Paparella: Volume IV: Plastic and Reconstructive Surgery and Interrelated Disciplines

Section 2: Disciplines Closely Associated With Otolaryngology

Chapter 21: Anesthesia for Otolaryngologic Procedures

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Anesthesia has been entwined with otolaryngology - head and neck surgery since its inception. The first public demonstration of the use of an anesthetic agent for surgery took place in 1846, when a 19-year-old patient had a tumor removed from his neck under ether anesthesia. Since then, the two specialties have matured together. Improvement in surgical technique has presented the anesthesiologist with ever more challenging patients. Similarly, better anesthetics and monitoring devices have enabled head and neck surgery to become more sophisticated and extensive.

This chapter is not intended to provide a complete review of anesthesia. Rather, it is designated to provide the basic information necessary to comprehend the function of anesthesia and to illuminate the anesthesiologist's point of view when approaching the otolaryngology patient. Our extensive discussion of preoperative assessment is intended to improve communication and in so doing promote patient safety and the avoidance of surgical delays and cancellations. A suggested reference list is provided to direct the reader on specific topics not covered.

The anesthesiologist's responsibilities are manifold. Traditional and highly visible functions, such as rendering the patient oblivious to surgical pain, protecting the vital functions, and avoiding psychological trauma, are now combined with state-of-the-art invasive monitoring, special techniques to facilitate surgery, supervision of the recovery period, and management of postoperative pain.

Preoperative Evaluation

The preoperative visit and evaluation provide the information on which the anesthetic plan is formulated. A cursory review of the chart or an incomplete history and physical examination can spell disaster as surely as a missed intubation or an unexpected surgical hemorrhage. The anesthetic preoperative evaluation is often perceived as compulsive and laborious, but in fact contributes to the reduction of perioperative morbidity and mortality. A routine preoperative evaluation should include the information in the Figure. Although it appears lengthy, it usually can be completed within 20 minutes. However, if current or past hospital records are extensive, it may take longer.

Figure: Anesthetic preoperative evaluation form. (NTG, nitroglycerin; MI, myocardial infarction; PND, paroxysmal nocturnal dyspnea; SOB, shortness of breath; TIA, transient ischemic attack; GC, gonorrhea; JVD, jugular vein distention; TMJ, temporomandibular joint.)

Name _____ Age _____ Sex _____
Diagnosis:
Operation Proposed:
Medical History and Review of Systems:
Cardiovascular: Hypertension (how long, medications, controlled)
Angina (frequency, exertional or rest, NTG)
MI (how many, when, sequelae)
Congestive failure (PND, SOB, orthopnea, edema)
Arrhythmia
Murmur (rheumatic fever)
Respiratory: Asthma (last attack, hospitalizations, steroids)
Emphysema, bronchitis, cold, flu, pneumonia (cough, fever, sputum)
TB (drug regimen)
CNS: Seizures (frequency, last one)
Stroke, TIA (when, residual, evaluation)
Headaches
GI: Hiatal hernia, ulcers, gastric reflux
Hepatic: Cirrhosis, hepatitis
Renal: Stones, infections, failure, dialysis
Endocrine: Diabetes (how long, ketoacidosis, insulin)
Thyroid
Hematologic: Sickle cell, transfusion history, easy bruising or bleeding
Infectious: Hepatitis, AIDS, herpes, syphilis, GC
Surgical and Anesthesia History: Procedure, date, type of anesthetic, associated problems
Activity Tolerance: Using dyspnea and angina as usual end points or activities of daily living
Unrestricted activity
Paced activity without difficulty
Onset with moderate exertion (stairs, walking, sweeping floor)
Onset with mild exertion (going across room)
Onset at rest
Family History: Anesthesia-related problems - apnea, hyperpyrexia, sudden or early death
Allergies: Drugs, tape
Medications: All current medications, past steroid or chemotherapy
Social History: Smoking, alcohol, illicit drugs
NPO Status: Last meal, content
Blood: Ordered
Physical Examination:
Vital signs _____ Height _____ Weight _____
H: Tracheal position, bruits, JVD, range of motion cervical spine and TMJ
E: Icterus, pupillary size

N: Patency and size of nares
T: Oral opening, dentition, degree of visualization
CV: Rhythm, rubs, gallops, murmurs
Respiratory: Barrel chest, breath sound, wheezes, rales
Extremities and Skin: Arthritis, turgor, lesions, vascular access sites
Laboratory Studies:
ASA Classification:
Anesthetic Risks and Plan:

Each operating room has minimal standards for preoperative laboratory testing. Table 1 lists the screening studies that should be performed on asymptomatic, healthy patients scheduled to undergo non-blood loss "peripheral" surgery. Those undergoing more extensive surgery may require additional testing.

Table 1. Preoperative Screening Studies

Age	**Test Indicated**	
	For Men	**For Women**
Under 40	None	Hemoglobin or hematocrit
40-50	Electrocardiogram	Electrocardiogram
	Bun/glucose	Bun/glucose
Over 60	All of the above plus chest x-ray	All of the above plus chest x-ray.

After compiling all the information noted above, the anesthetist categorizes the patient according to the standards of the American Society of Anesthesiologist (ASA) as shown in Table 2. In general, morbidity and mortality rates increase as ASA classification increases. One would not expect a poor anesthetic outcome in an ASA I or II patient undergoing a minor surgical procedure. Unfortunately, ASA classification categorizes only physical status and does not consider other pertinent factors, such as the invasiveness of the surgical procedure, the ease of intubation, or a history of malignant hyperthermia.

Table 2. ASA Classifications

Class	**Definition**
I	Normal healthy patient
II	Patient with mild systemic disease
III	Patient with severe systemic disease, limiting activity but not incapacitated
IV	Patient with incapacitating systemic disease or poorly controlled systemic disease that is a constant threat to life
V	Moribund patient not expected to survive 24 hours with or without surgery
VI	A declared brain-dead patient whose organs are being removed for donation
E	Designation added to above categories denoting emergency procedure, eg, IIIE.

The examination of the cardiovascular system merits the most attention. A positive finding or an abnormal ECG warrants further evaluation because the presence of coronary artery disease is the most lethal patient-related factor in anesthesia-related death.

Anesthesiologists request preoperative consultations from other specialists to address problems they may have neither the expertise nor the time to evaluate. The consultant should assess the patient's problems and differentiate those that can be medically improved preoperatively from those that cannot. Specific opinions are preferable to broad statements (eg, "cleared for spinal only", "oxygenate", "monitor", and "avoid hypotension"). The consultant should not recommend a specific anesthetic technique. A concluding statement to indicate that the patient is in the best possible condition is advisable.

Pharmacology

An attempt to review the pharmacology of anesthesia is beyond the scope of this chapter. In-depth information may be found in a general anesthesia reference text. The authors find the *Physician's Desk Reference (PDR),* the *American Hospital Formulary Service's Drug Information* book by the American Society of Hospital Pharmacists, and *The Pharmacological Basis of Therapeutics* by Goodman and Gilman to be integral with the day-to-day practice and anesthesiology. Cocaine and epinephrine are described individually because they are most frequently used by ENT surgeons during the course of an anesthetic. The authors would like to comment, however, on a few aspects of pharmacology.

Ours is a pill-oriented society. It is not often that we see patients who do not take medications. Even healthy patients often take aspirin, over-the-counter cold or sleep preparations, and, with increasing frequency, illicit drugs. Therefore, it is essential to know all the medications a patient is taking to avoid deleterious interactions with anesthetics. Although life-threatening and sudden-death reactions are rare, they have been noted in the medical literature.

Classic examples of drugs that interact with anesthetics are the monoamine oxidase (MAO) inhibitors and tricyclic antidepressants. MAO inhibitors cause markedly increased levels of circulating catecholamines. Severe reactions and death have resulted when patients on MAO inhibitors were given narcotics, especially meperidine or vasopressors. Therefore, it is usual to discontinue the MAO inhibitors for 2 to 3 weeks before elective surgery. Tricyclic antidepressants also increase catecholamines. Fatal dysrhythmias have resulted from their combination halothane and pancuronium bromide. Alternative anesthetic techniques are available to prevent these complications.

Every drug has its indications and contraindications. Appropriateness in administering a medication (eg, in a patient with a compromised airway) is as important as dosage. Preoperative anesthesia planning begins when it is decided which current medications should be continued. Most often, insulin, antihypertensives, immunosuppressants, antigout medication, ulcer preparations, vasodilators for coronary artery disease, eyedrops, and other "homeostatic" medications are continued. Stool softeners, diuretics, thyroid replacements, and oral

hypoglycemics are often omitted.

Preoperative medications are ordered to achieve several aims. Commonly, antisialogogues, anxiolytics, narcotics, sedatives, or hypnotics are given singly or in combination. Additional medications for specific problems, such as H_2 antagonists, antacids, and gastric motility stimulants for morbid obesity, may be indicated. Lastly, a check must be made before surgery to verify that medications were given as ordered.

Choice of Technique

Five types of anesthesia are given in the operating room (OR): straight local anesthesia, monitored anesthesia care (MAC), conduction anesthesia, major nerve block, and anesthesia general. Table 3 gives the definition of each.

Table 3. Types of Anesthesia Utilized in OR

Straight local	Local anesthetic by surgeon All medications under supervision of surgeon Monitoring by operating room nurses
Monitored anesthesia care (MAC) (formerly local with standby	Local anesthetic by surgeon or anesthesiologist Medications and monitoring by anesthesia personnel
Conduction anesthesia	Spinal, epidural, or caudal block Medications and monitoring by anesthesia personnel
Major nerve block	Brachial plexus, sciatic, ankle are examples Medications and monitoring by anesthesia personnel
General	Rendering patient unconscious with use of intravenous or inhalation agents; medications and monitoring by anesthesia personnel.

In adults, many procedures may be accomplished using local anesthetic agents topically, by infiltration, or by peripheral nerve block, provided the surgeon or anesthesia is capable and gentle. Some patients are not psychologically suited to having any surgical procedure performed with only regional block techniques. They are unable to cooperate because of anxiety, apprehension, or other personality traits. Some of these patients may be made cooperative and placid by the use of small parenteral doses of supplemental drugs such as narcotics or tranquilizers, or both. Children and hyperactive adults require such large doses of supplemental agents that procedures are frequently best done with general anesthesia techniques.

The choice of anesthesia must include factors other than the aforementioned patient factors. These include the site and duration of the proposed procedure and the physical status of the patient. Certainly, if the procedure is extensive in area and duration, it will likely exceed the safe pharmacologic actions of the local anesthetic drugs in relation to their total toxic dose or duration of action. The type of anesthesia employed does not necessarily reflect the relative

difficulty of the case. A tracheostomy in an ASA III or IV patient from the intensive care unit who needs vasoactive support and invasive monitoring under MAC is far more difficult than a mastoidectomy under general anesthesia in a healthy ASA I or II patient.

