

Cervical Spine Injuries in American Football

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Abstract

American football is a high-energy contact sport that places players at risk for cervical spine injuries with potential neurological deficits. Advances in tackling and blocking techniques, rules of the game and medical care of the athlete have been made throughout the past few decades to minimize the risk of cervical injury and improve the management of injuries that do occur. Nonetheless, cervical spine injuries remain a serious concern in the game of American football. Injuries have a wide spectrum of severity. The relatively common 'stinger' is a neuropraxia of a cervical nerve root(s) or brachial plexus and represents a reversible peripheral nerve injury. Less common and more serious an injury, cervical cord neuropraxia is the clinical manifestation of neuropraxia of the cervical spinal cord due to hyperextension, hyperflexion or axial loading. Recent data on American football suggest that approximately 0.2 per 100 000 participants at the high school level and 2 per 100 000 participants at the collegiate level are diagnosed with cervical cord neuropraxia. Characterized by temporary pain, paraesthesias and/or motor weakness in more than one extremity, there is a rapid and complete resolution of symptoms and a normal physical examination within 10 minutes to 48 hours after the initial injury. Stenosis of the spinal canal, whether congenital or acquired, is thought to predispose the athlete to cervical cord neuropraxia. Although quite rare, catastrophic neurological injury is a devastating entity referring to permanent neurological injury or death. The mechanism is most often a forced hyperflexion injury, as occurs when 'spear tackling'. The mean incidence of catastrophic neurological injury over the past 30 years has been approximately 0.5 per 100 000 participants at high school level and 1.5 per 100 000 at the collegiate level. This incidence has decreased significantly when compared with the incidence in the early 1970s. This decrease in the incidence of catastrophic injury is felt to be the result of changes in the rules in the mid-1970s that prohibited the use of the head as the initial contact point when blocking and tackling. Evaluation of patients with suspected cervical spine injury includes a complete neurological examination while on the field or the sidelines. Immobilization on a hard board may also be necessary. The decision to obtain radiographs can be made on the basis of the history and

physical examination. Treatment depends on severity of diagnosed injury and can range from an individualized cervical spine rehabilitation programme for a 'stinger' to cervical spine decompression and fusion for more serious bony or ligamentous injury. Still under constant debate is the decision to return to play for the athlete.

American football has long been recognized as an activity that places players at risk for cervical spine injuries with potential for neurological deficits. Cervical spine injuries affect players of all positions and levels of play, and can range in severity from a cervical strain with no neurological deficit to a catastrophic cervical fracture dislocation with a complete neurological deficit. Significant efforts have been made throughout the past few decades to minimize the risk of cervical injury and improve the management of injuries that do occur. These changes include changes in technique, changes in the rules of the game and improvements of the on- and off-field care of the injured athletes. Despite these efforts, however, cervical spine injuries remain a serious concern in the game of American football. The focus of this article is to review the demographics, mechanism, evaluation and treatment of cervical spine injuries with associated neurological deficit that occur in American football players. The injuries that will be covered include the 'stinger', cervical cord neuropraxia and catastrophic neurological injury.

A literature search was performed using PubMed. Our search period dated back to 1970. Searches included combinations of the following terms: 'cervical spine injury', 'American football', 'burners', 'stingers', 'neuropraxia', 'transient quadriplegia', 'spinal cord injury', 'catastrophic spine injury', 'diagnosis', 'management', 'treatment' and 'return to play'. Articles written about rugby and soccer players were excluded. Also excluded were articles written about the paediatric population. Athletes included high school through to professional level. Case reports were also excluded. The literature we used was chosen based on its utility in providing detailed explanations of the spectrum of injuries we wished to highlight.

1. Root/Brachial Plexus Neuropraxia

One of the most commonly occurring injuries in American football players is neuropraxia of the cervical nerve root(s) or brachial plexus, which is commonly referred to as a 'stinger'. This injury represents a reversible peripheral nerve injury of the upper extremity that results from a temporary physiological block in nerve conduction. It has been reported to occur in 50–65% of players over a 4-year collegiate career, and it most commonly occurs in linemen, defensive ends and linebackers.^[1] These 'stinger' or 'burner' injuries are characterized by unilateral burning pain radiating from the neck, down the arm to the hand. If the pain occurs in the bilateral upper extremities, further evaluation should be performed to rule out spinal cord injury.^[2] The pain usually lasts seconds to hours, and rarely beyond a 24-hour period. Players may experience associated weakness of the deltoid and/or spatati that typically resolves within 24 hours to 6 weeks following the injury.^[1] On physical examination, patients who have a stinger usually demonstrate pain-free active and passive range of motion of the neck and have no tenderness to palpation of the cervical spine or surrounding soft tissues.

