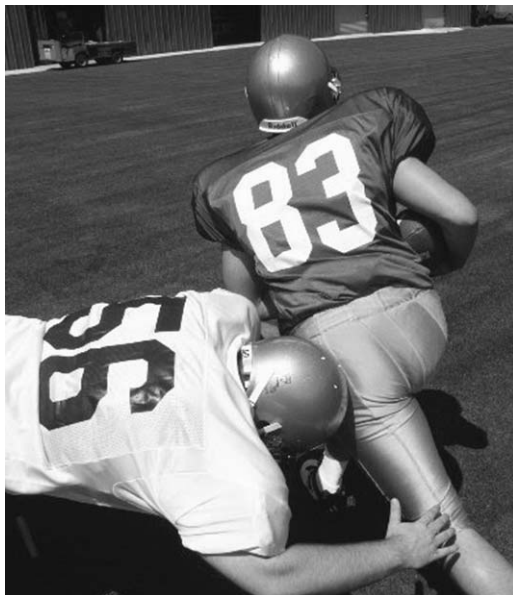


Fig. 5. Spear tackle. Notice flexed posture of cervical spine and loss of protective lordosis.

With large compressive forces, fractures may occur and the bony fragments or disc material may protrude into the spinal canal. This type of axial loading is the major mechanism of cervical fracture, dislocation, and quadraplegia, not only in football, but in diving and ice hockey as well [4,77].

Stingers

Stingers or burners are defined as unilateral radiating pain, burning, and parasthesias in an upper extremity with possible associated local weakness. The symptoms are transient and usually last for only seconds or minutes.

Stingers occur by one of two mechanisms: (1) traction on the brachial plexus, or (2) nerve-root impingement within the cervical neural foramen. The most common mechanism for a brachial plexus injury is a forceful blow to the side of the head. This increases the angle of the exiting brachial plexus cords in relation to the nerve roots and thereby causes injury. On the other hand, nerve root impingement usually occurs when the athlete's head is driven towards his shoulder pad, thereby causing a compression of the neural foramen and the resident dorsal root ganglion.

The clinical presentation of both types of stingers is identical. The symptoms are usually sensory alone and most commonly involve the C5 and C6 dermatomes. If weakness is present, it usually involves muscles of this neurotome as well. If stingers are not associated with any neck pain, limitation of neck movement, or residual neurologic symptoms in the arm, the athlete may safely return to competition. This is especially true if the athlete has had previous similar symptoms without sequelae. Persistent pain or residual arm symptoms should prompt further work-up with magnetic resonance imaging (MRI). If symptoms persist for more than 2 weeks, electromyography should be considered to assess the distribution and degree of the injury.

Athletes who experience two or more stingers, particularly in rapid succession, should be placed in a protective device to limit lateral neck flexion and hyperextension (see Fig. 1). Alternatively, modifying playing techniques or changing field position may decrease the risk of repeat injury. If stingers continue, the athlete may have to be suspended permanently.

Transient quadriplegia

Transient quadriplegia is characterized by a temporary loss of motor or sensory function, or both. Current hypotheses attribute the temporary spinal cord dysfunction to a contusion or ischemia. Although both hyperflexion and hyperextension injuries have been suggested as the cause of this syndrome, the classic mechanism is generally thought to involve a pincer injury to the spinal cord due to a relative decrease in spinal canal diameter and in-folding of the ligamentum flavum during hyperextension.

Torg and coworkers reported neurapraxia of the cervical spinal cord with transient quadraplegia in 1.3 percent of 10,000 athletes with a suggestive history

[47,78]. He also expanded on the previous work of Alexander and identified developmental spinal stenosis in athletes with these neurologic injuries [51,79]. From these studies, a ratio of spinal canal width to vertebral body diameter of 0.80 was suggested to indicate stenosis. Although a low ratio is found in most athletes sustaining transient neurapraxias of the cord, the positive predictive value is too low to be used as a screening tool in athletes [32,38,80]. This ratio has since been shown to be an unreliable prognostic index. Bilateral neurologic symptoms after a contact injury should alert the clinician to possible spinal cord injury. The diagnosis should be confirmed not only with plain radiography, but also with MR imaging or CT myelogram, which determines the functional reserve or space surrounding the cord. These advanced imaging modalities provide additional clues regarding disc protrusions, osteophytes, or other less common abnormalities.

There are no clear guidelines as to whether an athlete with documented cervical spinal stenosis is at increased risk for spinal cord injury. Proctor and Cantu state that those who have had spinal cord problems from sports-related injuries and are shown to have functional spinal stenosis on MR imaging or CT myelogram should not be allowed to return to contact sports [61]. This assertion is supported by other studies that propose spinal stenosis as a predisposing factor to spinal cord injury [79,81–83].

