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Abstract

Acute spinal cord injury is a devastating event associated with substantial morbidity worldwide. The pathophysiology of spinal cord injury involves the initial mechanical trauma and the subsequent inflammatory response, which may worsen the severity of neurologic dysfunction. Interventions have been studied to reduce the extent of primary injury to the spinal cord through preventive measures and to mitigate secondary insult through early specialized care. Management, therefore, is multifocal, interdisciplinary, and begins immediately at the time of injury. It includes the trauma triage, acute management of the circulatory and respiratory systems, and definitive treatment, mainly with surgical decompression and stabilization.

Keywords
► airway management
► bradycardia
► blood pressure targets
► intraoperative neuromonitoring
► neurogenic shock
► respiratory arrest
► resuscitation
► spinal cord injury
► spinal shock
► surgical decompression

Introduction

Acute spinal cord injury (SCI) is potentially irreversible and can be devastating, affecting between 250,000 and 500,000 people every year worldwide.¹ Severe (i.e., complete) SCI presents as a total loss of motor and sensory function and temporary depression of reflexes, reflecting an absence of communication between the nerves below the lesion and the brain. In an incomplete injury, some signals persist, which manifests in varying degrees of movement and sensation below the level of injury and rarely in extinction of reflexes.² The pathophysiology of injury occurs in a two-step process consisting of the primary mechanical insult and secondary pathobiological consequences.³

A greater understanding of these mechanisms and awareness of their potentially devastating consequences have led to primary prevention efforts and a surge of research in mitigating secondary injury. Protective measures such as improved safety equipment are reducing the effects of car accidents, sports-related injury, and falls.⁴⁻⁷ Early delivery of specialized medical and surgical care is now routine in high-resource settings.¹ This care comprises immediate spinal immobilization with simultaneous consideration of airway, breathing and circulatory demands, hemodynamic augmentation, and expedient neurologic and radiographic evaluation for early surgical intervention.⁸⁻¹⁰ These advances have likely played a role in both improved early survival and a decrease in incidence of severe (complete) SCI relative to incomplete SCI.¹⁰⁻¹¹ Developed countries such as the United States, Finland, and Australia are seeing greater percentages of incomplete quadriplegia from high cervical SCIs than in the past.¹²⁻¹⁴ This trend is thought to be attributed to advancements in early resuscitation and rapid airway management for an increasingly older population experiencing falls.¹¹⁻¹⁵
A lack of longitudinal data collected in low- and middle-income countries makes international trends in incidence and prevalence of SCI difficult to estimate and predict.\textsuperscript{15,16} However, if early survival rates climb globally, we can expect to see a growing population of high spinal cord injuries with concomitant ventilator dependency in extended care settings.\textsuperscript{11} Despite this increase in prehospital and hospital survival, there have been no meaningful reductions in long-term mortality rates since the 1980s, and the gap in life expectancy between the general population and individuals with SCI is increasing.\textsuperscript{13} Fortunately, the current focus of research efforts and care delivery now centers on preventing neural damage and promoting axonal regrowth through molecular agents and early, aggressive physical rehabilitation.

In this two-part review, we first provide a brief background on SCI and its sequelae, including the pathophysiology and timeline of injury, and then discuss the latest developments in initial management and anesthetic considerations during surgery. In the second part, we discuss the medical management of SCI in the intensive care unit, focusing on evidence-based current practices. We conclude by reviewing the current data on neurologic and functional recovery and promising neuroprotective approaches and neuroregenerative therapies.