Three mechanisms of injury have been described for the stinger. Injury can occur by compression of the cervical nerve root due to hyperextension, often with lateral flexion of the neck and an axial load. This can result in compression of the nerve root by narrowing of the intervertebral foramen. In addition, Penning^[3] described the 'pincer mechanism', in which there is some degree of spinal cord compression by the posterior-inferior margin of the superior vertebral body and the anterior-superior portion of the lamina of the vertebra below. The mechanism of compression of the cervical nerve root is often

suggested by a positive Spurling's test on physical examination.^[1] Meyer et al.^[4] reported that 85% of players with stingers had a mechanism of extension-compression. Levitz et al.^[5] echoed these results as 83% of stinger patients in their study suffered extension-compression injuries. These patients tend to have more severe symptoms and tend to be older, with radiographic evidence of cervical disk degeneration and spondylosis. From his study of 55 patients with recurrent burners, Levitz et al.^[5] reported that 53% had a narrowed cervical canal and 87% had evidence of degenerative disk disease. Another commonly reported mechanism of injury is that of traction or stretching of the brachial plexus due to depression of the ipsilateral arm and lateral bending of the head toward the unaffected side. As opposed to the pincer mechanism, these patients are often younger without prior cervical spine injury or cervical spondylosis.^[1] The Spurling's test is usually negative in these patients as the injury is not a result of nerve compression. Loss of cervical range of motion and neck pain is not characteristic of this mechanism of injury.^[6] The third mechanism of injury is that of direct trauma to the brachial plexus that results from the shoulder pads impinging on Erb's point, where the brachial plexus is most superficial.^[7]

Initial evaluation of patients with suspected nerve root or brachial plexus neuropraxia includes a complete neurological examination while on the field or the sidelines. Patients who have loss of consciousness, neck pain or any evidence of a neurological deficit on the field should have their cervical spine stabilized prior to transportation off the field. Once the player is removed from the field, a more thorough history and physical examination can be conducted, with attention given to the direction and mechanism of injury. Although cervical radiographs are often obtained in the setting of a stinger injury, the radiographs are usually normal.

Five to 10% of players with stinger symptoms have more serious injuries with prolonged neurological deficits.^[1] Patients with symptoms persisting beyond 2 weeks should be evaluated for the possibility of injury to the spinal cord. Anterior-posterior and flexion-extension radiographs

should be evaluated for fracture or gross instability. MRI evaluation serves to rule out significant ligamentous injury, disk herniation or cervical stenosis. There is some controversy as to the usefulness of electromyography (EMG), although this study may show a mild conduction block demonstrated by positive sharp waves and fibrillation potentials, demonstrating membrane instability due to axonal damage.^[8] The origin of injury can be identified with EMG as either the nerve root or the brachial plexus itself. Although Hershman et al.^[9] noted its limited utility, more contemporary management guidelines suggest EMG may have a role in determining return to play, as fibrillation potentials or moderate sharp waves in the presence of objective weakness indicate the need to refrain from participation in play.^[8]

Researchers have noticed a relationship between stingers and cervical stenosis. Meyer et al.^[4] reported that 47% of university football players with known stinger injuries had concomitant cervical stenosis. Patients with a congenitally narrow spinal canal have shortened pedicles and narrow intervertebral foramen. This foraminal narrowing is felt to increase the likelihood of nerve compression and cause a stinger injury.^[6] Cervical stenosis of the spinal canal can be identified using lateral radiographs. Measurement of the distance from the mid-point of the posterior aspect of the vertebral body to the nearest point on the spinolaminar line is considered the diameter of the spinal canal. Normal spinal canal diameter at the levels of C3–C7 ranges from 14 mm to 23 mm.^[10] Cervical stenosis is defined by a canal diameter of ≤ 13 mm.^[10,11]

In 1986 Torg et al.^[12] and in 1987 Pavlov et al.^[13] suggested the ratio method as a more accurate analysis of evaluation for canal stenosis that eliminates the variation and errors in measurement associated with simply measuring the diameter of the spinal canal. The ratio method measures the distance from the midpoint of the posterior aspect of the vertebral body to the nearest point on the spinolaminar line, divided by the sagittal diameter of the vertebral body. The normal ratio in asymptomatic controls was 1, while a ratio of ≤ 0.8 indicated cervical stenosis

