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It is estimated that approximately 10,000 new cases of spinal cord injury occur annually in the United States.[1-4] Acute traumatic spinal cord injury predominantly is a disease affecting young males. According to statistics from the National Spinal Cord Injury Database, 81% of these victims are male, with an average age of 31.5 years. Since 1991, motor vehicle accidents have accounted for 36% of all reported cases, followed by acts of violence, falls, recreational/ sporting activities, and other non-traumatic conditions. The most commonly injured area is the lower cervical spine (C_5-C_6), followed by the thoracolumbar junction,[3,5] although the elderly may be more likely to sustain injuries to the upper cervical spine.[6] At the present time, there roughly is an equal distribution between patients sustaining an injury resulting in quadraplegia (cervical) and an injury resulting in paraplegia (thoracic or lumbar), and there is a trend toward incomplete rather than complete injuries.[7]

The financial burden of spinal cord injuries is overwhelming. For example, it has been estimated that, for patients with a complete spinal cord injury, the cost of life-long medical expenses and lost wages exceeds $2,000,000.[2] In the first year post injury alone, it is estimated that health care and living expenses for a patient with a high cervical cord injury are in excess of $400,000.[1] With an estimated prevalence of 200,000 spinal cord injured patients in the United States requiring some form of medical care,[8,9] the yearly cost for these patients easily translates into billions of dollars.

The purpose of this review is to provide a categorization scheme for acute spinal cord injuries, and an outcome-effective evaluation and treatment protocol for emergency medicine practice.

— The Editor

Spinal Cord Injuries: A Practical Approach to Emergency Management

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Spinal Cord Injury: Clinical Pathophysiology and Anatomy

Spinal cord injury most often results from functional impairment of the cord, rather than from an actual anatomic transection.[2,4,10,11] The initial or primary insult usually occurs as a consequence of various physical forces that cause direct injury to the spinal cord. Forces responsible for producing impairment of function include compression, shear, laceration, and distraction.[2,12] As a result of primary injury, a cascade of secondary cellular changes occurs that can play a significant role in spinal cord injury. For example, after the primary event, there is a decrease in regional blood flow to the injured spinal tissue. This will lead to post-traumatic tissue ischemia and the initiation of a complex cascade of events causing an accumulation of free radicals, increased lipid peroxidation, increased intracellular calcium, and loss of cellular integrity.[2,4,8,10,13] The deficit in neuronal transmission responsible for the neurologic deficit can be caused by local spinal cord hypoxia, which may lead to ischemia, and, eventually, tissue infarction.[11]

The vertebral column consists of 33 vertabrae: 7 cervical vertebrae, 12 thoracic vertebrae, five lumbar vertebrae, five fused sacral vertebrae, and a variable number (3-5) of coccygeal vertebrae. The relationship of the vertebral column to the spinal nerve roots is presented in Figure 1. Within the confines of the vertebral column lies the spinal cord, which originates at the cervico-

medullary junction just caudal to the foramen magnum and, in adults, terminates at the level of the 2nd lumber vertebrae. The inferior portion of the spinal cord lies below the lumbosacral enlargement and is known as the conus medullaris, below which there is a continuation of nerve roots known as the cauda equina.

Blood supply to the spinal cord originates from the anterior spinal artery and the two, paired posterior spinal arteries, each of which originate as branches from the vertebral arteries. The anterior spinal artery extends for the entire length of the spinal cord and supplies the anterior two-thirds of the spinal cord. The two posterior spinal arteries supply the remaining, posterior one-third of the spinal cord. The spinal cord receives additional blood supply from radicular arteries of the aorta, including the radicular artery of Adamkiewicz, which enters the spinal canal in the lower thoracic region but sends branches as far cephalad as T_4.

When viewed in cross section, the spinal cord is composed of central gray matter containing cell bodies of neurons and surrounding white matter carrying ascending and descending motor and sensory tracts. *(See Figure 2.)* The white matter can be divided into three major motor and sensory tracts: 1) the dorsal (posterior) column; 2) the lateral corticospinal (pyrimidal) tract; and 3) the anterior spinothalamic tract. Each of these motor and sensory tracts can be clinically evaluated. The dorsal column carries nerve fibers for the transmission of proprioception, vibration, and touch. Ascending fibers will decussate at the cervico-medullary junction. The lateral corticospinal tract carries nerve fibers for the transmission of voluntary motor function. Ascending fibers for the upper extremities are medially located, while fibers for the lower extremities are positioned to more lateral locations at the periphery of the spinal cord. These ascending fibers also decussate at the cervico-medullary junction. The anterior spinothalamic tract carries fibers for transmission of pain, temperature, and light touch. These fibers will decussate in the anterior white commisure about one level above the point at which they enter the spinal cord. Understanding the topographic relationship and function of the spinal cord tracts will enhance accuracy of diagnosis of incomplete spinal cord injury syndromes.

