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STATEMENT OF FINANCIAL DISCLOSURE

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, Dr. Abraham (author) is a stockholder in American Biomed, Opko Pharmaceuticals, and Hologic. Dr. Dietrich (editor in chief), Dr. Brown (author), Dr. Perron (peer reviewer), Ms. Behrens (nurse reviewer), Ms. Mark (executive editor), Leslie Coplin (executive editor), and Mr. Landenberger (continuing education and editorial director) report no relationships with companies related to this field of study.

AHC Media

Spinal Cord Injury

Introduction

Spinal cord injury (SCI) is a devastating disease for both the patient and the caregivers. The spinal cord, although well protected, can be injured in a variety of ways, including motor vehicle collisions and sporting events. The inability of the neurons to regenerate, and their sensitivity to anoxia and hypoperfusion, makes the timely diagnosis and treatment of SCI imperative to preserve as much function as possible. This article will cover the basic epidemiology, physiology, and treatments for SCI in an attempt to prepare the reader to manage these complex injuries.

Epidemiology

Incidence and Prevalence

Spinal cord injury is a devastating result of trauma, with profound consequences on quality of life. In 2012, an estimated 12,500 to 17,000 people in the United States sustained acute, traumatic SCI, translating to an incidence of 40 to 54 per 1 million Americans.[1,2] Compared with the incidence in the 1970s,[3] SCI has become less common among people who are 16 to 45 years of age and more common among those older than 65 years of age.[2] Its prevalence is difficult to calculate, but it is estimated that 276,000 people in the United States are living with SCI.

The costs associated with SCI are staggering. First-year costs range from $342,000 to $1 million, and the lifetime cost of care for a 50-year-old high-tetraplegic patient can reach $2.5 million.[4] In addition to the financial burden, the cost in regard to loss of life is also considerable. The survival rate is difficult to calculate, given the underreporting of SCI within the first 24 hours after injury; however, patients with SCI who survive 24 hours and are still alive one year after injury have reasonable life expectancies, depending on their American Spinal Injury Association (ASIA) score and level of injury. Among 20-year-olds with ASIA D at any level, the median life expectancy is 52.3 years (the median survival rate for people at this age without SCI is 59 years). A 60-year-old who is ventilator-dependent after SCI has a life expectancy of two years (22.7 years without SCI).[1]

Gender

Most SCIs are sustained by men.[1,3] In 2014, the National Spinal Cord Injury Statistical Center (NSCISC) reported that 80.7% of all reported SCIs occurred in males.

EXECUTIVE SUMMARY

- The most common causes of spinal cord injury are motor vehicle collisions (31.5-47.6%), falls (21.8-40.6%), and violence (5.4-14.6%), primarily shootings.
- A recent epidemiologic study found an increase in spinal cord injury resulting from falls between 1997 and 2012, from 19.3% to 40.4%; the increase was most pronounced in the over-65 age group: from 28% between 1997 and 2000 to 66% between 2010 and 2012.
- There are four phases of spinal shock characterized by: 1) acute areflexia/hyporeflexia (0-1 day); 2) return of cutaneous reflexes (1-3 days); 3) early hyperreflexia (1-4 weeks); and 4) late hyperreflexia/spasticity (1-12 months). Lesions should not be referred to as "complete" prior to the resolution of acute hyporeflexia.
- The most common incomplete syndrome is acute traumatic central cord syndrome (ATCCS), which is characterized by weakness in the upper extremities (more than in the lower extremities) with some degree of sensory (decreased pain and temperature sensation) and bladder dysfunction.
- Anterior cord syndrome is rare, commonly occurring as a vascular insult to the anterior spinal arteries, resulting in ischemia of the anterior two-thirds of the spinal cord with motor weakness/paralysis, loss of pain and temperature sensation, and loss of voluntary bladder control, with preservation of vibration and position sense due to the sparing of the dorsal column medial lemniscus tracts.
- Brown-Séquard syndrome, also known as hemicord syndrome, occurs as the result of transection of half of the spinal cord with the resulting deficits of ipsilateral upper motor neuron weakness and impairment of vibration and position sense, with contralateral loss of pain and temperature sensation.
- The American Association of Neurological Surgeons and the Congress of Neurological Surgeons recommend aggressive treatment of hypotension by maintaining a mean arterial pressure (MAP) at or above 85-90 mm Hg for the first seven days following injury to improve spinal cord perfusion. Although, no formal recommendations regarding the selection of vasoactive medications exist, norepinephrine, phenylephrine, and dopamine are reasonable first-line agents.
- Venous thromboembolism (VTE) is a significant cause of morbidity and mortality, with pulmonary embolism accounting for nearly 10% of deaths within the first year after injury. Patients should have both mechanical and chemical VTE prophylaxis started upon admission and continued for three months.

Age

As the average age in the United States rises, so does mean age at the time of injury. The U.S. Census Bureau predicts that the proportion of Americans 65 years of age and older will increase from 13.7% in 2012 to 20.3% in 2030, and to 20.9% in 2050.[5] The NSCISC calculated that the mean age at time of injury increased from 28.7 years between 1973 and 1979 to 42.2 years between 2010 and 2014.[1]

Etiology

Between 1997 and 2012, the most common causes of SCI were motor vehicle collisions (31.5-47.6%), falls (21.8-40.6%), and violence (5.4-14.6%), primarily shootings.[1,2,6] The proportion of SCIs resulting from falls has grown significantly since 1997, most likely because of the rising mean age in the United States; the numbers of SCIs associated with violence and sports have fallen.[1] A recent epidemiologic study by Jain et al found an increase in SCI resulting from falls between 1997 and 2012, from 19.3% to 40.4%. This increase was most pronounced in the over-65 age group: from 28% between 1997 and 2000 to 66% between 2010 and 2012.[2]

Pathophysiology

Spine Anatomy

The human spine is composed of 33 vertebrae: seven cervical, 12 thoracic, five lumbar, five fused sacral, and four fused coccygeal bodies. The resulting 26 units are held together by a complex system of intervertebral disks, articulations, and structural ligaments. The spinal column functions as a load-bearing column for the thorax and provides protection for the spinal cord.

