Cervical Spine Injuries in Football Players

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Abstract

Cervical spine injuries have been estimated to occur in 10% to 15% of football players, most commonly in linemen, defensive ends, and linebackers. The overwhelming majority of such injuries are self-limited, and full recovery can be expected. However, the presenting symptoms of serious cervical spine injuries may closely resemble those of minor injuries. The orthopaedic surgeon frequently must make a judgment, on the field or later in the office, about the advisability of returning the athlete to the game. These decisions can have an enormous impact on the player and his family. Most severe cervical spine injuries share the common mechanism of application of an axial load to the straightened spine. Avoiding techniques that employ head-down "spear" tackling and wearing properly fitted equipment markedly reduce the risk of serious injury.

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More than 1.2 million individuals participate annually in high school football. Another approximately 200,000 individuals engage in college and professional play each year.[1] It has been estimated that cervical spine injuries occur in 10% to 15% of football players, most commonly in linemen, defensive ends, and linebackers.[2-4] Injuries may involve structural elements of the spine (bones, disks, ligaments) and/or neural elements (brachial plexus, nerve roots, spinal cord). The overwhelming majority of such injuries are self-limited, and full recovery can be expected.[5] However, in one study 50% of college freshman football players with a history of previous "neck injury" demonstrated radiographic changes including compression fractures, neural arch fractures, and abnormal motion segments.[4] In a National Collegiate Athletic Association (NCAA) study of football-related injuries incurred between 1977 and 1989, 128 players suffered permanent spinal cord injury.[6] Vigilance is required to detect those injury patterns that require immediate evaluation and treatment or long-term protection.

Clinical Syndromes

Root and Brachial Plexus Neurapraxia

The most frequent cervical spine injury in football is neurapraxia of the nerve roots or brachial plexus. In one study,[7] half of the members of a collegiate football squad reported one or more such episodes during a regular season. Linemen, defensive ends, and linebackers are most commonly affected.[2,8] "Stingers" and "burners" are the lay terms applied to this spectrum of injuries. There is no agreement on the specific clinical definitions for these subjective entities, which lack discernible signs. Objective findings may be subtle. A careful examination is required to prevent attribution of a burning or stinging sensation to a benign condition when, in fact, it may be the result of a more serious problem. Such symptoms, when present in both upper extremities, suggest spinal cord, rather than nerve root or plexus, involvement.

The transient stinging and burning in neurapraxias arise from compressive or traction injuries to multiple roots or to the brachial plexus.[2,7] The upper trunk of the brachial plexus is tensioned by a sudden shoulder depression and concomitant lateral head flexion toward the unaffected side. With simultaneous head rotation toward the affected arm, the neural foramen narrows, compressing exiting nerve roots.

Neurapraxia may also be caused by direct compression of the brachial plexus. A poorly fitting, mobile shoulder pad may be pushed

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into Erb's point (in the anterolateral portion of the neck, 2 to 3 cm above the clavicle), compressing the brachial plexus between the shoulder pad and the superior medial scapula.[8,9]

The athlete may complain of a "dead arm" with shoulder and/or arm pain as well as transient, unilateral muscle paresis. Symptoms are self-limited. Burning pain resolves in seconds to minutes. Strength usually returns in 24 hours. A variable degree of weakness in the muscles innervated by the upper trunk of the brachial plexus may last for up to 6 weeks. Examination of the cervical spine demonstrates pain-free full range of motion with no tenderness or palpable deformity.[5] If symptoms resolve quickly and the neurologic examination is normal with full motor strength, the patient may return to the game. Persistence of symptoms or lack of a pain-free range of motion requires further evaluation, including cervical spine radiographs. Players should be restricted from further play until they have recovered full muscle strength.

Wearing a thermoplastic total-contact neck-shoulder-chest orthosis beneath a well-fitting shoulder pad decreases the severity and recurrence of compressive brachial plexus injuries.[8] A U-shaped foam neck roll may also be effective by limiting neck motion and preventing the shoulder pad from being forced into the neck. Stiff yet comfortable thick pads at the base of the neck provide support against extension and lateral bending.

Acute Cervical Sprain

Acute cervical sprain, which is in fact a ligamentous injury with potential for instability, is the result of a direct collision. The athlete complains of a "jammed neck" sensation with pain localized to the neck without radiation into the arms. Typically, there is decreased cervical motion. Reproducible focal tenderness is indicative of a significant bone or soft-tissue injury. No neurologic deficits are demonstrable on examination. Individuals with a history of a collision who have pain and limited range of motion should be placed in cervical immobilization.

