LOCAL ANESTHETIC SYSTEMIC TOXICITY (LAST)

10 Fast Facts

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1. **Sodium channel blockade:** The analgesic effect of local anesthetics is primarily caused by inhibition of neural conductance secondary to sodium channel blockade.
2. **Lipid Solubility Link:** The more lipophilic/potent the agent (e.g., **bupivacaine**), the more cardiotoxic it is and the harder the resuscitation.
3. **"Fast-In, Slow-Out":** Bupivacaine binds rapidly to cardiac sodium channels but dissociates slowly during diastole, leading to profound, refractory arrhythmias.
4. **Atypical presentation:** Patients may present directly with seizures or cardiovascular collapse without prodromal symptoms.
5. **Absorption Hierarchy:** Absorption (and risk) is highest in areas with greater blood flow; for example, intravenous (IV) > tracheal > intercostal > epidural > subcutaneous.
6. **Acidosis Danger:** Hypercapnia and acidosis increase the unbound, active fraction of the drug and increase cerebral blood flow, worsening CNS toxicity.
7. **Intravenous Lipid Emulsion (ILE) mechanism:** acts as a "lipid sink," sequestering the drug from the heart/brain.
8. **Epinephrine Dosing:** In LAST-induced arrest, use **low-dose epinephrine** (<1 mcg/kg); high doses can impair the effectiveness of ILE and worsen acidosis.
9. **First-Line treatment of seizures: Benzodiazepines** are the preferred treatment. Avoid propofol in patients showing signs of hemodynamic instability.
10. **Resuscitation Duration:** Because LAs are metabolized/redistributed over time, successful resuscitation may require **prolonged effort** (often >60 mins) compared to standard ACLS.

References:

Hoffman, R. S., et al. (2019). Local Anesthetics. In *Goldfrank's Toxicologic Emergencies* (11th ed., pp. 936–948). McGraw-Hill Education.