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literature review

Management of Cervical Spine Injuries in Athletes

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Objective: Although the incidence of catastrophic cervical spine injury in sport has been significantly reduced over the past 3 decades, the injury warrants continued attention because of the altered quality of life that often accompanies such an injury. The purpose of our literature review was to provide athletic trainers with an understanding of the mechanisms, anatomical structures, and complications often associated with sport-related cervical spine injury. We also present the most current recommendations for management and treatment of these potentially catastrophic injuries.

Data Sources: A review of the most pertinent literature between 1970 and 2005 was conducted using MEDLINE and the search terms *spinal cord injury, cervical spine injury, neurosurgical trauma, cervical spinal stenosis,* and *catastrophic spine injury.*

Data Synthesis: Flexion of the head places the cervical spine into a straight line and prevents the neck musculature from assisting in force absorption. This mechanism is the primary cause of cervical fracture, dislocation, and quadriplegia. The most serious of the syndromes described in the literature involves a complete spinal cord injury with transverse myelopathy. This injury typically results in total loss of spinal function below the level of the lesion.

Conclusions/Recommendations: Spinal trauma may result in a variety of clinical syndromes, according to the type and severity of the impact and bony displacement, as well as subsequent secondary insults such as hemorrhage, ischemia, and edema. Athletic trainers should be prepared to promptly recognize these potentially catastrophic injuries and follow the recommendations of the Inter-Association Task Force for the Appropriate Care of the Spine Injured Athlete in managing such injuries.

Key Words: neck injury, spinal stenosis, quadriplegia, catastrophic injuries

Injuries to the cervical spine constitute uncommon but nonetheless devastating occurrences to those participating in athletic events. These injuries happen primarily to athletes involved in the contact sports of football, wrestling, and ice hockey, with football injuries constituting the largest number of cases. The important work of Schneider[1] in the mid 1960s, focusing on football-related head and neck injuries, resulted in a significant reduction in the incidence of these accidents owing to improvements in equipment, education in proper techniques, offseason conditioning, and rule changes. Reports of the frequency of serious neck injuries in football players vary, from 1 quadriplegic injury in 7000 participants to 1 in 58 000.[2,3]

Approximately 40 sport-related cases of vertebral column and 7 of spinal cord injury were reported annually in the United States from 1977 to 2004.[4,5] A disturbing trend seen in the late 1970s was a drop in the number of serious head injuries in football players but a simultaneous increase in the incidence of cervical injuries, including permanent quadriplegia. From 1971 to 1975, 259 cervical spine injuries occurred in football players. This correlates to 4.1 per 100 000 players, and, of those, 99 cases (1.58 per 100 000 players) resulted in permanent quadriplegia.[6] Mueller[7] noted an increase in cervical spine fatalities to 42 during the 1965–1974 decade. This was believed to be secondary to improvements in helmet design, which, despite providing protection against head injury, encouraged the device's use as a battering weapon. Torg et al[8] indicated that 85% of all cervical spine injuries from 1971 to 1975 were due to axial loading. After the no-spearing rule changes in high school and collegiate football were implemented in 1976, players were forced to change their tackling techniques, and the incidence of cervical spine injury started to decrease. The decline was dramatic, considering that between 1965 and 1974, 40 fatalities occurred as a result of spinal cord injury, 26 as a result of tackling. Between 1975 and 1984, 14 fatalities occurred from spinal cord injury, 10 as a result of tackling. Between 1985 and 1994, only 5 such fatalities occurred, all as a result of tackling.[7]

The purpose of this literature review was to provide athletic trainers with an understanding of the mechanisms, anatomical structures, and complications often associated with sport-related cervical spine injury. Additionally, we present the most current recommendations for management and treatment of these potentially catastrophic injuries to the spine.

CAUSES AND MECHANISMS OF SPINAL CORD INJURY

The mechanism of spinal cord injury can be sport related, but it is more commonly independent of the sport. The major mechanism of serious cervical injury is an axial load, or a large compressive force applied to the top of the head. This mechanism is more dangerous when the neck is slightly flexed, because the spine is brought out of its normal lordotic alignment, which does not allow for proper distribution of force to the thorax. Flexion puts the cervical spine in a straight line; thus, the musculature cannot assist in absorbing the force. Torg et al[8] indicated that injury occurs to the cervical spine when it is compressed between the body and the rapidly decelerating head. When a fracture results, if the bone fragments or herniated disc materials encroach on the spinal cord, neurologic damage will occur. This mechanism is the primary cause of cervical fracture, dislocation, and quadriplegia.

SYNDROMES OF SPINAL CORD INJURY

Spinal trauma may result in a variety of clinical syndromes, according to the type and severity of the impact and bony displacement as well as the subsequent secondary insults such as hemorrhage, ischemia, and edema. Complete spinal cord injury is a transverse myelopathy with total loss of spinal function below the level of the lesion. This insult is caused by either anatomic disruption of the spinal cord or, more commonly, hemorrhagic or ischemic damage at the site of injury. Although the spinal cord usually remains in continuity, a physiologic block to impulse transmission results in the complete injury. Complete injury patterns are rarely reversible, although with long-term follow-up, improvement of 1 spinal level may be seen when the initial segmental traumatic spinal cord swelling resolves.