Local or Regional Anesthesia

Local Anesthetic Agents

The flexibility of local or regional anesthesia lies not only in the variability of techniques but also in the properties of the various local anesthetic agents currently available. There are two major categories of local anesthetics: the esters and the anilides. Although the chemical differences are less relevant to the clinician, there are two major differences worth noting. First, the esters are metabolized in the plasma and liver primarily by the enzyme serum cholinesterase. The anilides are hydrolyzed, via a variety of pathways, within the parenchyma of the liver. Another difference is relative to allergic reactions. Although true allergic reactions to all local anesthetic agents are extremely rare, the well-documented cases thus far reported have occurred with the ester types of drugs.

Clinical Properties

It is now possible to select a drug that will fulfill most of the anesthetic requirements for each of the selected infiltration or nerve block techniques for any individual patient. Some of the clinical properties of these drugs that are of special concern are listed below.

1. *Effectiveness.* The local anesthetic agent but (more important) the concentration of that agent required for effective anesthesia depends on the type of nerve fibers to be blocked, ie, motor, sensory, or autonomic. The motor fibers are the largest and thus require concentrations; the autonomics, being small fibers, may be blocked with relatively weak solutions. With the introduction of the long-acting agent etidocaine, it was realized that some agents act preferentially on specific fibers; in this case, motor fibers, since etidocaine has a stronger affinity for motor blockade than its counterpart bupivacaine. However, since most otolaryngologic procedures do not require major motor blockade, ie, muscle relaxation, there is little reason to use the stronger concentrations of any drug for infiltration and nerve block. Topical anesthesia is not included in this clinical observation.

2. *Speed of onset (latency).* A long latency is not a disadvantage if the surgical procedure is not urgent. The most rapidly acting agents are 2-chloroprocaine, lidocaine, and etidocaine. Speed of onset is also affected by concentration, the stronger solutions being more rapid in their effect. It should be remembered, however, that toxicity also results more rapidly if sufficient volumes of concentrated solutions are used.

3. *Spreading power.* Most agents now in common use have the ability to spread along tissue planes. This property is of considerable importance in regard to the success rate of nerve blocks. Procaine, and to a lesser degree mepivacaine, does not diffuse through tissue as well as

lidocaine, for example, and therefore requires greater accuracy of needle placement for equivalent results.

4. *Duration of action.* Agents such as 2-chloroprocaine have very short durations are of benefit for short procedures on outpatients or when rapid return of normal function is desirable. Conversely, etidocaine and bupivacaine are extremely long-acting and are preferable for lengthy surgical procedures, prolonged post-operative analgesia, or both. Just as increasing the concentration of a solution hastens onset, it also prolongs duration. Again, one must determine if the increase in duration caused by increasing the concentration will be offset by an increased total dose and therefore an approach to toxic levels.

5. *Toxicity.* Although it is generally desirable to use drugs of the lowest possible toxicity, this may be of no importance if the dose used is small. Most nerve blocks of the head and neck require very small volumes, and toxicity from overdose is of little concern. However, when large areas are being anesthetized by infiltration, concentration should be reduced. In terms of "total dose toxicity", a dictum should be the inverse relationship of "the greater the volume, the lower the concentration". In terms of toxicity, local anesthetic drugs have two main advantages compared with most other drugs. First, they are applied at their site of action, allowing a very high local concentration around the nerves to be blocked but a low parenteral concentration. Second, they are, for the most part, administered by medical practitioners who will be in attendance at the time toxicity is likely to occur, it, within the first 1 to 2 minutes if injection is inadvertently made into a blood vessel, or up to 30 minutes after injection if toxicity occurs as a result of peak blood levels. Appropriate supervision and prompt treatment of any untoward reaction are thus possible, as such situations require prompt and informed action to prevent serious sequelae.

6. *Surface activity.* Although most local anesthetics are absorbed from mucous membranes, all are not clinically effective as topical anesthetic agents. The most effective are cocaine, tetracaine, dibucaine, and lidocaine. The most common exceptions are procaine, chloroprocaine, and mepivacaine, which have no clinical topical activity.

Epinephrine

Epinephrine is used in ENT surgery to improve hemostasis and decrease the absorption of local anesthetics. This decreased absorption prolongs the duration of action of the local anesthetic and may decrease toxic reactions.

Four questions commonly arise regarding epinephrine use in the operating room: What dose should be used? How do I mix it? Does the use of general anesthesia alter dosage? For what patients is epinephrine safe to use? We will attempt to answer these questions.

There has been some controversy in the past regarding "safe" dosage and "safe" concentration. Most authors and clinicians agree that a total dose of 200 microg in a concentration not greater than 1:200.000 is advisable. Some reference texts cite 250 microg as

the maximal safe dose, but it has been noted that increased dosage (> 200 microg) and/or concentration (> 1:200.000), is associated with a greater incidence of toxic sequelae. Concentrations greater than 1:100.000 offer no better hemostatic effects and carry the risk of ischemic tissue injury. The signs of toxic blood levels of epinephrine are palpitations, headache, hypertension, tachycardia, and arrhythmia.

The desired dosage and concentration can be mixed utilizing the following information:

1. The maximal recommended total epinephrine dose is 200 microg, which approximates 1.5 microg per kg.

2. A 1:200.000 concentration contains 5 microg epinephrine per mL of solution.

3. A 1:100.000 concentration contains 10 microg epinephrine per mL of solution.

4. Epinephrine is available in 1-mL ampules (1:1000) containing 1 mg per mL.

To achieve a 1:200.000 concentration with a total dose of 200 microg (0.2 mL of 1:1000 epinephrine), a total local anesthetic volume of 40 mL is used. If larger volumes of local anesthetic are needed, the epinephrine concentration will decrease. As noted above, a 1:100.000 concentration contains twice the dosage of epinephrine per mL as in a 1:200.000 concentration. Therefore, to achieve a 1:200.000 concentration, with a total dose of 200 microg, 0.2 mL of 1:1000 is added to 20 mL of local anesthetic.

Some volatile anesthetics may accentuate toxic reactions to epinephrine. Johnston and colleagues studied the dosage of epinephrine needed to produce cardiovascular symptoms in patients treated with various anesthetics. The dose producing a positive response of three premature ventricular contractions in 50 per cent of the patients (ED_{50}) is found in Table 4. The doses recommended above are well below those found to induce arrhythmias.

Table 4. Dosages of Epinephrine During Inhalation Anesthesia

Anesthetic Agent	Epinephrine Dose
Halothane	2.1 microg/kg
Halothane	3.7 microg/kg (epinephrine/lidocaine mixture)
Enflurane	10.9 microg/kg
Isoflurane	6.7 microg/kg.

Early reports of "halothane" hepatitis and the discovery of other agents have reduced halothane use in the USA. However, indications for the preferential use of halothane (especially in children and asthmatics) necessitates knowledge of specific dosage regimens for epinephrine during halothane anesthesia. The recommendations of several authors are noted in Table 5.

Table 5. Dosages of Epinephrine During Halothane Anesthesia

Author	Dose
Katz and Katz (1966)	1 microg/kg
Johnston et al (1976)	1 microg/kg
Moore (1981)	10 mL of 1:100.000 over 10 minutes (approximately 1.5 microg/kg)
Katz and Epstein (1968)	30 mL of 1:100.000 over 1 hour.

Patients with hypertension, a history of arrhythmia, thyrotoxicosis, coronary artery disease, or diabetes and elderly or debilitated individuals generally are not candidates for local vasoconstriction with epinephrine. Phenylephrine, an agent with primary alpha-adrenergic effects and minimal beta-adrenergic effects (epinephrine is both alpha and beta) is recommended as an alternative. A dose of 50 microg per mL of phenylephrine is equipotent to 5 microg per mL of epinephrine. A total phenylephrine dose of 2 to 4 mg is recommended by several authors. Phenylephrine is usually available as a 1 per cent solution containing 10 mg per mL. The maximal total recommended dose of 2 mg is achieved by adding 0.2 mL of 1 per cent solution to the total volume of local anesthetic to be used.

Cocaine

No drug in the pharmacopedia arouses as much concern and controversy today as cocaine. Some early investigators (Halstead) found themselves, like many today, addicted to this most euphoric of drugs. As the medical community contemplates its use in patient care, the world watches cocaine, a "new" and fashionable drug, become a social plague.

Despite the controversy regarding the use of cocaine as a topical anesthetic, many surgeons and anesthesiologists regard it as the near-ideal local anesthetic. Synthetic local anesthetics are all vasodilators that provide variable properties of topical anesthesia. Cocaine, the only naturally occurring local anesthetic, combines the properties of superb topical anesthesia with marked vasoconstriction. This combination remains unsurpassed in reducing bleeding in the highly vascular areas of the head and neck. Accordingly, the singular accepted indication for the use of cocaine is as a topical anesthetic and vasoconstrictor of mucous membranes, specifically the nose, pharynx, larynx, and lower respiratory tract.

Cocaine is an ester of benzoic acid that is hydrolyzed by plasma cholinesterase. Its breakdown products may induce an allergic reaction in a very few patients. It is not a stable compound and does not withstand heat autoclaving. It is available in solutions of the hydrochloride, varying in strength from 1 to 10 per cent. Concentrations of 1 per cent are suitable for corneal anesthesia. Concentrations of 4 to 5 per cent are adequate for anesthesia of the mucous membranes in the nose, mouth, and throat. Although further increasing the concentration to 10 per cent decreases the onset time of anesthesia from 4 to 2 minutes, it also doubles the risk of toxicity. It is seldom that a gain of 2 minutes justifies that risk. The duration of anesthesia is

approximately 60 minutes.

Cocaine inhibits the reuptake of epinephrine and norepinephrine by adrenergic nerve endings, thereby potentiating the response to exogenous and endogenous catecholamines. This action primarily affects the central nervous (CNS) and cardiovascular (CVS) systems and produces a hypermetabolic state.

The effect of cocaine on the CNS is biphasic. As inhibitory neurons are affected first, stimulation and euphoria manifest. Increasing blood levels may produce anxiety, headache, restlessness, tremor, vomiting, mydriasis, hyperreflexia, and clonic seizures. Thereafter, global cerebral depression ensues. Depression of the respiratory centers of the medulla causes irregular respiration, apnea, and subsequent coma.

Small systemic doses of cocaine are vagotonic, producing a diminution in heart rate. Increasing blood concentrations cause tachycardia, hypertension, ventricular tachycardia, and direct cardiac muscle depression, any of which may cause sudden death.

Cocaine has no effect when applied to intact skin but is very effective on mucous membranes and denuded epithelium. Its greatest topical absorption is from the trachea, followed by the pharynx, esophagus, and stomach. Donlon showed a 5- to 10-minute latency period after topical application to the nasal mucosa. Repeated small doses of 4 per cent cocaine appear to be safer than a large single dose. It is metabolized (via hydrolysis) by plasma pseudocholinesterase, but its pharmacokinetics are relatively unknown. The peak effect is at 1 hour and persists for 4 to 6 hours.