The initial radiographic examination should include anteroposterior, lateral, and odontoid views of the cervical spine. Once the acute symptoms have subsided, flexion-extension lateral views should be obtained if the initial static radiographs were normal. In cases of continuing limitation of motion, pain, or radicular symptoms, magnetic resonance (MR) imaging or bone scintigraphy may be indicated.

In general, treatment should be tailored to the degree of severity of the injury. A collar and analgesic agents can be used until there is pain-free full range of motion.

Intervertebral Disk Lesions

Acute traumatic disk herniation with resultant cord compression can result in transient quadriplegia or permanent quadriparesis or quadriplegia.[10,11] Affected players experience acute paralysis of all four extremities and a loss of pain and temperature sensation. Magnetic resonance imaging or the combination of computed tomography (CT) and myelography can confirm the diagnosis. Anterior diskectomy with interbody fusion is warranted for a patient with persistent radicular pain or myelopathy.

Cervical spondylolytic changes without herniation or neurologic findings are frequent in football players. In one study,[4] 5 of 75 (7%) college freshman football players demonstrated an abnormally narrow disk space. Early degenerative changes can be attributed to repetitive loading in the preceding years of play. An MR imaging study may demonstrate a bulge without herniation. Treatment is usually nonsurgical with activity modification. Severe spondylolytic changes may cause (1) uncovertebral joint hypertrophy with narrowing of the neural foramen affecting the exiting nerve root; and (2) disk-osteophyte occlusion of the central canal (acquired cervical stenosis).

Transient Quadriplegia

Ladd and Scranton[11] and Torg et al[12] have separately described the clinical entity of "neurapraxia of the cervical cord" with transient quadriplegia after an axial load with hyperflexion or hyperextension (Fig. 1). During the 1984 NCAA season, neurapraxia of the cord was reported in 1.3/10,000 players.[12] The symptoms include bilateral burning pain, tingling, and loss of sensation in the arms and/or legs. Motor symptoms vary from mild weakness to complete paralysis. Episodes are transient, and complete recovery usually occurs within 10 to 15 minutes but may take as long as 48 hours. Radiographs are negative for fractures or dislocations (Fig. 2) but frequently

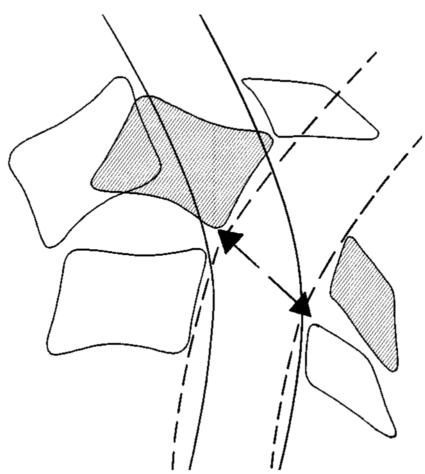

Fig. 1 Due to a pincer mechanism, injury to the cervical spinal cord may occur during extremes of flexion or extension. In hyperextension, the cord may be compressed between the posteroinferior portion of the vertebral body above and the anterosuperior lamina of the vertebra below.

show congenital stenosis, Klippel-Feil syndrome, or evidence of intervertebral disk disease or acquired stenosis.[12]

Maroon[13] has described the "burning hands" syndrome. This is believed to be a variant of the central cord syndrome. Edema and vascular insufficiency occur selectively within the medial aspect of the somatotopically arranged spinothalamic tracts.[13,14] Burning dysesthesias and paresthesias occur within a glovelike distribution, although strength, reflexes, and sensation are maintained. This clinical picture may be associated with a fracture-dislocation with or without a detectable radiographic abnormality.[14] In addition to plain radiography, MR imaging or postmyelography CT should be performed as part of the neural evaluation of all players who demonstrate the signs or symptoms of a cord injury.

Cord compression without residual radiographic abnormality may occur by means of a momentary pincerlike mechanism, originally described by Penning[15] (Fig. 1). When the cervical spine is in hyperextension, the cord is compressed between the posteroinferior margin of the superior vertebra and the anterosuperior lamina of the subjacent vertebra. In addition, infolding of the posterior longitudinal ligament and the ligamentum flavum contribute to central canal narrowing. With hyperflexion, a pinching effect is created between the lamina of the superior vertebra and the posterosuperior aspect of the subjacent vertebral body. Athletes with congenital or acquired cervical stenosis are predisposed to cord neurapraxia with hyperextension or hyperflexion loading.

