Lesson 280: PreAnesthetic Assessment of the Patient With Neurotrauma

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DATE REVIEWED: DECEMBER 2008

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TIME TO COMPLETE ACTIVITY: 2 hours
RELEASE DATE: June, 2009
TERMINATION DATE: June 30, 2010

TARGET AUDIENCE: Anesthesiologists

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Needs statement

Patients who have a cervical spine injury (CSI) are challenging cases for anesthesiologists. Proper management of the airway, control of circulation, and immobilization of the cervical spine are critical for achieving a favorable outcome. Systemic manifestations of spinal cord injury and associated head injuries are but a few of the issues that may present in cases involving cervical spine trauma.
Learning Objectives

At the end of this activity, the participant should be able to:

1. Discuss the epidemiology of CSI.
2. Explain the pathophysiology of CSI.
3. Recognize the characteristic injuries associated with abnormal movements of the spinal column.
4. Describe the systemic manifestations of CSI.
5. Outline the diagnosis and treatment of the patient with a CSI.
6. Summarize areas of current research involving the treatment of CSI.
7. Apply appropriate preoperative testing and evaluation.
8. Develop an anesthetic plan for the patient with suspected or diagnosed CSI.
9. Recognize and manage the patient with a difficult airway and CSI.
10. Explain the effects of anesthetic agents on the spinal cord that are observed during intraoperative monitoring.

Case History

A 17-year-old boy was admitted to the emergency department after a motor vehicle accident. The paramedics reported that he was the restrained driver of a small car that had sustained heavy damage on the driver’s side. He had experienced no loss of consciousness; however, he described pain in his neck and left upper quadrant. At the scene of the accident, a rigid cervical collar had been placed; in addition, 2 large-bore IV cannulae were inserted.

In the emergency department, the patient was tachycardic, with a heart rate of 110 beats per minute. His systolic blood pressure remained at approximately 100 mm Hg despite fluid challenges. On plain radiographs of his neck, several areas were difficult to visualize, and further evaluation with computed tomography was required. An abdominal scan revealed a splenic laceration requiring surgery.

Anesthesiologists frequently become involved in managing trauma patients with cervical spine injuries (CSIs). The role of the anesthesiologist in the management of these patients—including assessment of the airway, breathing, and circulation—is crucial to the neurologic outcome. As an acute care provider, the anesthesiologist may encounter such patients at any time during the perioperative period—in the emergency department, the operating room, and the intensive care unit—and should therefore be knowledgeable about suspected cases of CSI and how to evaluate them. A familiarity with the pathophysiology of primary and secondary CSI, and the clinical manifestations of each, is key. Furthermore, an understanding of the risks and benefits of the different approaches to airway management is imperative in caring for patients with CSI.
Epidemiology

The incidence of CSI associated with blunt trauma is about 0.9% to 3%. Types of trauma cases include those caused by motor vehicle accidents (50%-75%), falls -(6%-10%), and recreational injuries (5%-15%; Figure 1).1,2

Approximately 20% of patients have more than one cervical spine fracture. Approximately 20% to 75% of cervical spine fractures are considered unstable, and 30% to 70% are associated with neurologic injuries. Because the prognosis for recovery from a complete cervical cord lesion is poor, emphasis must be placed on preventing the worsening of a neurologic injury after trauma has occurred.3

Pathogenesis and Pathophysiology

In cases of CSI caused by blunt trauma, injury occurs most commonly at the second cervical (C2) vertebra; injuries at this level account for 24% of all fractures. Thirty-nine percent of fractures occur at C6 and C7, with the vertebral body the most common anatomic site of fracture.1 Instability occurs when physiologic loading causes patterns of vertebral displacement that jeopardize the spinal cord or nerve roots. To maintain stability, one element of the injured column must be preserved. The anterior column contributes more than the posterior column to the stability of the spine in extension. During flexion, it is the posterior column that contributes more to stability. In hyperextension injuries, the anterior elements tend to be disrupted, whereas in hyperflexion injuries, the posterior elements are disrupted. Both columns may be disrupted with extreme flexion or extension, or if either compressive or rotational forces are added.1

A primary mechanical injury caused by compression, penetration, laceration, shear, or distraction forces results in immediate neural damage due to the avulsion and devitalization of tissues. Fracture and dislocation may lead to persistent cord compression and ischemia (Figure 2). Cord injury also may be the consequence of laceration, contusion, or concussion by bony fragments.1 A compilation of the characteristic injuries sustained with abnormal movements of the spinal column appears in the Table.