Several patterns of incomplete spinal cord injury are common, usually produced on a vascular basis. The *central cord syndrome* causes incomplete loss of motor function with a disproportionate weakness of the upper extremities as compared with the lower extremities. This condition is thought to be the result of hemorrhagic and ischemic injury to the corticospinal tracts because of their somatotopic arrangement. Fibers of cervical nerves that innervate the upper extremities are arranged more medially than those subserving function to the lower extremities. The originally described central cord syndrome also includes nonspecific sensory loss and bladder and sexual dysfunction. This injury pattern is also often seen in older patients with vertebral osteophytes and in those with hyperextension injuries; in the latter, in the absence of fracture, a hyperextension mechanism causes an infolding of the ligamentum flavum with transient compression of the spinal cord, its blood supply, or both. Due to pre-existing degenerative narrowing within the spinal canal, even a relatively minor fall may produce a neurologic injury in these patients. The central cord syndrome describes the site of spinal cord injury, but clinical expression is wide ranging and overlaps with other incomplete spinal injury syndromes, especially the *Brown-Sequard syndrome.* Merriam et al[9] found in a younger group of patients (mean age of 34.6 years, with half of the patients being less than 30 years old) with central cord syndrome fewer long tract findings, motor deficits limited primarily to the upper limbs, and a good recovery. Overall, a good prognosis exists for some degree of recovery and often for total functional recovery.

The *anterior spinal cord syndrome* describes an injury that occurs to the anterior two thirds of the spinal cord in the region supplied by the anterior spinal artery. The neurologic deficit ordinarily consists of a complete loss of all motor function below the level of injury, in addition to loss of sensation conveyed by the spinothalamic tracts (pain and temperature). Compared with the disproportionate motor deficit seen in central cord syndrome, deficits in the upper and lower extremities are usually equal, along with sphincteric and sexual dysfunction. Although the precise mechanism of the pathologic process is not known, the final common insult is ischemia in the distribution of the anterior spinal artery, which is seen in a variety of spinal column injuries (unlike the strong association of hyperextension injury with central cord syndrome). Although the posterior funiculus of the spinal cord and the dorsal column function are relatively preserved, they are of little importance in determining functional outcome due to permanent motor function loss.

The *Brown-Sequard syndrome* has been classically described as hemisection of the spinal cord with loss of ipsilateral motor function and contralateral spinothalamic (pain and temperature) modalities. This latter finding occurs because of the decussation of the spinothalamic fibers 1 or 2 spinal levels above their entry into the cord, whereas the corticospinal tracts have previously crossed higher in the medullary pyramids and maintain their ipsilateral course to spinal levels of innervation of anterior horn cells. Although theoretically sound on an anatomic basis and occasionally seen as a result of penetrating injuries, the Brown-Sequard syndrome usually occurs not in an isolated form but as a combination with other types of incomplete injury. Often a mixture of central cord and Brown-Sequard syndromes is found, in which the patient has some degree of unilateral motor loss and contralateral sensory deficit but with a relatively greater degree of weakness in the upper extremities. The *posterior spinal cord syndrome* is an often mentioned but seldom seen clinical entity in which dorsal column function is lost but the corticospinal and spinothalamic tract functions are preserved, believed to be due to selective ischemia in the distribution of the posterior spinal artery.

Many patients have incomplete injuries that are not classifiable into any certain pattern. These injuries usually consist of loss of all or nearly all useful motor function below the level of injury, with a sensory loss that does not fit any specific pattern. This sensory preservation does, however, predict a better recovery than does complete functional loss. The *burning hands syndrome* is characterized by burning dysesthesias and paresthesias in both hands, commonly seen in athletes who participate in contact sports, especially football and wrestling, with repeated cervical trauma.[10] Burning hands syndrome was proposed to be a variant of central cord syndrome with selective injury to the central fibers of the spinothalamic tract that subserve pain and temperature sensation to the upper limbs. Because this injury did not result in permanent loss of either function or pain, it probably occurred as a result of edema or vascular insufficiency. Burning hands syndrome has been known to occur with both fractures and dislocations of the cervical spine and in patients without demonstrable radiographic abnormality.

In addition to persons with syndromes caused by blunt trauma directed to the spinal column and underlying neural structures, a small group of patients is at risk for neurologic injury resulting from *vascular trauma.* The carotid and vertebral arteries are at risk from direct compression or as a result of traumatic fracture-subluxation. However, a patient with a vascular injury may radiographically show only chronic degenerative changes or have a normal spine. An injury to any of these large arteries may cause a false channel to appear, with blood coursing distally within the vessel wall and causing a dissection, occlusion, thrombus, embolism, or pseudoaneurysm. The carotid arteries are rarely injured in athletic competition, but the clinician must keep them in mind whenever signs or symptoms suggest cerebral hemispheric dysfunction (hemiparesis, hemiplegia, hemianesthesia, dysphasia, homonymous visual field defects). A delay in the appearance of the neurologic defect, even up to several days, may occur. Transient ischemic attacks (TIAs) in the anterior or middle cerebral arteries may occur secondary to distal embolization of thrombotic material forming at a site of an intimal tear in the vessel.