No study examining the toxic level of cocaine in humans exists. We are guided by our clinical experience and anecdotal case reports. The general use of 3 mg per kg (200 mg) of topical cocaine as the maximal safe dose is not based on any scientific knowledge. In fact, Johns and colleagues reported that small doses of topical cocaine were associated with fatalities, whereas doses of up to 1 gm were used without incident. Barash reported that 1.5 mg per kg of cocaine applied to the nasal mucosa during general anesthesia does not cause sufficient sympathetic stimulation to alter hemodynamics.

Cocaine should not be used in patients with hypertension, coronary artery disease, dysrhythmias, decreased metabolic reserve (in the elderly or debilitated), or pseudocholinesterase deficiency. In addition, cocaine interaction with MAO inhibitors, tricyclic antidepressants, guanethidine, reserpine, alpha-methyl-dopa, and the inhalation anesthetic halothane may produce severe, sometimes fatal, cardiac dysfunction. Echothiophate eye drops, severe liver failure, and atypical pseudocholinesterase conditions affect the metabolism of cocaine, and may increase its duration of action or precipitate a toxic reaction with a "usual" dose.

The concomitant use of cocaine and epinephrine has been reported by several authors to provide no improvement in drug performance. The mechanism of action of cocaine prolongs the duration of action of both endogenous and exogenous catecholamines. The administration of

epinephrine, either concomitantly with or subsequent to that of cocaine, may lead to a severe cardiovascular reaction or collapse. As such, its combined use has been condemned by some, although approved by others.

The toxic effects of cocaine, as previously noted, should be treated in a straightforward manner. Airway, breathing, and circulation (the ABC's of CPR) are the first considerations, together with administration of 100 per cent oxygen, propranolol (1 to 5 mg) for tachyarrhythmias, diazepam (0.1 to 0.2 mg per kg), and usual seizure precautions.

The authors conclude, like others, that when cocaine is used in the proper concentration and volume in a carefully screened patient group, it can be effective and safe drug. Ironically, rising concern over the abuse of drugs by health care personnel, combined with difficulty in OR drug control and accountability, may make cocaine levels available for use in the OR than it is to the general public.

Lidocaine

Lidocaine is one of the amide group of local anesthetic agents that is hydrolyzed in the liver. It is an extremely stable compound and may be heat autoclaved. As mentioned previously, it has excellent spreading capabilities.

A 4 per cent solution of lidocaine is available for topical use and provides effective anesthesia of the nose, oropharynx, and tracheobronchial tree. Weaker concentrations (2 per cent) may be used for topical anesthesia, but are less effective. The 2 per cent concentration is prepared in viscous form for orally administered topical anesthesia. The preselected volume is usually retained in the oropharynx and mouth for as long as possible and then discarded. Similarly, ointments and gels containing 2.5 to 5 per cent lidocaine may be used on tubes and instruments to provide topical anesthesia. The onset of topical anesthesia occurs within 5 to 10 minutes and, like the onset for all anesthetic agents, may be reduced by increasing the concentration.

Infiltration and minor nerve block may be rapidly and effectively accomplished with 0.5 to 1 per cent lidocaine, plain or with epinephrine added. Onset of anesthesia occurs in 2 to 5 minutes and lasts 1 to 3 hours depending on the concentration and presence of epinephrine. The recommended safe dosage range is 5 mg of plain solution and 7 mg per kg of epinephrine-containing solutions. The recommendation not to exceed 500 mg of the latter solution, however, is still good advice. Many variables, such as multiple sites of injection, hypercarbia, hypovolemia, and liver disease, lower the toxic threshold and should always be kept in mind when calculating the total dose for any given patient.

Bupivacaine and Etidocaine

Bupivacaine is the first of two long-acting amide agents introduced in the past few decades (etidocaine being the other). Both drugs are highly protein bound and lipophilic. Their

outstanding clinical feature is a very long duration of action (6 to 8 hours for peripheral nerve blocks). Bupivacaine is approximately four times more potent than lidocaine, although this is offset by increased toxicity. Concentrations as low as 0.125 per cent may be used for infiltration when extremely large volumes of solution are required. The usual concentrations for infiltration or minor nerve block, however, are 0.25 to 0.5 per cent. Onset of anesthesia occurs in 5 to 10 minutes and lasts 3 to 4 hours for plain solutions, or 6 or more hours if epinephrine-containing solutions of 0.5 per cent are used for minor nerve blocks.

There are both advantages and disadvantages to be considered when using the long-acting local anesthetic agents. After peripheral nerve block, individual patients may have residual anesthesia or hypesthesia for 18 to 36 hours. This is not common, nor is it of consequence except in relation to the risk of injury to insensitive structures. Conversely, prolonged analgesia without the need for parenteral agents may be desirable and exploited in using these agents. The current recommended dosage for bupivacaine is 200 mg of the plain solution and 250 mg of solutions containing epinephrine.

Etidocaine too is effective in the 0.25 and 0.5 per cent concentrations for infiltration and nerve block, respectively. Its maximal recommended dose is 400 mg, providing an additional margin of safety in comparison with bupivacaine. Etidocaine is a more potent blocker of motor nerve fibers than bupivacaine for those blocks requiring muscle relaxation. In the aforementioned concentrations, its sensory block is less potent than that of bupivacaine, so duration will be reduced to the 4- to 6-hour range.

Other Local Anesthetic Agents

A number of other local anesthetic agents are available that can be just as effective as the examples cited. The compound *2-chloroprocaine* should be remembered as an extremely rapid-acting agent of very short duration - less than 60 minutes. Because it is an ester that is rapidly broken down by serum cholinesterase, it is perhaps the safest local anesthetic available. *Mepivacaine* is advantageous as an intermediate agent. It is of longer duration than lidocaine (2 to 3 hours) but of shorter duration than the very long-acting agents. Because of its poor tissue-spreading properties, it is slower in onset. Mepivacaine has inherent mild vasoconstrictor properties and is less affected by the addition of epinephrine. *Tetracaine* is said to be as effective topically in a 1 per cent solution as a 10 per cent cocaine solution. It does not have the vasoconstrictor action of cocaine, however. The maximal recommended dose for topical anesthesia is 100 mg.

Equipment

Although a great degree of flexibility is available in the selection of equipment for regional block procedures, a few basic principles should be observed. The smallest, shortest needle required to reach the neural target is the needle of choice. Specific needles are mentioned in the subsequent discussion of nerve block techniques. Disposable needles "spur" easily when contacting bone. Short bevel needles or sharp stainless steel needles are therefore preferred. A

25-gauge disposable spinal needle with stylet removed is excellent for extensive subcutaneous infiltration. With all needles, care should be exercised not to insert the entire needle under the skin surface. Needles do occasionally break at the hub, and recovery is most difficult if the entire shaft is buried in subcutaneous tissue.

A variety of disposable trays are available for infiltration and minor nerve block. Busy practitioners using regional anesthesia may prefer to assemble their own, but for less frequent use the disposables do offer guaranteed sterility and rapid access to the basic equipment. A thorough antiseptic preparation of the area to be blocked should be accomplished. Opened local anesthetic agents should always be kept well away from all prep solutions or other caustic agents. The maximal safe dose of the agent to be used should be measured out before inception of the block, so that there is no risk of exceeding this dosage and precipitating a toxic reaction.

Nerve Block Techniques

Because of the very compact anatomy of the head and neck and the close relationship of cranial and cervical nerves to many vital structures, meticulous placement of the needle and small discrete doses of anesthetic agent are usually required for accurate and safe regional anesthesia in this area.

The sensory supply of the face, head, and neck is primarily from the trigeminal nerve (cranial nerve V) and the cervical plexus. In addition, the glossopharyngeal (cranial IX) and vagus (cranial X) nerves supply the pharynx and larynx.

Some of the more common nerve blocks used for otolaryngologic procedures are described below.

Ophthalmic Nerve

The major branches of the first division of the trigeminal nerve are the supraorbital and supratrochlear nerves. With its intracranial and intraorbital course, the trunk of the ophthalmic nerve does not lend itself well to regional anesthesia.

Supraorbital Nerve. The supraorbital branch, like the infraorbital and mental nerves, lies in the same plane as that of the pupil when the patient is looking straight ahead. A block of this nerve is best accomplished above the eyebrow after the nerve has emerged from the orbit through the supraorbital notch. The supraorbital notch is easily palpated, and at that point 1 to 3 mL of local anesthetic is infiltrated through the skin and over the frontal bone.

Supratrochlear Nerve. The supratrochlear nerve emerges from the superomedial angle of the orbit and runs up onto the forehead parallel to the supraorbital nerve and one fingerbreadth or so medial to it. It may also be located half the distance between the supraorbital notch and the lateral margin of the root of the nose. There are two approaches to blockade of this nerve. It may be blocked as it emerges from the orbit, either by injection of 1 to 3 mL of local anesthetic at

that site or merely by medial extension of the infiltration of the supraorbital nerve.

These two nerve blocks, in combination, provide excellent analgesia of the forehead and may be used for minor surgical procedures, such as repair of lacerations.

Anterior and Posterior Ethmoidal Nerves. These too are terminal branches of the ophthalmic division. The anterior ethmoidal nerve supplies the mucous membrane of the upper and anterior part of the nasal septum, the lateral wall of the nose, and the frontal and anterior ethmoidal sinuses. Its terminal branch, the external nasal nerve, supplies the skin of the ala and nasal tip. The posterior ethmoidal and infratrochlear nerves branch off together; the former supplies the posterior ethmoidal and sphenoidal sinuses, and the latter supplies the skin of the lids and side of the nose, the conjunctiva, and the lacrimal sac.

The anterior ethmoidal nerve is ordinarily anesthetized topically on the lateral nasal wall, high in the dome and superior to the anterior end of the middle turbinate. The external nasal nerve may be blocked subcutaneously at the junction of the nasal base and upper lateral cartilage midway between the nasal dorsum and the base of the nose. The posterior ethmoidal nerve may be blocked externally by extending the external nasal nerve block procedure. Advancing the needle 1 to 1.5 cm allows infiltration of 1 to 3 mL of local anesthetic over the nose. Since the optic nerve lies at an average 4-cm depth from the superior orbital rim, the needle depth at no point should exceed 3 cm. The infratrochlear nerve is blocked by depositing 1 to 2 mL of local anesthetic midway between the inner canthus and the dorsum of the nose. The point of injection is on a plane that is a horizontal continuation of the medial canthus.

Maxillary Nerve

The maxillary nerve, the second division of the trigeminal nerve, innervates the skin and mucosa of the midface, including the maxilla, palate, and teeth. Indications for use of this nerve block are extensive midfacial procedures such as partial maxillectomy; extensive antral surgery, including the cryosurgical approach to the internal maxillary artery; palatal surgery; repair of midface fractures; and extensive dentoalveolar surgery.

