Optimal Use of Local Anesthetics and Tumescence

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ABSTRACT

It is mandatory for the cosmetic surgeon to use local anesthesia in a safe and effective manner. Current trends to perform more procedures in the office setting necessitate that the surgeon become facile with achieving anesthesia while minimizing complications. In a related theme, the use of tumescence during liposuction deserves respect, despite the ease with which it is applied. Too many unnecessary complications occur as a result of its careless use, resulting in a mortality rate higher than expected for an elective cosmetic procedure. Our goal is to describe the necessary characteristics, pharmacokinetics, physiologic effects, and overall safety guidelines for use of local anesthesia and tumescence. In addition, we highlight risk factors, newer anesthetics, and new methods for pain control.

KEYWORDS: Local anesthesia, safety guidelines, tumescence, liposuction

Safe and effective use of local anesthetic is paramount to the cosmetic plastic surgeon. Not only does it allow adequate anesthesia to perform procedures outside the operative theater, it also supplements postoperative analgesia and minimizes blood loss. Recent economic pressure to shift from operating theater to office-based procedures further drives the increase in local anesthetic utilization. This in turn augments the complications that may arise if special care is not demonstrated with anesthetic use. In addition, the application of tumescence during liposuction at first seems trivial and without consequence. Its apparent simplicity has led to widespread and cavalier use by surgeons as well as non-surgeons. However, mortalities occur more often than they should. A study suggests that the mortality rate may astonishingly approach nearly 1 in 5000 procedures.¹ This illustrates the absolute need for a thorough understanding of both pharmacokinetics and homeostatic alterations that occur with local anesthetic and tumescence use.

LOCAL ANESTHETIC USE

The basic pharmacologic mechanism of all local anesthetics entails interrupting axon depolarization by preventing Na⁺ influx into the neuron. Although it is not completely understood, it is thought that the anesthetics stabilize the membrane at resting potential, reduce the propagation of an excitatory impulse, and therefore block nerve conduction.² Unmyelinated nerve fibers associated with pain are most sensitive and immediately affected, with pressure and tactile sensation succumbing to the anesthetic effects late.

The two basic anesthetic groups are the ester and amide types, as seen in (Fig. 1). Both share similar properties in that they contain both hydrophilic and hydrophobic moieties. The hydrophobic portion is important in allowing the anesthetic to diffuse across the nerve cell membrane, and the hydrophilic portion allows binding with proteins to improve duration of effect. The link between these distinct segments can be either an ester or amide group. Esters were the first local

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Aromatic Group--Intermediate Chain--Amine Group

Figure 1 Basic anesthetic chemical groups.³

anesthetics but have fallen related due to one of their breakdown products. The more popular amides, which are metabolized in the liver, do not encounter this problem. They should, however, be used with caution in patients with liver disease.⁴

Each anesthetic possesses its own characteristic maximum safe dose and duration of clinical longevity, as seen in Table 1. However, note that individual variation can occur with regard to efficacy, duration, and toxicity. These numbers represent only guidelines for treatment.

Local complications of injection may result from direct nerve infiltration, nerve laceration, ecchymosis, hematoma, or infection. Minimizing pain associated with infiltration is accomplished by using smaller needles, preprocedure topical anesthetics, slow injection techniques, and gentle bedside manner. Anesthetic pH also affects pain response. Lidocaine with epinephrine contains acidic preservatives that yield a pH of 3.5 to 4.5, and plain lidocaine has a pH of 6.5 to 6.8. Their use often requires buffering with sodium bicarbonate to minimize the burning sensation.

Table 1 Characteristics of Local Anesthetics⁵

Anesthetic	Duration (min)	
	Without epi	With epi
Amide		
Lidocaine	30–120	60–400
Mepivacaine	30–120	60–400
Bupivacaine	120–240	240–480
Ropivacaine	120–360	N/A
Ester		
Procaine	15–30	30–90
Tetracaine	120–240	240–480
Chloroprocaine	30–60	N/A

N/A, not applicable; epi, epinephrine.