Vertebral artery injury may be seen with a fracture or fracture-dislocation at or above the C6 vertebra (Figure 1). This may result from direct compression by bony elements, by

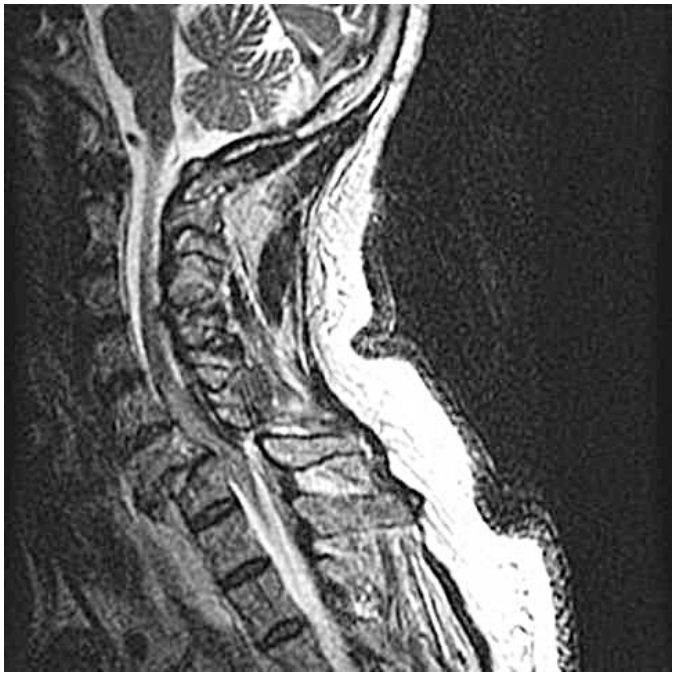

Figure 1. C6-C7 fracture-dislocation with cord edema, typical of complete quadriplegic injuries.

stretching of the artery by vertical movements, or by an expanding traumatic hematoma within the foramen transversarium. Any insult that compromises the structural component of the vessel wall, the tunica intima, or the bony foramen transversarium may lead to potentiation of thrombosis embolization or vasospasm, with resultant ascending thrombotic occlusion and hindbrain ischemia. Such an injury to the vertebral artery could be symptomatic immediately after the traumatic insult, with the developing neurologic deficit ranging from gradual and mild to sudden and severe. The clinical manifestation may be any of a variety of cerebellar or brainstem syndromes. The signs of vertebrobasilar insufficiency or infarction include dysarthria, emesis, ataxia, visual field deficit, cortical blindness, vertigo, diplopia, long tract deficits, and others. Complete brainstem infarction is rare but can occur. Computed tomography (CT) scanning is less likely to show an abnormality in hindbrain ischemic injury than in anterior circulation ischemia, but in either case, if a vascular injury is suspected, emergent angiography (catheter, magnetic resonance [MR] angiography, or CT angiography) must be performed to make the diagnosis.

IMMEDIATE EVALUATION

Prompt recognition of any potentially catastrophic injuries is paramount. Proper management ensures that excessive movement does not exacerbate any initial damage to the spine, thereby reducing the chance of a secondary injury. Initial assessment of injury to the cervical spine can be challenging if obvious signs and symptoms are not present. For assessment on the field, it is important to first conduct a primary survey checking for unconsciousness, airway, breathing, and circulation to identify any life-threatening injuries. If no immediate life-threatening conditions are present, then the level of consciousness of the individual should be determined and a neurologic screening conducted. The evaluation of conscious patients begins with questioning about extremity numbness, painful dysesthesias or paresthesias, weakness, and neck pain. A limited examination can identify obvious neurologic deficit if the patient is unable to move all or any limbs or has gross weakness, numbness, or significant pain to palpation of the cervical region. If any of these are present on history or examination, or if the athlete is unconscious, transportation should be performed carefully. In injured athletes with an altered level of consciousness, the initial evaluation should check for the possibility of associated head trauma. On-field evaluation should include an examination of level of consciousness, cognitive and memory processes, and cranial nerve function, with awareness that significant brain injury is possible.

Every unconscious athlete or injured athlete who complains of numbness, weakness, paralysis, or neck pain should be treated as if he or she has a cervical fracture and, thus, an unstable spine and should be stabilized and transported for further testing and diagnosis.

SPINE BOARDING AND TRANSPORTATION

The athlete should be properly secured to a rigid spine board in such a way that the cervical spine is immobilized and the airway is accessible.[11] Several groups have attempted to determine the optimal position for the cervical spine to allow the maximum amount of space for the spinal cord. DeLorenzo et al[12] determined optimal position of the cervical spine using MR imaging (MRI) and examining the cross-sectional area of the spinal canal versus the spinal cord. Maximum area was consistently obtained with slight flexion, corresponding to raising the occiput 2 cm. However, these authors did not investigate equipment considerations, and their finding is disputed by Tierney et al,[13] who indicated that the most space was present when the occiput was at zero elevation. Additionally, Tierney et al[13] tested a football helmet and shoulder pad condition and found no difference in spinal cord space between the zero elevation with or without the equipment. This result is consistent with many other studies and, therefore, leads us to the conclusion that leaving the shoulder pads and helmet on football players is the best plan.

For ice hockey equipment, LaPrade et al[14] determined that leaving the helmet and shoulder pads on an ice hockey player was the best choice for maintaining neutral spinal alignment. They found that, with the helmet removed but the shoulder pads remaining, the cervical spine was in a significant amount of lordosis. Therefore, for ice hockey players, the recommendation is to leave the helmet on. This advice is also consistent with the work of Metz et al,[15] who determined that the greatest amount of angular displacement occurred when the head rested on the backboard with the shoulder pads still in place.