There are two major approaches to blockade of the maxillary nerve, one externally via the infratemporal fossa and the other via an intraoral approach. With the infratemporal approach, the needle is inserted through the skin at a point below the midpoint of the zygomatic arch overlying the coronoid notch of the mandible. The notch in the mandible is usually palpable as the patient opens and closes the jaw. After being passed through the coronoid notch of the mandible, the needle is directed medially until it reaches the medial wall of the infratemporal fossa, at which time it will strike the lateral surface of the lateral pterygoid plate, usually at a depth of about 4 to 5 cm. The needle is then "walked" anteriorly off the lateral pterygoid plate until it enters the pterygopalatine fossa, at which point it is advanced an additional 1 to 3 cm into the fossa. Usually, a paresthesia is not sought but may be obtained, and 3 to 5 mL of local anesthetic solution is injected.

The intraoral route consists of anesthetizing the labiobuccal sulcus above the posterior to the third molar. A 7-cm, 25-gauge needle is bent to an angle of 60 degrees approximately 4 cm up the shaft. The needle is passed superomedially, with slight posterior inclination, along the posterior margin of the maxillary tuberosity, and advanced through the sphenomaxillary fissure into the pterygopalatine fossa. (Practice on a skull will serve to fix the spatial relationships in the anesthetist's mind.) If the needle meets bony obstruction prior to its complete entrance of 3 cm it likely has struck the base of the lateral pterygoid plate and should be partially removed and advanced in a more vertical path. After careful aspiration, 2 to 3 mL of anesthetic solution is slowly deposited.

Infraorbital Nerve. The infraorbital nerve constitutes a direct continuation of the maxillary nerve and supplies the tissue of the lower eyelid, anterior cheek, upper lip, and nasolabial fold, including gingiva and mucosa above the nasal vestibule. Blockade of this nerve also induces anesthesia of the maxillary incisors, cuspids, bicuspids, and mesobuccal root of the first molar on the injected side. This nerve too may be blocked via extra- or intraoral approaches.

Either approach utilizes the location of the infraorbital notch by having the patient gaze directly ahead and drawing an imaginary straight line passing through the supraorbital notch, the pupil of the eye, and the bicuspids. Extraorally, a skin wheal is made approximately 1 cm below the infraorbital rim directly below the midpoint of the pupil (where the imaginary line passes through the infraorbital notch). The needle is advanced in a slight upward position to the bone, and 2 to 3 mL of local anesthetic is injected. There is no need to enter the canal, since this causes pain or paresthesias, or both.

Intraorally, the surface landmarks used to locate the infraorbital notch are the same. The depression of the notch is located and fixed with the index finger while the thumb of the same hand retracts the lip upward and outward. A small wheal is made in the labiobuccal fold. The needle is aligned with the aforementioned imaginary line toward the notch under the palpating index finger. The distance is usually 1.5 to 2 cm. After careful aspiration, 2 to 3 mL of anesthetic solution is injected. At all times, care must be taken to prevent the needle entering the orbital cavity.

Nerves to the Palate (Anterior Palatine and Nasopalatine). The pterygopalatine (sphenopalatine) nerves unite the sphenopalatine ganglion with the maxillary nerve (see Chap. 5). Blockade of the major palatine branch, ie, the anterior palatine (or greater palatine) nerve, and the major nasal branch, ie, the nasopalatine (or long sphenopalatine) nerve, may be accomplished for intraoral anesthesia as follows:

Anterior Palatine (Greater Palatine) Nerve. The approach to the nerve at the greater palatine foramen is best made from the opposite side. A 2.5-cm, 25-gauge needle is inserted through a wheal at a right angle to the curvature of the palate between the second and third maxillary molars, approximately 5 to 10 mm above the gingival margin. When bone is reached, the needle is withdrawn 1 mm, and less than 0.5 mL of local anesthetic is deposited slowly. Palatal tissues are tense, and injections are quite painful if not made slowly. This nerve block

provides anesthesia of the posterior portion of the hard palate and its overlying structures up to the first bicuspid on the injected side.

Nasopalatine (Long Sphenopalatine) Nerve. Blockage of this nerve is accomplished as it emerges into the anterior hard palate through the incisive canal underlying the incisive papilla. A 1-cm 25-gauge needle is introduced into the incisive papilla at right angles to the central incisors. Resistance by the labial intraseptal plate is felt, and a small amount (less than 0.5 mL) of local anesthetic solution is slowly deposited. This allows a less painful advance of the needle into the incisive foramen, where another 0.5 mL of local anesthetic is injected. Blockade of this nerve should provide anesthesia of the anterior hard palate and its overlying structures back to the bicuspid area, where branches of the anterior palatine nerve overlap it. Blockade of both nerves should provide anesthesia of the entire palate.

Mandibular Nerve

The mandibular nerve, the third division of the trigeminal nerve, innervates the skin over the lower jaw, the superior two-thirds of the anterior surface of the auricle, and a strip of skin often extending up over the temporal area to the vertex of the skull. If sufficient concentration of local anesthetic is injected, motor blockade of the ipsilateral muscles of mastication will also result. The otic ganglion lies in intimate proximity posteriorly, just below the foramen ovale. Coincident blockade of the otic ganglion, which supplies secretomotor fibers to the parotid gland, results in impaired secretion from this gland.

The *extraoral* approach to blockade of the mandibular nerve is at the infratemporal fossa. The surface landmarks are identical to those used in the previously mentioned maxillary nerve block. The 6-cm needle is introduced through the skin wheal below the midpoint of the zygomatic arch, and passes through the coronoid notch of the mandible directed medially across the infratemporal fossa until it impinges on the bony medial wall, ie, the lateral aspects of the lateral pterygoid plate. At this point, the direction differs from the approach to the maxillary nerve because the needle must be "walked" posteriorly off the lateral pterygoid plate until a paresthesia into the lower lip area is obtained. Once the needle leaves the posterior aspect of the lateral pterygoid plate, it can pierce the attached superior constrictor muscle and enter the pharynx, causing the patient to swallow or spit out the anesthetic if injection is performed.

Inferior Alveolar Nerve. The continuing mandibular nerve proceeds downward, reaching the pterygomandibular space, where it lies between the sphenomandibular ligament and the medial surface of the ramus. At this point, it enters the mandibular foramen, where it becomes the inferior alveolar nerve, which can be blocked via the intraoral approach. Injection into this nerve provides anesthesia of the body of that mandible and an inferior portion of the ramus. It also anesthetizes the mandibular teeth, as well as the mucous membrane and underlying tissues anterior to the first mandibular molar. This includes the lower lip, both skin and mucosa.

The technique involves placement of a 4- to 5-cm, 25-gauge needle, the final position of which lies superior to the inferior alveolar nerve and blood vessels, superior to the insertion of

the internal pterygoid muscle, and superior to the nerve, while being medial to the inner ramus of the mandible and the sphenomandibular ligament. In order to achieve such a position, anatomic landmarks such as the anterior border of the ramus, external and internal oblique ridges, retromolar triangle, and pterygomandibular ligament and space must be recognized.

From a position in front and to the side of the patient, the anesthetist, using either the thumb or index finger, palpates the external oblique ridge and the anterior border of the ramus posteriorly from the mucobuccal fold. If the anesthetist assumes a position at the right front side of the patient, he will then palpate the right external oblique ridge and border of the ramus using his left index finger and the left external oblique ridge with the left thumb. In order to accomplish this, the hand is turned in an inverted position, so that while the thumb is used for palpation, the remaining fingers of the hand support the outside of the cheek. The forearm acts as a blindfold to the patient, preventing his visualization of the anesthetist's right hand with the approaching needle and syringe. When the patient is approached from the left front side, hand and finger positions are reversed, and the injected is made with the left hand while the right index finger palpates the left side, and the right thumb palpates the right side.

Palpating the anterior border of the ramus, the coronoid notch is located. This is an indentation indicating the greatest depth of the anterior border. At this level, the thumb or index finger is then moved medially across the retromolar pad onto the internal oblique ridge. The finger or thumb is then moved laterally again, tensing the tissue of the retromolar pad across the anterior border of the ramus. This gives a clearer exposure to the internal oblique ridge and pterygomandibular raphae. An aspirating dental syringe and a 1.62-inch (4 cm), 25-gauge needle are directed from the bicuspid (premolar) teeth of the opposite side of the jaw, and the needle is inserted at the level of the retracting finger or thumb. The injection site lies approximately 1 cm above the occlusal surfaces of the molars, medial to the retracting finger and lateral to the pterygomandibular raphae. The needle is advanced posterolaterally along the medial surface of the ramus. The syringe should be held in the horizontal position at all times. When the needle comes into contact with the middle portion of the ramus, it should be withdrawn slightly and aspirated to ensure that it is not intravascular, and the local anesthetic solution (1 to 2 mL) should be slowly deposited. Contact along the middle medial surface of the ramus should place the needle in close proximity to the mandibular sulcus, which funnels into the mandibular foramen. Placement of the needle too far posteriorly will result in anesthetic solution being deposited at the posterior margin of the ramus, which may cause anesthesia of branches of the facial nerve. During the mandibular block procedure, it is helpful to have the patient's mouth open at all times, because this greatly improves access to and definition of the anatomic landmarks.

Successful block of the inferior alveolar nerve results initially in a tingling of the lower lip on the affected side, rapidly progressing to a numbness of the lip. This numbness is often described by patients as a feeling of a fat lip. In most cases, the lingual nerve is also anesthetized during the block of the inferior alveolar nerve, and subjectively feels as if half the tongue is numb.

Lingual Nerve. The lingual nerve is a smaller terminal branch of the mandibular nerve. It provides sensory fibers to the mucous membranes of the floor of the mouth and gingiva on the lingual and sublingual salivary glands and their ducts. It occasionally supplies sensory fibers to the bicuspids and first molar teeth. Blockade of the lingual nerve also provides anesthesia over the anterior two-thirds of the tongue and the mucosa and mucoperiosteum on the lingual side of the mandible.

The lingual nerve block technique is actually a part of the aforementioned blockade of the inferior alveolar nerve. The lingual nerve is blocked by deposition of local anesthetic solution at a point approximately midway between the medial surface of the ramus and the end point described for injection into the inferior alveolar nerve. Injection may be made either as the needle enters along the pathway to the inferior alveolar nerve or while withdrawing from the mandibular foramen. Needle contact to the lingual nerve will elicit a paresthesia through the body of the tongue.

Long Buccal Nerve. The solitary sensory nerve among the anterior branches of the mandibular nerve is the long buccal nerve. It crosses the anterior ramus of the mandible at the level of the occlusal plane of the mandibular second and third molars. Crossing to a position lateral to the anterior border of the ramus, it is accessible to intraoral blockade.

The nerve itself is blocked by an injection into the buccal mucosa in the retromolar fossa approximately on line with the occlusal surface at the maxillary level of the third molar. This should be accomplished by injecting 0.5 to 1.0 mL of local anesthetic solution while the patient holds the jaw wide open. Alternatively, the terminal branches of the buccal nerve may be blocked by entering the mucosa above the buccal fold of the first mandibular molar. A 2.5-cm, 25-gauge needle is advanced in a horizontal position distally under the cheek toward the ramus of the mandible, depositing approximately 0.5 to 1.0 mL of the local anesthetic solution along the way.

