Blockade of Spinal Anesthesia

Anesthetic Pearls: Anesthetic Implications of Spinal Technique and Apnea

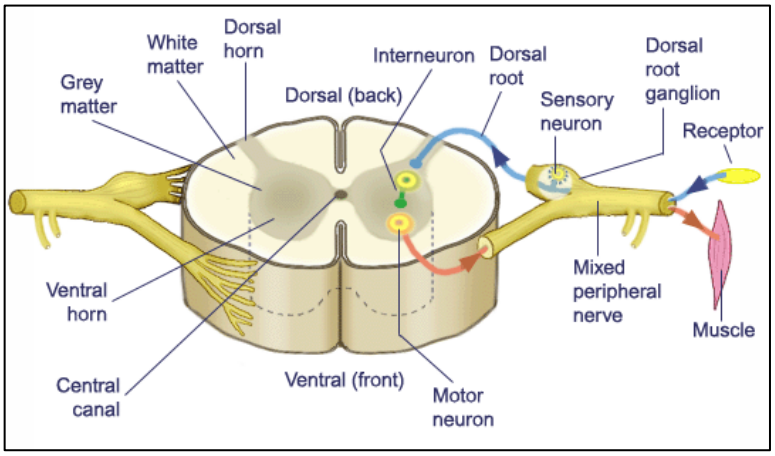
Spinal Anesthesia Site of Action:

Primary: Pre-ganglionic fibers leading to the spinal cord in the anterior rami
Secondary: Superficial spinal cord layers

Physiological Effects:

Consequences of sympathetic blockade:

1. Arteriolar dilation
   A. No significant effect on systemic BP due to compensatory upper extremity vasoconstriction (if adequately hydrated).
   B. No cerebrovascular vasoconstriction
   C. Total sympathetic blockade due to spinal anesthesia is associated with a reduction in systemic vascular resistance of less than 15% (arteriolar smooth muscle does not dilate maximally because of intrinsic tone)



2. Secondary cardiovascular responses to spinal anesthesia are due to:
   A. Effects on venous circulation (venules have minimal intrinsic tone retention and maximal dilation during spinal anesthesia)
   B. Consequences of venous dilation:
      - Reduced venous return to heart leads to decreased cardiac output and consequently decreased systemic blood pressure
      - Severe systemic hypotension in hypovolemic patients

Treatment:

1. Arteriolar vasoconstriction: Alpha-adrenergic receptor agonist (Phenylephrine, Ephedrine, Epinephrine), V-1 receptor agonist (Vasopressin)
2. Trendelenberg positioning
3. Fluid bolus

High Spinal:

A. Deleterious effect on spontaneous ventilation leading to respiratory compromise
B. Apnea may occur and likely due to abnormal medullary ventilatory center function (secondary to ischemia due to reduced cerebral blood flow)
C. Apnea unlikely due to Phrenic nerve blockade causing paralysis of diaphragmatic excursion.