Local Anesthetic Toxicity: Prevention and Treatment

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LESSON OBJECTIVES

Upon completion of this lesson, the reader should be able to:

1. List the signs and symptoms of local anesthetic toxicity.
2. Discuss the concept of "maximum safe doses of a local anesthetic."
3. Describe methods for reducing the risk of local anesthetic toxicity.
4. Explain the treatment of local anesthetic-related neurologic symptoms and toxic side effects.
5. Discuss the effects of epinephrine, clonidine, and the site of injection on peak local anesthetic concentrations in blood.
6. Describe the mechanism of local anesthetic central nervous system toxicity.
7. Identify the risk factors for local anesthetic toxicity.
8. Plan the treatment of local anesthetic-induced cardiac arrhythmias and cardiac arrest.
9. Describe the mechanism of local anesthetic cardiovascular toxicity.
10. Explain the dosage and proposed mechanism of lipid emulsion therapy.

Introduction

Cardiac and central nervous system (CNS) toxic side effects of local anesthetics are relatively rare but potentially catastrophic complications of local and regional anesthesia. Fortunately, the likelihood of a local anesthetic toxic event can be reduced by adherence to good technique. These reactions are in most cases readily treatable. This lesson will review the pharmacology, risk factors, presentation, and treatment of local anesthetic toxicity.

Mechanism of Local Anesthetic Toxicity

Local anesthetics normally produce their desired effects on peripheral nerves by binding and inhibiting voltage-gated sodium channels in neural cell membranes. When a sufficient fraction of these sodium channels are inhibited, the neuron cannot depolarize and cannot generate or conduct action potentials.
Local anesthetic molecules are weak bases. At physiologic pH, they exist in solution as a mixture of neutral, more lipid-soluble molecules and protonated, relatively lipid-insoluble molecules. In order to approach the local anesthetic binding site on Na channels, local anesthetic molecules must penetrate to the inner surface of the plasmalemma. The uncharged fraction of a local anesthetic molecule mix is therefore considered to be the fraction that most readily produces both desired and undesired drug actions. Curiously, once the molecule gains entry into the cytoplasm, it is the charged form that has greater potency. The different properties of the lipid-soluble and lipid-insoluble fractions are important when one considers treatments for local anesthetic toxicity.

There are three principal routes for entry of local anesthetic into the plasma: direct injection into an artery or vein, absorption from a depot dose in other tissues, and transcutaneous or transmucosal absorption.

In the plasma, all local anesthetics are bound to proteins to varying degrees, primarily to α1-acid glycoprotein (AAG) and albumin. Plasma protein levels are affected by disease states and age. The long-duration local anesthetics (bupivacaine and ropivacaine) are bound by plasma proteins to a greater extent than the less potent, shorter duration local anesthetics. Bupivacaine is approximately 95% protein-bound. Intermediate-duration local anesthetics (lidocaine and mepivacaine) have a smaller protein-bound fraction (60-70%). Protein binding helps to reduce the likelihood that local anesthetics in blood will enter brain or cardiac tissue causing either CNS or cardiac toxicity.

Local anesthetics may bind a wide variety of channels and enzymes in cardiac muscle and in the CNS, and increased blood concentrations of local anesthetics can cause toxic effects in these tissues. Possible mechanisms by which local anesthetics can produce cardiovascular collapse include depression of cardiac contractility, potentiation of cardiac arrhythmias, and peripheral vasodilation. The balance of these effects likely varies among local anesthetic agents. Lidocaine tends to produce vasodilation and negative inotropy while rarely producing arrhythmias. In contrast, bupivacaine has a greater tendency to produce ventricular arrhythmias in addition to vasodilation and negative inotropy.

For purposes of this lesson, we have made no assumptions as to the mechanism by which local anesthetics actually produce either CNS or cardiovascular toxicity. It is possible that these toxic side effects occur through binding to sodium channels; it is equally possible that another mechanism is operative.

Intravascular Injection
Intravascular injection of local anesthetic agents is possible with most regional anesthetic techniques due to the proximity of vascular structures to nervous tissue. For example, accidental injection of local anesthetic into the epidural venous plexus can occur when dosing an epidural catheter, and injection into the femoral artery or vein can occur during an attempted femoral nerve block. Injection into an artery feeding the brain must be considered when performing regional anesthetics in the neck, such as interscalene brachial plexus blocks. With accidental injection of local anesthetic into a carotid or vertebral artery, a bolus dose of local anesthetic at a relatively high plasma concentration is delivered directly to the brain. In this case, one should expect rapid onset of CNS toxic side effects, with rapid onset of seizures after even a very small intravascular injection. Fortunately, due to the typically small dose of local anesthetic injected, these seizures are generally short-lived and usually not accompanied by cardiac toxicity.