Systemic complications may result from either an allergic response or anesthetic toxicity. Allergic signs including urticaria, angioedema, and anaphylaxis should be treated with antihistamines, subcutaneous epinephrine, steroids, and airway support. Toxicity may result from infiltration of anesthetic directly into a large vessel, exceeding the maximum dose, or impaired metabolism of the drug (liver disease or pseudocholinesterase deficiency). Lidocaine in particular is metabolized in the liver by cytochrome P-450 CYP3A4. When this enzyme is saturated, lidocaine levels increase exponentially. The liver's clearance capacity for lidocaine has been estimated as 250 mg/hr. Known herbal remedies such as garlic, ginseng, and ginkgo may inhibit CYP3A4 and contribute to toxicity. In addition, medications such as midazolam may compete for CYP3A4 metabolism, slowing lidocaine degradation and masking signs of overdose.⁵ Lidocaine serum concentrations usually peak between 8 and 16 hours after infiltration. Therefore, any patient in whom the maximum dose is approached should be observed overnight.

Lidocaine is used intravenously to suppress myocardial automaticity and has a narrow therapeutic window with plasma concentrations between 2 and 5 mg/L. With concentrations between 5 and 9 mg/L, symptoms of central nervous system excitation prevail. These include metallic taste, tinnitus, and circumoral numbness. As levels increase, there is a progression to muscle twitching, shivering, grand mal seizures, and coma. When levels surpass 10 mg/L, hypotension, cardiac arrhythmias, and ultimately cardiorespiratory collapse occur. Treatment includes cardiopulmonary support using standard advanced cardiac life support protocol if necessary.

Bupivacaine is more cardiotoxic than equipotent doses of other local anesthetics, with malignant

Table 2 Maximum Dosage Quick Reference Guide⁹

Anesthetic	Conc. (%)	Maximum Dose (mg/kg)	Simple Equation
Lidocaine	1	5	$0.5 \times \text{wt (in kg)} = \text{max dose (in mL)}$
Lidocaine with epi	1	7	$0.7 \times \text{wt (in kg)} = \text{max dose (in mL)}$
Lidocaine	2	5	$0.25 \times \text{wt (in kg)} = \text{max dose (in mL)}$
Lidocaine with epi	2	7	$0.35 \times \text{wt (in kg)} = \text{max dose (in mL)}$
Marcaine	0.25	2.5	$\text{wt (in kg)} = \text{max dose (in mL)}$
Marcaine with epi	0.25	3.5	$1.4 \times \text{wt (in kg)} = \text{max dose (in mL)}$
Marcaine	0.50	2.5	$0.5 \times \text{wt (in kg)} = \text{max dose (in mL)}$
Marcaine with epi	0.50	3.5	$0.7 \times \text{wt (in kg)} = \text{max dose (in mL)}$
Ropivacaine	0.20	3	$1.5 \times \text{wt (in kg)} = \text{max dose (in mL)}$
Ropivacaine	0.50	3	$0.6 \times \text{wt (in kg)} = \text{max dose (in mL)}$

epi, epinephrine.

arrhythmias, seizures, and total cardiovascular collapse. Bupivacaine has been identified as a “fast in, slow out” type of local anesthetic that maintains a high affinity for myocardial sodium receptors. If cardiac arrest and electrical standstill occur, prolonged resuscitation efforts are required and are usually unsuccessful. Aggressive actions such as placing the overdosed patient on emergent cardiopulmonary bypass have been documented with success.⁶ Ropivacaine is a structural relative of bupivacaine in that it is a pure S-enantiomer. It is known to exhibit less cardiac toxicity while maintaining a long duration.⁷ Ropivacaine also has a vasoconstrictive effect, which allows it to be used without epinephrine, an added advantage. Although not yet widely used because of cost, it is likely to become more popular with time given its advantages and minimal side effects.

