Chapter 27: Local Anesthesia in Facial Plastic Surgery

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Local anesthesia plays a vital role in facial plastic and reconstructive surgery. Much of what we do today as surgeons would be impossible were it not for the discovery of local anesthetic agents more than 100 years ago. The initial acquisition of local anesthesia allowed the discipline of surgery the capability of developing procedures that required increasing precision and greater consumption of precious time. Today local anesthetic agents are often taken for granted, and for the most part, the vital role they play in the success of each operation passes unnoticed. Infiltration of local anesthetic agents is often thought of as the disdainful task that must precede the actual performance of an operation. In reality, it should be considered as the step that *begins* the actual procedure. Proper local anesthesia will significantly improve hemostasis, decrease patient discomfort, and effectively reduce surgical morbidity and mortality. For these reasons, the administration of anesthesia should be attended to as carefully as the technical nuances of the surgical procedure itself.

Surgeons who practice facial plastic and reconstructive surgery often find themselves performing the dual role of surgeon and anesthesiologist. Because of this added responsibility, it is imperative that during their training surgeons review basic information pertaining to both local anesthesia and intravenous sedation and become proficient in their administration.

History

The use of local anesthetic agents probably began thousands of years ago somewhere in the Andes Mountains, 3000 to 9000 feet above sea level. There, rugged mountain villagers chewed the leaves of the *Erythroxylon coca* bush for its euphoric properties and its magical ability to increase endurance and strength (Aldrich and Baker, 1976; Gay et al, 1973). Peruvian artifacts dating back to 2500 BC yield evidence of cocaine's widespread usage and its first (perhaps inadvertent) application as an anesthetic agent. Unfortunate Peruvians who became victims of skull fractures, headaches, and mental illnesses underwent the religious rite of trephination by the village shaman, whose cocaine-filled saliva was ritualistically spat into the wound. Whether or not the shaman or victim realized the benefits of topical cocaine, this ritual must have offered vital anesthesia and hemostasis to an otherwise painful and bloody procedure. Because of cocaine, these ancient physicians accomplished a head and neck operation that would elude physicians in Western medicine for thousands of years to come.

It was not until 1860 that Albert Niemann finally isolated the alkaloid cocaine from *Erythroxylon coca* and documented the anesthetic effect it produced when applied to the tongue. One of the biggest proponents of cocaine as a medicinal agent for humans was a then-young Sigmund Freud. He studied the drug diligently and in 1884 produced the widely read *Cocaine Papers,* in which he recommended cocaine for multiple ailments such as morphine addiction, fatigue, and depression (Byck, 1974). Freud then introduced cocaine to Karl Koller, a Viennese ophthalmologist, who began using it as a topical anesthetic agent on the surface of the eye (Koller, 1884). Koller's results were overwhelmingly successful and allowed ophthalmologic surgery to flourish as never before. News of Koller's breakthrough quickly spread throughout the ophthalmology specialty and into the greater surgical community. It was not long before cocaine was successfully used by surgeons as a topical anesthetic for

laryngeal (Semon, 1884) and nasal surgery (Bettman, 1884; Roe, 1887). Sporadic reports appeared describing anesthesia obtained by direct injection of cocaine into the operative field using a hypodermic syringe (Pritchard, 1884; Roe, 1891). Thus began the technique of anesthesia by local infiltration that we use today. In 1885 Halsted reported the first peripheral nerve block using an injection of cocaine at the root of the nerve, a technique that led to the development and practice of regional anesthesia.

Throughout the late 1800s and the early 1900s, cocaine became widely used as an anesthetic agent as well as a household medicinal tonic. Unfortunately, problems with tissue toxicity and psychologic addiction soon became tragically apparent. As was customary in that era of medicine, pioneers in anesthesia often experimented with new drugs on themselves or colleagues. Because of this, the forefathers of anesthesia became the first victims inflicted with the insufferable pain of psychologic and physical addiction to cocaine.

The search for safer agents continued and in 1905 Einhorn produced procaine, the first synthetic anesthetic agent. Like cocaine, this new agent was in the ester classification but did not cause physical or psychologic addiction; however, it still produced significant local tissue toxicity in patients allergic to esters. Finally, in 1948, Lofgren synthesized lidocaine, the first amide anesthetic. Lidocaine caused fewer allergic reactions and adverse responses than the ester compounds, and its success led to widespread synthesis of other amide anesthetics.

The growth of otolaryngology as a specialty was inextricably tied to the growth and development of local anesthesia. Advances in intranasal sinus surgery, endoscopic surgery, otology, head and neck surgery, and facial plastic and reconstructive surgery were made possible because of local anesthesia. As otolaryngologists, we probably administer more local anesthetic agents than any other surgical specialty in medicine. Over the years, a large number of different anesthetic agents have been used for a wide number of procedures. Many of these have fallen into disuse because of adverse reactions or poor efficacy. For this reason, it is important that surgeons remain current with literature and continue to search for newer agents that will minimize complications while maximizing the safety and comfort of our patients.

Mechanism of Action

Local anesthetics work by blocking sodium ion permeability, thus preventing the propagation of an action potential. An ideal local anesthetic agent would temporarily block sensory nerves with a short onset time, have excellent tissue penetration and minimal tissue reaction, and be painless to administer. To achieve some of these goals, various manipulations are made at distinct sites on the anesthetic molecule.

