SPINAL TRAUMA

Chapter 10

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Introduction

Combat casualty care (CCC) providers play a vital role in the diagnosis and initial management of acute spinal injury patients. Combat casualty care frequently presents patient care challenges that are beyond the scope of Advanced Trauma Life Support (ATLS) training.[1] Failure to recognize and appropriately manage unstable spinal injuries may result in the development or the progression of neurologic injury.[2] The optimal management of spinal injuries is dictated by a CCC provider's skill set, support staff, and access to equipment necessary to achieve desired outcomes.

Two broad categories of spinal trauma on the battlefield are penetrating and blunt injuries. Penetrating spinal injuries in combat are most often the result of gunshot wounds or fragmentation injuries from blast mechanisms.[3,4,5] Blunt trauma can result from falls, tertiary injury from blasts, combat vehicle collisions, and numerous other mechanisms. The early identification and management of open spinal injuries are critical. A direct path from the skin through the dura defines an open spinal injury. All spinal injuries associated with penetrating trauma should be deemed open spinal injuries, until proven otherwise (Fig. 1). Conversely, blunt

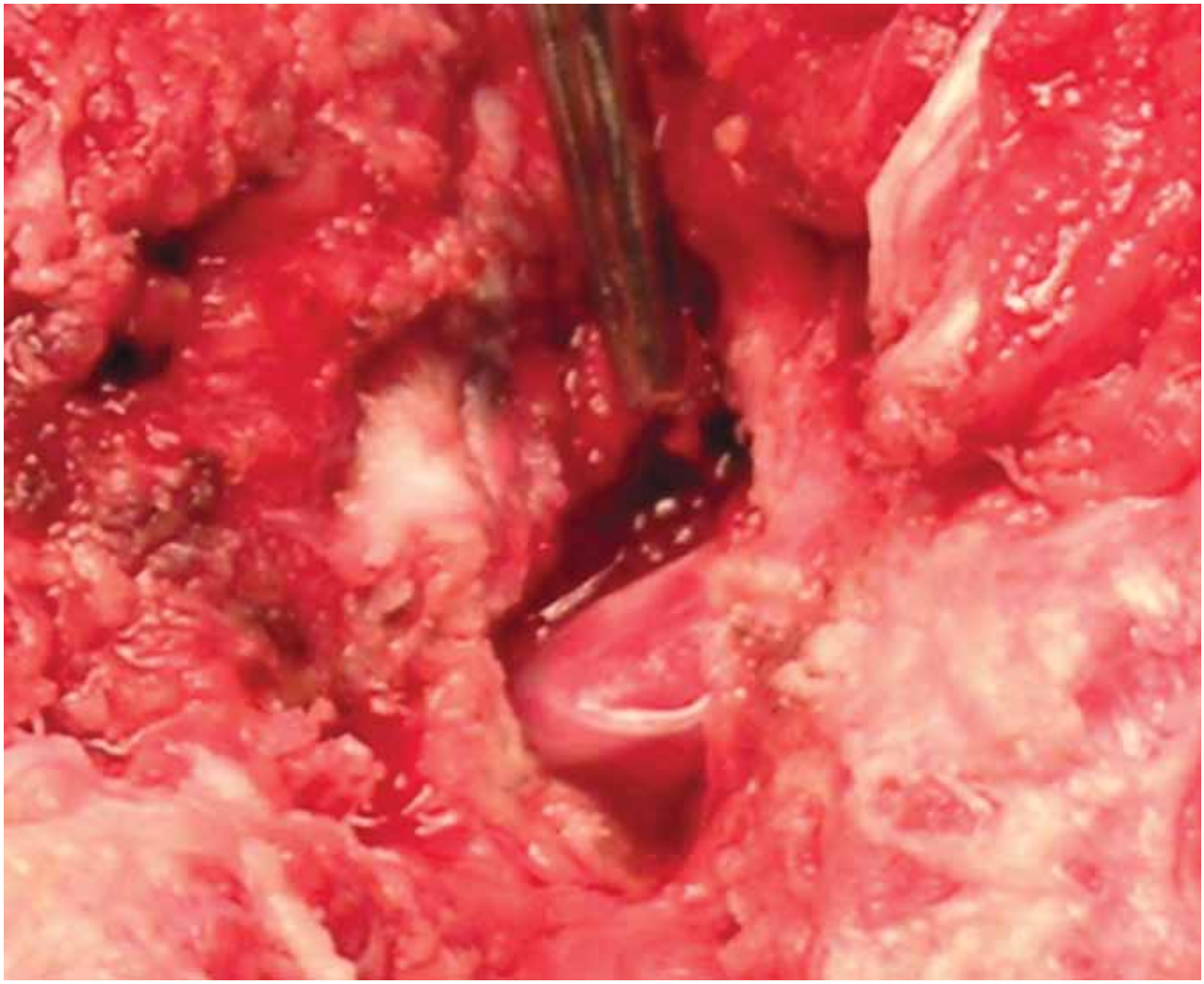

Figure 1. *Pediatric host nation patient with penetrating trauma to his back. Here, exposed spinal cord with cerebrospinal fluid leak is noted at the lumbar (L3) level. The spine was stable with only posterior spinal column involvement. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.*

trauma leading to spinal injury, needs to be classified on a case-by-case basis based on physical findings (e.g., open wound in proximity to spinal injury). Regardless of the etiology, spinal trauma represents a critical wounding pattern that increases the morbidity and mortality of soldiers injured on the battlefield.[6]

All soldiers injured on the battlefield, including soldiers with spinal cord injuries, are treated within the Health Service Support (HSS) system. This system comprises established levels of CCC. Each level of care, starting at the point of injury to medical centers within the continental United States (CONUS), varies in respective capacity to manage spinal trauma. Each level of care (Level I thru Level V) is designed to deliver a progressively greater degree of care and resources. Careproviders working in Level I thru Level III facilities will face the challenge of providing initial care for a significant number of penetrating spinal trauma casualties. This will be a markedly different experience for CCC providers trained in civilian centers, where blunt trauma mechanisms of spinal injury predominate.[7,8,9] This chapter will discuss the varying levels of CCC, relevant anatomical considerations and spinal cord injury patterns, and the optimal management of the spinal injury patient within a CCC environment.

Epidemiology

As of June 2010, 37,669 combatants were listed as wounded in action from Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF). Total soldier deaths from these two conflicts over the same time period was 5,425. In an attempt to report and study the injuries from this conflict, Holcomb et al. have created the Joint Theater Trauma Registry (JTTR) database. From this database, the rate of spine injury from OEF and OIF is 1.4 percent.[10] Therefore, CCC providers will need to be proficient in the management of combatants with spine injuries.

Levels of Combat Casualty Care

Civilian medical care for spinal injuries relies on the ability of emergency medical services (EMS) personnel to stabilize spinal injuries and directly transport the patient, using spinal immobilization, to a trauma center for definitive care. Typically, the civilian transport process takes less than an hour. In extenuating circumstances, if the patient is located in a remote location at the time of injury, transport time may be extended to several hours. In contrast, CCC provided from the point of injury to CONUS medical centers proceeds through varying stages of care. Typically, it takes 72 to 96 hours for a combat casualty to reach a Level V facility (Fig. 2).[11,12,13] Combat casualty care is not only complicated by long distance air evacuation (AIREVAC) through multiple facilities and careproviders, but also by unpredictable weather conditions and the inherent hostility of the battlefield.

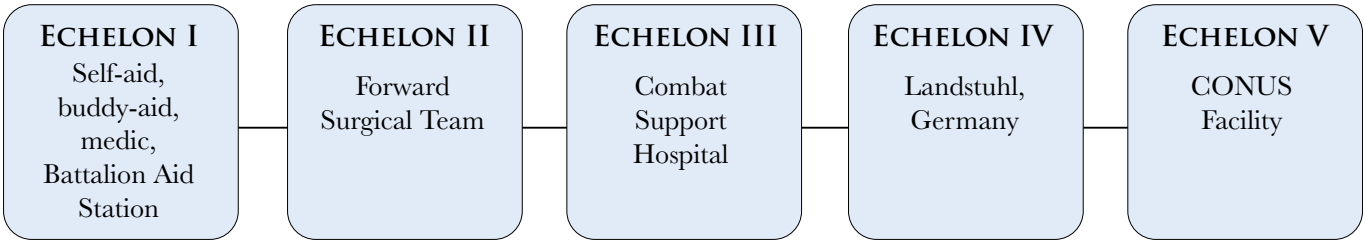

Figure 2. *Evacuation chain for combat casualties.*

Level I

Level I CCC refers to the initial care an injured soldier receives in the battlefield. This level of care is often provided by a combat medic trained at an emergency medical technician–basic (EMT-B) level. There are times when a careprovider with a higher level of training (e.g., special forces medic, physician assistant, or physician) is present at the point of injury. However, CCC providers at this level are limited by available resources and their environment. Level I care is often provided while the initial conflict or battle is in progress (Fig. 3). Casualty evacuation (CASEVAC) times may be delayed because of the ensuing battle or the hostility of the terrain.

Because of the possibility of exposing themselves to hostile fire, CCC providers may not be able to safely provide proper spinal immobilization for those injured on the battlefield (e.g., care-under-fire scenario). It is recommended that spinal immobilization be performed once the injured soldier has been moved to a safer location, such as a casualty collection point.

Figure 3. *On the battlefield (e.g., care-under-fire scenario), preservation of the lives of the casualty and medic is of paramount importance. Spinal immobilization of the injured soldier is performed once they have been moved to a safer location. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.*

Although battlefield heroics often prevail, careproviders risk injury or death by exposing themselves to direct fire from the enemy in order to provide CCC. Current ATLS guidelines recommend all patients with an injury above the clavicle or a head injury resulting in a loss of consciousness be immobilized in a semi-rigid cervical collar and a backboard.[1] This is not always appropriate on the battlefield because proper placement of a cervical collar and spinal immobilization could involve multiple personnel exposing themselves to hostile fire, thereby increasing the likelihood of more injured soldiers. In a retrospective study of penetrating trauma to the neck in the Vietnam War, Arishita et al. discovered that roughly 1.4 percent of the casualties treated with cervical spine precautions actually had a spinal injury that would have benefited from cervical spine immobilization.[14] The authors concluded that mandatory cervical spine immobilization of all penetrating neck trauma sustained in an environment hostile to careproviders had an unfavorable risk-to-benefit ratio. For this reason, it is recommended that spinal immobilization be performed once the injured soldier has been moved to a safer location, such as a casualty collection point.

Studies performed in civilian and combat settings suggest that most patients with normal neurological exams following penetrating trauma to the neck will not have a mechanically destabilized spinal column.[15,16]

Level II

Level II care expands upon Level I care by providing a designated CCC facility that is more removed from direct conflict. This facility is staffed with greater numbers of careproviders with access to laboratory and medical imaging technology. Radiographic studies are usually limited to plain radiography and hand-

carried ultrasonography. Computed tomography (CT) and magnetic resonance imaging (MRI) are typically not available at these facilities. The main mission of Level II facilities is to recognize and treat injuries that compromise a casualty's airway, breathing, and the circulatory system. Treatment initiated at this level typically follows the established protocols found in ATLS.[1] Combat casualties with suspected unstable spinal injuries undergo spinal immobilization at Level II facilities. Spinal injury patients who are experiencing neurogenic shock are treated with supportive care. Operative interventions aimed at stabilizing spinal injuries are not typically performed at these facilities.

Level III

Level III care facilities are represented by a field hospital environment such as a Combat Support Hospital (CSH), described as a Mobile Army Surgical Hospital (MASH) in previous conflicts. Level III facilities provide highly trained and specialized careproviders, extensive medical imaging capacity (e.g., CT scans), laboratory support, and blood bank services. In addition, they provide dedicated operating rooms with specialized surgical capabilities, and it is not uncommon to have vascular surgeons, neurosurgeons, orthopaedic surgeons, and trauma surgeons available.

This is the first CCC environment where more definitive stabilization of life-threatening injuries can be achieved. With regard to spinal injury, this level of care allows for better delineation of spinal column injury via CT imaging. Computed tomography has the ability to detect even the most subtle bone pathology. In addition, as the patient is further evaluated, tertiary examination of the casualty often confirms or refutes the presence of spinal injury. Once a spine injury is recognized, a Level III CSH provides the human and logistical resources needed to maintain strict spinal immobilization and ensure optimal patient transportation. If the patient cannot be treated definitively at this level with subsequent return-to-duty, the primary mission of the CSH is to stabilize all injuries such that the casualty can tolerate intertheater air evacuation to a higher level of care.

Level IV

Level IV facilities (e.g., Landstuhl Medical Center in Germany) are major medical centers that have the capacity to diagnose and treat complex injuries. They are often located out-of-theater and provide maintenance of stabilization and ongoing resuscitation. These treatment facilities provide a broad spectrum of specialty and subspecialty medical care, advanced diagnostic capability (e.g., MRI, angiography suites, nuclear medicine), surgical interventions, and stabilization prior to the patient reaching Level V care.

Level V

Level V facilities (e.g., Brooke Army Medical Center-BAMC) provide stabilization, definitive care, and reconstructive surgical capabilities. Level V is the highest level of medical treatment available to combat casualties. Definitive surgical repair and reconstruction of all injuries is provided at this level of care. With regard to spine injuries, definitive stabilization optimally occurs at Level V, unless it is medically necessary to perform at preceding levels of care (e.g., a case of progressive neurologic deterioration with documented cord compression).

Unless it is medically necessary to perform an operation at preceding levels of care (e.g., progressive neurologic deterioration with documented cord compression), a definitive stabilization procedure optimally occurs at Level V facilities.

Battlefield spinal injuries will challenge CCC providers. Given the limited capabilities to recognize and definitively treat spinal trauma in Level I and II settings, the main goal of management is to maintain spinal immobilization and initiate spinal cord resuscitation pending better delineation of spinal injury. Level III facilities will enable optimal radiographic imaging of spinal injuries, while definitive surgical stabilization typically occurs at Level IV and V facilities.

