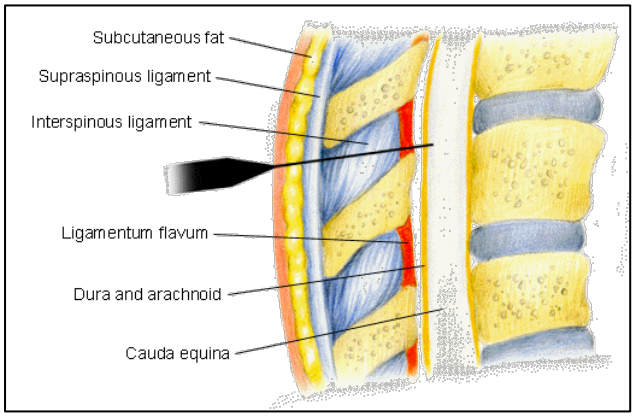
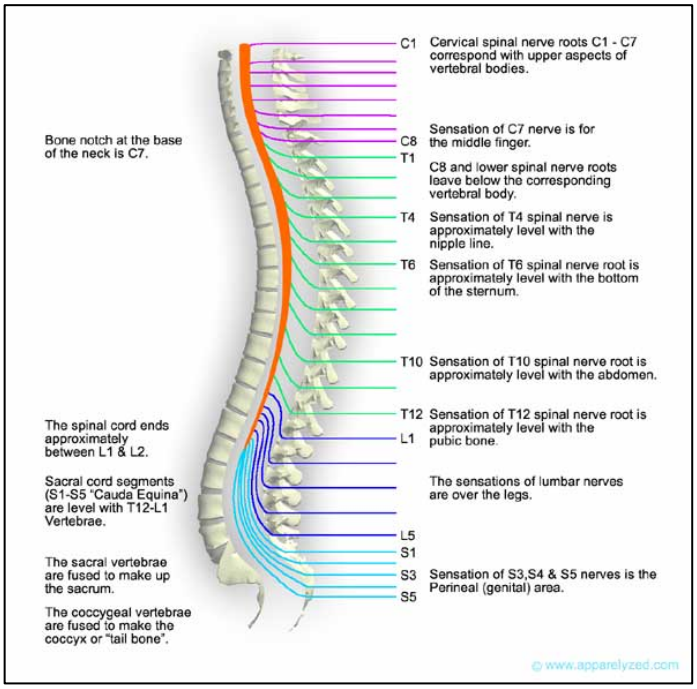
Spinal Block and Hypotension

Anesthetic Pearls: Anesthetic Implications and Management of Spinal Technique and Hypotension

During spinal anesthesia, the interruption of autonomic transmission at the spinal nerve roots produces sympathetic blockade and physiologic responses (decreased blood pressure, heart rate, and contractility) resulting form decreased sympathetic tone and unopposed parasympathetic tone. These effects are generally proportional to the level of the sympathectomy (the blood pressure deceases approximately 2.5% per segment blocked).



Vasomotor tone is determined primarily by sympathetic fibers arising from T5 – L1. The blockade of these nerves causes vasodilatation of the venous capacitance vessels, pooling of blood, and decreased venous return to the heart. In some cases, arterial vasodilatation may also decrease systemic vascular resistance. However, the arterial effects may be minimized by compensatory vasoconstriction above the level of the block. A high sympathetic block not only prevents compensatory vasoconstriction but also blocks sympathetic accelerator fibers that arise between T1-4. Profound hypotension may result from vasodilatation combined with bradycardia and decreased cardiovascular contractility. These effects are exaggerated if venous return is further compromised by a head-up position or from the weight of a gravid uterus on the aorta or vena cava.



What can be done to prevent hypotension?

1. Volume loading (10-20 ml/kg)
2. Left uterine displacement in the gravid female
3. Drugs:
    a. Phenylephrine, Ephedrine, Epinephrine, and Vasopressin for hypotension
    b. Atropine for bradycardia.