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Catastrophic Cervical Spine Injuries in the Collision Sport Athlete, Part 1

Epidemiology, Functional Anatomy, and Diagnosis

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Catastrophic cervical spine injuries can lead to devastating consequences for the collision athlete. Improved understanding of these injuries can facilitate early diagnosis and effective on-field management. This article is the first of a 2-part series. The first part reviews the current concepts regarding the epidemiology, functional anatomy, and diagnostic considerations relevant to cervical spine trauma in collision sports. In the second part, to be published later, the principles of emergency care of the cervical spine-injured athlete are reviewed. This article provides a rational approach to the early recognition of the different clinical syndromes associated with catastrophic cervical spine injury. Rapid on-field diagnosis can help to optimize the outcomes of these catastrophic injuries.

Keywords: cervical spine injury; collision athlete; football; hockey

Sporting events are the fourth most common cause of spinal cord injury (behind motor vehicle accidents, violence, and falls) and account for approximately 7.5% of the total injuries since 1990.¹⁴ In contrast to the other major causes of spinal cord injury, sports-related spinal cord injuries occur at a mean age of 24.⁴ In fact, in the first 3 decades of life, sports injuries are the second most common cause of spinal cord injury.¹⁵ Several sports have been identified as placing the participant at high risk for spinal cord injury, including football, ice hockey, rugby,^{19,41} skiing,^{10,26} snowboarding,^{10,26} and equestrian sports.²⁴ The collision sports of football and ice hockey account for a large number of these injuries and, historically, have been studied extensively because of the popularity and high visibility of these sports.

A *catastrophic cervical spine injury* can be defined as a structural distortion of the cervical spinal column associated with actual or potential damage to the spinal cord. This type of trauma can lead to devastating and irreversible neurologic consequences for the athlete. Although

advances in protective equipment and modifications in rules of play have led to a decreased incidence of athletic cervical spine trauma, this clinical problem continues to present a challenge for the sports medicine professional. Given the relative infrequency of the condition, few physicians are able to develop extensive experience in the emergency care of serious neck injuries. In addition, the initial management of a collision sport athlete with an injured cervical spine differs from that of a typical trauma victim. Specifically, the protective helmet and shoulder pads worn by the player complicate the medical evaluation and immobilization process.

Improper handling of the cervical spinal column on the field or during transport can worsen (or even cause) spinal cord dysfunction. Failure to appropriately manage a catastrophic neck injury may even compromise the athlete's cardiac and respiratory status. Therefore, this high-risk situation requires the responsible medical staff to have specialized knowledge and training to act efficiently and effectively in this unique clinical setting.

The primary purpose of this review is to describe a systematic approach to the emergency management of catastrophic cervical spine injury in football and ice hockey. This article is the first in a 2-part series. The goals of the first part of the article are to describe the epidemiology, functional anatomy, and diagnostic considerations relevant to cervical spine trauma in football and ice hockey. In addition, the specific clinical syndromes of cervical spine trauma encountered by the sports medicine physician will be reviewed in detail.

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EPIDEMIOLOGY

Football

Annual participation in the collision sport of football is estimated to be 1.8 million athletes per year. Approximately 1.5 million participate at the junior/senior high school level, 75 000 in college, and approximately 2000 in professional football.¹³ Spinal cord injury is uncommon in this sport, yet the burden of catastrophic cervical trauma is far from negligible. Although football has a lower rate of catastrophic cervical spine injuries (per 100 000 players) than ice hockey or gymnastics, the large number of participants in the United States has resulted in football being associated with the largest overall number of catastrophic cervical spine injuries.^{2,13}

The pattern of cervical spine injury in football has evolved over time. During the years 1959 to 1963, Schneider documented 56 cases (1.36/100 000 participants) of cervical fracture/dislocation, of which 30 (0.73/100 000) were associated with permanent quadriplegia.²⁵ From 1971 to 1975, the National Football Head and Neck Injury Registry compiled 259 (4.14/100 000) cervical fractures/dislocations and 99 cases (1.58/100 000) of quadriplegia.³⁶ This increased and disturbingly high rate of catastrophic cervical trauma coincided with the development of modern helmets. Improved head protection led to playing techniques that used the top of the helmet as the initial point of contact for blocking and tackling. It became apparent that this style of play placed the cervical spine at increased risk of injury.

In January 1976, the execution of headfirst contact was banned by the National Collegiate Athletic Association Football Rules Committee and high school football governing bodies.³⁸ This rule modification brought about changes in coaching and playing techniques that led to a progressive decrease over the next decade in the incidence of spinal cord injury related to football. From 1976 to 1987, the rate of cervical injuries decreased 70% from 7.72 per 100 000 to 2.31 per 100 000 at the high school level.³⁹ Similarly, traumatic quadriplegia decreased approximately 82% over the same time period. At the time of the rule change in 1976, the annual rate of permanent quadriplegia was 2.24 per 100 000 participants in high school football and 10.66 per 100 000 participants in college football. By 1984, the rate of this neurologic injury had decreased to 0.38 per 100 000 and 0 per 100 000, respectively.¹³ Current data indicate a plateau in the incidence of traumatic quadriplegia. In 2002, the incidence of this injury was 0.33 per 100 000 in high school football and 1.33 per 100 000 in college football.¹³

Cantu and Mueller reviewed the incidence of catastrophic spine injuries in American football from 1977 to 2001.² Over this 25-year period, 223 football players sustained a catastrophic cervical spine injury with no or incomplete recovery. Overall, 183 injuries occurred in high school athletes, 29 in college athletes, 7 in professional athletes, and 4 in recreational players. Based on these numbers, the incidence rates for catastrophic spine injury per 100 000 participants over the past 25 years are 0.52 in

high school, 1.55 in college, and 14 in professional football. Furthermore, 71% of catastrophic cervical spine injuries occurred in defensive players, and 69% of these injuries were the result of tackling. Fractures/dislocations occurred in 79% of these catastrophic injuries.