Emergency Management

Forty-seven percent of patients with spinal trauma and 64 percent of patients with spinal cord injuries have concomitant injuries in civilian studies.[17,18] Head, chest, and long-bone injuries are the most frequently associated injuries. Similarly, a majority of spinal injuries sustained in combat environments will be associated with concurrent injuries.[3,4,5,6]

Figure 4. *Open spinal injury. Image courtesy of Joint Combat Trauma Management Course, 2007.*

In the setting of multiple trauma, in which most spinal injuries occur, assessment and management of airway, breathing, and circulatory system compromise take priority. The assessment and management of airway, breathing, and circulation issues and the simultaneous identification of neurologic disability with exposure of the casualty constitute the primary survey. Immediately following initial stabilization, a careful neurologic examination is warranted. Spinal cord injuries have important physiologic consequences. A cervical or high thoracic spinal cord injury often results in a loss of phrenic and/or intercostal nerve function with resultant compromise of diaphragmatic and chest wall excursion. This is of special concern in patients with concurrent head, chest wall, or lung injury. All patients with high-level spinal cord injuries require close observation to ensure that ventilation and oxygenation remain adequate.

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Once primary survey issues are addressed, a secondary survey is performed. The secondary survey consists of performing a focused physical examination. Palpation of the entire spinal column and a more detailed neurological exam are performed during the secondary survey. Spinal injuries are typically documented during this phase of casualty management. Special attention must be paid to identifying physical findings suggestive of open spinal injuries (e.g., open wound in proximity to spinal injury) and detecting neurologic deficits suggestive of spinal injury (Fig. 4). The neurologic exam does not have to be extensive but should document the basic sensory and motor function of the combat casualty.

A direct path from the skin through the dura defines an open spinal injury. All spinal injuries associated with penetrating trauma should be deemed open injuries, until proven otherwise.

0	Total paralysis
1	Palpable or visible contraction
2	Active movement, full range of motion, gravity eliminated
3	Active movement, full range of motion, against gravity
4	Active movement, full range of motion, against gravity and provides some resistance
5	Active movement, full range of motion, against gravity and provides normal resistance
5*	Muscle able to exert, in examiner's judgment, sufficient resistance to be considered normal if identifiable factors were not present
NT	Not testable. Patient unable to reliably exert effort or muscle unavailable for testing due to factors such as immobilization, pain on effort, or contracture

Table 1. *American Spinal Injury Association Muscle Grading Score.*

In a responsive patient, light touch examination attempts to localize the lowest dermatome with normal sensation equilaterally. The sensation of pain may be of limited utility, as most casualties will have been made more comfortable with the use of narcotics or may have altered mental status due to concurrent injuries. However, motor function can be readily assessed in a cooperative patient. An intact motor level is recorded as that level that maintained enough muscular function to resist gravity (3 out of 5 motor strength) (Table 1).[19] Special attention must be paid to documenting rectal tone, sacral sensation, and bowel and bladder function to avoid missing injuries to the terminal spinal cord (conus medullaris) and cauda equina. Neurologic assessment in unresponsive patients is much more limited. The exam consists of observing spontaneous movements, the patient's motor responses to painful stimuli, the presence or absence of deep tendon reflexes, and rectal sphincter tone.

Figure 5. *During the secondary survey, patients should be logrolled, and the entire length of their backs should be inspected for tenderness, open wounds, deformities, or ecchymoses. Image courtesy of Defense Imagery Management Operations Center (DIMOC).*

A complete secondary survey includes careful examination of the entire length of the patient's spine. This is performed by gently logrolling the patient to his or her side while supporting the patient's spine with a cervical collar and in-line immobilization (Fig. 5). The patient's back should be inspected for open wounds, deformity, and ecchymosis. The spine should be palpated for a step-off or interspinous widening. The locations of lacerations and abrasions on the skull may help determine the mechanism of cervical injuries. Occipital lacerations suggest flexion injuries,

whereas frontal or superior injuries suggest extension or axial compression, respectively. The presence of a single spinal injury does not preclude careful evaluation of the rest of the spine. Eight to 28 percent of patients with a spinal column injury have additional noncontiguous spinal injuries, and it has been noted that up to 30 percent of these injuries are not initially recognized.[20,21,22,23,24] The upper and lower extremities are examined for motor function by nerve root level. Tables 2 and 3 outline how upper and lower extremity spinal nerve roots relate to muscle function, sensation, and reflex activity. The motor examination also includes a digital rectal examination for voluntary or reflex bulbocavernosus anal sphincter contraction.

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Root	Reflex	Muscles	Sensation
C5	Biceps	Deltoid, biceps	Lateral arm
C6	Brachioradialis	Wrist extension, biceps	Lateral forearm, thumb, index finger
C7	Triceps	Wrist flexion, finger extension, triceps	Middle finger
C8		Finger flexion	Medial forearm
T1		Finger abduction	Medial arm

Table 2. *Upper extremity spinal nerve roots with respective muscle function, sensation, and reflex activity.*

Root	Reflex	Muscles	Sensation
L1, L2		Hip flexion	Inguinal crease (L1), anterior thigh (L2)
L2, L3		Knee extension	Anterior thigh (L2), anterior thigh just above knee (L3)
L4	Patellar tendon	Ankle dorsiflexion	Medial leg, medial foot
L5	None	Extensor hallucis longus extension	Lateral leg, foot dorsum
S1	Achilles tendon	Ankle flexion	Lateral foot, sole

Table 3. *Lower extremity spinal nerve roots with respective muscle function, sensation, and reflex activity.*

Areas of sensory deficit should be accurately recorded, dated, and timed on the medical record progress note or spinal injury flow sheet and demarcated with ink at the affected level on the patient's skin.

The sensory examination includes testing of dermatomal pattern skin sensation. The light touch sensation

of a cotton-tipped applicator (Q-tip®) rubbed lightly over the skin is a quick and reliable examination maneuver. Sensation should also be tested in the perianal region. The areas of sensory deficit should be accurately recorded, dated, and timed on the medical record progress note or a spinal injury flow sheet. It is also recommended that the sensory level be marked, dated, and timed in ink on the patient's skin at the affected level. The practice of marking the sensory level on the skin can avoid much uncertainty when a number of examiners are involved. A known challenge of CCC is adequate hand-off or transfer of notes between the levels of care. When available, permanent markers can be used on the injured soldier's skin to aid the documentation process.

Spinal Shock

Spinal shock is defined as the loss of spinal reflexes after injury to the spinal cord, which affects the muscles innervated by the cord segments situated below the site of the lesion.[25] The loss of reflex function is most severe closest to the site of spinal cord injury. Patients with high-level cervical cord injuries may retain some distal sacral reflex function (bulbocavernosus reflex and anal wink) despite loss of more proximal reflex function.[26,27] The documentation of the bulbocavernosus reflex is a key early determinant of whether spinal shock has resolved in patients that had initially lost distal sacral reflex function. Return of spinal reflex function occurs in a caudal to rostral direction. The return of the bulbocavernosus reflex marking the end of spinal shock is variable but often occurs within 24 hours of cord injury.[26,27,28,29,30] The bulbocavernosus reflex is elicited by simultaneous digital rectal exam and lightly squeezing the glans penis (males) or by gently tugging on a placed Foley catheter in female or male patients. An involuntary increase in tone around the examiner's digit with these maneuvers indicates the presence of an intact bulbocavernosus reflex (normal). The absence of a bulbocavernosus reflex implies the patient is still in spinal shock. When the bulbocavernosus reflex is absent, the prognostic value of the motor exam following a spinal injury is inconclusive. The prognosis of a spinal injury patient with a complete cord syndrome, following the return of the bulbocavernosus reflex, is poor. Complete cord syndrome patients have a less than 5 percent chance of functional recovery if no motor function improvement is documented at 24 hours following return of the bulbocavernosus reflex.[29,31] Spinal shock following spine trauma should be differentiated from neurogenic shock, an injury syndrome characterized by flaccid paralysis, moderate hypotension, and varying degrees of bradycardia.[32]

The absence of a bulbocavernosus reflex implies the patient is still in spinal shock, and the prognostic value of the motor exam following a spinal injury is inconclusive. The return of the bulbocavernosus reflex marking the end of spinal shock is variable but often occurs within 24 hours of cord injury.

Spinal Immobilization

If following the initial patient evaluation suspicion exists for a spinal cord injury, strict spinal cord injury precautions must be observed until spinal injury has been ruled out (provided battlefield conditions allow). Civilian studies have suggested that neurologic deficits progress as a result of inadequate spinal immobilization in up to 5 percent of hospitalized patients during their initial stay.[2,33] Keeping the combat casualty immobilized on a full-length backboard during the initial resuscitation often facilitates patient care. This method of stabilization facilitates patient transportation and allows for rapid logrolling of the patient to prevent aspiration in case of vomiting.

Upon identifying a potentially unstable spinal injury on standard radiography, CT imaging is often the next

step in defining the injury. The most conservative management style is to maintain spinal immobilization until a CT scan is performed to better delineate the mechanical stability of the injured spine. Aggressive pain management will often be necessary to alleviate the discomfort resulting from immobilization on a hard backboard. Special care (e.g., padding hard surfaces) will need to be taken to avoid pressure necrosis of the skin and underlying tissue that can develop within hours of immobilization on a hard surface.[34,35]

Pressure necrosis of the skin and underlying tissue can develop within hours of immobilization on a hard surface.

The most effective method of initial cervical immobilization is the use of bilateral neck supports (e.g., sandbags or rolled towels) and taping of the patient across the forehead to a spine board, along with the use of a rigid cervical collar (which serves to limit extension) (Fig. 6).[36,37] In unstable cervical spine injuries, a soft collar, extrication collar, hard collar, or Philadelphia collar alone is not sufficient for immobilization.[36] A poster brace (e.g., four-poster brace) or sternal occipital mandibular immobilizer (SOMI brace) is not utilized on the battlefield. Securing a patient to a standard long spine board is also standard practice for immobilization of the thoracolumbar spine. This enables rotating of the patient (e.g., if they need to clear their airway and vomit) while providing maximal support to the thoracolumbar spine.[36] Cadaveric studies of recreated unstable spinal injuries suggest that significant translational and rotational spinal movement still occurs, despite in-line traction and backboard immobilization during logrolling maneuvers.[38,39,40] Hence, rotational movement of suspected spinal injury patients should be minimized. Spinal immobilization is discontinued only after radiographic and clinical evaluation have excluded unstable spinal injury. This usually occurs at the CSH where trauma surgeons and radiologists are available to interpret radiographic imaging and further assess patients.

Figure 6. *Image demonstrating appropriate initial cervical immobilization with the use of bilateral neck supports, taping of the patient's forehead to a spine board, and use of a rigid cervical collar.*

The most effective method of initial cervical immobilization is the use of bilateral neck supports (e.g., sandbags) and taping of the patient across the forehead to a spine board (stretcher or litter), along with the use of a rigid cervical collar.

Tactical Combat Casualty Care

Care-under-fire or at the point of injury is dictated by the tactical situation. The combat casualty may have to be moved to a safer location before an assessment can be accomplished. In battlefield conditions, rapid casualty evacuation is often a life-threatening process. When only one person is available to assist the combat casualty, the casualty is carried or often dragged to a safer location. Patients with suspected spine

injuries should undergo spinal immobilization as soon as it is feasible. Strict spinal cord injury precautions are ideally observed until spinal injury has been ruled out.

In care-under-fire tactical situations without direct confirmation of spinal injury, the presence or absence of associated risk factors should be identified. Associated risk factors for spinal injuries include bullet, fragmentation, and stab wounds, and direct trauma to the face, neck, head, or back. One may also include extreme twisting of the trunk and major blows to the head or chest that may occur from large blast injuries or body impact from landing following the blast wave (tertiary blast injury). Along with documented neurologic deficits, a Glasgow Coma Scale (GCS) score of 8 or below is associated with a higher risk (odds ratio = 2.77) of cervical spine injuries when associated with traumatic brain injuries.[41]

Figure 7. *Example of a rigid cervical collar applied to a combat casualty with head trauma.*

The standard battlefield cervical collar is the Vertebrace® Extrication Collar (VEC) (Fig. 7). Many Level I careproviders not only carry the VEC, but also have access to long spineboards that can be attached to the military litter prior to transport. Medical and rescue personnel should make every attempt to control the spine during initial extrication and evacuation until proper cervical and thoracolumbar spine precautions can be provided. Documentation of known injuries and neurological deficits must occur during the stabilization and resuscitative phases of treatment, often taking place prior to the injured combatant reaching a Level III care facility.

Spinal Resuscitation

Once spinal injury is recognized, it is important to begin resuscitative efforts as soon as possible. The ATLS guidelines have been designed to ensure full body resuscitation of the traumatized patient.[1] It is important to note that the injured spinal cord itself needs to be carefully resuscitated. In general terms, resuscitation of the spinal cord implies that perfusion with oxygenated blood to the injured area is restored to begin the process of healing and prevent further injury.[42,43]

Resuscitation of the spinal cord involves minimizing secondary injury due to hypoxemia, hypoperfusion, and mass effect.

