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AHC Media

An Update on Spinal Cord Injury: Epidemiology, Diagnosis, and Treatment for the Emergency Physician

A spinal cord injury can be devastating. The National Spinal Cord Injury Statistical Center (NSCISC) has been collecting epidemiologic data on spinal cord injury (SCI) for nearly 40 years and has been able to provide the public and medical community with information on the evolution of SCI, including demographics, injury patterns, long-term prognosis, and socioeconomic impacts. There have been some interesting trends and significant changes to the data as time, technology, and culture have progressed. According to the NSCISC website, as of 2011, there are approximately 12,000 new cases of SCI annually. This figure does not include those who die from their injuries at the scene. It is believed that there are as many as 316,000 people currently living with SCI in the United States.[1] *This article will provide an update of the recent literature and standard of care in the diagnosis and treatment of acute SCI.*

— Ann M. Dietrich, MD, Editor

Epidemiology

When data collected from the early 1970s are compared to current information, general trends are noted in age at time of injury and racial/ethnic distribution of SCI. In the early 1970s, SCI was a disease of young, Caucasian males with an average age in the late 20s, and close to 70% of those affected by SCI were Caucasian, with 14% and 0.9% affecting African Americans and Asians, respectively. Since 2005, the trends demonstrate an older population being affected, with the average age at time of injury approximately 40.7 years old, likely a reflection of the increasing age of society. An appreciable increase in the incidence of SCI in the African-American and Hispanic populations has also been observed, with African Americans now accounting for close to 27% and Hispanics for 8.3% of all SCI reported annually. These changes are likely a result of the general changes in the country's population, as well as improved referral, documentation, and reporting methods throughout the country.

Motor vehicle collisions (MVC) are persistently the most common cause of SCI and represent approximately 40.4% of all reported injuries. Falls (27.9%) are second to MVC, followed by violence (15.0%) and sporting injuries (8.0%). Interestingly, falls as an etiology of SCI appear to be increasing in prevalence, whereas sporting injuries are declining. As an etiology, violence peaked between 1990 and 1999 at a rate of 24.8%, but decreased in the following decade.

From a social perspective, approximately half of patients are married at the time of their injury. Compared to national averages, these marriages are only slightly more likely to end in divorce or separation. Those individuals who are not married at the time of injury are slightly less likely, overall, to get married. Close to 58% of people are employed at the time of their injury. Not surprisingly, at the end of their first year following injury, only approximately 11.6%

Executive Summary

- Since the injury to the cord is bilateral for anterior cord syndrome, the pattern of symptoms that accompanies this injury includes bilateral motor paralysis and loss of pinprick, temperature, and pain sensation below the level of injury. Since the posterior aspect of the cord is preserved, so is proprioception and vibratory sensation.
- Classic Brown-Séquard syndrome, in its purest form, is described as a loss of ipsilateral motor function, proprioception, vibratory and pressure sensation, and contralateral loss of temperature and pain sensation below the level of injury.
- Of all cord syndromes, Brown-Séquard syndrome has the best overall prognosis, with more than 80-90% of people recovering bowel and bladder function and more than 75% regaining their ambulatory status.
- The most common of all partial cord syndromes is central cord syndrome, which is distinguished from the other cord syndromes by the fact that the upper extremities are significantly more affected from the motor perspective than the lower extremities.

remain employed. Optimistically, this number increases to 35.2% after 20 years and holds steady for data collected after 30 years post-injury.

Spinal Cord Injury Model System Program

In the 1970s, the National Institute on Disability and Rehabilitation created the Spinal Cord Injury Model System Program through federally funded grants. The model systems are composed of 14 major medical institutions and three subcontracted facilities, which focus on the treatment of spinal cord injury, traumatic brain injury (TBI), and burn injury. The purpose is the collection of information and to conduct research surrounding the rehabilitation, long-term outcomes, and complications of SCI, TBI, and burn injuries, in addition to social aspects including health, wellness, and service delivery. Over the years, the model systems have provided a great deal of information about spinal cord injury, rehabilitation, and outcomes.[2]

Based on the 2011 National Spinal Cord Injury Database figures, the lifetime costs associated with SCI depend upon the severity/level of injury and the age at which the injury is sustained. The first year following the injury is always associated with a greater cost than subsequent years. It is estimated that a high quadriplegic (C1-C4) will expend approximately $985,000 in the first year alone, followed by approximately $171,000 annually. Injuries occurring earlier in life at higher spinal cord levels require the most financial expense. High quadriplegia sustained at age 25 results in an estimated $4.3 million lifetime expense, whereas the same injury sustained at age 50 is estimated to cost approximately $2.4 million. Lower cervical SCI and SCI resulting in paraplegia, on average, cost less for both the first year of treatment as well as for lifetime associated cost. (*See Table 1.*)

Grading Scales

There are two well-known scales used to grade and prognosticate SCI. The Frankel scale was developed during World War I, but is less commonly used today. It is a basic scale that grades the SCI based on level and is used to evaluate functional recovery. There are five grades used in the Frankel scale, which essentially divide complete versus incomplete spinal injuries as follows:

- A — complete paralysis (no motor/sensory below level of injury);
- B — sensory present below level of injury;
- C — incomplete injury with motor and sensory function below level of injury;
- D — fair to good motor function below level of injury; and
- E — normal function (no motor of sensory deficit).[3]

The American Spinal Injury Association (ASIA) Impairment Scale (AIS) is a more widely used and more refined scale. Based on the Frankel scale's five grading levels, the AIS was originally developed in 1982 and has undergone six revisions, with the most recent occurring in 2002. The AIS differs from the Frankel scale in that it more clearly defines complete and incomplete injury by determining sacral sparing (presence of rectal motor function or sensory function at S4-S5 dermatome), determining the presence of neurologic level of injury using sensory and motor evaluation in bilateral extremities, and by determining, in incomplete injuries, where partial zones of sensory or motor preservation exist.