To assess for congenital narrowing, the canal diameter is measured on a lateral radiograph from the midpoint of the posterior aspect of the vertebral body to the nearest point along the spinolaminar line (Fig. 3).[16] The normal midsagittal diameter is 14 to 23 mm. "Stenosis" is defined on the basis of a diameter of less than 13 mm. Variations in technique (e.g., use of different focus-to-film and object-to-film distances) and anatomy (e.g., variability in the triangular cross-sectional shape of the canal) often contribute to inaccurate measurements. To minimize these errors, Pavlov proposed using a ratio of the segmental

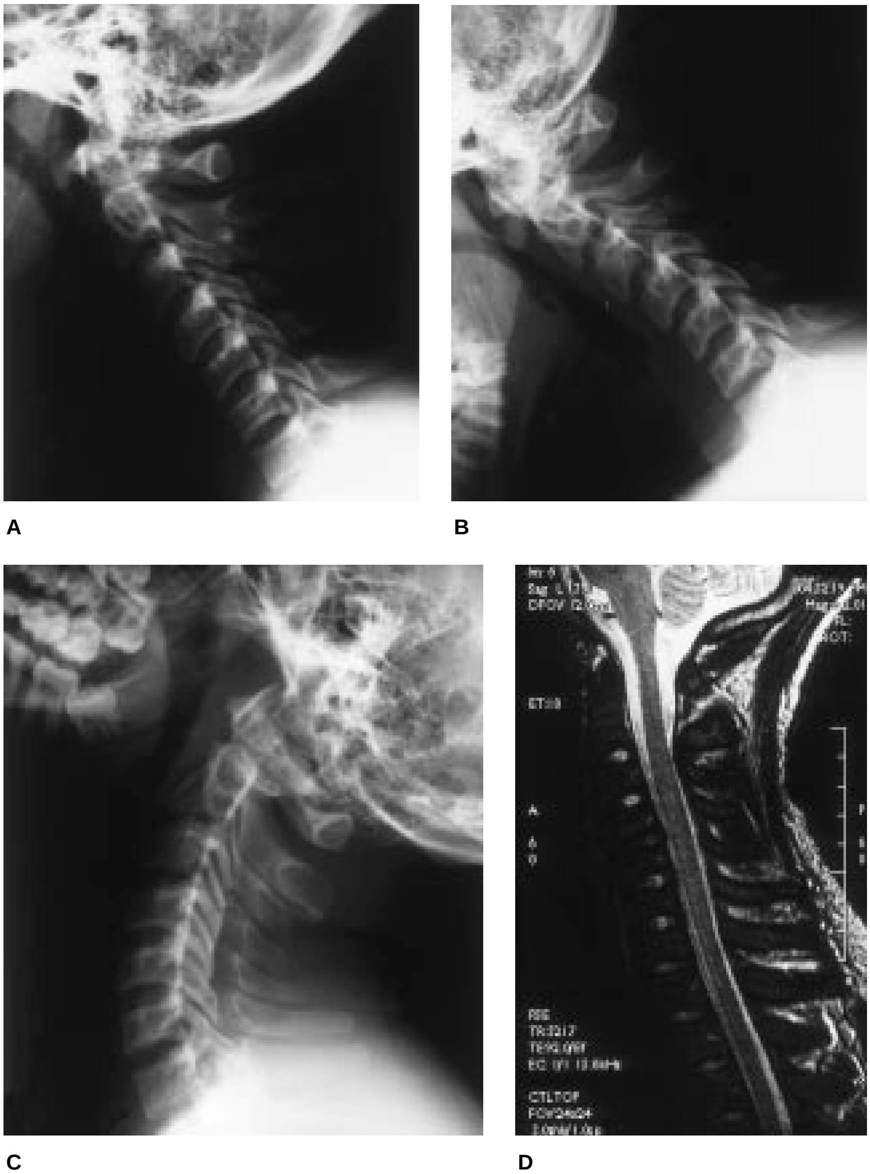

Fig. 2 A 19-year-old player received an axial load to the top of his helmet, which resulted in complete quadriplegia for approximately 10 minutes. All symptoms resolved rapidly and completely. Neutral lateral **(A)** and flexion **(B)** and extension **(C)** radiographs showed no abnormal soft-tissue swelling, no fractures or subluxations, and Pavlov ratios at C3 through C6 of 1.0. Sagittal MR imaging study **(D)** showed a disk-osteophyte complex at C6-7. No other degenerative changes, stenosis, or posterior ligamentous disruptions were noted. The spinal cord displayed no abnormal signal change. Subsequent flexion-extension radiographs showed no instability. The patient was allowed to participate in contact sports after demonstrating painless full range of motion.

