

# Anesthesia for Acute Spinal Cord Injury



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## KEYWORDS

- Spinal anatomy • Acute spinal cord injury • Spinal shock • ASIA impairment scale
- Autonomic dysreflexia

## KEY POINTS

- Spinal cord blood supply: The main blood supply to the anterior two-thirds of the spinal cord is via the single anterior spinal artery originating from the vertebral arteries, whereas the posterior one-third is supplied by the two posterior spinal arteries branching off the posterior inferior cerebellar artery.
- Spinal shock: Spinal shock refers to a clinical syndrome characterized by the loss of reflex, loss of motor and sensory function below the level of the injury, loss of autonomic tone leading to hypotension, hypothermia, urinary retention, and fecal incontinence.
- American Spinal Injury Association (ASIA) Impairment scale: The ASIA Impairment Scale (AIS) is a score assigning the functional level of the injury and the severity of sensory and motor impairment based on five degrees of severity (A to E).
- Autonomic dysreflexia: Autonomic dysreflexia is a potentially life-threatening emergency characterized by hypertension, bradycardia, headache, flushed face, nasal stuffiness, nausea, and sweating above the level of injury that affects patients with spinal cord injuries typically at the T6 level or higher.

## INTRODUCTION

Acute spinal cord injury (SCI) has multiple causes and can lead to significant morbidity and persistent, neurologic deficits, carrying significant long-term economic implications.<sup>1</sup> The annual incidence of acute SCI worldwide is about 15 to 40 cases per million and is predominantly seen in young adults. Trauma is an important cause of acute SCI; other causes of acute spinal cord compression are associated with oncologic conditions as a result of pathologic fractures or direct compression on the spine from mass effect. The most common causes of traumatic SCI (TSCI) include motor vehicle

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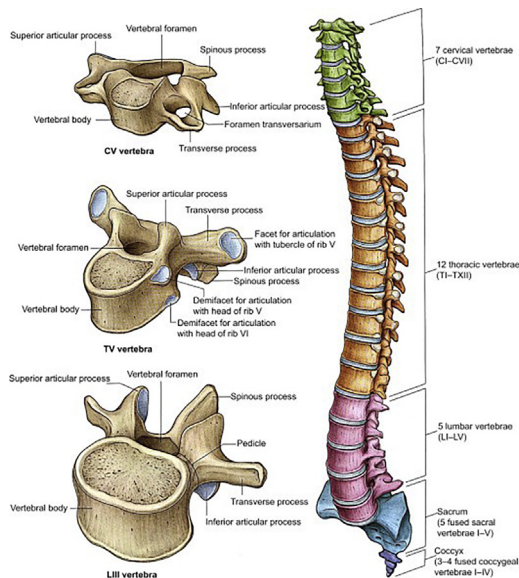
collisions, including whiplash injuries (48%), falls (16%), violent crime including gunshot wounds (12%), sports accidents (10%), and other causes (14%).<sup>2</sup> Nontraumatic acute SCIs can occur because of epidural hematomas, abscesses, degenerative diseases, or tumors causing spinal cord instability and compromised spinal cord perfusion. In this article, we discuss the spinal anatomy, blood supply of the spinal cord, pathogenesis of SCI, and expected complications of acute SCI. The discussion focuses on the peri-operative anesthetic management of these patients in the acute setting.

## A BRIEF OVERVIEW OF SPINAL ANATOMY AND FUNCTION

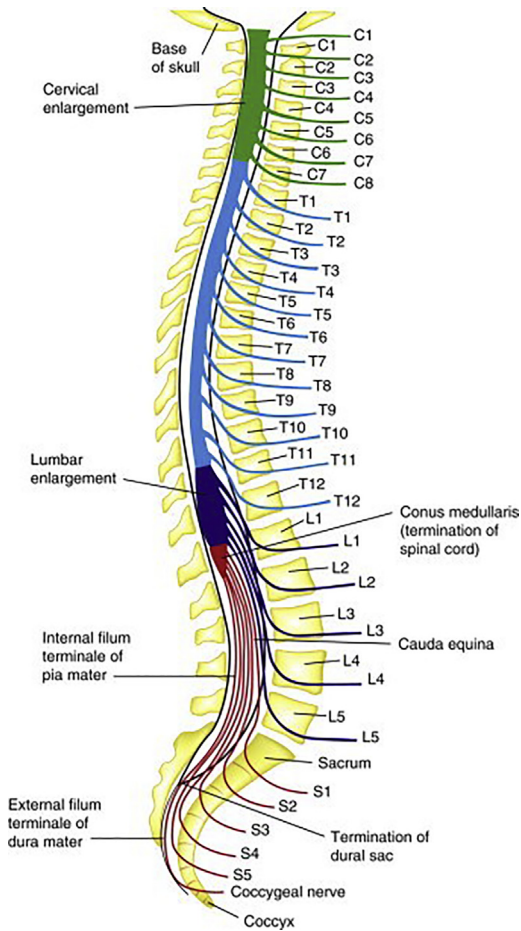
Longitudinally, the spinal cord extends from the base of the skull to the first lumbar vertebral body and continues as cauda equine at or below the second lumbar vertebra. The vertebral column is longer than the spinal cord and hence there is a difference in the segmental levels. The cord segments consist of cervical, thoracic, lumbar, and sacral areas.