In addition, helmet removal may create movement that is risky to the integrity of the spinal cord.[16,17] Thus, the Inter-Association Task Force for the Appropriate Care of the Spine-Injured Athlete recommended keeping the helmet and shoulder pads on while immobilizing the cervical spine.[11]

Equipment Consideration and Airway Access

Before transport, the airway needs to be made accessible in case of respiratory distress. This is not an issue for athletes who are not wearing a helmet, but for those who are it can be difficult to reach the airway. Some controversy exists over the best technique for doing this. The possible tools depend on

the style and type of helmet. All sports, including football, ice hockey, and lacrosse, have different styles and different brands of helmets with face masks. Unfortunately, these differences as well as the potential condition of the helmet have led to difficulty in making a recommendation about the best technique for removal. The most widely used tools include various types of cutting tools, such as the FM Extractor (Sports Medicine Concepts, Inc, Geneseo, NY) and the Trainer's Angel (Trainer's Angel, Riverside, CA), as well as a standard screwdriver. The obvious goal is to select a tool that that will allow removal of the face mask as quickly as possible with the least amount of head movement; therefore, it is best to use a screwdriver and have a backup cutting tool in case the screw cannot be removed.[18] Thus, it is important for the athletic trainer to assess the equipment and develop a plan before he or she has to perform the skill on the field.

Transfer to the Spine Board

Once the face mask has been removed and the head is still being stabilized by the initial person on the scene, the athlete needs to be transferred to a rigid spine board. This transfer can either be accomplished using a log roll or a 6-person lift (lift-and-slide) technique. When the athlete is supine, the 6-person lift is recommended, using a scoop stretcher placed under the athlete, who is lifted about 4 to 6 in (10.16 to 15.24 cm) while the spine board is slid underneath.[11] The athlete is then lowered down onto the spine board and strapped to it. At least 3 straps should be used to secure the torso, pelvis, and legs. Mazolewski and Manix[19] showed that an added strap across the pelvis significantly reduced the lateral motion of the torso. The head should be secured with towels, blankets, or commercial head immobilizers and then secured to the board with tape. The additional consideration is whether or not to use a cervical collar. The current recommendation is that a cervical collar should be put on the athlete if that can be done with the pads and helmet still in place. The best combination for head immobilization is a rigid cervical collar and supportive blocks on either side of the head, with adhesive tape across the forehead.[20] Given the circumstances and the equipment, however, it may be impossible to properly fit a cervical collar. Once the athlete is secured to the spine board and strapped down (body first, followed by the head), transport of the athlete can begin.

Equipment and Complications

Although the general recommendation is to leave the equipment on, removing it is acceptable in certain situations[11]:

1. If, after a reasonable time, the face mask cannot be removed to gain access to the airway.
2. If the design of the helmet and chin strap is such that even after removal of the face mask, the airway cannot be controlled or ventilation provided.
3. If the helmet and chin straps do not hold the head securely such that immobilization of the helmet does not also immobilize the head.
4. If the helmet prevents immobilization for transport in an appropriate position.

Although these circumstances may be difficult to determine while trying to manage the injury and may not be likely to occur in football, they may occur in other sports and can pose an interesting dilemma for the athletic trainer. Improved technology of protective equipment has resulted in an overall reduction in catastrophic injuries, but it potentially has made properly securing an athlete to a spine board more difficult. If the athlete's helmet must be removed, the shoulder pads and helmet should be removed as a unit. This can be safely accomplished by cutting the chin strap, removing the ear pads, and pulling outward on the helmet, which allows the helmet to be removed without flexing or extending the neck. The straps that attach the shoulder pads can also be cut along with the jersey to allow for removal around the head after the helmet.[11]

OFF-FIELD MANAGEMENT

If the athlete was not placed on a spine board, a detailed inquiry into any other potential nervous system dysfunction should take place at the sideline or athletic training facility. If no suspicion of a more serious spinal injury exists, the patient should still be questioned in greater detail about the presence of any neurologic symptoms. However, any persisting numbness or burning dysesthesias or paresthesias should alert one to look more closely for evidence of spinal cord injury. Certainly the presence of motor symptoms should alert the examiner to possible spinal injury. If an obvious deficit in movement, significant sensory disturbance, or neck pain occurs at any time, the athlete should be immobilized and cervical spine radiographs immediately obtained. Any athlete suspected of having neurologic involvement or vertebral column injury by history or physical examination should also have a complete radiologic evaluation.

Emergency Department/Hospital Treatment

The treatment begins with removal of the athlete from further participation until the exact nature and injury risk is known. After transport to a definitive care hospital with adequate facilities for diagnosis and treatment of such problems, cervical traction with a device such as Gardner-Wells tongs or a halo device may be required for adequate bony reduction and maintenance of alignment in the anatomically neutral position. This must be done in conjunction with constant radiographic assessment and repeated examinations of the patient. At this time, methylprednisolone is administered if it had not been previously administered by the emergency medical services personnel. The clinicians must be aware that respiratory and cardiovascular alterations can occur after spinal cord injury and must be prepared to treat them effectively as they arise. An intensive care setting is usually the optimal place to care for such injuries in the acute phase. Patients must also be assessed for associated lesions such as head injury, which may have an effect on the clinical course and outcome.

