K. J. Lee: Essential Otolaryngology and Head and Neck Surgery (IIIrd Ed)

Chapter 25: Head and Neck Anesthesia

Local Anesthesia

Definitions

Local anesthesia is the loss of sensation in a circumscribed area. Local anesthetics are drugs thart block nerve conduction when applied locally to nerve tissue in appropriate concentrations. In addition, clinically useful local anesthetics have the following properties:

1. The nerve block is reversible.

2. The time of onset, and duration of blockade of the nerve fiber is predictable for common usage.

3. The drug is nonirritating to the tissue to which it is applied.

4. The drug is permeable and diffusable.

5. The drug has a high therapeutic index.

6. The drug is water soluble and chemically stable.

Mechanism of Action

Local anesthetics prevent the conduction of nerve impulses. They act by interfering with ionic exchange at the nerve cell membrane and by stabilizing the membrane against the generation of an action potential.

Chemistry

The local anesthetics consists of three parts: aromatic lipophilic group, intermediate chain, hydrophilic group.

The common local anesthetics have as an intermediate chain either an ester, i.e. cocaine, procaine, or an amide linkage, i.e. lidocaine (Xylocaine), mepivacaine.

The terminal hydrophilic (amine) group is able to combine with an acid and form a water soluble salt.

The base unionized form is more lipid soluble and is the form that penetrates the neural membrane and produces anesthesia. The pKa of the particular drug and the pH of the solution determine the ratio of salt to base (as dictated by the Henderson-Hasselbalch equation) and therefore, the amount of drug in the pharmacologically active form. When the tissues into which a local anesthetic is introduced are acidotic, a larger proportion of the drug is in the inactive (salt) farm. This accounts for the diminished activity of local anesthetics in

infected areas.

Uptake, Metabolism, and Excretion

Most local anesthetic agents are absorbed rapidly into the bloodstream from the mucous membranes and subcutaneous tissues. Certain sites of particular interest to the otolaryngologist, such as the laryngeal and tracheal mucous membranes, are associated with such rapid uptake of local anesthetics that blood levels approach that achieved with intravenous administration.

Amide type drugs are metabolized by the liver in a complex series of steps beginning with N-dealkylation. Ester type drugs are hydrolyzed by cholinesterases in the liver and plasma. Both degradation processes depend on enzymes which are synthesized in the liver and therefore, both will be compromised in a patient with parenchymal liver disease.

Many of the end products of catabolism of both esters and amides are water soluble and are excreted to a large extent in the kidneys.

Toxicity

Local Toxicity

Local toxicity is a reaction of tissue at the site of injection. These include reactions of the skin and mesenchymal tissues (cellulitis, ulceration, abscess formation, tissue slough) as well as lesions of the peripheral nerves (neuropathy). The most common causes of local tissue reactions include:

1. Faulty technique: contamination of the local anesthetic agents and traumatic administration.

2. Reactions from the local anesthetic agent itself.

3. Reactions from preservatives and vasoconstrictor agents added to the local anesthetic.

General Toxicity

General toxicity includes systemic reactions which occur due to absorption of a given drug into the general circulation (Table 25-2). These may be due to an excessively high blood level, allergy, or miscellaneous causes.

Table 25-2. Rate of Topical Absorption in Decreasing Order

Tracheobronchial tree, nose, pharynx, larynx, esophagus.

1. A toxic blood level is the result of a drug overdose. This can be achieved by rapid absorption, excessive dose, and/or inadequate metabolism and redistribution. Most often a toxic overdose is a result of carelessness in exceeding the recommended dosage for a

particular drug, or inadvertent intravenous administration. Ninety-eight percent of systemic toxic reactions to local anesthetics are due to drug overdose.

Significant symptoms of toxic overdose of local anesthetic agents are confined to the central nervous system and cardiovascular system (see Tables 25-3 and 25-4). The central nervous system responses to local anesthetic agents are biphasic with stimulation followed by depression. Clinically, patients may appear agitated with confused and rambling speech. This excitation may proceed to seizures and coma. The direct cardiovascular effects of local anesthetics are those of depression. Both myocardial performance and peripheral vascular tone are diminished by increasing levels of local anesthetic agents.