Definitive evidence of discrete hemodynamic resuscitation parameters leading to improved clinical outcomes following spinal injury does not exist. A series of animal and human studies do support the concept of optimizing spinal cord perfusion following spinal cord injury.[44,45] Suggested treatment options include ensuring hemodynamic stability and maintaining mean arterial pressures greater than or equal to 80 mm Hg to optimize spinal cord perfusion.[30,42,43,46,47,48,49,50,51] Similarly, ensuring adequate oxygen delivery

to the spinal cord is important. This often requires supplemental oxygenation via breathing apparatus and the application of pulse oximetry. Likewise, airway, breathing, and circulatory compromises need to be corrected to ensure optimal resuscitation of the injured spine. Maintaining a minimum hemoglobin value of 7 grams per deciliter in trauma patients is recommended, and the timing of blood transfusion (triggers) should be based on bedside clinical findings rather than absolute hemoglobin values.[52]

Neurogenic Shock

Spine trauma may result in neurogenic shock, a syndrome characterized by flaccid paralysis, moderate hypotension, and varying degrees of bradycardia.[32,53] The typical neurogenic shock patient will have suffered a traumatic spinal cord injury, resulting in disruption of T1 to L2 sympathetic outflow.[54] Vagal tone is unopposed, and moderate hypotension and bradycardia ensue. Bradycardia is a distinguishing sign in neurogenic shock as opposed to other shock states where tachycardia is often observed.

Neurogenic shock is a diagnosis of exclusion and should be made only after life-threatening hemorrhage has been excluded, as hypovolemic shock is the most common shock state observed after battlefield trauma. Recognition of neurogenic shock will minimize excessive use of crystalloid fluids and resultant dilutional coagulopathy and pulmonary edema. Vasopressors may be used following the restoration of intravascular volume to maintain normal blood pressures and minimize excessive fluid administration.[55]

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Restoring Spinal Alignment

An immediate initial treatment priority is to realign the spine. Subluxed or dislocated spinal segments often result in additional mechanical stress and ischemic injury to the spinal cord. Alignment of the spine is accomplished through a variety of interventions ranging from traction devices to operative interventions. Spinal realignment requires specialized equipment, spine surgeons, and support staff. Hence, attempts at spinal realignment must typically await transfer of the spinal injury patient to Level III or higher facilities.

Role of Glucocorticosteroids

The use of steroids in the battlefield for treatment of spinal injury is not recommended.[56,57,58,59] The role of glucocorticosteroids in the treatment of the acute spinal cord injury has long been controversial. The purported mechanisms through which steroids exert their effects following spinal cord injury are unclear. There is no evidence to suggest administration of glucocorticosteroids results in neurologic improvement following penetrating spinal trauma, and some studies suggest increased harm.[58,60,61,62] Inconsistency in study methodology and reporting has largely discredited any pre-existing civilian data, such as the National Acute Spinal Cord Injury Studies (NASCIS), suggesting benefit to the administration of glucocorticosteroids following blunt spinal trauma.[56,57,58,62,63,64,65,66,67,68]

> The use of steroids following penetrating spinal injury is not recommended as there is no evidence to suggest improved neurologic outcomes, and some studies suggest increased harm.

Detailed Assessment of Spinal Injury

A complete and thorough understanding of spinal anatomy and spinal cord function is integral to the appropriate recognition and subsequent management of spinal injuries. In-depth assessments of spinal injuries are performed after patients are removed from immediate danger, immobilized, hemodynamically stabilized, and transferred to appropriate levels of care. This level of assessment and evaluation is typically performed in Level III and higher facilities. Computed tomography evaluation and serial neurological evaluations constitute the critical elements that help careproviders further assess and subsequently manage spinal injuries.

Spinal Anatomy

Due in large part to its flexibility and exposure, the cervical segment is the most commonly injured part of the spinal cord.[69] In contrast, the thoracic spine (T1 to T10 vertebral bodies) is a rigid and fixed structure. This results from ribs attaching to their respective transverse processes and forming articulations anteriorly with the sternum. Cervical nerve roots exit the spinal canal above their respective vertebral bodies. Thoracolumbar nerve roots exit the spinal canal below their respective vertebral bodies. The neural canal is narrower in the thoracic spine than in the cervical or lumbar spine. These anatomic characteristics and the fact that great force is required to damage the thoracic spine probably account for the high incidence of significant neurologic injuries following fractures of the thoracic spine.[69,70] Because of its unique anatomy, the thoracolumbar junction is the second most injured area of the spine.[69,70] Unlike the other ribs, the 11th and 12th ribs do not articulate with the sternum, nor do they attach to their respective transverse processes.

Due in large part to its flexibility and exposure, the cervical segment is the most commonly injured part of the spinal cord, while the thoracolumbar junction is the second most injured area.

The orientation of lumbar vertebral body articulating facets and thicker lumbar intervertebral discs allow for more flexion, extension, and lateral bending of the lumbar spine. Thus, the rigidly fixed thoracic spine abruptly transitions to a less rigidly supported lumbar spine. This abrupt transition likely explains the susceptibility of the thoracolumbar junction (T11 to L2 vertebral bodies) spinal segment to injury.[69] The spinal canal is relatively wide at this region. Hence, thoracolumbar junction injuries often result in incomplete cord lesions. The spinal cord's terminal segment is called the conus medullaris. It terminates at the first lumbar (L1 vertebral body) level in adults and at the L2 or L3 vertebral body level in pediatric patients. Individual nerve roots extending distal to this segment constitute the cauda equina. The lower lumbar and sacral segments are less prone to spinal cord injury, and the neurologic sequelae are usually less severe.[69,71]

The blood supply to the spinal cord consists of the anterior and posterior spinal arteries and radicular arteries (Fig. 8). The anterior spinal artery perfuses the anterior and central cord, and the posterior spinal artery supplies the posterior one-third of the spinal cord. The anterior and posterior spinal arteries ascend from the vertebral arteries and travel downward along the anterior and posterior aspects of the spinal cord. With the exception of the cervical region, these small arteries inadequately maintain the viability of the spinal cord. Radicular arteries serve to augment the blood supply to the spinal cord in areas where the anterior

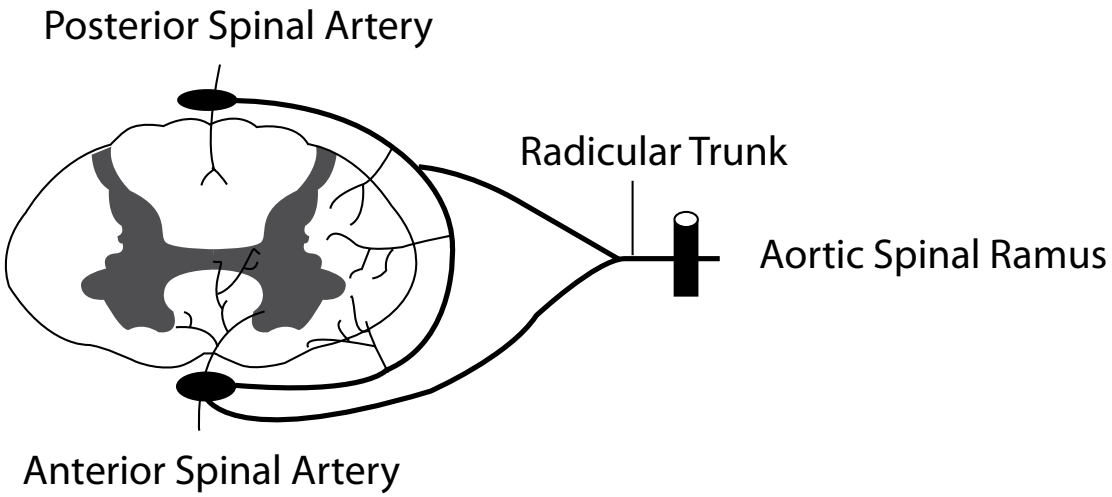

Figure 8. *Cross-section illustration of spinal cord anatomy demonstrating its vascular supply.*

and posterior arteries alone are insufficient. The radicular arteries arise from the thoracoabdominal aorta, among other sources. They form anastomotic tracts with the spinal arteries. The midthoracic region of the spinal cord is described as a watershed area. This region has limited blood flow and is located between the well-perfused superior and inferior segments of the spinal cord. One of the larger radicular arteries is the great radicular artery of Adamkiewicz. It enters the spinal canal between the T10 and L2 vertebral bodies. Injury to this artery explains how neurologic deficits resulting from spinal cord ischemia may extend cephalad from a more caudal vertebral body fracture or dislocation.[29,72]

Radiographic Considerations

Plain radiography, CT imaging, and MRI are used in the evaluation of spinal injuries. In civilian studies, up to 5 percent of trauma patients who are unable to give a reliable history or have a painful distracting injury have a spinal injury.[73,74,75] Conversely, the patient who has neither spinal pain or midline tenderness on palpation nor neurologic signs or symptoms and is awake, alert, and without major distracting injuries does not require routine spinal radiographs.[76] While indications for plain radiography of the thoracolumbar spine have not been clearly defined, the application of criteria developed for cervical spine radiography appears reasonable.[77,78]

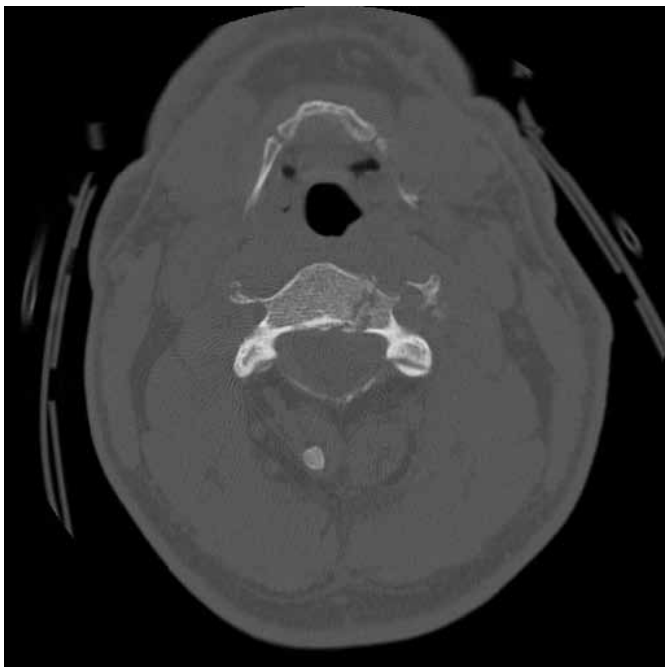

Figure 9. *Axial CT image demonstrating C3 vertebral body fracture.*

A standard trauma spinal series should include a cross-table lateral and an anterior-posterior (AP) view radiograph. An open-mouth odontoid view is needed for cervical spine evaluation. While the

sensitivity and specificity of these views are controversial, a normal and technically optimal study is widely considered adequate to exclude major fractures and dislocations. The primary exceptions include patients with unexplained neurologic deficits or underlying musculoskeletal conditions (e.g., severe spondylosis). Patients with unexplained neurologic deficits or a major fracture or dislocation identified on plain radiographs should undergo CT scan to further define the extent of injury. Patients with inadequate plain films or areas of suspicion on plain radiographs typically undergo CT scanning.[76,79]

Patients with inadequate plain films, unexplained neurologic deficits or a major fracture or dislocation identified on plain radiographs should undergo CT imaging to further define the extent of injury.

Computed tomography imaging is more accurate than plain radiography in delineating the extent of fractures and bone fragment displacement.[76,79,80] A CT scan is also useful in identifying minor fractures, many of which are missed on plain radiography. Standard axial CT images are adequate to delineate most injuries (Fig. 9). Chance fractures and odontoid fractures are exceptions.[18,81,82,83,84] Their detection often requires sagittal reconstruction of CT or MRI images and concurrent plain radiographs (Fig. 10).[83,84] Computed tomography imaging fails to adequately visualize the spinal cord, hence it cannot give direct evidence of spinal cord injury. The excellent bone visualization, wide accessibility, and speed of CT imaging make it the primary supplementary method of imaging the spine.

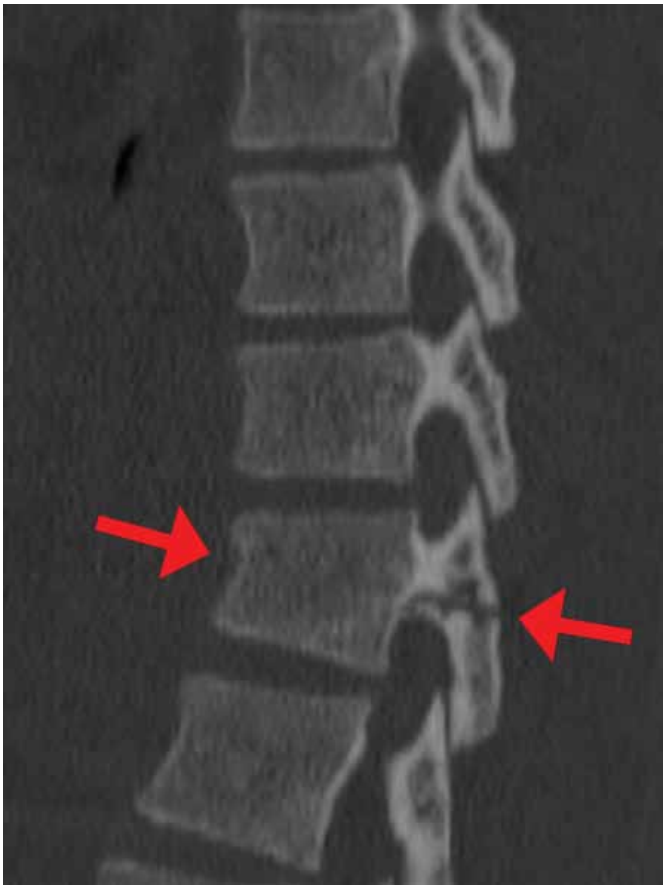

Figure 10. *Sagittal reconstruction of CT images demonstrating a Chance fracture. Adapted image courtesy of LearningRadiology.com.*