Initial Assessment and Evaluation

Bystanders and prehospital personnel often are the first people to encounter individuals who may have sustained a spinal cord or spinal column injury. The initial care that is provided may determine whether a patient will ultimately regain normal neurologic function or suffer permanent injury. As in all trauma patients, an initial survey focusing on respiratory and cardiovascular stabilization is essential.[14] It is imperative that proper, spinal immobilization is implemented early if this type of injury is suspected. The head should be maintained in a neutral position and all attempts should be made to limit flexion or axial loading of the cervical spine.[15] Many iatrogenic spinal injuries that occur following trauma result from a combination of flexion and axial loading.[15] A properly sized rigid cervical collar should be applied to all patients suspected of having a spine injury. Lateral stabilization of the head should be maintained with either sand bags or application of towel rolls and taping of the head. Although helpful, these maneuvers do not completely immobilize the cervical spine, since some flexion may still occur. It is recommended that all patients with suspected spine injuries be transported on a spine board.[15] Prolonged exposure of any bony prominence (i.e., the occiput, scapula, sacrum, and calcaneus) may lead to the development of pressure sores. Hence, if a patient will be immobilized for an extended period of time, consider padding these areas. In helmeted patients, a two-person technique can be employed to remove the helmet to gain better access to the airway and to permit further inspection of the head.[16] One author recommends transporting spinal cord injured patients in the Trendelenberg position in order to minimize the risks of aspiration and shock, two major causes of death in the prehospital phase of treatment.[14,17] In the quadriplegic patient, vital capacity may be diminished by Trendelenberg positioning because the mass of the abdominal compartment can displace the diaphragm rostrally. Appropriate management of the airway may be required.

Prehospital personnel should have a low threshold for determining which patients should be immobilized in order not to miss a potential spine injury. In this regard, it is also important to consider spinal immobilization in high-risk patient groups, including individuals who are post-ictal or intoxicated, and patients with an altered mental status who may be victims of

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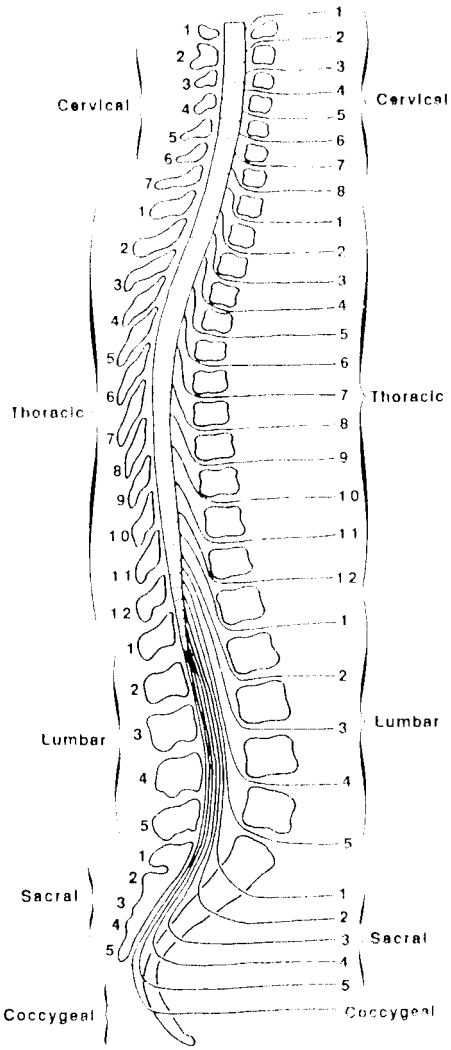
Figure 1. Diagram Illustrating the Relationship of Spinal Column Segments to Spinal Cord Segments



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occult trauma. Because spinal cord injuries are not always evident, it is preferable to be conservative when the decision is made to immobilize the spine. As a general rule, the following patients usually require spinal immobilization: any potential victim of trauma with neurologic deficits, and/or head, neck, or back pain; or any trauma victim with an altered mental status. In the field, a brief neurologic examination should be performed on all patients and should include an assessment of the level of consciousness, pupillary function, and evaluation of gross motor and sensory function of the upper and lower extremities.