Practically, the ASIA is a scale that scores motor and sensory function on different scales. There is also a letter that is assigned based on degree of motor, sensory, and/or presence of sacral function. Although for completeness sake the motor and sensory scales are important in the evaluation of an acute SCI, from a functional perspective, the letter grade is most often used and conveys substantial information.

The motor scale is a 100-point scale that uses 10 muscle groups (five groups in the upper extremities and five in the lower) that are tested bilaterally. It is important to recognize that the muscle strength is not what is tested, but rather the range of motion. Range of motion is

Table 1. Economic Impact of SCI

Severity of Injury	Average Yearly Expenses (in 2010 dollars)		Estimated Lifetime Costs by Age at Injury (discounted at 2%)	
	First Year	Each Subsequent Year	25 years old	50 years old
High Tetraplegia (C1-C4)	$985,774	$171,183	$4,373,912	$2,403,828
Low Tetraplegia (C5-C8)	$712,308	$105,013	$3,195,853	$1,965,735
Paraplegia	$480,431	$63,643	$2,138,824	$1,403,646
Incomplete Motor Functional at Any Level	$321,720	$39,077	$1,461,255	$1,031,394

Data Source: Economic impact of SCI , *Topics in Spinal Cord Injury Rehabilitation* 2011;16(4).

scored using the standard 0-5 point scale, with 0 indicating no movement and 5 indicating movement against full resistance. Assessment of strength in each motor group instead of range of motion across the joint can lead to inappropriately high scores that will inevitably overestimate the final motor score and, therefore, final ASIA score. A motor index score of 100 is evidence of no motor deficit, or a normal exam. The motor level is defined as the lowest level at which there is 3/5 movement.

The sensory grading system is a 112-point scale that tests 28 dermatomes bilaterally for both light touch and pinprick sensation. The sensory level is defined as the most caudal level with normal light touch and pinprick sensation. Again, a sensory index score of 112 demonstrates a normal sensory examination. (*See Table 2.*)

Complete injuries are those in which there is no motor or sensory function below the level of injury. Specifically, AIS A injuries are those in which there is no sacral motor function or sensation. Incomplete injuries are grades B, C, or D, and the specific letter assignment depends on the presence of motor, sensory, or grade of muscle strength. AIS E is a normal neurologic exam with no deficits appreciated. Zone of partial preservation is a term only applied in complete SCI (those in which there is no sacral motor or sensory function) and describes areas of preserved segments below the neurologic level of injury.

The AIS may suggest the potential for minimal to some neurologic recovery, but has not been validated as a true prognostic scale. It does, however, provide the medical community with a common language to communicate the severity and level of SCI and, for this reason, in addition to the thoroughness of the AIS, it has been adopted by the majority of major medical intuitions both nationally and internationally.[4]

Pathophysiology

The normal structure, neuroprotective mechanism, and metabolism of the spinal cord are interrupted as a result of injury. There are many theories on the pathophysiology of the spinal cord following injury, but most theories include the belief that there are two main phases of evolution, primary and secondary injury, and it is likely that the actual process encompasses components of all theories. The progression of injury is believed to occur over the course of the first week, but successive histologic changes can be seen as far out as one month following injury.

Normally, the gray matter of the spinal cord has a much higher rate of metabolism and contains the neuronal bodies directly within the tissue itself, as opposed to the white matter that has a slower metabolic rate and contains its neuronal bodies at distant sites. This difference makes the gray matter more susceptible to injury than the white matter.

Primary injury occurs at the time of the initial insult and can be either penetrating or blunt. Primary injury to the spinal cord is commonly due to trauma and affects the cord level at which the trauma occurs. Primary injury also occurs to neighboring levels adjacent to the primary site secondary to edema, hemorrhage, concussive, stretching, or shearing forces and ischemia.

In the minutes, hours, and first several days following SCI, metabolic changes take place in the spinal cord that are believed to lead to secondary injury. It is at this point that the many theories on SCI diverge. After direct injury to the cord, there is a period of inflammation that occurs within the gray and white matter, which leads to the metabolic and biochemical changes that can be toxic to the spinal cord tissue, leading to further damage. Following spinal cord injury, clinically it is not uncommon to have hypotension and bradycardia as a result of autonomic dysfunction, which can contribute to decreased cord perfusion and, ultimately, continued cord damage. Although there are many theories that describe

Table 2. ASIA Scale/Grading

AIS Impairment Scale	
A	Complete — Absence of motor and sensory function in sacral segments (S4-S5)
B	Incomplete — Preserved sensory function, but absent motor function below neurologic level of injury. Sacral sensation intact (S4-S5)
C	Incomplete — Preserved motor function below neurologic level of injury. More than half of muscle groups below injury have muscle grade of 3 or less.
D	Incomplete — Preserved motor function below neurologic level of injury. More than half of muscle groups below injury have muscle grade of 3 or greater
E	Normal Examination — No motor or sensory deficits

different biochemical complications as a result of injury, they all conclude that ultimately a cascade of toxic mediators (excitatory amino acids, free radicals, or calcium ions, for example) results in neuronal cell damage and death propagating secondary spinal cord injury.[5]

Spinal Cord Syndromes

The anatomy of the spinal cord must be appreciated to identify the patient's specific spinal cord syndrome. Patterns of symptoms can give the examiner an idea of the injury sustained, prognosis, and outcome even prior to imaging.