After a primary CSI, several mechanisms are responsible for secondary or progressive cord injury, including vascular compromise leading to reduced blood flow, loss of autoregulation, vasospasm, thrombosis, and hemorrhage. Electrolyte shifts, permeability
changes, loss of cellular membrane integrity, edema, and loss of energy metabolism all contribute to progressive cord injury after initial trauma. Biochemical changes—including the accumulation of neurotransmitters, release of arachidonic acid, production of free radicals and prostaglandin, and lipid peroxidation—cause axonal disruption and cell death. Glutamate released from damaged cells of the central nervous system is responsible for the excitotoxic component of secondary injury. Increased protease activity, loss of mitochondrial function, and increased oxidative stress as a result of overactivation of glutamate receptors initiate a cascade of events resulting in selective cell death and demyelination around the site of injury; as a result, the lesion increases in size and scarring develops.  

### Table: Characteristic Injuries Caused by Abnormal Movement of the Spinal Column

<table>
<thead>
<tr>
<th>Type of Injury</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperflexion</td>
<td>Subluxation or fracture–dislocation of a vertebral body with disruption of the posterior longitudinal ligament (eg, compression fracture, compression with burst fracture). Herniation of intervertebral disks common.</td>
</tr>
<tr>
<td>Hyperextension</td>
<td><strong>Most common in cervical injuries.</strong> Cause reduction of anterior–posterior diameter of the spinal canal with disruption of the anterior longitudinal ligament. Vertebral arteries may be damaged. Injuries associated with a normal-appearing radiograph are most likely of this type.</td>
</tr>
<tr>
<td>Compression</td>
<td>Caused by a significant &quot;axial load&quot; (eg, a fall onto the occiput); associated with compression or burst fracture of a vertebra and retropulsion of bone fragments.</td>
</tr>
<tr>
<td>Rotation</td>
<td><strong>Most often involves intervertebral disks.</strong> In combination with hyperflexion can result in locked facet joints. Associated with major neurologic injury requiring surgical reduction. Most often seen in C5–C7.</td>
</tr>
<tr>
<td>Combined</td>
<td>Rotation plus flexion/extension. <strong>Ligament tears commonly seen</strong> (eg, &quot;whiplash&quot; accident, or traumatic spondylolisthesis when face/chin hits steering wheel, most commonly dislocating C2 from C3).</td>
</tr>
</tbody>
</table>

Adapted from reference 2.

### Clinical Features and Manifestations

Primary and secondary injuries of the spinal cord occur after traumatic disruption of the cervical spine. A primary spinal cord injury can result from any of several mechanisms, including shear, compressive, ballistic, and distracting forces. Spinal cord blood flow is severely reduced within the first 30 to 60 minutes after injury as a consequence of hypertensive vasogenic edema (the result of an initial release of catecholamines). The loss of autoregulation leads to ischemia and tissue hypoxia. Compromised global perfusion resulting from systemic hypotension, and tissue hypoxemia resulting from hypoventilation associated with a head injury, exacerbate existing perfusion deficits.