Figure 1. Relative Absorption Rates of Local Anesthetic at Various Locations
Table 1
Factors Increasing Risk of Local Anesthetic Toxicity

- Administration in a site with rapid absorption
- Young age
- Large total dose of local anesthetic
- Renal dysfunction
- Hepatic dysfunction
- Heart failure
- Pregnancy

Absorption from Tissues
When local anesthetic is injected into perineural connective tissue (as in a peripheral nerve block) or the epidural space, the nerve-blocking action is terminated by gradual absorption from the nerve into the systemic circulation. Local anesthetic metabolism has almost no effect on the duration of nerve blocks. Injecting a given dose of local anesthetic into highly vascular tissue will lead to greater plasma drug concentrations than placing the same dose of local anesthetic into a poorly vascularized site. Epinephrine is effective in retarding the rate of absorption of local anesthetic and reducing peak plasma levels of local anesthetic. The effect of clonidine is less clear, with some studies showing increased plasma local anesthetic concentrations when clonidine is included in local anesthetic solutions. Figure 1 shows the relative absorption rates for local anesthetics after injection into various sites.

Tumescent liposuction techniques present a scenario in which large amounts of local anesthetic solution may be absorbed into the systemic circulation in an unpredictable manner. Typically, dilute lidocaine with epinephrine is injected through the liposuction cannula. Dangerously elevated plasma lidocaine levels have been reported when large volumes of injectate are used.

Transcutaneous and Transmucosal Absorption
Local anesthetics can be absorbed across cutaneous and mucosal surfaces, most commonly in the oral, nasal, and tracheo-bronchial mucosa. Topical local anesthetic formulations may contain concentrated local anesthetic and introduce the possibility of delivering many milligrams of drug in a small volume.

Toxic blood concentrations are possible following transcutaneous absorption of local anesthetic creams or gels, particularly in small children. There have been several deaths reported after patients applied large amounts of topical local anesthetic products to provide anesthesia for hair removal procedures.

Risk Factors for Local Anesthetic Toxicity (see Table 1)

Route of Administration
The location of local anesthetic injection affects its absorption rate and peak plasma concentration. The local anesthetic dose may need to be reduced when it is placed into an area with especially rapid absorption, such as the airway or in intercostal nerve blocks, as compared to sites with relatively slow absorption, such as subcutaneous injections or sciatic nerve blocks.

Young Age
Infants, particularly those aged 0-3 months, have reduced concentrations of plasma proteins to which local anesthetics bind, such as AAG. This leads to greater peak levels of free (unbound) local anesthetic after single injections, such as caudal epidural blocks. The unbound form is largely responsible for toxic side effects. Infants also have a reduced capacity to metabolize local anesthetic drugs, with lower plasma clearance rates than adults. This may lead to greater plasma levels when continuous infusions of local anesthetics are given. Due to these factors, both bolus doses and infusion rates of local anesthetics should be reduced in infants.

Local anesthetic toxicity symptoms are caused by the free fraction of the local anesthetic drug (the fraction not bound to plasma proteins). Any condition that reduces plasma protein levels may increase a patient’s risk of local anesthetic toxicity.

Total Dose of Local Anesthetic Administered
All other factors being equal, administering larger doses of local anesthetic will lead to increased plasma concentrations. Patient size should be considered when determining a local anesthetic dose. Of note, it is the product of the concentration and the volume of the local anesthetic solution that is important, not either in isolation; plasma levels of local anesthetic correlate with the total mass of drug given. For example, in most cases 20 ml of a 0.25% ropivacaine solution and 10 ml of a 0.5% ropivacaine solution will produce the same peak plasma concentration.

Presence of Epinephrine
Addition of epinephrine to local anesthetic solutions will normally reduce the rate of absorption and peak plasma levels. Epinephrine 1:400,000 to 1:200,000 (2.5-5 μg/ml) is as effective as more concentrated solutions, while having a reduced risk of epinephrine side effects such as hypertension, tachycardia, and arrhythmias. Less data exist on
the effect of clonidine on local anesthetic absorption, and some studies suggest that clonidine may increase rather than decrease peak plasma levels of local anesthetics.

