Pearls and Pitfalls
The Role of Intralipid During ACLS

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Objective: Upon completion of this presentation, participants will be able to describe the role of intralipid in local anesthetic toxicity, and its interactions with vasopressors used in ACLS.

Summary: Local anesthetic toxicity following epidural anesthesia occurs with an incidence of approximately 4 per 10,000. Until the introduction of lipid therapy, supportive therapy and cardiopulmonary bypass were the only treatment options available for local anesthetic-induced cardiac toxicity. Intralipid was first shown to be an effective treatment in animal studies; however, almost 10 years passed until it was used to resuscitate a patient suffering from a bupivacaine-induced cardiac arrest who had failed standard advanced cardiac life support (ACLS) measures. Since then, lipid therapy has been used successfully in various clinical settings, so few would doubt the efficacy of this treatment in the setting of local anesthetic toxicity. While intralipid has not been incorporated into ACLS guidelines yet, it has been incorporated into guidelines for the management of severe local anesthetic toxicity by the Association of Anaesthetists of Great Britain and Ireland.

While the early studies and case reports made lipid therapy seem like a panacea, recent studies have created controversy as to where lipid therapy belongs in the setting of ACLS. Weinberg et al using a rat model, demonstrated that epinephrine and vasopressin resulted in less hemodynamic and metabolic recovery, compared to lipid alone. In contrast, Mayr et al found that a combination of epinephrine and vasopressin were superior to lipid therapy using an asphyxiated swine model. More recently, it has been shown that doses of epinephrine greater than 10 mcg/kg impairs lipid-based resuscitation. Furthermore, it is not known whether lipid interferes with other lipophilic medications used in ACLS or cardiopulmonary bypass, such as amiodarone. Clearly, additional studies are needed to determine the best timing of intralipid in the setting of local anesthetic toxicity, but also, what combination of medications promotes the best outcomes.

Key Points:
1. If local anesthetic systemic toxicity occurs, stop injecting local anesthetic and begin ACLS, as hypoxemia and acidosis will worsen the local anesthetic toxicity. Consider intralipid therapy early in the resuscitation, as evidence suggests that lipid infusion should not be delayed until other resuscitative measures have failed.
2. The recommended dose of 20% intralipid for local anesthetic toxicity is 1.5 mL/kg as a bolus. This dose can be repeated 1-2 times for persistent asystole. After the initial bolus, an infusion should be started at 0.25 mL/kg/min for 30-60 minutes, and this infusion can be increased to 0.5 mL/kg/min if there is persistent hypotension.
3. Potential complications of intralipid administration include: allergic reaction, acute lung injury, pancreatitis, and fat emboli.
4. Caution is always best, when performing regional anesthesia/analgesia use the lowest effective dose necessary, use incremental injections, and remember to aspirate prior to each injection.
5. Further research is needed to determine what intralipid treatment protocol (timing, dose, and combination of medications) optimizes outcomes.

Key References: