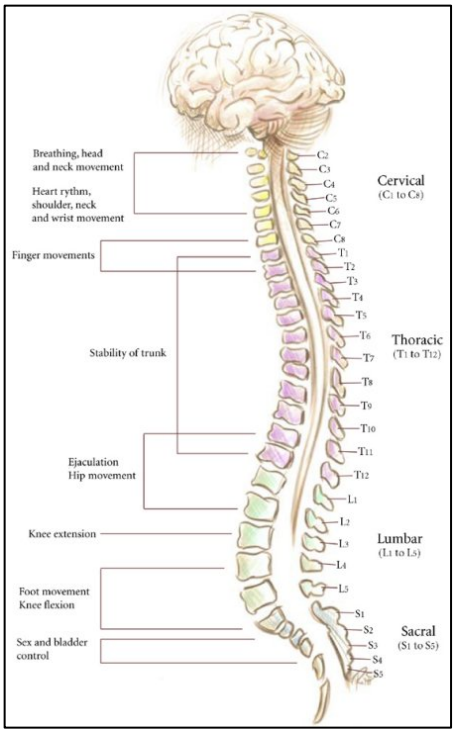
Acute Spinal Cord Injury

Anesthetic Pearls: Anesthetic Implications of the Cardiovascular Effects of Acute Spinal Injury

Acute spinal injuries resulting in spinal cord dysfunction have variable effects depending upon the location of injury (cervical, thoracic, lumber). Complete cervical spinal cord injury (SCI) results in the most pronounced physiologic effects consisting of cardiovascular instability, cardiac dysrhythmias, and ventricular dysfunction, whereas SCI below T5 spinal levels results in varying degrees of hypotension.



I. Cardiovascular Instability

1. **Hypotension** is common after SCI above the mid-thoracic spinal cord level. The hypotension is due to the withdrawal of sympathetic neural outflow between TI and L2 traveling via the splanchnic nerves to reach the viscera and peripheral blood vessels and innervating alpha- and beta-receptors. The loss of sympathetic tone results in a generalized peripheral venous pooling in the lower extremities and abdominal viscera. Approximately 60% of patients demonstrate hypotension after complete cervical injury, with treatment being required in up to 35% of patients.
2. **Bradycardia** is present in virtually all patients with complete cervical SCI, however, it is less likely with SCI involving the thoracic and lumbar regions. Bradycardia results from the interruption of the sympathetic cardiac accelerator nerves (Tl-4) leaving an unopposed parasympathetic influence. Approximately 70% of patients with high SCI demonstrate a heart rate less than 45 beats per min, with 30% of these patients requiring treatment with Atropine. Bradycardia usually resolves over a 3-5 week period post injury. More profound degrees of bradycardia, including cardiac arrest, may occur during stimulation of the patient such as turning the patient or tracheal suctioning. Patients appear particularly susceptible to these interventions during the first two weeks after injury. An awareness of the factors precipitating bradycardia should lead to preventive interventions (proper sedation, administration of 100% oxygen prior to suctioning, limiting the time allowed for suctioning). Although most episodes are effectively treated with Atropine (0.5-1.0 mg IV/IM), temporary pacemaker therapy may be required in selected cases with severe refractory bradycardia.
3. **Spinal shock** is the term used to describe the phenomenon seen with physiologic or anatomic transection (or near transection) of the cervical to high-thoracic spinal cord. The condition consists of the loss of somatic motor and sensory function below the level of injury, loss of voluntary rectal contraction, and loss of sympathetic autonomic function with hypotension and bradycardia.

Absent Neurological Component	Clinical Finding
Motor	Paralysis, flaccidity, areflexia; loss of voluntary rectal contraction
Sensory	Anesthesia to all modalities
Autonomic	Systemic hypotension, bradycardia, skin hyperemia, priapism

Rx: The more severe the functional spinal cord transection and the higher the level of injury, the greater the severity and duration of spinal shock. Autoregulation of spinal cord blood flow is lost after SCI, thus **spinal cord perfusion becomes flow-dependent**. Consequently, maintaining spinal cord perfusion is paramount to minimizing further secondary SCI. Spinal shock is treated initially with prompt fluid boluses of isotonic crystalloid fluid replacement (several fluid boluses consisting of 5-10 mL/kg crystalloid solution over 5 minutes). **The goal is to obtain a MAP of greater than 70 mmHg.** Be careful to limit the overall volume of fluid administered, as patients with cervical SCI are prone to the development of pulmonary edema (cardiogenic and non-cardiogenic). If hypotension persists despite adequate fluid administration, vasopressor therapy is recommended and should be started early. Up to 35% of patients require pressor therapy to support blood pressure. The vasopressor of choice is one with some beta agonist properties such as Dopamine (5-10 mcglkglmin) or Dobutamine (5-20 mcg/kg/min). Alpha agonists (Phenylephrine or Norepinephrine) may be needed in combination with low-dose Dopamine for optimal support of blood pressure. The use of vasopressor therapy should be considered only a temporizing measure until hemodynamic monitoring can be instituted. Invasive central venous hemodynamic monitoring is best managed utilizing a pulmonary artery catheter, which can provide a more accurate assessment of left ventricular filling pressures and cardiac outputs than a CVP. In patients with previously normal cardiac function, maintaining a pulmonary artery occlusion ("wedge") pressure in the normal to slightly elevated range (12-15 mm Hg) should best optimize spinal cord perfusion. Newer technology such as the Flow Track device that give stroke volume variation analysis may also be of benefit in the management of the hemodynamic volume status.

II. Cardiac Rhythm Disturbances in SCI

Cardiac rhythm disturbances are observed after both experimental and clinical SCI. Disturbances may include bradycardia (most common), primary cardiac arrest, supraventricular dysrhythmias (atrial fibrillation, reentry supraventricular tachycardia), and ventricular dysrhythmias. It is speculated that the cardiac abnormalities are caused by an acute autonomic imbalance resulting from a disruption of sympathetic pathways located in the cervical cord, while the parasympathetic influences via the Vagus nerve (CN-X) remain undisturbed. In patients with chronic SCI, the risk of cardiac dysrhythmias decreases over time and may eventually disappear altogether.

III. Left Ventricular Dysfunction

Left ventricular impairment has been noted in complete cervical SCI most likely due to the loss of cardiac sympathetic neural input and autonomic imbalance.