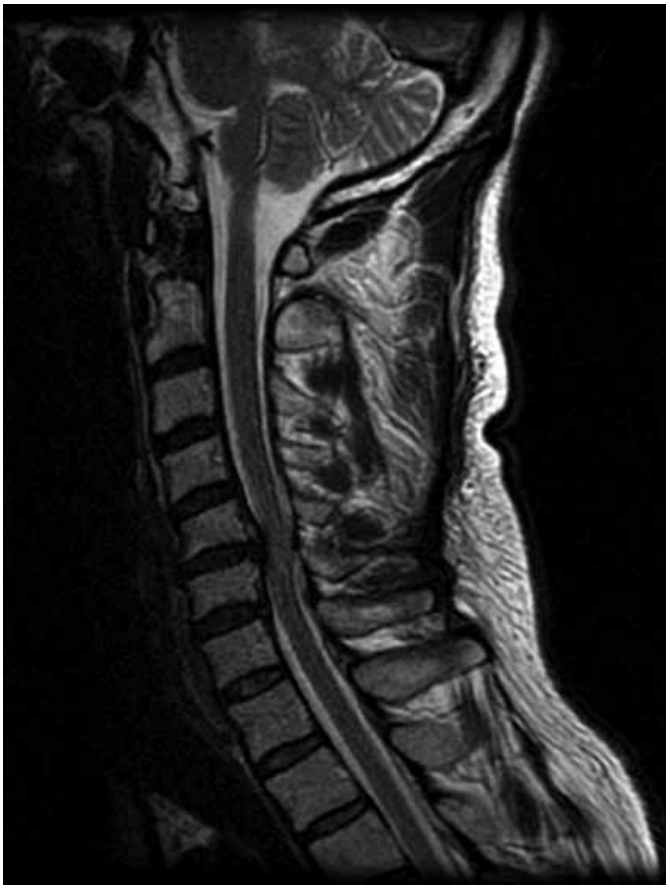

Figure 11. *MRI, available at Level IV or V facilities, allows precise visualization of the spinal cord. MRI imaging is more sensitive than CT imaging for ligamentous and soft-tissue injury.*

Magnetic resonance imaging plays a vital role in spinal cord injury evaluation (Fig. 11). More sensitive than CT scan for ligamentous and soft-tissue injury, MRI also allows precise visualization of the spinal cord.[85,86] Logistical difficulties, including limited accessibility, limited ability to monitor the patient while in the scanner, and prolonged imaging time, currently prevent routine use of MRI in the evaluation of acute spinal injury patients. MRI is not available until Level IV or V care for combat casualties.

Determining Spinal Stability

Determining the stability of the spinal column is an important initial step in the evaluation of spinal injuries. White and Panjabi provided a widely accepted definition of spinal stability.[87,88] They defined spinal stability as the ability of the spine, under physiologic loads, to limit patterns of displacement, to preclude damage and irritation to the neural elements, and to prevent incapacitating deformity or pain due to structural changes.

While White and Panjabi's definition lends itself to the subjectivity of the examiner, the use of CT allows for a much more objective approach to determining mechanical stability. The advent of CT led to an evolution of theory regarding the determinants of spinal column stability. In 1983, Denis devised an anatomic three-column theory of stability based on a retrospective review of 412 spinal injuries and their CT features (Fig. 12).[89] Denis divided the spinal column into anterior, middle, and posterior columns. The anterior column consists of the anterior longitudinal ligament, anterior half of the vertebral body, and the annulus fibrosus. The middle column consists of the posterior half of the vertebral body, posterior longitudinal ligament, and posterior part of the annulus fibrosus. The posterior column consists of the neural arch, ligamentum flavum, facet joint capsules, and the supraspinous and interspinous ligaments. Denis concluded that the integrity of the middle column determines the stability of the spine. Experience over subsequent years has generally supported this concept. Therefore, a definitive assessment of the integrity of the middle column is made at a facility where CT imaging is available, typically at a Level III CSH.

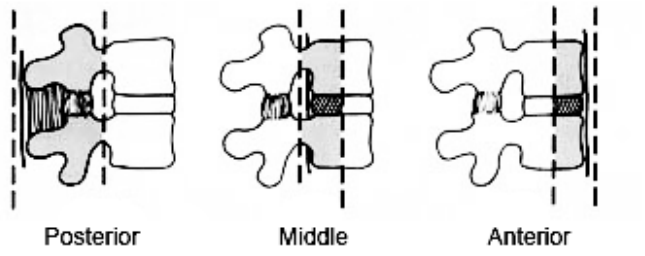

Figure 12. *Three-column Denis classification of the spine. The posterior column consists of the posterior ligamentous complex. The middle column includes the posterior longitudinal ligament, posterior annulus fibrosus, and posterior wall of the vertebral body. The anterior column consists of the anterior vertebral body, anterior annulus fibrosus, and anterior longitudinal ligament.*

Spinal stability is defined as the ability of the spine, under physiologic loads, to limit patterns of displacement, preclude damage and irritation to neural elements, and prevent incapacitating deformity or pain due to structural changes.

Spinal Cord Injury Patterns

Complete Cord Syndrome

A complete spinal cord syndrome is characterized by flaccid paralysis and loss of sensation below the level of spinal cord injury.[78] Deep tendon reflexes and the bulbocavernosus reflex are absent in the acute

phase. This reflex is useful in assessing the integrity of the lower sacral cord segments. The return of the bulbocavernosus reflex marking the end of spinal shock is variable but often occurs within 24 hours of cord injury.[26,27,30,90] The development of spasticity, clonus, hyperreflexia, and Babinski reflexes is more variable. These findings develop days to months after injury. Functional motor recovery following a complete cord syndrome injury is extremely poor. Complete injuries have a less than 5 percent chance of functional motor recovery if no improvement occurs within 24 hours of injury, and virtually no chance of recovery after 48 hours.[29,31,90] Therefore, intervening surgically on a complete injury typically is futile if the goal is neurologic recovery. The restoration of mechanical stability following a complete cord syndrome injury can be delayed until the combat casualty reaches Level IV and V facilities.

Restoration of mechanical stability following a complete cord syndrome injury can be delayed until the combat casualty reaches Level IV or V facilities. With incomplete cord syndromes, a delay in decompression and stabilization is tolerated until the evacuee arrives at Level IV or V facilities, unless the combatant's neurologic condition is deteriorating.

Incomplete Cord Syndromes

A spinal cord injury is termed incomplete if there is some sparing of motor or sensory function below the level of injury. Incomplete spinal cord syndrome patients usually improve from their presenting condition, with some patients regaining the ability to ambulate.[28,78,91,92,93,94] The decision of where and when to surgically intervene remains a challenge for battlefield surgeons. Unless the combatant's neurologic condition is deteriorating, a delay in decompression and stabilization is tolerated until the evacuee arrives at Level IV and V facilities. In an unpublished analysis of combat-related spine injuries, zero of 52 patients evacuated from OEF/OIF had deterioration in neurologic status (Bellabarba, personal communication, 2009). Therefore, it stands to reason that if a combat casualty has tolerated evacuation from the point of injury to a Level III facility without neurologic deterioration, decompression with or without spinal fusion can often be delayed until evacuation to Level IV and V centers. In the presence of neurologic deterioration, the judgment of whether to proceed with surgery in-theater rests with the careprovider at that given level.

Anterior Cord Syndrome

The anterior cord syndrome is the result of an injury to the anterior two-thirds of the spinal cord (Fig. 13). The syndrome typically results from anterior spinal cord compression by adjacent bone or disc fragments following a hyperflexion injury.[93] Hyperextension is a less common mechanism of injury. Anterior cord syndrome is characterized by immediate, complete paralysis with hypesthesia to the level of the lesion, with preservation of light touch, motion, position, and part of vibration sense. This syndrome has a better prognosis for recovery than a complete spinal cord syndrome.[92,93] In one of the earliest descriptions of anterior cord syndrome, Schneider reported that five of 11 patients in his case series regained the ability to ambulate.[92] This potential for recovery underscores the importance of a careful neurologic examination aimed at detecting any residual neurologic function in spinal cord injury patients.[95]

Central Cord Syndrome

The central cord syndrome commonly occurs in the cervical region and typically occurs in the setting of preexisting degenerative spine disease. The spinal cord is thought to be compressed between the ligamentum flavum posteriorly and osteophytes anteriorly during hyperextension. As a result, centrally located spinal tract fibers are injured. Weakness is greater distally than proximally and worse in the upper extremities than

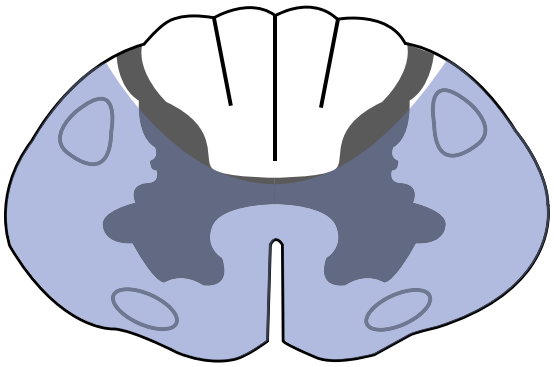

Figure 13. *The anterior cord syndrome is typically the result of anterior spinal cord compression following a hyperflexion injury. It is characterized by immediate, complete paralysis with hypesthesia to the level of the lesion, with preservation of light touch, motion, position, and part of vibration sense.*

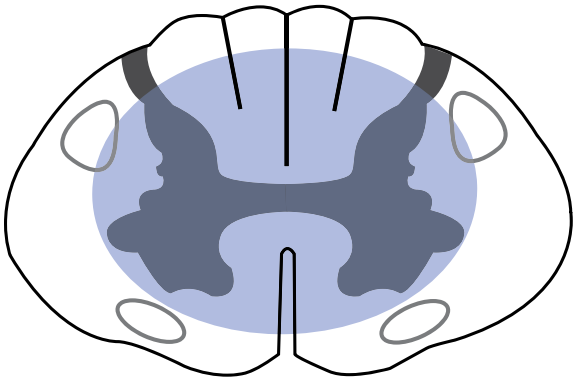

Figure 14. *The central cord syndrome is typically seen following a hyperextension injury. Weakness is greater distally than proximally and worse in the upper extremities than in the lower extremities. Sensory loss is variable.*

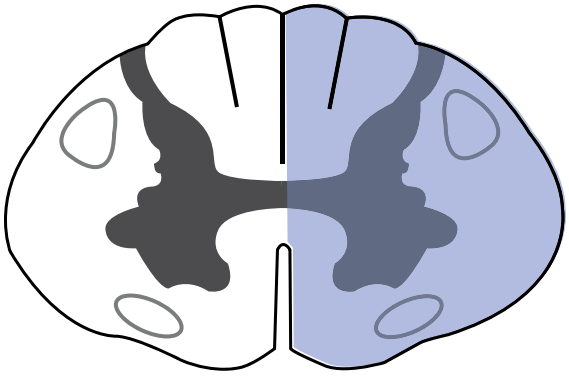

Figure 15. *The Brown-Séquard syndrome is characterized by ipsilateral proprioceptive and motor loss and contralateral loss of pain and temperature sensation.*

in the lower extremities. Sensory loss is variable. In severe cases, upper extremity paralysis, loss of sensation, and urinary retention may occur.[96] The lower extremities are relatively spared. The recovery potential with this syndrome is favorable.[70] Previous studies of central cord syndrome patients have revealed that more than 50 percent become ambulatory and regain functional use of their hands (Fig. 14).[94,97,98]

Brown-Séquard Syndrome

The Brown-Séquard syndrome is characterized by ipsilateral proprioceptive and motor loss and contralateral loss of pain and temperature sensation (Fig. 15).[99] Anatomically, only one-half of the spinal cord is damaged. This results in spinothalamic tract, corticospinal tract, and dorsal column injury. This syndrome, which was previously thought to occur only in penetrating trauma, is increasingly described following blunt trauma. The prognosis for recovery in this syndrome is good. Several case series have documented functional recovery in up to 75 percent of patients with Brown-Séquard syndrome.[91,94,98]

Cauda Equina Syndrome

The cauda equina syndrome results from injury to the lumbosacral nerve roots within the neural canal. The clinical manifestations vary and include sensorimotor deficits of the lower limbs and bowel and bladder areflexia. The prognosis for recovery is similar to peripheral nerve injuries and is significantly better than that for spinal cord injuries.

Spinal Nerve Root Syndromes

Nerve root syndromes deserve special mention because they often occur in the setting of spinal fractures and facet dislocations. The spinal nerve root can be injured along with the spinal cord, or an isolated nerve root injury can occur. Motor and sometimes sensory deficits will be found in the distribution of one or several contiguous nerve roots. The prognosis for recovery from these lesions is good, provided adequate reduction and anatomic restoration of the vertebral column occurs.[100,101,102] Given that nerve root injuries have a good prognosis for recovery, nerve root decompression may be delayed until patients reach Level IV and V facilities, unless a progressive loss of neurologic function occurs.

> Nerve root decompression may be delayed until patients reach Level IV or V facilities unless a progressive loss of neurologic function occurs.

Spinal Cord Injury Without Radiographic Abnormality

Spinal cord injury without radiographic abnormality (SCIWORA) is a post-traumatic myelopathy with no radiographic evidence of fracture or dislocation on plain radiography and CT scan evaluation.[86,103,104,105] Although the syndrome was initially described in children, it can occur in the adult population as well.[106] Anatomic differences of the pediatric spine allow for significant intersegmental movement without bony column disruption. The spinal cord does not share the same degree of elasticity, thus contusions, transections, infarctions, and stretch injuries result. While the exact mechanism for SCIWORA in adults is varied, patients with underlying stenosis, either congenitally or degeneratively, are at considerably much higher risk.[106]

Spinal Injury Management Considerations

Spinal Immobilization and Transport Considerations

Once an unstable spinal injury is identified at a Level III facility, full spinal immobilization pending definitive stabilization is indicated. The medical evacuation network in the theater of operation has greatly reduced the evacuation and transport time for injured combatants. If a prolonged delay in definitive care exists, moving the patient to a bed using strict spinal cord injury precautions is often attempted. The risk of worsening the patient's neurologic status must be weighed against the risk of pressure necrosis of the skin and degree of patient discomfort resulting from the backboard.[34,35,107] Level I care is often limited to hard cervical collars, Kendrick Extrication Device (K.E.D.®) short boards, and long spineboards placed on a military stretcher. Due to the space limitations inherent in casualty evacuation (ground ambulance and helicopter), the equipment used on the battlefield for spine immobilization is limited.

