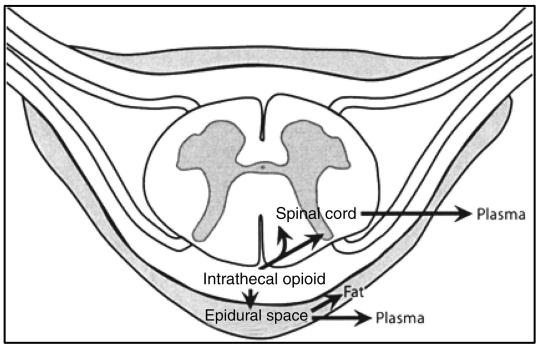
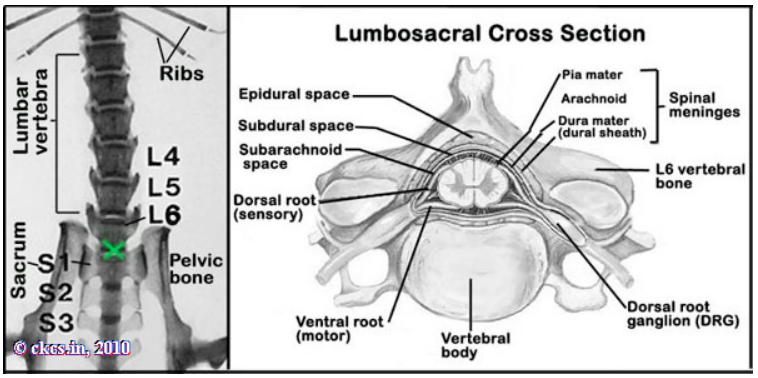
Intrathecal Opioids

Anesthetic Pearls: Anesthetic Implications and Management for Intrathecal Opioids



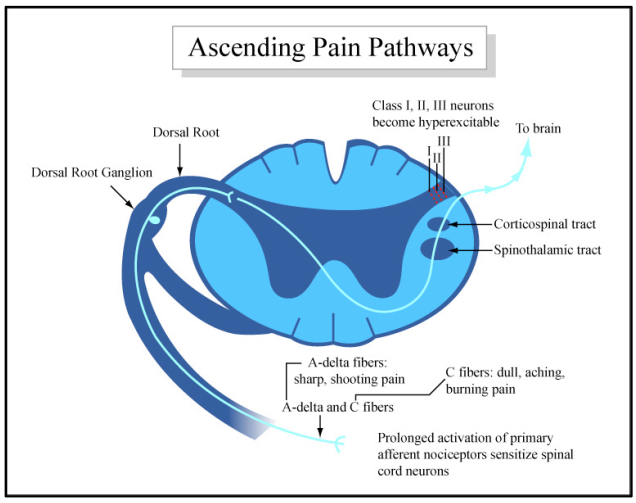
Placement of opioids in the epidural or subarachnoid space to manage acute or chronic pain is based on the knowledge that opioid receptors (principally **Mu** [μ] receptors) are present in the substantia gelatinosa of the spinal cord. Analgesia is dose related (epidural dose is 5 - 10 times the subarachnoid dose) and specific for visceral rather than somatic pain.

Analgesia that follows epidural placement of opioids reflects diffusion of the drug across the dura to gain access to Mu opioid receptors on the spinal cord as well as systemic absorption to produce effects similar to those that would follow IV administration of the opioid. Spinally administered opioids provide powerful regional analgesia without associated motor blockade or excessive CNS depression.



Morphine selectively suppresses neurons of Dorsal Horn Laminae - I and -V, mediating noxious information, while having no effect on Lamina - IV neurons (known to process light touch and proprioceptive input). Enkephalin-containing neurons are almost exclusively localized to the Substantia Gelatinosa, offering evidence that there is an intrinsic modulatory system whose activation could attenuate release of Substance-P and other nociceptive transmitters.

Three major opioid receptor subtypes are involved in nociceptive input modulation. **Mu** (μ) and **Delta** (δ) receptors produce dose-dependent inhibition of cutaneous-thermal responses, whereas activation of **Kappa** (κ) receptors results in powerful suppression of visceral-chemical responses while having no effect on somatic nociception.



The onset of analgesic effects is directly proportional to the lipid solubility of the opioid. Preservative-free Morphine (along with Hydromorphone and Meperidine) has a relatively low lipid solubility and its onset of action is delayed for typically 20 to 40 minutes after administration. The hydrophilic nature of the opioid also determines its duration of action. Preservative-free Morphine is very hydrophilic and poorly lipid soluble, which extends its duration of analgesic effect up to 12 to 24 hours. Because of its poor lipid solubility, intrathecal Morphine remains in the cerebrospinal fluid (CSF) for a prolonged period of time. It is circulated through cerebral spinal bulk flow and eventually rises rostrally to supraspinal levels. This is in contrast to intrathecal Fentanyl which is highly hydrophilic and lipid soluble. Intrathecal Fentanyl has an extremely fast onset of action at the level of the nerve roots and spinal cord but does not have an extended analgesic effect.

Intrathecal Morphine has **bimodal analgesic effects**. The first peak is soon after administration and is due to spinal opiate receptor binding. The second peak occurs 12 to 24 hours later and is due to supraspinal binding as the drug is circulated. The side effects, however, are important to recognize and treat. Respiratory depression can be delayed up to 24 hours after administration and is due to the cephalad spread of intrathecal Morphine to the opioid receptors in the medullary centers of the brain stem. Thus, patients receiving intrathecal Morphine must be closely monitored for up to 24 hours afterward for signs of respiratory depression. Patients with post-op pain despite having received intrathecal Morphine present a management dilemma. Giving the patient additional systemic opioids must be done cautiously, as it may increase and potentiate the risk of respiratory depression.