Airway. It is critical that cervical spine control be maintained if, at any time, it is determined that the patient requires intubation. If the front of the cervical collar needs to be removed for inspection of the neck, application of Sellick's maneuver, or airway management, an assistant will be required to maintain manual, in-line cervical stabilization. The assistant's primary goal is to maintain the neck in a neutral position. The airway must be evaluated for patency and adequacy of ventilation and oxygenation. Any patient who has a vital capacity less than 15 mL/kg, a PaO_2 of less than 60-70 mmHg, or a $PaCO_2$ above 45 mmHg will likely require urgent intubation.[2,18] Hypoxia and hypercarbia may also cause an excessive increase in blood flow to the spinal cord leading to tissue edema.[18]

A number of intubation techniques have been used to secure an airway in patients suspected of having spinal cord injuries. These include orotracheal intubation; nasotracheal intubation; light wand assisted, retrograde, fiberoptic intubation; and cricothyroidotomy.[5,17-19] The optimal technique used will vary according to specific circumstances and the comfort level of the operator. If succinylcholine is used for rapid sequence intubation, it should be stressed that this agent may cause an increase in the serum potassium in patients who have spinal cord injuries. This can result from the hypersensitization of denervated muscle below the level of injury.[18] Typically, because this reaction does not occur prior to 48 hours, succinylcholine is safe to use in acute spinal cord injuries.

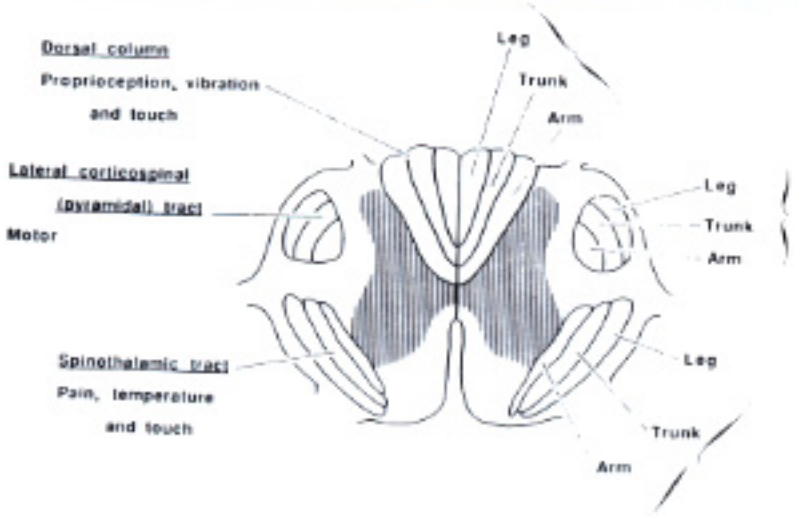
Cardiovascular System. All patients with suspected spine injuries should have large bore intravenous access, supplemental oxygen, and cardiac monitoring (i.e., the "ED safety net"). Vital signs should be closely monitored. In patients with a high thoracic or cervical cord injury, neurogenic shock may occur as a result of impaired sympathetic innervation to the heart.[2] These patients may present with hypotension, absolute or relative bradycardia, and warm peripheral extremities. Hypotension may occur in patients who are normovolemic as a result of increased venous capacitance. Cardiovascular stabilization should be directed at reversing hypoxia and hypotension to help prevent further secondary tissue injury.[14] Autoregulation is usually lost in the acutely injured spinal cord, and as a guideline the mean arterial pressure should be kept above 70 mmHg to prevent further ischemia.[2] Initially, these patients may be resuscitated with crystalloids, but vasopressor support eventually may be needed. Atropine can be used to treat symptomatic bradycardia.[18] An acute spinal cord injury may also leave the patient poikilothermic. This mandates the need for close monitoring of body temperature.

A careful evaluation should be sought for associated injuries that may be masked by the spinal cord injury. In any patient with sensory abnormalities, it may be necessary to perform a bedside diagnostic peritoneal lavage or an abdominal/pelvic CAT scan to rule out an associated intra-abdominal injury.[17] All patients should have an indwelling foley catheter placed to monitor urine output and prevent urinary retention. A nasogastric tube may need to be placed to decrease the likelihood of aspiration.

Radiographic Studies. After all life threatening conditions are identified and treated, radiographic evaluation may begin. Never delay initial management and stabilization to obtain radiographic studies. The cross-table lateral radiograph, which has long been considered the gold standard for evaluating the cervical spine, is not sensitive enough to radiographically clear the cervical spine.[20-24] Moreover, no single radiographic view can completely exclude an injury to the cervical spine.[17,20-24] In fact, various studies have shown that a single lateral cervical spine view is only 70-80% sensitive for identifying cervical spine fractures.[20-22,25] An initial cervical spine trauma series often consists of a cross-table lateral cervical spine radiograph followed by an anteroposterior and odontoid view. The sensitivity of this series has been noted in two studies to be as high as 93%.[21,23] One author recommends adding bilateral supine trauma oblique radiographs to the cervical spine series.[24]