Spinal shock develops after a complete lesion of the spinal cord. Symptoms include hypotension, loss of function of the bowel and bladder, loss of sensation and deep tendon reflexes, and total paralysis below the level of the lesion with flail limbs (Figure 3). Systemic vascular disturbances of blood flow after an acute spinal cord injury include reduced heart rate and mean arterial pressure, in addition to decreased peripheral vascular resistance as a result of interrupted sympathetic outflow below the level of injury. Respiratory insufficiency and pulmonary dysfunction are common after injuries to the cervical spinal cord. In severely injured patients, marked reductions in expected vital capacity and inspiratory capacity, as well as relative hypoxemia, exacerbate cord ischemia.
Diagnosis, Therapy, and Treatment

Although there is considerable variation in imaging of the patient at risk for CSI, most centers rely on multiple plain radiographs (at least 3 views) of the cervical spine supplemented by computed tomographic (CT) scans of areas that are difficult to visualize or in which injury is suspected. Obtundation, coexisting distracting (ie, painful) injuries, head injuries, and intoxication make it impossible to assess many patients clinically without imaging. If evidence of neurologic deficit suggests cervical injury despite a normal appearance on radiographs and CT scans, magnetic resonance imaging (MRI) may be useful.\(^1\)

The goals in treating spinal cord injuries are to protect the cord from secondary damage, maintain the alignment of bony structures, and stabilize the spinal column to allow rehabilitation of the patient. During the acute phase of injury, surgical indications include decompression with or without fusion in a patient with neurologic deterioration, reduction and stabilization when conservative management has failed, and surgical intervention for other life-threatening conditions.\(^4\)

Methylprednisolone was advocated some years ago for the treatment of acute primary and secondary injuries of the spinal cord.\(^5\) However, the current opinion among spine surgeons has been to move away from this therapy. A survey published in the *Canadian Journal of Neurological Sciences* in 2008 reported that 76% of spine surgeons do not use methylprednisolone for acute spinal cord injury, a reversal of the practice 5 years previously. In fact, one-third of physicians reported that they administered methylprednisolone only out of fear of litigation.\(^7\)
Practice guidelines and the role of methylprednisolone in the treatment of acute spinal cord injuries had been based on the National Acute Spinal Cord Injury Studies I, II, and III (conducted in 1984, 1990, and 1997, respectively). Now, however, evidence-based medicine suggests that although methylprednisolone results in neurologic improvement in certain types of acute spinal cord injury, its role in preventing secondary spinal cord injury remains unclear. In light of proven harmful side effects of high-dose steroids (including increased incidence of wound infection, pulmonary embolism, hyperglycemia, and gastrointestinal hemorrhaging), additional studies are needed to define the benefits and limitations of treating acute spinal cord injuries with steroids.

Attention has shifted to research focusing on other treatments. In a recent study by Jellish et al, a single intrathecal administration of magnesium sulfate was found to attenuate glutamate release and reduce neurologic injury after transient ischemia in animal models. Another promising area of research involves the potentially protective effects of intraperitoneal octreotide and melatonin in rats after spinal cord injury. Erol et al found that although octreotide had significant effects on edema and congestion following injury, melatonin was superior in preventing edema, congestion, axonal degeneration, and necrosis.

Yet another area of research involves the modulation of growth factors (eg, epidermal growth factor, transforming growth factor-β1 and -β2) to improve motor function after spinal cord injury and prevent neuronal death in animal models. Variables such as temporal expression and cellular distribution of growth factors are still under investigation, but current research is encouraging.

An important area of study pertains to optimizing the determinants of regeneration in the central nervous system, in the hope of repairing and regenerating the injured cord. Enhancing conduction in remaining axons, or mitigating damage to some extent, would lessen the degree of incapacity after an injury. One strategy under investigation in early and late phases of spinal cord injury in rats is to treat with cells differentiated from embryonic stem cells. The cells are delivered by direct injection to the injured area or by lumbar puncture with immune suppression to lessen the chance of rejection. Figure 4 depicts some of the key variables and strategies aimed at developing future curative treatments.