Ice Hockey

Ice hockey is immensely popular in North America. Despite the large number of participants, before 1980 the sport was a relatively rare cause of catastrophic spinal trauma. Since then, there has been a marked increase in the occurrence of hockey-related cervical spine injuries. The Canadian surveys performed by Tator et al²⁷ from 1966 to 1993 documented a total of 241 spinal fracture and dislocations related to hockey, of which approximately 90% involved the C1 through T1 vertebral levels. Major disruptions of the spinal column occurred at an increased rate between 1982 and 1993, with a mean of 16.8 fractures/dislocations per year during that time period. Of the 207 athletes in the Canadian registry with adequate documentation of neurologic status, 108 (52.2%) sustained a permanent spinal cord injury, and in 52 (25.1%) the cord lesion was complete. Eight players died as a result of complications of their spinal cord injuries.

Although football is associated with a higher total number of nonfatal catastrophic neck injuries each year, the annual incidence of spinal cord damage with paralysis is at least 3 times greater in Canadian hockey than in American football.^{13,27}

Checking an opponent from behind has been identified as an important causative factor of cervical spine trauma in hockey. This playing tactic typically produces a headfirst collision of the checked player with the boards.^{12,27} In an effort to prevent these injuries, rule changes have been adopted that prohibit checking from behind and checking of an opponent who is no longer controlling the puck.^{27,28} Data from the Canadian registry suggest that fewer cases of major spinal column trauma and complete quadriplegia have been caused by illegal playing techniques since the rule changes have been instituted.²⁷

CLINICAL ANATOMY

Effective management of a catastrophic neck injury requires a basic understanding of the regional anatomy. The cervical spine acts to support the head and protect the neural elements while simultaneously allowing for complex motion. The cervical spinal column can be conceptually divided into an upper region and a lower region that differ in terms of anatomical design and function.

The upper cervical spine consists of the occiput and the first 2 vertebrae (Figure 1). The atlas (C1) is a bony ring that articulates with the occipital condyles. Motion in the sagittal plane is the major function of the atlantooccipital joint, which accounts for 40% of all cervical flexion-extension and 5° to 10° of lateral bending. The axis (C2) has a true vertebral body from which projects the odontoid process, or dens. The midline atlantodens articulation is

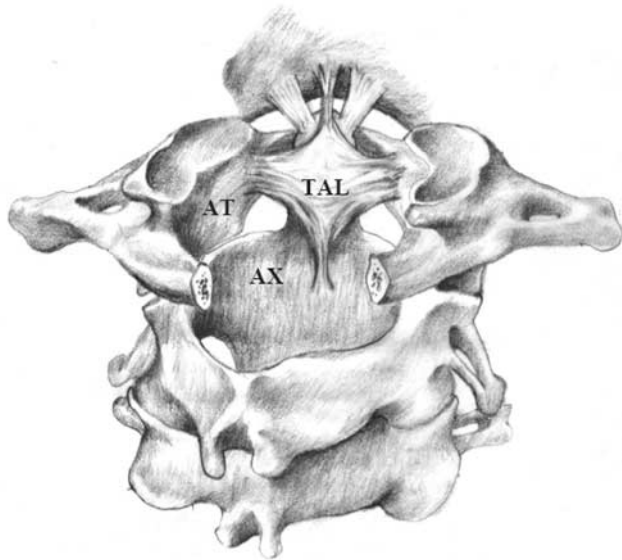


Figure 1. Anatomy of the atlantoaxial complex. The transverse atlantal ligament (TAL) stabilizes the median atlanto-dens articulation. The dens acts as a pivot about which the atlas (AT) rotates. AX, axis.

stabilized by the transverse atlantal ligament, which serves as an effective restraint against forward translation of the atlas. This specialized osseoligamentous anatomy allows the second cervical vertebra to act as a bearing surface on which the atlas rotates in a highly unconstrained manner. The atlantoaxial complex is responsible for 60% of all cervical rotation.⁷

The lower cervical spine, composed of the C3 through C7 vertebrae, accounts for the remaining arc of neck flexion, extension, lateral bending, and rotation. The 2 contiguous vertebrae and supporting soft tissues of each motion segment can be separated into an anterior and a posterior column (Figure 2). Stability of a cervical segment is derived mainly from the anterior spinal elements. Compression of the spinal column is primarily resisted by the vertebral bodies and intervertebral disk. Shear forces are opposed primarily by paraspinal musculature and ligamentous support. The facet articulations also restrain forward vertebral translation by virtue of the coronal plane orientation of the articular processes. The ligamentous structures are the primary restraints to distraction of the spine. Tensile forces are countered mainly by the annulus fibrosus and longitudinal ligaments, with additional resistance provided by the ligamentum flavum, facet capsules, and the supraspinous and interspinous ligaments.⁷

The spinal cord, extending downward from the foramen magnum, is protected circumferentially by the osseoligamentous structures of the cervical spine. In the midsagittal plane, the cervical spinal canal can be conceptualized as having the shape of a funnel (Figure 3).¹⁷ The luminal tolerance in the upper cervical region is such that the cord occupies less than half of the canal's cross-sectional area at the level of the atlas. Progressive narrowing of the bony canal combined with a gradual widening of the spinal cord