Most anesthetic agents contain three distinct moieties: (1) a tertiary amine that is the hydrophilic portion, (2) an aromatic ring that is the hydrophobic portion, and (3) a connection that is either an amide or ester linkage. An anesthetic compound falls under one of two broad classifications depending on whether it has an ester or an amide linkage. Changes in the hydrophilic portion affect the anesthetic agent's solubility, whereas changes in the hydrophobic portion affect the agent's ability to penetrate nerve membranes. The tertiary amine portion of the agent is found in both the protonated quaternary ammonium salt form and the unprotonated amine base form (Fig. 27-1). It is the uncharged form that is capable of nerve membrane penetration, whereas the charged form is believed to enhance water solubility and

possess the bioactivity that is responsible for blocking sodium channels and preventing nerve conduction. The distribution of these two forms of a particular agent is dictated by the unique pKa (dissociation constant) of each agent. The Pka is the pH at which both forms are found in equal proportions. Most agents have a pKa of 8 to 9, which indicates that 5% to 35% of their molecules will be in the uncharged form at physiologic pH (Covino, 1978). The exact mechanism by which local anesthetic agents block sodium influx and nerve conduction is unknown. Early investigators proposed a "membrane expansion theory", which held that local anesthetic agents caused a nonspecific expansion of the nerve membrane, which in turn constricted sodium channels. The membrane expansion blocked the influx of sodium ions, limiting membrane depolarization and thus preventing the subsequent conduction of action potentials down the nerve. However, more recent investigators believe there is a specific membrane receptor on, in, or near the sodium channel that regulates sodium permeability (Covino, 1978, 1987; Covino and Vassallo, 1976; Hille et al, 1975; Lee, 1979; Ritchie and Greengard, 1966; Strichartz, 1976).

Nerves are classified into three major categories (A, B, and C) based on their relative size. Cutaneous sensory nerves of the face are small, nonmyelinated C fibers that are rapidly blocked by local anesthetic. The B fibers are small myelinated preganglionic sympathetic nerves that are slightly resistant to anesthetic blockade. Finally, A fibers are large myelinated fibers that conduct motor function, proprioception, pressure sensation, and, to a lesser degree, pain and temperature. These large myelinated A fibers are the most resistant to anesthetic agents. Because of these differences, anesthetics produce a sequential block of nerve conduction beginning with the loss of pain and temperature sensitivity, then proprioception, and ending with loss of motor nerve function. With the usual concentrations of local anesthesia, one is able to limit the block to pain fibers without affecting motor function.

Excretion and Elimination

Esters and amides differ dramatically in their metabolic breakdown. The esters are hydrolyzed by plasma pseudocholinesterases (Covino, 1978). Procaine and tetracaine hydrolysis results in para-aminobenzoic acid, which is then excreted in the urine. It is this metabolite, and not the intact anesthetic molecule, that accounts for the unwanted allergic reactions. Cocaine is different and undergoes degradation by plasma esterases to benzoylecgonine and ecgonine (Mistra et al, 1974).

Metabolism of amides is generally more complex than ester degradation and results in multiple metabolites that require excretion. Amides are primarily metabolized by the liver, and only small amounts are excreted unchanged in the urine. Therefore patients with significant hepatic disease or taking drugs that affect hepatic metabolism may reach toxic blood levels of anesthetic after administration of nontoxic anesthetic dosages. Most noted among these drugs that alter hepatic degradation of amides is cimetidine. Lidocaine blood concentrations have been found to be significantly higher in those patients concurrently receiving cimetidine. The effect does not appear to be present in patients receiving ranitidine.

Epinephrine

Most local anesthetic agents are relatively insoluble at physiologic pH and are made water soluble by being first dissolved in weak solutions of hydrochloric acid. In addition, anesthetics often contain epinephrine to promote vasoconstriction and offer hemostasis. These drugs must be stored at a low pH of 3.5 to 5.0 to prevent degradation of light-sensitive epinephrine to adenochrome (DeJong and Cullen, 1963). Unfortunately, a low pH solution produces a great deal of pain when administered by infiltration. But adding an 8.4% sodium bicarbonate buffer to the anesthetic agent in a 1:10 ratio raises the pH to 7.2 to 7.4 (Fig. 27-2) and dramatically reduces the pain caused by the low pH (Arndt et al, 1983). If this is done by the operating surgeon just before infiltration, there is little degradation or precipitation of anesthetic agent or epinephrine. Theoretically, a buffered solution will contain more anesthetic agent in the unprotonated, uncharged form and will promote nerve penetration (Fig. 27-3) and quicken the onset of anesthesia without increasing the duration of nerve blockade (Buckley et al, 1985; Byck, 1974; DiFazio et al, 1986; Hilgier, 1985; Strobel and Bianchi, 1970). This characteristic has been of benefit in regional spinal anesthesia, but for practical purposes this minor increase in nerve penetration that buffered anesthetics appear to have is of little consequence in facial surgery. The converse situation, however, suggests that low pH environments might inhibit nerve penetration and the onset of anesthesia by driving more anesthetic into the protonated, charged form. This may in part explain the difficulty one encounters anesthetizing infected tissue, which is usually acidic. Whether buffered anesthetics are more effective than unbuffered anesthetics in structures where tissue is infected remains unknown.

