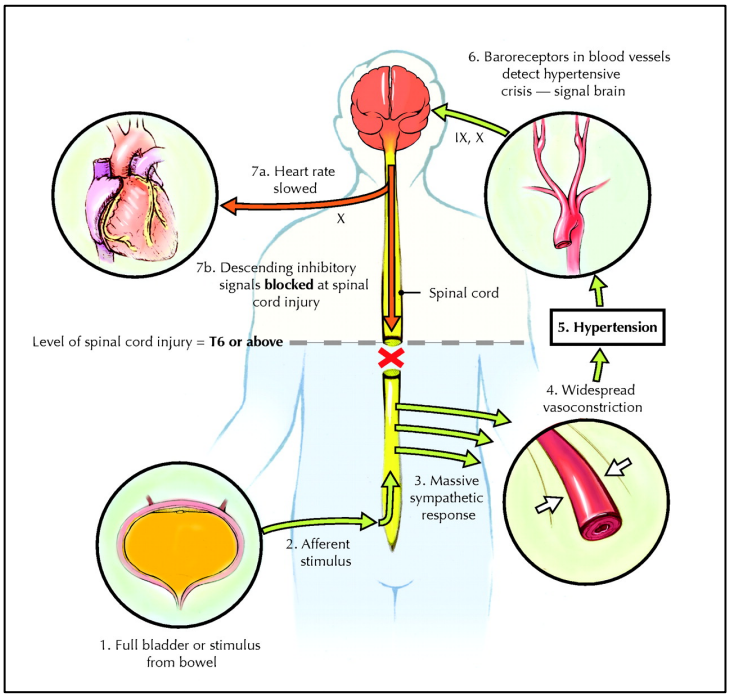
Autonomic Hyperreflexia

Anesthetic Pearls: Anesthetic Implications and Management of Autonomic Hyperreflexia

Autonomic Hyperreflexia (Autonomic Dysreflexia) occurs in approximately 85% of patients with a sensory level lesion above T-6 but is unlikely to occur in patients with lesions below T-10. In patients with lesions below T-10, stimulation of the autonomic reflexes causes vasoconstriction below this level. However, hypertension does not develop as a result of compensatory vasodilation above the level of the lesion. This results in flushing, pilomotor erection, sweating, and congestion of mucous membranes above the level of the injury. However, in patients with lesions at T-6 and above, the vascular territory available for dilatation is limited, and stimulation of these reflexes causes hypertension. Hypertension may be severe enough to result in loss of consciousness, convulsions, and cerebral hemorrhage, and should therefore be treated as a medical emergency. The hypertension can stimulate the carotid sinus and cause a reflex bradycardia, cardiac dysrhythmias, and potentially cardiac arrest.



Painful cutaneous stimulation of peripheral sensory receptors causes reflex activation of efferent sympathetic fibers that originate in the spinal cord. Neuroanatomically speaking, the sympathetic response occurs at and below the level of the stimulation. In normal individuals, cortical fibers tone down these spinal reflexes. However, in patients with chronic spinal cord injuries, there is interruption of this cortical inhibition, and painful stimulation or bowel / bladder distension can lead to the development of autonomic hyperreflexia.

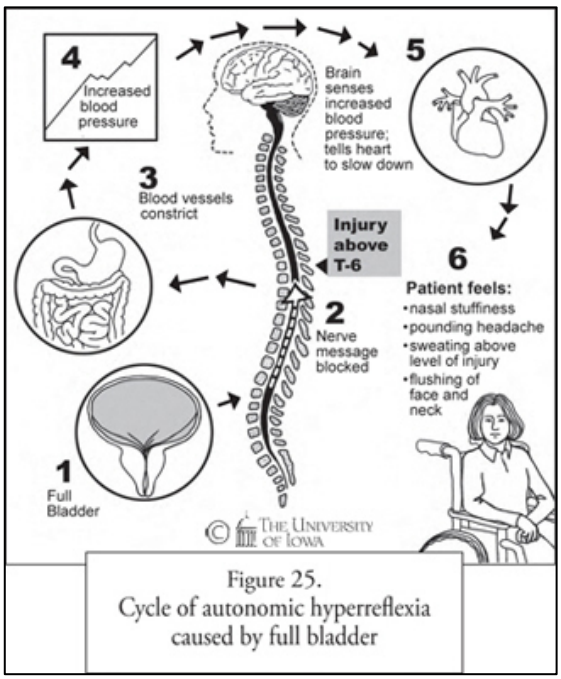


Figure 25.
Cycle of autonomic hyperreflexia caused by full bladder

The best management is prevention. Many patients with high spinal lesions give a history consistent with autonomic hyperreflexia in response to cutaneous stimulation or bowel / bladder distension. Adequate analgesia should be given in anticipation of any invasive procedure. Management of autonomic hyperreflexia involves removing the stimulus, treating the afferent limb of the response (adequate analgesia), and treating the efferent limb of the reflex (ganglionic blockers [Trimethephan], alpha blockers [Prazosin or Phentolamine], direct vasodilators [Sodium Nitroprusside]). Deep general anesthesia or spinal anesthetic techniques are an option, although many clinicians are reluctant to administer spinal anesthesia because of the difficulties encountered with determining the anesthetic level, exaggerated hypotension, and technical problems resulting from anatomic deformities. Epidural anesthesia is usually ineffective secondary to decreased block density and is especially ineffective for urologic cases because of sacral sparing.