**Renal Dysfunction**

Patients with uremia have a hyperdynamic circulatory state, and they have a more rapid absorption of local anesthetic, with higher peak plasma levels than in non-uremic patients. Many authors have assumed that these two are causally related. Partially offsetting this effect, uremic patients have greater levels of AAG than non-uremic patients. The AAG tends to bind local anesthetic in the plasma, reducing the concentration of free drug. It has been recommended that a dose reduction of 10-20% be applied when administering regional anesthetics to patients with renal dysfunction.

**Liver Dysfunction**

While mild hepatic dysfunction appears to have a minimal effect on local anesthetic levels, patients with end-stage liver dysfunction (ESLD) may have significantly reduced hepatic clearance rates for local anesthetics. In ESLD, patients have an increased volume of distribution for local anesthetics. This, plus the continued presence of AAG in the plasma even in ESLD, leads to a recommendation that normal doses of local anesthetic may be used for single-dose techniques in patients with liver dysfunction.

Continuous infusions of local anesthetics, however, must be significantly reduced in patients with hepatic dysfunction due to their lower rate of clearance of these drugs. Dose reductions of 10-50% have been suggested based on the severity of the hepatic dysfunction.

**Heart Failure**

Patients with mild, well-controlled heart failure may not require any reduction in local anesthetic dosing. In patients with severe heart failure, however, clearance of local anesthetic drugs may be substantially reduced due to decreased hepatic blood flow and clearance.

**Pregnancy**

Pregnant patients have increased sensitivity to local anesthetics, allowing dose reductions. They also have a reduced degree of protein binding of local anesthetics. Because of the increased sensitivity and higher risk of toxic effects, local anesthetic doses should be reduced in pregnant patients. Other factors, including the reduced spinal CSF volume in pregnancy, lead to an exaggerated spread of spinal and epidural local anesthetics.

### Signs and Symptoms of Local Anesthetic Toxicity

#### CNS Signs and Symptoms

After an accidental intravenous injection, local anesthetics produce signs and symptoms of CNS toxicity at lower doses and earlier than signs and symptoms of cardiovascular toxicity. However, the longer-acting local anesthetics (particularly bupivacaine) may produce toxicity in the CNS and myocardium simultaneously, and there are even reports of cardiovascular toxicity without any CNS side effects. One should also keep in mind that drugs commonly given for sedation (e.g., midazolam or propofol) may increase the seizure threshold.

The effects of elevated systemic levels of local anesthetics are generally divided into early and late signs and symptoms, as shown in Table 2.

#### Cardiovascular Signs and Symptoms

There is evidence from animal studies that bupivacaine and lidocaine cardiotoxicity may take different forms. Lidocaine tends to decrease myocardial contractility and cause peripheral vasodilation, leading to hypotension as a prominent sign of lidocaine toxicity. In animal studies, animals receiving lidocaine to the point of cardiovascular collapse will almost never demonstrate cardiac arrhythmias. In contrast, bupivacaine tends to produce aberrant conduction and arrhythmias (in addition to producing vasodilation and myocardial depression) leading to cardiac arrest.
In general, cardiac signs of local anesthetic intoxication include:
- Transient hypertension and tachycardia (especially if epinephrine is present)
- Hypotension
- Bradycardia
- Cardiac arrhythmias, including premature ventricular contractions, ventricular tachycardia, ventricular fibrillation, pulseless electrical activity, and cardiac arrest.

Techniques for Reducing the Likelihood of Injury from Local Anesthetic Toxicity (see Table 3)

Basic Preparations
Whenever one administers local anesthetic to a patient in doses sufficient to produce toxicity, one should apply appropriate monitors (pulse oximetry and non-invasive blood pressure at a minimum) and be certain that emergency resuscitation drugs and equipment are available. The latter should include airway management devices, an electrical defibrillator, and a lipid emulsion solution.

The long-acting local anesthetics (particularly bupivacaine) may produce toxicity in the CNS and myocardium simultaneously.

Use of Lowest Effective Doses
The lowest practical dose or volume of local anesthetic solution that will produce the intended therapeutic effect should be used. In patients with medical conditions that predispose them to local anesthetic toxicity, one should choose the peripheral nerve block locations and techniques that permit reduced doses of local anesthetic. For example, studies suggest that ultrasound guidance permits the use of significantly lower doses of local anesthetic for supraclavicular blocks compared with nerve stimulator-guided axillary blocks.