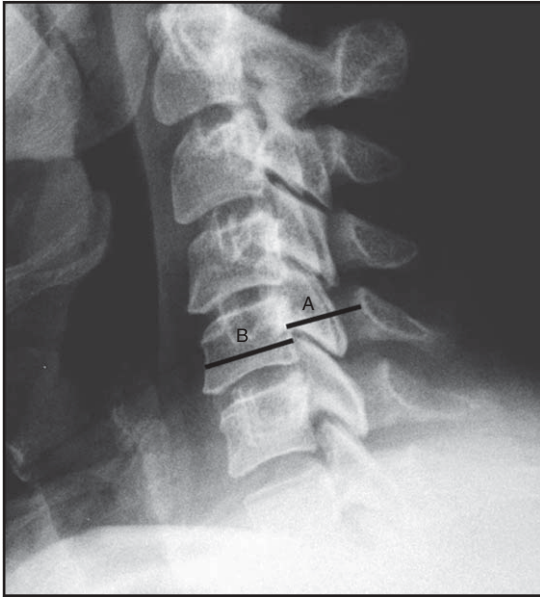


Fig. 1. A lateral cervical spine radiograph depicting the measurements for cervical spinal stenosis. Line 'A' indicates the anteroposterior dimension of the cervical spinal canal, measured from the posterior vertebral body to the spinolaminar line. Line 'B' indicates the anteroposterior width of the vertebral body. The Torg ratio is defined as A/B. Cervical spinal stenosis has been defined by a cervical spinal canal diameter of ≤ 13 mm or a Torg ratio of < 0.8 .

(figure 1).^[12,13] A reported limitation of the ratio method is that football players tend to have increased vertebral body size compared with the general population. This leads to a reduced Torg ratio despite an adequate canal diameter, thus over-reporting the incidence of cervical stenosis in this population.^[12-15]

Treatment of stinger injuries involves an individualized and comprehensive cervical spine rehabilitation programme that is sufficiently aggressive without causing further injury to the patient. The step-wise goals of therapy are to provide protection to the neck and injured nerves, control pain, correct imbalances in strength and flexibility, correct posture, re-condition the patient and prevent further or recurrent injury prior to return to play.^[16]

2. Cervical Cord Neuropraxia

Cervical cord neuropraxia, also referred to as transient quadriplegia, was first described in 1986

by Torg et al.^[12] as the clinical manifestation of neuropraxia of the cervical spinal cord due to hyperextension, hyperflexion or axial loading. It is characterized by temporary pain, paraesthesias and/or motor weakness in more than one extremity with a rapid and complete resolution of symptoms and a normal physical examination within 10 minutes to 48 hours after the initial injury. Torg et al.^[12] reported an incidence of cervical cord neuropraxia of 1.3 per 10 000 1984 National Collegiate Athletic Association (NCAA) football players with transient weakness with paraesthesias and an additional 6 per 10 000 players with transient paresthesias, totaling an estimated 7 per 10 000 players with signs and symptoms of cervical cord neuropraxia per year in this athletic population. Boden et al.^[17] more recently reported on 23 high school and 20 collegiate football players who sustained cervical cord neuropraxia between the years of 1989 and 2002. This translated into 3.3 cervical cord neuropraxia injuries per year and 0.17 cervical cord neuropraxia injuries per 100 000 participants at the high school level, and 1.5 per year and 2.05 per 100 000 participants at the collegiate level. The rate of cervical cord neuropraxia reported by Boden et al.^[17] is considerably lower than that documented by Torg et al.,^[12] which is likely reflective of rule changes that have been implemented in high school and collegiate football.

The mechanism of injury that typically results in cervical cord neuropraxia includes hyperextension or hyperflexion of the cervical spine resulting in a temporary physiological conduction block within the spinal cord. Torg et al.^[18] explain the temporary disruption of cervical spinal cord function as the result of local anoxia and an increase in intracellular calcium that results from cord compression at the time of the injury. Extension-compression injuries (i.e. Penning's^[3] 'pincer mechanism') are felt to be responsible for the most severe spinal cord compression in the anteroposterior direction. Hyperextension injuries may be further exacerbated by infolding of the ligamentum flavum, which can cause an additional 30% decrease in the anteroposterior diameter of the spinal canal.^[6,19] Hyperflexion injury is felt to result from impingement of the spinal

cord by the superior vertebra and the anterior superior lamina of the subjacent vertebra.^[2] The degree of compression depends on the sagittal diameter of the spinal canal, presence of any spondylotic changes and the degree of soft tissue hypertrophy and infolding.^[3,20]

Symptoms of neuropraxia include burning pain, tingling and loss of sensation in more than one extremity, with no associated neck pain other than a burning sensation. The motor changes range from no or partial weakness to complete paralysis.^[2,20] Frequently, however, motor and sensory symptoms will coexist in patients. Symptoms are completely transient and resolve within 10 minutes to 48 hours following the injury.^[6,12,17] The duration of symptoms in the study of Boden et al.^[17] was documented in 12 of the 43 athletes with cervical cord neuropraxia and included the following: <15 minutes in five patients, between 15 minutes and 24 hours in five patients and >24 hours in two patients.