The spinal cord is divided into tracts. These tracts contain bundles of nerve fibers that run together in either an ascending or descending fashion, cross over the spinal cord in varying locations, and have clinically and characteristically significant findings when damaged. There are three particular tracts of the spinal column that are critically important. The dorsal column is an ascending, ipsilateral tract that is located in the posterior aspect of the cord and contains information on proprioception and vibration. The corticospinal tract is a descending, ipsilateral motor tract. Lastly, the spinothalamic tract is an ascending, contralateral tract that contains pain, temperature, and light touch nerve fibers.

Anterior Cord Syndrome

Anterior cord syndrome is usually sustained due to a hyperflexion injury to the cervical cord, but can occur anywhere in the spinal column. Hyperflexion of the cord causes direct contusion to the cord or can result in the protrusion of disc contents, bony fragments that have fractured, or, rarely, can cause direct laceration or thrombosis to the anterior spinal artery. Since the injury to the cord is bilateral, the pattern of symptoms that accompany this injury includes bilateral motor paralysis and loss of pinprick, temperature, and pain sensation below the level of injury. Since the posterior aspect of the cord is preserved, so is proprioception and vibratory sensation.

The overall prognosis for anterior cord syndrome is poor. Improvement in motor function can be seen within the first 24 hours following injury, but usually does not occur after the first day. After 30 days following injury, there is little to no additional recovery of function.[6]

Brown-Séquard Syndrome

Brown-Séquard syndrome is an anatomic or functional hemisection of the cord, which has several potential causes. From a trauma perspective, Brown-Séquard is commonly the result of penetrating trauma to the spinal cord. However, more commonly it is due to inherent spinal or compressive lesions such as tumors or epidural hematomas. Classic Brown-Séquard syndrome, in its purest form, is described as a loss of ipsilateral motor function, proprioception, vibratory and pressure sensation, and contralateral loss of temperature and pain sensation below the level of injury. Although the pure form of Brown-Séquard syndrome is rarely seen, a partial form of Brown-Séquard is more common. Interestingly, because the fibers of the lateral spinothalamic tract decussate one or two levels above or below where the injury may occur, it is possible to see ipsilateral pain and temperature sensory loss above the level of injury.

Of all cord syndromes, Brown-Séquard syndrome has the best overall prognosis, with more than 80-90% of people recovering bowel and bladder function, and more than 75% regaining their ambulatory status.[7]

Central Cord Syndrome

The most common of all partial cord syndromes is central cord syndrome, which is distinguished from the other cord syndromes by the fact that the upper extremities are significantly more affected from the motor perspective than the lower extremities are. The most common mechanism of injury is a hyperextension injury, and it is usually seen after a fall in an older population with preexisting spinal stenosis or arthritis. The injury to the spinal cord affects the central portions of the corticospinal and spinothalamic tracts, resulting in the disproportionate pattern of symptoms between the upper and lower extremities. Patients typically have greater weakness in the proximal muscles than in the distal ones. Sensory symptoms are also appreciable, with some patients presenting with dysesthesias of their upper extremities as their predominant symptom.

The prognosis of central cord syndrome is dependent upon the severity of the cord contusion after injury

and the age at the time of injury. Patients younger than 50 years old at the time of injury tend to have more significant bladder continence recovery (80%) and achieve ambulatory recovery (90%) as compared to their older counterparts. Those older than 50 years at the time of injury have approximately a 50% chance of regaining ambulatory function, and only 30% regain bladder continence.[8]

Acute Management of SCI

The acute management of spinal cord injury occurs in several settings. In the prehospital setting, providers must maintain a high index of suspicion for spinal injury when presenting on the scene of any motor vehicle collision, fall, assault, or various other mechanisms in which injury to the spinal cord, occult or obvious, may have occurred. As with any trauma, airway, breathing, and circulation (the ABCs) always come first in the stabilization of a patient. In the event of high cervical spine injuries, severe trauma, or head injuries in which the patient's respiratory effort, mental status, or ability to maintain his/her own airway may be compromised, it may be indicated to bring the patient to the closest local facility for airway protection. Inline cervical spine stabilization should be utilized in every trauma patient for intubation, regardless of a high or low suspicion for SCI. A single person should be dedicated to maintaining the integrity of spinal stabilization, while another is cautiously securing the airway. If a high suspicion for cervical spine injury exists, fiberoptic intubation, when available, should be considered as the safest means of intubation.

Proper prehospital precautions include cervical spinal immobilization with a rigid cervical collar, full spinal precautions, log-rolling, and transporting patients on a backboard. Minimizing movement of the patient while still maintaining the ability to provide care should be balanced.