Ultrasound Imaging
The use of ultrasound guidance for peripheral nerve blocks may reduce the risk of local anesthetic toxicity in several ways. As mentioned above, the use of ultrasound guidance may allow the use of lower volumes of local anesthetic solutions while maintaining acceptable surgical anesthesia. Unlike nerve stimulator or landmark techniques, the nerve block needle is visualized during positioning and injection, potentially reducing the risk of accidental vascular puncture and potentially reducing the volume needed to detect an accidental intravascular injection.

Fractionated Doses with Multiple Aspirations
Nerve block needles and epidural catheters can move, and an initially well-positioned needle or catheter may become intravascular. Doses of local anesthetic should be divided into aliquots ≤ 5 mL, with aspiration between injections. Always observe the patient’s condition and vital signs during injection because negative aspiration for blood does not guarantee that local anesthetic is not entering a vein. This is especially important if a small gauge needle is utilized.

Slow Injection Speed
Fast, forceful injection of local anesthetic may increase the risk of local anesthetic channeling into veins or other vascular structures. It also may increase the risk of pressure injuries to nerves during peripheral nerve blocks. We normally inject local anesthetic no faster than 5 mL at a time and will usually not administer more than three such increments per minute.

Use of Test Doses
Accurate placement of a needle in the epidural space or perineurally does not guarantee that a catheter passed through it will not enter a vascular structure. Dilute epinephrine (2.5 to 5 µg/mL) may be used as a marker for intravascular injection. Test doses of medication (generally containing epinephrine) should be given when initiating use of an epidural or nerve block catheter and should be repeated if there is any indication that the catheter position may have changed.

<table>
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<tr>
<th>Table 3</th>
<th>Techniques for Reducing Local Anesthetic Toxicity Risk</th>
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<td></td>
<td>• Have appropriate monitors and resuscitation equipment available whenever regional anesthetics are placed</td>
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<td></td>
<td>• Use the lowest effective dose to achieve the desired anesthetic endpoint</td>
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<td></td>
<td>• Use ultrasound guidance when it will facilitate the use of lower doses of local anesthetic</td>
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<td></td>
<td>• Inject local anesthetic slowly, with frequent aspirations</td>
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<td>• Use test doses to verify placement of epidural and nerve block catheters</td>
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<td>• Avoid redosing of local anesthetics whenever possible</td>
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Avoidance of Repeated Dosing

When repeating nerve block injections (e.g., after a failed block) over a short time interval, one must be aware of the cumulative dose of local anesthetic that has been administered.

Treatment of Systemic Local Anesthetic Toxicity
(see Tables 4 and 5)

Treatment for systemic local anesthetic toxicity should be guided by the form of toxicity the patient is experiencing (CNS vs. cardiovascular vs. allergy) and the local anesthetic agent used (bupivacaine and related drugs appear to require a different approach to cardiac resuscitation than the less potent local anesthetics). In general, milder symptoms should be treated with more conservative actions. Nevertheless, mild CNS symptoms may rapidly progress into local anesthetic cardiotoxicity with arrhythmias and cardiac arrest, and one should frequently re-evaluate whether more aggressive therapies are required.

<table>
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<tr>
<th>Table 4 Treatments for Local Anesthetic Toxicity</th>
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<tr>
<td>• Mild neurologic symptoms (tinnitus, lightheadedness, confusion) may be treated with observation and reassurance if they are expected to be brief in duration.</td>
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<td>• Local anesthetic-induced seizures should be treated with small doses of benzodiazepines, barbiturates, or propofol. Phenytoin and fosphenytoin should be avoided.</td>
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<td>• When treating local anesthetic-induced arrhythmias, lidocaine, beta blockers, and calcium channel blockers should be omitted.</td>
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<td>• When local anesthetic cardiotoxicity has been caused by bupivacaine, levobupivacaine, or ropivacaine, doses of epinephrine and vasopressin should be reduced during resuscitation.</td>
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<tr>
<td>• Lipid emulsion therapy should be initiated as soon as possible in cases of local anesthetic cardiotoxicity.</td>
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<td>• Cardiopulmonary bypass should be considered as a treatment of last resort when other therapies have failed.</td>
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Treatment of Local Anesthetic CNS Toxicity

If a patient exhibits mild-to-moderate symptoms and signs of local anesthetic toxicity (e.g., tinnitus, lightheadedness, tremulousness, myoclonic jerks, or confusion) without seizure activity or signs of cardiac toxicity, it may be appropriate to provide conservative treatment in the form of reassurance and mild sedation and anxiolysis with benzodiazepines. A conservative approach is most appropriate with toxicity symptoms due to intermediate-duration local anesthetics (lidocaine, mepivacaine), and when the symptoms are expected to be brief and of limited severity, such as occurs commonly after release of a Bier block.