A CAT scan may be indicated in patients with normal films

Figure 2. Cross Sectional View of the Spinal Cord and Diagram of Major Spinal Tracts



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but a high index of clinical suspicion, in patients with inadequate or equivocal plain films, to delineate fractures noted on plain film, or in patients with neurologic deficits. Magnetic resonance imaging (MRI) may be helpful in determining the extent of acute spinal cord compression, disc herniation, soft tissue changes, or ligamentous injury.[2] Flexion-extension views may be indicated in patients who are alert, non-intoxicated, and cooperative, but who have persistent neck pain, negative radiographs, and who are neurologically intact. If these views are performed, a physician should accompany the patient into the x-ray suite and assist with active range of motion. Under no circumstance should the patient's head be passively moved.[24] If a patient has a cervical spine fracture identified radiographically, there is approximately a 10% incidence of a second, non-contiguous vertebral fracture.[2,16] Therefore, it is important to obtain full spine radiographs if a fracture is identified, or if the patient is intoxicated, has a neurologic deficit, or an alteration in consciousness that precludes a complete physical examination.[26]

It is not uncommon to have an associated head and spine injury. In fact, it has been reported that 5-10% of head injured patients have an associated spinal injury, and that 25-50% of patients with a spine injury have an associated head injury.[16,28] Alcohol use is often associated with spinal cord injury. In one series, 87% of patients with spinal cord injuries were intoxicated upon arrival to the trauma center.[5] This may also present a diagnostic challenge when trying to identify major associated injuries.

After completion of the primary survey and initiation of resuscitative efforts, a detailed and systematic secondary survey should to be performed. While conducting the secondary survey, it is important to continue proper spinal immobilization. It has been reported that up to 10% of spinal cord injuries can occur when patients with unstable fractures are moved.[3,4,13,16] In patients with a suspected spinal cord injury, an understanding of the mechanism of injury may help reveal injury patterns. It is important to question the patient about complaints of neck pain, paresthesias, paralysis, weakness, or loss of consciousness at the time of the injury. The presence of any of these findings may suggest spinal cord involvement. Any patient with an altered mental status or who is intoxicated should heighten the physician's index of suspicion for spinal injury. A complete head to toe secondary survey should be performed in accordance with ATLS guidelines, including a careful examination of the head, neck, and axial skeleton.[16]