Sensory fibers from the buccal nerve supply the mucous membranes and skin of the cheek. Other branches provide sensation to the buccal gingiva located about the mandibular molars and to the mucous membrane of the lower part of the buccal vestibule. Occasionally, the buccal nerve contributes to the sensation of the second bicuspid and the first molar of the lower jaw. It *may not* cover the posterior superior alveolar nerve but may extend a short distance into the mucous membranes of the upper and lower lips near the corner of the mouth.

Mental Nerve. The mental nerve is one of two terminal branches of the previously described inferior alveolar nerve. Near the mental foramen of the mandible, the inferior alveolar nerve divides into the mental nerve and a small incisive branch. Having emerged through the mental foramen, the mental nerve sends a few fine fibers to form a delicate plexus on the surface of the bone. The mental foramen usually lies at the apex of and just anterior to the second bicuspid root.

Anesthesia of the mental nerve includes the incisor teeth of the mandible, as well as the skin of the chin and lower lip and the mucous membrane of the lower lip. Because block of the

mental nerve usually includes the incisive nerve, anesthesia of the anterior portion of the mandible from the bicuspids to the incisors usually results, including anesthesia of the overlying labial structures anterior to the mental foramen.

As with certain other nerves, the mental nerve may be blocked either intra- or extraorally. For the *intraoral* approach, the lower lip and cheek are pulled to the buccal side and a 2-cm, 25-gauge needle is inserted into the labial fold. The needle is directed toward the periosteum of the mandible to a point between the bicuspid teeth at a level just below their root apices. It is often helpful to palpate the foramen before inserting the needle, since the needle must be "walked" gently along the periosteum until it enters the mental foramen. After careful aspiration, 0.5 to 1.0 mL of local anesthetic solution is deposited.

Identification of the mental foramen is essential to the performance of the *extraoral* technique. As mentioned previously, the mental foramen lies in the same vertical line as the supraorbital and infraorbital foramen and the pupil in the midposition. The actual location of the foramen on the mandible varies with age, being more caudally situated on the ramus in youth and much nearer the alveolar margin in edentulous elderly patients. After a skin wheal is made over the foramen, a 2- to 3-cm, 25-gauge needle is directed anteriorly and cephalad toward the foramen. It is not necessary actually to enter the foramen. Infiltration after paresthesia is elicited empirically over the midpoint of the mandible in a vertical plane is usually ample to effect analgesia of the ipsilateral lower lip and chin.

Auriculotemporal Nerve. This nerve is seldom blocked. The auriculotemporal nerve is one of the sensory posterior divisions of the mandibular nerve. It sends terminal branches to the parotid gland, temporomandibular joint, anterior portion of the ear, external auditory meatus, tympanic membrane, and scalp over the temporal region. The auriculotemporal nerve may be blocked extraorally by infiltration of local anesthetic solution into the area of tissue at the posterior margin of the zygomatic arch.

Glossopharyngeal Nerve (Cranial Nerve IX)

This nerve, as the name implies, is distributed to the tongue and pharynx. It is a mixed nerve, as its sensory fibers are both visceral and somatic, and its motor fibers are both somatic and special visceral efferents. The somatic sensory fibers supply the mucous membrane of the pharynx, fauces, palatine tonsil, and posterior part of the tongue.

The glossopharyngeal nerve emerges from the cranial cavity via the jugular foramen in very close relationship to the vagus nerve (cranial nerve X) and spinal accessory nerve (cranial nerve XI) along with the internal jugular vein. It is blocked just below this point and therefore usually involves blockade of the other two nerves as well. All three nerves lie in the groove between the internal jugular vein and the internal carotid artery. These two large vascular conduits may easily be punctured during attempts to block these nerves at this site, resulting in either intravascular injection or hematoma. Even very small amounts (ie, 0.25 mL) of local anesthetic injected into the carotid artery at this point can produce quite profound effects of

convulsion and loss of consciousness. Therefore, as always, aspiration tests must be meticulous. The landmarks for this block involve locating the styloid process of the temporal bone. This osseous process represents the calcification of the cephalic end of the stylohyoid process of the temporal bone. This osseous process represents the calcification of the cephalic end of the stylohyoid ligament. This fibrous band passing from the base of the skull to the lesser cornu of the hyoid bone ossifies to a variable extent in different individuals. It is relatively easy to identify in people with a large styloid process, but if ossification has been limited, the styloid process sometimes cannot be located with the exploring needle.

A 2-inch, 22-gauge needle is inserted at a point midway on a line joining the angle of the mandible to the tip of the mastoid process of the occipital bone. The needle is advanced directly medially until it locates the styloid process. If the styloid process is not located, the needle is inserted to a depth of 3 cm. In patients who have had a radical neck dissection and therefore are often candidates for this type of block, removal of the sternomastoid muscle places the styloid process and its adjacent nerves and vessels at a much more superficial location; in fact, in such individuals the styloid process can often be palpated in the interval between the styloid process and the posterior border of the mandible. In these people, the needle needs to be inserted only to 1.5 cm. Ideally, the styloid process is located as bony end point. If the needle is adjusted posteriorly to this at the same depth as the process, an injection of 1 to 2 mL of local anesthetic will effect anesthesia of the glossopharyngeal nerve as well as the vagus and accessory nerves. At this site it is not possible to selectively block one of these three nerves.

An alternative approach for blocking the glossopharyngeal nerve via an intraoral route has been reported and involves injecting local anesthetic into the midpoint of the posterior pillar of the fauces. This technique appears to have considerable promise as a means of numbing the glossopharyngeal nerve distribution to the oropharynx, and in combination with laryngeal nerve blocks and topical anesthesia poses a great potential for endoscopic procedures under regional anesthesia.

Vagus Nerve (Cranial Nerve X)

The vagus is the longest of the cranial nerves and contains both somatic and visceral afferent fibers. The somatic sensory fibers supply the external auditory canal and the posterior aspect of the pinna. The visceral afferents directly relevant to otolaryngology include those supplying the mucous membranes of the pharynx, larynx, bronchi, and lungs. Special visceral efferents supply the striated muscles of the larynx, pharynx, and palate.

Although neural blockade of the vagus itself is possible because of its long course and multiple sites of innervation, this is seldom done. However, the sensory branches of the vagus to the pharynx and larynx may be blocked, rendering the laryngeal inlet analgesic. This is very useful for intubations and endoscopic procedures.

Superior Laryngeal Nerve

The superior laryngeal nerve arises from the inferior ganglion of the vagus (nodose ganglion) and passes downward and medially, deep to the internal carotid artery to the lateral aspect of the thyrohyoid membrane, where it divides into internal and external branches. The internal branch pierces the thyrohyoid membrane with the superior laryngeal artery and veins, and supplies sensory fibers to the mucous membranes and parasympathetic-secreting fibers to the glands of the epiglottis, base of the tongue, aryepiglottic folds, and intrinsic parts of the larynx as far as the vocal cords. It communicates with the recurrent nerve inferiorly.

The superior laryngeal nerve is easily blocked as it sweeps around the inferior border of the greater cornu of the hyoid bone. This is readily palpable. A 1.5- to 2-cm, 25-gauge needle is "walked" off the inferior border of the greater cornu of the hyoid, and 2 to 3 mL of local anesthetic solution is deposited there. By reversing the process, the contralateral nerve may be blocked. This produces anesthesia over the inferior aspect of the epiglottis and the laryngeal inlet to the level of the vocal cords. It also produces motor blockade of the cricothyroid muscles.

To accomplish anesthesia of the trachea below the vocal cords, the simplest method is transtracheal puncture. A short 1- to 2-cm, 20- to 22-gauge needle is introduced in the midline through the cricothyroid membrane, as close to the superior border of the cricoid as possible to avoid trauma to the cords. Insertion of the needle into the trachea is confirmed by aspiration of air. Rapid injection of 2 to 4 mL of a topical anesthetic solution (4 per cent lidocaine) usually produces a forceful cough and disperses the solution through the trachea and ventral surface of the vocal cords.

Cervical Nerves

Spinal nerves in general separate into dorsal and ventral divisions almost as soon as the two roots have joined to form a single spinal nerve. The dorsal divisions of the cervical nerves supply the dorsal surface of the head and neck, and the ventral divisions supply the ventral and lateral parts of the neck.

Greater Occipital Nerve. The dorsal division of the second cervical nerve is the largest of the cervical dorsal divisions and is both motor and sensory. It emerges between the atlas and axis and divides into a large medial branch, the greater occipital nerve, and a smaller motor lateral branch. The greater occipital nerve gives off a few muscular fibers and then become subcutaneous in the neck to supply sensation to the scalp posteriorly and superiorly.

The greater occipital nerve may be blocked by making a wheal 2 cm below and slightly lateral to the occiput. A 2-cm, 25-gauge needle is advanced through the wheal to the skull, and 2 to 3 mL of local anesthetic solution is deposited as the needle is gradually withdrawn from the bone.

Lesser Occipital Nerve. A branch of the ventral cervical division supplies the skin of the side of the head behind the ear and may be blocked by a similar infiltration of local anesthetic solution at the posterior aspect of the mastoid process.

Cervical Plexus. The cervical plexus originates high in the neck opposite the first four cervical vertebrae. It emerges anterior to the deep prevertebral muscles but deep and posterior to the sternocleidomastoid muscle. In addition to the aforementioned lesser occipital branch, other branches of the superficial cervical plexus include the great auricular nerve to the skin over the parotid gland, the mastoid process, and the back of the ear and the anterior cutaneous nerve to the skin of the submandibular region and the anterior and lateral portions of the neck. Descending branches of this nerve supply the skin of the lateral and anterior aspects of the lower part of the neck to the sternum. Nerve block of these superficial branches may be accomplished by infiltration of 5 to 10 mL of local anesthetic solution at the midpoint of the posterior border of the sternocleidomastoid muscle.

Blockade of the deep cervical plexus is actually a paravertebral nerve bloc of C2, C3, and C4 spinal nerves as they emerge via the foramina in the cervical vertebrae. Each nerve lies in the sulcus in the transverse process of these vertebrae. Usually three needles are used, inserted at C2, C3, and C4 vertebral levels. The sites of insertion are located by reference to a line joining the tip of the mastoid process with the Chassaignac tubercle of C6, which is readily palpated at the level of the cricoid cartilage. A further line is drawn parallel and posterior to this at a distance of 1 cm. The C2 transverse process is usually located approximately one fingerbreadth caudad to the mastoid process on this line, and the C3 and C4 transverse processes are at similar intervals caudally on the same line. A horizontal line through the lower border of the ramus of the mandible intersects this line at C4, Two 5-cm, 22-gauge needles are used and directed in a medial and caudad fashion. The reason for the caudad direction is to avoid inadvertently entering the paravertebral foramen and producing a peridural or spinal block. The end point is the bony landmark of the transverse process, and paresthesias are obtained. A bolus of 3 to 4 mL of local anesthetic at each nerve is usually adequate for anesthesia. Fortunately, the paravertebral space communicates freely in the cervical region, and the anesthetic solution can spread easily to adjacent levels. Thus, it often is quite conceivable to effect deep cervical block with injections at just one level, but using a larger bolus, ie, 6 to 8 mL. This block is most useful for procedures such as thyroidectomy under local anesthesia and is also used effectively for carotid endarterectomy.

