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Complications of sympathetic blocks for extremity pain

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Sympathetic blockade is often undertaken to diagnose or treat sympathetically maintained pain in painful extremities. A thorough understanding of the anatomy and physiology of the sympathetic nervous system is essential to understand complications that may arise as a result of sympathetic blocks and to develop strategies to prevent them. Complications of sympathetic blockade vary depending on the sympathetic ganglia blocked, the location, the approach, and the agents used. Complications related to sympathetic blocks can be generally divided into unwanted results related to blockade of sympathetic fibers and untoward events related to injuring other organs as a result of the procedure. This article describes complications that may occur with sympathetic blocks performed for extremity pain and potential strategies to minimize such events.

An understanding of both anatomy and physiology is necessary to appreciate the basis for complications associated with sympathetic blockade. A brief review of the anatomy follows.[1-4] Of greatest interest clinically are the four main components of the sympathetic nervous system. These include the cervico-thoracic, splanchnic, celiac plexus, lumbar ganglia, and the superior hypogastric and inferior hypogastric plexuses. These systems are connected centrally and distally by two ganglionated trunks extending from the first cervical vertebrae to the coccyx. Although the ganglia are variable in number, they are connected centrally by small myelinated nerve fibers, the rami communicans (white), and distally to the spinal nerves via gray rami. Considerable variation of these synaptic connections, both cranially and caudally, is apparent. Also, there is considerable divergence of preganglionic fibers with multiple postganglionic neurons.

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The cervicothoracic chain distributes sympathetic fibers to the head, neck, and upper extremities. Those fibers to the head synapse with postganglionic fibers in the superior cervical ganglion to join other fibers on the carotid vessels and nasociliary nerves. Both vasoconstrictor and vasodilator fibers to the head, neck, and upper extremity pass through the stellate ganglion. Vasodilation of the face, for example, is therefore not a passive process. Many fibers (up to 20%), so-called Kuntz's nerves, do not pass through the stellate ganglion.[5] This probably explains the much lower incidence of Horner's syndrome after section of the upper thoracic chain at T2 and T3.[6]

The stellate ganglion is a fusion of the last cervical and first thoracic ganglia. It is situated at the anterolateral aspect of the 7th cervical and 1st thoracic vertebrae, specifically in a groove between the vertebral body and the transverse process. However, it should be noted that there is considerable variation. The cervical sympathetic chain is variable and may also be included in the sheath of the carotid artery. As the sympathetic trunk enters the chest, it lies more lateral in close proximity to the junction of the neck and head of each rib. The T2 ganglion is generally found in the second intercostal space, whereas the T3 ganglion is anterior to the neck of the third rib. As the sympathetic trunks descend,

they assume a gradual anterolateral position in relation to the vertebral bodies. This relationship continues throughout their descent into the lumbar region.

The celiac plexus consists of efferent and afferent neurons, as well as both pre- and postganglionic sympathetic efferent fibers. Other fibers include parasympathetic fibers and visceral sensory afferents. Block of this plexus, therefore, interrupts traffic subserving the foregoing modalities in both directions. Preganglionic sympathetic efferents originate in the three splanchnic nerves: T5–T10, greater splanchnic; T10–T11, lesser; and T12, least splanchnic nerve. The postganglionic fibers originate in the upper lumbar splanchnic ganglia, and most parasympathetic nerve fibers descend in the vagus nerves. Splanchnic nerve blocks are most effective for pain of malignant and benign origins in the upper abdominal viscera.

The lumbar sympathetic chain runs anterolateral to the lumbar vertebrae, the ganglia of which are variable in both position and number.[7] These have been well studied. The L3 ganglia tend to be in the middle of the body of the L3 vertebrae, whereas the others are either absent or fused, laying more anterolateral. The 5th ganglion is frequently discrete as a large contribution to the 5th lumbar nerve. Connections between the sympathetic chains have been described and possibly explain the less than optimal results of unilateral sympathetic trunk interruption.