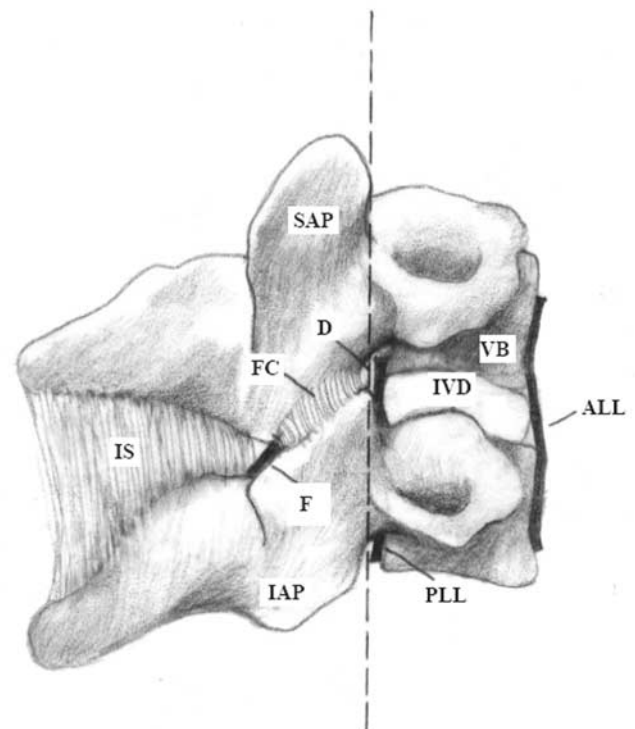


Figure 2. Anatomy of the lower cervical spine. The elements of the anterior column include the posterior longitudinal ligament and all structures ventral to it. The posterior column consists of those structures dorsal to the posterior longitudinal ligament. VB, vertebral body; IVD, intervertebral disk; ALL, anterior longitudinal ligament; PLL, posterior longitudinal ligament; F, facet articulation; FC, facet joint capsule; IS, interspinous ligament; SAP, superior articular process; IAP, inferior articular process; D, division between anterior and posterior column.

diameter reduce the space available for the neural elements between the C4 and C7 levels. At the lower cervical levels, the spinal cord normally fills approximately 75% of the cross-sectional area of the canal.¹⁷

In the subaxial cervical spine, the dimensions of the spinal cord remain relatively constant among humans.¹⁶ The average midsagittal cord diameter is in the range of 8 to 9 mm. In contrast, the size of the vertebral canal in the lower cervical region shows substantial individual variation. Normally, the midsagittal diameter of the lower cervical spinal canal is in the range of 14 to 23 mm. The canal is considered stenotic when its anteroposterior dimension measures less than 13 mm on a lateral radiograph⁶ or the Pavlov ratio is less than 0.8 (Figure 3B). In the presence of a narrow canal, the space available for the spinal cord is reduced. Cord compression can be predicted when the midsagittal canal diameter is 10 mm or less.⁵ These facts are clinically relevant because the available space for the neural elements has been identified as an important predictor of acute neurologic dysfunction.⁸ Cervical canal stenosis has been implicated as a risk factor for the burner phenomenon (see later) in college football players.⁹