A major complication of the block is caused by the proximity of the vertebral artery, injection into which can produce profound toxic side effects of convulsions and unconsciousness. Therefore, aspiration tests are mandatory. Extension of the anesthetic into the epidural or subdural spaces is theoretically possible via either dural sleeves or leakage through the paravertebral foramen. Thus, patients who undergo such procedures must be observed very carefully. When performed bilaterally, phrenic nerve block is a very real risk. However, if less concentrated solutions are used specifically for sensory block rather than administering stronger solutions that produce motor block, this complication may be avoided. Because the deep cervical plexus lies under the deep cervical fascia, spread to the cervical sympathetic chain should not occur. If,

however, infiltration has spread anterior to the prevertebral fascia, the cervical sympathetic chain will be involved, with subsequent Horner's syndrome and spread to the recurrent laryngeal nerve, resulting in hoarseness. Both of these complications following a failed block indicate that the anesthetic has been injected at too superficial a site, superior to the deep cervical fascia.

Complications

Textbooks dealing with nerve block anesthesia tend to perpetuate lists of possible complications of regional anesthesia. Practically speaking, with the exception of toxic reactions, complications of the nerve blocks described have been extremely rare. Discussion here will be limited to hematomas and toxic reactions.

Hematomas

The head and neck are very vascular areas and represent a complicated anatomic network of arteries, veins, and nerves. It is possible, therefore, that needle perforation of a vessel and resultant hematoma may occur. Because of the bony cavity and lack of loose tissue areas, small collections of blood may expand, with resultant ischemia to nerves and tissues. Especially dangerous areas are the orbit and the neural foramina. The risk of hematoma may be minimized by the following: (1) careful attention to detailed anatomic landmarks; (2) precise knowledge of the block technique to be employed; (3) use of the shortest and smallest needle possible; (4) careful aspiration before injection as an indication that a blood vessel has been perforated; (5) injection of small, non-tissue-distending volumes of solution; (6) possibly, use of a vasoconstrictor in the injected solution; and, most important, (7) careful post-block follow-up.

Toxic Reactions

Toxic reactions to local anesthetics either are confined to the site of injection or are systemic with manifestations in distant organs. When injected extraneurally, commonly used local anesthetics are singularly free of neurotoxicity. The higher the local anesthetic blood level and the longer it remains elevated, the greater is the risk of an adverse systemic reaction. Although convulsions are the most conspicuous signs of toxicity, serious respiratory depression and hypotension may occur. As stated earlier in the discussion of local anesthetic agents, allergy to lidocaine and the amide-linked agents is exceptionally rare. Systemic toxicity thus remains the major concern and may be classified into two types: (1) direct intravascular injection (immediate) and (2) relative total overdose (delayed).

Direct Intravascular Injections. The discussion of hematoma formation identified the risk of vascular puncture. Of greater consequence is the failure to recognize the presence of the needle in a vessel and the subsequent direct intravascular injection of local anesthetic agent. Fortunately, most of the block techniques previously described use volumes of less than 5 mL, and even if injected directly into a vessel would produce a very transient CNS reaction, if at all. It is the level of local anesthetic in the blood going to the brain that precipitates a reaction. Therefore, if injection is made directly into an artery going to the brain, eg, the vertebral artery,

even 1 to 3 mL of drug may precipitate a seizure. Careful placement, fixation, and aspiration of the needle before injection prevents many toxic reactions.

Relative Overdose. This type of reaction is highly unlikely for nerve blocks of the head and neck, because it results from the use of total doses of local anesthetics in excess of maximal doses. The absorption of local anesthetic agents from the injection site into the bloodstream varies according to site. It is well known that uptake is increased in vascular areas and from mucous membranes. Therefore, the recommend safe dose should be reduced in these areas. Of greater risk to the otolaryngologic surgeon is the local infiltration of large volumes of local anesthetic agents, as may occur in plastic procedures, rotation of flaps, and so forth. Most of the local anesthetic agents require 15 to 30 minutes after injection to reach peak blood levels. This means that CNS toxicity or seizures, or both, may not occur for that period of time after administration of the block; ie, it is a delayed reaction. Furthermore, with many agents the blood level is cumulative. Therefore, the first dose may be tolerated, but 2 to 3 hours later, after two or three additional infiltrations have been accomplished, toxicity will result. This type of reaction may be minimized by calculating and preparing the total allowable dose in advance and not injecting more than the allowed solution. The use of vasoconstrictors with most agents has been shown to provide lower blood levels per given dose of drug than similar doses of plain solution.

Clinical Features and Diagnosis of Toxicity. The clinical features of toxicity manifest themselves primarily by the CNS reactions, whereas the cardiovascular system may be involved secondarily as a result of hypoxia or primarily as a consequence of gross overdosage.

In diagnosing toxicity, the time course from the injection to the appearance of symptoms is important. Thus, accidental intravenous injection causes a rapid onset (within 15 to 20 seconds), whereas overdosage is associated with symptoms occurring 5 to 20 minutes later. Evidence of toxicity appearing later than this should be viewed cautiously, since it is unlikely that signs and symptoms will be so delayed. After treatment has been instituted, it is valuable to withdraw blood to determinate the plasma concentration of the local anesthetic agent in any patient in whom toxicity is suspected. Because the signs and symptoms of toxicity tend to be nonspecific (with the exception of numbness of the tongue and circumoral tissues, which is diagnostic), there is a chance of considerable error in the diagnosis. Thus, muscular twitching may be the result of shivering or nervousness rather than toxicity. Convulsions may be due to epilepsy or eclampsia, and cardiovascular collapse may have a wide variety of causes. It is possible that hypotension is due to the local anesthetic procedure, ie, high extradural or spinal block, rather than the local anesthetic drug itself. On the other hand, toxicity may be missed, especially in the presence of anticonvulsive drugs such as general anesthetics or diazepam. Prolonged unconsciousness due to lidocaine given intravenously for postcardiac surgical care has been seen, muscular twitching having been abolished by sedative drugs.

Treatment of Toxicity. It is seldom necessary to treat the signs and symptoms of toxicity short of convulsions, provided that adequate respiration and cardiovascular functions are maintained. If convulsions occur, the aim of treatment is to stop them and treat any respiratory or cardiovascular depression before further cerebral hypoxia occurs.

Currently, three pharmacologic approaches to controlling convulsions are available.

First, *intravenous barbiturates* such as thiopentone. Even in small doses (50 to 100 mg), these drugs rapidly abort convulsions. They have been criticized in that they may exaggerate both respiratory and cardiovascular depression, but in the small doses required, such effects would be minimal and short-lived. These agents have the great advantage of being extremely familiar (and generally readily available) to anesthetists.

Second, *suxamethonium* (50 mg) given intravenously. This also stops convulsions rapidly, but this is accompanied by paralysis and cessation of respiration. In the hands of a competent anesthetist who can intubate and respire the patient, this is no disadvantage, but the drug is better avoided by the less experienced. Suxamethonium has less deleterious effects on the cardiovascular system than intravenous barbiturates. There is a theoretical objection that suxamethonium will not affect the convulsive process going on in the brain and that this might increase the oxygen demand of the brain, which is depressed by barbiturates. If respiration is assisted and cardiovascular function is adequate, this is most unlikely to affect the outcome.

Third, *diazepam* (5 to 10 mg) given intravenously. This drug is a powerful anticonvulsant and exerts its effect in little more time than does thiopentone (although even 20 to 30 seconds can seem a long time in these circumstances). It is virtually free from depressive effects on the circulation. The respiration must be carefully observed and a clear airway obtained, if necessary, by endotracheal intubation. If depression or apnea supervenes, artificial ventilation with oxygen is required. Provided oxygenation is maintained, it is very unlikely that serious consequences will ensue. The prevention of hypoxia is the single most important feature of treatment. Cardiovascular depression as evidenced by a weak pulse and hypotension should be treated by:

1. Correction of hypoxia, if present.

2. Elevation of the legs and an increased rate of intravenous solution.

3. Intravenous injection of a vasopressor. Since the hypotension is due to a combination of myocardial depression and vasodilatation, it is preferable to use a vasopressor that stimulates both alpha- and beta-adrenergic receptors (such as ephedrine, 15 to 30 mg).

General Anesthesia

Adult Patients

Patient Preparation and Monitoring

Incomplete patient workup often delays the start of surgery, as does waiting to obtain informed consent. With the increase in outpatient surgery, we have found that matters are expedited by having consent forms readily available and signed before the day of surgery.

Increased public awareness of blood-borne disease has created more questions concerning blood transfusions, AIDS, and hepatitis. Hospital policies dictating that physicians inform patients of transfusion-related risks and alternatives have been established.

Preparation for cases has become complex and time-consuming. It is no longer a matter of moving an ether bottle and mask from bed to bed. Consideration has to be given to the use of invasive monitors, as well as the position of the patient for induction of anesthesia and for surgery. Seemingly inconsequential last-minute decisions such as changing the final direction of the head of the table can create substantial logistical problems in rearranging monitoring equipment and has machines. In addition, any change in the patient's position carries the risk of loss of vascular access and extubation.

All patients should be monitored with a minimum of blood pressure cuff and ECG for straight local anesthesia. MAC monitoring includes a precordial stethoscope and, if available, a pulse oximeter. General anesthesia monitoring usually involves all the above, plus end-tidal CO_2 (mandatory in some states), temperature measurement, an esophageal stethoscope, a peripheral blockade monitor (to assess muscle relaxation), an FiO_2 meter, and a disconnect alarm (for disconnection of the patient from the anesthesia system).

Unfortunately, owing to the medicolegal climate, it is difficult to establish an accurate analysis of anesthetic morbidity and mortality. An anesthesiologist may expect to lose one to two patients in a lifetime as a result of anesthetic mishap; it is estimated that 50 to 95 per cent of these are preventable. Vigilance is the key and there should be no short cuts concerning patient safety. During a surgical procedure, multiple interruptions to have the anesthesiologist check "under the drapes" for breath sounds or problems with monitors may be tiresome, but are essential.

Concerns Related to Specific Procedures

Procedures involving the ear include mastoidectomy, myringotomy and middle ear surgery. If acute infection is present, every attempt should be made to avoid coughing. Microscopic procedures call for complete immobility and consideration of the use of vasoconstrictors and deliberate hypotension to diminish bleeding in the field. Nitrous oxide (N_20) is best avoided if work is done on the middle ear. Graft lifting may occur because of N_20's greater diffusibility compared with nitrogen. Nausea and vomiting associated with surgery of the inner ear are treated with antiemetics.