Approximately 85% of all SCI are fractures, and 10% involve subluxation without associated fracture. The remaining are SCI without radiographic abnormalities (SCIWORA) of the bones or ligaments.[7]

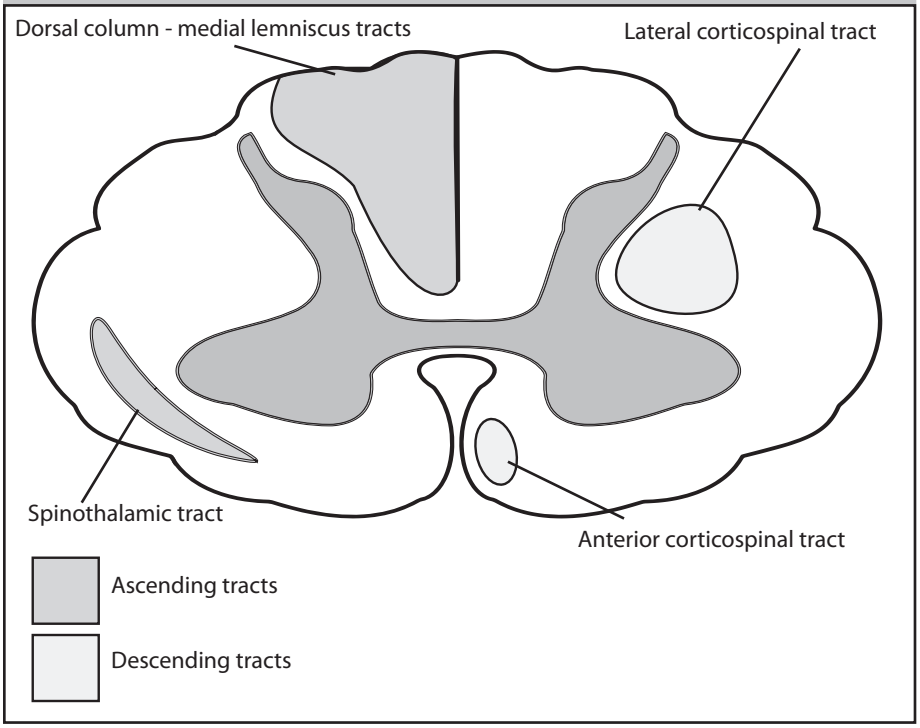
Acute spinal trauma is most likely to occur in areas of maximal mobility and generally is classified based on its location (craniocervical, subaxial cervical, or thoracolumbar) and the mechanism of injury. Either individually or in combination, flexion, extension, rotation, and axial compression are the most common mechanisms of injury,[8,9] resulting in fractures, disruption of ligamentous structures, and instability of the spinal column. The "three-column" concept of the spine, developed in 1984 by Denis, states that at least two of the three spinal columns must be disrupted to result in spinal instability.[10] However, in patients with underlying pathology (e.g., elderly patients with cervical spondylosis), this rule might not apply because forces can compress the spinal cord against pathologically enlarged elements.

The anterior column is composed of the vertebral bodies and their stabilizing, ligamentous structures: the annulus fibrosus capsule and the anterior longitudinal ligament. The middle column is delineated anteriorly by the posterior longitudinal ligament and posteriorly by the articulating surfaces of the paired laminae and pedicles. The posterior column is largely ligamentous, consisting of the spinous processes, nuchal ligament, infra- and supra-spinous ligaments, and ligamentum flavum.[11]

Spinal Cord Anatomy

The spinal cord is the principal reflex center and conduit of information traveling between the body and the brain. Understanding the anatomy and physiology of the spinal cord is crucial in the determination of injury patterns.

Figure 1. Anatomy of the Spinal Cord



The cord begins as a continuation of the medulla, originating at the foramen magnum in the occipital bone, and extending to the L1–2 vertebral level. It is protected from injury by the cerebrospinal fluid (CSF); the meninges; the fat-filled epidural space; and the vertebral bodies, ligaments, and muscles that compose the spinal column.[11]

The spinal cord can be divided further into gray matter and white matter. Gray matter is composed largely of axonal bodies and acts as an intermediary between the peripheral nervous system and the central nervous system. White matter is composed mainly of highly myelinated axons, with very few axonal bodies, and is designed to carry information quickly.

Spinal gray matter is arranged in an "H" shape running centrally down the length of the spinal cord and is divided into anterior, lateral, and posterior columns. The anterior column contains motor neurons derived from the pyramidal tract, which are responsible for purposeful movement of muscles. The posterior column is largely committed to the synapse of sensory neurons coming from the body and intended for the brain. Information from the body enters through the dorsal root ganglion, synapses within the posterior gray column, and is transmitted via the dorsal column-medial lemniscus tract (fine touch, vibration, two-point discrimination, and proprioception) and the spinothalamic tract (pain and temperature). The lateral column is composed of neurons dedicated to the sympathetic division of the autonomic nervous system.

White matter is organized into groups of ascending (sensory) and descending (motor) tracts (*see Figure 1*). The major descending tract is the corticospinal tract, which is divided into the lateral (control of contralateral muscles) and anterior (control of contralateral axial and girdle muscles) tracts. The lateral corticospinal tracts are organized somatotopically (cervical fibers are more medial than thoracic than lumbar) and play a large role in the motor symptoms of spinal cord injury. The main ascending tracts are the spinothalamic and dorsal column-medial lemniscus tracts. The fibers of the spinothalamic tract enter the spinal cord and cross to the contralateral ascending tract within a few levels of entrance; the fibers of the dorsal columns do not cross until they reach the medulla. This leads to sensory dissociation in incomplete spinal cord injuries, as described below.

Spinal Cord Injury

Spinal cord injury occurs through two pathways: primary and secondary.[12,13] Primary injury occurs mechanistically, at the time of injury, and is irreversible.[14] It has four characteristic mechanisms.[15] The most common is impact that causes persistent compression of the spinal cord by extradural elements. Laceration or transection occurs most frequently via penetrating trauma or extreme blunt trauma, resulting in significant displacement of the spinal column. Distraction injuries arise as a result of stretching of the spinal cord and/or its blood supply and may occur without overt fracture or ligamentous injury. SCIWORA often is observed in children because of ligamentous laxity[16] but is being recognized more frequently in adults.[17] The last mechanism is impact with transient compression, as seen in patients with degenerative cervical spine disease who sustain hyperextension injuries.

Secondary injury results from a cascade of vascular, cellular, and biochemical events initiated by the primary (mechanical) injury. Initial cord compression leads to hemorrhage and vasospasm, inhibition of microcirculatory autoregulation, increased permeability of the plasma cell membrane, and an increase in calcium influx, all of which ultimately contribute to ischemia and cell death. This scenario is exacerbated by systemic hypotension from spinal and/or neurogenic shock as well as hypoxia resulting from respiratory system compromise. An uncontrolled local inflammatory response ensues, with influx of immune cells and release of proinflammatory cytokines and free radicals. In combination, these factors contribute to the accumulation of neurotoxic substances such as glutamate, oxygen free radicals, and tumor necrosis factor (TNF)-alpha.[13] This cytotoxic cascade is a perpetual vicious cycle that causes more cellular death and, thus, more cytotoxicity, which repeats and amplifies with each cycle.

Clinical Features

Spinal injuries should be considered in trauma victims who are injured by a high-risk mechanism or unknown

Table 1. Normal Muscles of Respiration and Their Innervation

Innervation	Muscle Group	Function
Phrenic nerve (C3-5)	Diaphragm	Most important muscle of inspiration; pushes abdominal cavity downward to increase vertical capacity of the thoracic cavity
Intercostal nerves (T1-6)	Intercostal muscles	During inspiration, external intercostals serve to lift the ribs, increasing thoracic cavity volume During forced expiration, the internal intercostals contract to reduce the thoracic cavity volume
Spinal nerves (T5-L1)	Rectus abdominus, external/internal oblique muscles	Contraction during forced expiration causes intraperitoneal contents to push the diaphragm superiorly
CN XI, spinal nerves (C2-8)	Scalenes, sternocleidomastoid (inspiration) Pectoralis major, latissimus dorsi (expiration)	Elevation of the ribs 1-6 and sternum Flexion reduces thoracic cavity volume

scenario. These injuries should be suspected in patients with spinal tenderness, neck pain, significant facial/head trauma, altered mental status, focal neurologic deficit, or signs of neurogenic shock. Patients should be resuscitated according to the American College of Surgeon's Advanced Life Support algorithm.[18] Airway, breathing, and circulation should be assessed and dealt with prior to evaluation of disability (the neurologic examination).