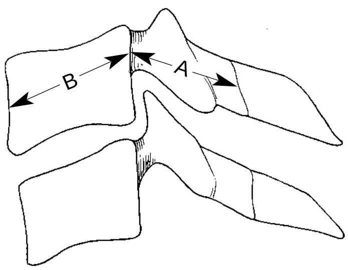

Fig. 3 The Pavlov ratio is calculated with the use of measurements on a lateral radiograph. The spinal canal is measured at its narrowest distance between the posterior aspect of the vertebral body and the most anterior point on the spinal laminar line. This distance (A) is divided by the width of the vertebral body (B).

sagittal diameter of the canal to the width of the vertebral body.[16] A ratio of less than 0.8 has been used to define a developmentally narrow canal. In one study,[17] that value was documented in 93% of players with transient quadriplegia, 12% of asymptomatic nonathletes, and 48% of asymptomatic football players.[17] A threefold increase in the incidence of stingers has also been seen among subjects with a ratio of less than 0.8, but this difference is considered to be secondary to foraminal, rather than central, stenosis.[2]

This ratio must be interpreted with caution, however, as some football players with relatively large vertebral bodies have a low ratio despite ample canal dimensions.[18] In addition, the ratio may be insensitive if the canal is narrow because of compression by soft-tissue elements (disk, ligamentum flavum). Thus, "stenosis" cannot be accurately diagnosed on the basis of bone measurements alone.

To clarify the risk to players with this entity, Torg et al[12,17] used data from the National Football Head and Neck Injury Registry to compare groups of males who had participated in tackle football with a control group of nonathletes. Players with cervical canal stenosis (as determined on the basis of a canal–vertebral body ratio of less than 0.8) were no more susceptible to neurologic injury than members of the general population (positive predictive value, 0.2%).[17] However, this study should be viewed with caution because of the previously discussed problems that may arise when the Torg ratio is used to define stenosis. A survey of 177 athletes who had been rendered quadriplegic by football-related accidents documented the absence of antecedent cord symptoms.[12] Therefore, screening with plain radiography to assess for stenosis in high school, college, or professional football players is not routinely recommended.[12,17,19]

There is a subset of players, however, in whom radiographs may be predictive of the risk of quadriplegia. These players have all regularly employed tackling techniques involving "spearing" (i.e., using the top of the helmet to intentionally ram an opponent). In addition, developmental stenosis, loss of the normal lordotic curve of the cervical spine, and posttraumatic abnormalities are all demonstrated radiographically. This dangerous constellation has been referred to as "spear tackler's spine" by Torg et al[20] and is an absolute contraindication to participation in football.

Congenital Anomalies

In general, the presence of cervical congenital anomalies alters the mechanical stability of the spine and greatly elevates the risk of severe cervical spine injury from minor trauma. There are two broad categories of congenital anomalies of the cervical spine: failure of segmentation and failure of formation.

Klippel-Feil syndrome encompasses a spectrum of failure of segmentation ranging from the absence of one motion segment to the absence of many motion segments. For the purposes of differentiating the risks to football players, Torg and Glasgow[19] have defined two types: type I, in which there is a long fusion mass, and type II, with only one or two fused segments. The more segments involved, the greater the loss of motion and the greater the stresses on adjacent normal segments; the ability of the cervical spine to absorb and dissipate loads is clearly diminished. In athletes with an atlanto-occipital congenital failure of segmentation, insidious compression of the posterior column of the spinal cord may develop at the posterior margin of the foramen magnum (Fig. 4).

Failure of formation leading to odontoid agenesis or hypoplasia and developmental os odontoideum can cause substantial atlantoaxial instability (Fig. 5). Spina bifida occulta is a failure of formation of the posterior arch. The spinal biomechanics in spina bifida are not typically or substantially altered. These conditions are frequently asymptomatic, and the diagnosis is made incidentally on examination of a radiograph obtained for other reasons.

Unstable Cervical Fractures and Dislocations

Although there has been much discussion about the influence of canal geometry on the risk of spinal cord injuries, there does not appear to be a direct relationship. In fact, most patients with football-related spinal cord injuries have had concomitant unstable fractures and dislocations. In a retrospective study of a collection of cases from the membership of the Congress of Neurological Surgeons, Schneider[21] found 78 severe cervical spine injuries that resulted in 16 deaths between 1959 and 1963. During the same interval, 69 cases of intracranial subdural hematoma resulted in 28 deaths. Surprisingly, well-outfitted professional athletes sustained a greater proportion of in-

A B

Fig. 4 A 38-year-old man with Klippel-Feil syndrome presented with transient quadriplegia, which resolved after 15 minutes. **A,** Lateral radiograph shows congenital failure of segmentation at C5-6 (Torg type II) with no acute fractures or subluxations. **B,** Sagittal T2-weighted MR image demonstrates signal change within the cord. Subsequent flexion-extension radiographs showed a stable spine. The patient was permanently restricted from contact sports.

juries compared with their "pickup"-play counterparts. It was evident that the plastic football helmets used at that time lacked sufficient resiliency for energy dissipation, prompting improvements in material and design.