Key anatomic structures of the cervical spinal cord region include C1 (atlas), which supports the atlantooccipital junction, and the section C3–C5, which forms the phrenic nerve and innervates the diaphragm. Important aspects of the thoracic spinal cord region include the intercostal nerves that arise from the thoracic spinal cord and innervate the accessory muscles of respiration. Importantly, the thoracic section of the spinal cord also provides sympathetic innervation to the heart and abdominal organs. Key aspects of the lumbosacral spinal cord region are the role in motor and sensory innervation of the lower extremity. Lastly, sacral nerves also provide parasympathetic innervation to the pelvis and abdominal organs (Figs. 1 and 2).

The cross-sectional anatomy of the spinal cord is comprised of gray matter in the center, surrounded by white matter tracts. The principal white matter tracts include: (1) dorsal columns that carry sensory information pertaining to joint position (proprioception) and vibration, which ascend upwards and cross in the medulla to the contralateral



**Fig. 1.** Vertebral column. (From Drake R, Vogl A, Mitchell AWM. Back. In: Drake R, Vogl W, Mitchell AWM, et al, editors. Gray's Atlas of Anatomy, Second Ed. Philadelphia: Elsevier; 2020, P. 22; with permission.)



**Fig. 2.** Spinal cord and cauda equina. (From Joshua M. Rosenow. *Anatomy of the Nervous system*. In: Krames ES, Peckham PH, Rezaei AR, Editors. *Neuromodulation: Comprehensive Textbook of Principles, Technologies, and Therapies*, Second Edition. London: Elsevier; 2018. P. 25-29; with permission. (Figure 7 in original).)

cerebral hemisphere; (2) anterolateral spinothalamic tracts that are ascending pathways carrying sensations of pain, temperature, and touch; and (3) corticospinal tracts that are descending pathways for motor neurons. The fibers of the corticospinal tracts cross cranially at the level of cervicomedullary junction (pyramids). The sympathetic fibers do not have a well-defined tract and lie at the spinal levels between T1 and L3. Noncranial parasympathetic cell bodies originate in the sacral spinal cord at S2-S4.

## OVERVIEW OF BLOOD SUPPLY TO THE SPINAL CORD

The chief blood supply of the spinal cord comes from a single anterior spinal artery and two posterior spinal arteries. The anterior spinal artery supplies the anterior two-thirds of the cord, and the two posterior spinal arteries supply the posterior one-third (dorsal columns). Both of these arteries arise from the vertebral arteries at the base of the skull. An important radicular branch arising from the aorta artery of Adamkiewicz enters the spinal cord usually between T9 and T12.

Spinal cord autoregulation is not robust and is more pressure dependent than cerebral autoregulation. In contrast to cerebral tissues, oxygenation of the spinal cord is more sensitive to hypotension and decreases as mean arterial pressure (MAP) decreases less than 60 mm Hg.<sup>3</sup>

## MECHANISMS OF SPINAL CORD INJURY

SCIs are often associated with vertebral bone fractures, ligament tears, and/or associated head injury, or as part of a polytrauma scenario. There is often a primary injury that has occurred at the time of initial insult, including complete or partial transection, penetrating injury, and/or contusion to the spinal cord itself. This situation is subsequently exacerbated by a secondary insult caused by ischemia, edema, hypotension, hypoxia, excitotoxicity, apoptosis, or further movement of the spinal cord/vertebral column. The phenomenon of secondary injury is sometimes clinically manifest by neurologic deterioration over the first 8 to 12 hours in patients who initially present with an incomplete cord syndrome.<sup>1</sup> Systemic hypotension is a hallmark physiologic response immediately following SCI.