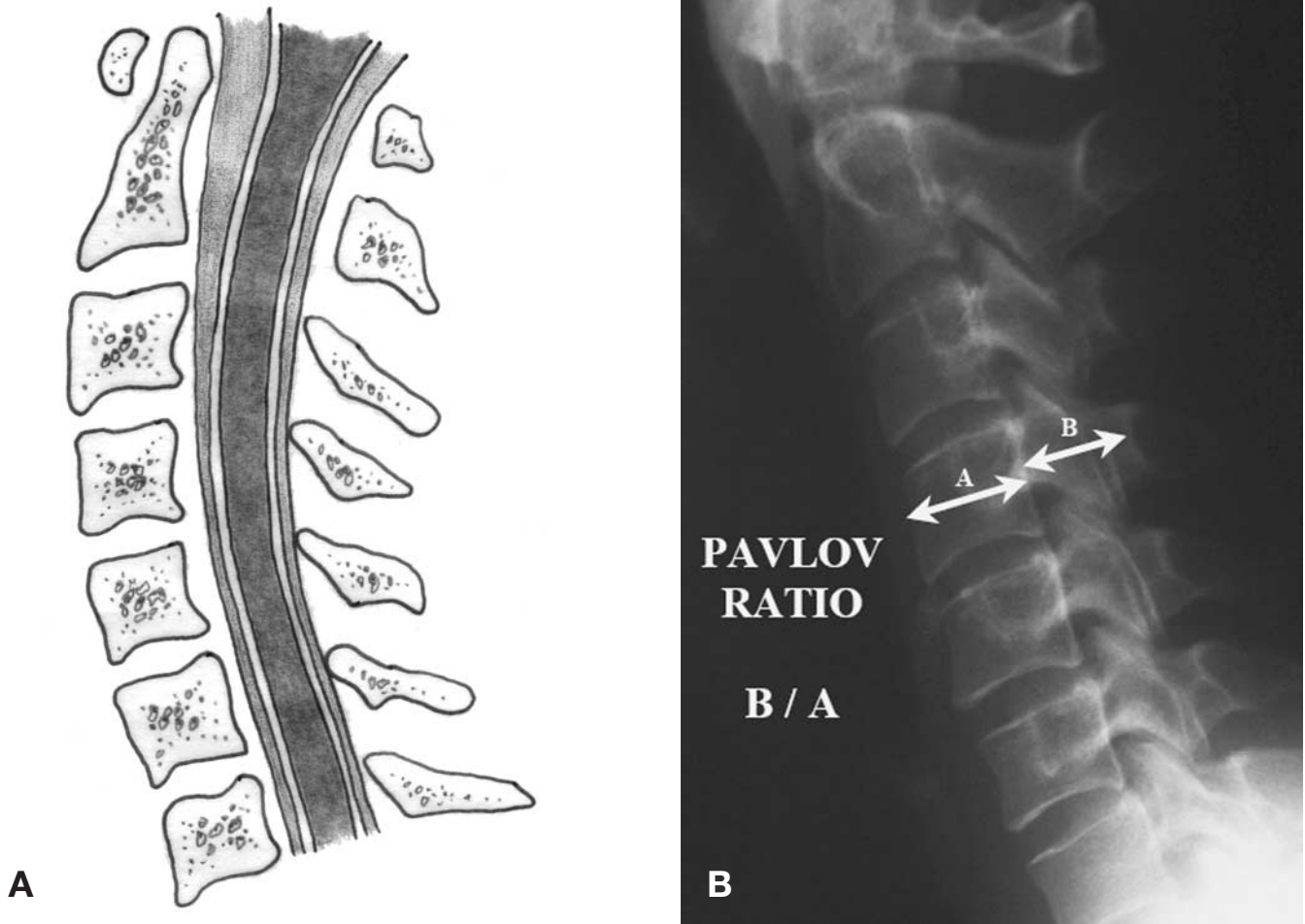


Figure 3. The cervical vertebral canal and spinal cord. A, midsagittal schema of the cervical spine. The diameter of the spinal canal progressively narrows in a cranial to caudal direction with the space available for the spinal cord at a minimum between C4 and C7. B, lateral cervical spinal radiograph. At each cervical level, the sagittal diameter of the spinal canal is measured from the middle of the posterior vertebral body cortex to the nearest point on the spinolaminar line. The Pavlov ratio is calculated by dividing the anteroposterior width of the vertebral body (A arrow) into the diameter of the spinal canal (B arrow). Cervical stenosis is indicated when the canal diameter measures less than 13 mm or the Pavlov ratio is below 0.8.

CATASTROPHIC CERVICAL SPINE INJURIES

A spectrum of cervical spine pathologic abnormalities can result from participation in a collision sport. Initial evaluation of the athlete requires familiarity with the mechanism, pathologic anatomy, and clinical presentation of the neck injuries common in football and hockey. The traumatic conditions that typically result in a catastrophic cervical spine problem include unstable fractures and dislocations, transient quadriplegia, acute central disk herniation, and congenital spinal anomalies. These 4 diagnostic entities, by virtue of a shared underlying disturbance of cord function, produce neurologic symptoms and signs that involve the extremities in a bilateral distribution.

Unstable Fractures and Dislocations

Unstable fracture and/or dislocation are the most common causes of catastrophic cervical spine trauma in the colli-

sion sport athlete. An osseous or ligament injury is considered unstable when it results in loss of the ability of the spine under physiologic loads to maintain its premorbid patterns of motion so that there is no initial or additional damage to the spinal cord or nerve roots, no major deformity, and no incapacitating pain.⁴²

Understanding the potential mechanisms of athletic injury to the spine allows one to predict the typical patterns of structural damage. The mechanism of spinal column damage is best described by using the concept of a major injury vector. In football and hockey, the injury vector most frequently associated with cervical spinal cord injury is compression.^{27,31,39} Flexion is the primary disruptive force in a smaller percentage of cases.

When an axial force is applied to the vertex of the athlete's helmet, the cervical spine is compressed between the instantly decelerated head and the mass of the oncoming body. The response of the spine to the applied axial load depends largely on neck position at the time of impact.

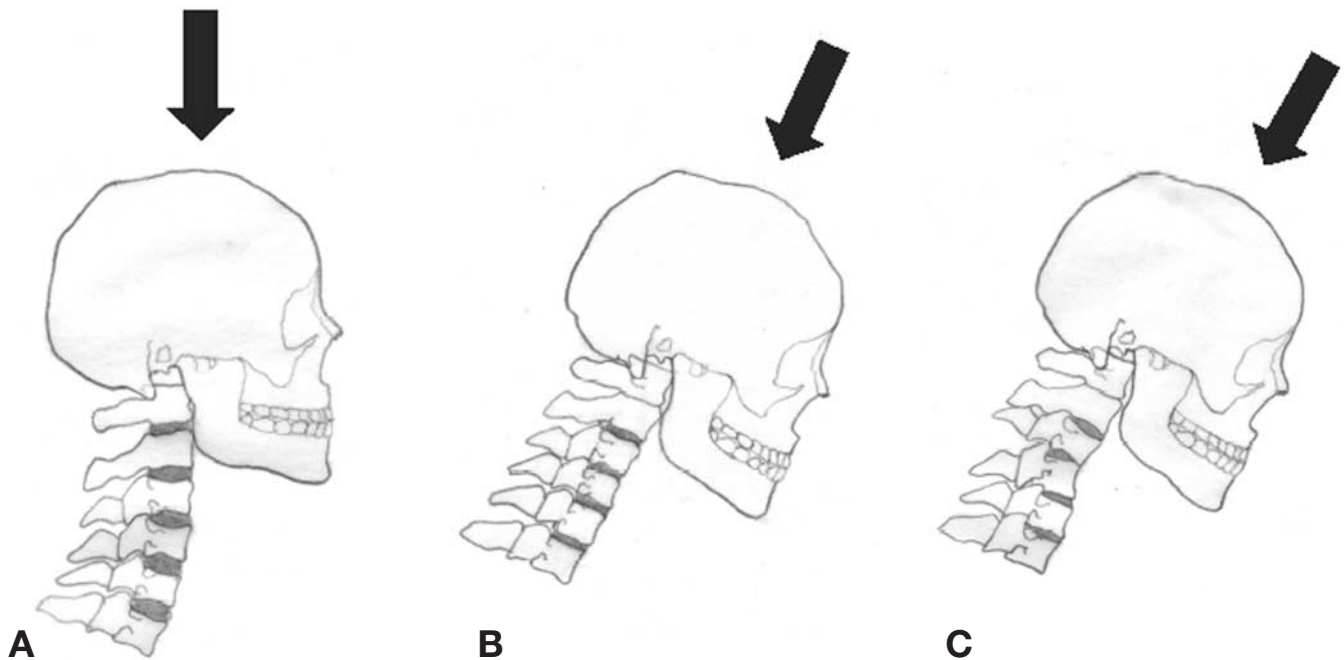


Figure 4. Response of the cervical spine to applied axial load. A, with the neck in neutral alignment, the vertebral column is extended. The compressive force can be dissipated by the spinal musculature and ligaments. B, with the neck in a flexed posture, the spine straightens out and becomes collinear with the axial force. C, at the time of impact, the straightened cervical spine undergoes a rapid deformation and buckles under the compressive load.