Physical examination findings that should raise suspicion for spinal cord injury include diaphragmatic breathing, hypotension without obvious cause, bradycardia, priapism, flaccid areflexia, and loss of pain response below a dermatomal level.[19]

The 2010 American Spinal Injury Association (ASIA) Standards[20] are the preferred evaluation tool for the neurologic assessment of adult patients with SCI.[21] The examination consists of a comprehensive evaluation of motor function, sensation, and rectal tone/sensation. Sensation is evaluated via pinprick for pain and light touch through the dermatomes from head to toe on the right and left sides. Motor function is then tested through 10 muscle groups, bilaterally, on a scale of 0 (total paralysis) to 5 (full range of motion against gravity and resistance). The neurologic level of injury (NLI) then is determined by identifying the most caudal segment of the cord with intact sensation and antigravity (motor score > 3) motor function. For instance, a patient with absent, bilateral sensation from the C7 dermatome and with 3/5 wrist extension bilaterally would be considered to have an NLI of 6.

The next step is to determine whether the injury is complete or incomplete. This is accomplished via rectal examination. A lesion is considered complete (ASIA A) if the patient has an absence of voluntary anal contraction, a lack of sensation in S4–5, and a lack of sensation to deep anal pressure. Incomplete lesions can be classified as sensory incomplete (ASIA B, preserved S4–5, or deep anal pressure sensation) or motor incomplete (ASIA C/D), depending on muscle grading below the NLI. For example, a patient with an NLI of 6 (insensate from C7 dermatome down, with 3/5 wrist extension) but with preserved S4/5 sensation and > 50% of muscles below C6 being graded > 3/5 would be an ASIA 6C.

Neurologic function should be assessed in serial fashion over the days and weeks following the acute incident, keeping in mind that secondary SCI are likely to occur.

The NSCISC has tracked neurologic level at discharge since its creation in 1983 (from 1973 to 1981, it operated as the National Spinal Cord Injury Data Research Center at the University of Alabama at Birmingham). In 2014, the center reported that 53.9% of patients had sustained cervical lesions, 35.2% had thoracic lesions, 10.5% had lumbar lesions, and the remaining 0.4% had sacral lesions. The most common lesions involved C4 (14.8%), C5 (15.3%), C6 (10.4%), T12 (6.3%), C7 (5.1%), and L1 (4.9%). Over the past 40 years, the most common neurologic deficits at discharge were incomplete tetraplegia (31.6%), complete paraplegia (24.6%), complete tetraplegia (19.3%), and incomplete paraplegia (18.6%). The incidence of complete tetraplegia and paraplegia has decreased over the past four decades, from 25.3% and 27.7% in the 1970s to 12.4% and 18.5% between 2010 and 2014, respectively.[1]

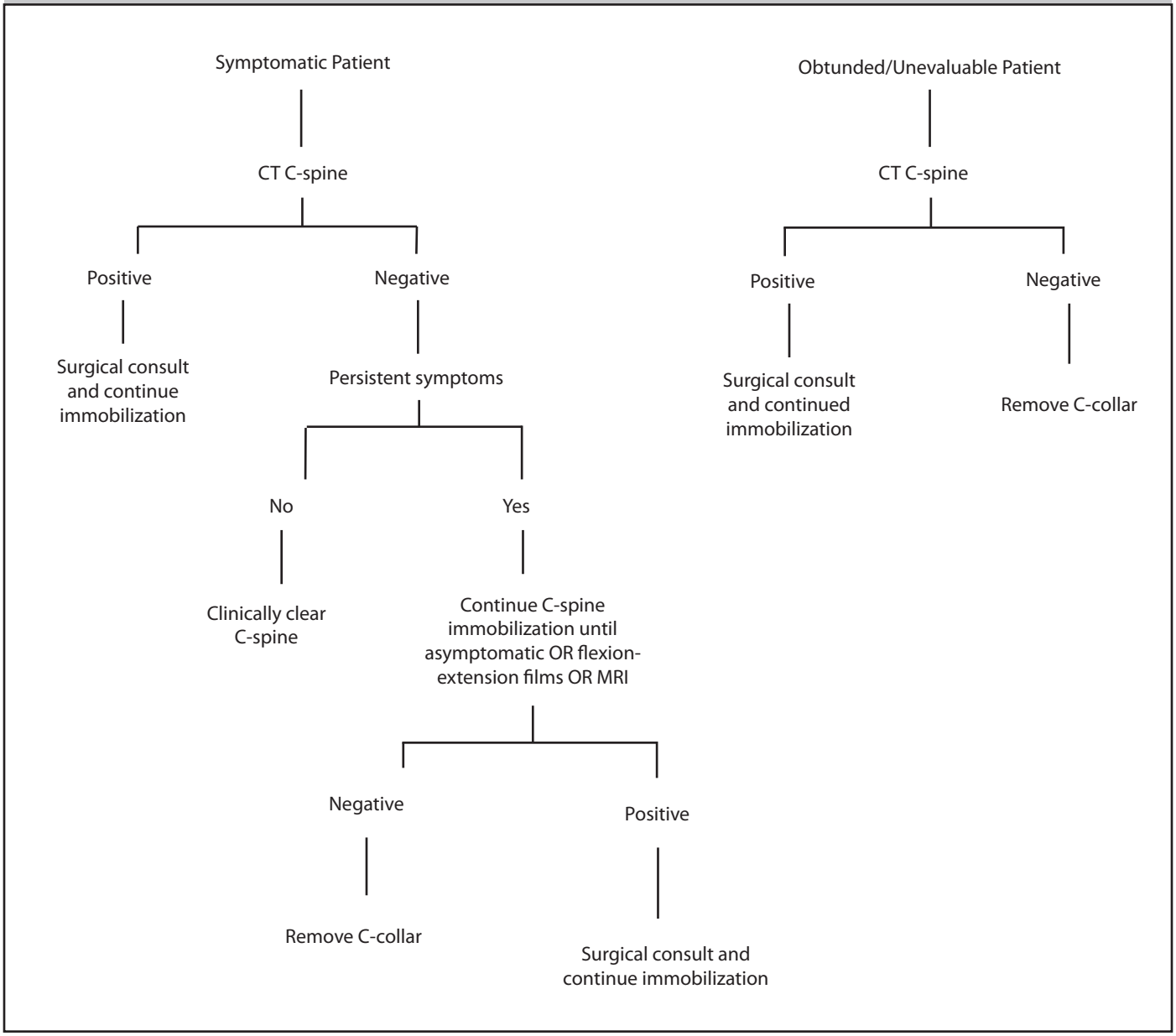
Complete Cord Syndrome

Complete cord syndrome results in disruption of all descending and ascending pathways and is characterized by acute flaccid paralysis and loss of sensation below the level of spinal cord injury.[22] This pattern often is mimicked by spinal shock, wherein concussive forces stun afferent and efferent tracts with concurrent loss of spinal reflexes. Ditunno[23] described four phases of spinal shock characterized by 1) acute areflexia/hyporeflexia (0–1 day); 2) return of cutaneous reflexes (1 day–3 days); 3) early hyperreflexia (1–4 weeks); and 4) late hyperreflexia/spasticity (1–12 months). Lesions should not be referred to as "complete" prior to the resolution of acute hyporeflexia. It is widely observed that the bulbocavernous reflex marks the end of the first phase of spinal shock, after which a true assessment of neurologic deficits can be performed. The bulbocavernosus reflex is elicited by simultaneous digital rectal exam and lightly squeezing the glans penis of male patients or gently tugging on a Foley catheter placed in male or female patients. An involuntary increase in tone around the examiner's digit with these maneuvers indicates the presence of an intact bulbocavernosus reflex (normal).