Upon arrival in the hospital setting, ABCs should be reevaluated and intervention at any step should be taken to stabilize the trauma patient. The airway should be secured, while maintaining cervical spine precautions, in any patient with significant facial trauma, head injury, or GCS less than 8. In those patients who are believed to have a significant or isolated spinal injury requiring urgent rather than emergent intubation, consideration of fiberoptic or other advanced method of intubation is warranted. Patients with high cervical spine injuries (C5 and above) are at high likelihood of requiring mechanical ventilation and should be closely monitored and considered for early intubation and airway control.[9]

A primary survey as part of a trauma evaluation should be rapidly but thoroughly preformed. Part of the primary survey should include examination of the spine. With the patient's cervical spine being properly immobilized, the patient should be log-rolled and the emergency physician should palpate the spine for bony tenderness, obvious step-offs, or crepitus that would heighten the suspicion for spinal injury. The presence of any of these findings should prompt the physician to perform further diagnostic evaluation. If none of these exist and there are no concerning neurologic findings, the backboard can be removed.

Hemodynamic Status

In evaluation of a patient's hemodynamic status, sufficient vascular access should be obtained while keeping in mind additional injuries, which may limit optimal locations for access placement. What is considered sufficient may vary based on injuries sustained and presenting hemodynamics and should be determined based on the physician's clinical judgment. It is important to remain vigilant to the fact that many spinal cord-injured individuals develop a significant amount of hemodynamic lability in the first few hours, days, and even weeks following their injuries, and what seems sufficient may quickly become otherwise. The hemodynamic lability will often require vasoactive support and, therefore, placement of central venous access and an arterial line are reasonable even if a patient is initially stable. Frequent reevaluation of vital signs in a patient with SCI is warranted.

Spinal Shock vs. Neurogenic Shock

Initial vital signs can vary depending on an isolated spinal fracture or spinal cord injury or multi-system trauma. In a hemodynamically unstable patient with what appears to be isolated spinal trauma, the medical provider should still be concerned about and exclude additional internal injuries, particularly abdominal injuries. Hypovolemic shock can present similarly to spinal and neurogenic shock; however, the management varies considerably. Once other injuries have been excluded as the cause of shock (i.e., intra-abdominal bleeding, thoracic trauma, etc.), it is safe to attribute hemodynamic instability to acute spinal cord injury. In SCI, vital signs can vary dramatically as well and can reflect two well-known but different entities: spinal shock and neurogenic shock.

Spinal Shock. Spinal shock is a presentation of acute SCI that occurs within the first 24 hours following injury and describes a transient constellation of symptoms that include total loss of reflexes below the level of injury, flaccid paralysis including loss of rectal tone, and complete loss of sensation. These findings are often accompanied by autonomic dysfunction, but spinal shock is not a hemodynamic phenomenon.[10] The presence of priapism in a patient with acute SCI is suggestive of spinal shock. There are several theories that attempt to explain both the pathophysiology as well as define the duration of spinal shock. No clear consensus exists, but many theories suggest that the end of spinal shock occurs when the first reflexes return. Often, the first reflex to return is the bulbocavernosus reflex. Despite the lack of consensus on the pathophysiology and termination of spinal shock, all theories agree that spinal shock begins within 24 hours, and

usually within minutes to hours of the acute injury. Most theories suggest that spinal shock begins to resolve within 72 hours of onset, but there are several schools of thought that suggest termination based on the return of deep tendon reflexes several weeks after injury or the return of detrusor function, which can take several months to recover.[11]

Neurogenic Shock. Neurogenic shock is a hemodynamic phenomenon that is also associated with acute SCI. A classic triad of symptoms including hypotension, bradycardia, and peripheral vasodilation, secondary to a profound loss of systemic vascular resistance, is seen. Neurogenic shock is often referred to as "warm shock" because of the vasodilation, but as a result of the spinal injury, it is not uncommon for these patients to be hypothermic. Onset is usually within 30 minutes of injury and can last several days to weeks following injury.[12] Neurogenic shock is most commonly seen in association with spinal cord injury above the level of T6, but can occur at any level. A recent study suggests that neurogenic shock is seen in approximately 19% of cervical spinal injuries, 7% of thoracic spinal injuries, and in only 3% of lumbar spine injuries.[13] As previously stated, any trauma patient with hypotension should be quickly assessed to ensure there is no alternative cause. Neurogenic shock should, therefore, essentially be a diagnosis of exclusion once hemorrhage and thoracic trauma have been effectively excluded.

Treatment of shock in SCI is dependent upon identification of spinal shock or neurogenic shock. It is important to make the distinction between the two in each individual, but to also keep in mind that a single patient can have both conditions concomitantly. Treatment of spinal shock is primarily supportive. In contrast, the treatment of neurogenic shock requires volume resuscitation to help compensate for the massive vasodilation that occurs, in addition to pharmacologic support. Early use of vasopressors is recommended to avoid fluid overload, although no specific goal of mean arterial or systolic blood pressures has been identified as optimal. Pharmacologic support in the form of vasopressors should be initiated once fluid resuscitation has failed to improve the patient's hemodynamics. Norepinephrine should be used as a first-line agent, followed by medications like dopamine and dobutamine. Medications like phenylephrine, with unopposed alpha activity, should be avoided, given the pathophysiology of neurogenic shock and the potential for reflexive bradycardia, which may further worsen the spinal cord injury. Atropine is used to treat bradycardia.[14]

Overall, the goal of treating neurogenic shock is the same as treatment for any form of shock: adequate tissue perfusion. Most importantly, however, treatment of neurogenic shock should be aimed at maintaining perfusion of the injured region of the spinal cord in hopes of minimizing any additional secondary injury.