Once the athlete has been initially evaluated and placed in cervical traction to reduce the fracture, a decision must be made as to whether surgical fusion or external orthosis will be used for spinal stabilization. The current trend in most cases of neck injuries is to attempt nonsurgical fusion by applying a halo vest cervical orthosis. For most patients treated in this manner, adequate bony healing occurs within 12 weeks of halo immobilization. Surgical treatment continues to be preferred for severe comminuted fractures of the vertebral body, fractures of the posterior elements with extreme instability, type II odontoid fractures, and incomplete spinal cord injuries with

compromise of the spinal cord as documented on diagnostic studies and in patients who have neurologic deterioration by loss of higher spinal levels of function.

Patients with minor fractures that are stable radiographically as documented by flexion-extension films and who do not have spinal cord injury are allowed to return to their normal daily activities. Athletes with brachial plexus trauma or burning hands may be considered healed when their symptoms resolve and they have no neurologic deficits on examination. The question of whether to allow an athlete to return to contact sports after a documented or suspected spinal injury has always been an issue. Any athlete who suffers a neurologic injury to the spinal cord ordinarily should not be allowed to return. Also, athletes who have had fractures and dislocations of the spine that have required halo brace or surgical stabilization probably are best considered not to have adequate strength to withstand subsequent contact sports. In addition, one must consider the loss of normal movement of the spinal motion segments above and below the area of injury and fusion. Some spinal fractures, however, are inherently stable, and when they have occurred without neurologic injury, they do not preclude the athlete from further contact sports. These include isolated laminar or spinous process fractures. Depending on the situation, a healed minor vertebral body fracture, stable according to flexion-extension films, may permit further participation in contact sports.

On the other hand, strong consideration should be given to disallowing further participation of patients without fractures, instability, or neurologic deficit who suffer repeated injuries with symptoms suggestive of spinal cord involvement (eg, bilaterality). An MRI and sometimes somatosensory evoked potential testing should be performed in an attempt to demonstrate spinal cord injury, either radiographically or physiologically. These studies may aid in documenting actual neurologic compromise, which would preclude further participation in contact sports.

OTHER SPINAL CORD AND PERIPHERAL NERVE CONDITIONS AND PREDISPOSITIONS TO SPINAL CORD INJURY

Whether an injury represents involvement of the central nervous system (spinal cord) or peripheral nerves may be uncertain. This situation is best represented by the injury known as ''burners'' or ''stingers'' and is seen most commonly in football players but also in wrestlers. This injury results from head and shoulder contact in which the head is laterally flexed to the opposite side with downward traction to the ipsilateral shoulder, causing traction on the upper trunk of the brachial plexus, or by axial loads to the head or shoulders causing injury to the cervical root within its foramen. Typically transient and lasting for up to 15 to 30 minutes, weakness and a searing, lancing pain in the arm may occur. Residual pain or neurologic deficit corresponding to the upper trunk of the plexus or cervical nerve root may persist for days or months, and proper management is essential.[11]

The examiner must consider whether the complaints and any detectable neurologic abnormalities characterize involvement of a cervical nerve root, the brachial plexus, or the spinal cord. Nerve root symptoms include pain radiating into a specific dermatomal pattern, with the possibility of neurologic deficits related to that dermatomal sensory pattern or to the muscle innervated. Plexus involvement is predicted by persistent pain, often in more than one dermatome and sometimes in the entire arm, or by weakness of more than one major muscle group. Most seriously, one must discern the likelihood of spinal cord injury, which is more common if the symptoms are bilateral, occur in the lower extremities, reflect the long tract or include bladder or sexual disturbance. A disturbance of motor or sensory function (or both) on one half of the body may also be of spinal origin. Electromyography may help in distinguishing root and plexus injury from spinal cord involvement but ordinarily is not diagnostic until 3 weeks or longer postinjury. When evidence suggests spinal cord and not root or plexus injury, a thorough search must be performed to identify a possible occult bony injury or spinal cord contusion. Although rare, acute traumatic rupture of an intervertebral disc or traumatic hematoma or edema must be considered, and MRI is superior for diagnostic evaluation (Figure 2).

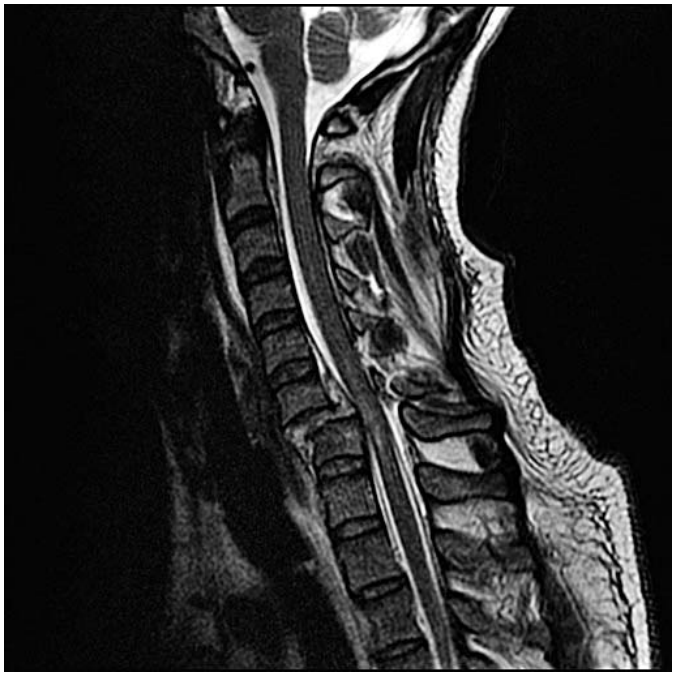

Figure 2. C6-C7 acute, traumatic herniated nucleus pulposus with a fracture-dislocation and spinal cord edema.