Incomplete Cord Syndromes

The incomplete spinal cord

Figure 2. Spinal Imaging of Symptomatic and Obtunded Patients



syndromes are central cord syndrome, anterior cord syndrome, and Brown-Séquard syndrome.

Central Cord Syndrome

The most common incomplete syndrome is acute traumatic central cord syndrome (ATCCS).[24] Clinically, it is characterized by weakness in the upper extremities (more than in the lower extremities) with some degree of sensory (decreased pain and temperature sensation) and bladder dysfunction. Burning sensory dysesthesias are particularly common with central cord syndrome. It is often described as a "cape-like" distribution of deficits. The injury traditionally occurs after a hyperextension injury in an elderly patient with preexisting cervical spondylosis and osteoarthritis with or without disc herniation or fracture subluxation.[25] Disruption of the ligamentum flavum then leads to damage of the central portion of the spinal cord. Motor deficits are preferentially confined to the upper extremities because of the somatotopic organization of the lateral corticospinal tract. Sensory deficits are manifested by disruption of the spinothalamic fibers as they cross through the anterior commissure, resulting in loss of pain and temperature but preservation of fine touch, vibration, two-point discrimination, and proprioception.[22]

Anterior Cord Syndrome

Anterior cord syndrome is a rare finding in patients with traumatic SCI; it more commonly occurs as a vascular insult to the anterior spinal arteries, resulting in ischemia of the anterior two-thirds of the spinal cord. Traumatic anterior cord syndrome usually results from anterior spinal cord compression by adjacent extradural elements following a hyperflexion injury.[26] Patients have motor weakness/paralysis, loss of pain and temperature sensation, and

Table 2. Mechanisms of Respiratory Dysfunction

Impaired inspiratory capacity 1. Decreased respiratory muscle strength and fatigue 2. Paradoxic chest wall movement causing an increase in effort of breathing 3. Decreased inspiratory capacity 4. Atelectasis 5. Chest wall rigidity	Autonomic nervous system dysfunction 1. Increased secretions 2. Bronchospasm 3. Pulmonary edema
Retained secretions and development of mucus plugs 1. Increased secretion production 2. Decreased cough effectiveness	

Table 3. Causes of Hypotension

- Hemorrhage
- Tension pneumothorax
- Myocardial injury
- Pericardial tamponade
- Sepsis
- Adrenal insufficiency
- Other traumatic or medical causes

loss of voluntary bladder control, with preservation of vibration and position sense due to the sparing of the dorsal column-medial lemniscus tracts.

Brown-Séquard Syndrome

Brown-Séquard syndrome, also known as hemicord syndrome, occurs as the result of transection of half of the spinal cord. Resulting deficits are ipsilateral upper motor neuron weakness and impairment of vibration and position sense, with contralateral loss of pain and temperature sensation. Injury is usually the result of penetrating trauma to the spinal cord but also can occur after lateral mass fracture. Hemisection in the cervical region can result in ipsilateral Horner syndrome (proptosis, miosis, anhidrosis) due to disruption of descending autonomic (sympathetic) pathways located within the lateral horn.[22]

Diagnostic Studies

In 2013, the Joint Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons published an extensive review on imaging of suspected spine trauma. This review states that, following a brief neurologic examination, three broad categories of trauma patients should be considered when making imaging decisions: 1) the awake, asymptomatic patient; 2) the awake, symptomatic patient; and 3) the obtunded or unevaluable patient.[27] Imaging and further immobilization are not required for patients who are awake; are not intoxicated; do not have neurologic symptoms, neck pain, or distracting injuries; and have painless range of motion.[27] This recommendation is supported by the Canadian C-Spine Rule (CCR) and the National Emergency X-Radiography Utilization Study Group (NEXUS) criteria.

The CCR, developed in 2001 by Stiell et al,[28] is based on three criteria: 1) the presence of a high-risk factor that mandates radiography (i.e., age > 65 years, dangerous mechanism, or paresthesia in extremities); 2) the presence of a low-risk factor allowing safe assessment of range of motion (i.e., simple rear-end motor vehicle collision, sitting position in ED, ambulatory at any time following injury, delayed onset of neck pain, or absence of midline C-spine tenderness); and 3) the ability to actively rotate the neck 45˚ to the left and right. Application of the CCR resulted in 100% sensitivity in the detection of significant cervical spine injuries, so the authors concluded that C-spine radiography is unnecessary in patients who meet CCR criteria. The study excluded patients younger than 18 years and older than 65 years of age and is not validated for use in those patient populations.

The NEXUS study[29] designed a protocol consisting of five criteria used to identify patients as having a low probability of injury: no midline cervical spine tenderness, no focal neurologic deficit, normal alertness, no intoxication, and no painful, distracting injury. Application of these criteria resulted in 100% sensitivity for ruling out C-spine injury. The relative strength of NEXUS in comparison to CCR is that the NEXUS population included children and the elderly, making it more applicable to emergency department (ED) populations.

In 2009, the Eastern Association for the Surgery of Trauma (EAST) concluded that CT has become the primary imaging modality for trauma patients requiring cervical spine imaging, supplanting three-view plain films.[30] If CT is unavailable, then three-view cervical spinal plain films (anteroposterior, lateral, and odontoid views) should be obtained.[31]

Awake, symptomatic patients with negative C-spine CT imaging but with persistent symptoms should be managed in one of three ways (*see Figure 2*)[31]:

1. Continue cervical immobilization until the patient becomes asymptomatic.
2. Discontinue cervical immobilization after normal and adequate dynamic flexion/extension radiographs have been obtained.
3. Discontinue cervical immbolization if a normal MRI is obtained within 48 hours after injury.