Neurologic Evaluation

In the patient with an acute spinal cord injury, it is imperative to obtain a thorough neurologic evaluation as soon as possible. After initial stabilization, it is important to perform the neurologic examination as completely as possible, fully assessing both motor and sensory deficits. Rectal sensation and tone are imperative in the neurologic evaluation of an acute SCI. All reflexes should be evaluated, as they can provide important clues to diagnosing spinal shock. As previously described, the ASIA scale is the most commonly used and most widely accepted scale for determining the extent of neurologic injury in patients with SCI and should be performed at 72 hours. The ASIA can be performed earlier, but in patients with spinal shock it may not be accurate and should be redone once reflexes return. Specifically, the diagnosis of complete versus incomplete injury (AIS A versus B, C, or D) cannot be made until spinal shock has resolved, and this diagnosis carries significant implications for the patient's prognosis.

Polytrauma

In patients with spinal cord injury in the setting of additional injuries or hemodynamic instability, it is reasonable to defer definitive treatment of the spinal cord injury until other life-threatening injuries are addressed (i.e., abdominal trauma/hemorrhage, or thoracic or cardiac injury), assuming the cervical spine is properly immobilized using a hard collar and log-roll precautions are used when moving the patient.

Imaging of the Spine

Imaging of the spine in a trauma patient can be accomplished using plain films, computed tomography (CT), or magnetic resonance imaging (MRI). The type of imaging chosen by the emergency physician should be based on the physician's clinical suspicion, level of risk stratification, or pretest probability for SCI.

In the past several decades, many studies have evaluated close to 40,000 patients with an asymptomatic cervical spine examination. From these studies, class I evidence exists that states that in an asymptomatic patient, there is no benefit in obtaining cervical spine imaging. In addition to the fact that imaging provides no benefit in the asymptomatic population, it also exposes patients to unnecessary radiation, is costly to the medical system, and utilizes unnecessary resources. In 2001, the Congress of Neurological Surgeons and the American Association of Neurologic Surgeons published the Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injuries, which define an asymptomatic patient by the following criteria:

- Neurologically normal (GCS 15, no focal motor or sensory deficits, appropriate response to external stimuli, and appropriate orientation);
- Free of intoxication or substances, which can alter a patient's level of alertness;
- Absence of midline neck tenderness from the nuchal ridge to the first thoracic vertebrae;

• Absence of associated, distracting injury (including, but not limited to a long bone fracture, large lacerations or degloving injuries, burns, or injuries that require surgical consultation).[15]

In emergency medicine, there are two clinical rules commonly used to evaluate the cervical spine in patients with blunt trauma. The National Emergency X-Radiography Utilization Study (NEXUS) used the same criteria mentioned above to evaluate patients with blunt trauma and demonstrated 100% sensitivity in excluding cervical spine trauma. If all of the criteria are met, the authors recommend that imaging not be performed.[16] The Canadian C-Spine (CCR) rule includes additional criteria, which more clearly risk stratify patients based on the mechanism of injury, previous spinal disease or injury, as well as clinical examination findings. Although a lengthier rule, the CCR may allow for clearance of cervical immobilization devices in patients who may have otherwise kept the collar based on NEXUS.[17]

Alternatively, the symptomatic patient is the patient with pain on examination of the midline cervical spine, an altered level of consciousness due to trauma or intoxication, or patients with neurologic deficit. These are the patients who should undergo radiologic evaluation prior to clearance of the immobilization device. Plain radiographs are sufficient when three views are completed and the radiograph quality is optimal.[18] Injuries may be missed in those who have suboptimal films that do not adequately show the region of injury. These missed injuries secondary to suboptimal imaging most commonly occur at high cervical levels (C2) as well as at the cervical-thoracic junction (C7-T1). The emergency physician knows that in the acutely injured patient wearing a hard cervical collar, optimal plain films are often hard to obtain. For this reason, CT has been studied as an adjunct mode of imaging to plain radiographs and has been found to be a cost-effective means of evaluating for cervical spine injury.[19]

Computed tomography of the spinal column has been shown to be an efficient and sensitive modality to assess for bony injury. However, in 2005 Holmes suggested that only in patients with a depressed mental status and high likelihood of cervical spine injury should CT be the initial mode of imaging. In populations with less significant injuries and a lesser likelihood of cervical spine injury, plain films still should be used as the initial screening method.[20]

In the past several years, there has been a movement to make CT the standard of care in the evaluation of the cervical spine in the trauma patient. The Eastern Association for the Surgery of Trauma (EAST) has started to recommend CT as the standard way to evaluate the cervical spine in those patients who do not meet NEXUS criteria and are suspected of having a cervical spine injury. In patients with neurologic deficit, EAST recommends the addition of an MRI in the evaluation of a cervical spine injury.[21]

In 2011 Duane et al studied trauma patients to evaluate the accuracy of NEXUS criteria using CT scan as the gold standard and found NEXUS to miss a number of significant injuries. They looked at approximately 2600 patients and found 26 with missed injury based on the NEXUS criteria. Of the 26 missed, 19 patients required additional intervention in the treatment of their injuries, including several who required operative repair and one who required placement of a halo.[22]