Major concerns during tonsillectomy include placement of the endotracheal tube and its possible dislodgement intraoperatively. Various mouth gags are available. The decision whether to extubate when the patient is in a deep plane of anesthesia or awake is an individual one. For awake extubation, the patient must be able to swallow and cough and have full protective reflexes. Extubation during stage II (the excitement stage) of anesthesia is dangerous, because vomiting and laryngospasm may occur.

Fortunately, post-tonsillectomy hemorrhage is infrequent, but it poses a significant anesthetic risk because the patient has a full stomach and is usually hypovolemic. The patient should be hemodynamically stabilized with blood, colloid, and crystalloid for volume replacement before the induction of anesthesia. The preferred induction technique is an awake intubation under topical anesthesia.

Maxillary and frontal sinus surgery require oral endotracheal tube (ETT) placement. Blood loss can be high and should be monitored during surgery. The head-up position immediately after surgery is helpful to limit facial edema.

Surgery of the parotid gland involves dissection about the facial nerve. The use of a nerve stimulator by the surgeon precludes the use of total muscle relaxation by anesthesia, and this must be understood before induction.

Cocaine and vasoconstrictors are often employed in nasal and adenoid surgery; a discussion may be found elsewhere in this chapter. Blood loss during these procedures can be substantial, and the use of a throat pack is advisable.

Endoscopic evaluations include laryngoscopy, nasopharyngoscopy, bronchoscopy, and esophagoscopy. Except for lesions involving the vocal cords, most examinations under anesthesia are best done on the paralyzed patient. Care must be given to protect the eyes, teeth, and gums. Although major bleeding is rarely a problem, minimal postbiopsy bleeding can induce laryngospasm. The patient should be fully awake at the end of procedure, and a full sitting position is often used for transport to the recovery room (RR). Racemic epinephrine and humidified oxygen by face mask should be available on arrival to the RR.

Uvulopalatoplasty (UPP) involves the same considerations as a tonsillectomy. Additional problems include difficult intubation, airway obstruction, sleep apnea, and the complications of morbid obesity. Pretreatment with H2 blockers and gastric motility agents are suggested. Sedatives and narcotics are not recommended because of the central regulatory problems of sleep apnea. If morbid obesity is present, a cardiac evaluation may be necessary, because these patients may experience substantial reductions of cardiac output upon assuming the supine position. In morbidly obese patients with sleep apnea, the surgeons at the authors' institution are inclined to start UPP with an elective tracheostomy under local anesthesia.

One of the first anesthetic considerations in major head and neck surgery is how the airway is to be managed. If there is any doubt as to whether the patient can be intubated, an initial tracheostomy under MAC should be considered.

Other concerns of the anesthesiologist are the possibility of large blood loss, hypothermia, the use of deliberate hypotension, invasive monitoring (Foley, CVP, A-line), and the management of a long-acting anesthetic. Venous air embolism is a possible complication because ENT surgery often involves open neck veins and a head-up position. There are no data of the incidence of venous air embolism in ENT surgery. Another potential problem results from pressure on the

carotid sinus causing vagal stimulation, decreased heart rate and blood pressure, and subsequent cardiac arrest. When this occurs, the surgeon must stop stimulation of the area and apply local anesthetic. Intravenous atropine may be used to treat bradycardia. Surgical ablation of the right stellate ganglion or trauma to the cervical autonomic nervous system may cause prolongation of the Q-T interval and lowering of the ventricular threshold. Life-threatening ventricular dysrhythmias can occur even into the postoperative period.

Pediatric Patients

The number of pediatric ENT procedures performed on an outpatient basis is increasing rapidly. Several problems and considerations particular to the practice of outpatient surgery have emerged. Specifically, difficulties regarding premedication, NPO status, the evaluation of children with upper respiratory infections (URIs), and coordination of anesthesiologist-surgeon-parent-child communication must be resolved to ensure good patient care and speedy, cost-efficient management.

Several large centers across the nation are using the telephone for anesthesia preoperative evaluation with great success. This format eliminates the need for an additional patient visit and decreases the amount of non-OR anesthesia time.

The use of premedication for outpatient pediatric surgery is variable. Some institutions instruct the parent to administer a premedicant drug at home, others administer it in the holding area, and still others prefer no premedication at all. Since drug absorption by the oral route is unpredictable, the practice of at-home medication administration is not recommended. Practical experience demonstrates that no medication regimen achieves the calming effect of a short period of constructive "play" among the anesthesiologist, patient, and parents. No premedication should be given to a patient with sleep apnea or a compromised airway.

NPO status appears to be more difficult to enforce in the pediatric patient. Failure to comply with standard fasting orders has become a common reason for surgical cancellation. Careful explanation of the reasons for fasting and the consequences of not doing so (cancellation of surgery and possible aspiration) is essential. The usual NPO guidelines are listed in Table 6. It is imperative to remember that children dehydrate quickly, making them more sensitive to the depressant effects of anesthetics. If a procedure is delayed, measures should be taken to ensure adequate hydration. A list of standard fluid replacement guidelines for pediatric patients can be found in Table 7.

Table 6. NPO Guidelines in Children According to Age

	Age	
Liquids	Less than 1 year old	Clear liquids and breast milk up to 4 hours preoperatively
	Over 1 year old	Clear liquids and breast milk 6 to 8 hours preoperatively
Solids	Over 1-2 years old	NPO after midnight.

Pediatric patients presenting for surgery with upper respiratory infections (URIs) pose a dilemma. Currently, cancellation of surgery is recommended if the child is acutely ill, although a "runny nose" associated with unchanged chronic rhinitis or in a child who is not acutely ill, it is no longer an indication for cancellation. However, patients with a history of prematurity, asthma, or other conditions predisposing to respiratory complications must be carefully evaluated. Surgery should be postponed if there is any suspicion of an acute respiratory problem.

Table 7. Pediatric Intravenous Fluid Replacement Guidelines

Weight	Replacement
O-10 kg (first 10 kg)	4 ml/kg/hr
10-20 kg (second 10 kg)	2 ml/kg/hr
20+ kg (each kg thereafter)	1 ml/kg/hr.

Anesthesia for ENT surgery in children presents an exaggeration of a problem also found in adults, namely, the need to share the airway in a smaller physical space. Children are more prone to laryngospasm and more easily extubated inadvertently than adults. Therefore, it is imperative for the surgeon and anesthesiologist to communicate before the patient is positioned or the operating table moved.

A frequently encountered difficulty in the 5- to 9-year-old population is that of loose teeth. A lost tooth during surgery, especially during anesthetic induction or emergence, is a source of great stress to all. Failure to locate a displaced tooth requires subjecting the child to a chest film. If the tooth is visualized in the stomach, there is no further treatment. However, if it is seen in the tracheobronchial tree, bronchoscopy is mandatory. The consequences of tooth aspiration include pneumonia, chronic infection, and bronchiectasis.

A surgeon noting a loose tooth during the preoperative examination should inform the anesthesiologist. The surgeon or anesthesiologist must inform the parents of the possible dangers associated with this finding and obtain informed consent for possible tooth extraction. If the tooth is only slightly mobile, additional care with the mouth gag may be sufficient. It is wise to note the condition of a loose tooth on the anesthetic and OR records, both after induction and after emergence, as well as on the RR record. If an extraction is necessary, it is a good practice whenever possible to give the tooth to the child postoperatively for future collection by the "tooth fairy".

Foreign body aspiration is most common between the ages of 1 and 3 years. Intravenous or inhalation induction may be used, but it is best to keep the patient breathing spontaneously. The use of atropine or glycopyrrolate is advisable to oppose vagal stimulation. Postoperative care includes aerosolized racemic epinephrine, intravenous dexamethasone, and a chest x-ray.

Tonsillectomy and adenoidectomy (T & A) are now being performed in sicker children, thus creating more anesthetic difficulties. To protect the airway, patients must be extubated either

deeply asleep or completely awake. Hypovolemia associated with the postoperative bleeding tonsil must be corrected before anesthesia is induced.

Thirty-three per cent of patients with cleft lip and 14 per cent of those with cleft palate have additional congenital anomalies. Aspiration, URI, chronic otitis, and anemia are commonly associated medical problems. Anesthetic selection is affected by the use of epinephrine. Endotracheal tube dislodgment, conjunctivitis, and corneal abrasions are notable intraoperative complications. If a palatoplasty is performed, postoperative airway problems are diminished by suturing the tongue.

Malignant Hyperthermia

Malignant hyperthermia (MH) is a relatively rare, pharmacogenic syndrome occurring with one of every 15.000 pediatric and one of 50.000 adult general anesthetics. This syndrome is associated with several triggering agents, notably halothane and succinylcholine. Two-thirds of cases occur on first exposure to anesthesia, but one-third may be noted when there is a history of previously uncomplicated anesthesia. Since familial association is very high, inquiries regarding anesthesia-related death or unusual instances of high fever, especially those related to exercise, are advisable. Pretreatment with dantrolene and the avoidance of known MH triggers have made anesthesia in patients with positive family histories or previous MH relatively safe. However, special preparations (eg, availability of ice in the OR, use of a cooling blanket) must be made and the OR team should be prepared for rapid action.

The initial sign of MH is tachycardia. Tachycardia during anesthesia is fairly common, but when it occurs in children, especially those undergoing anesthesia for the first time, a high state of readiness must ensure. In MH, tachycardia is almost immediately followed by a rapid increase in temperature with resultant high mortality. For this reason, the observation of possible MH symptoms by the anesthesiologist is to be taken seriously. For this reason, the observation of possible MH symptoms by the anesthesiologist is to be taken seriously. The surgical team may assist in the rapid treatment of this problem, which includes stopping all anesthetic gases, giving 100 per cent oxygen, sending an immediate blood gas determination, and (in the presence of any increase in temperature or increased pCO_2 or decreased pH) administering bicarbonate, dantrolene, and furosemide and packing the patient in ice. A history of malignant hyperthermia must be actively sought during the preoperative interview.

Deliberate Hypotension

The controversy surrounding the merits of deliberate hypotension (DH) has continued over 40 years. The initial enthusiasm accompanying the discovery of the "bloodless field" in the mid-1940s lasted a short 8 years. Initial studies revealed that early methods of inducing hypotension (including arteriotomy and spinal anesthesia), combined with poor patient selection and indiscriminate use, caused a high incidence of morbidity and mortality. Fear of inducing major organ damage or death resulted in near-total abandonment of the technique.

Interest was rekindled in 1960 when a study reviewing 9000 cases using DH revealed a significantly lower complication rate, and concluded that early complications were a result of poor vigilance and inappropriate anesthetic management. The intervening years have seen an improved understanding of hypotension and an increased availability of well-trained anesthesia personnel and sophisticated monitoring devices. However, the use of controlled hypotension remains largely within the confines of the university OR. This limited utilization becomes all the more unlikely when the attributes of DH anesthesia are examined.