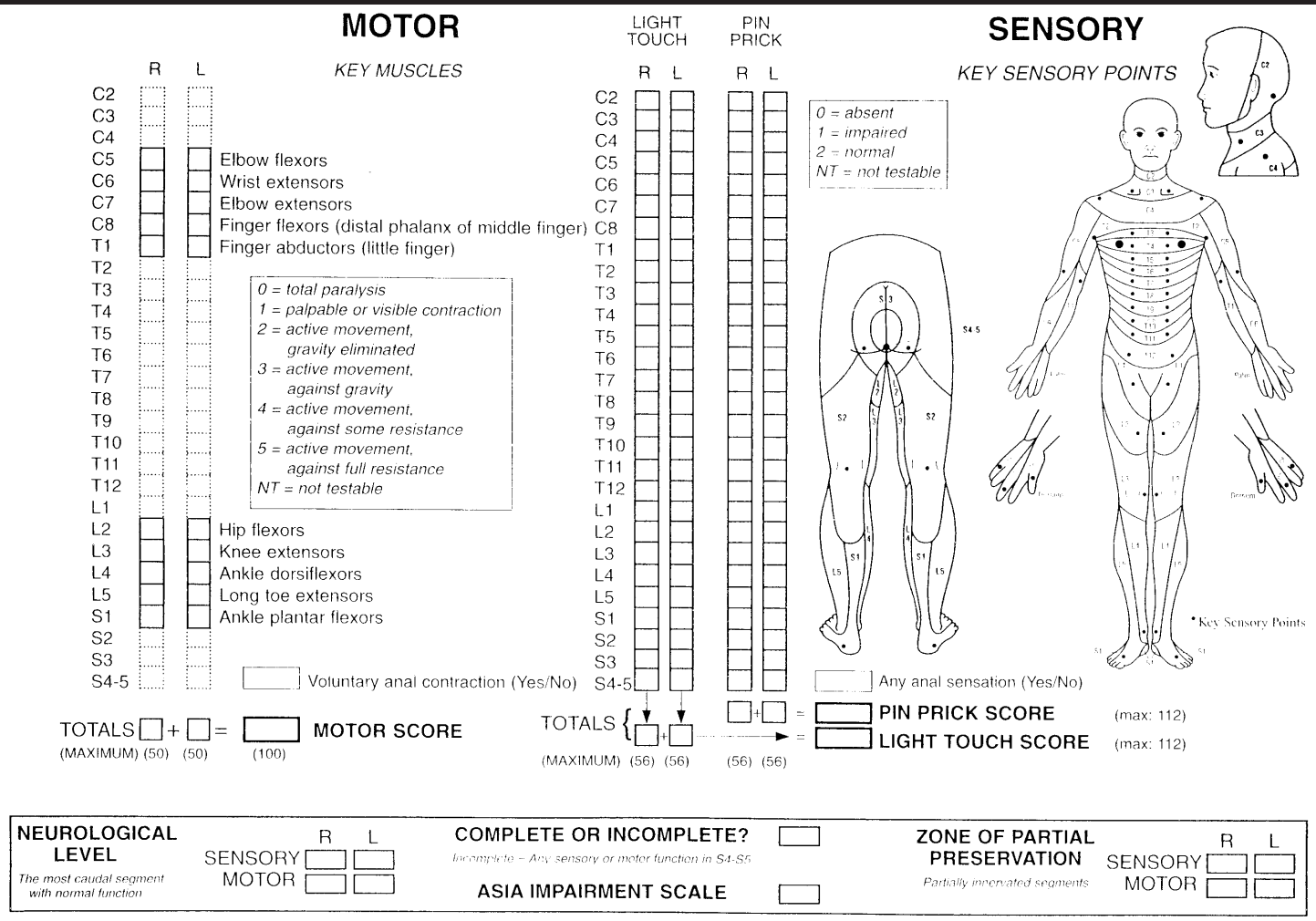
Neurological Examination. A thorough and detailed neurologic examination should attempt to determine the presence, severity, and level of neurologic dysfunction—these findings will be used as a basis for future comparison. The neurologic examination can begin with simple patient observation. How is the patient positioned? How does the patient hold his or her extremities? Abnormal extremity positioning in a conscious and alert patient may be a clue to a cervical cord injury The patient's breathing pattern will also need close observation. In lower cervical and upper thoracic cord injuries, there may be loss of innervation to the intercostal muscles resulting in a paradoxic (diaphragmatic) respiratory pattern. With a high cervical cord injury at or above the level of C_4, hypercapneic respiratory failure may ensue because of impaired phrenic nerve innervation to the diaphragm.

A detailed motor and sensory examination (pinprick and light touch) should also be performed as outlined in the American Spinal Cord Injury Association (ASIA) standard neurologic classification form. *(See Figure 3 and Table 1.)* Additional dorsal column functions (vibration and joint position) can be tested using a 128 Hz tuning fork or by evaluating proprioception of the great toe or index finger. Cord-mediated deep tendon reflexes, cranial nerve testing, and an evaluation of sacral sparing and islands of sparing should be noted. Sacral sparing is the presence of voluntary anal sphincter contraction or sensory function in the perianal region (S_{3-5}) or slight flexor toe movement of the great toe (S_1).[16] If present, this indicates that the lesion is incomplete and improves the prognosis for functional recovery. The presence of "islands of sparing" of sensation within an affected dermatome or below the level of apparent total dysfunction, even in the presence of complete motor paralysis, indicates a chance of functional motor recovery. The preservation of pinprick sensation between the level of injury and the sacral dermatomes may be the best prognostic indicator for useful motor recovery.[29]

Early neurosurgical consultation should be obtained in patients with spine injuries. Carefully screened patients with minor, stable vertebral column injuries such as thoraco-lumbar compression fractures, who are neurologically intact, can be discharged from the emergency department (ED). Bedside neuro/surgical consultation should be obtained for all patients with an unstable vertebral column or an acute spinal cord injury. Early stabilization and transfer of these patients to a regional spinal cord injury treatment center is advocated. These centers are able to provide the specialty care that is required which consists of a team of specially trained physicians, nurses, and other allied health professionals.

Additional Complications of Spinal Cord Injuries. It is not uncommon for a patient with a prior spinal cord injury to present to the ED seeking medical attention. These patients are at higher risk than the general population for a variety of medical illnesses and conditions. In patients with high thoracic and cervical cord injuries, an impaired ability to clear respiratory secretions increases the incidence of pneumonia. Venous stasis secondary to paralysis also increases the incidence of deep venous thrombosis

Figure 3. Standard Neurological Classification of Spinal Cord Injury



Source: American Spinal Injury Association

leading to pulmonary embolism. Pressure on insensate skin often leads to the development of decubitus ulcers. Heterotopic ossification occurs in some spinal cord injury patients, leading to ossification of muscle tissue and a decreased range of motion of the affected joint. Joint contractures and muscle spasticity are also common in this patient population. All patients with chronic indwelling foley catheters are at risk for urinary tract infections. Autonomic hyperreflexia or "mass reflex" is a unique condition affecting spinal cord injury patients. Patients may present with severe hypertension, diaphoresis, or pallor that can lead to seizures. This can occur as a result of hollow organ over distention (bladder or rectum)—initial treatment is directed at relieving the underlying problem (urinary retention or fecal impaction).