Another group of patients may have congenital spinal canal stenosis, posterior ligamentous compromise, resolved (either medically or surgically) herniated nucleus pulposus (Figure 3), or congenital vertebral body fusion. These individuals have a heterogeneous assortment of abnormalities, which by themselves may not require treatment but yet may be associated with a higher risk of injury in contact sports. In general, any patient who has had neck surgery for disc herniation is cleared for further contact sports after postoperative bony fusion is documented. However, the disposition of each patient should be made on an individual basis.

Transient Spinal Cord Injury

Transient spinal cord injury (TSCI) during athletic competition is one of the most complicated situations the athletic trainer, team physician, paramedic, and neurosurgeon may encounter. The initial neurologic signs are often complex, which can make on-field management and subsequent triage difficult. Although usually seen in athletes in traditional contact sports

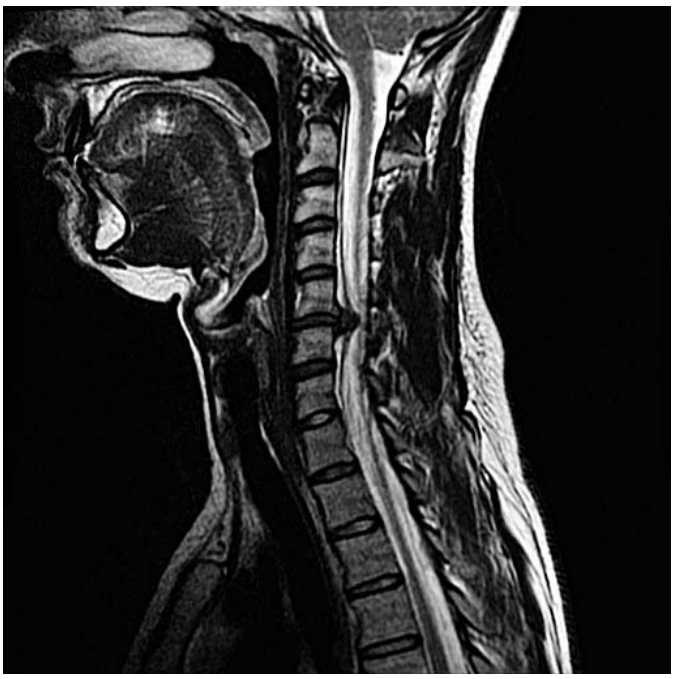

Figure 3. C5-C6 herniated nucleus pulposus.

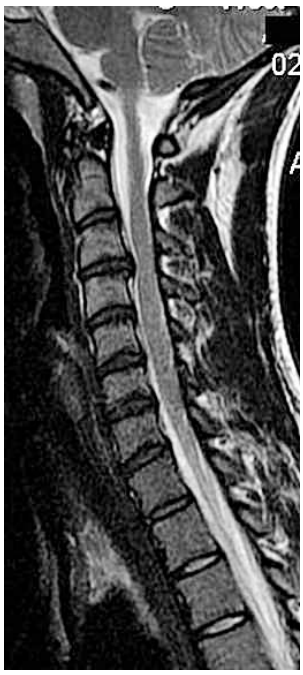

Figure 4. Sagittal view on magnetic resonance imaging showing congenital spinal stenosis in a collegiate gymnast with transient spinal cord injury and a focal lesion at C3-C4. Because of recurrent symptoms and the focal lesion superimposed on the widespread stenosis, the athlete was medically disqualified from further contact sport participation.

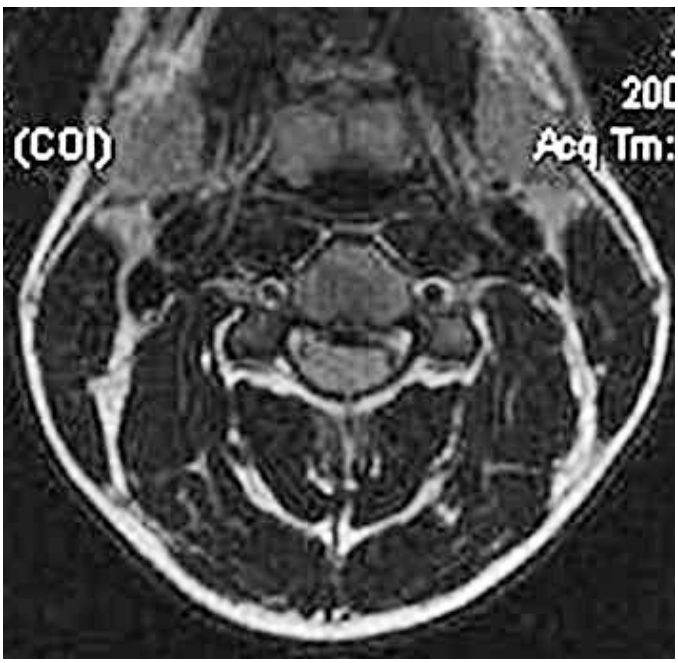

Figure 5. Axial view on magnetic resonance imaging of the same gymnast shown in Figure 4, demonstrating severe spinal canal narrowing with spinal cord compromise.

such as football, wrestling, and ice hockey, TSCI may also be seen in other sport activities in which collisions occur, such as basketball, soccer, gymnastics, and baseball.