The addition of epinephrine also increases anesthetic duration by 30% to 50% and decreases systemic toxicity by reducing the rate of absorption. Epinephrine is both an alpha and beta receptor stimulant but is primarily used for the local alpha receptor stimulation on vascular smooth muscle. When using the commercial epinephrine concentrations of 10 microg/mL (1:100.000) or 5 microm/mL (1:200.000), epinephrine toxicity is rarely a problem as long as nontoxic doses of local anesthetic agent are administered. However, other medications or medical conditions may sensitize patients to epinephrine, and these particular situations should be reviewed (see section on adverse reactions). Epinephrine concentrations of 1:100.000 or 1:200.000 are equally effective in achieving hemostasis as are more concentrated forms (Larrabee et al, 1987; Millay et al, 1991; Siegel et al, 1973). In most facial soft tissue operations, the degree of vasoconstriction caused by these concentrations of epinephrine in the local anesthetic does not affect flap survival. The exception is in flaps that are delayed before transposition. In this situation, flap survival *is* adversely affected and anesthetics without epinephrine are recommended (Reinisch and Meyers, 1974; Wu et al, 1978).

Local Anesthetic Agents

Over the past 100 years, numerous local anesthetic agents from both the ester and amide groups have been used in facial plastic and reconstructive surgery. Rather than attempting to review all of these, this discussion will limit itself to those agents currently used on a regular basis (Table 27-1).

Table 27-1. Local anesthetics

Drug	Class	Onset	Duration	Dose
Cocaine	Ester	Rapid	1-2 hr	3 mg/kg
Tetracaine	Ester	Delayed	2-3 hr	1 mg/kg
Lidocaine	Amide	Rapid	1-2 hr	3-5 mg/kg plain 5-7 mg/kg & epinephrine
Bupivacaine	Amide	Delayed	2-4 hr	3 mg/kg.

Esters

Cocaine is still the most frequently used ester because of its rapid onset and excellent hemostatic effect. It is unique among the local anesthetic agents because of its ability to vasoconstrict vessels and to elicit a euphoric effect through stimulation of the CNS. By preventing uptake of norepinephrine from terminal synapses, cocaine not only causes vasoconstriction, but also sensitizes the subject to catecholamines. Excessive stimulation of the sympathetic system can result in multiple untoward cardiovascular responses (tachycardia, blood pressure changes, diaphoresis, and vasospasm). Cocaine is one of the more toxic anesthetics commonly used today, and the recommended dosage is up to 3 mg/kg (= 200 mg for the average adult; Katz and Lee, 1987), although some sources allow for use of even higher dosages (Henderson and Johns, 1977; Verlander and Johns, 1981). It is administered as a 4% to 10% solution, and toxicity correlates with the total amount of drug administered, not the concentration. In most cases, 4 to 5 mL of a 4% solution provides adequate anesthesia for an intranasal procedure. Current commercial preparations of cocaine are usually in one-unit dose vials with an identifying dye marker to avoid overdosing or accidental infiltration. The combination of cocaine with 1:1000 epinephrine to form the so-called "cocaine mud" is dangerous and is no longer recommended because of the combined catecholamine effects of these ingredients (Henderson and Johns, 1977; Mayer, 1924; Ritchie and Green, 1980). Initially, it was believed that the addition of epinephrine might decrease the toxicity of cocaine by decreasing cocaine absorption. However, this does not appear to be true when cocaine or any other anesthetic is applied topically to mucosal surfaces (Campbell and Adriani, 1958; Schenck, 1975).

Unfortunately, significant systemic effects have also been noted with amounts of cocaine well within the clinical dosages. Coronary artery vasospasm, for example, has been documented with intranasal doses as low as 2 mg/kg (Chiu et al, 1986; Isner and Chokshi, 1989; Lange et al, 1989). For these reasons, cocaine must be administered with a great deal of care and one should always be frugal with its usage. Pledgets should be vigorously squeezed to eliminate excess drug before they are applied to the nasal mucosa and should be removed after 5 to 10 minutes to prevent unnecessary absorption of cocaine and drainage of excess drug into the posterior oral pharynx. Because of the problems associated with cocaine administration, a growing number of surgeons have abandoned cocaine and adopted a less toxic combination of lidocaine mixed with either oxymetazoline or phenylephrine.

Tetracaine (Pontocaine) is another topical ester anesthetic used for head and neck anesthesia. It is an extremely potent anesthetic that must be used judiciously to avoid the life-threatening complications documented in the past. Maximum dosage is 1 mg/kg (Katz and