In symptomatic patients, ligamentous spinal injuries should also be a consideration. Once radiographs and/or CT have evaluated and are negative for bony injury, flexion/extension films can be performed in the awake patient to assess for subluxation, reflecting the presence of ligamentous injury. Three cervical views and a negative flexion/extension film have been cited as having a negative predictive value of 99%.[23] In neurologically impaired individuals, those who cannot flex or extend their neck secondary to pain or muscle spasm, or those who cannot undergo flexion/extension films for any other reason but in whom a concern for cervical spine injury exists, flexion/extension films with fluoroscopic guidance or MRI evaluation should be performed.[24]

MRI evaluation of patients is costly and time consuming. MRI should not be performed in unstable patients. In the evaluation of the obtunded trauma patient, the cervical spine can be cleared using MRI. The current data support the use of MRI within 48 hours of the trauma event to evaluate specifically for ligamentous injury. Benzel et al studied 174 patients with suspected cervical spine injuries who underwent MRI evaluation. Thirty-six percent of patients were found to have MRI evidence of injury, but, more importantly, none of the patients with a negative MRI had later spinal instability and were all cleared of their immobilization device.[25]

Despite the evidence that supports the use of plain radiography in evaluation and clearance of the cervical spine, it is quite common and acceptable for the emergency physician to order a CT in trauma patients, especially in the trauma patient who is going to require other CT imaging. Simultaneous imaging of the cervical, thoracic, and/or lumbar spine is more efficient and reliable at assessing for injury.[26] It is also important to remember that any patient with a single spinal injury is at risk for an additional spinal injury, and the presence of a single spinal injury should prompt radiographic evaluation of the remainder of the spinal column. Plain radiographs (AP and lateral images of the thoracic and lumbar spine) are sufficient if the patient is asymptomatic.

As with many conditions evaluated by the emergency physician, follow-up within a reasonable period of time is essential for the trauma patient. Patients with symptoms but negative films and/or CT scan should continue the use of their rigid neck immobilizer until neurosurgery can evaluate the patient.

Surgical Consultation

In the patient with spinal injury, appropriate neurosurgical or orthopedic consultation is essential and should be obtained in a timely matter. Every hospital has specific guidelines as to which surgical services should be consulted based on the level or severity of the injury or specific deficits associated with the injury. The goal of surgical intervention is spinal stabilization and decompression of the spinal cord to prevent additional or ongoing injury. Early decompression, if indicated, within 4-6 hours of injury may improve outcomes.

Steroids

The use of high-dose steroids in the presence of spinal injury used to be the standard of care and was included as part of many SCI guidelines. In the past several years, this practice has been called into question and many studies have addressed this controversy. Although currently controversy still exists surrounding the use of steroids, the practice has been removed from guidelines and is no longer considered the standard of care.

The purpose of steroids in spinal cord injury was originally thought to be of benefit in three distinct ways. Steroids were thought to improve blood flow to the injured area of the spinal cord, assist in limiting the inflammatory response to injury, and reduce vasogenic shock and edema.

The National Acute Spinal Cord Injury Study (NASCIS) I, II, and III are the three largest prospective, double-blinded, randomized studies on the use of steroids in spinal cord injury. NASCIS I studied 330 patients with acute spinal cord injury with two groups: a high-dose methylprednisolone group against a standard-dose methylprednisolone regimen. The patients were evaluated at 6 weeks and 6 months following injury. NASCIS I found no difference in outcome (recovery of sensory or motor function between the two groups, but although not statistically significant, both a higher rate of wound infection and early case fatality were found in the high-dose methylprednisolone group).[27]

NASCIS II studied three treatment groups with a total of 487 patients: a high-dose methylprednisolone group (different dose than used in NASCIS I), a naloxone group, and a placebo group. In the high-dose methylprednisolone group, statistically significant improvements were found with respect to pinprick and light touch sensation at 6 weeks and 6 months following injury. Unfortunately, this finding was lost at one year post-injury. Post-hoc analysis revealed improved neurologic outcomes in groups receiving steroids within 8 hours of their acute injury. Interestingly, patients receiving steroids more than 8 hours following injury were found to have worse neurologic outcomes than the comparison groups, despite this finding not reaching statistical significance. Similarly to NASCIS I, there were more wound infections, pulmonary complications (including an increase in pulmonary embolism), and adverse events noted in the steroid group.[28]

NASCIS III compared methylprednisolone given for 24 hours, methylprednisolone given for 48 hours, and tirilazad mesylate given for 48 hours in patients with acute spinal cord injury. Patients were assessed at 6 weeks and 6 months for motor function change, in addition to being scored on the Functional Independence Measure (FIM). The group receiving 48 hours of methylprednisolone and whose therapy was started within 3-8 hours of injury was found to have a statistically significant improvement in motor score at 6 weeks and 6 months (one full motor grade) and were more likely to show improvement in the FIM scores. However, analysis of the groups reveals a disproportionate number of patients in the group receiving 48 hours of steroids who had minor motor deficits on initial examination as compared to the other two treatment groups. This discrepancy alone could account for the statistical findings in the 48-hour treatment group and, in fact, once this discrepancy was controlled for, the statistical significance disappeared. The group receiving tirilazad had similar outcomes in motor recovery rates to the treatment group receiving methylprednisolone for 24 hours. Like NASCIS I and II, there were higher complication rates noted in the groups receiving high-dose steroids. The 48-hour treatment group had higher rates of pneumonia and severe sepsis as compared to the 24-hour methylprednisolone and tirilazad groups. The conclusion drawn by the authors of NASCIS III was that patients who are started on a high-dose steroid regimen within 3 hours of injury should receive only a 24-hour course of steroids, but those receiving steroids within 3-8 hours of injury should be maintained on steroids for 48 hours.[29]