The evaluation of an obtunded patient can be more challenging, as unrecognized cervical spine injuries are associated with worsening neurologic status, paralysis, and death (*see Figure 2*). There has been much debate about the utility of MRI in the clearance of these patients, culminating with a recent practice management guideline from EAST with the recommendation to discontinue cervical spine immobilization with a negative, high-quality C-spine CT scan result.[32]

The identification of a fracture within the cervical spine should prompt full-spine CT, because 10% to 40% of patients have another, noncontiguous vertebral injury.[33,34]

Management

Prehospital Care

Identification and treatment of all spinal injuries begin at the scene of the incident by experienced and well-trained

Table 4. Vasopressor Options for Hypotension

Drug	Dose (mcg/kg/min)	Receptor	Effect	Cautions
Dopamine	5–10 10-20	Pure β α = β	Increases heart rate and cardiac contractility Preserves increase in cardiac output, peripheral vasoconstriction	Causes mild vasodilation; low dose is not recommended; higher risk of arrhythmias
Norepinephrine	0.01-3	α > β	Increases cardiac output and causes vasoconstriction Agent of choice	
Phenylephrine	0.5–9	Pure α-1	Peripheral vasoconstriction, most useful in lesions below T4-6	May worsen bradycardia
Epinephrine	0.01–1	α = β	Increases cardiac output	Risk for arrhythmias
Dobutamine	2–20	Pure β	Increases HR and cardiac contractility	May cause mild hypotension
Vasopressin	0.03–0.04 units/min	V1	Peripheral vasoconstriction	Causes volume expansion and hyponatremia

emergency medical system personnel. It is estimated that 3% to 25% of all spinal cord injuries occur after the initial trauma and, despite an abundance of high-quality evidence, current recommendations for any patient with a suspected spinal cord injury resulting from blunt trauma are to immobilize the patient with a cervical collar and rigid backboard for transportation.[35] Spine immobilization should not be used for patients who have sustained penetrating trauma, because it is associated with a higher mortality rate.[36] This increase is thought to be due to the delay in resuscitation that occurs during immobilization.

Emergency Department Care

Upon arrival at the ED, the patient should be triaged and evaluated according to Advanced Trauma Life Support (ATLS) protocols. The primary survey — airway, breathing, and circulation — is paramount, as a significant number (20-57%) of SCI patients will have associated brain, chest, and/or extremity trauma.[37] The primary survey is also important because hypoxia and hypotension should be avoided in neurologically injured patients.

Respiratory Management

Airway compromise in SCI patients is often multifactorial, owing to profound neurologic dysfunction and high rates of concomitant injuries. Table 1 illustrates the normal muscles of respiration and their innervation. Due to diaphragmatic dysfunction, patients with high cervical lesions (above C5) have profound compromise of their respiratory muscles.[38] Those with complete lesions above C3 usually experience respiratory arrest within minutes after the initial injury.[39] Because of the high risk of respiratory failure and the need for mechanical ventilation, all patients with complete injuries at C5 or higher should be intubated as soon as possible.[40] Additionally, 51% of patients with high thoracic SCI injuries (T1–T6) and 28% of those with injuries involving T7 to T12 experience serious respiratory complications due to impairment of their respiratory mechanics.[41] The result of impaired diaphragm and intercostal muscle function is paradoxic abdominal breathing.

Respiratory complications are the leading cause of morbidity and mortality among patients with SCI[6] and are highly correlated with ASIA score. As cord edema spreads and secondary SCI occurs, most patients can lose as much as one ASIA level.[40] Therefore, it is recommended that patients displaying any respiratory distress, hypoxemia despite oxygenation, or hypercarbia should be intubated urgently rather than emergently.[39,42]

Aggressive management of any potentially compromised airway is recommended to avoid catastrophic airway loss. Traditionally, direct laryngoscopy with manual inline stabilization has been the standard of care and remains the recommendation of EAST.[43] Video laryngoscopy should be considered when stabilizing a patient with a difficult airway in cervical spine immobilization.[44,45] There are currently no formal recommendations on either induction agents or paralytic agents for rapid sequence intubation in SCI patients. Care should be taken to maintain adequate blood pressure during intubation attempts.

In addition to their immediate need for airway control, patients have severely altered breathing mechanics after SCI. The most common complications — atelectasis, pneumonia, and ventilatory failure — occur within the first five days.[46] Lesions between C5 and T11 will result in varying levels of respiratory dysfunction due to impaired inspiratory capacity, retained secretions, and autonomic dysfunction.[47] (*See Table 2.*)

Respiratory failure is caused by muscle fatigue, atelectasis, decreased surfactant production, and increased secretion production.[47] Paralysis of the intercostal muscles produces a paradoxic inward chest wall movement with respiration, leading to increased work of breathing. Furthermore, paralysis of the abdominal musculature impairs forced expiration and the patient's ability to cough and expel secretions. Pulmonary compliance decreases as secretions accumulate and atelectasis becomes more profound, leading to further increased work of breathing.

As spinal shock resolves and flaccid paralysis is replaced by spasticity, ventilatory mechanics tend to improve. The paradoxic inward chest contraction on inspiration is replaced by chest wall rigidity, and a significant increase in vital capacity is seen within 90 days after injury.

Circulation

SCI commonly is associated with systemic hypotension, most often due to neurogenic shock and/or hypovolemia. Hypotension, regardless of its cause, contributes to secondary SCI and can worsen prognosis.[48] After other causes of hypotension have been excluded (*see Table 3*), neurogenic shock becomes a serious concern in lesions above T4.[33] It is characteristically a distributive form of shock with warm extremities, hypotension, and bradycardia. Hypotension stems from sympathetic denervation and pooling of blood within the venous system. It can worsen as a result of unopposed vagal tone, bradycardia, and reduced myocardial contractility.[49] The severity of neurogenic shock generally correlates with level of injury, with higher levels associated with more severe symptoms.[50]

Neurogenic shock always should be treated first and foremost with fluid resuscitation.[33] Vasopressors should be used after volume resuscitation. The agent chosen depends on the patient's hemodynamic status and neurologic level of injury. Alpha-agonists cause vasoconstriction and increases in blood pressure (vasopressor action), while beta-agonists cause an increase in myocardial contractility and heart rate (inotropy and chronotropy).

Persistently hypotensive patients with an NLI above T6 require both chronotropic and inotropic agents[33] because the innervation of the heart exits the spinal cord from T1 to T4. An agent that increases myocardial contractility and heart rate and provides vasoconstriction (e.g., dopamine or norepinephrine) is a reasonable choice.[50] Lesions below the level of T4 to T6 generally cause hypotension through peripheral vasodilation. A pure alpha-1 agonist such as phenylephrine causes peripheral vasoconstriction and is an appropriate choice for patients with low thoracic and lumbar lesions but not those with cervical lesions.[50]

Bradycardia from neurogenic shock is very common in SCI patients. However, they also have a degree of autonomic dysfunction, so exogenous stimulation (tracheal suctioning and tracheal intubation) can induce bradycardia and, in some cases, cardiac arrest.[51] Atropine should be used in the acute treatment of symptomatic bradycardia. Beta-agonists can be used for longer-term treatment, with pacemaker insertion being required in as many as 17% of patients with cervical injuries.[51]

The American Association of Neurological Surgeons and the Congress of Neurological Surgeons recommend aggressive treatment of hypotension by maintaining a mean arterial pressure (MAP) at or above 85 to 90 mm Hg for the first seven days following injury to improve spinal cord perfusion.[33] There are no formal recommendations regarding the selection of vasoactive medications; however, norepinephrine, phenylephrine, and dopamine are reasonable first-line agents. Epinephrine should be used with caution because of its higher propensity to induce arrhythmias. Vasopressin (antidiuretic hormone [ADH]) causes water retention and hypervolemic hyponatremia, so its use should be considered carefully only when catecholamines are not sufficient. Dobutamine may be considered for its inotropic effects, but only after adequate volume resuscitation, because of its ability to cause mild vasodilation and hypotension.