Through the late 1960s and early 1970s, the incidence of severe head injuries decreased while the incidence of severe cervical spine injuries increased.[3] In a study of catastrophic spine injuries in football players in the period from 1977 through 1989, Cantu and Mueller[6] found that the act of tackling by defensive players was associated with the greatest risk of injuries resulting in quadriplegia. Most catastrophic events resulted from either a combined fracture-dislocation (33%) or an anterior compression fracture (22%).[6]

Since 1975, the National Football Head and Neck Injury Registry has prospectively gathered important epidemiologic information.[3] Through the analysis of injury reports, media clippings, medical records, video recordings, and radiographs, the predisposing factors and mechanisms of specific injury patterns have been elucidated. Needed modifications of rules and equipment have followed.

Improvements in helmet design and construction effectively decreased head injuries while encouraging playing techniques, such as spearing, that use the head as the point of contact, thus placing the cervical spine at substantial risk.[21] Axial loading of the cervical spine is the primary mechanism for severe neck injuries in football.[3,10] Between 1971 and 1975, 52% of the injuries resulting in permanent quadriplegia were attributed to spearing.[3]

The cervical spine can absorb much of the imparted energy of collisions by dissipation through the paravertebral musculature, the intervertebral disks, and the normal lordotic curve of the cervical spine. However, when the neck is flexed approximately 30 degrees, the normal lordotic curve is flattened, and forces applied to the top of the helmet are directed to a straight segmented column (Fig. 6).[3] In this situation, the cervical spine is less able to disperse the forces being exerted. With mounting axial load, compressive deformation occurs within the intervertebral disks, causing angular deformation and buckling. The spine fails in flexion with a resultant fracture, subluxation, or dislocation (Fig. 7).

Biomechanical studies replicating this proposed mechanism support this theory. Axial load to failure requires an average of 3,500 N (range, 645 to 7,439 N).[22] Less energy to failure under axial load is needed in straight spines than in those with a normal lordotic curve.[22] A direct vertex load imparts a larger force to the cervical spine than a force applied farther forward on the skull.

Although axial loading accounts for most fracture-dislocations, it does not account for all of the patterns seen. The combination of rotation and compression can produce a variety of recognized spinal injuries.[23] As a result of complex coupled motions, deformations occurring during impact may give rise to a number of different local mechanisms, including concomitant flexion, extension, rotational, and shear forces, within adjacent regions of the cervical spine.

As a result of the detailed analysis of the National Football Head and Neck Injury Registry,[3] two recommendations were made to the NCAA Football Rules Committee in February 1976: (1) No player

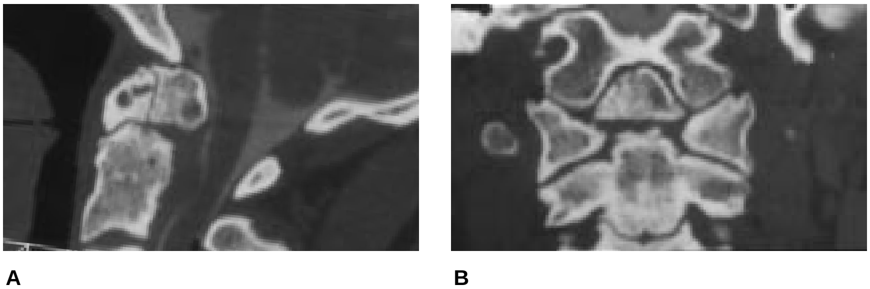

Fig. 5 A 26-year-old man presented with transient quadriplegia that lasted 15 minutes before gradual and complete resolution. Sagittal **(A)** and coronal **(B)** CT reconstructions demonstrate discontinuity of the dens with the C2 body. The dens–anterior ring of the C1 unit is posteriorly displaced with a sclerotic junction, which indicates its long-term presence. Soft-tissue swelling posterior to C2 displaces the cord. The patient was treated with a posterior C1-2 fusion and restricted from all participation in contact sports.

should intentionally strike an opponent with the crown or top of the helmet. (2) No player should deliberately use his helmet to butt or ram an opponent. Similar rules were later adopted by the National Football High School Athletic Association during the same year.