Pathophysiology

The mechanisms causing neurologic dysfunction in acute SCI involve a temporal sequence of pathobiological events (Fig. 1).\textsuperscript{3,17} Primary injury to the cord by acute compression, contusion (most common), distraction, laceration, rotation, or severing results in immediate neurovasculature damage, for example, through shearing of axons or ischemia from blood vessel compression.\textsuperscript{19} The extent of primary injury depends on the degree of the initial insult and the underlying condition of the cord and nerve roots (i.e., preexisting degenerative disease). In most cases, functional deficits are disproportionate to the initial anatomical damage, owing to the deleterious phases of injury and progressive neuronal deterioration.\textsuperscript{1} Surgical and medical interventions during the acute and intermediate phases of SCI have the greatest promise for halting and potentially partially reversing the damage caused by secondary injury.\textsuperscript{19}

Initial Assessment

According to the Advanced Trauma Life Support guidelines, initial management of a patient with potential SCI includes early transfer to a specialized center and rapid assessment of injuries using the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach.\textsuperscript{20} Patients must be assessed in the field and transported using full spinal immobilization of the cervical, thoracic, and lumbar regions until each region is cleared or definitively stabilized. “Spinal precautions” must be taken and are intended to limit further mechanical injury to the highly vulnerable cord.\textsuperscript{21} These include placement of an adequately fitting rigid cervical collar, use of a backboard,
and log rolling patients every 4 hours. If there is concern for intracranial injury with elevated intracranial pressure (ICP), the bed or stretcher should be tilted in the reverse Trendelenburg position to elevate the head.

Airway, Breathing: The highest priority is to ensure an unobstructed airway and adequate oxygenation and ventilation. Unlike current guidelines for advanced cardiac life support, airway management in the trauma patient remains the first essential step. Respiratory compromise in traumatic SCI can occur due to associated facial, neck, or chest injuries. Complete SCI above the C3 level prompts apneic respiratory arrest. Cervical or thoracic cord injury with phrenic or intercostal nerve paralysis leads to hypoventilation and hypercapnic–hypoxic respiratory insufficiency. A definitive airway with positive-pressure ventilation should be provided as needed. The standard of care is rapid-sequence intubation (RSI) with in-line spinal immobilization. In some patients, direct laryngoscopy can be used successfully without manipulating the neck. However, video laryngoscopy obtains better views of the glottis in less time than standard laryngoscopy when intubation is performed in a neutral neck position unless active bleeding or copious secretions compromise image clarity. A hyperangulated video laryngoscope or optical stylet may provide better glottic visualization in cervical SCI patients with additional risk factors for difficult intubation, such as obesity and restricted mouth opening.

Circulation: Simultaneously, the hemodynamic status of the patient should be assessed. Occult hemodynamic instability may be unmasked by induction agents and positive-pressure ventilation if not yet apparent. Cervical and thoracic SCI can interrupt intermediolateral cell column outflow with undeterred parasympathetic activity. Peripheral vascular tone, cardiac contractility, and heart rate may abruptly diminish, resulting in circulatory collapse that can develop immediately and may last for weeks. In polytrauma patients, circulatory shock from hypovolemia and cardiogenic causes must be considered first before the symptoms of profound hypotension and bradycardia are attributed to neurogenic shock. Immediate management of neurogenic shock involves fluid resuscitation to restore intravascular volume followed by vasopressors, if necessary, to avoid hypotension. If an endotracheal tube is not immediately warranted, resuscitation prior to intubation may help blunt the effects of RSI. Class III evidence supports maintaining a mean arterial pressure (MAP) of 85 to 90 mm Hg for a minimum of 5 to 7 days, as discussed later in Part II.

Disability (neurologic status): The next step after airway, breathing, and circulation concerns are addressed is a neurologic assessment to help determine the extent of injury. In general, impairment of motor and sensory function occurs in a distribution below the level of injury. For example, injury to the cervical cord can lead to tetraplegia, whereas injury to the thoracic or lumbar spine would result in lower extremity paraplegia. Additionally, cervical injuries may affect the diaphragm and respiratory function. However, injury to either the upper or lower cord can result in bowel and bladder dysfunction. The neurologic manifestations of SCI can be assessed using the American Spinal Injury Association (ASIA) Impairment Scale (AIS) consisting of a dermatomal-based sensory examination, myotomal-based motor examination, and a rectal examination (Fig. 2). Severity and level of injury are assigned based on the findings of these examinations. The level of neurologic injury is defined as the lowest functioning nerve root with intact sensation and grade 3 or greater motor function. (See Fig. 2 for a description of motor grades.) Severity is classified as complete or incomplete: from ASIA A, representing complete SCI with no motor and sensory function below the level of injury, to ASIA E, corresponding to normal sensory and motor function (but other neurologic phenomena, such as abnormal reflexes, may be present).

