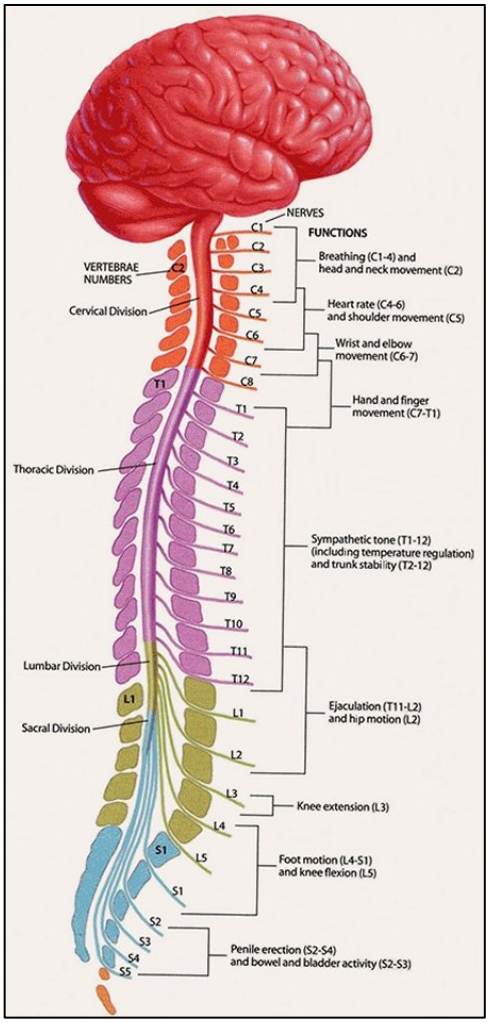
T4 Spinal Anesthetic

Anesthetic Pearls: Anesthetic Implications of T4 Spinal Anesthetic



Cardiovascular Effects

Arterial hypotension is commonly associated with spinal anesthesia. Blood pressure decreases approximately 2.5% per spinal segment blocked. The amount of blood pressure change often directly correlates with the total amount of sympathetic blockade. Spinal anesthetics cause the reduction of arteriolar resistance (SVR) 5-20%, stroke volume 5-20%, heart rate 5- 25%, cardiac output 10-30%, and arteriolar blood pressure 15-30%. Occasionally spinal anesthesia results in bradycardia caused from blocking cardio-accelerator nerves (T4) and from reduced venous return to the heart.

If T1 – T4 levels are blocked, there is a resultant total blockade of preganglionic sympathetic fibers as well as inhibition of cardio-accelerator nerves. This interruption of neural flow produces venodilatation and reduction of venous return. The decreased venous return causes a reflexive slowing of the heart by activating stretch receptors in the right heart and great veins. The fall in mean artery blood pressure is then caused by a decrease in cardiac output resulting from venodilatation and decreased stroke volume.

If the patient is volume depleted and has cardiac impairment (ischemia or infarction), the patient is then predisposed toward having a cardiac event. Care must be taken to guard against severe enough cardiac depression that can lead to cardiac arrest.

Respiratory Effects

Respiratory arrest can be caused by high levels of spinal anesthesia and are attributed to brain stem ischemia from resultant hypotension. Spinal anesthesia may also at times produce dyspnea, which is caused by decreased afferent input to the abdomen and chest walls. Diaphragmatic function is usually not affected because the Phrenic nerve is spared (C3 – C5). However, expiration may be difficult for patients with COPD secondary to weakened abdominal muscles (primary muscles involved in expiration).