The occurrence of TSCI is uncommon in athletes, with the incidence estimated to be 7.3 per 10 000 participants in American football.[21] The condition typically presents with involvement of both arms, all 4 extremities, or an ipsilateral arm and leg, consisting of either motor or sensory or combined symptoms. The most frequent pattern, seen in approximately 80% of cases, is involvement of all 4 extremities with weakness or quadriplegia and combined sensory deficits. The symptoms usually resolve in 15 to 30 minutes but may last for 24 to 48 hours. Subsequently, the symptoms resolve completely, with pain-free, full range of motion of the cervical spine. Once recovery has occurred, the neurologic examination is typically normal, without residual long tract findings.

The phenomenon of TSCI has been noted for more than a century, with several mechanisms offered to explain the pathophysiology. Obersteiner,[22] in 1879, described neurologic dysfunction termed *spinal cord concussion,* also subsequently identified in wartime injuries. In 1941, Denny-Brown and Russel[23] and later Groat et al[24] reported that temporary spinal cord injury resulted from spinal cord neural transmission failure. Penning[25] postulated that an extreme movement can occur in high-velocity injuries, resulting in rapid compression of the spinal cord by the posterior-inferior cervical vertebral body and the subjacent spinal lamina. He termed this the ''pincers effect,'' altering spinal cord impulse transmission via momentary impingement without causing a structural failure and, thus, resulting in no lasting radiographic injury.[26] Spinal cord concussion has become accepted to mean those instances in which sufficient forces result in temporary inhibition of spinal cord impulse transmission without causing structural damage to the vertebral column or spinal cord.

Spinal Stenosis

In our experience and that of others, spinal stenosis is the most common radiographic finding in the athletic population presenting with TSCI (Figures 4 and 5).[21,27–29] The sagittal diameter of the cervical canal averages 18.4 mm at C3 and 17.8 mm from the C4 to C7 levels.[26,30] Defined as a spinal canal diameter of 14 mm or less, spinal stenosis may be of congenital or acquired forms[26,29,31] and asymptomatic.[32] Stenosis often occurs in athletes secondary to degenerative osteo-

phyte formation, which narrows the canal, probably resulting from repetitive contact sport stresses, and is referred to as acquired rather than congenital stenosis. A 3-fold increase in the incidence of peripheral nerve ''burner'' or ''stinger'' injuries, which occur from compression within the foramen, has been associated with spinal stenosis.[33,34] Spinal stenosis also has been claimed to result in an increased risk of spinal cord injury due to less available space to accommodate excursions of the spinal cord with elongation, compression, or momentary impingement.[35,36] Spinal stenosis is associated with a greater chance of neurologic involvement in any person presenting with vertebral column injury.[36]

The radiographic assessment of the athlete with suspect or proven vertebral column or spinal cord injury includes plain radiographs, MRI, and CT. The vertebral canal–body ratio has been previously proposed as a valid measure of the available space in which the spinal cord resides and moves to escape permanent injury. A canal-body ratio of <0.80 has been considered indicative of spinal stenosis.[37] Measurements of the spinal cord among individuals are fairly uniform, but bony anatomy varies significantly among different-sized individuals. Persons with a large body habitus and size are associated with relatively larger vertebral bodies.[38] This fact, as well as the ability of MRI to directly image the vertebral column, intervertebral discs, spinal canal, and spinal cord, have made MRI (and not bony landmarks) the preferred method for assessing the relative anatomy. In a previous report[39] of 63 athletes with cervical spine injuries in the pre-MRI era, Bailes et al cited 29% who sustained TSCI, with cervical stenosis being the predominant radiographic finding. More recent experience with additional athletes has led to the current emphasis on MRI assessment of cerebrospinal fluid (CSF) signal around the spinal cord, termed the *functional reserve.* This imaging allows direct visualization of the neural elements, in particular the spinal cord, which improves on the previously known advantage of contrast myelography to discern compression and not bony anatomy alone. The visualization of the CSF signal, its attenuation in areas of stenosis, and changes on dynamic flexion-extension MRI studies are paramount for thorough analysis and decision making in this patient population. Patients who do not have a CSF pattern on axial and sagittal MRI views have *functional stenosis.*[27,30]