Ultimately, the use of steroids in acute SCI is controversial and should be the decision of the institution and treating physician. It is not currently clear if there is a significant benefit in neurologic outcome with steroid use, and more studies need to be done, but it is clear that patients who receive steroids have higher complication rates, including severe sepsis, pneumonia, pulmonary embolism, gastrointestinal bleeding, and wound infections, than their counterparts. For these reasons, as mentioned above, high-dose steroids are not currently a part of treatment guidelines for acute SCI. The Congress of Neurological Surgeons states that treatment of acute SCI with steroids "should only be undertaken with the knowledge that the evidence suggesting harmful side effects is more consistent than any suggestion of clinical benefit."[30]

Blood Pressure Management

Blood pressure management in acute spinal cord injury is another area in which class I evidence is lacking. It is clearly understood that hypotension can directly contribute to hypoperfusion and result in further injury to the spinal cord. Many studies over the previous two

decades have attempted to determine the optimal blood pressure, or mean arterial pressure (MAP), in acute spinal cord injury and the duration of treatment to maintain the proposed blood pressure. The theory is similar to that for steroids, in that the goal is to reduce secondary injury to the spinal cord from hypoperfusion.

It is well understood and accepted that, as with traumatic brain injury, autoregulation of blood flow to the spinal cord is compromised in acute SCI. Animal models suggest that hypotension and, therefore, hypoperfusion are detrimental in acute SCI and worsen neurologic outcomes. Because the treatment of hypotension and shock is a basic tenet in medicine, class I evidence supporting or refuting the use of a goal MAP in SCI will never be obtained.

In 1993 Levi et al studied hemodynamic parameters in acute cervical SCI with 50 consecutive spinal cord-injured patients. The patients were aggressively supported using fluids and vasopressors to achieve a goal systolic blood pressure of greater than 90 mm Hg. All patients received invasive monitoring with an arterial line, central venous access, and Swan-Ganz catheters. Data for the first week following injury, including hemodynamic, neurologic, and demographic information, were analyzed. Of the 50 patients included in the study, 31 had complete (grade A) injuries, 20 had no improvement in function, 21 had appreciable improvement, and 9 died.[31]

Four years later, Vale et al studied acute cervical and thoracic SCI in 70 patients. These patients underwent similar monitoring and hemodynamic augmentation strategies as in the Levi study and were followed to one year post-injury. The goal systolic blood pressure was maintained above 85 mm Hg for 7 days following injury using fluids and vasopressors. Patients in whom decompression and spinal stabilization were indicated underwent those procedures. The authors found that after one year, 60% of patients with complete injuries had improvement by one grade on the ASIA AIS, and 20% had return of bowel function. Thirty percent of these patients had recovered the ability to walk. Of those patients who sustained incomplete cervical spine injuries, 92% recovered their ability to ambulate and 88% regained bladder control. Of the thoracic injuries, both complete and incomplete, improvements were noted and, albeit smaller percentages than their cervical counterparts, some demonstrated the ability to walk and regained bladder function. A majority of patients with thoracic injuries underwent surgical decompression or stabilization of the spine. The authors controlled for the timing of surgical intervention (early vs. late), and found no significant correlation between the timing of surgery and neurologic outcome.[32]

In 2010, a literature review examined the evidence supporting the use of vasopressors to augment blood pressure in acute spinal cord injury. Unfortunately, the researchers were unable to draw any definitive conclusions about the use of vasopressors, reporting that currently there is "no gold standard on vasopressors support." They did note, however, that cervical spine injuries require vasopressor support much more frequently than thoracic or lumbar injuries.[33] In both of the above mentioned studies, minimal morbidity associated with invasive monitoring and vasopressors administration was observed. The authors of both studies recommend the use of blood pressure augmenting medications and fluids in the acute period (the first 7 days) following spinal cord injury. The authors thought that the neurologic improvements noted in each study were the result of improved perfusion and prevention of secondary injury from hypotension.

The Congress of Neurological Surgeons states that there is "insufficient evidence" to support treatment standards or guidelines, but does affirm that hypotension in acute SCI should be avoided and/or remedied as soon as possible. Also recommended is maintenance of a MAP between 85 and 90 for the first 7 days following acute injury to help with perfusion and prevent secondary injury.[15]

Additional Issues in Acute SCI

Once the patient has been stabilized and admitted to the hospital and surgical consultations have been completed, there are several issues that patients face with new SCI, in addition to the emotional and physical adjustments of a new life.

Patients have significant autonomic instability, often for several weeks following their initial injury, which requires vasopressors and, frequently, long-term medications for stabilization. The general rule is, the higher the injury, the more autonomic instability can be seen and the longer the duration of instability.