While immediate neurologic assessment using the AIS provides standardized, detailed information that guides radiographic assessment and treatment, determining whether injuries are complete or incomplete often requires resolution of spinal shock. "Spinal shock" is the term describing the immediate depression of all fundamental functions of

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**Fig. 2** International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) Worksheet from the American Spinal Injury Association (ASIA) and the International Spinal Cord Society (ISCoS). (©2019 American Spinal Injury Association. Reprinted with permission.)
The anesthesiologist’s main task is to decompress the cord and to realign and stabilize the spine. Early relief from compressive forces reduces ischemia and alleviates trauma from bone fractures and disc fragments. This limits the effects of secondary injury and optimizes the environment for neurologic recovery. The surgical team may place an alert and oriented patient with a cervical fracture dislocation injury in traction either instead of or prior to surgical intervention.

Operative Management of Neurologic Injury

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Central respiratory depression from benzodiazepines can intensify respiratory muscle weakness, leading to alveolar hypoventilation. Furthermore, benzodiazepines are associated with delirium and cognitive impairment in older individuals due to increased sensitivity and decreased metabolism and are best avoided in elderly SCI patients. Preoxygenation is paramount and can be continued through apneic oxygenation after induction in patients at risk for aspiration.

Hypotension must also be averted and insertion of an arterial catheter prior to induction is recommended. While the reverse Trendelenburg position is best for lung expansion, to reduce aspiration risk, and for traumatic brain injury patients with elevated ICP, this position may worsen hypotension in patients with high SCI. Guidelines suggest that MAP be maintained within 85 to 90 mm Hg throughout the case. However, this is not supported by class I or II evidence. MAP exceeding 90 mm Hg increases the risk of bleeding and obscures visualization of the operative field due to vascular congestion, whereas MAP less than 85 mm Hg is believed to worsen secondary spinal cord injury.

Table 1: Suggested anesthetic guidelines for the management of surgery for acute spinal cord injury

<table>
<thead>
<tr>
<th>Preoperative preparation</th>
<th>Expediuous chart review, including medical history, circumstance of injury, hospital course, and images of the injury</th>
<th>Directed physical examination and detailed airway assessment</th>
<th>Difficult airway equipment readily available</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premedication</td>
<td>Early preoxygenation</td>
<td>Limit anxiolytic premedication</td>
<td>Provide preinduction anticholinergic medication in high SCI</td>
</tr>
<tr>
<td></td>
<td>Limit anxiolytic premedication</td>
<td>Preinduction arterial catheterization</td>
<td>Preinduction arterial catheterization</td>
</tr>
<tr>
<td>Induction</td>
<td>Careful titration of induction agents with prophylactic fluid/vasopressors</td>
<td>Succinylcholine is contraindicated &gt;48 h postinjury</td>
<td></td>
</tr>
<tr>
<td>Airway</td>
<td>Video/direct laryngoscopy and endotracheal intubation with MILS in emergent scenarios or an uncooperative patient</td>
<td>Awake fiberoptic bronchoscope intubation with topicalization of the airway can be considered in cooperative patients</td>
<td></td>
</tr>
<tr>
<td>Maintenance</td>
<td>MAP of 85–90 mm Hg with fluids and vasopressors</td>
<td>IONM with TcMEP, SSEP, and EMG using a tailored anesthetic technique</td>
<td></td>
</tr>
<tr>
<td>Emergence</td>
<td>Wane sedation for neurologic assessment</td>
<td>Leave ETT in situ for complete high cervical SCI</td>
<td></td>
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<tr>
<td></td>
<td>Extubate with consideration of patient and surgical factors (length, positioning, volume administered, future interventions necessary)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: EMG, electromyography; ETT, endotracheal tube; IONM, intraoperative neuromonitoring; MAP, mean arterial pressure; MILS, manual in-line stabilization; SCI, spinal cord injury; SSEP, somatosensory evoked potentials; TcMEP, transcranial motor evoked potentials.