## SIGNS AND SYMPTOMS OF ACUTE SPINAL CORD INJURY

Pain at the site of injury is one of the most common presenting clinical signs of acute SCI. There may be associated injuries, such as fractures, hemothorax, intra-abdominal bleeding, or head injury with associated altered consciousness, which may impair neurologic assessment. There may also be associated acute sensory or motor changes according to the American Spinal Injury Association (ASIA) Impairment Scale (AIS).<sup>1</sup>

### *American Spinal Injury Association Impairment Scale*

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Grade A = Complete. No sensory or motor function is preserved in the sacral segments S4-S5.<sup>4</sup>

Grade B = Sensory Incomplete. Sensory but no motor function is preserved below the neurologic level and includes the sacral segments S4-S5 (Light Touch [tests posterior column] or Pin Prick [tests spinothalamic tract] at S4-S5 or Deep Anal Pressure), and no motor function is preserved more than three levels below the motor level on either side of the body.

Grade C = Motor Incomplete. Motor function is preserved at the most caudal sacral segments for voluntary anal contraction or the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments [S4-S5] by Light Touch [tests posterior column], Pin Prick [tests spinothalamic tract], or Deep Anal Pressure), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body. (This includes key or nonkey muscle functions to determine motor incomplete status.) For AIS C, less than half of key muscle functions below the single neurological level of injury have a muscle grade  $\geq 3$ .

Grade D = Motor Incomplete. Motor incomplete status as defined previously, with at least half (half or more) of key muscle functions below the single NLI having a muscle grade  $\geq 3$ .

Grade E = Normal. If sensation and motor function as tested with the International Standards for Neurologic Classification of Spinal Cord Injury are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.

For individuals to receive a grade of C or D (ie, motor incomplete status), they must have either voluntary anal sphincter contraction or sacral sensory sparing with sparing of motor function more than three levels below the motor level for that side of the body.

Depicted in **Fig. 3** is the dermatomal distribution of different levels of SCI, and the chart used to determine the level of injury and the severity of impairment.

## INITIAL EVALUATION AND TREATMENT OF SPINAL CORD INJURY

Along with the presenting signs and symptoms mentioned previously, imaging can aid in diagnosing associated injuries, such as ligament and/bone fractures. Plain radiographs in the anteroposterior, lateral, and odontoid (in suspected cervical spine injury) views provide information on spinal cord alignment, bony fractures, and other possible displacements of anatomic structures. Computed tomography (CT) has been suggested to be more cost-effective in trauma patients, because often these patients receive thoracic and abdominal CT scans to look for other injuries. CT imaging is particularly helpful for assessment of bone injury. MRI of the spinal cord provides accurate assessment of the extent of injury. The chief advantage of MRI is that it provides a detailed image of the spinal cord and spinal ligaments, intervertebral disks, and paraspinal soft tissues that is superior to CT and is more sensitive for detecting epidural hematomas or masses.<sup>5</sup>

## PRINCIPLES OF MANAGEMENT

The initial assessment of a patient suspected to have acute SCI should always prioritize airway, breathing, and circulatory support. These patients often require transfer to a tertiary care center because of the complexity of injuries.

### *Patient Monitoring*


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Large-bore intravenous (IV) lines and/or central venous access may be required for adequate resuscitation. Care must be taken to minimize neck movement when placing central venous access. Ultrasound guidance may help in accurate placement of central lines. Arterial catheter placement aids in monitoring MAPs and drawing frequent blood gas samples to assess adequacy of resuscitation. A Foley catheter is required to monitor urine output and should be placed as early as possible. Patients with traumatic cervical and high thoracic SCI are critically ill and require intensive care unit (ICU) admission, along with continuous monitoring of vitals, and mechanical ventilation. Often, these patients are admitted with “spinal shock” or neurogenic shock. This is a form of distributive shock characterized by hypotension with bradycardia, because of disruption of autonomic sympathetic fibers, causing decreased systemic vascular resistance and lack of compensatory tachycardia. This type of shock may be compounded with hemorrhagic shock because of trauma-induced blood loss. In the resuscitation of patients with SCI, it is critical to maintain adequate perfusion to the spinal cord.

### *Airway Management*

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Cervical and high thoracic SCI compromises the primary muscles of respiration, and these patients should be immediately intubated and placed on mechanical ventilator support. Extreme caution must be taken to minimize any movement of the head and neck during laryngoscopy and intubation, to prevent further injury to the spinal cord. Some of the techniques that are used to achieve this include manual in-line axial stabilization and placement of an immobilizing collar on the neck (C-collar). Similar caution must be exercised in the operating room during transfer of patients from



**INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY (ISNCSCI)**

Patient Name \_\_\_\_\_ Date/Time of Exam \_\_\_\_\_

Examiner Name \_\_\_\_\_ Signature \_\_\_\_\_

**RIGHT**

**MOTOR KEY MUSCLES**

**UER** (Upper Extremity Right)

Elbow flexors C5

Wrist extensors C6

Elbow extensors C7

Finger flexors C8

Finger abductors (little finger) T1

**LER** (Lower Extremity Right)

Hip flexors L2

Knee extensors L3

Ankle dorsiflexors L4

Long toe extensors L5

Ankle plantar flexors S1

(VAC) Voluntary Anal Contraction (Yes/No)