Spinal Cord Injury Syndromes

A complete spinal cord injury is one in which there is a complete loss of motor and sensory function below the level of the lesion. If any evidence of sacral sparing or islands of sparing is present, then the lesion is incomplete and there is some chance of functional recovery. In addition, a condition known as spinal shock may mimic a complete cord injury. Spinal shock, which usually occurs in conjunction with a severe spinal cord injury, is a concussive injury to the spinal cord producing total neurologic dysfunction distal to the site of the injury. Clinically, a patient may present with flaccid paralysis, areflexia, and complete anesthesia distal to the injury. Spinal shock also can result in autonomic dysfunction because of interruption of sympathetic innervation and can lead to hypotension, relative bradycardia from unopposed vagal stimulation, and warm peripheral extremities. Cessation of spinal shock is usually heralded by return of the bulbocavernosus reflex. To test this reflex, one has to perform a digital rectal examination and then gently tug on a properly placed foley catheter or squeeze the glans of the penis. A positive reflex is elicited if one feels contraction of the anal sphincter. The symptoms of spinal shock are often variable, but it can be assumed that motor and sensory deficits last only about one hour, although the reflex and autonomic nervous system deficits may persist for days to months.[27]

Incomplete spinal cord lesions can often be classified into one of three clinical syndromes: 1) anterior cord syndrome; 2) central cord syndrome; or 3) the Brown-Sequard syndrome.[13] The anterior cord syndrome often occurs as a result of forced hyperflexion. These patients typically present with a loss of motor function and loss of pain and temperature sensation below the injury; dorsal column functions (joint position, touch, and vibration) are preserved. Prognosis for this type of injury remains poor. The central cord syndrome usually occurs as a result of forced hyperextension. This injury may be associated with buckling of the ligamentum flavum or underlying degenerative arthri-

tis. Patients will present with a neurologic deficit that is more pronounced in the upper extremities than in the lower extremities because of the central location of the ascending and descending motor fibers of the upper extremities. The classic finding is loss of distal upper extremity pain, temperature, and strength, with relative preservation of lower extremity strength and sensation. The Brown-Sequard syndrome can be thought of as a functional hemisection of the spinal cord. The mechanism is often secondary to penetrating trauma, and these patients classically present with loss of ipsilateral motor strength, vibratory and joint position sense, and loss of contralateral pain and temperature sensation below the level of the injury. Usually, some bowel and bladder function is retained, and the prognosis is good.

Other less common, incomplete cord syndromes also have been noted. The cervico-medullary syndrome may mimic a central cord lesion, and can present with sensory loss over the face conforming to the onion skin or Dejerine pattern. The conus medullaris syndrome can present with bilateral lower extremity motor and sensory deficits, in addition to bowel and bladder deficits. This type of injury may appear similar to the cauda equina syndrome, but the former usually is associated with little or no radicular pain, while the latter often has radicular pain as a major complaint.

Another syndrome that is somewhat unique to children is the syndrome of spinal cord injury without radiographic abnormality (SCIWORA). The cause of injuries in this patient population seems to be similar to their adult counterparts.[30] Although often less commonly reported, one series of pediatric spinal cord injuries reported a 67% incidence of SCIWORA.[30] Young children also appear to be more likely to suffer a severe, upper cervical spinal injury.[30] Interestingly, in this group of patients, 27% had delayed onset of neurologic deficits, and some children were noted to have recurrent neurologic injury days to weeks after the initial trauma. These findings raise the concern of prolonged spinal instability. The majority of the cases associated with delayed deficits had transient but definite neurologic symptoms immediately following trauma. The transient symptoms included subjective paralysis, distal parathesias, and the Lhermitte phenomenon. The increased incidence of SCIWORA, as compared to adults, may be related to the relative elasticity of the spinal supporting ligaments and the immature osseous structure of the pediatric spinal column.[4,30]