Moreover, there are cases of permanent quadriplegia in athletes with cervical canal stenosis without evidence of fracture or overt instability. Therefore, functional spinal stenosis may be a contraindication to further participation in contact sports [32,38,80]. Recommendations for return to sport must be individualized based on symptoms, degree of canal stenosis, and risk of further neurologic injury.

On-field evaluation and management

The initial assessment of a head or neck injury has inherent challenges. Athletes with fatal head or neck injuries or those with immediate neurologic consequences are easy to identify. The challenge lies in identifying those athletes with a mild head injury with no immediate overt signs or symptoms. It is essential that all individuals involved in the treatment of athletes understand the diagnosis, prognosis, and management of the aforementioned head and neck injuries and are adequately prepared to handle such injuries. The mechanism of injury and the energy of the injury are essential in the evaluation of the head or neck injured athlete and must be considered in their ensuing diagnosis.

In 1998, the multidisciplinary Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete was formed to develop guidelines for the appropriate management of the catastrophically injured athlete [84]. This collaboration outlined specific issues for the care the spine-injured athlete based on current research and data, and should be a required resource for any individual involved in the care of athletes.

On-field management of the head- or neck-injured athlete can be distilled into five categories: (1) preparation for any neurologic injury; (2) suspicion and recognition; (3) stabilization and safety; (4) immediate treatment and possible secondary treatment; and (5) evaluation for return to play.

All personnel involved in the care of an athlete should be prepared to care for any of the previously described head or neck injuries. Optimal preparation includes education of all members involved in care of the athlete and availability of all necessary emergency equipment at the site of the potential injury, including a spine board, cervical collar, and cardiopulmonary resuscitation equipment. Specific equipment to remove protective gear quickly and safely should also be readily available. An emergency plan should be outlined during practice and competition and should include ambulance availability for all contact and high-energy sports.

Injuries as a result of acceleration-deceleration impact to the head should immediately raise suspicion of a head or neck injury. The athlete should immediately be assessed for level of consciousness while still on the field if a head or neck injury is suspected. The Glasgow Coma Scale has been used extensively and provides a prognostic indicator for recovery (Table 3) [85]. A score of 11 or higher on this scale is associated with an excellent prognosis for recovery, whereas a score of 7 or less is considered serious with a less favorable prognosis.

Once the suspicion of a head or neck injury has been entertained or proven to be correct, it is imperative to stabilize the athlete to ensure that no further injury will occur. Any athlete with a head injury should be assumed to have a cervical injury as well. The face mask should be removed quickly and safely. Neither the helmet nor shoulder pads should be removed in an athlete with a suspected cervical injury (Fig. 6) [52]. Proper precautions, such as a hard cervical collar and

Table 3
Glasgow Coma Scale

Assessment area	Score
Eye opening (E)	
Spontaneous	4
To speech	3
To pain	2
None	1
Best motor response (M)	
Obeys commands	6
Localizes pain	5
Normal flexion (withdrawal)	4
Abnormal flexion (decorticate)	3
Extension (decerebrate)	2
None (flaccid)	1
Verbal response (V)	
Oriented	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
None	1

a spine board, should always be used to prevent further injury to the player until a cervical injury can definitely be excluded.

The Inter-Association Task Force for Appropriate Care of the Spine-Injured Athlete has outlined four areas where the athletic helmet and chin strap should be removed: (1) if the helmet and chin strap do not hold the head securely, such that immobilization of the helmet does not also immobilize the head; (2) if the design of the helmet and chin strap are such that, even after removal of the face mask, the airway cannot be controlled nor ventilation provided; (3) if the face mask cannot be removed after a reasonable period of time; and (4) if the helmet prevents immobilization for transportation in an appropriate position [84]. Spine immobilization must be maintained while removing the helmet.

If a cervical injury has been excluded, but a head injury is still suspected, the athlete may be assisted into a sitting position. This upright posture should decrease the intracranial pressure and ameliorate some of the neurologic symptoms. If stable in a seated position, the athlete may be helped to stand and then helped off the field for further evaluation and observation.

Further evaluation on the bench is of paramount importance, and the athlete should be observed according to the type of injury sustained. If it is determined that a player is stable and safe, he should undergo a complete neurologic evaluation on the sidelines. If the athlete is determined to be unstable or at risk for further deterioration, he should be transported emergently to a hospital.

Unconscious athletes must be stabilized before any neurological evaluation is performed. This includes advanced cardiac life support guidelines of securing and protecting an airway, and providing cardiopulmonary resuscitation if the patient is not breathing independently or does not have an adequate pulse. If the athlete is



Fig. 6. Spine stabilization of an injured football player. Note that the face mask has been removed for airway access and the neck has been immobilized with tape to prevent further head or neck injury. The helmet and shoulder pads are left in place.

in this compromised state, the face mask should be removed immediately for ventilation, and the front of the shoulder pads should be opened to provide chest compressions and possible defibrillation [84]. Respiratory compromise may require emergency on-field intubation. Blood pressure should be monitored continuously, and hyperventilation of the patient is recommended if focal neurologic findings develop. Initial on-field treatment of grade II and III concussions should include safe placement of the patient on a fracture board, with the head and neck immobilized and the face mask removed where appropriate. Once the unconscious athlete is stabilized and secure, emergency transport to a hospital for evaluation is indicated. Again, the face mask should always be removed during transportation.