**RIGHT TOTALS (MAXIMUM)**

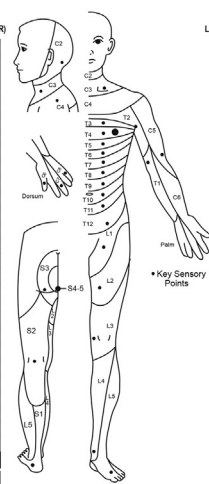
MOTOR SCORES: UER (25) + UEL (25) = UEMS TOTAL (50)

LER (25) + LEL (25) = LEMS TOTAL (50)

NEUROLOGICAL LEVELS: 1. SENSORY (R/L), 2. MOTOR (R/L)

**SENSORY KEY SENSORY POINTS**

Light Touch (LTR) Pin Prick (PPR)



Key Sensory Points

**SENSORY SUBSCORES**

LTR (56) + LTL (56) = LT TOTAL (112)

PPR (56) + PPL (56) = PP TOTAL (112)

4. COMPLETE OR INCOMPLETE? (Injury with absent motor OR sensory function in S4-5)

5. ASIA IMPAIRMENT SCALE (AIS)

**SENSORY KEY SENSORY POINTS**

Light Touch (LTR) Pin Prick (PPL)

**LEFT**

**MOTOR KEY MUSCLES**

**UEL** (Upper Extremity Left)

Elbow flexors C5

Wrist extensors C6

Elbow extensors C7

Finger flexors C8

Finger abductors (little finger) T1

**MOTOR (SCORING ON REVERSE SIDE)**

0 = Total paralysis

1 = Palpable or visible contraction

2 = Active movement, gravity eliminated

3 = Active movement, against gravity

4 = Active movement, against some resistance

5 = Active movement, against full resistance

NT = Not testable

0' = 1', 2', 3', 4', 5', NT' = Non-SCI condition present

**SENSORY (SCORING ON REVERSE SIDE)**

0 = Absent

1 = Altered

2 = Normal

NT = Not testable

0', 1', NT' = Non-SCI condition present

**LEFT TOTALS (MAXIMUM)**

MOTOR SCORES: UEL (25) + UEM (25) = UEMS TOTAL (50)

LER (25) + LEL (25) = LEMS TOTAL (50)

NEUROLOGICAL LEVELS: 1. SENSORY (R/L), 2. MOTOR (R/L)

**MOTOR SCORES** UER (25) + UEL (25) = UEMS TOTAL (50) LER (25) + LEL (25) = LEMS TOTAL (50)

**SENSORY SCORES** LTR (56) + LTL (56) = LT TOTAL (112) PPR (56) + PPL (56) = PP TOTAL (112)

**Muscle Function Grading**

- 0 = Total paralysis
  - 1 = Palpable or visible contraction
  - 2 = Active movement, full range of motion (ROM) with gravity eliminated
  - 3 = Active movement, full ROM against gravity
  - 4 = Active movement, full ROM against gravity and moderate resistance in a muscle specific position
  - 5 = (Normal) active movement, full ROM against gravity and full resistance in a functional motion expected from an otherwise unpaired person
- NT = Not testable (i.e. due to immobilization, severe pain such that the patient cannot be graded, amputation of limb, or contracture of > 50% of the normal ROM)
- 0', 1', 2', 3', 4', 5', NT' = Non-SCI condition present \*

**Sensory Grading**

- 0 = Absent
  - 1 = Altered, either decreased/impaired sensation or hypersensitivity
  - 2 = Normal
  - NT = Not testable
  - 0', 1', NT' = Non-SCI condition present \*
- \*Note: Abnormal motor and sensory scores should be tagged with a "\*" to indicate an impairment due to a non-SCI condition. The non-SCI condition should be explained in the comments box together with information about how the score is related for classification purposes (at least normal / not for classification).

**When to Test Non-Key Muscles:**

In a patient with an apparent AIS B classification, non-key muscle functions more than 3 levels below the motor level on each side should be tested to most accurately classify the injury (differentiate between AIS B and C).