![Barriers to regeneration and common strategies of spinal cord repair](image-url)
**Anesthetic Management**

Successful surgical management can be achieved only after a comprehensive preoperative evaluation. The patient’s airway, breathing, and circulation must first be assessed and addressed. Resuscitation and stabilization, followed by prevention of secondary damage to the spinal cord via spinal immobilization and airway management, are priorities. CSI should be suspected in all injuries involving blunt trauma. Patients at increased risk include those with injuries above the clavicles and those with head injury and a Glasgow Coma Scale score of less than 9.3

In both complete and incomplete lesions of the spinal cord, manipulation can aggravate the injury and cause ascending deterioration. Therefore, the goal is to establish endotracheal intubation without causing further damage to the spinal cord. Perhaps the most important factor in determining the best technique for intubation is the urgency of the situation. The anesthesiologist must evaluate and assess the risk for further cord injury, taking into consideration the patient’s head and neck movement, degree of cooperation, and airway anatomy and trauma. The anesthesiologist must also consider his or her expertise with airway techniques. Collars, whether soft or rigid, do not effectively eliminate neck movement during intubation. Manual in-line stabilization is more effective in immobilizing the neck during intubation, but it may cause excessive distraction in C1-C2 fractures.3

Opinions differ on the optimal way to secure the airway in patients with CSI; some experts in neuroanesthesia advocate the use of a fiber-optic bronchoscope to facilitate endotracheal intubation, especially in elective situations. Currently, there are no data to suggest that better neurologic outcomes are achieved by this means. The technique can be more risky if attempted by inexperienced practitioners. Failed awake intubation has been identified as a cause of morbidity and mortality according to a recent analysis of difficult airways claims in the American Society of Anesthesiologists Closed Claims Project.1

The use of direct laryngoscopy after induction of anesthesia is considered an acceptable option by most experts in trauma, anesthesia, and neurosurgery. Manual in-line stabilization with an assistant holding both sides of the neck and mastoid processes prevents neck movement and allows mouth opening with minimal risk. The anesthesiologist should have alternative devices available—including conventional and intubating supraglottic airways, lighted wands and stylets, esophageal–tracheal airways, video laryngoscopes, and cricothyrotomy devices—in the event of a failed or difficult intubation. A bougie is a less expensive and perhaps more readily available option for airway management, especially in the patient with a difficult airway (Figure 5).3

After induction and endotracheal intubation of the patient, anesthesia is maintained by continuing adequate perfusion to prevent further damage. With a loss of autoregulation, spinal cord perfusion depends on systemic perfusion. Hypotension may cause further secondary injury; hypertension may lead to hemorrhage and edema. Normal systemic perfusion and gas exchange are the objectives as spinal cord blood flow cannot be monitored. Drugs should be titrated slowly because of cardiovascular lability. Bradycardia, which frequently develops, should be treated with atropine. The administration of vasopressors may be necessary to maintain circulation. Normocapnia or mild hypocapnia is recommended; the efficacy of hyperventilation in cases of cord injury has not been substantiated.3

The dorsal column of the spinal cord of patients with vertebral column injuries but no neurologic deficits is often monitored by using somatosensory evoked potentials (SSEPs). The interpretation of intraoperative findings is difficult in patients with incomplete or abnormal preoperative SSEPs. The
SSEP signal is abolished by high doses of inhaled anesthetics. The recommended anesthetic regimen for intraoperative SSEP monitoring is a continuous IV opioid infusion supplemented with a low-dose inhaled anesthetic. Motor evoked potentials with either electrical or magnetic stimulation of the motor cortex complement the SSEPs and allow the anterior and posterior columns to be monitored simultaneously.

Emergence from anesthesia should be rapid to facilitate an early postoperative neurologic assessment. If reintubation is likely to be difficult because of instability, cervical fixation, or the general anatomic features, extubation should be delayed until the patient is fully awake and meets standard extubation criteria.