With the neck in neutral alignment, the cervical spinal column is slightly extended as a result of its normal lordotic posture. In this situation, compressive force can be effectively dissipated by the paravertebral musculature and vertebral ligaments (Figure 4A). When the head and neck are prepositioned in slight flexion, cervical lordosis is eliminated (Figure 4B). Biomechanically, the straightened spine behaves like a segmented column. Axially directed force is transmitted along the spine's longitudinal axis, causing large amounts of energy to be transferred directly to the vertebrae as opposed to the surrounding soft tissues.³⁹ Cadaveric studies have shown that the cervical spine, when straight and collinear with the applied load, responds to compression by buckling (Figure 4C).⁴⁵

The majority of fractures and dislocations occur in the lower cervical region. With respect to catastrophic trauma, there are 2 major patterns of spinal column damage caused by the compression injury vector. The most common variant is classified as a compressive-flexion injury that results from the combination of axial force and a bending moment. Progressive deformation results in shortening of the anterior column because of compressive failure of the vertebral body and lengthening of the posterior column because of tensile failure of the spinal ligaments (Figure 5A). Often referred to as a "flexion teardrop" injury, this mode of structural failure is highly unstable and is frequently associated with spinal cord injury.³⁵

When the point of load application is such that a cervical vertebra is subjected to a relatively pure compression force, both the anterior column and posterior column shorten, resulting in a vertical compression (or "burst") fracture

(Figure 5B).¹ In this scenario, axial loading causes a progressive rise in intradiskal pressure such that the adjacent endplate fails. With extrusion of disk material through the fractured endplate, the vertebral body essentially explodes, displacing bone fragments in all directions. Spinal cord compromise often results from the retropulsion of osseous material into the spinal canal.

Catastrophic cervical trauma caused by a flexion vector generally results from either a direct blow to the occipital region or rapid deceleration of the torso. In both situations, a bending moment in the sagittal plane causes the head and neck to flex forward over the trunk, subjecting the ligamentous structures of the involved motion segment to tensile forces. In simplest terms, progressive force application disrupts the stabilizing ligaments of the involved motion segment in a posterior-to-anterior direction. Although a spectrum of osseoligamentous damage is possible, the flexion-distraction injury most likely to result in spinal cord dysfunction is a bilateral facet dislocation (Figure 6A).^{3,20,44} The addition of axial rotation to the distractive force can produce a unilateral facet dislocation that is associated with cord injury in up to 25% of cases (Figure 6B).³

An athlete who sustains an unstable lower cervical fracture and dislocation can exhibit a spectrum of neurologic dysfunction. In the worst-case scenario, there is complete quadriplegia with no preservation of sensory or motor function below the level of the cord lesion. Less severe cord damage can manifest as an incomplete spinal cord injury syndrome with partial preservation of sensory or motor function in the extremities and torso. In this regard, the

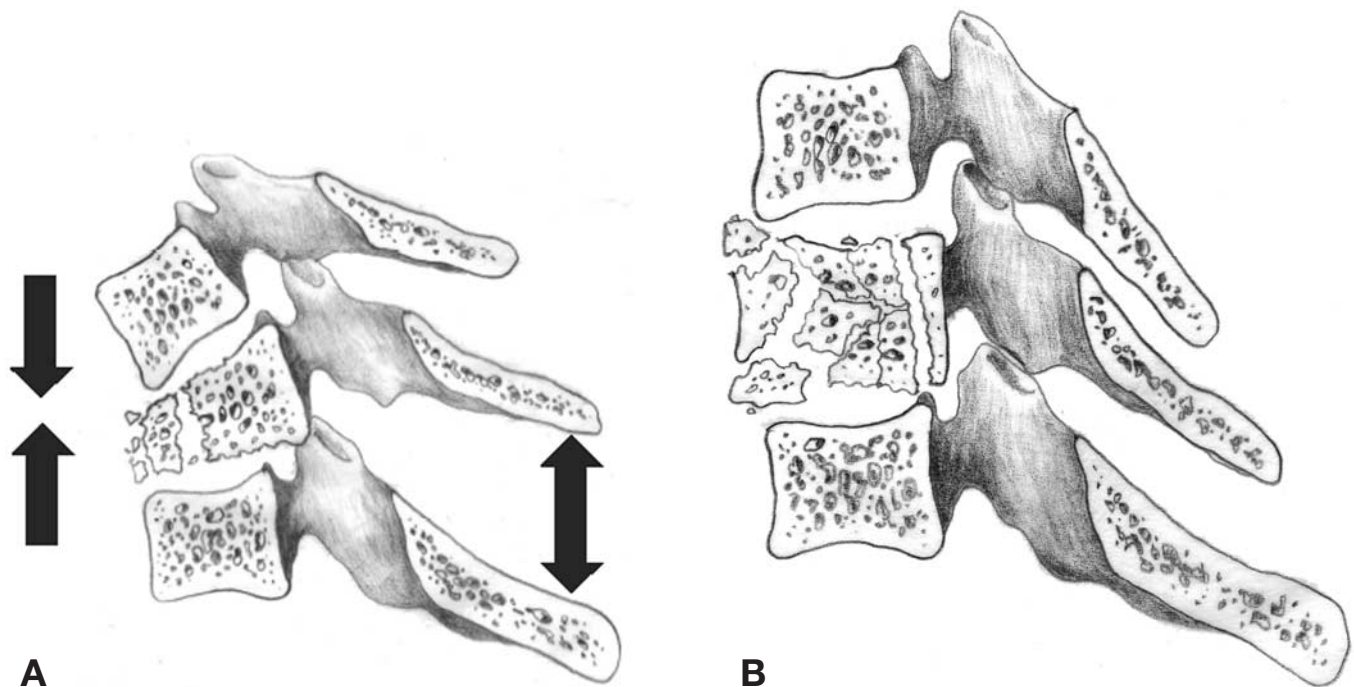


Figure 5. Compression injuries of the lower cervical spine. A, compressive-flexion pattern. The “teardrop” fracture variant is characterized by compressive failure of the anterior column with a coronal plane fracture extending through the vertebral body. Tensile forces cause disruption of the posterior spinal ligaments. B, vertical compression pattern. In the “burst” fracture variant, comminution of the vertebral body can be associated with retropulsion of osseous fragments into the spinal canal.

central cord syndrome is the most common pattern, followed in frequency by the anterior cord syndrome. Maroon et al have described a variant of the central cord lesion characterized by dysesthesias in both hands without loss of strength or sensation.^{11,43} This “burning hands” syndrome is likely caused by edema and vascular insufficiency affecting the medial aspect of the somatotopically arranged spinothalamic tracts.

