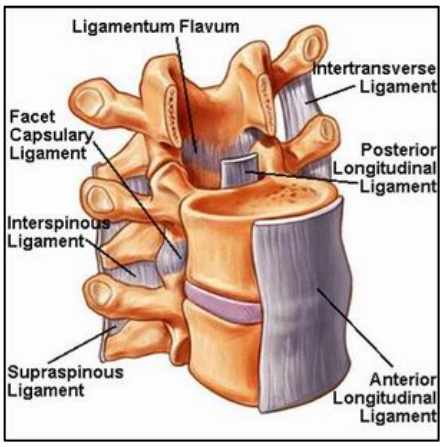
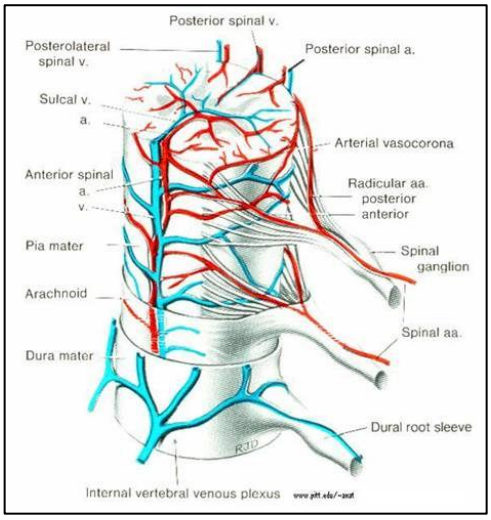
Spinal Anesthesia: Transient Neurologic Effects

Anesthetic Pearls: Anesthetic Implications of Transient Neurologic Syndrome from Spinal Anesthesia



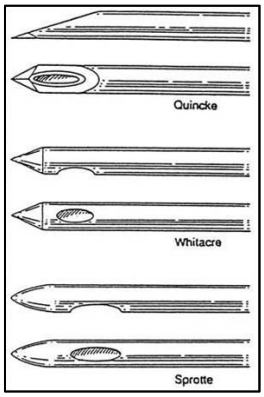
Transient Radicular Syndrome / Transient Neurological Syndrome (TNS) is defined as self-resolving pain related to the use of spinal Lidocaine, lithotomy position, and early ambulation post-op after spinal anesthesia.

The past 20 years has seen a large number of case reports and incidence studies that implicate Lidocaine as being more neurotoxic than Bupivacaine, Ropivicaine, and Tetracaine. Available studies have suggested a risk of persistent lumbosacral neuropathy after spinal Lidocaine by single injection in approximately 1 in 1300 procedures and a risk as high as about 1 in 200 after continuous spinal anesthesia with Lidocaine (very uncommonly used currently in the US). While the syndrome is uncommon, this risk is probably an order of magnitude higher than the risk reported for other commonly used local anesthetics in general anesthesia. Spinal Lidocaine is also implicated in the syndrome of **Transient Neurologic Symptoms** (previously referred to as Transient Radicular Irritation), manifest by pain or dysesthesia in the buttocks or legs after recovery from anesthesia. Although the pain typically resolves within 1 week without lasting sequelae, it can be severe in up to 33% of patients with the syndrome. In addition to clinical studies, animal studies have shown that Lidocaine can be neurotoxic at clinically available concentrations and can be more neurotoxic than equipotent concentrations of other commonly used local anesthetics. The mechanism of this neurotoxicity has been hypothesized to involve changes in cytoplasmic calcium homeostasis and mitochondrial membrane potential.



Repeated applications of local anesthetics via an indwelling intrathecal catheter or by multiple single shot spinal injections to improve on a patchy or failed block have been associated with cauda equina syndrome.

Neurologic complications of regional anesthetics are usually discovered after the patient has left the recovery room. Persistent motor blockade during recovery from sensory anesthesia may indicate anterior spinal artery occlusion or spasm. Lack of recovery from spinal blockade in the expected time interval may indicate spinal cord compression due to epidural hematoma. Since early intervention is the key to success in managing these potentially devastating complications, prompt diagnosis (MRI or CT) and early surgical management is indicated. Spinal hematomas associated with indwelling epidural catheters and intrathecal bleeding with continuous spinal anesthesia in patients receiving thrombolytic agents are well reported in the literature. Spinal anesthesia should definitely be avoided in patients who will receive or have received thrombolytic therapy. Allowing the local anesthetic to wear off prior to instituting continuous post-op infusions and deliberation of appropriateness of narcotic infusions will permit ongoing evaluation of the neurologic status during the post-op period. The patient should be monitored closely for early signs of cord compression such as complaints of back pain, an increase in intensity of motor or sensory blockade, or the development of new paresis. If spinal hematoma is suspected, the treatment of choice is <u>immediate</u> decompressive laminectomy. Neurologic recovery is unlikely if surgery is postponed for more than 8 hours.



Suggested Precautions with Spinal Technique:

1. Aspiration of CSF before and after drug injection.
2. Evaluation of the extent of sacral blockade to ascertain preferential distribution to that site.
3. Limit the drug dosage to a maximum pre-calculated "safe" dosage.
4. If injection is repeated, avoid reinforcement of the same drug distribution (change patient position, drug baricity, etc.)
5. If CSF cannot be aspirated after injection, do <u>not</u> repeat with a "full" dose unless there are <u>no</u> signs of neural blockade (including the sacral area) are present.