Guidelines for return to sport

Severe head injuries

Athletes with severe head injuries will rarely return to sports. Although severe diffuse axonal injuries usually do not require surgical intervention, the sequelae can be neurologically devastating, and if the athlete survives, he will need long-term care and rehabilitation. Epidural hematomas, significant subdural hematomas, and large intracranial hematomas are usually managed surgically. It is generally accepted that any athlete with a head injury requiring surgical management should not be allowed to return to competition [86]. Other severe head injuries should be managed on an individual basis.

Concussions

When to allow athletes to return to sports after a concussion is a more difficult question to answer. Because of second-impact syndrome, no player should be allowed to return to play until all neurologic symptoms have resolved, both at rest and with exertion at the level of normal competition [33]. All athletes with concussions should have an evaluation by a physician and be educated regarding their injury and their prognosis. Although there is no uniform agreement regarding return-to-play guidelines, Cantu has provided a general outline for management (see Table 2) [34].

Athletes with grade I (mild) concussions require initial acute observation for 20 to 30 minutes by a health care provider. If there is no deterioration of neurologic function and the athlete is completely asymptomatic, both at rest and with exertion, the athlete may return to play [33]. Cantu, however, suggests that an athlete should be suspended from competition until he or she is asymptomatic for at least 1 week following a concussion [34]. A subsequent concussion in the same season should sideline the athlete for at least 2 weeks. Evaluation for repeat concussions should include neurodiagnostic imaging to evaluate for intracranial pathology. If the imaging studies are normal and the athlete remains asymptomatic,

matic for 2 weeks, he or she may return to competition. A third concussion should mark the end of that season for the athlete [33,87].

Grade II (moderate) concussions should sideline the athlete until he is asymptomatic for at least 1 week. A subsequent concussion should prompt suspension of the player for 1 month and evaluation by a physician with diagnostic imaging. A third moderate concussion should mark the end of the season and may mark the termination of participation in the sport.

Grade III (severe) concussions should sideline the athlete for a month. A second severe concussion should mark the end of the season and probably the end of a career. Multiple head injuries can lead to significant long-term sequelae and should be avoided.

The waiting period before return to sports has been challenged by neuropsychiatric tests and tests of balance and coordination, in an attempt to return the athlete to the sport sooner [58]. These tests may be useful in the very high-level athlete, but for the majority of athletes these measures are not useful.

Cervical spine injuries

Return-to-play recommendations after a cervical spine injury should be delayed until the athlete has no neurologic symptoms, and has regained all of his preinjury strength, especially in his neck. Also, lateral cervical spine radiographs should show normal lordotic curvature, indicating resolution of cervical muscle spasm and malalignment. Functional spinal stenosis should be evaluated and recommendations should be individualized for each athlete.

Many authors have outline return-to-football criteria for athletes with cervical spine injuries [31,45,47,80,84,88–90]. Additionally, Morganti attempted to define return-to-play criteria after a cervical spine injury for all sports [91]. This study subclassified types of play and attempted to use this classification as a guide to acceptable postinjury sports activity. Unfortunately, there is little consensus among physicians regarding return to play and what level of play is acceptable [91].

In an attempt to help with return-to-play criteria, Maroon and Bailes developed a classification of cervical spine injuries in athletes [9]. They defined type I injuries as permanent cord injuries and excluded these athletes from further play. Athletes with type II injuries had transient injuries with abnormal radiographs and were returned to play over time. Type III injuries showed radiographic abnormality without neurologic deficit and return to play was individualized based on the abnormality. The return-to-play criteria for stingers and transient quadriplegia are outlined in their respective sections.

The wide range of recommendations regarding return to play after a cervical injury confuses both the athlete and health care providers. Absolute contraindications to return to contact or collision sports include neck injuries resulting in permanent central nervous system dysfunction, permanent and significant peripheral nerve root dysfunction, and injuries resulting in spinal fusion above the C5 level [84]. Conditions such as spinal stenosis represent relative contraindications for return to play [81,84].

Summary

Head and cervical spine sports-related injuries are intimately associated. The on-field evaluation and management of the athlete with these injuries is of paramount importance to stabilize the athlete and prevent further injury. Clinicians need to be aware of the differential diagnoses and consider each possibility based on the mechanism of injury. Although recognition of head and cervical spine injuries has resulted in significant reductions of catastrophic neurological injuries, especially in the cervical spine, further advances to decrease the incidence and long-term sequelae of head and neck injuries are needed. The first step is education of the athlete and the individuals involved in the care of that athlete.

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