Lee, 1987), which makes this agent approximately six times more toxic than lidocaine with epinephrine. Tetracaine was at one time widely used as a topical agent in endoscopy and tonsillectomy, but dosages were not strictly monitored and there were frequent complications (Mayer, 1924). Today it is used in a limited number of ways with strict dosage schedules. A mixture of tetracaine with adrenaline and cocaine (TAC) can be safely used topically to anesthetize small cutaneous wounds (Cannon et al, 1989; Pryor et al, 1980). Absorption through cutaneous wounds is believed to be slower and safer than absorption through mucous membranes (Adriani and Campbell, 1956; Campbell and Adriani, 1958). Therefore TAC is not recommended for use on nasal, oral, or conjunctival mucosal lacerations. The usual concentration of TAC is 0.5% tetracaine, 1:2000 (0.5 mg/mL) adrenaline, and 11.8% cocaine. However, we currently use a weaker solution with equal success (0.5% tetracaine, 1:2000 adrenaline, and 2% cocaine). The dosage schedules of this weaker solution is listed in the box. This "needle-free" anesthetic has become especially popular for treating pediatric lacerations in which the administration of local anesthesia is often more difficult than the wound repair itself (Cannon et al, 1989; Pryor et al, 1980; White et al, 1986). Although experimental studies in animals have demonstrated that TAC diminishes the cellular defense to bacterial inoculations (Barker et al, 1982), this has not been found to be of clinical significance in humans (Cannon et al, 1989).

Box: TAC dosages

10 kg = 0.5 mL
20 kg = 1.0 mL
30 kg = 1.5 mL
40 kg = 2.0 mL
Maximum dose of cocaine is 2.5 mg/kg.
Maximum dose of tetracaine is 5 mg/kg.
TAC = 0.5% tetracaine, 1:2000 adrenaline, 2% cocaine.

Amides

By far the most frequently used anesthetic agent in soft tissue surgery today is lidocaine (Xylocaine). This agent has a rapid onset, excellent tissue penetration, a 1- to 3-hour duration, and minimal local and systemic toxicity. It is used as 0.5%, 1%, or 2% solution. In most circumstances, lidocaine is used with 1:100.000 or 1:200.000 epinephrine to provide hemostasis, increase anesthetic duration, and reduce systemic toxicity. The toxic dose is 3 mg/kg without epinephrine (approximately 200 mg in the average person) and 5 to 7 mg/kg with epinephrine (approximately 500 mg) (Katz and Lee, 1987). Although it is usually administered by infiltration, lidocaine is also effective as a topical 4% solution when applied to mucosal membranes. Recent investigations have also been conducted on topical administration of lidocaine through intact skin. Lidocaine and prilocaine mixed in an oil-in-water emulsion cream (EMLA) is an experimental drug that can be topically applied to skin to reduce the pain of needle sticks, cannula insertion, and harvesting of skin grafts (Arthur and Covino, 1988). Although effective, it must be applied 45 to 60 minutes before surgery and only results in superficial anesthesia; thus its use in facial surgery is rather limited. At the present time EMLA is not available on the American market.

Bupivacaine (Marcaine) is an amide agent used for longer procedures because of its extended duration of 2 to 4 hours. It is used in concentrations of 0.25% or 0.50% with or without 1:200.000 epinephrine and is generally safe and effective. When compared to lidocaine, it has a slower onset time but has an increased potency and toxicity. The recommended dose is 3.0 to 3.5 mg/kg, or approximately 225 mg (Covino, 1981; Katz and Lee, 1987; Wilkinson, 1989).

Administration Technique

Administration of local anesthetics is generally simple, safe, and straightforward but is usually associated with some degree of temporary discomfort. The discomfort of local anesthetic administration is clearly multifactorial. Initially, there is the pain elicited by the needle puncture itself, then the pain caused by the anesthetic's acidity, and finally the pain caused by hydrostatic pressure. These factors are further accentuated by each patient's anxiety and the sympathomimetic effects of epinephrine. There are a number of simple things that can be done by the surgeon to minimize some of these factors. For small procedures, one can effectively blunt the pain caused by the initial needle puncture by pretreating the area with ice. Next, the pain of infiltration is significantly reduced by neutralizing the acidity of local anesthetics with sodium bicarbonate (Arndt et al, 1983). Finally, the hydrostatic effect is minimized by simply slowing the rate of injection. One must wait 5 to 10 minutes after the injection to allow enough time for effective anesthesia and hemostasis. A good way to avoid beginning the procedure prematurely is to administer the local anesthetic before the surgical prep. Patients appreciate an extra effort by surgeons to minimize their discomfort by attending to these details.

The goal of anesthesia is to temporarily block sensation provided primarily by cranial nerve V and cervical nerves II and III (Fig. 27-4). One should note how the foramina of these nerves relate to the pupil, orbital rim, medial canthus, canine dentition, sternocleidomastoid muscle, and other anatomic landmarks.

For most procedures, use a No. 27 or No. 30 gauge needle, which is 1.5 inch long. It is a common misconception that a short 0.5-inch needle is less painful. In fact, a short needle requires a far greater number of puncture sites and often results in areas that are inadequately anesthetized.

One should use a 3 mL, 5 mL, or 10 mL syringe and avoid the use of larger syringes. Since syringes operate on the same hydraulic principles as a plunger, smaller syringes actually generate more injection pressure and require less work than larger syringes. Using a "control" type of syringe allows one to easily aspirate before injections placed near large vessels of the head and neck. It is important to distribute the anesthetic evenly and precisely to avoid tissue distortion with unnecessary volumes. Advancing the needle to its full depth and injecting as the needle is withdrawn will place a thin line of anesthetic droplets that will not distort the tissue; this practice will also avoid intravascular injection of anesthetic agent.