Patients found to have a fracture dislocation in the cervical spine with neurologic deficit require immediate consultation with a spine surgeon for anatomic reduction and surgical management. In an awake patient with worsening neurologic status, facet dislocation, or bilateral locked facets, anatomic reduction should be performed as soon as possible. When closed reduction cannot be achieved under fluoroscopic guidance, further imaging is warranted to delineate the anatomy in preparation for open reduction. Any obtunded patient with a cervical fracture dislocation should undergo an MRI prior to attempted reduction.[52]

The timing of surgical decompression has been debated since the 1960s. As surgical techniques and hardware have advanced, there has been a shift from conservative to aggressive management. In 2012, Fehlings et al[53] published results from the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS), in which they investigated early (< 24 hours) versus late (> 24 hours) decompression. Their primary outcome was recovery of at least two AIS grades at six-month follow-up. The study showed a statistically significant difference between the two groups (19.8% early vs 8.8% late [odds ratio 2.8]) in regard to primary outcome.

The surgical management of patients who do not require decompression remains controversial. Surgical management is preferred to non-surgical, but the optimal timing and approach have not been defined. Consultation with a spinal surgeon is recommended.[54]

Additional Immediate Considerations

Venous thromboembolism (VTE) after SCI is a significant cause of morbidity and mortality. The incidence lies somewhere between 12% and 64%, with pulmonary embolism accounting for nearly 10% of deaths within the first year after injury.[55] Patients should have both mechanical and chemical VTE prophylaxis started upon admission and continued for three months.[56] Within 72 hours after admission, low-molecular-weight heparin should be administered instead of heparin for chemical prophylaxis because of its favorable half-life, relative reduction in bleeding complications, and more predictable dose effect.[57] Inferior vena cava filters are not recommended as routine prophylaxis and should be reserved for patients in whom chemical prophylaxis is contraindicated.[56]

Patients with SCI have a high risk of gastrointestinal bleeding for four weeks following injury. Injuries above the sympathetic outflow tract result in an imbalance of sympathetic and parasympathetic tone within the gastrointestinal tract. This results in increased secretion of gastric acid and delayed gastric emptying, leading ultimately to ulceration of the gastric and proximal small bowel mucosa. All patients with SCI should be given ulcer prophylaxis with either a proton pump inhibitor or

H2-receptor antagonists.[33]

Additional Aspects

Steroids

Modulation of the inflammatory cascade has long been a therapeutic goal. Early investigations in animal models showed promise for methylprednisolone in augmenting secondary SCI outcomes.[58] This prompted investigations by Bracken and colleagues and the creation of the National Acute Spinal Cord Injury Study (NASCIS).

NASCIS I was released in 1984 and, overall, was a negative study with no significant difference in the neurologic outcomes between the groups (high- vs. low-dose methylprednisolone).[59] It was determined later through animal models that the dosing was below any potential therapeutic threshold.

Consequently, NASCIS II was developed and released in 1990,[60] examining methylprednisolone vs. naloxone vs. placebo, with the primary outcomes of neurologic status at six weeks, six months, and one year. The study revealed no difference in motor score at any point, with a transient improvement in pinprick and light touch scores at six months, which were lost at one year. This would be considered a negative study; however, a post hoc subgroup analysis that examined steroid administration before and after eight hours showed a significant improvement (5 points) in motor score at six months and one year. Despite the suspect validity of this post hoc analysis, as well as the overall methodology of the study, steroids gained mainstream acceptance as therapy for SCI.

NASCIS III was released in 1997[61] and compared high-dose methylprednisolone duration (24 vs. 48 hours) with tirilazad (a medication that, at the time, was a novel treatment for stroke). This study had the same outcome measures as NASCIS II. Again, NASCIS III showed no improvement in neurologic outcomes between the groups. Post hoc analysis showed the same 5-point motor improvement in patients receiving steroids three to eight hours after injury without changes in sensory outcomes. In addition to providing no neurologic benefit, high-dose methylprednisolone therapy has been associated with numerous complications, including infection, respiratory compromise, gastrointestinal hemorrhage, and death.[62]

A Cochrane review published in 2012 continued to advocate the use of methylprednisolone in high doses and administered within eight hours of injury.[63] The current recommendation by the Joint Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons is not to use methylprednisolone in the treatment of SCI.[62]

Hypothermia

Mild to moderate hypothermia (32-35° C) has been a recent focus of research in the treatment of SCI. It has been well demonstrated that therapeutic hypothermia mitigates many of the mechanisms of secondary SCI, such as excitotoxicity, neuroinflammation, apoptosis, and free radical production.[64] A substantial number of studies in animal models support the beneficial neuroprotective effects of mild to moderate hypothermia therapy after SCI.[65] Limited human studies[66-69] have shown great promise but offer insufficient evidence to recommend for or against the routine use of therapeutic hypothermia.

Disposition

Patients who have been diagnosed with acute spinal cord injury potentially may do better at a facility equipped to deal with the injury and the resultant complications. These facilities include trauma centers and centers where neurosurgical or spinal orthopedic specialties are available. Patients with confirmed spinal cord injuries should be admitted to areas where neurologic checks can be performed as frequently as every hour during the first 24 hours after injury, which in most institutions would be an intensive care setting. Depending on institutional abilities, some patients may need to be admitted or transferred for further diagnostic testing, such as MRI. If there is a high suspicion of injury, transfer to a capable facility is the preferred choice. Patients who have a negative diagnostic evaluation and a normal neurologic examination can be discharged from the ED, with the understanding that they should have a plan for good follow-up or otherwise arrange for a repeat examination in a short time.

Summary

Spinal cord injury can be a debilitating and devastating consequence of a traumatic incident. The medical costs associated with its diagnosis and treatment are almost always overwhelming. Having a thorough understanding of anatomy will help the clinician understand the short- and long-term ramifications of SCI. Although the best option is prevention, when SCI does occur, immediate recognition and treatment can limit long-term sequelae. Every attempt should be made to limit hypotension, hypoglycemia, hypoxia, and hyperthermia in patients with SCI, as they are all associated with adverse outcomes. Finally, patients with high-grade SCI should be cared for in institutions with immediate access to either neurosurgical or spinal orthopedic specialists.