As in most iatrogenic complications, the most effective treatment of local anesthetic toxic overdose is avoidance. This requires care in the choice of agent and administration. When preliminary signs of overdose appear, O_2 should be administered and a venous cannula should be secured. Symptoms of excitement may be treated with hypnotics (diazepam, barbiturates), although this should be done with caution so as not to exacerbate the subsequent cerebral depression. Likewise if seizures occur, antiseizure medication should be employed but with the realization that the subsequent coma may be exacerbated. The physician should be prepared for general supportive measures in the case of ultimate cardiovascular and respiratory collapse. This may include endotracheal intubation, mechanical ventilation and intravenous fluid and pressor therapy.

Table 25-3. Local Anesthetic Toxic-Symptoms.

1. CNS:	Excitation.
Cerebral cortex -->	excitement, disorientation rambling speech, seizures.
Brain stem -->	tachycardia, hypertension, vomiting, sweating.
2. CNS:	Depression.
Cerebral cortex -->	coma.
Brain stem -->	bradycardia, hypotension, apnea.
3. Cardiovascular system:	Depression.
Bradycardia.	
Hypotension.	
Shock.	
4. Cardiorespiratory arrest.	
5. Death.	

Table 25-4. Prevention and Treatment of Toxicity.

1. Prophylaxis.

a. Avoid overdose.

b. Diazepam (Valium) premedication.

2. Maintain verbal contact with patient throughout surgery. Must be alert to early signs and symptoms of excitation.

3. Have an IV in place before administration of local anesthetics.

4. When toxic symptoms appear, stop surgery, give O_2.

5. Maintain airway and ventilation.

6. Avoid giving further depressants if possible. However, IV Valium or Pentothal may be required to terminate seizure.

7. Fluid, pressor resuscitation as required.

2. True allergic reactions to local anethetics are an infrequent occurrence (2% of reported complications), and most commonly occur with ester derivatives. These may present as any of the gamut of allergic syndromes from relatively innocuous dermatologic signs to anaphylactic shock. The treatment of allergic reactions to local anesthetic agents involves the same strategies as for any allergic reaction.

Trying to choose an anesthetic technique for a patient with a history of "allergy" to local anesthetics is a frequent clinical problem. A careful history with documentation, if possible, should help sort out those with reactions to toxic overdose (see above) or miscellaneous reactions (see below) from those with true allergy. If allergy is confirmed, some authors suggest that utilization of the opposite class of drug (i.e. amide if ester was previously used) is a relatively safe approach. Dyclonine, which is neither amide nor ester, may be safely used in some cases where allergy to both classes of drugs are suspected. If doubt exists, one must consider alternative techniques (i.e. general anesthesia).

3. Miscellaneous reactions included those adverse reactions which are not specific to the local anesthetic agent per se. An inappropriate response to the needle used for administration or an increased sensitivity to the preservative in the drug are examples. A unique adverse reaction occurs with the local anesthetic prilocaine. When used in excess of 500 mg in an adult, a significant fraction of the patient's hemoglobin is reduced to the methemoglobin state. Methemoglobin has a diminished ability to transport oxygen to peripheral tissues. The treatment of methemoglobinemia caused by prilocaine overdose is the slow intravenous administration of methylene blue, 1% solution, total dose 1-2 mg/kg.

Local Anesthetic Agents (See Table 25-1)

Cocaine

Cocaine was the earliest recognized local anesthetic and is the only agent that is naturally occurring. It was introduced into clinical practice for topical anesthesia by Sigmund Freud and Karl Koller in 1884 and for nerve trunk blockade by William Halsted in 1885.

Cocaine is unique among local anesthetic agents in its ability to block the reuptake of norepinephrine at adrenergic nerve endings. It is this metabolic action which accounts for its side effects of vasoconstriction, tachycardia, hypertension, "sensitization of the myocardium to catecholamines", mydriasis, cortical stimulation, and addiction.