The evaluation of patients with cervical cord neuropraxia includes a complete history and physical examination with particular attention given to the neurological examination and the mechanism of injury, including the direction of the traumatic force. Anteroposterior, lateral and open mouth odontoid radiographs are performed initially to rule out any obvious fracture or dislocation. In the absence of neck pain, flexion and extension cervical radiographs are obtained to rule out instability. If the patient does have neck pain, flexion and extension radiographs should not be performed until a cervical MRI rules out any ligamentous injury. Radiographic findings of fracture or dislocation are usually absent in cases of cervical cord neuropraxia. Often seen on radiographs, however, are signs of congenital cervical stenosis and congenital abnormalities such as Klippel-Feil syndrome.^[2] Furthermore, intervertebral disk disease, acquired cervical stenosis and/or cervical instability (defined as >3.4 mm of anterior-posterior translation or angulation of 11° between adjacent vertebrae on a lateral view) are relatively common radiographic findings in patients with neuropraxia.^[20,21] The mechanism of injury that causes cervical cord neuropraxia is usually the 'pincer mechanism', in

which hyperextension with an associated axial load to the cervical spine results in spinal cord compression. MRI allows a more thorough evaluation of the discoligamentous complex, the spinal cord itself and the cerebrospinal fluid surrounding the spinal cord. The cerebrospinal fluid space has been referred to as the functional reserve of the spinal canal, and the absence of this space is felt to place the spinal cord at risk for injury in the event of a trauma.^[10,22,23] MRI or CT myelogram must be performed in any patient demonstrating an abnormal neurological examination, signs or symptoms of neuropraxia, or translation >3 mm on x-ray. These findings are suggestive of intrinsic or extrinsic cord compression, nerve root compression and/or ligamentous injury.

Stenosis of the spinal canal, whether congenital or acquired, is thought to predispose the athlete to cervical cord neuropraxia.^[12,14] In 1996, Torg et al.^[14] published an analysis of 45 football players who suffered from cervical cord neuropraxia. This analysis included cohorts of asymptomatic football players, asymptomatic non-athlete males, football players who had an episode of cervical cord neuropraxia and football players who sustained cervical injuries that resulted in permanent quadriplegia. These authors reported that 93% of football players with cervical cord neuropraxia had a Torg ratio of <0.8.^[14] The notion that football players have increased vertebral body size compared with the general population, however, suggests that the Torg ratio may overestimate the presence of significant spinal stenosis in this population.^[14] This notion is supported by Odor et al.^[24] and Herzog et al.,^[25] who reported that 34% and 49% of asymptomatic professional football players, respectively, had a Torg ratio of <0.8. Torg et al.^[14] reported that 48% of asymptomatic football players and only 12% of asymptomatic non-athletes had Torg ratios of <0.8 and that the positive predictive value of football players with a Torg ratio of <0.8 for sustaining cervical cord neuropraxia is only 0.2%. It is, therefore, not recommended that the Torg ratio be used as a factor when determining whether or not an athlete should be allowed to participate in football.^[14]

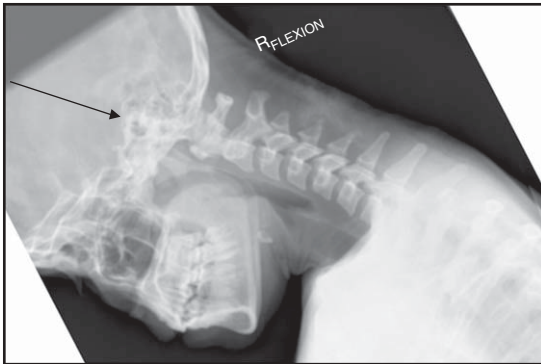


Fig. 2. A flexion lateral cervical radiograph demonstrating the mechanism of cervical injury that occurs with spear tackling. When the neck is flexed, as is the case when a player spear tackles, contact is initiated with the crown of the helmet. With the neck in a flexed position, the alignment of the cervical spine is straight to slightly kyphotic, as demonstrated in the lateral cervical radiograph. Almost the entire applied load (black arrow) is axially applied to the cervical spinal column. When this load is excessive, the cervical spinal column fails in a flexion and compression (see figure 3).