Central venous catheterization may be indicated if injuries to extremities preclude access or for the administration of vasoactive agents.

For patients with cervical and high thoracic SCI, an airway management plan must involve maintenance of spinal immobility. Techniques for mask ventilation and instrumentation of the airway may need to accommodate cervical spine immobilization devices such as a halo or rigid collar. Manual in-line stabilization (MILS) of the cervical spine provided by an assistant standing at the head of the bed or reaching across the chest can be performed with the anterior portion of a rigid collar removed and is associated with less movement of the spine during intubation than collar immobilization alone. A properly performed jaw thrust maneuver, achieved by translating the condyles of the mandible out of the temporomandibular joint and then pulling the mandible forward, is the safest method to open the airway of a patient with cervical SCI and can be performed in patients with a rigid collar without moving the head or neck. Awake fiberoptic bronchoscopy is commonly used to intubate stable, cooperative patients prior to cervical spine surgery. Alternative devices such as intubating laryngeal mask airway, rigid optical stylet, and hyperangulated video laryngoscopy with MILS are associated with decreased first attempt failure rate and less cervical spine motion than direct Macintosh laryngoscopy with MILS and are preferred in emergency situations or for patients who would not tolerate an awake procedure due to their mental state. Cricoid pressure during RSI is best avoided in patients with lower cervical SCI. Instead, a gentle backward-upward-rightward pressure maneuver can be used to facilitate laryngoscopic view if necessary. Airway manipulation and tracheal suctioning in patients with complete high thoracic and cervical SCI can trigger severe bradycardia and sinus arrest due to unopposed efferent parasympathetic activity. Pretreatment with anticholinergic medications...
prior to induction is advised in these high-risk patients. Resting bradycardia associated with acute upper SCI can be treated with small doses of glycopyrrolate, or with ephedrine for simultaneous MAP augmentation. Atropine should be readily available throughout the surgical procedure for severe bradycardia. Cutaneous pacing with a placement of anterolateral pacer pads prior to surgical preparation and draping may be necessary in patients with high SCI.

Routinely used induction agents include propofol, ketamine, etomidate, and sodium pentothal. Agents that decrease systemic vascular resistance, such as propofol, should be used with caution in SCI due to the risk of profound hypotension from sympathetic denervation and decreased venous return to the heart. Concurrent intravenous fluid bolus administration for preload augmentation and vasopressor infusion during induction may be required as prophylaxis for hypotension. In patients with lower SCI, pure vasoconstrictors such as phenylephrine can be safely used alongside fluid to mitigate vasodilation from the peripheral sympathetic cause of the injury further compounded by anesthetic agents. However, with injuries disrupting the sympathetic cardiac-accelerator fibers (T1 to T4), vasopressors with inotropic properties are needed, such as norepinephrine, dopamine, or epinephrine. Depolarizing neuromuscular blocking agents, such as succinylcholine, should be avoided in patients with SCI after 24 hours postinjury due to an exaggerated and potentially life-threatening intracellular potassium efflux. This is because acetylcholine receptors (AChRs) are upregulated and occupy all of the muscle membrane after denervation injury, not only the neuromuscular junction. Furthermore, immature AChRs have a longer open channel time when depolarized and a greater potential for sustaining a more prolonged potassium leak. Choice of maintenance anesthetic agents depends on multiple factors such as the duration of the planned procedure, level of hemodynamic stability, patient comorbidities, and intraoperative neuromonitoring (IONM) techniques (see the IONM section below).