Spinal cord damage due to fractures or dislocations involving the upper cervical spine is rare. Neurologic compromise is not usual because there is proportionately greater space available within the spinal canal compared with the lower cervical segments. Cord compression is highly improbable in relation to burst fracture of the atlas (Jefferson fracture) and traumatic spondylolisthesis of the axis (Hangman fracture) because these osseous injuries further expand the dimensions of the spinal canal. The traumatic conditions most likely to result in upper cervical cord injury are those that destabilize the atlantoaxial complex and include fracture of the odontoid and rupture of the transverse atlantal ligament. Along with quadriplegia, an unstable upper cervical injury can cause diaphragmatic paralysis with acute respiratory insufficiency by traumatizing the anterior horn cells that give rise to the phrenic nerve.

Last, it should be recognized that unstable cervical fractures/dislocations do not always result in upper motor neuron dysfunction. In some situations, only a lower motor neuron lesion will be present. For example, 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 neurologic impairment. Spinal cord injury in these scenarios is potential rather than actual based on the loss of structural integrity of the spinal column.

Transient Quadriplegia

Neurapraxia of the cervical spinal cord resulting in transient quadriplegia has been estimated to occur in 7 per 10 000 football players.³³ Athletes with developmental or acquired cervical stenosis are predisposed to this clinical entity.³⁰ In one study, a Pavlov ratio of less than 0.8 was documented in 93% of football players with cervical cord neurapraxia.³⁴ From a mechanical perspective, momentary cord compression can occur at the extremes of neck extension or flexion owing to a “pincer mechanism.”¹⁸ As described by Penning, forced hyperextension of a lower cervical motion segment causes an approximation of the posterior margin of the endplate of the cranial vertebral body to the spinolaminar line of the subjacent vertebra (Figure 7). Infolding of the ligamentum flavum may contribute to this dynamic narrowing of the spinal canal. In response to a hyperflexion force, the pincer effect is created by the endplate of the caudal vertebral body and the lamina of the cranial vertebra.

The pathophysiology of spinal cord dysfunction in this condition is thought to be due to a physiologic conduction block without true anatomical disruption of neuronal tis-

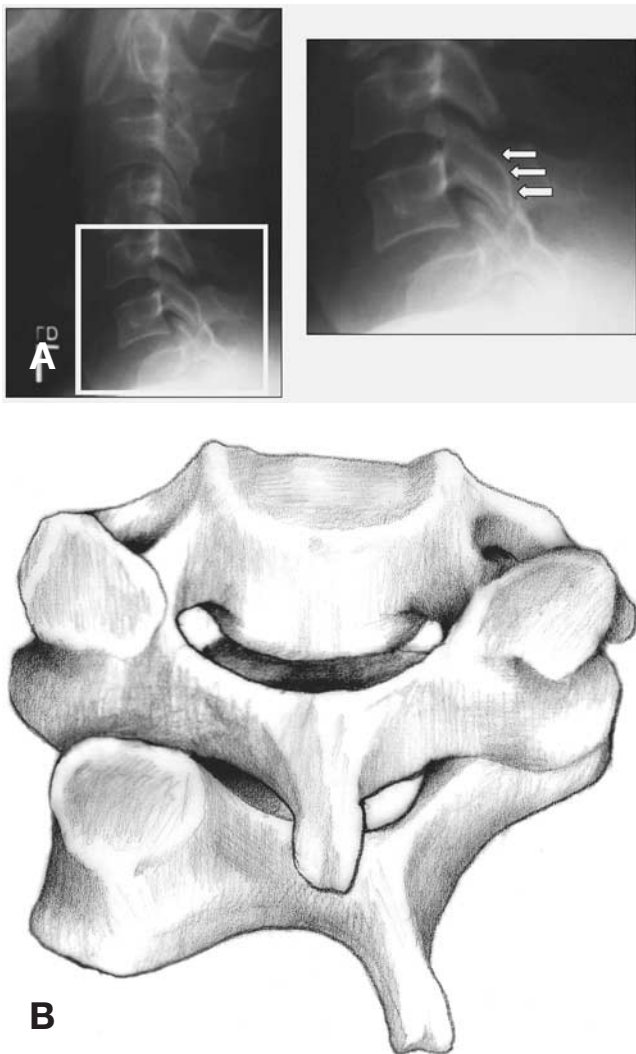


Figure 6. Flexion-distraction injuries of the lower cervical spine. A, bilateral facet dislocation. Lateral cervical spine radiograph showing bilateral facet dislocation of C6 on C7. This pattern of injury results from disruption of the supraspinous and interspinous ligament, facet capsules, ligamentum flavum, posterior longitudinal ligament, and the dorsal portions of the annulus fibrosus. The soft tissue damage can be associated with fractures of the superior articular processes. B, unilateral facet dislocation. This injury is usually caused by the combination of flexion and rotational forces. The addition of shear or compressive forces can cause fracture of the articular process.

sue. The neurapraxia is related to segmental demyelination and prolongation of the refractory periods of long-tract axons. As a result of the traumatic cord compression or contusion, the axons become unresponsive to stimulation for a variable period of time, creating a “post-convulsive” effect.⁴⁶ Based on experiments involving mechanical deformation of squid axons, Torg et al have theorized that local anoxia and increased intracellular calcium concentration