Table 25-1. Concentration and Maximum Safe Doses of Local Anesthetics

	Topical Concentration/Max dose	Infiltration Concentration/Max dose
Esters		
Cocaine	&4-10% / 3 mg/kg	Not used
Procaine (Novocaine)	Not effective	1-2% / 14 mg/kg adults / 5 mg/kg children
Tetracaine (Pontocaine)	0.5-2% / 1 mg/kg	0.1-0.25% / 1-1.5 mg/kg
Chloroprocaine (Nesacaine)	Not effective	2% / 14 mg/kg
Hexylcaine (Cyclaine)	5% / 3 mg/kg	1-2% / 7 mg/kg
Amides		
Lidocaine (Xylocaine)	2-4% / 3 mg/kg	1-2% / 3 mg/kg sine epi / 7 mg/kg cum epi
Mepivacaine (Carbocaine)	Not effective	1-2% / 7 mg/kg
Prilocaine (Citanest)	Not effective	1-2% / 7 mg/kg
Bupivacaine (Marcaine)	Not effective	0.25-0.75% / 3 mg/kg
Piperidine		
Dyclonine (Dyclone)	0.5% / 4 mg/kg	Not used
Epinephrine	1:1000-1:100.000 / 1 mg	1:1000-1:100.000 / 1 mg

with halothane anesthesia 10 mL of 1:100.000 (0.1 mg) can be used over a 10 minute period, or 30 mL over 1 hour (0.3 mg).

& (10% solution = 100 mg/mL; 1% solution = 10 mg/mL).

Other drugs which interfere with catecholamine catabolism, such as monoamine oxidase inhibitors (MAOI) may interact with cocaine and cause a hypertensive crisis.

Cocaine is an extremely potent topical anesthetic agent and an extremely toxic drug. The maximum permissible dose topically is 2-3 mg/kg. The onset of action is immediate, and the duration is 45 minutes. It is decomposed by autoclaving.

Procaine Hydrochloride (Novocaine)

Procaine was first synthesized in 1905 by Einhorn as a result of a concerted effort to find a safer substitute for cocaine.

Procaine hydrochloride is a relatively weak local anesthetic agent of the ester type. It is inactive when applied topically. When used for infiltration, it is associated with a rapid onset (2-5 minutes) and a brief duration of action (45-60 minutes). It has a relatively toxicity, and a maximum recommended dose of 1000 mg. It is commonly used in 2% solution for infiltration.

Procaine is rapidly hydrolyzed by intravascular cholinesterase. Procaine may prolong the effect of succinylcholine (Anectine) which is also catabolized by cholinesterase.

Tetracaine Hydrochloride (Pontocaine)

Tetracaine is a potent anesthetic of the ester family. Its potency and toxicity are approximately 10 times those of procaine. It is effective when applied topically in a concentration of 1-2% and is associated with a rather delayed onset (6-12 minutes) and prolonged duration of action (1.5-2 hours). No more than 80 mg should be used for topical anesthesia of the upper respiratory tract.

Chlorprocaine Hydrochloride (Nesacaine)

Chlorprocaine is a halogenated derivative of procaine, and as such has similar pharmacologic properties. It is hydrolyzed more rapidly than procaine and is therefore less toxic. It is not useful for topical anesthesia. It is used in a 2% concentration for infiltration and the maximum recommended dose is 1 g.

Hexylcaine (Cyclaine)

Hexylcaine is an ester having somewhat greater potency and toxicity than procaine. It is most frequently used for topical application, where it provides a rapid onset (2-3 minutes) and moderate duration of action. Infiltration use has been limited by a high incidence of local irritation. The solution is stable and may be autoclaved.

Lidocaine Hydrochloride (Xylocaine)

Lidocaine is an aminoacetylamide. It has excellent penetrating powers and is effective by all routes of aministration, providing a rapid onset and a moderate duration of action (1 hour). The action may be prolonged by the addition of epinephrine in a concentration of 1:100.000 (1 mg of epinephrine per 100 mL of solution). For infiltration or nerve block, 1 and 2% solutions are used. A 4% solution is employed for topical anesthesia. The maximum recommended dose for topical anesthesia in an adult is 200 mg (5 mL of the 4% solution) and

for infiltration is 200 mg (without epinephrine) and 500 mg (with epinephrine).

The enhanced ability of lidocaine to suppress automaticity in ectopic myocardial foci has encouraged its use in the acute management of ventricular arrhythmia. A dose of 50-100 mg as an intravenous bolus is used for the purpose.

Mepivacaine Hydrochloride (Carbocaine)

Mepivacaine is an amide chemically related to lidocaine. It shares with lidocaine many clinical features. It is associated with less vasodilatation than that seen with lidocaine and has a slightly longer duration of action.

Prilocaine Hydrochloride (Citanest, Propitocaine)

Prilocaine has similar clinical properties to those of lidocaine except that it is more rapidly metabolized. When the maximum dose of 500 mg is exceeded, methemoglobinemia may result (see miscellaneous reactions, above).