Athletic Mechanism and Stenosis

Most athletes who sustain catastrophic spinal cord injury have structural failure of the vertebral column that is not caused by or related to stenosis.[4,21,28,30] Catastrophic spinal cord injury in athletes with fracture-dislocation is believed not to result from an athlete's spinal anatomy but instead from the use of techniques that involve making initial contact with the top or crown of the helmet or head. These include being driven into the mat in wrestling, spearing in football, and being knocked into the boards in ice hockey.[4,33,40–42] These vertex impacts cause an axial load, most commonly with a flexion component, which leads to structural failure of the vertebral column and spinal cord injury.[4,21,28,30] Athletic spinal cord injury does not appear to follow a pattern of repetitive transient injuries culminating in a catastrophic event of vertebral column failure.[30] In a survey of 117 football players who sustained permanent quadriplegia, none recalled having had a prior prodromal experience of motor symptoms; only one (0.9%) had previous sensory symptoms referable to the spinal cord.[21] The syndrome of *spear tackler's spine* is an exception and consists of 4 characteristics: reversal of cervical lordosis; radiographic evidence of previous, healed, minor vertebral body fractures; canal stenosis; and habitual use of spear-tackling techniques.[43] In some instances, the factors of spear tackler's syndrome may be corrected and the athlete's ability to safely return to contact sport participation may be considered. Although rare, with likely incomplete documentation, at least 4 reported instances of spinal cord injury have occurred after prior symptoms; however, 2 of these were known spear tacklers.[21,30,44]

The syndrome of TSCI occurs in 2 categories of athletes. The first group represents those athletes who sustain temporary spinal cord dysfunction but have normal radiographic studies and lack cervical spinal stenosis.[21,39,45] In the series of Zwimpfer and Bernstein,[45] 5 of 19 individuals (mostly young males) sustained TSCI during contact or collision athletic activities. All had normal radiographic evaluations, with cervical canal diameters ranging from 17 to 20 mm. The authors reported that this phenomenon represented spinal cord concussion, reminiscent of cerebral concussion.[45] This condition may be similar in some respects to the syndrome of spinal cord injury without radiographic abnormality (SCIWORA). In contrast, cervical stenosis is associated with TSCI in the athletic population, with likely brief impingement causing a physiologic disturbance in axonal action potentials, expressed as long tract dysfunction. Certain high-velocity impacts may exceed the tolerance threshold in these predisposed individuals without causing a structural failure in the vertebral column. In general, it also seems that this phenomenon does not lead to permanent injury in the vast majority of these athletes, nor do they subsequently experience cervical fracture-dislocation. Currently, no readily acceptable corrective treatment exists for spinal stenosis in this population; cervical laminoplasty has been proposed as a potential solution but is thus far unproven. For other structural lesions, such as herniated nucleus pulposus with a lateral impingement component, posterior microsurgical decompression is an attractive alternative that minimizes disruption of normal spinal anatomy.

For the clinician attempting to provide advice regarding return-to-play recommendations for athletes involved in contact sports, available management decision data are mainly experiential. Based on the available published information, it appears that catastrophic spinal cord injury is not usually associated with a prodrome or with recurrent symptoms and, furthermore, that spinal stenosis alone does not result in a high risk of future catastrophic spinal cord injury. It is now recommended that this assessment be based on sagittal MRI images that reveal preservation of the CSF signal without a focal lesion.

Once permanent spinal cord injury and surgically or otherwise correctable abnormalities have been eliminated, cervical canal stenosis remains a common finding in athletes with TSCI. Athletes with additional radiographic abnormalities, such as cord contusion, herniated nucleus pulposus, focal stenosis, compressive osteophyte formation, ligamentous instability or intrusion within the spinal canal, or spear tackler's spine, should not be allowed to participate further in their sports. However, most patients in whom MRI documents preservation of the CSF signal are able to return safely to contact sports participation (Figure 6). No current findings in the literature suggest that a single episode of temporary spinal cord dysfunction in an athlete with spinal stenosis substantially in-

Figure 6. Sagittal view on magnetic resonance imaging of a collegiate football player with a single episode of transient spinal cord injury but smooth, nonfocal stenosis and preservation of the cerebrospinal fluid signal throughout. This player was permitted to return to competition and experienced no recurrent symptoms.

creases the risk of future catastrophic spinal cord injury. Although the presence of an MRI-documented CSF signal may not be ''protective,'' it may correlate with the lack of irreversible spinal cord compression in a high-velocity injury. Multiple episodes of TSCI involving symptoms of spinal cord dysfunction, regardless of the presence of the CSF signal, may be better treated by more conservative approaches. The subsequent risk of catastrophic spinal cord injury secondary to TSCI in athletes with cervical stenosis is uncertain. The findings in this small series of cases demonstrate that catastrophic injury did not develop secondary to TSCI and cervical stenosis in 10 athletes; of the 4 with MR imaging-documented CSF signal preservation who returned to participate, none suffered recurrent symptoms.[31] Neurologic sports medicine is highly individualized, and many factors must be considered before an injured athlete is allowed to return to competition. In returning to play a contact or collision sport, however, the athlete with previous TSCI must accept that the injury was not necessarily benign and that an apparently small, but nonetheless present, risk of permanent spinal cord injury exists.

PREVENTION

Athletic cervical spine injuries may be preventable with proper technique and instruction. In contrast to recreational sports such as diving, organized team sports such as football, wrestling, and gymnastics have the advantages of ritualized training and competition phases that allow coaches and trainers to convey proper methods to athletes. Correct positioning and techniques of blocking and tackling in football avoid hyperflexion and initial contact with the head. Rule changes to avoid ''spearing'' contact have helped to reduce the incidence of spinal and head injuries and, we hope, will continue to evolve. Gymnastics, ice hockey, and wrestling coaches must emphasize proper maneuvers to reduce accidents. Particularly in football, but also in other sports, offseason neck-strengthening exercises are a vital part of the injury prevention programs. Improvements in equipment design and manufacture have also made great strides for safety in athletic competition, and future developments may prove beneficial in injury reduction.

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