Movement	Root level
Shoulder: Flexion, extension, abduction, adduction, internal and external rotation	C5
Elbow: Supination	C5
Hip: Pronation	C6
Wrist: Flexion	C6
Finger: Flexion at proximal joint, extension	C7
Thumb: Flexion, extension and abduction in plane of thumb	C7
Finger: Flexion at MCP joint	C8
Thumb: Opposition, adduction and abduction perpendicular to palm	C8
Finger: Abduction of the index finger	T1
Hip: Adduction	L2
Hip: External rotation	L3
Hip: Extension, abduction, internal rotation	L3
Knee: Flexion	L4
Ankle: Inversion and eversion	L4
Toe: MP and IP extension	L5
Hallux and Toe: DIP and PIP flexion and abduction	L5
Hallux: Adduction	S1

**ASIA Impairment Scale (AIS)**

- A = Complete.** No sensory or motor function is preserved in the sacral segments S4-5.
  - B = Sensory incomplete.** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-5 (light touch or pin prick at S4-5 or deep anal pressure) AND no motor function is preserved more than three levels below the motor level on either side of the body.
  - C = Motor incomplete.** Motor function is preserved at the most caudal sacral segments for voluntary anal contraction (VAC) OR the patient meets the criteria for sensory incomplete status (sensory function preserved at the most caudal sacral segments S4-5 by LTR, PPR or DAP), and has some sparing of motor function more than three levels below the ipsilateral motor level on either side of the body. (This includes key or non-key muscle functions to determine motor incomplete status.) For AIS C – less than half of key muscle functions below the single NLI have a muscle grade ≥ 3.
  - D = Motor incomplete.** Motor incomplete status as defined above, with at least half (half or more) of key muscle functions below the single NLI having a muscle grade ≥ 3.
  - E = Normal.** If sensation and motor function as tested with the ISNCSCI are graded as normal in all segments, and the patient had prior deficits, then the AIS grade is E. Someone without an initial SCI does not receive an AIS grade.
- Using ND:** To document the sensory, motor and NLI levels, the ASIA Impairment Scale grade, and/or the zone of partial preservation (ZPP) when they are unable to be determined based on the examination results.



**Steps in Classification**

The following order is recommended for determining the classification of individuals with SCI.

1. Determine sensory levels for right and left sides. The sensory level is the most caudal, intact dermatome for both pin prick and light touch sensation.
  2. Determine motor levels for right and left sides. Defined by the lowest key muscle function that has a grade of at least 3 (on supine testing), providing the key muscle functions represented by segments above that level are judged to be intact (graded as a 5). Note: in regions where there is no myotome to test, the motor level is presumed to be the same as the sensory level, if testable motor function above that level is also normal.
  3. Determine the neurological level of injury (NLI). This refers to the most caudal segment of the cord with intact sensation and antigravity (3 or more) muscle function strength, provided that there is normal (intact) sensory and motor function rostrally respectively. The NLI is the most cephalad of the sensory and motor levels determined in steps 1 and 2.
  4. Determine whether the injury is Complete or Incomplete. (i.e. absence or presence of sacral sparing) If voluntary anal contraction = No AND any S4-5 sensory scores = 0 AND deep anal pressure = No, then injury is Complete. Otherwise, injury is Incomplete.
  5. Determine ASIA Impairment Scale (AIS) Grade. Is injury Complete? If YES, AIS=A
- NO ↓
- Is injury Motor Complete? If YES, AIS=B
- NO ↓ (No voluntary anal contraction OR motor function more than three levels below the motor level on a given side, if the patient has sensory incomplete classification)
- NO ↓ YES ↓
- AIS=C AIS=D

Are at least half (half or more) of the key muscles below the neurological level of injury graded 3 or better?

- If sensation and motor function is normal in all segments, AIS=E
- Note: AIS E is used in follow-up testing when an individual with a documented SCI has recovered normal function. If at initial testing no deficits are found, the individual is neurologically intact and the ASIA Impairment Scale does not apply.
6. Determine the zone of partial preservation (ZPP). The ZPP is used only in injuries with absent motor (no VAC) OR sensory function (no DAP, no LT and no PP sensation) in the lowest sacral segments S4-5, and refers to those dermatomes and myotomes caudal to the sensory and motor levels that remain partially preserved. With sacral sparing of sensory function, the sensory ZPP is not applicable and therefore "NA" is recorded in the block of the worksheet. Accordingly, if VAC is present, the motor ZPP is not applicable and is noted as "NA".

Fig. 3. Dermatomal distribution of different levels of spinal cord injury. (© 2020 American Spinal Injury Association. Reprinted with permission.)

stretcher to operating room table and/or ICU bed. These precautions must be exercised even if there is no obvious visible cervical spine injury, such as in a trauma patient. Rapid sequence induction and intubation is often used as the method of choice in emergent intubations, because of concerns with the possibility of aspiration. If time permits, the authors recommend flexible fiberoptic intubation or use of a videolaryngoscope in the anesthetized and paralyzed patient, to minimize neck movement during intubation, as opposed to traditional laryngoscopy. However, disagreements still exist on the optimal approach to airway management.