Rhinoplasty

Once the patient is lightly sedated, the nasal cavity is anesthetized with approximately 3 mL of 4%or 10% cocaine. Each pledget is vigorously squeezed to eliminate excess drug, which would otherwise drain down the posterior pharynx. Infiltration of nasal soft tissue using 1% lidocaine (Zylocaine) with 1:100.000 epinephrine is then initiated. Just before injection, one may choose to administer a short-acting intravenous sedative such as methohexital (Brevital) or propofol (Diprivan). First, the infraorbital nerves (injection sites 1 and 2 in Fig. 27-5, A) are anesthetized through an intranasal or percutaneous approach using approximately 0.5 mL per side. The nasal root and lateral nasal wall are infiltrated with 1 mL per side, keeping the depth of injection near the periosteum. Small amounts of anesthetic may also be injected on the internal surface of the nasal bones to improve anesthetic blockade and hemostasis.

Next, a volume of 1 to 2 mL of anesthetic is used to anesthetize the nasal tip, columella, and ala (injection site 3) through a percutaneous injection. The percutaneous approach affords access to both sides of the nose under direct visualization. A single 1 mL injection is placed across the base of the nose (injection site 4), anesthetizing the remaining portions of the nasal spine, lateral ala, and inferior columella. The average rhinoplasty will require no more than 6 mL of anesthesia (Dingman, 1961; Tardy, 1986).

If septal work is necessary, the septum (injection site 5) is infiltrated with an additional 2 to 3 mL in a submucoperichondrial plane (Fig. 27-5, B). This hydrostatic dissection allows easier elevation of septal flaps and supplies greater anesthesia over the maxillary crest, where manipulation of bone is extremely sensitive.

This anesthetic technique should supply adequate anesthesia for much of the case. During osteotomies an additional intravenous bolus of methohexital or propofol may be necessary to increase the amount of sedation.

Blepharoplasty

Blepharoplasty operations require minimal amounts of local anesthetic. Most surgeons use 1% lidocaine with 1:100.000 epinephrine to anesthetize the eyelids and provide adequate hemostasis. To avoid unnecessary distortion of the delicate eyelid tissue, only 2 mL of anesthetic should be used on each lid. If the eyelid tissue is not distorted with unnecessary amounts of anesthetic, it is easier to determine the precise amount of excess skin and fat that requires resection. Dissection, traction, and cauterization of postseptal orbital fat may require an additional 0.5 mL of anesthetic. This injection is placed at the base of the fat pedicle before its clamping, resection, and cauterization.

Rhytidectomy

The patient is preoperatively sedated with 5 to 10 mg of oral diazepam. Once in the operative suite, the patient is further sedated with midazolam and fentanyl until comfortable. It is important to remain in verbal contact with your patients to reassure them and eliminate unnecessary anxiety. Verbal contact is also a good way to monitor the depth of sedation. A solution of 1% lidocaine with 1:100.000 epinephrine is mixed with 0.5% bupivacaine with

1:200.000 epinephrine in a 1:1 ratio. This mixture has a quick onset and a 2- to 3-hour duration of anesthesia. Some surgeons will also add hyaluronidase in an attempt to increase the rate at which anesthetics penetrate through tissue; the effectiveness of this practice, however, has not yet been investigated in controlled studies. The drug is deposited using a 10 mL control syringe and a No. 25 gauge spinal needle.

The face is sequentially anesthetized in units as the operation progresses (Fig. 27-6). The first unit anesthetized is the submental triangle and the anterior neck. This initial series of injections, which requires approximately 10 to 20 mL of anesthetic, allows for liposuction and surgical access to the anterior platysma. The injection begins in the submentum (injection site 1 in Fig. 27-6) and radiates across the anterior part of the neck to the level of the hyoid, over the angle of the mandible, and across to the sternocleidomastoid muscle.

After liposuction is performed and the anterior platysmal bands have been surgically addressed, one side of the face and neck is anesthetized in the following manner: Injections are begun in the periauricular region (injection site 2) and then extended across the anterior part of the face in a subcutaneous plane. Avoid a deeper injection that could temporarily paralyze the facial nerve and worry both the patient and surgeon. The temporal region is infiltrated from a site just superior to the root of the helix (injection site 3). The postauricular region is injected from a site posterior to the superior aspect of the conchal bowl (injection site 4). Finally, the posterior part of the neck is infiltrated from a site along the posterior margin of the sternocleidomastoid muscle (injection site 5). A total of 20 to 25 mL of anesthetic is used to anesthetize one side of the face. Both sides of the face are not anesthetized simultaneously to avoid complications caused by rapid absorption of large amounts of local anesthetic.

The opposite side is done in an identical manner using the final 20 to 25 mL of local anesthetic. Usually, the opposite side requires less anesthetic since much of the anterior part of the neck was initially anesthetized for the liposuction. A total of 40 to 50 mL of local anesthetic is used for the entire face, and this amount of drug should not result in toxic levels. A 70 kg patient will tolerate up to 490 mg (7 mg/kg) of lidocaine alone over a 30-minute period. If 0.5% lidocaine with 1:100.000 epinephrine is mixed with 0.5% bupivacaine with 1:200.000 epinephrine in a 1:1 ratio, a 50 mL solution will contain 250 mg of lidocaine and 125 mg of bupivacaine. Of one then estimates that bupivacaine (3 mg/kg) is twice as potent as lidocaine with epinephrine (7 mg/kg), a 50 mL volume of this lidocaine:bupivacaine mixture should be roughly equivalent to 500 mg of lidocaine. If this is given over a 2- to 3-hour period, blood concentrations will be well below toxic levels. Trouble-free anesthesia, however, is never guaranteed, and one must always be prepared for complications with every patient.