All local anesthetics are vasodilators by nature with the exception of cocaine and ropivacaine. Therefore, epinephrine is often used for its vasoconstrictive properties, which decrease anesthetic dissolution into

the blood stream and ultimately allow a higher maximum anesthetic dose. Traditional surgical dogma has always strongly warned against epinephrine use in end arterial organs such as the fingers, penis, nose, and ears. However, a study involving large numbers of digital blocks have refuted this concern as long as the concentration is more dilute than 1:100,000.⁸ One could probably extrapolate this to these other end organs with equally safe results. Systemic consequences of epinephrine use may also occur. They can include tachycardia, hypertension, and chest pain and should be appropriately treated with vasodilating agents (e.g., hydralazine, clonidine, nitroglycerine). Extreme caution should be used with patients taking monoamine oxidase inhibitors as clinically significant hypertension may result.

General administrative guidelines for local anesthetics include using the weakest concentration and smallest volume to obtain adequate anesthesia. In addition, utilizing epinephrine whenever it is not

A

B

Figure 2 Continuous anesthetic delivery system and catheter.¹⁰

contraindicated and waiting the appropriate 5 to 10 minutes for maximum effect are appropriate. Prevention of large-vessel injection by frequent aspiration and incremental injection techniques are useful. Nerve blocks allow a smaller total anesthetic volume for the same effect and should be used when suitable.⁵ A simple reference guide to calculate maximum dosages is provided in Table 2.

In an effort to improve postoperative pain, reduce narcotic consumption, and shift more procedures to an outpatient setting, many surgeons are beginning to utilize continuous local anesthetic infiltration. Their function is based on a simple pump attached to a "soaker-hose" type of catheter (Fig. 2). The attraction to these instruments lies in the ease of their use. The placement is quick and straightforward, the delivery system is simple and relatively infallible, and patients may remove the device at home. Potential complications include pump malfunction, wound infection, and hematoma but are rare. Pacik et al used indwelling catheters to allow self-administered bupivacaine for 200 consecutive augmentation mammoplasty patients with sound results.¹¹ Lu and Fine used anesthetic infusion pumps for breast reduction patients with a significant decrease in oral narcotic usage.¹² Finally, Mentz frequently uses regional infusion pumps during routine abdominoplasty, allowing patients to ambulate sooner and thus probably reducing the incidence of deep venous thrombosis.¹³

TUMESCENCE

The use of epinephrine-containing subcutaneous infiltration is well known for decreasing blood loss associated with suction lipectomy. Furthermore, high-volume infiltration enables the operator to minimize blood loss to as low as 1% of the total aspirate with the superwet and tumescent techniques.¹⁴ As the number of patients undergoing liposuction approaches 200,000 annually, safety becomes the primary issue.¹⁵ Complications, although rare, may be severe and are unacceptable with such an elective procedure. Indeed, liposuction is much safer since the introduction of wetting solutions, but a thorough knowledge of the pharmacologic and physiologic effects of this technique is paramount. Large-volume lipoaspiration (>4–5 L aspirations) has been associated with rare but devastating lidocaine and epinephrine toxicity, fluid overload, pulmonary edema, pulmonary embolism, and hypothermia and should be performed with prudence.

The maximum dose of lidocaine with epinephrine is often cited as 7 mg/kg during routine use. However, when used for tumescence during liposuction, this ceiling is dramatically increased to 35 mg/kg (others report up to 55 mg/kg).^{14,16} This has proved acceptable because plasma concentrations of lidocaine still remain below

toxic levels (<5 mg/L) despite these high infiltrative dosages. The reasons for this tolerance are severalfold. Of course, some of the anesthetic is immediately suctioned after infiltration (approximately 22 to 29%),^{17,18} preventing its systemic absorption. In addition, the extremely dilute concentrations (usually 0.025 to 0.05%) allow the anesthetic to be sequestered totally in the subdermal tissue of the widely undermined skin. Therefore, the lidocaine is absorbed slowly and should not overwhelm the hepatic breakdown rate.¹⁹