When a prolonged delay in definitive care of unstable spinal injuries exists, the risk of worsening the patient's neurologic status during transfer to a bed must be weighed against the risk of pressure necrosis of the skin, and degree of patient discomfort, resulting from full spinal immobilization on a backboard.

Patients at greatest risk for pressure sores are those who remain on the spine board for more than two hours without being repositioned.[34,35] Careproviders must use their discretion. If enough manpower is not present to move the patient safely, or there is any doubt about the patient's ability to cooperate with spinal injury precautions, the patient should be kept immobilized on the backboard. The patient should be moved as little as possible as even minimal movement may worsen the neurologic deficit in an unstable spinal injury. If a spine injury is excluded or deemed to be a stable injury, prompt removal of the patient from the backboard to a more comfortable setting is indicated.

Timing of Surgical Interventions

The role and timing of operative intervention in patients with acute spinal cord injury are controversial.[108,109,110,111,112] The paucity of prospective randomized trials defining operative indications for acute spinal injury results in a disparate approach to these injuries among spine specialists. Issues germane to CCC providers may be addressed by using three clinical categories: (1) patients with complete spinal cord syndromes; (2) patients with incomplete but progressive spinal cord syndromes; and (3) patients with incomplete but nonprogressive spinal cord syndromes.

Emergency spine surgery for penetrating or closed injuries of the spinal cord is indicated only in the presence of neurologic deterioration.

If a spinal cord neurologic injury can be determined to be a complete injury (i.e., the bulbocavernosus reflex is intact and complete loss of sensorimotor function, including proprioception exists), spinal segment realignment may proceed at a less urgent pace. There does not seem to be any evidence to indicate that early surgical treatment can alter the prognosis of patients who present with complete cord syndromes.[113] Although this subset of patients may need subsequent stabilization of the spine, they do not require emergency surgery. An exception may be in the cervical spine, where urgent reduction may improve the rate of "root-sparing" recovery.[114,115]

There is broad support for emergency surgery for patients with incomplete but progressive spinal cord syndromes. This syndrome is rare, but may result from progressive spinal cord injury via fracture displacement, bone fragment compression, expanding hematoma, spinal cord edema, or infarction.[116] Animal studies have indicated that immediate decompression of neural elements is associated with reduction of permanent neurologic sequelae.[117,118] In combat environments, transport times and access to Level III, IV, and V care must be weighed by CCC providers when considering the timing of surgical interventions.

While there does not seem to be any evidence to indicate that early surgical treatment can alter the prognosis of patients who present with complete cord syndromes, there is broad support for emergency surgery for patients with incomplete but progressive spinal cord syndromes.

Management of incomplete and nonprogressive spinal cord syndromes involves rapid spinal segment reduction and stabilization to minimize neurologic injury. In the cervical spine, such management frequently involves the application of skull traction devices at Level III or higher facilities. In the thoracolumbar spine, traction is less successful, so if neutral supine body positioning does not restore anatomic alignment, definitive correction of the malalignment will typically be performed at the time of stabilization surgery (typically at Levels IV and V).

Spinal Reduction Interventions

Axial Traction

Prior to any attempt at reduction of a malaligned cervical spine, the entity of atlanto-occipital disassociation must be excluded (Fig. 16). When severe hyperflexion or hyperextension combined with distraction occurs in the upper cervical spine, atlanto-occipital disassociation may occur. Atlanto-occipital disassociation (dislocation) is characterized by complete disruption of ligamentous attachments between the occiput and the atlas.[119,120] Death commonly occurs due to concurrent injury to the brainstem.[119,121,122] Radiographically, pathologic separation between the base of the occiput and the arch of atlas is noted.[120] Cervical traction is absolutely contraindicated with atlanto-occipital disassociation since further stretching of the brainstem can occur. Atlanto-occipital disassociation may initially be evaluated with lateral plain radiographs, but it is most reliably detected with CT imaging.

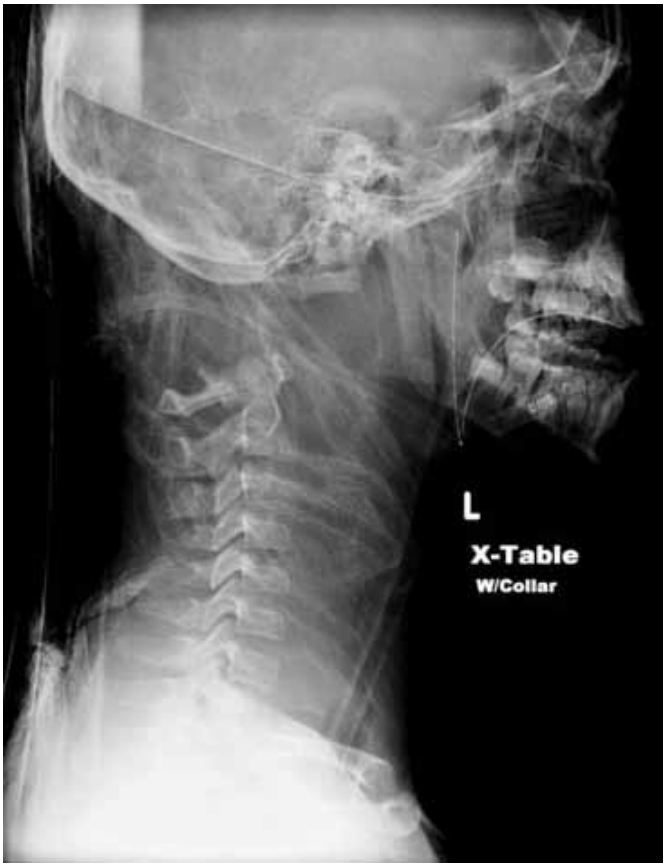

Figure 16. *Atlanto-occipital disassociation.*

An unstable or malaligned cervical spine requires either more stable immobilization or axial traction to achieve reduction.[123,124,125] Two types of axial

traction devices are available: Gardner-Wells tongs and the halo ring apparatus (Tables 4 and 5).[126,127,128] Gardner-Wells tongs are a simple, effective means of applying axial traction for reduction, but they do not significantly limit voluntary rotation, flexion, or extension in an uncooperative patient (Figs. 17 and 18). Gardner-Wells tongs can be applied with minimal skin preparation and without assistance. In contrast, the halo ring allows axial traction for reduction and provides rather stable immobilization with the application of a vest but its application requires an assistant, and it takes longer to apply than Gardner-Wells tongs. Traction devices are typically utilized at the Level III CSH where appropriate radiographic support and a more secure setting are found.

Gardner-Wells Tong Application
• After the patient is placed in a supine position, the provider identifies the external auditory meatus, bilaterally • A position on the skin is marked one centimeter (cm) superior and one cm anterior to the external auditory meatus • The skin is infiltrated with lidocaine with epinephrine to assist with pain control and bleeding from the scalp • The tongs are placed over the crown of the head with the pins positioned on the skin prepared area • The pins are inserted into the skull by symmetrically tightening the knobs • Weights are then applied to the tongs such that the traction vector is directed superiorly • Traction should be initiated at 10 pounds (lbs) and increased by five- to 10-lb increments (5 lb weight added for each spinal level) • Reduction should be performed in awake patients with administration of intravenous sedation and analgesia, as necessary • Fluoroscopy or serial radiographs and serial neurologic examinations should be performed to detect excessive distraction of spinal segments • In patients with neurologic symptoms or signs or one centimeter distraction of a disk space, closed reduction should be stopped and further images taken

Table 4. *Gardner-Wells Tong application.*

Halo Ring Application
• The correct ring size is selected according to head circumference • The ring is placed around the head, at a level one centimeter above the eyebrows and is held temporarily with plastic pod attachments • After the patient's eyelids are closed, the skin is prepared, and local anesthetic is infiltrated through the ring holes • Pins are then placed through the ring holes and are torqued down in an opposing fashion to 8 inch-pounds in adults and 4 inch-pounds in pediatric patients 8 years or younger • After 24 hours, these pins will require reevaluation of proper torque as they often loosen early on

Table 5. *Halo Ring application.*

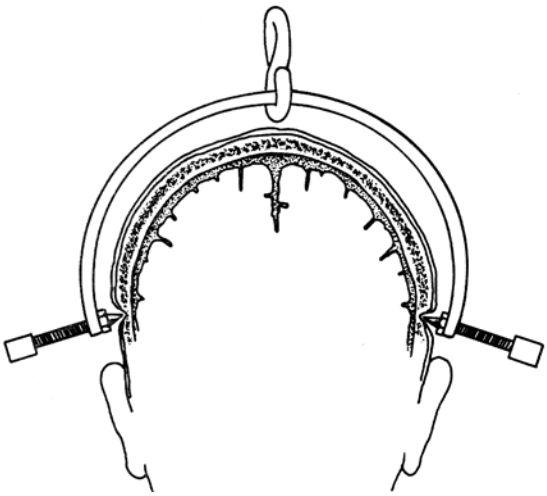

Figure 17. *Gardner-Wells tongs are applied when cervical spine traction is desired. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.*

Figure 18. *Atlanto-occiptal disassociation must be ruled out prior to application of cervical traction with Gardner-Wells tongs. Image courtesy of Jonathan Martin, MD, Connecticut Children's Medical Center.*

If traction is applied, radiographs must be obtained to ascertain that no undiagnosed ligamentous injury has been exacerbated by the added weight.

A halo ring can be applied when definitive treatment is anticipated to be in a halo or in cases in which distraction cannot be achieved with Gardner-Wells tongs. Placing a halo vest underneath the patient, prior to or during transfer to the bed, can help attach the ring to the vest while the patient is in traction after reduction. Open halo rings offer the advantage over previous whole rings in their ability to be placed without putting the patient's head on a head holder off the stretcher.

Penetrating Injury to the Spine

Management

Mechanical Stability

The majority of gunshot wounds to the spine in patients with normal neurological exams are mechanically stable.[14,15,16] In assessing the stability of the cervical spine, it has been noted that 36 percent of the weight of the head is carried by the anterior vertebral bodies and disks and 32 percent by each of the two posterolateral columns, which are composed of facet joints and lateral masses.[129] In the vast majority of cases, the projectile does not destabilize the spine, and collars or any other type of bracing are not necessary. However, the cervical collar does have the benefit of maintaining a neutral neck position that may be important in the setting of head injuries.

The majority of gunshot wounds to the spine are mechanically stable in patients with normal neurological exams. Unlike closed spinal cord injuries, it is rarely necessary to operate on gunshot wounds to the spine for purposes of establishing mechanical spinal stability.

In the thoracic and lumbar spine, the three-column concept of Denis can be applied, but the careprovider

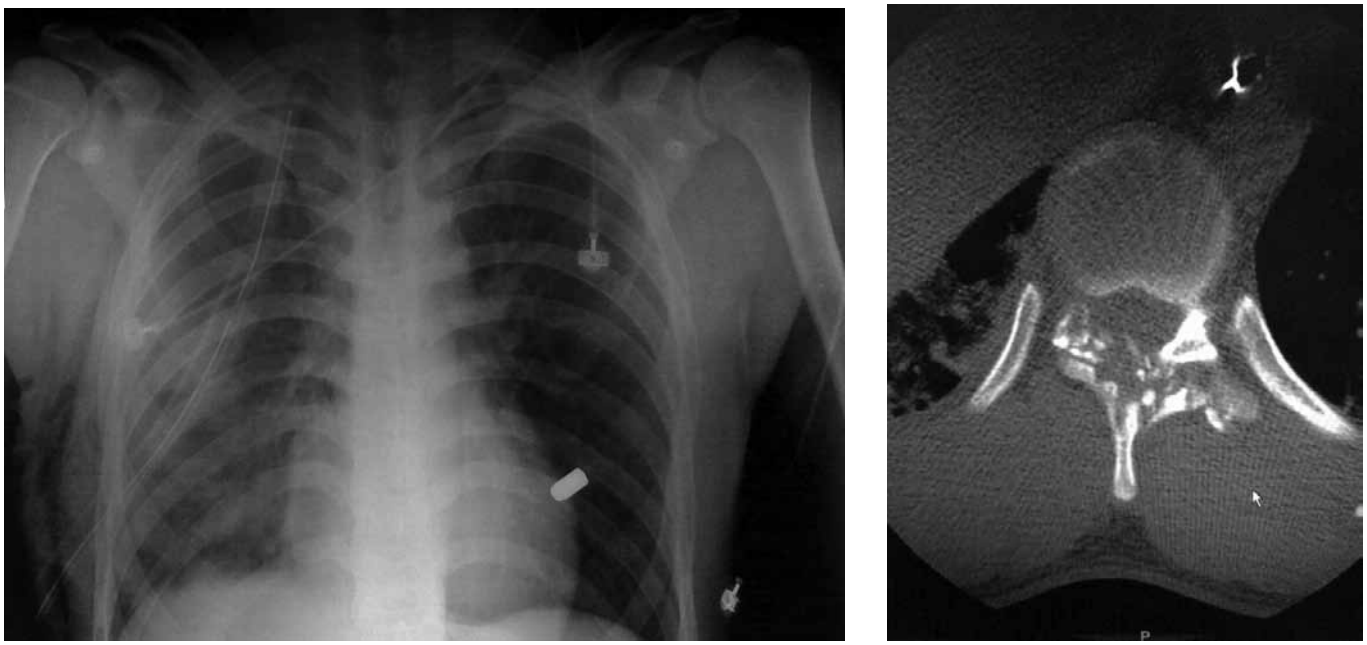

Figure 19. *The majority of gunshot wounds to the spine are mechanically stable in patients with normal neurologic exams:* (Left) *This patient sustained a transthoracic gunshot wound. Right-sided hemothorax is noted and the missile is resting in the subcutaneous space of the posterior thorax.*(Right) *Mechanical stability was determined when CT images demonstrated isolated injury to the posterior column.*

should understand that the mechanism of destruction is considerably different from that in the closed injuries for which this classification was designed.[129] When destruction is limited to one of the three columns, then no particular immobilization is needed (Fig. 19). If two or three columns are compromised by the gunshot wound, then use of spine precautions and immobilization are recommended until definitive management occurs at Level IV and V facilities.