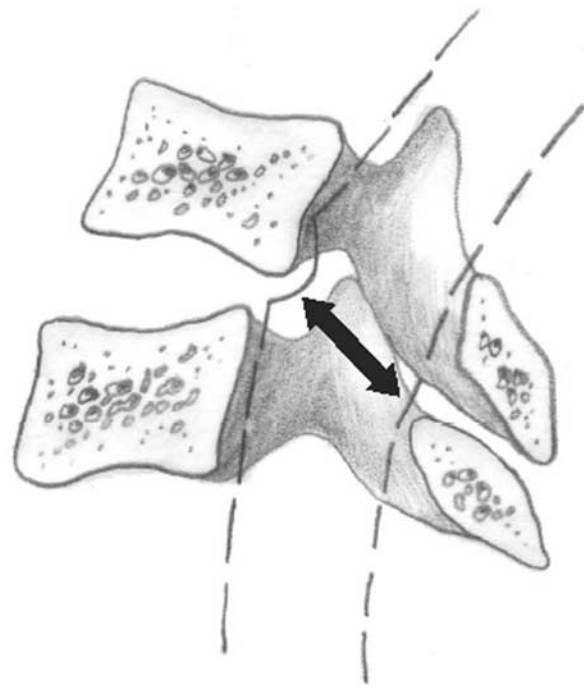


Figure 7. Transient quadriplegia. The “pincer mechanism” effect of hyperextension causes dynamic compression of the spinal cord between the endplate of the cranial vertebral body and the spinolaminar line of the subjacent vertebra.

are responsible for the temporary disturbance of cord function.³⁷

Clinically, the affected athlete complains of pain, tingling, or loss of sensation bilaterally in the upper and/or lower extremities. A spectrum of muscle weakness is possible, varying from no motor deficit, to mild quadriplegia, to complete quadriplegia. Neck discomfort is not present at the time of injury, and there is a full, pain-free range of cervical motion. By definition, the neurologic symptoms and signs are temporary; complete resolution generally occurs within 15 minutes but may take up to 48 hours. In players who return to football, the rate of recurrence has been reported to be as high as 56%.³²

Intervertebral Disk Herniation

Acute herniation of a lower cervical intervertebral disk can occur secondary to high-energy head or neck trauma during collision sport activities. With extrusion of the gelatinous nucleus pulposus through the annulus fibrosus and into the central spinal canal, the space available for the spinal cord is compromised. Acute cord compression by the soft nuclear material can produce a transient or permanent cord injury syndrome. The athlete may present with acute paralysis of all 4 extremities and a loss of pain and temperature sensation. A traumatic central disk herniation is also typically accompanied by the sudden onset of posterior neck pain and paraspinal muscle spasm.

Referred pain to the periscapular area or true radicular arm pain may also be present.

Congenital Spinal Anomalies

By altering the structural integrity of the spinal column, congenital anomalies predispose the collision sport athlete to reversible and permanent forms of spinal cord damage. Klippel-Feil syndrome, a failure of segmentation characterized by fusion of 2 or more vertebrae, reduces the number of motion segments available to dissipate loads applied to the cervical spine (Figure 8A). Over time, the increased stress on adjacent segments can result in mechanical instability and/or degenerative stenosis. Hypoplasia of the dens (a failure of formation involving the second vertebra) and developmental os odontoideum can both result in atlantoaxial instability (Figure 8B). Although frequently asymptomatic, these preexisting osseous abnormalities can increase the risk of a traumatic upper cervical cord injury.

ADDITIONAL DIAGNOSTIC CONSIDERATIONS

In reality, only a very small percentage of football and hockey players will sustain a catastrophic injury. The most common syndromes related to collision sport participation are not the result of a spinal cord injury and do not produce neurologic deficits that affect the extremities in a bilateral fashion. Rather, in the majority of neck-injured athletes, clinical findings are distributed to (1) a single upper extremity, (2) the neck and arm, or (3) the neck only.

When symptoms and signs are restricted to one upper limb, the primary diagnostic consideration is neurapraxia of a cervical nerve root or the brachial plexus. The anatomical basis of this well-known “stinger” or “burner” can be foraminal compression of a nerve root in response to forceful neck extension and rotation toward the affected side. Alternatively, a collision can result in a neurapraxia of the brachial plexus. Tensile forces can damage the plexus when the shoulder is depressed and the head deviates away from the involved side. An upper trunk lesion can also be caused by direct compression of the nerve structures between the shoulder pad and the ipsilateral scapula.²¹ Clinically, the athlete complains of burning pain, weakness, or paresthesias in the shoulder girdle and arm. Spinal examination will show a full, pain-free arc of neck motion with no midline palpation tenderness.⁴⁰ Neurologic dysfunction in the form of a motor, sensory, and/or reflex deficit can be present but is generally a self-limited process. The radiating arm pain tends to resolve within several minutes, with strength usually returning in 24 to 48 hours. Permanent sensorimotor deficits are rare, though a variable degree of muscle weakness can last up to 6 weeks in a small percentage of cases. The incidence of a burner in the careers of college football players has been reported to be 65%.²²

Clinical findings distributed to the neck and a single upper limb can be produced by a paracentral disk hernia-



Figure 8. Congenital anomalies of the cervical spine. A, Klippel-Feil syndrome. Sagittal MRI scan showing fusion of the C3-4 and C5-6 segments. This patient was myelopathic because of cord compression at the adjacent levels. B, os odontoideum. Anteroposterior and dynamic lateral radiograph of the cervical spine (flexion view) demonstrating atlantoaxial instability due to os odontoideum.