Bupivacaine (Marcaine)

Bupivacaine is an amide chemically related to lidocaine. It shares with Xylocaine many clinical features. It is associated with an extremely long duration of action (2-4 hours). It is tightly bound to tissue and plasma protein, and is not associated with high blood levels when appropriately administered. Bupivacaine is used for infiltration and nerve block ina 1-2% solution with a maximum recommended dose of 225 mg. Its high potency and long duration of action make it a useful agent for prolonged procedures.

Dyclonine Hydrochloride (Dyclone)

Dyclonine is neither an ester nor an amide. Therefore, it has been recommended for use in those patients who are allergic to both families of local anesthetics. It has a rapid oset of action (3-10 minutes) and a brief duration (30 minutes). It is used in a 0.5% solution for topical anesthesia and the recommended maximum safe dose is 300 mg in an adult.

Dibucaine Hydrochloride (Nupercaine)

Dibucaine is of the amide group. It is extremely potent for topical and infiltrative use. However, it has fallen out of common use because of a reported high incidence of local toxicity.

Piperocaine (Metycaine)

Piperocaine is similar to procaine but more toxic and with a longer duration of action. The concentration used is 0.5-1% solution for infiltration and 2-10% solution for topical use.

Miscellaneous

Cetacaine is a mixture of tetracaine and ethyl and butyl aminobenzoate. Forestierre's solution is a mixture of cocaine (4%), phenol, potassium chloride, and epinephrine, 1:1000. Bonnaine's solution is a mixture of cocaine (4%), methol, and phenol.

Premedication

Drug premedication is only a supplement to a supportive and informative preoperative visit.

Hypnotics

Barbiturates. The barbiturates are probably the most frequently employed of the hypnotics. They act principally by depressing cerebral cortical activity, but also may be associated with respiratory and cardiovascular depression. Pentobarbital (Nembutal) and secobarbital (Seconal) are the most commonly used of the short-acting barbiturates. They are administered orally or intramuscularly in a recommended dose of 50-200 mg for adult patients.

Chloral Hydrate. This is one of the oldest and safest hypnotics. It is especially useful in elderly patients in whom barbiturates may be contraindicated. The recommended dosage for adults is 0.5-1.0 g by mouth.

Antihistamines. Antihistamines such as hydroxyzine (Vistaril) and diphenhydramine (Benadryl) are useful for their sedative, antihistaminic, and antiemetic properties. They are commonly used to supplement the action of a narcotic premedicant. They are well tolerated and relatively safely administered to all age groups. Hydroxyzine and diphenhydramine are administered in dosages of 25-100 mg intramuscularly.

Narcotics

Morphine sulfate (10 mg) and meperidine (Demerol) (50-100 mg) are commonly used in premedication. These drugs are especially useful when pain is a component of the preoperative condition. Both these agents are associated with central nervous system and respiratory depression and on occasion nausea and vomiting.

Tranquilizers

Phenothiazines. These are useful preoperative medications, contributing excellent sedative, antiemetic, and antihistaminic properties. Many of the phenothiazines can be given orally as well as intramuscularly for preoperative medication. Commonly utilized premedicants in this group include: chlorpromazine (Thorazine) 15-50 mg, prochloroperazine (Compazine) 5-10 mg, and promethazine (Phenergan) 25-50 mg.

Benzodiazepines. Benzodiazepines are especially effective premedicants for local anesthesia because of the prophylactic protection they provide against seizures. Effective in both oral and intramuscular administration, diazepam (Valium) 5-10 mg, and chlordiazepoxide

(Librium) 25-50 mg are the most frequently used of this family.

Belladona Derivatives

Atropine sulfate (0.5 mg) and scopolamine (0.5 mg) are the two most commonly employed belladona agents. Used for their antimuscarinic properties, their most beneficial action is that of drying of secretions of the upper airway. Scopolamine is associated with more frequent central nervous system effects (sedation, excitation) and is less effective in preventing reflex bradycardia than is atropine.

One of the authors (KJL) uses 100-mg secobarbital (Seconal) PO 2 hours preoperatively; 8- to 10-mg morphine IM, on call; 8- to 10-mg diazepam (Valium) IM, on call. The use of valium also serves as an adjunct to protect against local anesthetic toxicity reactions, particularly in rhinoplasty or other procedures in which larger quantities of local anesthetic agents are used. The use of morphine cause pylorospasm, thus preventing absorption of the Seconal if given simultaneously. Besides the preoperative medication, it is essential to inform the patient preoperatively of the procedure "step by step" and what to expect throughout. This preoperative counseling has been referred to by Jackson as the "sermon".