With implementation of these rules, a dramatic decrease was seen almost immediately in the rate of fractures, subluxations, and dislocations of the cervical spine in both high school and college athletes. The incidence of severe neck injury in college athletes decreased from 30/100,000 players in 1975 to 20/100,000 players in 1977.[3] The incidence of permanent quadriplegia also declined, from 5.3/100,000 players in 1975 to 1.6/100,000 players in 1977.[3,6] This beneficial trend has been sustained in recent years.[6,24] Overall, a 70% reduction in high school injuries and a 65% reduction in college injuries have been realized.[24]

Field Evaluation and Early Treatment

Initial involvement of the orthopaedic surgeon in the care of a football player with a cervical spine injury frequently begins on the field. Essential sideline equipment should include a spine board, a stretcher, and tools necessary to remove face masks from helmets and to perform cardiopulmonary resuscitation. Preparedness is paramount to timely, successful management.

It is necessary to remove the face mask for airway control of the unconscious athlete while simultaneously protecting the cervical spine. The type of mask determines the method of removal. The older double- and single-bar masks are removed with bolt cutters. Newer cage-type masks can be removed by cutting the plastic attachment loops with a scalpel or utility knife.[5] The chin strap and helmet are best left in place. The jaw thrust and chin lift are the safest ways of opening the airway in a patient with a suspected cervical injury. The head-tilt method is not considered safe.

Transportation to a medical facility is necessary for the player with altered mental status, neck pain or tenderness, limited cervical motion, and symptoms referable to a cord injury. The patient should be fully immobilized on a spine board with helmet and shoulder pads remaining in place. Marked alterations in the position of the cervical vertebrae can occur with either helmet or shoulder pad removal.[25,26] If desired, cervical radiographs can be obtained with all of the protective gear still in place. The helmet should be removed only when permanent immobilization in a con-

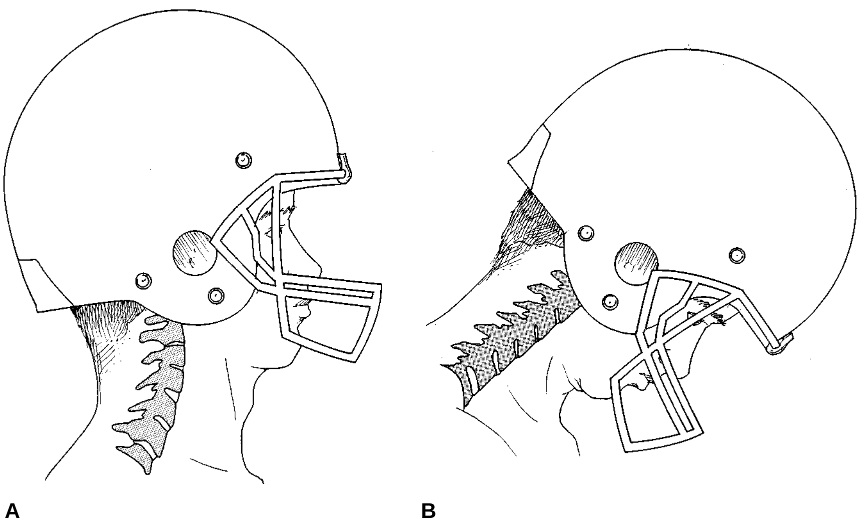

Fig. 6 **A,** Normal lordosis of the cervical spine. **B,** When the neck is flexed approximately 30 degrees, the cervical spine is straightened, assuming the configuration of a segmented column. (Adapted with permission from Torg JS, Vegso JJ, O'Neill MJ, Sennett B: The epidemiologic, pathologic, biomechanical, and cinematographic analysis of football-induced cervical spine trauma. *Am J Sports Med* 1990;18:50-57.)