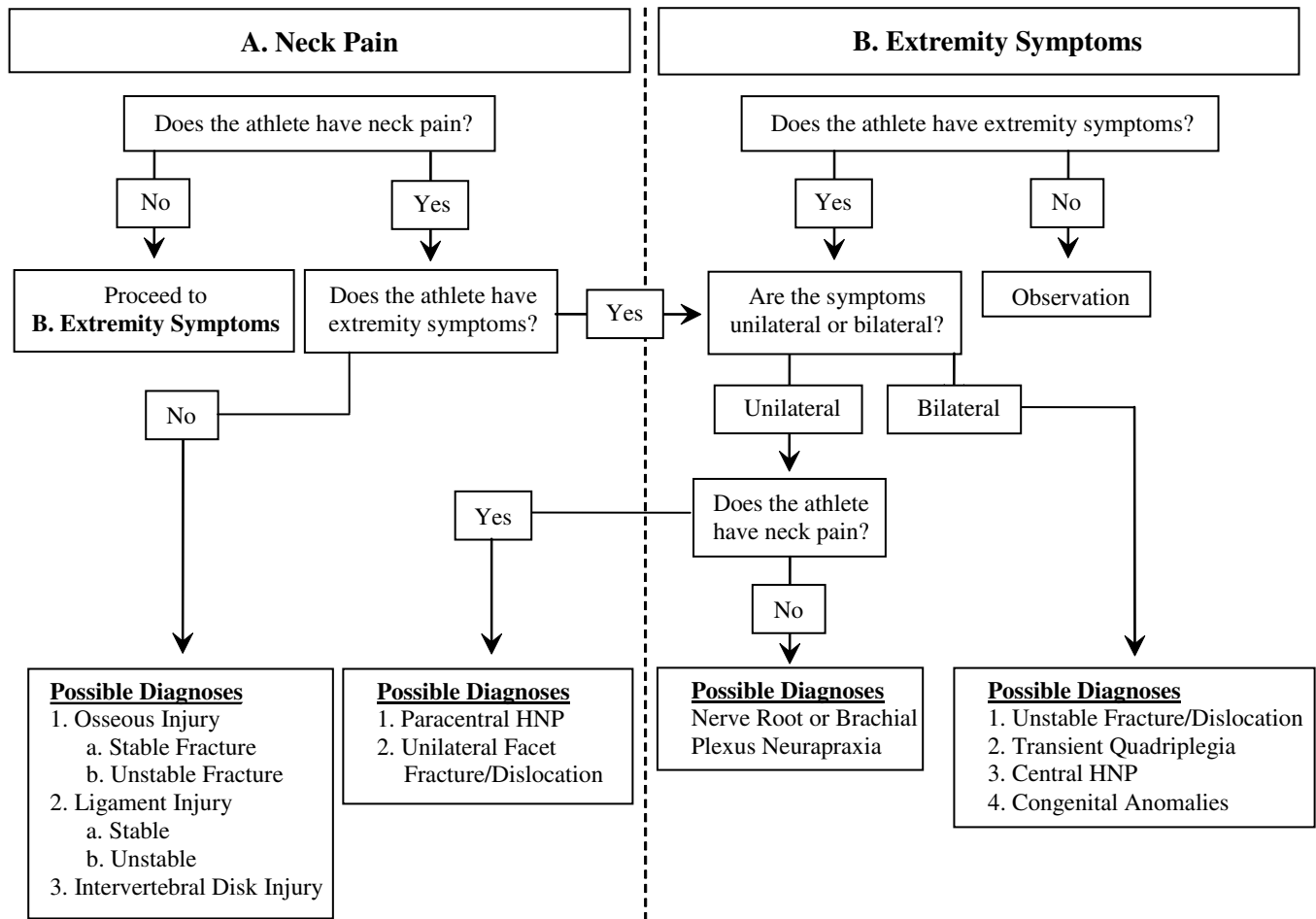


Figure 9. Algorithm for on-field evaluation of cervical spine injury. HNP, herniated nucleus pulposus.

tion with associated nerve root compression. The causative event, when identifiable, can be a high-energy impact to the head or even a minor twisting injury to the neck. The pathologic anatomy involves protrusion of a portion of the nucleus pulposus through a tear in the posterolateral aspect of the annulus fibrosus. Acute nuclear herniation is usually accompanied by the sudden onset of posterior neck pain and spasm. Compression and inflammation of the cervical root produce a monoradiculopathy characterized by radiating pain, paresthesias, and/or weakness in the upper extremity. The presence of a sensory, motor, and/or reflex deficit attributable to a specific nerve root allows for a presumptive determination of the affected disk level.²³

Symptoms and physical findings restricted to the neck can be caused by damage to the osseous structures, the spinal ligaments, or the intervertebral disk. The majority of these injuries are caused by a collision in which the energy of head impact is less than that required to destabilize the spinal column. Stable fractures of the anterior column usually occur in response to compression, whereas those of the posterior elements are typically owing to an extension injury vector. The athlete who sustains a spinal ligament injury (ie, cervical sprain) can also present with

complaints of pain localized to the neck without radiation into the extremities. When the ligamentous damage is minor, the stability of the spinal column is maintained and the neural elements are not placed at risk. A decrease in the player's active range of neck motion is common, and there may be focal tenderness to palpation over the posterior cervical spine. Last, an intervertebral disk injury can produce axial pain in the absence of radiculopathy or myelopathy. The pathologic anatomy usually involves an annular tear or a disk protrusion that does not result in neural element compression.

Figure 9 summarizes the diagnostic algorithm for the rapid identification of these clinical syndromes in the athlete with a potential catastrophic cervical spine injury.

SUMMARY

Catastrophic cervical spine injury can be an overwhelming event in a young athlete's life. Effective on-field and emergency department management of these injuries requires a concerted effort from the athletic team care providers. An improved knowledge and understanding of this compli-

cated problem can lead to improved prevention and emergent treatment strategies.

In these situations of actual or potential spinal cord injury, effective pre-event planning, early diagnosis, and adherence to strict management protocols can prevent secondary injury and worsening of the problem. This article has reviewed the current concepts on the epidemiology, functional anatomy, and diagnostic considerations in collision sport-related cervical spine trauma. We have provided a rational approach to the early recognition of the different clinical syndromes associated with this type of injury. Rapid on-field diagnosis can help to optimize the outcomes of these catastrophic injuries. The second article in this series will review the principles of on-field emergency care of the spine-injured collision athlete.

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