References

1. NSCISC National Spinal Cord Injury Statistical Center. 2014 Annual Report Complete Public Version. Available at: https://www.nscisc.uab.edu/annualstatreportsarchives.aspx. NSCISC Annual Statistical Report Complete Public Version.pdf. Accessed May 23, 2016.
2. Jain NB, Ayers GD, Peterson EN, et al. Traumatic spinal cord injury in the United States, 1993-2012. *JAMA* 2015;313:2236-2243.
3. Bracken MB, Freeman DH, Hellenbrand K. Incidence of acute traumatic hospitalized spinal cord injury in the United States, 1970-1977. *Am J Epidemiol* 1981;113:615-622.
4. National Spinal Cord Injury Statistical Center. Facts and Figures at a Glance. Birmingham, AL: University of Alabama at Birmingham, February 2016. Available at: https://www.nscisc.uab.edu/Public/Facts%202016.pdf). Accessed Oct. 5, 2016.
5. Ortman JM, Velkoff VA, Hogan H. An aging nation: The older population in the United States. *Current Population Reports* May 2014.
6. DeVivo MJ. Epidemiology of traumatic spinal cord injury: Trends and future implications. *Spinal Cord* 2012;50:365-372.
7. Demetriades D, Newton E. *Color Atlas of Emergency Trauma*, 2nd ed. Cambridge, UK; Cambridge University Press; 2011.
8. Allen BL Jr, Ferguson RL, Lehmann TR, et al. A mechanistic classification of closed, indirect fractures and dislocations of the lower cervical spine. *Spine* (Phila Pa 1976) 1982;7:1-27.

9. Maroon JC, Abla AA. Classification of acute spinal cord injury, neurological evaluation, and neurosurgical considerations. *Crit Care Clin* 1987;3:655-677.
10. Denis F. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. *Spine* (Phila Pa 1976) 1983;8:817-831.
11. Moore KL, Dalley AF, Agur AMR. *Clinically Oriented Anatomy,* 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2014.
12. Kwon BK. Tetzlaff W, Grauer JN, et al. Pathophysiology and pharmacologic treatment of acute spinal cord injury. *Spine J* 2004;4:451-464.
13. Oyinbo CA. Secondary injury mechanisms in traumatic spinal cord injury: A nugget of this multiply cascade. *Acta Neurobiol Exp* (Wars) 2011;71:281-299.
14. Dalbayrak S, Yaman O, Yılmaz T. Current and future surgery strategies for spinal cord injuries. *World J Orthop* 2015;6:34-41.
15. Dumont RJ, Okonkwo DO, Verma S, et al. Acute spinal cord injury. Part I: Pathophysiologic mechanisms. *Clin Neuropharmacol* 2001;24:254-264.
16. Boese CK, Oppermann J, Siewe J, et al. Spinal cord injury without radiologic abnormality in children: A systematic review and meta-analysis. *J Trauma Acute Care Surg* 2015;78:874-882.
17. Boese CK, Lechler P. Spinal cord injury without radiologic abnormalities in adults: A systematic review. *J Trauma Acute Care Surg* 2013;75:320-330.
18. American College of Surgeons. *ATLS: Advanced Trauma Life Support for Doctors*, 9th ed. Chicago; 2008.
19. Bonner S, Smith C. Initial management of acute spinal cord injury. *Contin Educ Anaesth Crit Care Pain* 2013;13:224-231.
20. Kirshblum SC, Burns SP, Biering-Sorensen F, et al. International standards for neurological classification of spinal cord injury (Revised 2011). *J Spinal Cord Med* 2011;34:535-546.
21. Hadley MN, Walters BC, Aarabi B, et al. Clinical assessment following acute cervical spinal cord injury. *Neurosurgery* 2013;72(3 suppl 2):40-53.
22. Cho TA. Spinal cord functional anatomy. *Continuum* (Minneap Minn) 2015;21: 13-35.
23. Ditunno JF, Little JW, Tessler A, Burns AS. Spinal shock revisited: A four-phase model. *Spinal Cord* 2004;42:383-395.
24. McKinley W, Santos K, Meade M, et al. Incidence and outcomes of spinal cord injury clinical syndromes. *J Spinal Cord Med* 2007;30:215-224.
25. Aarabi B, Hadley MN, Dhall SS, et al. Management of acute traumatic central cord syndrome (ATCCS). *Neurosurgery* 2013;72:195-204.
26. Foo D, Subrahmanyan TS, Rossier AB. Post-traumatic acute anterior spinal cord syndrome. *Paraplegia* 1981;19:201-205.
27. Ryken TC, Hadley MN, Walters BC, et al. Radiographic assessment. *Neurosurgery* 2013;72:54-72.
28. Stiell IG, Wells GA, Vandemheen KL, et al. The Canadian C-spine rule for radiography in alert and stable trauma patients. *JAMA* 2001;286:1841-1848.
29. 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: 94-99.
30. Como JJ, Diaz JJ, Dunham CM, et al. Practice management guidelines for identification of cervical spine injuries following trauma: Update from the Eastern Association for the Surgery of Trauma practice management guidelines committee. *J Trauma* 2009;67:651-659.
31. Walters BC, Hadley MN, Hurlbert RJ, et al. Guidelines for the management of acute cervical spine and spinal cord injuries: 2013 update. *Neurosurgery* 2013;60(suppl 1):82-91.
32. Patel MB, Humble SS, Cullinane DC, et al. Cervical spine collar clearance in the obtunded adult blunt trauma patient: A systematic review and practice management guideline from the Eastern Association for the Surgery of Trauma. *J Trauma Acute Care Surg* 2015;78:430-441.
33. Consortium for Spinal Cord Medicine. Early acute management in adults with spinal cord injury: A clinical practice guideline for health-care professionals. *J Spinal Cord Med* 2008;31:403-479.
34. Nelson DW, Martin MJ, Martin ND, et al. Evaluation of the risk of noncontiguous fractures of the spine in blunt trauma. *J Trauma Acute Care Surg* 2013;75:135-139.
35. Theodore N, Hadley MN, Aarabi B, et al. Prehospital cervical spinal immobilization after trauma. *Neurosurgery* 2013;72(suppl 2):22-34.
36. Haut ER, Kalish BT, Efron DT, et al. Spine immobilization in penetrating trauma: More harm than good? *J Trauma* 2010;68:115-121.
37. Sekhon LH, Fehlings MG. Epidemiology, demographics, and pathophysiology of acute spinal cord injury. *Spine* (Phila Pa 1976) 2001;26(24 suppl):S2-S12.
38. Terson de Paleville DGL, McKay WB, Folz RJ, et al. Respiratory motor control disrupted by spinal cord injury: Mechanisms, evaluation, and restoration. *Transl Stroke Res* 2011;2:463-473.
39. Stein DM, Pineda JA, Roddy V, et al. Emergency neurological life support: Traumatic spine injury. *Neurocrit Care* 2015:23(suppl 2):S155-164.
40. Como JJ, Sutton ER, McCunn M, et al. Characterizing the need for mechanical ventilation following cervical spinal cord injury with neurologic deficit. *J Trauma* 2005;59:912-916.
41. Cotton BA, Pryor JP, Chinwalla I, et al. Respiratory complications and mortality risk associated with thoracic spine injury. *J Trauma* 2005;59:1400-1409.
42. Hassid VJ, Schinco MA, Tepas JJ, et al. Definitive establishment of airway control is critical for optimal outcome in lower cervical spinal cord injury. *J Trauma* 2008;65:1328-1332.
43. Mayglothling J, Duane TM, Gibbs M, et al. Emergency tracheal intubation immediately following traumatic injury: An Eastern Association for the Surgery of Trauma practice management guideline. *J Trauma Acute Care Surg* 2012;73(5 suppl 4):S333-S340.
44. Mosier JM, Stolz U, Chiu S, et al. Difficult airway management in the emergency department: GlideScope videolaryngoscopy compared to direct laryngoscopy. *J Emerg Med* 2012;42:629-634.
45. Bathory I, Frascarolo P, Kern C, et al. Evaluation of the GlideScope for tracheal intubation in patients with cervical spine immobilisation by a semi-rigid collar. *Anaesthesia* 2009;64:1337-1341.
46. Jackson AB, Groomes TE. Incidence of respiratory complications following spinal cord injury. *Arch Phys Med Rehabil* 1994;75:270-275.
47. Berlly M, Shem K. Respiratory management during the first five days after spinal cord injury. *J Spinal Cord Med* 2007;30:309-318.
48. [No authors listed.] Blood pressure management after acute spinal cord injury. *Neurosurgery* 2002;50:S58-S62.
49. Weaver LC, Fleming JC, Mathias CJ, et al. Disordered cardiovascular control after spinal cord injury. *Handb Clin Neurol* 2012;109:213-233.
50. Ploumis A, Yadlapalli N, Fehlings MG, et al. A systematic review of the evidence supporting a role for vasopressor support in acute SCI. *Spinal Cord* 2010;48: 356-362.
51. Hector SM, Biering-Sørensen T, Krassioukov A, et al. Cardiac arrhythmias associated with spinal cord injury. *J Spinal Cord Med* 2013;36:591-599.
52. Gelb DE, Hadley MN, Aarabi B, et al. Initial closed reduction of cervical spinal fracture-dislocation injuries. *Neurosurgery* 2013;72:73-83.
53. Fehlings MG, Vaccaro A, Wilson JR, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: Results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). *PLoS One* 2012;7:e32037.
54. Gelb DE, Aarabi B, Dhall SS, et al. Treatment of subaxial cervical spinal injuries. *Neurosurgery* 2013;72:187-194.
55. Furlan JC, Fehlings MG. Role of screening tests for deep venous thrombosis in asymptomatic adults with acute spinal cord injury: an evidence-based analysis. *Spine* (Phila Pa 1976) 2007;32:1908-1916.