3. Catastrophic Neurological Injury

It has been estimated that each year approximately 11 000 neck injuries that are sustained while playing American football present to emergency departments in the US.^[26] Fortunately, permanent neurological injury and death as a result of these injuries are infrequent.^[27,28] Athletic activity is the fourth most common overall cause of spinal cord injuries after motor vehicle accidents, violent crime and falls, and the second most common cause in the first three decades of life.^[29-31] The level of risk is particularly high in contact sports like American football and hockey or high-energy sports such as gymnastics.^[8]

In American football, the mechanism of catastrophic cervical injury is most often a forced hyperflexion injury, as occurs when ‘spear tackling’.^[17,28,32] Spear tackling refers to a technique of tackling or blocking in which the player initiates contact with the crown of the helmet, with the neck in a slightly flexed position (figure 2). Axial force applied to the helmet is transmitted to the cervical spine, and the subaxial spine fails in flexion in the form of a fracture and/or subluxation or dislocation (figure 3). Although upper cervical spine injuries (i.e. occiput to C2) have been reported to occur in football and result in per-

manent quadriplegia, they are not as common as injuries of the subaxial cervical spine.^[17,28,33,34] Combined injuries of the upper cervical and subaxial cervical spine have also been reported.^[17] This must be considered when evaluating the patient radiographically. The majority of cervical cord injuries occur during actual games and are most common in defensive players.^[14,17,28,32,34] Over the last 30 years, defensive players have sustained 70.3% of the 269 permanent neurological injuries reported.^[28,34] Defensive back is the position at highest risk of injury, representing 35.3% of all spinal cord lesions reported, followed by linebackers (9.7%) and special teams players (8.2%). The overwhelming majority of the injuries occur while tackling or blocking.^[28,34]

Since the first intercollegiate American football game between Princeton and Rutgers in 1869, repeated attempts have been made to decrease



Fig. 3. Lateral radiograph of a 21-year-old collegiate defensive end who sustained a cervical injury when making a tackle. This represents a flexion-compression injury at the C4-5 level, with compression of the C5 vertebral body and a bilateral C4-5 facet dislocation. He underwent an anterior C5 corpectomy and fusion and a posterior spinal fusion from C4 to C6 within 24 hours of the injury.

the frequency of death and catastrophic injury sustained in the course of play.^[35] In 1964, Schneider^[36] reported that 56 cervical fractures or dislocations occurred during the years 1959–63 (1.4/100 000), 30 of which resulted in permanent quadriplegia (0.73/100 000). The National Collegiate Athletic Association and the National Federation of State High School Associations have collected injury data since the 1970s, which has been reported as part of the Annual Survey of Football Injury Research since 1980.^[30] The primary purpose of these efforts was to make the game of football safer for the participants.

In 1975, the National Football Head and Neck Injury Registry was established in order to document the extent of head and neck injuries. In 1979, Torg et al.^[37] retrospectively gathered information dating back to 1975 and found an increase in the number of players rendered quadriplegic.^[37] The cause for this increase was attributed to better protective capabilities of modern helmets. These helmets decreased the incidence of head injuries, but also led to the use of the helmet as a primary point of contact in blocking, tackling and head butting, placing the cervical spine at risk (i.e. spear tackling).^[37] Fifty-two percent of the cervical injuries with associated permanent quadriplegia were the result of spear tackling or direct force applied to the helmet.^[37] Although hyperflexion^[36,38,39] and hyperextension^[40,41] have traditionally been implicated in the development of cervical spine injuries, pathological, biomechanical and cinematographic analyses have determined that axial loading is the primary mechanism of football related injuries.^[42,27]

As a consequence of these findings, the National Collegiate Athletic Association and the National Federation of State High School Associations implemented major rule changes in 1976 prohibiting the use of the head as the initial contact point when blocking and tackling; these rules were later supported by the American Football Coaches Association Ethics Committee.^[43] These changes had a dramatic effect on the incidence of permanent cervical quadriplegia. According to the National Football Head and Neck Injury Registry^[27,37,44] in 1975, prior to the

rules change, there were a total of 89 cervical fractures or dislocations (6.65/100 000 and 29.3/100 000 at the high school and college levels, respectively) resulting in 28 cases of quadriplegia (2.1/100 000 and 8.0/100 000 at the high school and college levels, respectively). By 1987, the number of cervical fractures and dislocations had fallen to 22 (2.31/100 000) at high school and eight (10.66/100 000) at college level. The number of cases of permanent quadriplegia had fallen to eight (0.73/100 000) at the high school level and none at the collegiate level.^[44,27]