Spinal Cord Injuries in Athletes

Sports and recreational activities account for 5-15% of all spinal cord injuries.[7,9,31-33] Two-thirds of these injuries occur as a result of diving accidents and an overwhelming majority occur in the lower cervical spine, leading to complete spinal cord injury.[7,9,34] In organized sports, football accounts for the greatest number of spine injuries.[32,35] Generally speaking, spinal injuries in athletes can be divided into three types.[35-37] Type 1 injuries consist of athletes who suffer permanent spinal cord injury. This group of patients can have either a complete or incomplete injury, or any of the previously mentioned spinal cord injury syndromes. It is safe to assume that either the clinical or radiographic evidence of a spinal cord injury will preclude return to contact sports.[33,36,37] A type 2 injury is one in which a transient neurologic deficit occurs without an identifiable bony injury. The presence of transient quadriplegia or the presence of bilateral neurologic symptoms should raise the suspicion of spinal cord compromise. This injury may occur as a result of an underlying spinal canal stenosis. The burning hands syndrome, as described by Maroon, is an example of this type of injury.[38] It can be considered analogous to the central cord syndrome seen in older adults. The mechanism of this injury usually is hyperextension. A key point is that painful dysesthesias or burning hands may be the only complaint of patients with spinal cord injuries. One author recommends that if an athlete has spinal cord symptoms from a sports-related injury and is shown to have functional spinal canal stenosis on MRI, he or she should not be allowed to return to contact sports.[39]

In addition, there is a commonly encountered football-related injury called a stinger or burner. This type of injury is not a true spinal cord injury. The mechanism of injury is usually related to traction on the brachial plexus or nerve root impingement.[36,37,40] In most situations, symptoms last seconds to minutes and involve pain, burning, or tingling down an arm; this may be accompanied by localized weakness. These symptoms are most commonly noted in the C_5-C_6 nerve root distribution,[34,35] are always unilateral, and never involve the lower extremities. When evaluating an athlete who has suffered a suspected stinger injury, a careful distinction must be made between this type of injury and a true spinal cord injury. A type 3 injury includes an injury to the vertebral column without neurologic deficits. Also included in this category are ligamentous injuries and herniated intervertebral discs.

Another entity called spear tackler's spine also has been identified. Permanent neurologic injury has occurred in athletes with the following abnormalities noted on cervical spine films: 1) Developmental narrowing of the cervical spinal canal; 2) straightening or reversal of the normal cervical lordotic curve; and 3) pre-existing minor post-traumatic radiographic evidence of bony or ligamentous injury. In addition, the athletes were documented as using spear tacking techniques.[33,36,37] The decision as to whether to allow an athlete to return to contact sports after he or she has experienced a type 2 or 3 injury is often complex and will need to be made on an case-by-case basis.

Pharmacologic Therapy of Acute Spinal Cord Injury

Initial animal studies simulating spinal cord injury models per-

Table 1. ASIA Impairment Scale

- ❑ **A = Complete:** No motor or sensory function is preserved in the sacral segments S4-S5.
- ❑ **B = Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.
- ❑ **C = Imcomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.
- ❑ **D = Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.
- ❑ **E = Normal:** Motor and sensory function is normal.

CLINICAL SYNDROMES

- ❑ Central Cord
- ❑ Brown-Sequard
- ❑ Anterior Cord
- ❑ Conus Medullaris
- ❑ Cauda Equina

formed in the 1970s served as the basis for future clinical trials. Glucocorticoids have long been known for their anti-inflammatory properties. Initially, it was thought that glucocorticoids could help prevent secondary spinal cord edema.[41] In 1979, the National Acute Spinal Cord Injury Study Group (NASCIS) performed the first multicenter, randomized, clinical trial evaluating the use of methylprednisolone (MPS) in the treatment of acute spinal cord injury.[42] The study, NASCIS I, compared the efficacy of a 1 gm bolus dose of MPS followed by 1 gm daily for 10 days with a similar dosing regimen of 0.1 gm of MPS. There was no significant difference between the two dosing regimens observed in neurologic recovery of motor or sensory function (pinprick and touch) one year after injury. At the time that the study was conducted, it was felt that steroids were beneficial and that withholding them would be unethical; as a result, there was no placebo control.

NASCIS II was based on extensive studies of MPS in an experimental model of spinal cord injury.[41] The mechanism by which steroids work is still somewhat unclear, but a leading theory is that steroids limit post-traumatic lipid peroxidation.[43] NASCIS II, published in 1990, was the first randomized, double-blind, placebo-controlled trial that unequivocally demonstrated that steroids could modify recovery from a severe, non-penetrating spinal cord injury.[44,45] This study compared three treatment arms: MPS 30 mg/kg bolus, followed by 5.4 mg/kg/hr for 23 hours, naloxone hydrochloride, and a third placebo arm. Initial data analysis did not reveal a difference between the three treatment groups. Subgroup stratification showed that increased recovery rates of neurologic function at six weeks, six months, and one year occurred in patients treated with MPS within eight hours of injury, as compared to those treated with either naloxone or placebo.