56. Dhall SS, Hadley MN, Aarabi B, et al. Deep venous thrombosis and thromboembolism in patients with cervical spinal cord injuries. *Neurosurgery* 2013;72:244-254.
57. Christie S, Thibault-Halman G, Casha S. Acute pharmacological DVT prophylaxis after spinal cord injury. *J Neurotrauma* 2011;28:1509-1514.
58. Braughler JM, Hall ED. Lactate and pyruvate metabolism in injured cat spinal cord before and after a single large intravenous dose of methylprednisolone. *J Neurosurg* 1983;59:256-261.
59. Bracken MB, Collins WF, Freeman DF, et al. Efficacy of methylprednisolone in acute spinal cord injury. *JAMA* 1984;251:45-52.
60. Bracken MB, Shepard MJ, Collins WF, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. *N Engl J Med* 1990;322:1405-1411.
61. Bracken MB, Shepard MJ, Holford TR, et al. Administration of methylprednisolone for 24 or 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. National Acute Spinal Cord Injury. *JAMA* 1997;277:1597-1604.
62. Hurlbert RJ, Hadley MN, Walters BC, et al. Pharmacological therapy for acute spinal cord injury. *Neurosurgery* 2013;72(suppl 2):93-105.
63. Bracken MB. Steroids for acute spinal cord injury. *Cochrane Database Syst Rev* 2012;1(1):CD001046.
64. Polderman KH. Mechanisms of action, physiological effects, and complications of hypothermia. *Crit Care Med* 2009;37(suppl):S186-S202.
65. Wang J, Pearse D. Therapeutic hypothermia in spinal cord injury: The status of its use and open questions. *Int J Mol Sci* 2015;16:16848-16879.
66. Levi AD, Green BA, Wang MY, et al. Clinical application of modest hypothermia after spinal cord injury. *J Neurotrauma* 2009;26:407-415.
67. Levi AD, Casella G, Green BA, et al. Clinical outcomes using modest intravascular hypothermia after acute cervical spinal cord injury. *Neurosurgery* 2010;66:670-677.
68. Dididze M, Green BA, Dietrich WD, et al. Systemic hypothermia in acute cervical spinal cord injury: A case-controlled study. *Spinal Cord* 2013;51:395-400.
69. Hansebout RR, Hansebout CR. Local cooling for traumatic spinal cord injury: Outcomes in 20 patients and review of the literature. *J Neurosurg Spine* 2014;20:550-561.

CME/CE Questions

1. What is the most common etiology of spinal cord injury?
   a. Sporting injury
   b. Iatrogenic injury
   c. Motor vehicle collision
   d. Malignancy
2. In the acute evaluation of a SCI patient, what is the first system that should be evaluated?
   a. Neurologic disability
   b. Circulatory dysfunction
   c. Electrolyte abnormalities
   d. Patency of airway
3. Which of the following is *not* a sign of spinal shock?
   a. Hypertension
   b. Hypotension
   c. Areflexia
   d. Flaccid paralysis
4. Which reflex is the first to return and typically marks the end of the period of spinal shock?
   a. Bulbocavernosus
   b. Patellar
   c. Babinski
   d. Biceps
5. Which of the following injuries is *not* consistent with a central cord injury pattern?
   a. Associated with a hyperextension injury
   b. Lower motor neurons affected greater than upper motor neurons
   c. Described as a "cape-like" distribution of paresthesias
   d. There is associated bladder dysfunction
6. Which of the following is the classic description of an anterior cord syndrome?
   a. Patients will have motor weakness/paralysis, loss of pain and temperature, and loss of voluntary bladder control with preservation of vibration and position sense.
   b. Patients will have motor weakness/paralysis, loss of pain and temperature, and loss of voluntary bladder control with disruption of vibration and position sense.
   c. Patients will have motor weakness/paralysis, loss of vibration and position sense.
   d. Patients will have normal motor exam, with loss of pain and temperature, and loss of voluntary bladder control with preservation of vibration and position sense.
7. In a patient with a presumed SCI, a normal neurologic exam and negative CT cervical spine, and who continues to have cervical spine tenderness, which is the appropriate next step?
   a. Discharge because of the low likelihood of significant cervical spine injury
   b. Admission for serial cervical spine examinations
   c. Discharge without cervical immobilization for outpatient MRI
   d. Obtain flexion and extension cervical spine radiographs
8. Which of the following is true about SCI and associated neurogenic shock?
   a. Initial treatment should be limited to vasopressors.
   b. It is classified as a hemorrhagic type of shock.
   c. It is synonymous with spinal shock.
   d. Patients with injuries above T6 will often require both chronotropic and inotropic support.
9. According to NASCIS concerning the use of steroids on SCI patients, all of the following are true *except*:
   a. NASCIS I showed no significant difference in neurologic outcomes with high-dose methylprednisolone.
   b. NASCIS II showed no difference in naloxone, methylprednisolone, or placebo in SCI patients.
   c. NASCIS II subgroup analysis resulted in significant changes in methylprednisolone administration.
   d. Methylprednisolone has very minimal side effects when given at high doses.
10. When considering intubation of an SCI patient, which of the following should be avoided?
    a. Hypothermia
    b. Hypertension
    c. Hypotension
    d. Hyperglycemia

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