While excessive intraoperative fluid resuscitation to meet MAP goals should be avoided to prevent pulmonary and spinal cord edema, precise clinical endpoints to guide therapy have not been established. Euvolemic is a good initial goal, and fluid losses, both urine and blood, should be calculated frequently. Lactate levels, acid-base status, and trends in pulse pressure variation can further guide fluid management. Choice of intravenous fluid is often dictated by regional and institutional availability. While there is insufficient evidence that colloid solutions are superior to balanced electrolyte solutions, colloids may be useful to minimize total volume of administered fluid while improving intravascular retention and microcirculatory hemodynamics with minimal capillary leakage.

Fluids are typically the primary volume replaced until transfusion thresholds are crossed. This could be ongoing large-volume surgical bleeding or a specific Hb or hematocrit value. There is a lack of data on values below which blood should be transfused in acute SCI. However, spinal cord tissue offers little anaerobic reserve, and the injured cord has a very low neurologic threshold to hypoxia/ischemia in the face of anemia. It, therefore, can be argued that the risk of anemia in acute SCI weighs more heavily than the risks associated with blood transfusion, and a liberal (Hb of 10 g/dL) transfusion approach may be preferred to a restrictive one.

**Intraoperative neuromonitoring:** Patients with SCI benefit from IONM, particularly those with incomplete cord injury, which helps ensure that surgery does not exacerbate motor or sensory deficits. Ideally, IONM should be initiated immediately after the airway is secured and prior to surgical positioning on the operating room table. Level I evidence supports the use of intraoperative somatosensory evoked potential (SSEP) and transcranial motor evoked potential (TcMEP) monitoring. Additionally, electromyography can be used to detect and prevent nerve root injury from decompression or pedicle screw fixation.

One way in which anesthetic agents induce unconsciousness is by depressing synaptic activity. While anesthetic effects vary by synapse location, the greatest effects occur on TcMEP muscle responses and cortical potentials. Inhalational agents including isoflurane, sevoflurane, and desflurane have greater effects than intravenous anesthetics. Inhalational anesthetics cause a dose-related decrease in amplitude and an increase in the latency of cortical SSEPs. In healthy patients, adequate SSEPs can usually be recorded at 0.5 to 1 MAC of volatile agents. However, for patients with SCI and neurologic impairment, even low levels of inhalation agents may abolish potentials and make monitoring impossible. TcMEP responses are even more sensitive to the effects of volatile anesthetic than SSEPs. Total intravenous anesthesia is encouraged when TcMEP responses are monitored during surgical treatment for SCI. Whichever the choice of anesthetic, its level must remain constant during critical monitoring periods to avoid iatrogenic changes from baseline and misleading interpretation of responses.

Regardless of IONM signal changes, a neurologic assessment may be requested by the surgical team after emergence from anesthesia but before exiting the operating room. Bispectral index (BIS) and other processed electroencephalographic monitoring are helpful in determining the depth of anesthesia to facilitate safe emergence and a timely spinal assessment prior to extubation. Patients with a complete, high cervical SCI will require assisted ventilation and therefore should be left intubated if a tracheostomy is not already in place. Multiple factors influence the decision to extubate patients with incomplete and lower SCI, including the length of surgery, positioning during surgery, volume of blood and fluid administered, and associated injuries that may require additional interventions in the near future.

**Conclusion**

Patients with suspected SCI should be brought to facilities that specialize in their care to help decrease mortality and improve prognosis. Initial management involves early identification of injuries, aggressive resuscitation with attention to associated conditions, and definitive surgical treatment. The anesthesiologist plays a vital role in preventing further injury to the vulnerable spinal cord during surgical decompression.
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Conflict of Interest
None declared.

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