In 1991, a study was published that reported the results of a randomized, double-blind, placebo-controlled study evaluating the use of GM-1 gangliocide in acute spinal cord injury.[11] Experimental evidence suggests that these agents augment neurite growth in vitro, and may induce regeneration of neurons and restore neuronal function after injury in vivo.[4] The study consisted of two treatment arms: GM-1 gangliocide 100 mg administered intravenously daily for 18-32 days, and a second placebo arm. In addition, both groups received MPS 250 mg intravenously followed by 125 mg every six hours for 72 hours. Data analysis showed a statistically significant improvement in neurologic function at one year in patients treated with GM-1 gangliocide as compared to placebo. Improvement in patient scores resulted from greater recovery for the lower extremities as compared to the upper extremities, and it appeared that a pattern of recovery was noted in patients who initially had complete paralysis as compared to weak muscles.

In 1997, the results of NASCIS III were published.[46] This study was a randomized, double-blind trial evaluating the use of a 24- or 48-hour protocol of MPS or tirilazad mesylate, a potent lipid peroxidase inhibitor. All three treatment arms received a 30 mg/kg intravenous bolus of MPS prior to randomization. One arm then continued a MPS infusion of 5.4 mg/kg/hr for 23 hours, the second arm continued the infusion for 48 hours, and the third arm received tirilazad as a 2.5 mg/kg bolus every six hours for 48 hours. The 48-hour MPS regimen showed statistically significant improvement in motor recovery at six weeks and six months among patients whose therapy was initiated 3-8 hours after injury. Patients treated with tirilazad for 48 hours showed motor recovery rates equivalent to patients who received MPS for 24 hours.

At the present time, administration of high-dose glucocorticoids has become standard of care in patients with acute, non-penetrating spinal cord injury. All patients should receive an initial intravenous bolus of 30 mg/kg of MPS within eight hours of injury, followed by 5.4 mg/kg/hr for 23 hours.[2,12,17] Steroid administration should be initiated as soon as the neurologic deficit is recognized. Recently published studies, including NASCIS III and other investigational agents, will require further validation before these recommendations are changed.

Summary

In a previously healthy person, an acute spinal cord injury can be devastating. Continued prevention strategies aimed at young adults who are most likely to suffer a spinal cord injury may help reduce the incidence of these injuries occurring. Federal and state legislation addressing the use of seat belts, motorcycle helmets, and drunk driving are playing a role in the reduction of spinal cord injuries.[9] As emergency medicine specialists we must continue to be systematic in our approach to these patients; prompt, efficient, and expert care can play a significant role in improving outcomes.

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Physician CME Questions

97. Central cord syndrome will classically present with which of the following clinical findings?
    A. Paralysis below the injury with preservation of position sense and vibratory sensation
    B. Distal upper extremity weakness greater than proximal upper extremity weakness greater than lower extremity weakness
    C. Paralysis below the injury with preservation of pain and temperature sensation
    D. Complete motor and sensory loss below the injury

98. A C_8 cervical cord injury will present with which of the following motor findings?
    A. Decreased elbow flexion
    B. Decreased wrist extension
    C. Decreased finger flexion
    D. Decreased elbow extension

99. An elderly patient with underlying cervical spine arthritis suffers a spinal cord injury as a result of excessive hyperextension of the cervical spine. Which of the following incomplete spinal cord injury syndromes would be most likely to occur?
    A. Anterior cord syndrome
    B. Posterior cord syndrome
    C. Central cord syndrome
    D. Brown-Sequard syndrome

100. Spinal shock often presents with which of the following vital sign patterns?
    A. Hypertension and bradycardia
    B. Hypotension and bradycardia
    C. Hypertension and tachycardia
    D. Hypotension and tachycardia

101. What percentage of head injured patients have an associated spinal injury?
    A. 0-5%
    B. 5-10%
    C. 10-15%
    D. 15-20%

102. How sensitive is a single, cross table, lateral view of the cervical spine in identifying a cervical spine fracture?
    A. 90-100%
    B. 80-90%
    C. 70-80%
    D. 60-70%

103. What is the suspected level of spinal cord injury in a patient who cannot extend his arms at the elbow?
    A. C_4
    B. C_5
    C. C_6
    D. C_7

104. When should methylprednisolone be administered to a patient with an acute spinal cord injury?
    A. After neurosurgical consultation
    B. As soon as the neurologic deficit is recognized
    C. Just prior to transfer to a regional spinal cord center
    D. After definitive open reduction of a vertebral fracture is performed

In Future Issues

Deep Venous Thrombosis