The method of infiltration of wetting solution differs from surgeon to surgeon. The basic methods are shown in Table 3 with associated blood loss. Since the advent of superwet and tumescent techniques, megadoses of lidocaine (500 to 1000 mg) have been administered, and many suggest that this trend puts patients at higher risk for lidocaine toxicity. The American Society of Plastic and Reconstructive Surgeons Task Force on Lipoplasty warned that the increase in complications is a result of high-volume tumescence and its accompanying fluid shifts and lidocaine dosages. They stated that removal of more than 5 L of fat demands skill, experience, and postoperative vigilance.^{21,22} Kenkel et al proposed that local tissue levels of lidocaine become greatly diminished by 4 to 8 hours, whereas plasma levels peak at 8 to 16 hours and persist for 36 hours.²³ This suggests that the postoperative analgesic effects are negligible. On that note, some question the need for lidocaine at all in tumescence when general anesthesia is utilized, arguing that the postoperative pain relief is insignificant while the risk of toxicity remains. If lidocaine is to be used, some would suggest removing lidocaine from infiltrate solution beyond 4 L to avoid toxicity.²⁴

Physiologic alterations are also an important aspect of liposuction tumescence. A study using pulmonary artery catheters after high-volume liposuction revealed basic hemodynamic changes that should be noted. Heart rate, cardiac index, and pulmonary artery (PA) pressures increased while mean arterial pressure and systemic vascular resistance decreased. They theorized that cardiac efficiency decreased as a result of higher epinephrine levels. This occurs in the setting of vasodilating effects of general anesthesia.²⁴ Such stresses are well tolerated by a young healthy heart but must be respected in patients with comorbidities.

Fluid overload resulting in pulmonary edema and cardiac failure is certainly a concern in large-volume

Table 3 Infiltrative Techniques²⁰

Technique	Amount Infiltrated	Blood Loss (% of Lipoaspirated)
Dry	None	20–45
Wet	300 mL/region	25
Superwet	1:1 infiltrate to aspirate	3–10
Tumescent	2–3:1 infiltrate to aspirate	1

lipoaspiration. Although no concrete data exist to drive appropriate fluid resuscitation, Trott et al provided recommendations based on a study of 53 consecutive patients. They suggested using the superwet technique followed by 0.25 mL of intravenous crystalloid for each milliliter aspirated over 4000 mL.²⁴ Although most would agree that the use of wetting solutions is safe and superior to dry liposuction, the amount of infiltration (wet, superwet, or tumescent) is controversial. Some would propose that the superwet technique lessens the disposition toward cardiac and pulmonary decompensation that exists with the larger volume tumescent technique.²⁵

Patients are also known to become hypothermic during liposuction, despite use of accepted warming procedures. Postoperative infections, arrhythmias, and coagulation problems may result from this. Every effort should be made to minimize this, at least by using warmed tumescent fluid, covering exposed body areas, using forced air (Bair Hugger), and warming the operating room.²⁶

High-volume tumescence of the abdomen and lower extremities may cause poor venous outflow, resulting in venous stasis and deep venous thrombosis. In addition, the obstruction of venous return may promote the release of tissue clotting factors and, in combination with patient inactivity, culminate in pulmonary thromboemboli.⁵ A poll concerning liposuction-related deaths rated pulmonary embolism as the most common cause of mortality.¹ Mechanical compression devices should be used whenever possible. An encouraging retrospective study of 296 consecutive cases of large-volume liposuction and abdominoplasty revealed zero cases of thromboemboli when low-molecular-weight heparin was administered 1 hour postoperatively through 72 hours.²⁷

Mechanical disruption of fat manifesting as pulmonary fat embolism was described in several postliposuction autopsy specimens. The result of fat emboli syndrome is damage to pulmonary endothelial cells resulting in massive inflammation and poor oxygen exchange, usually resulting in death.²⁸ Although it is true that macroglobulinemia appears with less than 1 L of lipoaspiration, the incidence of clinically significant fat embolism remains quite low.¹ Some speculate that postoperative intravenous fluids should be administered for 24 hours to clear the circulation of this fatty material and prevent this potential complication.²⁹

The use of tumescence should not be trivialized, and a thorough evaluation of the patient's medical background and general health status should be made. Furthermore, patients with even mild cardiovascular risk factors should be considered for the operating room to minimize perioperative complications. Combining this comprehensive approach with a thorough

understanding of the tumescent technique should yield safe, predictable results.

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