The most recent report from The National Center for Catastrophic Sport Injury Research and the Annual Survey of Catastrophic Football Injuries show that football has been responsible for 269 permanent cervical spinal cord injuries since 1977.^[28,34] High school players represent the vast majority of these injuries accounting for 222 cases, college players for 33, recreational players for 5, and professional players for only 9.^[28,34] When per-participant incidence is considered, however, there is an increase in incidence with the level of player. These data also showed a dramatic decrease in the number of injuries since the reports from the 1960s and early 1970s.^[28,34] During the 1990s, the average figure was 6.9 injuries per year and a mean incidence over the last 30 years of 0.52/100 000 participants at high school level and 1.47/100 000 at college level.^[28,34] Nevertheless, in four of the last eight seasons, the number of permanent spinal cord lesions has surpassed ten cases.^[28,34] The incidence in 2006 is above average, especially at the collegiate level (i.e. 2.66/100 000).^[28,34] Recent changes in the rules have been implemented to hopefully decrease the incidence of catastrophic injury. In 2005 the word 'intentional' was dropped from the spearing rule, so that now no forms of spearing, intentional or not, are permitted. Additional rule changes effective from 2006 and 2007 include the requirement of at least a 4-point chinstrap to secure the helmet, coloured mouth guards and revision of illegal helmet contact rules.^[28,34] Continuous surveillance of sustained injuries will help us to realize the impact of these recent rule changes on the incidence of catastrophic cervical spine injuries.

4. Early Evaluation and Management

Evaluation and management of a football player with a cervical spine injury begins on the field. In fact, there should be extensive pre-event planning and preparations for dealing with these types of injuries. A standard protocol for pre-hospital care, proper organization of equipment and a trained group of personnel are essential to ensure the best care for the injured athlete. Basic sideline equipment should always include a spine-board, stretcher, tools necessary to remove protective gear and maintain cervical spine immobilization, and items for airway management and cardiopulmonary resuscitation. In addition, efficient communication with the emergency room will optimize care.^[45]

As with any traumatic injury, the primary objective in early management is to address any life-threatening conditions and to prevent further injury. The ABCDE approach should be utilized. Airway is first assessed while maintaining cervical spine stability. Breathing and ventilation are next assessed, following by circulation and disability (neurological status). Lastly, the athlete should be exposed for the secondary survey (although this usually doesn't occur until the player is in the emergency room). Findings during the primary survey should be addressed and appropriate resuscitation efforts should be made. During the on-field evaluation, the athlete's helmet and shoulder pads should remain in place with immobilization of the cervical spine.^[46] In fact, the helmet and shoulder pads serve to provide support and alignment to the injured cervical spine.^[45] It may be necessary to remove the face mask for airway control. This should be accomplished using the proper face mask shears or bolt cutters. A quick on-field history and examination should reveal if the athlete is experiencing unilateral or bilateral arm pain, neck pain, weakness or paraesthesias. Transportation to a medical facility is required for the player with an altered mental status, neck pain or tenderness, limited cervical motion or any neurological symptoms suggesting a spinal cord injury.^[2] The helmet and shoulder pads should only be removed once the patient is in a controlled setting

and in the hands of personnel trained in such procedures.

Once the athlete's care has been transferred from the on-field team to the emergency room medical staff, the primary and secondary survey will be repeated, including a full neurological examination. At this time, cervical antero-posterior, lateral and odontoid radiographs should be obtained. If plain radiographs are inadequate (i.e. the C7-T1 level cannot be visualized), then a cervical CT should be performed. If warranted, a consultation with a spine surgeon should be quickly obtained. Any signs or symptoms suggestive of a spinal cord injury warrant a cervical MRI.