### ***Fluid Management and Circulatory Support***

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Hypotension is an important cause of secondary SCI, and can result from bleeding caused by associated injuries and spinal shock caused by lack of sympathetic tone. The latter can occur because of disruption of sympathetic chain, leading to loss of sympathetic tone and pooling of blood in the lower extremities. Prevention of hypotension (systolic blood pressure <90 mm Hg) is an essential component of the early management of victims of SCI. Although it is well-documented that early aggressive management in an ICU setting involving respiratory and cardiovascular support has been accompanied by considerable improvement in vital and functional outcomes in patients with SCI, the goals of hemodynamic resuscitation have not been investigated in a systematic manner.

Recently, the Consortium for Spinal Cord Medicine released a guideline on the acute management of SCI in adults.<sup>6</sup> These guidelines include standard volume resuscitation to euvolemia and early use of vasopressor agents for the treatment of neurogenic shock (SCI above the level of T6) for the first 72 hours. Resuscitation is performed with crystalloids and/or blood transfusion as indicated.

The Association of Neurologic Surgeons released SCI guidelines in 2013 recommending artificial elevation of an MAP between 85 and 90 mm Hg for the first 7 days following SCI.<sup>7-10</sup> MAP is the average arterial pressure throughout one cardiac cycle, systole, and diastole, and is calculated using the formula:

$$\text{MAP} = \text{Diastolic Pressure} + 1/3 (\text{Pulse Pressure}).$$

However, this recommendation is only based on level III evidence from case series, nonexperimental, and noncomparative studies. There is, in theory, a strong rationale for blood pressure manipulation to prevent secondary spinal cord insult. A series of animal-based investigations performed in the 1970s and 1980s suggested that systemic hypotension after SCI was associated with reduced spinal cord perfusion and worsened neurologic outcomes.<sup>11,12</sup> The concept of blood pressure augmentation in neurologic resuscitation assumes that the spinal cord blood flow is pressure passive when there is loss of autoregulation. However, not only is the extent of impairment of spinal cord autoregulation unknown, but potent vasoconstrictors could impair spinal cord blood flow by limiting collateral flow through intercostal anastomosis when increased pressure occurs at the expense of excessive vasoconstriction. The inability to measure or monitor spinal cord blood flow while manipulating blood pressure with potent vasoconstrictors raises concerns regarding the beneficial effects of blood pressure augmentation.

There is substantial debate regarding the optimal vasopressor to induce blood pressure augmentation. Although norepinephrine might have direct toxic effect on myocytes by inducing apoptosis,<sup>13</sup> phenylephrine use might be limited by the occurrence of reflex bradycardia. Treatment of refractory bradycardia is achieved using temporary pacing pads or IV injection of atropine. A recent retrospective cohort



study evaluated the short-term effects of vasopressor use on patients with acute SCI.<sup>14</sup> Patients were treated by maintaining an MAP greater than 90 mm Hg ( $n = 131$ ) and the outcome of interest was complications associated with vasopressors. The authors reported an overall vasopressor-related complication rate of 74% in this patient population with the most common adverse event being cardiac dysrhythmia. Other complications included ST-segment elevation, cardiac troponin elevation, and skin necrosis. A similar study by Martin and colleagues<sup>15</sup> demonstrated that episodes of hypotension and the need for vasopressors did not affect the change in the ASIA motor score during acute hospitalization, regardless of an MAP goal set-point. With the caveat of lacking long-term follow-up, they concluded that their arbitrarily elevated MAP goals may not be efficacious.

### ***Role of Glucocorticoids***

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Two blinded, randomized controlled trials have studied the efficacy of glucocorticoid therapy in patients with acute TSCI. The National Acute Spinal Cord Injury Study II compared methylprednisolone, 30 mg/kg IV, followed by 5.4 mg/kg per hour over 23 more hours, naloxone, and placebo in 427 patients with acute TSCI.<sup>16</sup> At 1 year, there was no significant difference in neurologic function among treatment groups. However, within the subset of patients treated within 8 hours, those who received methylprednisolone had a modest improvement in motor recovery compared with those who received placebo. Wound infections, however, were somewhat more common in patients who received methylprednisolone.