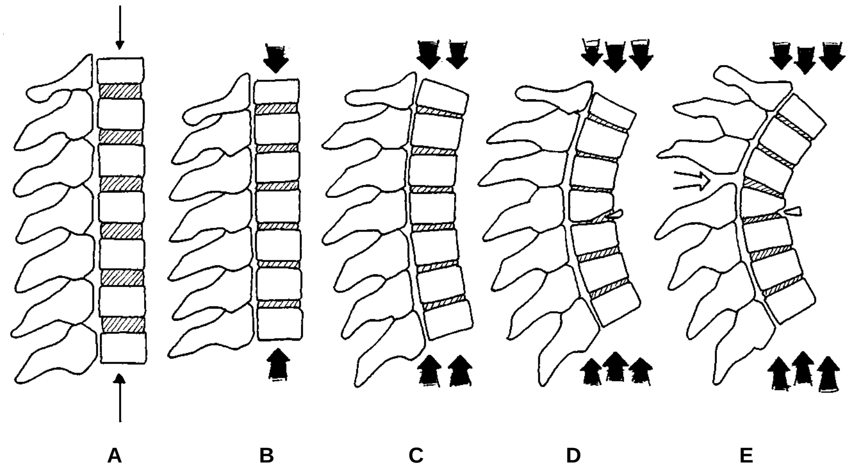

Fig. 7 Compared with normal lordotic posture, the straight segmented column is less able to dissipate the energy imparted during a substantial axial load. The sequence begins with compression of the intervertebral disks **(A, B)**. With continuing load, angular deformity occurs **(C)**. Fracture, subluxation, or dislocation follows **(D, E)**. (Adapted with permission from Torg JS, Vegso JJ, O'Neill MJ, Sennett B: The epidemiologic, pathologic, biomechanical, and cinematographic analysis of football-induced cervical spine trauma. *Am J Sports Med* 1990;18:50-57.)

trolled setting can be instituted. At that time, the chin strap should be detached, and the ear flaps of the helmet spread. The helmet is gently pulled in line with the cervical spine while the head is supported under the occiput.

Rehabilitation

Optimal head position, neck mobility, and paraspinal muscular strength are important factors for both playing performance and prevention of further injury. Proper rehabilitation is instrumental in recovery of range of motion, posture, and strength. The program begins with isometric contractions with the head maintained in the midline and resisting forces being applied perpendicular to the neck. Once the patient is pain-free with midline isometrics, a concentric resistive program, allowing increased arcs of motion against progressive loads, can begin. Advancement should be slow, avoiding the return of pain.

Stretching exercises should not be instituted acutely, as they may cause reactive paraspinal muscle spasm and stiffness. Gentle passive stretching, avoiding eccentric muscle loads by staying within the painless arc of motion, may begin after resolution of the acute inflammatory phase (usually within 72 hours). The pace of rehabilitation is dictated by the clinical recovery. When painless full range of motion has been obtained, eccentric muscle strengthening may commence.

Timing of Return to Play

The sideline evaluation of the ambulatory player is frequently a delicate matter. The desires of the coach, teammates, and cheering crowds should not unduly influence the team physician. The mechanism of the injury must be reconstructed in detail from information obtained from the player and observers. The player should be queried regarding the specific location of pain, numbness, tingling, or weakness, and the duration of these subjective symptoms should be recorded. A complete motor and sensory neurologic evaluation should then be performed.

A player with a stinger may return to play when the paresthesias resolve and full strength and painless full neck mobility are demonstrated.[5,27] It is essential that the athlete with anything less than pain-free full range of cervical motion must be protected with immobilization and excluded from further activity. Appropriate radiographs should be obtained expeditiously.

Acute cervical strains are treated with a collar and analgesic agents. If plain radiographs and flexion-extension lateral views are normal, the patient may return to football when there is pain-free normal range of motion and full motor strength. Proper rehabilitation is essential. However, comparative data gauging the "normal" neck paraspinal strength, endurance, and power required in football players are not yet available. Reinjury is always a possibility when the player returns to the field. At the high school level, a reinjury rate of 17% has been reported.[4]

Cervical disk herniations can have serious permanent neurologic complications. The decision to return to high-level play must be made carefully. A disk bulge without herniation as demonstrated by MR imaging, can be treated conservatively with activity modification. Return to play may occur when pain-free full range of motion is demonstrated and radicular symptoms are completely resolved. Symptomatic disk herniation with cord or root impingement may require anterior diskectomy with interbody fusion. A limited fusion (one or two levels) of the subaxial cervical spine is not considered a contraindication to future play if the segments above and below the fusion are normal.[27] A return to play cannot be recommended until there is radiographic evidence that the graft is well incorporated, the symptoms are completely resolved, and the player demonstrates a painless range of motion and full motor strength. Otherwise, contact sports are not recommended.

Watkins et al[9] created a rating scale to assess patients with transient quadriparesis and spinal canal stenosis for return to play. A score of 1 to 5 points can be assigned in each of three categories: extent of neurologic deficit, duration of symptoms, and degree of canal narrowing (Table 1). Those with a summary score of 6 points or less are considered to be at minimal risk; 6 to 10 points, moderate risk; and 10 to 15 points, severe risk. The authors stressed that this is only a guideline; each case must be considered individually.

The combination of congenital stenosis with instability, disk disease (bulge or herniation), degenerative change (osteophytes), MR imaging evidence of cord abnormality, neurologic findings lasting longer than 36 hours, or more than one recurrence is considered an absolute contraindication to sports participation.[27] With the exception of spear tackler's spine, there is no evidence that transient neurapraxia of the cord predisposes an individual to subsequent permanent quadriplegia or quadriparesis.[12] Congenital stenosis (Pavlov ratio less than 0.8) without instability is not considered a contraindication to play.[27] However, players and families should be thoroughly counseled regarding the specific condition and the potential risks.