Treatment of cervical spine injury depends on the spectrum of symptoms and the presence of fracture, dislocation, ligamentous injury or spinal cord injury. Although athletes with unstable cervical spine injuries require surgical treatment, the majority of athletes suffer from stable cervical injuries that can be treated conservatively, with rehabilitation. A thorough rehabilitation programme allows for restoration of motion, posture, strength and the prevention of further injury. Most programmes follow a protocol starting with isometric exercises. Forces applied to the head without motion are followed by concentric resistive programmes, gradually allowing greater degrees of neck motion. Progression should be slow and extra care should be taken to avoid the return of pain. Stretching exercises should be avoided in the early inflammatory phase, as they may exacerbate muscle spasm and stiffness. Only after a full painless arc of motion is attained should the athlete partake in eccentric muscle strengthening.^[2]

Long a controversial topic, the use of glucocorticosteroids in the treatment of acute spinal cord injury is guided by the National Acute Spinal Cord Injury Study (NASCIS) I and II in 1985 and 1990, respectively.^[47,48] Although NASCIS I showed no improvement, analysis of NASCIS II using a higher dose of methylprednisolone (i.e. 30 mg/kg IV over 15 minutes, followed by a 45-minute rest and then 5.4 mg/kg/h for 23 hours) showed that the group receiving methylprednisolone within 8 hours of injury had

significant neurological score improvement. Interestingly, methylprednisolone given after 8 hours of injury was actually found to cause significant worsening of neurological score. The authors also conceded that the improvements in neurological score did not equate with improvements in functional status. Additionally, patients with coexisting life-threatening injuries and spinal cord injuries secondary to gun-shot wounds were excluded. NASCIS I showed increased rate of infection with corticosteroids whereas NASCIS II did not.

Bracken et al.,^[49] in a more recent study, showed that patients who receive high-dose corticosteroids between 3 and 8 hours after injury had improved neurological outcomes when treated with 48 hours of corticosteroids rather than 24 hours. This group of patients had higher rates of severe sepsis and severe pneumonia, but otherwise did not show increased complications or mortality. The authors concluded that patients with acute spinal cord injury who present within 3 hours of their injury should be treated with the high-dose methylprednisolone regimen (i.e. 30 mg/kg loading dose followed by 5.4 mg/kg/h) for 24 hours. Patients who present between 3 and 8 hours after their spinal cord injury should be maintained on the high-dose corticosteroid regimen for a total of 48 hours. Administration of high-dose corticosteroids is not indicated in the patient with life-threatening co-morbidity and may be harmful if given after 8 hours of injury.^[49]

The results of the NASCIS studies and the efficacy of methylprednisolone as a neuroprotective agent following acute spinal cord injury have been heavily debated over the years. Many authors feel as though high-dose methylprednisolone causes more harm than good and that it should not be administered in the setting of acute spinal cord injury.^[50-52] This remains a controversial topic, although many spinal cord injury centres continue to treat acute spinal cord injury patients according to the above protocol.^[53]

5. Return to Play

Despite significant efforts to develop guidelines for return to play for the spectrum of cervi-

cal spine injury, the issue of return to play remains controversial. Currently, no set of guidelines for return to play is agreed upon. This issue is often complicated by extrinsic pressures placed on the physician from coaches, players, families and other involved parties. Players with injuries desiring to return to play must be evaluated thoroughly to minimize the risk of recurrent injury. Evaluation includes a detailed history and physical examination and a complete neurological examination. The patient must be able to demonstrate a full, painless cervical range of motion and have no evidence of neurological deficit prior to returning to play.^[20] The decision of when, if at all, to return an athlete to contact sports is often difficult. This decision should be based on the mechanism of injury, objective findings by clinical examination and radiographic evaluation, extent of treatment required (i.e. surgical vs nonsurgical) and the ability of the athlete to successfully complete a comprehensive rehabilitation programme.^[54]

Traditionally, the athlete sustaining a stinger may return to play when the paraesthesias resolve and full strength and painless full range of neck motion are appreciated.^[2] It is essential that there is no pain in the neck with motion. If this criterion is not met, the athlete should be immobilized and excluded from activity until radiographs and possibly an MRI are obtained and a definitive diagnosis is reached. After return to play, properly fitting protective equipment can help prevent recurrent injury.^[42] The risk of sustaining a recurrent compressive injury can be minimized by wearing a thermoplastic total contact neck-shoulder-chest orthosis under properly fitting shoulder pads. Although it doesn't protect against a compressive injury, a U-shaped neck roll can protect the neck by limiting extreme range-of-motion and preventing hyperextension and excessive lateral bending.^[2]