Intravenous Sedation

Diazepam

Diazepam (Valium)(2.5-5 mg) given in slow intravenous increments, is a relatively safe and effective sedative. It is useful in supplementing local anesthesia to optimize clinical conditions. Its effectiveness for tranquilization and prophylaxis against local anesthetic-induced seizures has been previously stressed. Very rapid administration is occasionally associated with transient respiratory depression.

Innovar

Innovar is a mixture of droperidol (2.5 mg/mL) and fentanyl (0.5 mg/mL). When given slowly in small increments (o.5 mL) it may enhance intraoperative sedation during local anesthesia. When given too rapidly, it may be associated with hypotension and chest wall spasm.

Barbiturates

Sodium pentobarbital (Nembutal) and secobarbital (Seconal) when given in small increments intravenously (25-50 mg) may provide safe sedation to supplement local anesthesia. Too rapid administration may produce respiratory depression.

Block Techniques

In virtually all blocks, eliciting an appropriate paresthesia before injection of the agent helps to insure success.

Laryngoscopy, Tracheoscopy

The larynx and trachea receive their sensory nerve supply from the superior and inferior laryngeal nerves, which are branches of the vagus nerve.

1. Anesthesia may be provided to the larynx by the topical application of local anesthesia (using a laryngeal syringe) to the mucous membrane of the pyriform fossa (dep to which runs the superior laryngeal nerve) and to the laryngeal surface of the epiglottis and the vocal folds (Fig. 25-1).

2. Local anesthesia of the larynx and trachea also may be accomplished by the percutaneous infiltration of local anesthetic solution around the superior laryngeal nerve and the transtracheal application of local anesthetic to the tracheal mucosa.

For percutaneous infiltration, the superior laryngeal nerve is located as it pierces the thyrohyoid membrane (Fig. 25-2).

a. Palpate the greater cornu of the hyoid bone.

b. Insert a 25-gauge needle approximately 1 cm caudal to this landmark.

c. The needle is inserted to a depth of approximately 1 cm until the firm consistency of the thyrohyoid membrane is identified.

d. Inject of 3 mL of local anesthetic solution.

The transtracheal application of local anesthesia requires the insertion of a 25-gauge needle through the cricothyroid membrane in the midline (Fig. 25-3).

a. Introduce the 25-gauge needle in the midline between the thyroid and cricoid cartilages.

b. Puncture the cricothyroid membrane. This is readily felt as a "pop". Free aspiration of air with the attached syringe verifies the intratracheal position of the needle tip.

c. Instill 4 mL of local anesthetic solution. In addition to anesthesia of the larynx and trachea (1 and 2 above), the topical application of local anesthesia to the oropharynx is required for adequate visualization for laryngoscopy and tracheoscopy.

Reduction of Dislocated Temporomandibular Joint

In the common presentation of temporomandibular dislocation, the condyle rests on the anterior slope of the articular eminence (Fig. 25-4). There is intense pain and severe spasm of the surrounding mandibular musculature. Reduction of this dislocation may frequently be accomplished by the unilateral, intracapsular injection of local anesthesia.

1. With the head of the condyloid process locked anteriorly, the depression of the glenoid fossa is easily palpated.

2. The needle is inserted into the depression of the glenoid fossa, and directed anteriorly towards the head of the condyloid process.

3. When the condyloid process is contacted, the needle is slightly withdrawn.

4. Instill 2 mL of local anesthetic solution into the capsule.

Reduction and Fixation of a Mandibular Fracture

Complete anesthesia for reduction and fixation of a mandibular fracture requires adequate anesthesia of the maxillary and mandibular branches of the trigeminal nerve and superficial branches of the cervical plexus (Fig. 25-5).

1. The mandibular branch of the trigeminal nerve is readily anesthetized near its exit from the skull through the foramen ovale (Fig. 25-6).

a. A skin wheal is raised just over the posterior inferior surface of the mandibular notch.

b. An 8-cm needle is inserted transversely and slightly anteriorly to a depth of 4-5 cm where it comes into contact with the lateral pterygoid plate.

c. The needle is withdrawn slightly and directed in a more anterior superior direction to pass anterior to the pterygoid plate into the pterygopalatine fossa.

d. The needle is advanced another 0.5-1.5 cm until paresthesia is elicited. A total of 5-10 mL of local anesthetic solution is deposited.

