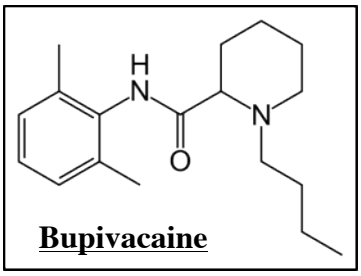
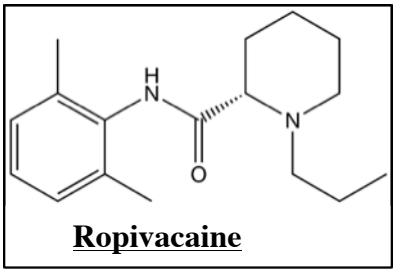
# Bupivacaine Toxicity

**Anesthetic Pearls:** Anesthetic Mechanism and Treatment of Bupivacaine Cardiac Toxicity

**Bupivacaine** cardiac toxicity is manifest in the development of refractory dysrhythmias (V-tach / V-fib) leading to cardiovascular collapse. Multiple studies have suggested that several mechanisms of cardiotoxicity are involved.



**Bupivacaine**



**Ropivacaine**

Proposed Cardiotoxic Mechanisms:

1. **Blockade of sodium channels thereby leading to prolongation of the recovery phase of sodium channels**.
2. Marked depression of the rapid phase of depolarization (V-max) of the cardiac action potential. The depressed V-max results in slowed conduction of potentials, which leads to unidirectional block and development of reentrant ventricular dysrhythmias. All local anesthetics do this to some extent but it is much more profound with Bupivacaine.
3. Significant increase in the effective refractory period, leading to polymorphic ventricular tachycardia that is similar to "*torsades de pointes*".
4. Direct myocardial depression.

**The best treatment is <u>PREVENTION</u>**. Always inject small doses (3 ml) with frequent aspirations to confirm needle / catheter position. Consider the use of a test dose with an intravascular marker (1:200,000 Epinephrine) and do <u>not</u> exceed maximum recommended doses.

Most Bupivacaine related cardiovascular complications in humans have occurred in pregnant women and studies in pregnant animals have confirmed that pregnancy does increase the sensitivity to the cardiotoxic effects of Bupivacaine.

**If cardiotoxic effects are observed, immediate and aggressive treatment is essential.**

1. ABC's of ACLS
2. Epinephrine (+/- Bretylium [large doses])
3. Cardioversion
4. Intralipid (20%) regimen:
   - Administer 1 ml/kg over 1 minute.
   - Repeat twice more at 3 – 5 minute intervals.
   - Then (or sooner if stability is restored), convert to an infusion at a rate of 0.25 ml/kg/min, continuing until hemodynamic stability is restored (increasing the dose beyond 8 ml/kg has not been found to be useful).
   - The proposed mechanism of Intralipid benefit involves the lipophilicity of Bupivacaine following a concentration gradient from the arrested myocardium into the Intralipid solution.