Management of the Case Presented

The patient was transported to the operating room for splenectomy and exploratory laparotomy. Standard monitors and a radial arterial catheter were placed, in addition to the existing large-bore IV cannulae. The patient was preoxygenated; manual in-line stabilization was maintained during rapid-sequence IV induction and intubation. The anesthetics midazolam and desflurane were titrated according to hemodynamic parameters. After consultation with the surgeon, muscle relaxation was maintained with rocuronium. A mean arterial pressure of 60 mm Hg was maintained throughout the case with intermittent use of a vasopressor (phenylephrine) after normovolemia had been ascertained. At the conclusion of surgery, the patient emerged from anesthesia in the operating room and was extubated while fully awake. His neurologic assessment was unchanged and revealed no deficits. He was transported to the intensive care unit for further postoperative monitoring.
Summary

Patients presenting with cervical spine trauma can be especially challenging for the anesthesiologist. Concurrent head and systemic injuries often complicate the care administered to these patients. The anesthesiologist must be familiar with the mechanisms of primary and secondary cervical spine trauma to select appropriate techniques. In addition, skill at different techniques in airway management is necessary, given that clinical situations may involve elective or emergency procedures. Furthermore, to minimize secondary neurologic injury and protect the cord, the anesthesiologist must understand the effects of anesthetic techniques on the spinal cord physiology that are observed during intraoperative monitoring. Finally, anesthesiologists should remain abreast of scientific advancements and evolving research on the treatment of acute spinal cord injuries because they may be asked to provide anesthesia in cases that involve innovative procedures.

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REFERENCES


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**Post-test**

1. The majority of cervical spine injuries (CSIs) associated with trauma are caused by:
   a. motor vehicle accidents
   b. falls
   c. sports injuries
   d. diving accidents

2. At which spinal level do injuries to cervical vertebrae most commonly occur?
   a. C2
   b. C3
   c. C6
   d. C7

3. Mechanisms of secondary or progressive spinal cord damage include:
   a. loss of autoregulation
   b. vasospasm
   c. electrolyte shifts
   d. all of the above

4. Within how many minutes after a spinal cord injury will spinal cord blood flow be severely reduced because of initial cate-cholamine release and subsequent hypertensive vasogenic edema?
   a. 5-15
   b. 15-30
   c. 30-60
   d. 60-90

5. Disruption of the posterior longitudinal ligament is most commonly seen in injuries related to:
   a. hyperflexion
   b. hyperextension
   c. compression
   d. rotation
6. Which of the following is a goal when a spinal cord injury is treated?
   a. Protecting the cord from further damage
   b. Maintaining the alignment of bony structures to allow maximum recovery
   c. Stabilizing the spinal column for rehabilitation
   d. All of the above

7. All of the following are potentially harmful side effects of methylprednisolone, except:
   a. wound infection
   b. pulmonary embolism
   c. gastrointestinal hemorrhage
   d. pulmonary fibrosis

8. Which of the following is true regarding assessment of the patient at risk for CSI before intubation?
   a. Movement of the head and neck is not an important consideration.
   b. The patient does not need to be able to cooperate to facilitate fiber-optic intubation.
   c. The anesthesiologist need not consider his or her own expertise in techniques of intubation.
   d. The anatomy of the airway and degree of trauma to the airway are important considerations before intubation is attempted.

9. Which statement is true regarding intubation of the patient with CSI?
   a. Direct laryngoscopy with manual in-line stabilization is a proven technique to improve neurologic outcomes.
   b. Some neuroanesthesiologists advocate the use of a fiber-optic bronchoscope to facilitate intubation, especially in elective procedures.
   c. Regardless of the practitioner’s level of expertise in fiber-optic bronchoscopy, fiber-optic intubation is superior to direct laryngoscopy with manual in-line stabilization.
   d. Direct laryngoscopy is not an acceptable means of intubation.

10. Somatosensory evoked potentials:
    a. are not affected by volatile anesthetics
    b. that are incomplete or abnormal preoperatively are nevertheless useful in monitoring patients for spinal cord ischemia
    c. are used to monitor the anterior column of the spinal cord
    d. in combination with motor evoked potentials allow the anterior and posterior columns of the spinal cord to be monitored simultaneously