When the CNS symptoms and signs include loss of consciousness or seizures, standard resuscitation measures should begin including control of the airway and breathing. Medications that are useful for treatment of local anesthetic-induced seizures include benzodiazepines, barbiturates, and propofol. Small doses of these drugs are recommended (e.g., midazolam 2-4 mg, propofol 0.5-1 mg/kg). Propofol doses in particular should be kept low due to propofol's cardiodepressant activity, which may worsen any subsequent local anesthetic cardiotoxicity. Phenytoin and fosphenytoin have generally been avoided in the treatment of local anesthetic toxicity because they share the sodium channel blocking actions of local anesthetics and may potentiate their toxicity.

Lidocaine (and related agents with moderate potency and duration) tends to decrease myocardial contractility and cause peripheral vasodilation, leading to hypotension as a prominent sign of lidocaine toxicity. In contrast, bupivacaine will often produce aberrant conduction and arrhythmias leading to cardiac arrest.

Treatment of Local Anesthetic Cardiac Toxicity: Antiarrhythmics

If unexplained hypotension, bradycardia, or arrhythmias are detected, treatment for suspected local anesthetic cardiac toxicity should begin without delay. Resuscitation of a patient with local anesthetic-induced cardiovascular collapse can require prolonged and extensive resuscitative efforts, particularly with bupivacaine-induced toxicity, and may prove unsuccessful. Treatment may begin with standard advanced cardiac life support (ACLS) methods, but we recommend some minor changes to the standard protocols.

We omit lidocaine from resuscitation of patients with local anesthetic-induced arrhythmias due to its potential to produce an additive effect with the "intoxicating" local anesthetic agent. While we suspect that amiodarone may be a better choice for treatment of ventricular arrhythmias, conclusive data are lacking.
Table 5

Recommended Treatment of Severe Local Anesthetic Toxicity

1. **Call for help.** Initiate a rapid response system ("Code Blue"), if available and adequate assistance cannot otherwise be obtained.

2. **Start ACLS treatment:**
   a. **Airway + Breathing:** Administer 100% oxygen and assure adequate ventilation. Place advanced airway devices, if appropriate, based on the patient and the circumstances.
   b. **Circulation:** Assess blood pressure, heart rate, and cardiac rhythm. Start chest compressions if indicated.
   c. **Do not administer lidocaine, calcium channel blockers, or beta-blockers.**
   d. If the local anesthetic is *bupivacaine, levobupivacaine, ropivacaine, tetracaine, etidocaine, or cocaine:* administer epinephrine (and/or vasopressin) incrementally, in "just sufficient" doses.*
   e. If the local anesthetic is *lidocaine, mepivacaine,* or related agents: epinephrine and vasopressin may be administered in a standard resuscitative fashion with less concern about epinephrine (or vasopressin) interactions with the local anesthetic.*

3. **Treat seizures:**
   a. Benzodiazepines are preferred.
   b. Avoid phenytoin.
   c. Avoid propofol in patients showing signs of cardiotoxicity.

4. **Start lipid emulsion therapy as soon as it is feasible:**
   a. **Use a 20% lipid emulsion (such as Intralipid™).**
   b. **DO NOT USE PROPOFOL IN PLACE OF LIPID EMULSION.**
   c. Give a bolus dose of 1.5 ml/kg IV over one minute (~100 ml for adult patients).
   d. When the bolus dose is complete, start an infusion of 0.25 ml/kg/min (~20 ml/min for adult patients).
   e. Administer up to two additional bolus doses for continued severe cardiovascular symptoms (severe hypotension, ventricular arrhythmias).
   f. Increase the infusion rate to 0.5 ml/kg/min if hypotension persists.
   g. The lipid infusion should be continued for at least 10 minutes after the cardiac rhythm and circulation have stabilized.

5. If the lipid emulsion therapy fails, **contact the nearest facility with cardiopulmonary bypass capability,** and prepare the patient for emergent bypass.

*These recommendations are somewhat speculative. Further research should determine their appropriateness.