National Acute Spinal Cord Injury Study III compared three treatment groups: (1) methylprednisolone administered for 48 hours, (2) methylprednisolone administered for 24 hours, and (3) tirilazad mesylate (a potent lipid peroxidation inhibitor) administered for 48 hours in patients with acute complete or incomplete TSCI.<sup>17</sup> All 499 patients received an initial IV bolus of 30 mg/kg methylprednisolone and were treated within 8 hours of TSCI. For patients treated within 3 hours, there was no difference in outcomes among treatment groups at 1 year. For patients treated between 3 and 8 hours, 48 hours of methylprednisolone was associated with a greater motor but not functional recovery compared with other treatments. Patients who received the longer duration infusion of methylprednisolone had more severe sepsis and severe pneumonia compared with the shorter duration of infusion; mortality was similar in all treatment groups.<sup>18</sup>

More recently, in 2013, based on the available evidence, the American Association of Neurologic Surgeons and Congress of Neurologic Surgeons stated that the use of glucocorticoids in acute SCI is not recommended.<sup>8</sup>

### ***Prevention of Complications from Critical Illness***

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Concomitant interventions are directed at prevention of potential complications, such as ventilator-associated pneumonia, deep vein thrombosis prevention, pressure ulcers, and gastric stress ulcers. Importantly, attention should be paid to nutritional support with insertion of feeding tube if the patient is incapable of oral intake, and pain control with appropriate evaluation and judicious opioid usage.

## **ANESTHETIC CONSIDERATIONS FOR EMERGENT SPINAL CORD SURGERY**

Patients with acute SCI may present to the operating room for decompression and fixation, in an attempt to stabilize the spinal cord. These patients may arrive on an emergent basis, with a C-collar. All C-spine precautions mentioned previously should be undertaken during laryngoscopy, intubation, positioning, and transport of the patient.



In addition, these patients may have associated orthopedic or soft tissue injuries, which may complicate line placement and positioning. They may also have associated head injuries with increased intracranial pressure, and this has to be kept in mind during anesthetic management. These patients are usually positioned prone for surgical access; hence intubation and lines may have to be performed on a stretcher/hospital bed. Pretreatment with glycopyrrolate is recommended in patients with cervical cord injuries to prevent severe bradycardia, which is associated with intubation in these patients. A rapid sequence induction and intubation with videolaryngoscope is usually preferred to safely secure the airway, if the patient is not already intubated.

In addition to standard American Society of Anesthesiology monitors, which must be used at all times, large-bore venous access and arterial catheter are required for intraoperative fluid resuscitation and monitoring. Most of the surgeries involving cervical and thoracic spinal cord use extensive neuromonitoring, to aid in stabilizing the spinal cord without exacerbating injury. Commonly used modalities include somatosensory evoked potentials and motor evoked potentials (MEPs), which test the integrity of the dorsal columns and motor pathways respectively. Inhaled anesthetic agents, such as sevoflurane, have a dose-dependent depressant effect on somatosensory evoked potentials and MEPs, and are usually avoided.

Intubation with a short-acting or no muscle relaxant technique is preferred to facilitate MEP monitoring. Rocuronium is usually reversible with neostigmine/glycopyrrolate if neuromonitoring will be initiated only after turning prone and final positioning. However, a desire to initiate neuromonitoring before turning the patient prone requires awake intubation, use of succinylcholine, rocuronium/sugammadex, or high-dose remifentanyl. The authors recommend maintenance with total IV anesthesia using a balanced combination of propofol infusion and an opioid, such as fentanyl, to facilitate monitoring, and provide anesthesia and analgesia. Vasopressor infusions, such as phenylephrine or norepinephrine, may be required to maintain MAPs, to aid perfusion of the spinal cord, as previously discussed.

Decision to extubate at the end of the surgery depends on multiple factors, such as respiratory muscle involvement in the injury, anticipation of spinal cord swelling during the immediate postsurgical period, associated injuries, and need for multiple subsequent surgeries. Patients with high cervical SCI are usually intubated in the postoperative period with mechanical ventilatory support. These patients may require a tracheostomy for long-term care.

## **LONG-TERM COMPLICATIONS**

### ***Autonomic Hyperreflexia***

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This phenomenon is seen in SCIs above the level of T6, and is usually seen within the first year of injury, in about 20% to 70% of patients.<sup>19,20</sup> It is characterized by exaggerated and uncoordinated sympathetic responses to noxious stimuli below the level of injury, leading to vasoconstriction and severe hypertension. An insufficient compensatory parasympathetic response occurs because of the SCI, leading to bradycardia and vasodilation above the level of lesion. Inciting stimuli include bladder or bowel distention, attempts to catheterize the bladder, pressure ulcers, and other similar stimuli. Clinical features include bradycardia, flushing, headache, sweating, and increased blood pressure. In severe cases, hypertensive crisis can occur, leading to intracranial hemorrhage, seizures, and cardiac arrest because of severe bradycardia. Anticipation, early recognition, and early treatment is essential for successful management of this medical emergency. Hypertensive crisis can occur in the operating room because these patients frequently present for procedures below the level of lesion.