Unlike closed spinal cord injuries, it is rarely necessary to operate on gunshot wounds of the spine for purposes of establishing stability. The length of immobilization for mechanically unstable injuries of the cervical, thoracic, or lumbar spine is normally six to eight weeks. At that time, further radiographic imaging of the affected region is performed to establish whether the spine has adequately healed and is stable.

Infection Control

Disruption of the dura (e.g., open spinal injury) is associated with a significant risk of central nervous system infection.[130] Reports from combat settings (i.e., Iran-Iraq, Vietnam, and Lebanese conflicts) have documented infection rates of 4 to 11 percent following penetrating brain injuries.[131,132,133] The benefits of administering prophylactic antibiotics prior to elective neurosurgical procedures have been used to justify the administration of prophylactic antibiotics following penetrating injuries to the spine.[134]

Prophylactic antibiotics may be of benefit following open spinal injury given the significant risk of central nervous system infection.

If a hollow viscus is not violated with penetrating spine injuries, the administration of three to five days of prophylactic parenteral antibiotics has been recommended.[135] Cefazolin, one gram intravenously every 8 hours, is often given in these situations.[136] If a concern for meningitis exists, as in the case of persistent

cerebrospinal fluid (CSF) leakage after dural violation by a projectile, ceftriaxone (one gram intravenously every 12 hours) is often prescribed.[137] Better defining the role (e.g., optimal duration of therapy, choice of antibiotic) of these treatment regimens requires future study.

It is also important to consider associated hollow viscus injuries in patients with penetrating projectile injury to the spine. If the projectile has potentially penetrated the pharynx, esophagus, or colon, then extra precautions should be taken to prevent spine infection.[129] This is essential only when the bullet has first penetrated the viscus and then penetrated the spine, and it does not seem to be clinically important if the bullet first traversed the spine before perforating the viscus. In contrast to prior recommendations, which promoted radical spine debridement, the best results have been reported by Roffi and coworkers, who recommended minimal spine debridement and one to two weeks of prophylactic parenteral antibiotics.[138] The parenteral antibiotics should be broad-spectrum agents directed at the particular bacteria normally associated with hollow viscus injury.[139]

In contrast to prior recommendations that advocated radical spine debridement for cases of penetrating spinal injury with associated hollow viscus violation, current evidence supports minimal spinal debridement in conjunction with parenteral antibiotics.

Complications: Penetrating Spine Trauma

Complications from penetrating injuries to the spine are a concern for CCC providers. Even in the face of proper initial triage and management, these complications may present either early in the treatment course or up to several weeks later. It is absolutely critical that CCC providers promptly recognize and treat these complications as they can have devastating effects on patients. Cerebrospinal fluid fistulae, infection, and vascular injuries are some of the more common situations that may be faced by the battlefield medical team.[3,4,5,131,132,133]

Cerebrospinal Fluid Fistulae

Cerebrospinal fluid fistulae have been recognized after penetrating injuries, and they are defined as an abnormal CSF conduit within either the skin or body cavities.[4,31] Stauffer et al. studied bullet removal following penetrating spinal injury and found that CSF fistulae commonly occurred after surgical treatment with laminectomy.[140] The incidence of CSF fistulae was 6 percent in patients treated with laminectomy, debridement, irrigation and bullet removal.[140] As previously stated, routine surgical intervention following spine injuries with stable neurological exams does not change neurologic outcomes.[141] The fact that most CSF fistulae occur following surgical interventions is yet another reason to reserve surgical intervention for later in the treatment course, unless emergent decompression due to neurological deterioration is indicated.

If emergency spinal surgery is performed (e.g., due to neurologic deterioration), it is imperative that meticulous dural repair occurs. Augmentation of dural repair with tight closure of the paraspinal muscles, fascia, and skin will minimize the occurrence of postoperative CSF fistulae.

Meningitis is a devastating complication of persistent CSF leakage and it is imperative to avoid this dreaded complication.[4,142] When spinal surgery is performed, meticulous dural repair is essential. Augmentation of dural repair with tight closure of the paraspinal muscles, fascia, and skin will minimize the occurrence of postoperative CSF fistulae (Fig. 20). Likewise, it is advised that an intraoperative valsalva

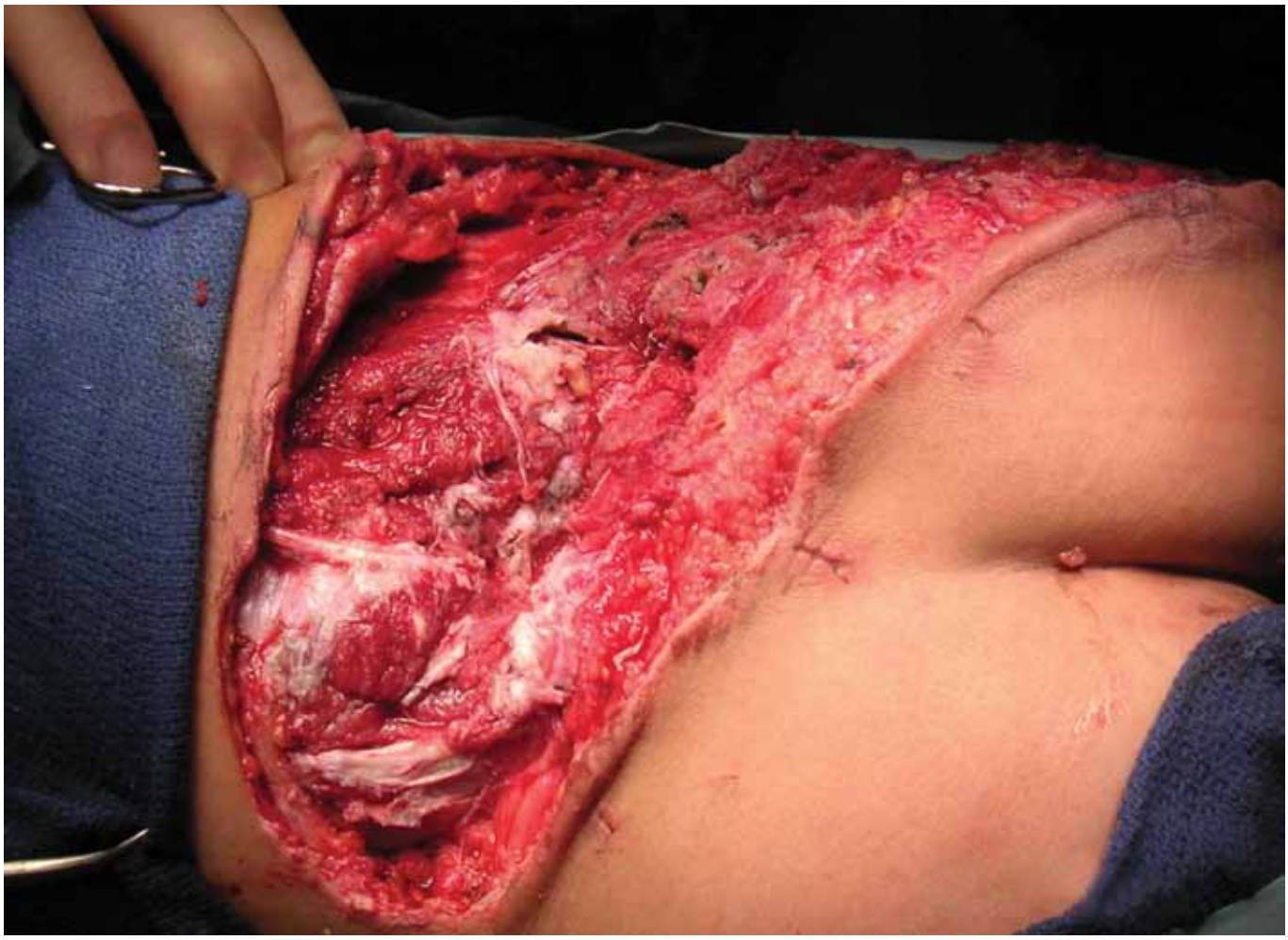

Figure 20. *Pediatric host nation patient with penetrating spinal injury (patient's lumbar region). After wound debridement, a gluteal flap was created and rotated over the spinal canal to prevent CSF leakage and infection. Image courtesy of the Borden Institute, Office of The Surgeon General, Washington, DC.*

maneuver should be performed after the dural leak is repaired to ensure the seal is watertight, and if persistence of the leakage occurs, a lumbar drain should be placed. Most Level II and III facilities will not have subarachnoid (lumbar) drains on-hand, thus providing another reason to delay elective surgical interventions.

CSF fistulae can either track cutaneously (externally) or into other body cavities (internally). The cutaneous variety typically are easily identifiable. However, fistulae emptying into a deep body cavity can be overlooked. When a penetrating spinal injury patient has persistent postural headaches, the existence of a CSF fistulae with a course that tracks to an internal cavity should be suspected. Radionucleotide studies can be effective in confirming the diagnosis and localizing the tract.[143] However, these studies require introduction of the radionucleotide into the subarachnoid space and are not available to Level II and III CCC providers. When a CSF fistula is suspected, the placement of a lumbar (subarachnoid) drain is recommended followed by supine patient body positioning pending resolution of the leak.

Spinal Infections

Spinal infections following penetrating spinal injury are often accompanied by perforation of a hollow

viscus.[138] Postoperative spine infections can be another common scenario in association with penetrating spinal injury. Stauffer found the rate to be 4 percent after decompressive laminectomy with projectile removal.[140] For these reasons, perioperative antibiotic prophylaxis and the administration of antibiotics for up to 14 days post-injury have been recommended when a dural tear is in communication with a hollow viscus injury.[144] In such patients, an internal fistula may also be present in association with the spine infection. It is virtually impossible to resolve a spine infection unless the fistula is corrected. General surgery techniques such as diversion of the drainage with nutritional hyperalimentation may be necessary when a deep space CSF fistula is present.

Local surgical exploration, debridement, or diversion may be required in cases of penetrating spinal injury associated with hollow viscus involvement, given the risk of infection.

In the face of progressive paralysis or deformity in association with spine infection, the necessity to identify an organism, problematic foreign body, or failure of nonoperative management are all indications to proceed with exploratory surgery. In most cases, spine infections may not be readily apparent until later in the evacuation of the battlefield trauma patient, and CT-guided biopsy with abscess drainage along with parenteral antibiotic administration is the typical management strategy. Routinely, the Level V facility has this capability.

Vascular Injuries

Vascular injury must be considered in all patients with penetrating injury to the spine. The vertebral arteries within the transverse foramina of the cervical spine, the thoracic aorta associated with the thoracic spine, and the iliac vessels anterior to the lumbar spine are vulnerable to injury following penetrating spine trauma. Suspicion for vascular injury should be further heightened in the face of progressive anemia or persistent hypotension in the spine trauma patient. Current recommendations advise that wounds should be explored when significant warning signs for vascular injury are present.[145,146] Hard signs such as pulsatile bleeding, neurovascular compromise, an expanding hematoma, and a palpable thrill are obvious warning signs for vascular injury.

Projectile in the Disc Space

Several factors have traditionally been considered in deciding whether surgery is indicated when projectiles are located in the disk space. The first consideration is whether lead poisoning (plumbism) or other projectile-associated toxicity will develop. Reports in the literature suggest that the lead is leached out of a bullet that is bathed in synovial fluid, and lead poisoning can subsequently occur.[129] Plumbism is a late complication and is typically not an immediate concern for CCC providers. Second, the careprovider should determine whether mechanical disruption of physiologic spinal segment movement has resulted from the presence of a projectile within the disk. Mechanical stability is an issue when the patient is placed upright and typically can be dealt with at higher levels of care outside of the battlefield. Lastly, it must be determined whether disk extrusion has resulted from a penetration of the disk space by the projectile. If disk extrusion leading to symptomatic neural compression occurs, neural decompression and removal of the disk fragments are indicated. This occurrence is extremely uncommon, but it has been reported in the literature.[129]

The need to remove a projectile resting in the disk space is rare. If this operative intervention is required, it should be performed at Level IV or V facilities, unless progressive neurologic deterioration is noted at a Level III facility and skilled spine surgeons are available to operatively intervene.

Projectile in the Spinal Canal

Many anecdotal articles have been written concerning removal of bullets (projectiles) from the spinal canal.[97,140,147,148,149,150] Prior to the 1990's, this topic had not been studied in a methodologically rigorous manner. For study conclusions to be robust, the two study groups must have equivalent pathology, with one group having bullets removed and the other group having bullets left in place. It is also important that this study be done on a prospective basis, recording adequate neurologic information as well as quantitative assessment of clinical variables (e.g., pain). A well-designed study of the removal of bullets from within the spinal canal was performed by Waters et al.[31] The study reviewed 90 cases of patients with bullets lodged within their spinal canals, of whom 32 had bullets removed and 58 had bullets left in place. They concluded that between the T12 (thoracic) to L5 (lumbar) spinal levels statistically significant neurologic motor improvement occurred with removal of the bullet from the spinal canal. There was no difference, however, in sensation or in pain experienced by the patients. In thoracic spine injuries, from T1 to T11, no statistical difference was seen for either complete or incomplete injuries, whether or not the bullet was removed. Similarly, no difference was seen with bullet removal in the cervical spine; however, the authors suggest that the patient numbers were too small to be able to draw statistical conclusions about the cervical spine.