Congenital anomalies of the upper cervical spine are an absolute contraindication to participation in all contact sports. This includes os odontoideum, odontoid hypoplasia or aplasia, and atlanto-occipital fusion, even if asymptomatic.[20,27] Torg type I Klippel-Feil deformity is also a contraindication to play. Players with type II anomalies associated with limited motion, occipitocervical abnormalities, or secondary instability as a result of degenerative changes should also be excluded. However, a type II deformity below C3 in an otherwise asymptomatic player is a relative contraindication.

Determining when a player can return to contact sports after an "unstable" injury can often be a difficult decision, as comprehensive guidelines are lacking. A detailed analysis of congenital, degenerative, and posttraumatic factors is recommended on a case-by-case basis.

Bailes et al[28] divided cervical injuries into three prognostic categories on the basis of their shared experience in treating 63 athletes with acute cervical injury. Type I injuries, which occurred in 58% of the cohort, involve a permanent spinal cord injury, most commonly at the C5 level. Also included within this group are minor neurologic deficits, spinal cord hemorrhage, contusion, and swelling demonstrated on MR imaging. Players with type I injuries should not return to contact sports.

Type II injuries, which occurred in 30% of the study group, are associated with transient symptoms referable to the cervical cord. The neurologic examination and radiographic studies are normal. There is no evidence of fracture, instability, or intrinsic cord lesion. This group includes those players with transient brachial plexopathy, burning hands syndrome, or transient quadriplegia. Return to play

Table 1
Cervical Spine Injury Rating Scale of Watkins et al[9]*

Criterion	Point Value†
Neurologic deficit	
Unilateral arm numbness or dysesthesia, loss of strength	1
Bilateral upper extremity loss of motor and sensory function	2
Loss of motor and sensory function in arm, leg, and trunk on one side of body	3
Transient quadriparesis	4
Transient quadriplegia	5
Duration of neurologic deficit	
Less than 5 minutes	1
Less than 1 hour	2
Less than 24 hours	3
Less than 1 week	4
More than 1 week	5
Central diameter of neural canal	
>12 mm	1
10-12 mm	2
10 mm	3
8-10 mm	4
8 mm	5

* Adapted with permission from Watkins RG, Dillin WH, Maxwell J: Cervical spine injuries in football players. *Spine State Art Rev* 1990;4:391-408.
† A total score for all three criteria of less than 6 points represents minimal risk; 6 to 10 points, moderate risk; 10 to 15 points, severe risk.

is acceptable if there is no residual neurologic deficit and no radiographic abnormality, including any congenital anomaly. Patients with recurrent injuries may be at higher risk and should be restricted from play.

Type III lesions are vertebral column injuries demonstrated only on radiographic imaging. The neurologic examination is normal. This is a heterogeneous group in which some patients may return to play and others should not. Those who have unstable fractures or dislocations that require bracing or surgery are restricted from further participation. Players with stable healed fractures (isolated lamina fractures, spinous process fractures, or minor injury of the vertebral body) should be evaluated with flexion-extension radiographs. Unfortunately, the direct data currently available are inadequate for use in determining whether a fracture is stable enough after treatment to allow a return to contact sports. Prospective use of this system has not been described.

If any fracture or unstable ligamentous injury of the upper cervical spine requires an atlantoaxial fusion, restriction from contact sports is necessary. Relative contraindications include healed nondisplaced Jefferson fractures, type I and type II odontoid fractures, and asymptomatic lateral-mass fractures.[27]

Subaxial injuries are assessed with use of the principles of stability described by White et al.[29] Combined disruption of anterior and posterior elements, more than 3.5 mm of horizontal segmental displacement, and more than 11 degrees of angulation difference between adjacent levels in the sagittal plane precludes further participation. Patients with healed, nontender, stable compression fractures; spinous process fractures; or endplate fractures without sagittal deformity may play. Residual pain, neurologic findings, and limited motion are always excluding factors. A limited fusion of the cervical spine is not considered a contraindication if the segments above and below the fusion are stable.[30]

Summary

Most cervical spine injuries in football players are self-limited. Both minor and severe injuries may present with nonspecific complaints. Most severe cervical spine injuries share the common mechanism of application of an axial load to the straightened spine. Avoiding techniques that employ head-down "spear" tackling and wearing properly fitting equipment substantially reduce the risk of serious injury. The return of the injured athlete to collision sports is a complex issue and needs to be evaluated carefully on an individual basis with consideration of the known principles of cervical spine stability.

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