Local anesthetic cardiac toxicity will often include depressed contractility. One should avoid administering other negative inotropes in this circumstance.

**Treatment of Local Anesthetic Cardiotoxicity:**
**Epinephrine and/or Vasopressin**

Recent animal studies indicate that in cases of bupivacaine cardiotoxicity, high-dose epinephrine may exacerbate resuscitation efforts and worsen outcomes. Based on the available animal and human data we recommend that epinephrine be administered incrementally (even in resuscitation) starting with 1 μg/kg and basing additional doses on the patient's response, rather than immediately giving 10-15 μg/kg as a bolus.

Whether vasopressin should be used routinely in combination with epinephrine (as some animal results would suggest) or avoided completely due to a propensity for producing pulmonary edema (as it does in rodents) remains highly controversial. We recommend that if vasopressin is used, it should be dosed incrementally rather than as a single large bolus.

**Local anesthetic injections should always be fractionated and frequent aspirations for blood should be performed during injection. Test doses should always be given prior to initiating use of an epidural or peripheral nerve catheter.**

**Treatment of Local Anesthetic Cardiotoxicity:**
**Lipid Emulsion**

Infusion of a 20% lipid emulsion solution (such as Intralipid™) appears to be a surprisingly effective treatment for severe local anesthetic cardiotoxicity. Multiple case reports and animal studies indicate the efficacy of this treatment and its apparent lack of noxious side effects. The evidence supporting lipid emulsion therapy is still being gathered, and animal
Techniques for reducing the risk of injury from local anesthetic drugs have been widely used. The regimen described below has been widely reported, and is based on discussions with investigators and clinicians who have used lidocaine successfully for cardiac toxicity. Boluses and short-term infusions of 20% lipid emulsion appear to have almost no important side effects. In the setting of local anesthetic toxicity, the benefits of fairly liberal lipid emulsion dosing will likely outweigh any risks or side effects.

The mechanism by which 20% lipid emulsion negates local anesthetic toxicity is not clear. Most likely, microscopic lipid micelles provide a large lipid-rich component in the plasma. Local anesthetic molecules bind these lipid micelles, and the micelles likely act as a "lipid sink", effectively sequestering the active drug from the circulation. The local anesthetic drug is then gradually removed from the bloodstream and metabolized as the lipid micelles are broken down by the liver.

Administration of propofol will NOT provide sufficient lipid to assist in resuscitation from local anesthetic-induced cardiovascular toxicity. In order to administer sufficient lipid, cardiotoxic doses of propofol would be required.

Infusion of a 20% lipid emulsion solution (such as Intralipid™) appears to be an effective treatment for severe local anesthetic cardiotoxicity.

Treatment of Local Anesthetic Cardiotoxicity: Cardiopulmonary Bypass

The arrhythmias, hypotension, and cardiac collapse associated with bupivacaine (and associated long-acting local anesthetic agents) toxicity have been reported to be quite refractory to standard ACLS resuscitation methods. Resuscitation efforts may be prolonged, with some patients potentially not responding to standard drugs and therapies. When all other methods have failed, several surviving patients have been emergently placed on cardiopulmonary bypass for cardiopulmonary support and to allow time for clearance of a sufficient quantity of the local anesthetic drugs from target tissues to permit survival. There are case reports of successful resuscitations using cardiopulmonary bypass after all other therapies have failed.

Summary

Local anesthetic toxicity can be a catastrophic complication of regional anesthesia. It may present with CNS symptoms, cardiovascular symptoms, or both. Patients' size, age, and comorbidities may increase their risk of local anesthetic toxicity. Techniques for reducing the risk of injury from local anesthetic toxicity include having appropriate monitors and resuscitation equipment available, using the lowest effective dose of local anesthetic, injecting slowly and with multiple aspirations, and using test doses prior to dosing epidural and peripheral nerve catheters. Treatment of CNS toxicity includes suppression of seizures and supportive care. Treatment of cardiovascular symptoms consists of ACLS techniques (with omission of lidocaine, calcium channel blockers, beta-blockers, and consideration of reduced doses of epinephrine and vasopressin). Lipid emulsion therapy should be initiated as rapidly as possible. In cases of refractory cardiovascular collapse, cardiopulmonary bypass should be instituted.

References


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Weinberg GL: http://lipidrescue.org (Dr. Guy Weinberg's website focusing on lipid emulsion therapy for local anesthetic toxicity, accessed on 26 May 2010)