Return to contact sports following an episode of cervical cord neuropraxia is a highly debated issue. Boden et al.^[17] reported on 76 athletes who sustained permanent quadriplegia playing football. Forty-six were available for questioning, 38 of whom reported no previous injury and eight of whom reported a previous stinger. None of the

athletes reported a previous episode of cervical cord neuropraxia. Of the 43 players in the series of Boden et al.^[17] who sustained cervical cord neuropraxia, two reported a prior episode of cervical cord neuropraxia and two reported a prior stinger. Torg et al.^[14] surveyed 77 athletes who had sustained permanent quadriplegia playing football, none of whom reported a previous episode of cervical cord neuropraxia. These authors also studied 45 athletes who had sustained an episode of cervical cord neuropraxia, none of whom had a subsequent injury resulting in permanent neurological deficit. In another study, Torg et al.^[55] reported on 110 athletes who sustained cervical cord neuropraxia, 87% of which occurred playing football, with 105 of these athletes available for an average follow-up of 3.3 years. No permanent neurological deficit occurred as a result of their episode of cervical cord neuropraxia, and no permanent neurological deficit occurred subsequently in those who returned to contact sports. Of those that returned to contact sports, however, 56% had a recurrent episode of cervical cord neuropraxia. Decreased space for the cervical spinal cord (i.e. cervical stenosis) strongly correlated with the risk of experiencing a recurrent episode of cervical cord neuropraxia. These data suggest that an episode of cervical cord neuropraxia does not increase the risk of sustaining subsequent permanent quadriplegia, but does increase the risk of having subsequent episodes of cervical cord neuropraxia with return to contact sports, particularly in athletes with cervical stenosis. For this reason, players at an elevated level of play (professional) who have an episode of cervical cord neuropraxia are often permitted to return to contact sports, but warned of the increased risk of recurrence.

Although return to play following cervical cord neuropraxia is controversial, many agree that relative contraindications to return to play include recurrent episodes of cervical cord neuropraxia, an episode of cervical cord neuropraxia with symptoms lasting >24 hours and cervical cord neuropraxia with associated congenital or acquired spinal stenosis.^[20,54,56-58] An absolute contraindication to return to play is an episode of cervical cord neuropraxia that is associated with congenital spinal anomalies (e.g. Klippel-Feil), spinal in-

stability, ligamentous injury, persistent neck pain or loss of motion and/or oedema in the spinal cord.^[20,54,56-58] In general, congenital anomalies of the upper cervical spine, including os odontoidum, odontoid hypoplasia or aplasia and atlanto-occipital fusion, are an absolute contraindication to participation in contact sports.

Bailes et al.^[59] created three prognostic categories based on characteristics of the cervical spine injury. Type I injuries included those with permanent spinal cord injury, cord haemorrhage, cord contusion or swelling on MRI. These players are not to return to contact sports. Type II injuries included transient neurological symptoms referable to the cervical cord. Neurological examination and radiographs are normal. Diagnoses in this group include brachial plexopathy, burning hands syndrome and cervical cord neuropraxia. As long as there are no neurological deficits or radiographic abnormalities, players may return to play. Type III injuries include those vertebral column injuries only demonstrated on radiographs in a patient with a normal neurological examination. Unstable fractures or dislocations requiring bracing or surgery should not return to play. Any player with an injury requiring atlantoaxial fusion is restricted from play. White et al.^[21] devised a set of principles to follow for subaxial injuries. If any of the following criteria are met, the player's cervical spine can be considered unstable and may require surgical stabilization: combined disruption of the anterior and posterior elements, 3.5 mm of horizontal segment displacement or more than 11° of angulation between adjacent levels. Patients with non-tender, healed, stable compression fractures or spinous process fractures may return to play. As with all injuries, any neurological examination abnormalities or painful motion should restrict an athlete from return to play.

An athlete who sustains a disk herniation may require an anterior disectomy with interbody fusion. If limited to one or two levels, this operation is not a contraindication to eventual return to play given that the segments above and below the fusion are normal.^[58] The athlete can return to play when symptoms have resolved, the graft is radiographically well incorporated and the athlete exhibits a full painless range of neck motion

and full strength. Although surgical options provide the opportunity to return to sport in patients with focal disease, Maroon et al.^[23] have reported a 25% increase in adjacent segment disk herniation within 10 years following a single level fusion.

6. Summary

American football is a high-energy contact sport that places participants at risk of a variety of injuries, including those of the cervical spine. Injuries to the cervical spine range from a simple sprain to a catastrophic injury with permanent quadriplegia. Over the last three decades, improved understanding of injury mechanism and subsequent rule changes have led to an overall decrease in the number of annual catastrophic cervical spine injuries sustained playing football. Advances in on- and off-field evaluation and management, rehabilitation strategies and return-to-play guidelines have improved the care of athletes that sustain cervical injuries. Continued surveillance of cervical injuries in football and other contact sports will hopefully lead to further improvements in preventative strategies.

Acknowledgements

No sources of funding were used in the preparation of this review, and the authors have no conflicts of interest that are directly relevant to the content of this review.

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