Adverse Drug Interactions

There are a number of agents that should not be used with certain local anesthetic agents because of adverse reactions. Halogenated inhalation anesthetics used for general anesthesia sensitize the myocardium to epinephrine. Current recommendations are that under general anesthesia with halogenated agents the maximum doses of epinephrine to which patients should be exposed are, with halothane, 4 microg/kg; with isoflurane, 6 microg/kg; and with enflurane, 8 microg/kg.

Patients taking monoamine oxidase (MAO) inhibitors for treatment of depression should avoid epinephrine, which might incite a hypertensive crisis. These patients are especially sensitive to the narcotic meperidine (Demerol). To a lesser degree, tricyclic antidepressants also sensitize patients to epinephrine. If patients are taking these medications, it is best to seek recommendations from their treating physicians as to the possibility of discontinuing the medication perioperatively.

In the past, it was feared that antihypertensive beta blockers would adversely affect the administration of epinephrine by creating an unopposed alpha environment. Initial recommendations were made to discontinue the use of beta blockers in the preoperative period, but problems with rebound hypertension arose. Current recommendations are to continue the use of beta blockers through the preoperative period and into the immediate postoperative period (Cooke, 1985; Lynch, 1980).

Intravenous Sedation

Intravenous sedation has developed over the past 50 years and is now used in a multitude of locations inside and outside the hospital, for numerous minor surgical procedures, and with a plethora of drugs. The goals of sedation in the patient undergoing minor surgical procedures under local anesthesia are (1) patient safety, (2) modification of patient behavior, (3) positive patient response to treatment, and (4) return to pretreatment level of consciousness by the time of discharge.

All local anesthetics are CNS depressants. There are potentially hazardous interactions between local anesthetic agents and sedative/narcotic drugs. The physician who utilizes sedation during surgical procedures must have available proper personnel and equipment to manage any reasonably foreseeable emergency situation. An emergency cart must be readily accessible and should include the necessary drugs and equipment to resuscitate a nonbreathing and unconscious patient. Included in this equipment must be a positive-pressure oxygen delivery system that is capable of administering greater than 90% oxygen ad a 5 L/min flow rate for at least 60 minutes.

Before the surgical procedure the patient should undergo a thorough anesthetic history and physical examination. Particular attention should be paid to reporting of allergies, and current medications and identification of troublesome cardiovascular or respiratory complaints. The physical examination should focus particular attention on the patient's airway: its patency, obvious deformities, and ease of airway management or intubation if that was to become necessary. Patients should not undergo surgical procedures using local anesthesia with intravenous sedation if the following conditions or similar problems are identified: history of severe sleep apnea, poorly controlled seizures, craniofacial anomalies with difficult airway access, cerebral palsy with abnormal swallowing, severe gastroesophageal reflux, chronic hypoxia, or poorly controlled hypertension.

Sedation for surgical procedures with the patient under local anesthesia can be delivered either as deep or conscious sedation. Deep sedation is a controlled state of depressed consciousness or unconsciousness from which the patient is not easily aroused. This may be accompanied by a partial or complete loss of protective airway reflexes and the ability to respond to physical stimulation or verbal command. Conscious sedation is a minimally

depressed level of consciousness that retains the patient's ability to maintain a patent airway independently and continuously and respond appropriately to physical stimulation or verbal command. The separation between these two types of sedation is often narrow; therefore administration of intravenous sedation requires experience and close monitoring. For procedures that require relatively heavy sedation it is recommended that sedation be administered by an anesthesiologist or trained nurse anesthetist.

During sedation the heart rate, respiratory rate, and blood pressure should be continuously monitored and recorded at specific intervals by the anesthesiologist, anesthetist, or a nurse specifically assigned to this task. At a minimum, patients should have continuous ECG and digital pulse oximeter monitoring.

The patient should be discharged only when the following criteria are met: cardiovascular and airway stability are assured, and the patient is alert and can ambulate safely with minimal assistance.

Potential serious complications cannot be ignored; these include idiosyncratic drug reactions, anaphylaxis, respiratory depression, airway obstruction, hypoxemia, aspiration, and cardiac arrhythmias. The risk of death from intravenous sedation is reported to be 1 in 314.000 cases. Proper drug selection and administration are paramount to a successful operation and should be reviewed by the operating surgeon on a regular basis.

Drugs for sedation

The injectable drugs that are chosen for conscious sedation play a significant role in determining the success of the surgical procedure and the speed of the postoperative recovery. Four classifications of intravenous agents are generally employed to provide conscious sedation. These are benzodiazepines, barbiturates, opioids, and the newer alkylphenols (Table 27-2). The goals of conscious sedation are anxiolysis, amnesia, modified consciousness, and analgesia. The clinician must be familiar with each class of drug's specific attributes to attain these objectives. For example, if anxiolysis or amnesia is desired, then a benzodiazepine or similar agent would be the drug of choice. If analgesia is desired, then an opioid should be chosen. In this situation a barbiturate would be relatively contraindicated since barbiturates exhibit antianalgesic properties that might increase the patient's perception of pain.