The medical literature does not support the routine removal of projectiles resting in the spinal canal when located between T1 to T12 vertebral bodies. For projectiles in the canal inferior to T12 vertebral body, removal of the projectile in-theater should only be attempted if the neurological exam is deteriorating. If the exam remains unchanged or improves, then removal may occur at Level IV or V facilities. There is insufficient data to provide more definitive recommendations regarding cervical spinal canal bullet fragment removal.

Elective removal of projectiles from within the spinal canal should ideally be performed seven to 10 days following the injury. This time lag will minimize many cases of CSF leakage and considerably simplify dural tear management.

Once the decision has been made to surgically remove the projectile from the spinal canal, it is essential that a scout radiograph be taken in the operating room before the incision is made. The reason for this is that the projectile can occasionally migrate within the spinal canal, depending on the position of the patient.[129] This is especially true for patients with large spinal canals and relatively small embedded projectiles. Elective removal of projectiles from within the spinal canal should ideally be performed seven to 10 days following the injury. This time lag will minimize many cases of CSF leakage and considerably simplify dural tear management.[129]

Summary

Spinal injuries cause significant morbidity. Spinal injury patients often have multiple coexisting, life-threatening injuries. Knowledge of spinal anatomy is necessary to accurately evaluate and manage spinal injury patients. Combat casualty care providers must have an accurate understanding of the emergency management, diagnostic evaluation (e.g., radiological studies), transport considerations, and therapeutic management of spinal injury patients.

References

1. American College of Surgeons Committee on Trauma. Advanced trauma life support program for doctors. 7th ed. Chicago, IL: American College of Surgeons; 2004.

2. Marshall LF, Knowlton S, Garfin SR, et al. Deterioration following spinal cord injury. A multicenter study. J Neurosurg 1987;66(3):400-404.

3. Hammoud MA, Haddad FS, Moufarrij NA. Spinal cord missile injuries during the Lebanese civil war. Surg Neurol 1995;43(5):432-437; discussion 437-442.

4. Kahraman S, Gonul E, Kayali H, et al. Retrospective analysis of spinal missile injuries. Neurosurg Rev 2004;27(1):42-45.

5. Splavski B, Vrankovic D, Saric G, et al. Early management of war missile spine and spinal cord injuries: experience with 21 cases. Injury 1996;27(10):699-702.

6. Weaver FM, Burns SP, Evans CT, et al. Provider perspectives on soldiers with new spinal cord injuries returning from Iraq and Afghanistan. Arch Phys Med Rehabil 2009;90(3):517-521.

7. Kraus JF, Franti CE, Riggins RS, et al. Incidence of traumatic spinal cord lesions. J Chronic Dis 1975;28(9):471-492.

8. Ergas Z. Spinal cord injury in the United States: a statistical update. Cent Nerv Sys Trauma 1985;2(1):19-32.

9. Price C, Makintubee S, Hemdon W, et al. Epidemiology of traumatic spinal cord injury and acute hospitalization and rehabilitation charges for spinal cord injuries in Oklahoma, 1988-1990. Am J Epidemiol 1994;139:37-47.

10. Eastridge BJ, Jenkins D, Flaherty S, et al. Trauma system development in a theater of war: experiences from Operation Iraqi Freedom and Operation Enduring Freedom. J Trauma 2006;61(6):1366-1372; discussion 1372-1373.

11. Carlton PK Jr, Jenkins DH. The mobile patient. Crit Care Med 2008;36(7 Suppl):S255-257.

12. Ling GS, Rhee P, Ecklund JM. Surgical innovations arising from the Iraq and Afghanistan wars. Annu Rev Med 2010;61:457-468.

13. Harman DR, Hooper TI, Gackstetter GD. Aeromedical evacuations from Operation Iraqi Freedom: a descriptive study. Mil Med 2005;170(6):521-527.

14. Arishita GI, Vayer JS, Bellamy RF. Cervical spine immobilization of penetrating neck wounds in a hostile environment. J Trauma 1989;29(3):332-337.

15. Ramasamy A, Midwinter M, Mahoney P, et al. Learning the lessons from conflict: pre-hospital cervical spine stabilisation following ballistic neck trauma. Injury 2009;40(12):1342-1345.

16. Barkana Y, Stein M, Scope A, et al. Prehospital stabilization of the cervical spine for penetrating injuries of the neck—is it necessary? Injury 2000;31(5):305-309.

17. Saboe LA, Reid DC, Davis LA, et al. Spine trauma and associated injuries. J Trauma 1991;31(1):43-48.

18. Anderson PA, Rivara FP, Maier RV, et al. The epidemiology of seatbelt-associated injuries. J Trauma 1991;31(1):60-67.

19. American Spinal Injury Association. Standard neurological classification of spinal cord injury. 2006 [cited 2010 May 12]. Available from: URL: http://www.asia-spinalinjury.org/publications/2006_Classif_worksheet.pdf.

20. Gupta A, el Masri WS. Multilevel spinal injuries. Incidence, distribution and neurological patterns. J Bone Joint Surg Br 1989;71(4):692-695.

21. Hadden WA, Gillespie WJ. Multiple level injuries of the cervical spine. Injury 1985;16(9):628-633.

22. Henderson RL, Reid DC, Saboe LA. Multiple noncontiguous spine fractures. Spine 1991;16(2):128-131.

23. Korres DS, Katsaros A, Pantazopoulos T, et al. Double or multiple level fractures of the spine. Injury 1981;13(2):147-152.

24. Vaccaro AR, An HS, Lin S, et al. Noncontiguous injuries of the spine. J Spinal Disord 1992;5(3):320-329.

25. Dorland's Illustrated Medical Dictionary. 29th ed. Philadelphia, PA: W.B. Saunders; 1980. p. 1633.

26. Atkinson PP, Atkinson JL. Spinal shock. Mayo Clin Proc 1996;71(4):384-389.

27. Abdel-Azim M, Sullivan M, Yalla SV. Disorders of bladder function in spinal cord disease. Neurol Cin 1991;9(3):727-740.

28. Stauffer ES. Neurologic recovery following injuries to the cervical spinal cord and nerve roots. Spine 1984;9(5):532-534.

29. Schneider RC, Crosby ED, Russo RH, et al. Chapter 32. Traumatic spinal cord syndromes and their management. Clin Neurosurg 1973;20:424-492.

30. Stevens RD, Bhardwaj A, Kirsch JR, et al. Critical care and perioperative management in traumatic spinal cord injury. J Neurosurg Anesthesiol 2003;15(3):215-229.

31. Waters RL, Adkins RH. The effects of removal of bullet fragments retained in the spinal canal. Spine 1991;16(8):934-939.

32. Zipnick RI, Scalea TM, Trooskin SZ, et al. Hemodynamic responses to penetrating spinal cord injuries. J Trauma 1993;35(4):578-582; discussion 582-583.

33. Colterjohn NR, Bednar DA. Identifiable risk factors for secondary neurologic deterioration in the cervical spine-injured patient. Spine 1995;21:2293-2297.

34. Curry K, Cassidy L. The relationship between extended periods of immobility and decubitus ulcer formation in the acutely spinal cord-injured patient. J Neurosci Nurs 1992;24(4):185-189.

35. Linares HA, Mawson AR, Suarez E, et al. Association between pressure sores and immobilisation in the immediate post-injury period. Orthopedics 1987;10(4):571-573.

36. Gupta MC, Benson DR, Keenen TL. Initial evaluation and emergency treatment of the spine-injured patient. In: Browner BD, Green NE, editors. Skeletal trauma. 4th ed. WB Saunders Co; 2008. p. 730-778.

37. Podolsky S, Baraff LJ, Simon RR. Efficacy of cervical spine immobilization methods. J Trauma 1983;23(6):461-465.

38. Conrad BP, Horodyski M, Wright J, et al. Log-rolling technique producing unacceptable motion during body position changes in patients with traumatic spinal cord injury. J Neurosurg Spine 2007;6(6):540-543.

39. DiPaola CP, DiPaola MJ, Conrad BP, et al. Comparison of thoracolumbar motion produced by manual and Jackson-table-turning methods. Study of a cadaveric instability model. J Bone Joint Surg Am 2008;90(8):1698-1704.

40. McGuire RA, Neville S, Green BA, et al. Spinal instability and the log-rolling maneuver. J Trauma 1987;27(5):525-531.

41. Holly LT, Kelly DF, Counelis GJ, et al. Cervical spine trauma associated with moderate and severe head injury: incidence, risk factors, and injury characteristics. J Neurosurg 2002;96(3 Suppl):285-291.

42. Tator CH. Hemodynamic issues and vascular factors in acute experimental spinal cord injury. J Neurotrauma 1992;9(2):139-140; discussion 141.

43. Rengachary SS, Alton SM. Resuscitation and early medical management of the spinal cord injury patient. In: Tator CH, Benzel EC, editors. Contemporary management of spinal cord injury: from impact to rehabilitation. 2nd ed. Park Ridge, IL: American Academy of Neurologic Surgeons Publishing; 2000. p. 61-73.

44. Dolan EJ, Tator CH. The effect of blood transfusion, dopamine, and gamma hydroxybutyrate on

posttraumatic ischemia of the spinal cord. J Neurosurg 1982;56(3):350-358.

45. Ducker TB, Saleman M, Perot PL, et al. Experimental spinal cord trauma, I: correlation of blood flow, tissue oxygen and neurologic status in the dog. Surg Neurol 1978;10(1):60-63.

46. Kobrine AI, Doyle TF, Rizzoli HV. Spinal cord blood flow as affected by changes in systemic arterial blood pressure. J Neurosurg 1976;44(1):12-15.

47. Tator CH, Fehlings MG. Review of the secondary injury theory of acute spinal cord trauma with emphasis on vascular mechanisms. J Neurosurg 1991;75(1):15-26.

48. Levi L, Wolf A, Belzberg H. Hemodynamic parameters in patients with acute cervical cord trauma: description, intervention, and prediction of outcome. Neurosurgery 1993;33(6):1007-1016; discussion 1016-1017.

49. Zach GA, Seiler W, Dollfus P. Treatment results of spinal cord injuries in the Swiss Paraplegic Centre of Basel. Paraplegia 1976;14(1):58-65.

50. Stratman RC, Wiesner AM, Smith KM, et al. Hemodynamic management after spinal cord injury. Orthopedics 2008;31(3):252-255.

51. Vale FL, Burns J, Jackson AB, et al. Combined medical and surgical treatment after spinal cord injury: result of a prospective pilot study to assess the merits of aggressive medical resuscitation and blood pressure management. J Neurosurg 1997;87(2):329-246.

52. Napolitano LM, Kurek S, Luchette FA, et al. Clinical practice guideline: red blood cell transfusion in adult trauma and critical care. Crit Care Med 2009;37(12):3124-3157.

53. Piepmeier JM, Lehmann KB, Lane JG. Cardiovascular instability following acute cervical spinal cord trauma. Cent Nerv Sys Trauma 1985;2(3):153-160.

54. Albuquerque F, Wolf A, Dunham CM, et al. Frequency of intraabdominal injury in cases of blunt trauma to the cervical spinal cord. J Spinal Disord 1992;5(4):476-480.

55. Grundy D, Swain A, Russell J. ABC of spinal cord injury: early management and complication—I. Br Med J (Clin Res Ed) 1986;292(6512):44-47.

56. Short DJ, Masry WS, Jones PW. High dose methylprednisolone in the management of acute spinal cord injury—a systematic review from a clinical perspective. Spinal Cord 2000;38(5):273-286.

57. Hugenholtz H. Methylprednisolone for acute spinal cord injury: not a standard of care. CMAJ 2003;168(9):1145-1146.

58. Nesathurai S. Steroids and spinal cord injury: revisiting the NASCIS 2 and NASCIS 3 trials. J Trauma 1998;45(6):1088-1093.

59. Eck JC, Nachtigall D, Humphreys SC, et al. Questionnaire survey of spine surgeons on the use of methylprednisolone for acute spinal cord injury. Spine 2006;31(9):E250-253.

60. Prendergast MR, Saxe JM, Ledgerwood AM, et al. Massive steroids do not reduce the zone of injury after penetrating spinal cord injury. J Trauma 1994;37(4):576-579; discussion 579-580.

61. Bracken MB, Shepard MJ, Collins WF, et al. A randomized controlled trial of methylprednisolone or naloxone in the treatment of acute spinal cord injury: the results of the National Acute Spinal Cord Injury Study. N Engl J Med 1990;322(20):1405-1411.

62. Pharmacological therapy after acute spinal cord injury. Neurosurgery 2002;50(3 Suppl):S63-72.

63. Bracken MB, Shepard MJ, Collins WF Jr, et al. Methylprednisolone or naloxone treatment after acute spinal cord injury: 1 year follow-up data. Results of the Second National Acute Spinal Cord Injury Study. J Neurosurg 1992;76(1):23-31.

64. Bracken MB. Pharmacological treatment of acute spinal cord injury: current status and future projects. J Emerg Med 1993;11 (Suppl 1):43-48.

65. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methylprednisolone for 24 and 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury: results of the third National Acute Spinal Cord Injury Randomized Controlled Trial. JAMA 1997;277(20):1597-1604.

66. Bracken MB, Collins WF, Freeman D, et al. Efficacy of methylprednisolone in acute spinal cord injury. JAMA 1984;251(1):45-52.