The superior hypogastric plexus consists of afferent nociceptive fibers from pelvic organs, descending sympathetic fibers, and parasympathetic fibers from the pelvic splanchnic nerves ascending from the inferior hypogastric plexus. Most of these latter parasympathetic fibers pass to the left of the superior hypogastric plexus for distribution to the inferior mesenteric vessels. Other parasympathetic fibers arising from the pelvic splanchnic nerves supply the distal colon. The superior hypogastric plexus is actually prelumbar and not presacral as usually described. Hypogastric nerves descend from the superior hypogastric plexus contain mainly sympathetic fibers and afferent sensory fibers from the pelvic organs and the colon as far as the splenic flexure. The preganglionic sympathetic cell bodies reside in the lower three thoracic and upper two lumbar segments. The parasympathetic cell bodies are in the S2–4 segments of the spinal cord.

Side effects with complications

The side effects that accrue from interruption of sympathetic nerves should be understood and distinguished from complications or unwanted results of sympathetic blockade. A further distinction should be made between those complications that result from local anesthetic block and those from more permanent interruption, or neurolysis, of the sympathetic chains using chemical or radiofrequency techniques.

Side effects such as Horner's syndrome that includes ptosis, myosis, enophthalmos, anhidrosis of neck and face,

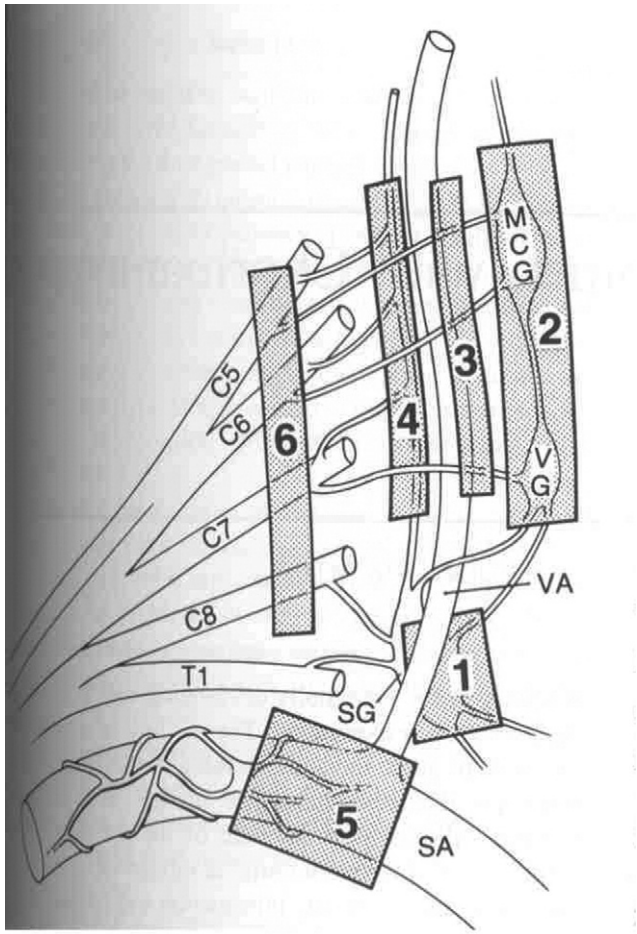

Figure 1 Sites demonstrated by investigation of sympathetic block after stellate ganglion block. (1) stellate ganglion; (2) cervical sympathetic chain; (3) gray rami communicantes; (4) vertebral plexus; (5) subclavian sympathetic plexus; (6) brachial plexus; (7) MCG, middle cervical ganglion; SA, subclavian artery; SG, stellate ganglion; VA, vertebral artery; VG, vertebral ganglion.[9]

conjunctival injection, unilateral flushing, and nasal congestion are expected reactions to block of the stellate ganglion. The most common complications of stellate ganglion blockade result from the diffusion of the local anesthetic to adjacent neural structures. This fact, acknowledging the extensive spread of local anesthetic solutions after injection of the stellate ganglion, is well described by Malmqvist and coworkers and Hogan and coworkers.[8,9] (Figure 1). Local anesthetic block of the recurrent laryngeal nerve will cause hoarseness, a sensation of having a lump in the throat, and shortness of breath. For this reason, bilateral stellate ganglion blocks are not advised, as interruption of both laryngeal nerves and loss of laryngeal reflexes may significantly interfere with respiration and protective laryngeal reflexes, allowing the inhalation of particulate matter or fluids. Diffusion to the phrenic nerve similarly may result in temporary paralysis of the diaphragm with resultant respiratory compromise, particularly in those with respiratory insufficiency. Bilateral blockade would obviously require emergency ventilation.