Benzodiazepines as a group are useful adjunctive drugs to use during minor surgical procedures with the patient under local anesthesia because they possess antianxiety, sedative, amnesic, anticonvulsant, and skeletal muscle relaxant properties. However, one must be aware of possible idiosyncratic reactions, especially present in the very your or elderly, such as increased anxiety, restlessness, and disinhibition. Lorazepam, diazepam, and midazolam are the currently available benzodiazepines. The duration of Lorazepam is too long for it to be useful for the ambulatory patients since Lorazepam can produce prolonged amnesia up to 6 to 8 hours when given parenterally. Diazepam administered in doses of 2.5 to 5.0 mg increments (up to 0.3 mg/kg) can produce conscious sedation adequate for outpatient procedures. Because diazepam is insoluble in water, it is commercially available in solutions of propylene glycol at a pH of 6.6 to 6.9. As a result of this acidity, intramuscular or intravenous injections are often painful. Dilution with water or saline decreases the irritation without altering the potency. Preceding the injection of diazepam with a small dose of

lidocaine (1 mL, 20 mg) will also significantly reduce the irritation. Midazolam is an excellent sedative that is approximately four times more potent than diazepam. In doses of 1 to 2.5 mg (up to 0.1 mg/kg) midazolam can produce sedation appropriate for use in outpatient surgery. In the pediatric population there has been reported success using intranasal midazolam that is absorbed through mucous membranes (Wilson et al, 19880. It is superior to diazepam because of its more rapid onset, shorter duration, and lack of venous irritation. Midazolam causes more amnesia than diazepam, and this may occur with relatively light levels of sedation. Midazolam-induced amnesia may extend antegrade such that patients may not be able to recall presurgical events that occurred while they were fully conscious.

Table 27-2. Sedatives

Drug	Diazepam (Valium)	Midazolam (Versed)	Propofol (Diprivan)	Methohexitol (Brevital)	Fentanyl (Sublimaze)
Class	Benzodiaz	Benzodiaz	Alkylphenol	Barbiturate	Opiate
Water sol	No	Yes	No	Yes	Yes
Pain	Yes thrombophlebitis	No	Yes	Slight	No
Analgesia	No	No	No	No	Yes
Amnestic	Yes	Yes	Minimal	Yes	No
Duration (min)	Long (60-120)	Short (15-30)	Short (5-10)	Short (5-10)	Short

The analgesic properties of short-acting opioids such as fentanyl and alfentanil are especially useful in outpatient surgery. Because of the rapid onset and short duration, these drugs can be closely titrated to alleviate patient discomfort without oversedation. Fentanyl is the most widely used agent because of its relative potency (80 times more potent than morphine; Jaffe and Marin, 1980). It is given in 12.5 to 25 microg doses every 3 to 5 minutines until pain is relieved. Alfentanil is an analog of fentanyl that is less potent (1/5 to 1/10) and has one third the duration of action. It also has less respiratory depression (1/13) than fentanyl (Scamman et al, 1984). Both of these drugs work well in combination with a benzodiazepine such as midazolam for outpatient sedation (Ben-Shlomo et al, 1990).

Barbiturates, as a group, are very useful during procedures requiring conscious sedation. Thiopental is the most commonly used barbiturate in these types of procedures; it has a rapid onset, and its duration is short (5 to 6 minutes) because of its rapid redistribution from brain to muscle. However, detoxification is slow (10% to 15%) and thiopental may cause prolonged postoperative sedation when multiple doses are given. Methohexital is similar to thiopental but appears to have a quicker recovery time, which makes it particularly useful if large or repeated doses of sedation are necessary. Dosages must be carefully titrated to avoid apnea and patients with a history of asthma should probably avoid thiopental because of its tendency to induce histamine release and incite bronchospasm.

Unfortunately, barbiturates do not contain analgesic properties and they may actually be antianalgesic. For this reason, barbiturates are usually administered with an opioid.

The alkylphenols are the newest classification of drugs being used for intravenous sedation. Propofol is the only agent in this group currently available and produces a dose-dependent central nervous system, cardiovascular, and ventilatory depression very similar to the kind caused by the barbiturates. Propofol's onset of action is almost identical to that of thiopental or methohexital; however, recovery is much faster and is associated with few postanesthetic side effects such as nausea, confusion, drowsiness, and restlessness (Doze et al, 1986; MacKenzie and Grant, 1987; Rodrigo and Jonsson, 1989; Valtonen et al, 1989). It appears to have cardiorespiratory depression effects similar to methohexital (White, 1988). Propofol has extremely poor water solubility and is mixed with a lecithin emulsion (10% intralipid) that causes local irritation. Side effects are rare but include thrombophlebitis, involuntary skeletal muscle movements, coughing, and hiccoughs. Because of propofol's short duration of 5 to 10 minutes, prolonged sedation must be performed using an infusion pump and a continuous drip or by repeated boluses at regular intervals.