67. Ducker TB, Zeidman SM. Spinal cord injury: role of steroid therapy. Spine 1994;19(20):2281-2287.

68. Hall ED. The neuroprotective pharmacology of methylprednisolone. J Neurosurg 1992;76(1):13-22.

69. Fife D, Kraus J. Anatomic location of spinal cord injury relationship to cause of injury. Spine 1986;11(1):2-5.

70. Meyer PR Jr, Cybulski GR, Rusin JJ, et al. Spinal cord injury. Neurol Clin 1991;9(3):625-61.

71. Riggins RS, Kraus JF. The risk of neurologic damage with fractures of the vertebrae. J Trauma 1977;17(2):126-133.

72. Wagner FC Jr. Management of acute spinal cord injury. Surg Neurol 1977;7(6):346-350.

73. Terregino CA, Ross SE, Lipinski MF, et al. Selective indications for thoracic and lumbar radiography in blunt trauma. Ann Emerg Med 1995;26(2):126-129.

74. Samuels LE, Kerstein MD. 'Routine' radiologic evaluation of the thoracolumbar spine in blunt trauma patients: a reappraisal. J Trauma 1993;34(1):85-89.

75. Reid DC, Henderson R, Saboe L, et al. Etiology and clinical course of missed spine fractures. J Trauma 1987;27(9):980-986.

76. Hoffman JR, Mower WR, Wolfson AB, et al. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. National Emergency X-Radiography Utilization Study Group. N Engl J Med 2000;343(2):94-99.

77. Winslow JE III, Hensberry R, Bozeman WP, et al. Risk of thoracolumbar fractures doubled in victims of motor vehicle collisions with cervical spine fractures. J Trauma 2006;61(3):686-687.

78. Savitsky E, Votey S. Emergency department approach to acute thoracolumbar spine injury. J Emerg Med 1997;15(1):49-60.

79. Holmes JF, Akkinepalli R. Computed tomography versus plain radiography to screen cervical spine injury: a meta-analysis. J Trauma 2005;58(5):902-905.

80. Ross SE, Schwab CW, David ET, et al. Clearing the cervical spine: initial radiographic evaluation. J Trauma 1987;27(9):1055-1060.

81. Groves CJ, Cassar-Pullicino VN, Tins BJ, et al. Chance-type flexion-distraction injuries in the thoracolumbar spine: MR imaging characteristics. Radiology 2005;236(2):601-608.

82. Pepin JW, Bourne RB, Hawkins RJ. Odontoid fracture, with special reference to the elderly patient. Clin Orthop 1985;193:178-183.

83. Lin JT, Lee JL, Lee ST. Evaluation of occult cervical spine fractures on radiographs and CT. Emerg Radiol 2003;10(3):128-134.

84. O'Callaghan JP, Ullrich CG, Yuan HA, et al. CT of facet distraction in flexion injuries of the thoracolumbar spine: the "naked" facet. AJR Am J Roentgenol 1990;134(3)563-568.

85. Qaiyum M, Tyrrell PNM, McCall IW, et al. MRI detection of unsuspected vertebral injury in acute spinal trauma: incidence and significance. Skeletal Radiol 2001;30(6):299-304.

86. Grabb PA, Pang D. Magnetic resonance imaging in the evaluation of spinal cord imaging without radiographic abnormality in children. Neurosurgery 1994;35(3):406-414.

87. White AA, Panjabi MM. Clinical biomechanics of the spine. 2nd ed. Philadelphia, PA: JB Lippincott; 1990.

88. White AA III, Johnson RM, Panjabi MM, et al. Biomechanical analysis of clinical stability in the cervical spine. Clin Orthop 1975;109:85–96.

89. Denis F. The three-column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine 1983;8(8):817-831.

90. Stauffer ES. Diagnosis and prognosis of acute cervical spinal cord injury. Clin Orthop Relat Res 1975;112:9-15.

91. Roth EJ, Park T, Pang T, et al. Traumatic cervical Brown-Sequard and Brown-Sequard-plus syndromes: the spectrum of presentations and outcomes. Paraplegia 1991;29(9):582-589.

92. Schneider RS, Thompson JM, Bebin J. The syndrome of acute anterior cervical spinal cord injury. J Neurosurg 1958;21:216.

93. Foo D, Subrahmanyan TS, Rossier AB. Post-traumatic acute anterior spinal cord syndrome. Paraplegia 1981;19(4):201-205.

94. Bosch A, Stauffer ES, Nickel VL. Incomplete traumatic quadriplegia: a ten-year review. JAMA 1971;216(3):473-478.

95. Schrader SC, Sloan TB, Toleikis R. Detection of sacral sparing in acute spinal cord injury. Spine 1987;12(6):533-535.

96. Scheider RC, Cheery G, Pantek H. The syndrome of acute central cervical spinal cord injury; with special reference to the mechanisms involved in hyperextension injuries of cervical spine. J Neurosurg 1954;11(6):546-577.

97. Heiden JS, Wess MH, Rosenberg AW, et al. Management of cervical spine cord trauma in Southern California. J Neurosurg 1975;43(6):732-736.

98. Mortara RW, Flanagan M. Acute central cervical spinal cord syndrome caused by a missile injury: case report and brief review of syndrome. Neurosurgery 1980;6(2):176-180.

99. Gentleman D, Harrington M. Penetrating injury to the spinal cord. Injury 1984;16(1):7-8.

100. Nissen SJ, Laskowski ER, Rizzo TD Jr. Burner syndrome: recognition and rehabilitation. Phys Sportsmed 1996;24(6):57-64.

101. Robertson WC Jr, Eichman PL, Clancy WG. Upper trunk brachial plexopathy in football players. JAMA 1979;241(14):1480-1482.

102. Poindexter DP, Johnson EW. Football shoulder and neck injury: a study of the "stinger." Arch Phys Med Rehabil 1984;65(10):601-602.

103. Pang D, Wilberger JE Jr. Spinal cord injury without radiographic abnormalities in children. J Neurosurg 1982;57(1):114-129.

104. Pollack IF, Pang D, Sclabassi R. Recurrent spinal cord injury without radiographic abnormalities in children. J Neurosurg 1988;69(2):177-182.

105. Hayashi K, Yone K, Ito H, et al. MRI findings in patients with a cervical spinal cord injury who do not show radiographic evidence of a fracture or dislocation. Paraplegia 1995;33(4):212-215.

106. Hendey GW, Wolfson AM, Mower WR, et al. Spinal cord injury without radiographic abnormality: results of the National Emergency X-Radiography Utilization Study in blunt cervical trauma. J Trauma 2002;53(1):1-4.

107. Chan D, Goldberg R, Tascone A, et al. The effect of spinal immobilization on healthy volunteers. Ann Emerg Med 1994;23(1):48-51.

108. Burke DC, Murray DD. The management of thoracic and thoraco-lumbar injuries of the spine with neurological involvement. J Bone Joint Surg Br 1976;58:72-78.

109. Tator CH, Rowed DW. Current concepts in the immediate management of acute spinal cord injuries. Can Med Assoc J 1979;121(11):1453-1464.

110. Wilmot CB, Hall KN. Evaluation of acute surgical intervention in traumatic paraplegia. Paraplegia 1986;24(2):71-76.

111. Tator CH, Duncan EG, Edmonds VE, et al. Comparison of surgical and conservative management in 208 patients with acute spinal cord injury. Can J Neurol Sci 1987;14(1):60-69.

112. Krompinger WJ, Frederickson BE, Mino DE, et al. Conservative treatment of fracture of the thoracic and lumbar spine. Orthoped Clin North Am 1986;17(1):161-170.

113. Bohlman HH, Freehafer A, Dejak J. The results of treatment of acute injuries of the upper thoracic spine with paralysis. J Bone Joint Surg 1985;67:360-369.

114. Yablon IG, Palumbo M, Spatz E, et al. Nerve root recovery in complete injuries of the cervical spine. Spine 1991;16(10 Suppl):S518-521.

115. McQueen JD, Khan MI. Evaluation of patients with cervical spine lesions. In: Cervical Spine Research Society, Editorial Subcommittee, editors. The cervical spine. Philadelphia: JB Lippincott; 1983.

116. Chapman JR, Anderson PA. Thoracolumbar spine fracture with neurologic deficit. Orthoped Clin North Am 1994;25(4):595-612.

117. Dolan EJ, Tator CH, Endrenyi L. The value of decompression for acute experimental spinal cord compression injury. J Neurosurg 1980;53(6):749-755.

118. Rivlin AS, Tator CH. Effect of duration of acute spinal cord compression in a new acute cord injury model in the rat. Surg Neurol 1978;10(1):38-43.

119. Harmanli O, Koyfman Y. Traumatic atlanto-occipital dislocation with survival: a case report and review of the literature. Surg Neurol 1993;39(4):324-330.

120. Govender S, Vlok GJ, Fisher-Jeffes N, et al. Traumatic dislocation of the atlanto-occipital joint. J Bone Joint Surg Br 2003;85(6):875-878.

121. Papadopoulos SM, Dickman CA, Sonntag VK, et al. Traumatic atlantooccipital dislocation with survival. Neurosurgery 1991;28(4):574-579.

122. Matava MJ, Whitesides TE Jr, Davis PC. Traumatic atlanto-occipital dislocation with survival. Serial computerized tomography as an aid to diagnosis and reduction: a report of three cases. Spine 1993;18(13):1897-1903.

123. Star AM, Jones AA, Cotler JM, et al. Immediate closed reduction of cervical spine dislocation using traction. Spine 1990;15(10):1068-1072.

124. Cotler JM, Herbison GJ, Nasuti JF, et al. Closed reduction of traumatic cervical spine dislocations using traction weights up to 140 pounds. Spine 1993;18(3):386-390.

125. Crutchfield WG. Skeletal traction for dislocation of the cervical spine: report of a case. South Surg 1933;2:156-159.

126. Botte MJ, Byrne TP, Garfin SR. Application of the halo device for immobilization of the cervical spine utilizing an increased torque pressure. J Bone Joint Surg Am 1987;69(5):750-752.

127. Gardner W. The principle of spring-loaded points for cervical traction. Technical note. J Neurosurg 1973;39(4):543-544.

128. Garfin SR, Botte MJ, Centeno RS, et al. Osteology of the skull as it affects halo pin placement. Spine 1985;10(8):696-698.

129. Eismont F, Roper JG. Gunshot wounds of the spine. In: Browner BD, Green NE, editors. Skeletal trauma. 4th ed. Edinburgh: WB Saunders Co; 2008. p. 431-452.

130. Al-Haddad SA, Kirollos R. A 5-year study of the outcome of surgically treated depressed skull fractures. Ann R Coll Surg Engl 2002;84(3):196-200.

131. Aarabi B, Taghipour M, Alibaii E, et al. Central nervous system infections after military missile head wounds. Neurosurgery 1998;42(3):500-507; discussion 507-509.

132. Rish BL, Caveness WF, Dillon JD, et al. Analysis of brain abscess after penetrating craniocerebral injuries in Vietnam. Neurosurgery 1981;9(5):535-541.

133. Taha JM, Haddad FS, Brown JA. Intracranial infection after missile injuries to the brain: report of 30 cases from the Lebanese conflict. Neurosurgery 1991;29(6):864-868.

134. Haines SJ. Efficacy of antibiotic prophylaxis in clean neurosurgical operations. J Neurosurg 1989;24(3):401-405.

135. Bayston R, de Louvois J, Brown EM, et al. Use of antibiotics in penetrating craniocerebral injuries. "Infection in Neurosurgery" Working Party of British Society for Antimicrobial Chemotherapy. Lancet 2000;355(9217):1813-1817.

136. Klekner A, Ga'spa'r A, Kardos S, et al. Cefazolin prophylaxis in neurosurgery monitored by capillary electrophoresis. J Neurosurg Anesthesiol 2003;15(3):249-254.

137. Velanovich V. A meta-analysis of prophylactic antibiotics in head and neck surgery. Plast Reconstr Surg 1991;87(3):429-434.

138. Roffi RP, Watera RL, Adkins RH. Gunshot wounds to the spine associated with a perforated viscus. Spine 1989;14(8):808-811.

139. Lin SS, Vaccaro AR, Reisch S. Low-velocity gunshot wounds to the spine with an associated transperitoneal injury. J Spinal Disord 1995;8(2):136-144.

140. Stauffer ES, Wood RW, Kelly EG. Gunshot wounds of the spine: the effects of laminectomy. J Bone Joint Surg Am 1979;61(3):389-392.

141. Bowen TE, Bellamy RF. Emergency war surgery. Second US revision of the Emergency War Surgery NATO Handbook. Washington, DC: US Government Printing Office; 1988.

142. Kitchell S, Eismont FJ, Green BA. Closed subarachnoid drainage for management of cerebrospinal fluid leakage after an operation on the spine. J Bone Joint Surg Am 1989;71(7):984-987.

143. Gellad FE, Paul KS, Geisler FH. Early sequelae of gunshot wounds to the spine: radiologic diagnosis. Radiology 1988;167(2):523-526.

144. Buxton N. The military medical management of missile injury to the spine: a review of the literature and proposal of guidelines. J R Army Med Corps 2001;147(2):168-172.

145. Sheely CH 2nd, Mattox KL, Reul GJ Jr. Current concepts in the management of penetrating neck trauma. J Trauma 1975;15(10):895-900.

146. Bishop M, Shoemaker WC, Avakian S. Evaluation of a comprehensive algorithm for blunt and penetrating thoracic and abdominal trauma. Am Surg 1991;57(12):737-746.

147. Cybulski GR, Stone JL, Kant R. Outcome of laminectomy for civilian gunshot injuries of the terminal spinal cord and cauda equina: review of 88 cases. Neurosurgery 1989;24(3):392-397.

148. Kupcha PC, An HS, Colter JM. Gunshot wounds to the cervical spine. Spine 1990;15(10):1058-1063.

149. Simpson RK Jr, Venger BH, Narayan RK. Treatment of acute penetrating injuries to the spine: a retrospective analysis. J Trauma 1989;29(1):42-46.

150. Yashon D, Jane JA, White RJ. Prognosis and management of spinal cord and cauda equina bullet injuries in sixty-five civilians. J Neurosurg 1970;32(2):163-170.