Abstract 19

Perioperative ACLS Recommendations Should Be Modified for the Treatment of Local Anesthetic Toxicity

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Case 1: A patient scheduled for a carotid endarterectomy received a preoperative cervical plexus block and 30 mL of 0.5% bupivacaine. Thirty minutes later, three beats of ventricular tachycardia was noted and 100 mg of IV lidocaine was administered. Within minutes the patient suffered a cardiac arrest consisting of ventricular fibrillation (VF) and pulseless wide QRS bradycardia. The patient required more than 25 minutes of chest compressions and multiple vasopressor doses before a stable heart rate and blood pressure returned.

Case 2: A patient underwent a total knee replacement. Postoperatively a sciatic nerve block was attempted and 20 mL of 0.5% bupivacaine was administered. Before completion of the procedure, the patient developed seizures, profound hypotension, bradycardia, and a markedly widened QRS complex. As part of her resuscitation she received a rapid bolus of 500 mL 20% intralipid. Within 20 minutes her QRS complex narrowed and her vital signs normalized.

Discussion: The large volumes of local anesthetics typically administered during peripheral nerve blocks can lead to local anesthetic toxicity, including malignant cardiac arrhythmias. The onset of cardiac arrest may be instant (intravascular injection) or delayed (systemic absorption). Perioperative care providers should be aware that current advanced cardiac life support (ACLS) recommendations need to be modified when local anesthetic toxicity is suspected. The first consideration is the role of lidocaine, which is classified as a Class IIb treatment for VF/pulseless ventricular tachycardia. While it may seem obvious that a local anesthetic should not be administered to treat cardiac arrest caused by local anesthetic toxicity, a prominent anesthesia publication recently included such a recommendation.[1] The proper designation should instead be Class III, defined as ". . . a treatment (which) is not useful/effective, and . . . may be harmful."[2] The other modification in the treatment of local anesthetic toxicity involves the role of administering intralipid. More than a decade of animal research and many recent case reports suggest the value of initiating this therapy along with standard ACLS recommendations.[3] Intralipid most likely acts as a "lipid sink" for lipophilic local anesthetics and may also have a protective effect at the cellular level.

Conclusion: Perioperative caregivers should modify current ACLS recommendations when local anesthetic toxicity is suspected. Lidocaine is contraindicated, and intralipid therapy should be considered

1. *Schwartz DA. Perianesthetic management of the ex-premature infant. Anesthesiology News 2010; 36:41–44. Lesson 289.*

2. *2010 AHA Guidelines for CPR and Emergency Cardiovascular Care Science. Circulation 2010; 122(suppl 3).*

3. *Rowlingson JC. Resuscitation of local anesthetic toxicity with intralipid. American Society of Anesthesiologists Web site. http://www.asahq.org/Knowledge-Base/Subspecialty-Interests/ASA/Resuscitation-of-Local-Anesthetic-Toxicity-with-Intralipid.aspx. Published October 2007. Accessed January 27, 2011.*