Complications

Complications from local anesthetics vary from being relatively minor and self limited to ones that are major life-threatening events. Both types must be expeditiously diagnosed and treated. Unfortunately, it can be difficult separating the complications secondary to sedative agents from the complications caused by local anesthetic agents. Minor complications of local infiltration usually consist of local tissue injury secondary to bleeding, infection, or allergic reactions. Minor changes in wound healing have been demonstrated in animal studies but do not appear to be a major problem in the clinical setting (Chvapil et al, 1979). True allergic reactions to anesthetics are, fortunately, rare and are usually caused by anesthetic agents from the ester classification. These agents have PABA-like (para-aminobenzoic acid) metabolites that can be antigenic. Most allergic reactions to ester agents are mild and result in cutaneous hives, but a small number of patients may actually manifest severe anaphylaxis (Ritchie and Greene, 1980). Allergic reactions to amide agents are even less common than reactions to esters and tend to be mild. Patients with an unclear history of allergy to local anesthetics should avoid ester compounds. Those individuals suspicious for a mild allergy to both amide and ester compounds should avoid anesthetics that contain the preservative methylparaben, as it is often this molecule, and not the anesthetic agent itself, that is responsible. Local anesthetic skin-testing techniques are not reliable when selecting an agent for regional anesthesia (Baker and Blackmon, 1985) but may offer reassurance when selecting an agent for minor procedures that only require minute amounts of anesthetic agent. Infiltration with an antihistamine such a diphenhydramine (Benadryl) causes local anesthesia; this technique may be useful in patients who have demonstrated severe reactions to the usual local anesthetics. This substitute works because of a structural similarity with local anesthetic compounds (Ritchie and Greene, 1980).

The more dangerous complications of local anesthetics are caused by their effects on the central nervous and cardiovascular systems. Systemic toxicity is unusual when nontoxic doses are administered but is dependent on the rate of absorption, the PCO_2 (Covino and Vassallo, 1976), and the cardiopulmonary hemodynamics of each individual patient.

Most anesthetic complications are easily avoided if one is careful to limit administration to nontoxic amounts. The vast majority of local anesthetic fatalities are the direct result of drug overdosage and poor anesthetic techniques (Adriani and Campbell, 1856; Mayer, 1924). Direct injection of normally nontoxic dosages into a large vessel can produce toxic symptoms, but this danger is easily avoided with careful infiltration techniques. It should be the surgeon's personal responsibility to check all drug concentrations and dosages and not rely on the surgical staff for this important task.

Most anesthetics initially cause CNS stimulation, which is thought to occur by selective depression of inhibitory neurons (Frank and Saunders, 1963). Initial symptoms of toxicity may present as tinnitus, blurred vision, and dizziness while signs of toxicity may manifest as slurred speech, shivering, muscle twitches, and tremor. Moderately toxic levels result in seizure activity, which is exacerbated by elevated levels of carbon dioxide and acidosis. Agents such as bupivacaine, cocaine, and tetracaine are more potent and consequently have lower seizure thresholds than lidocaine. Increasing anesthetic blood levels into higher toxic levels will result in CNS depression, severe respiratory depression, and death.

Local anesthetic agents can also affect the electrical conductivity of the myocardium. Nontoxic doses may demonstrate antiarrhythmic properties, but toxic doses can result in myocardial depression, decreased cardiac output, and circulatory collapse. The myocardium is also sensitive to epinephrine, which can cause hypertension, tachycardia, ectopic beats, and other deleterious arrhythmias when toxic levels are reached.

It is essential that these signs and symptoms of systemic toxicity be recognized immediately and treated appropriately to avoid major complications or life-threatening injury. Once a minor reaction is recognized, the surgical procedure is terminated, all intravenous sedatives are stopped, intranasal pledgets that contain anesthetics or epinephrine are removed, and the patient is given oxygen by mask. Verbal contact is maintained with the patient to encourage respirations and monitor the depth of sedation. In most circumstances, this is all that is necessary and the procedure is continued after a brief waiting period to ensure the patient is stable and drugs have returned to nontoxic levels.

Elimination of hypoxia and hypercarbia not only prevents CNS injury but also reduces the risk of seizures. If seizures do occur, they are treated effectively with intravenous diazepam or barbiturate. Naloxone is given intravenously to reverse the respiratory depression and sedation caused by narcotics. If the airway is obstructed or the respirations are dangerously depressed despite marked ventilation, the patient must undergo immediate oral intubation with assisted ventilation. Tachycardia and hypertension are treated with small doses of propranolol, whereas hypotension usually responds to intravenous fluids and Trendelenburg positioning. Once stabilized, the patient should continue to undergo close observation and cardiac monitoring until awake. If patients remain unstable or their conditions questionable, a decision should be made to immediately transfer the patient to an intensive care facility for closer monitoring and the appropriate consultation.

Summary

Local anesthetics are a vital part of facial surgery and are responsible for many of our past and present achievements. Anesthetic agents and techniques for their administration have continuously evolved over the past 100 years, but the search for better agents and methods continues. Each new drug has its own characteristic onset time, duration, penetration, and complication rates that must be addressed. A few of these drugs will undoubtedly replace some of the more toxic ones we currently use. It is important for otolaryngologists to remain abreast of these changes and continue to incorporate new drugs or techniques that demonstrate a higher degree of efficacy or safety.