The most frequent complications of mandibular and maxillary nerve block is hemorrhage into the cheek. This usually is managed conservatively. Subarachnoid injections and facial nerve blocks are two other rarely reported complications.

3. The superficial branches of the cervical plexus are easily blocked as they emerge along the posterior margin of the sternocleidomastoid muscle. Starting at the midpoint of the posterior margin of the sternocleidomastoid muscle, infiltration is accomplished along the posterior margin of this muscle using 10-15 mL of anesthetic solution.

Otology

The sensory innervation of the external ear is illustrated in Fig. 25.8. The middle ear receives its sensory innervation through the tympanic plexus (V3, IX, and X).

V3 --> Auriculotemporal nerve.
IX --> Jacobson's nerve.
X --> Auricular nerve.

Myringotomy

1. Inject the cartilaginous and bony junction of the external auditory canal.

2. Instead of introducing the local anesthetic through the classical 12 o'clock, 3 o'clock, 6 o'clock, and 9 o'clock infiltration, infiltrate at 12 o'clock, 2 o'clock, 4 o'clock, 6 o'clock, 8 o'clock, and 10 o'clock. In this manner, other than the first injection site, the subsequent injection sites are already anesthetized before the needle prick. The patient feels one needle prick instead of the classic four pricks. For myringotomy alone, it is not necessary to infiltrate the skin of the bony canal wall, thus no local anesthetic agent should infiltrate into the middle ear cavity. (See: Complications of Local Anesthetic in Stapedectomy.)

Stapedectomy

In addition to the technique described for myringotomy, it is necessary to infiltrate the tympanomeatal flap. Besides assuring adequate anesthesia, this provides vasoconstriction (1% lidocaine (Xylocaine) with epinephrine 1:100.000) for hemostasis).

Complications. Two transient complications arising from the lidocatine, which migrated from the tympanomeatal flap to the middle ear cavity, have been noted in local anesthetic infiltration for stapedectomy:

1. Temporary facial nerve paralysis. This is due to the local anesthetic coming into contact with the dehiscent facial nerve. Patience and reassurance for a few hours will resolve the problem.

2. Violent vertigo with nystagmus (similar to Ménière's attack) can occur 45 minutes after the infiltration. Provided no damage has been done to the vestibular labyrinth, this is secondary to the effect of lidocaine on the membranous labyrinth through the oval or round windows.

These two complications are particularly distressing if they occur after an office myringotomy. Hence, it is the author's (KJL) advice that no infiltration in the skin of the bony canal wall is needed for myringotomy. The infiltration at the junction of the bony and cartilaginous canal will not reach the middle ear cavity.

Tympanoplasty and Mastoidectomy (Canalplasty, Meatoplasty)

This procedure is usually performed under general anesthesia. However, it is quite possible to have it performed under local anesthesia. In addition to the stapedectomy infiltration, postauricular and conchal infiltration are necessary (see Fig. 25-8 for the sensory innervation). The skin of the anterior canal wall needs to be anesthetized if surgery is to include that anatomic site.

Nasal Surgery

Nasal Polypectomy

Cocaine pledgets along the mucosal surfaces, as well as in contact with the sphenopalatine ganglion, supply adequate anesthesia for polypectomy. Occasionally it is necessary to supplement this with external infiltration, as in rhinoplasty.

Septoplasty and Rhinoplasty

The sensory innervation of the septum and external nose is illustrated in Figs. 25-9, 25-10, 25-11, 25-12, 25-13. Besides local infiltration as shown i nFig. 25-13, cocaine pledgets along the mucosal surfaces and sphenopalatine ganglion are used. For best hemostasis and anesthesia results, it is wise to wait at least 20 minutes before performing the surgery.

Table 25-5a. Nasal Sensory Innervation

V1 --> Lacrimal nerve.
V1 --> Frontal nerve --> Supraorbital nerve.
V1 --> Frontal nerve --> Supratrochlear nerve.
V1 --> Nasociliary nerve --> Anterior ethmoid --> Anterior ethmoid cells.
V1 --> Nasociliary nerve --> Cartilaginous nose, internally and externally.
V1 --> Nasociliary nerve --> Med int nasal branch --> Upper and ant septum.
V1 --> Nasociliary nerve --> Lat int nasal branch --> Lat wall of nose.
V1 --> Nasociliary nerve --> Infratrochlear --> Skin of the root of the nose.
V1 --> Nasociliary nerve --> Post ethmoid nerve --> Sphenoid / post eth cells.