An inadequate depth of anesthesia for surgical stimulation can trigger intraoperative hypertensive crisis. Treatment involves deepening the anesthetic and lowering the blood pressure with rapid-onset medications, such as nicardipine. An arterial catheter may be required for continuous intraoperative blood pressure monitoring.

### ***Coronary Artery Disease***

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Studies suggest that the prevalence of coronary artery disease is 3 to 10 times higher in patients with SCI than it is in the general population. Coronary artery disease risk factors, such as adverse lipid profile (low levels of high-density lipoproteins, elevated low-density lipoprotein cholesterol) and abnormal glucose metabolism (impaired glucose tolerance, insulin resistance, and diabetes), are more prevalent in patients with chronic SCI than in the able-bodied population.<sup>21</sup>

### ***Chronic Respiratory Insufficiency and Tracheostomy Dependence***

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Patients with cervical spinal and high thoracic cord injuries have significant long-term respiratory muscle involvement. Impaired cough and inability to clear lung secretions, along with chronic long-term ventilator care, places them at high risk of acquiring pneumonia. Aggressive chest physical therapy and improving respiratory muscle strength is vital in preventing this condition.

### ***Chronic Urinary Tract Infections***

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Patients with TSCI are prone to frequent urinary tract infections because of chronic indwelling catheters. The urinary tract is the most frequent source of septicemia in patients with SCI and has a high mortality rate (15%).<sup>22</sup>

### ***Musculoskeletal Contractures***

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Musculoskeletal contractures can result from chronic immobility and shortening and reorganization of the collagen fibers. Patients with SCI are also prone to bone fractures because of weakened muscles and ligaments. This may prove challenging to place IV lines or to position the patient intraoperatively.

### ***Pressure Ulcers***

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Pressure ulcers can occur because of chronic immobility and tissue damage on bony prominences.

### ***Chronic Pain***

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Chronic neuropathic pain is often seen in these patients. The underlying mechanism is poorly understood, and medical treatment involves a combination of antidepressants, gabapentin, and intrathecal morphine/baclofen. Patients with a history of SCI and a combination of one or more long-term complications may present to the operating room for procedures, such as debridement of wound ulcers, suprapubic catheter placement, removal of kidney stones, or any acute abdominal condition, such as appendicitis. As always, the principles of anesthetic management should be guided by airway, breathing, and circulatory support. If the patient has a long-term tracheostomy, appropriate suctioning may be required to clear secretions. If the patient arrives with an uncuffed tracheostomy tube, it may need to be changed to a cuffed tracheostomy tube to facilitate mechanical ventilation in the operating room.

Adequate depth of anesthesia and analgesia is required to prevent autonomic dysfunction from occurring intraoperatively. General anesthesia is used for most procedures, although procedures on the lower torso, such as debridement of sacral

pressure ulcer or suprapubic catheter placement, or lower extremity procedures may be performed under spinal anesthesia.

## SUMMARY

Patients with acute SCI are typically encountered for decompression in clinical anesthesia practice, often in an emergency setting. There may be insufficient time for a detailed preoperative history and examination. However, it is important to assess the patient's airway and be prepared to use difficult airway equipment, if necessary. It is also important to quickly assess other associated injuries that may influence intraoperative anesthetic management and line placement. The key to successful management is recognition of possible airway difficulty, and providing adequate hemodynamic support to maintain spinal cord perfusion pressure. If neuromonitoring modalities are being used, appropriate anesthetic management needs to be chosen and agents titrated accordingly. Because these patients return for subsequent surgeries or procedures, members of the anesthesia care team have to be cognizant of possible long-term complications, especially autonomic hyperreflexia, and be prepared to treat it.

## CLINICS CARE POINTS

- Trauma is one of the most common causes of acute spinal cord injury.
- Delayed resuscitation is associated with worse clinical outcomes and secondary injury after acute high spinal cord injury. The origin of spinal hypoperfusion is typically multifactorial, and includes bleeding, loss of sympathetic tone, and spinal shock. These causes must be promptly recognized and treated.
- Use of high dose glucocorticoids in acute spinal cord injury is no longer recommended.
- It is important to minimize C-spine movement at all times during airway management. Some of the recommended techniques include manual in line axial stabilization, immobilization in a C-collar, and/or use of traction in Halo.
- Autonomic hyperreflexia can develop as a late complication in up to 70% of patients after injuries above T6 level and needs to be immediately recognized and treated.

## DISCLOSURE

No conflicts of interest.

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