Early tracheostomy in patients with cervical spine injuries has been studied and is becoming more accepted. Patients with cervical and thoracic spinal trauma often require prolonged mechanical ventilation while hospitalized, and long-term mechanical ventilation is common in patients with high cervical spine injury.[9] In 2010 Romero et al looked at 152 consecutive patients with acute spinal injury and did a retrospective study on the timing of tracheostomy. Seventy-eight percent of patients were grade A injuries. Early tracheostomy (preformed within 7 days of injury) was shown to be beneficial for reducing the number of days that mechanical ventilation was required, decreasing ICU stays, and decreasing the risk of orotracheal complications from intubation. The authors were not able to show a change in the risk of mortality associated with injury or a reduction in the risk of ventilator-associated pneumonia.[34] The timing of tracheostomy has also been controversial due to concerns of infection following anterior spinal stabilization. Berney et al evaluated early tracheostomy after anterior spinal stabilization and found there is a low risk of infection, further supporting the idea that early tracheostomy is beneficial.[35]

Secondary infection risks are significant in SCI patients. As stated above, patients with spinal injuries often require prolonged mechanical ventilation and, therefore, have a significant risk of ventilator-associated pneumonia. Urinary tract infections are common among these patients, as well, given their need for prolonged catheterization. Skin breakdown and decubitus ulcers are a common complication of immobility. Aggressive mobilization and physical and occupational therapy are imperative in the prevention of ulcers as well as for the patient's general recovery.

Patients with SCI are at risk for deep vein thrombosis (DVT) and pulmonary embolism (PE). The incidence of DVT in the SCI population has been quoted between 12-64%. Consequent PE results in approximately 10% of deaths in the first year following SCI, despite the use of chemical thromboprophylaxis.[36] Studies have been done to evaluate the effectiveness of inferior vena cava (IVC) filter placement for prophylaxis. Whereas some studies suggest the placement of IVC filters for secondary prophylaxis in patients with a PE or a contraindication to chemical prophylaxis,[37] no strong evidence exists to support the routine placement, and some studies suggest that IVC filter placement may actually increase the risk of DVT development.[38]

Conclusion

Acute spinal cord injury is a complex disease process that is often complicated by additional traumatic injury. Although physicians know a significant amount about spinal cord injury, there are many key areas still to be fully understood. As a result, over the past decade, guidelines have changed. The use of steroids and vasopressors is controversial and requires additional study. The emergency physician should be prepared to assess and treat the acute spinal cord injury regardless of level and should be aware of the differences in clinical presentation of injuries at a specific level. The emergency medicine physician is the critical first step in the management of SCI, including airway management, imaging and clearance of the spinal cord, identification of other critical injuries, and initiation of pharmacologic management that will minimize the long-term morbidity and mortality for this devastating injury.

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CME/CNE Questions

1. Which of the following is the most common cause of spinal cord injury?
   A. penetrating injury
   B. sporting injury
   C. motor vehicle collision
   D. falls

2. People with spinal cord injury are:
   A. slightly more likely to have relationships that end in divorce than the unaffected population
   B. likely to remain employed following their injury
   C. will likely never get married if they are single at the time of injury
   D. report less fulfilling lifestyles than the unaffected population

3. Which of the following is true of anterior cord syndrome?
   A. It is usually the result of hyperextension.
   B. Patients have a good prognosis.
   C. It results in loss of bilateral motor function, pinprick, temperature, and pain sensation below the level of injury.
   D. Proprioception, vibratory sense, and pinprick sensation are lost.

4. Which of the following is true of central cord syndrome?
   A. The lower extremities are affected more than the upper extremities.
   B. Distal muscle groups are affected more than proximal muscle groups.
   C. The prognosis is based on age at the time of injury and severity of cord contusion.
   D. The majority of patients affected, regardless of age, regain bladder function.

5. Which of the following is true of spinal shock?
   A. It is a hemodynamic phenomenon.
   B. It is classified by loss of reflexes, flaccid paralysis, and sensation.
   C. It is clearly understood and defined.
   D. It is usually completely resolved by 24 hours after injury.

6. Which is true of neurogenic shock?
   A. It should be treated with IV fluids and vasopressors.
   B. It most commonly occurs with spinal injuries below T6.
   C. It is defined by hypotension, tachycardia, and peripheral vasodilation.
   D. It is often accompanied by hyperthermia.

7. In which patients can cervical spine radiographs be used to clear a cervical collar?
   A. all patients with low-risk mechanisms
   B. symptomatic patients
   C. patients who meet NEXUS criteria
   D. patients who want to leave the emergency department AMA

8. Which of the following is true about the use of steroids in spinal cord injury?
   A. Steroid use is associated with improved neurologic outcomes.
   B. If steroids are given for 48 hours with tirilazad, FIM scores show improvement.
   C. Steroids should only be given if within 3-8 hours of injury.
   D. Steroid use is highly controversial, and they should be given based on institutional and provider practices.

9. Which is true regarding blood pressure management in patients with spinal cord injury?
   A. BP management should be aimed at maintaining a MAP of > 90 mm Hg.
   B. BP management should be aimed at preventing hypotension and secondary injury.
   C. BP management is clearly associated with improved neurologic outcomes.
   D. BP management is important, but vasoactives should not be used to augment blood pressure.

10. Once the acute injury has been stabilized:
    A. Autonomic instability in cervical spine injuries resolves quickly.
    B. Patients with spinal cord injury are at increased risk of DVT, PE, and pulmonary infection.
    C. Mechanical ventilation is uncommonly needed.
    D. Patients integrate back into society without problems.

CNE/CME Objectives

Upon completing this program, the participants will be able to:

- discuss conditions that should increase suspicion for traumatic injuries;
- describe the various modalities used to identify different traumatic conditions;
- cite methods of quickly stabilizing and managing patients; and
- identify possible complications that may occur with traumatic injuries.

CNE/CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

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