The subjective and objective advantages of DH are as follows:

1. Improved surgical conditions, namely, a clearer, cleaner field.

2. Decreased usage of suture ligatures and electrocautery.

3. Decreased tissue trauma with improved healing and cosmetic results.

4. Reduction of blood loss and perioperative transfusion.

5. Decreased operating time.

Most investigators and clinicians agree that DH provides a drier surgical field. However, controversy was again incited by a 1982 clinical study of DH, in which surgeons assessing the operating field with a scoring system failed to correlate the quality of the surgical field with blood pressure. It follows that a clearer field would lessen tissue trauma, require fewer hemostatic maneuvers, and possibly produce a better aesthetic outcome. The reduction of blood loss remains the clearest, largely undisputed advantage of DH. In light of the emergence of AIDS, the ability to minimize blood loss, and therefore transfusion risks, will undoubtedly become the major motivation for DH use.

Although all donated blood in the USA is screened for hepatitis and HIV antibodies, the risk of non-A, nod-B hepatitis (one in ten) and infection via the HIV "window" (one in 10.000)0 remains. The HIV "window" refers to the time period during which a blood donor is infected but not yet producing detectable antibody.

A reduced duration of surgery with the use of DH has never been proved. Rather, the seniority and experience of the principal surgeon remain the major determinants of operative duration.

DH involves alterations in patient positioning, ventilation, and anesthetic dosing, as well as the addition of cardiovascular drugs (arterial and venous dilators, beta blockers, calcium channel blockers, propranolol). The complications and contraindications may easily be noted in relation to these four interventions. Studies have found the incidence of complications to be the same in normotensive patients as in those undergoing induced hypotension. Complications attributed to DH range from mild (dizziness, prolonged awakening) to severe (cerebral

thrombosis, air embolism, death) and are believed often to be related to faulty technique.

A 1986 review of 1800 ENT procedures employing controlled hypotension described cerebral morbidity of four in 1802 (0.22 per cent) and a mortality rate of one in 1802 (0.06 per cent). The only mortality therein wa a total cerebral death secondary to an undiagnosed congenital hypoplasia of the right vertebral artery. Table 8 cites the best-known studies of mortality in hypotensive anesthesia.

Table 8. Mortality in Hypotensive Anesthesia

			Mortality	
Author	**Years**	**No of Cases**	**No of Cases**	**Incidence (%)**
Little (1955)	1950-1953	27.930	96	0.34
Enderby (1961)	1950-1960	9107	9	0.10
Larson (1964)	1958-1964	13.264	113	0.85
Kerr (1977)	Not noted	700	0	0
Enderby (1980)	1960-1976	9256	2	0.02
Pasch (1986)	1977-1984	1802	1	0.06.

Contraindications to DH are major organ dysfunction (hepatic, renal, CNS, or cardiovascular), severe peripheral vascular disease, hypovolemia, anemia, sickle cell disease, hypertension (especially uncontrolled), uncorrected polycythemia, and inexperience of the anesthesiologist. Angina and myocardial infarction remain controversial contraindications.

In summary, DH is a decidedly advantageous technique in certain situations. Since the "safe" lower limit of blood pressure has never been established, DH should be reserved for procedures in which patient factors or copious operative blood loss make it advisable.

Laser Surgery

The anesthesiologist has two major concerns when surgery involves the use of a laser: protection of the patient from the hazards of inadvertent laser exposure, and prevention of laser-related fires. These concerns have become the topic of much discussion.

Table 9A. Anesthetic Technique for ENT Procedures

ETT	Advantages	Disadvantages
Rusch tube		
Aluminum wrapped	Controlled airway Able to use inhalation agents Able to scavenge Able to measures $ETCO_2$	Kinking of tube Tape obstructs airway Tape traumatizes airway Use of wrong tape that will burn Tape reflects and burns Unable to wrap cuff or distal tip Bulky
Muslin / cloth wrapped	As in aluminum wrapped	Needs repeat wetting / soaking Dries out, will burn Expensive Disposable
Silicone laminated	As in aluminum wrapped Single use Cuff and distal end laminated	Most flammable tube with CO_2 laser
Polyvinylchloride	As in aluminum wrapped Least expensive tube for general use	
Transparent unmarked polyvinylchloride	As in aluminum wrapped Best for Nd-YAG laser use Noncombustible	Uncuffed Aspiration
Norton - noncombustible flexible metal		Unable to scavenge well Large out diameter obstructs view Small inner diameter: increased airway resistance Reflective.

It is widely known that the eyes are most susceptible to unintentional laser exposure. OR staff should wear appropriate glasses and the patient's eyes must be carefully taped and covered. Fire occurs in 0.4 to 1.5 per cent of cases involving the CO_2 laser. Any nonhypoxic (> 21 per cent O_2) gas mixture supports combustion. In addition, surrounding tissue as well as most commonly used OR materials (eg, drapes, dry gauze, Cottonoids, endotracheal tubes) will ignite.

Table 9B. Anesthetic Technique for ENT Procedures

ETT	Advantages	Disadvantages
Apneic	Uninterrupted view of larynx Elimination of ETT	Aspiration Laryngospasm: too light anesthetic level Hypoxia and hypercarbia Shared airway with surgeon Fire hazard
Spontaneously breathing	Uninterrupted view of larynx Elimination of ETT	Aspiration Laryngospasm: too light anesthetic level Hypoxia and hypercarbia Vocal cords not immobile Inadequate scavenging Shared airway with surgeon Fire hazard
Bronchoscope with side arm	Better visualization Elimination of ETT Able to measure $ETCO_2$ Able to use potent inhalation agents Vocal cords immobile	Shared airway with surgeon Hypoxia and hypercarbia Inadequate scavenging Fire hazard Leak around bronchoscope Aspiration Gastric distention Pneumothorax / pneumomediastinum Recall
Bronchoscope with jet ventilation	Better visualization Elimination of ETT	Unable to use potent volatile anesthetics Prolong ventilation postoperatively Fire hazard Above with jet ventilation.
Bronchoscope Sanders venturi	Better visualization Elimination of ETT Better ventilation over bronchoscope with side arm	

Current recommendations are to keep the inspired oxygen concentration as low as possible, usually between 25 and 30 per cent. A pulse oximeter must be used to ensure an oxygen saturation greater than 90 per cent. The use of helium, nitrogen, or air mixed with oxygen is advocated because nitrous oxide (commonly used) supports combustion almost as well as oxygen. Helium is probably the most advantageous choice: its low density produces little turbulence, improving gas flow, and its flame-retardant property is thought to be related to high thermal

diffusivity.

All endotracheal tubes are hazardous in the presence of laser. They either burn or reflect the laser beams, causing burning of adjacent tissues. The advantages and disadvantages of currently available ETTs are listed in Table 9.

The management of a laser-initiated fire in the surgical field includes immediate cessation of ventilation, dousing with water when possible, extubation of the trachea, maintenance of the airway, ventilation with 100 per cent O_2, reintubation, and evaluation of damage with bronchoscopy and chest x-ray. The use of topical anesthesia with lidocaine at the time of extubation is suggested to prevent laryngospasm. Subsequent edema of the airway is a potential hazard.

After laser resection of tumor of the airway, the development of postoperative edema is a concern. Patients should be observed for several hours (up to 24) in the RR. Humidified O_2, corticosteroids, and racemic epinephrine are recommended.

Several anesthetic techniques for laser surgery of the airway have been described and are outlined in Table 9. In reviewing this information, it should be kept in mind that an ideal anesthetic technique should provide adequate visualization, ensure the absence of airway reflexes and vocal cord motion, and allow a rapid return of normal function when desired.

Recovery Room: After Anesthesia

Recovery from general anesthesia usually begins in the OR. After the anesthetic is discontinued, emergence occurs in stages. Thus, the anesthetic depth at the conclusion of surgery and the time to complete recovery from anesthesia are variable.

The emergence in some instances may prove the most difficult part of the anesthetic. Assistance from the surgeon may be needed, especially in cases involving the airway, and should continue during patient transportation to the RR.

The patient's position during transportation from OR to RR is influenced by anesthetic depth, the level of consciousness, and the type of procedure. The lateral decubitus position, with or without the head down, is frequently used, especially if the potential for vomiting and aspiration is increased. The tonsil position (right side down, head lower than hips and slightly extended, hand beneath the chin) is popular in the pediatric group. Positioning should be such that the airway can be maintained by the patient and monitored by the anesthesiologist.

An extensive list of possible complications after ENT surgery is given in Table 10.

Table 10. Complications After ENT Surgery

More Frequent Problems	Less Frequent Problems
Emergence excitement	Aspiration
Respiratory obstruction	Oliguria
Hypoventilation	Postsurgical bleeding
Hypoxia	Arrhythmia
Hypotension / hypertension	Congestive heart failure
Nausea and vomiting	Convulsion
Pain	Cardiac arrest.
Shivering	

Emergence from anesthesia usually involves restlessness and agitation. Pain, the use of ketamine, hypercarbia, preoperative anxiety, behavioral type, abdominal distention, and urinary retention are believed to contribute to emergence-related excitement. Whatever the cause, hypoxia must be ruled out. Hypoxia may be monitored by pulse oximetry, which is easy and non-invasive, or by analysis of arterial blood gases (ABG).

Postoperative respiratory obstruction is commonly due to pharyngeal soft tissue, and is usually relieved by the jaw thrust maneuver or alteration of head position. There should be no hesitation in reintubating a patient if it appears that the airway cannot be maintained easily and safely.

Immediate postoperative hypotension is most frequently due to hypovolemia. Volume replacement is therefore more appropriate than the use of vasoconstrictors.

Vomiting is more common in patients who swallow blood or undergo ear procedures. The potential for emesis is often anticipated intraoperatively and antiemetics are given. If aspiration is suspected, a chest x-ray, serial physical examination, and ABG analysis are necessary.

Pain is often associated with agitation. Once the causes of agitation that may heighten pain are eliminated, analgesics may be administered. Intravenous narcotics are often used for a faster onset. Maximal narcotic-associated respiratory depression and drowsiness do not occur simultaneously. Therefore, a sleepy patient may be aroused and complain of pain.

Shivering can be a serious postanesthesia problem because of the enormous increase it causes in oxygen consumption. Unfortunately, the mechanism is not well defined and methods of treatment are inconsistent.

Discharge from the RR is effected when the patient meets preset criteria: vital signs stable, effects of local anesthetics reversed, level of consciousness regained, and reflexes present. Outpatient surgical patients should also be ambulatory, have adequate pain relief, be able to micturate, demonstrate no nausea and vomiting, and have someone competent to care for them

for the following 12 to 24 hours. Since psychomotor skills can be impaired with even small amounts of medication, patients are cautioned not to drive, not to operative heavy machinery, and to postpone major decision making for 12 to 24 hours.