Table 25-5b.

V2 --> Infraorbital nerve --> Ant sup dental nerve branches before the infraorbital foramen. It exits from the region of the anterior nasal spine to innervate that region. Thus it is necessary to inject this area to achieve anesthesia for rhinoplasty.
V2 --> Infraorbital nerve --> The rest of the nerve exits at the infraorbital foramen to innervate the palpebra, conjunctiva, nasal, labial areas.

Table 25-5c.

V2 - Sphenopalatine ganglion --> Greater palatine nerve --> Soft and hard palate.
V2 - Sphenopalatine ganglion --> Greater palatine nerve --> Nasal branches to the floor of the nose.
V2 - Sphenopalatine ganglion --> Greater palatine nerve --> Anastomose with long sphenopalatine nerve.
V2 - Sphenopalatine ganglion --> Lesser palatine nerve --> Soft palate and tonsil.
V2 - Sphenopalatine ganglion --> Long sphenopalatine nerve --> Root of nose --> Septum and vomer --> Incisive foramen --> Hard palate mucosa --> Anastomose with greater palatine nerve.
V2 - Sphenopalatine ganglion --> Long sphenopalatine nerve --> Superior and middle conchae and posterior septum.

V2 - Sphenopalatine ganglion --> Short sphenopalatine nerve --> Superior and middle conchae and posterior septum.
V2 - Sphenopalatine ganglion --> Posterior superior dental nerve --> Gingiva, cheek, teeth, maxillary sinus mucosa.

Sinus Surgery

Caldwell-Luc

To achieve good anesthesia for this procedure, one needs to block the infraorbital nerve, the sphenopalatine ganglion, and the posterior superior dental nerve. The posterior superior dental nerve exits from the maxillary nerve adjacent to the sphenopalatine ganglion. To block the sphenopalatine ganglion and posterior superior dental nerve, introduce the local anesthesia through the greater palatine foramen via a curved needle.

Further topical anesthesia is applied with cocaine pledgets intranasally against the sphenopalatine ganglion. Local infiltration of the mucosa in the canine fossa will supply the hemostasis needed over the line of incision.

Ethmoid Sinuses

The sensory innervation of the ethmoid sinuses is intertwined with that of the nose and septum. In addition, it is innervated by the anterior ethmoid nerve (branch of the nasociliary, V1) and the posterior ethmoid nerve (branch of infratrochlear, V1).

Sphenoid Sinuses

The sensory innervation is from the pharyngeal branch of the maxillary nerve as well as the posterior ethmoid nerve.

General Anesthesia

Definition

General anesthesia is the chemically induced, reversible loss of conscious.

Mechanism of Action

The mechanism of action of general anesthetics remains a controversial issue. Most likely, these agents block multisynaptic neuronal pathways, such as in the reticular activating system of the brain stem. General anesthetics act on virtually all cell membranes and, therefore, affect all organs and systems.

General Anesthetics Agents

Inhalation Anesthetic Agents

These anesthetics are administered via an inhalation route and absorption occurs through the alveolar-pulmonary capillary interface.

1. Nitrous oxide is one of the oldest and remains one of the most useful of inhalation anesthetics. It usually is employed in an inspired concentration of 50-75% with oxygen. Nitrous oxide is a relatively safe but weak anesthetic agent.

2. Diethyl ether is among the older of the clinically useful general anesthetics and remains a relatively safe and useful agent. One of the major advantages of diethyl ether is the ventilatory stimulation which occurs at anesthetic stages. This is in marked contrast to the respiratory depression seen with virtually all other general anesthetics. This property makes diethyl ether a useful agent for bronchoscopic examination. The flammability hazard with diethyl ether has limited its use in recent years.

3. Cyclopropane is a potent anesthetic, commonly used in inhalation concentrations from 6-20%. Its usefulness is also limited by its flammability characteristics.

4. Halothane is an example of the series of halogenated hydrocarbons that have been synthesized and introduced into clinical practice during the recent 2 or 3 decades. It is a potent agent providing profound anesthesia when administered in concentrations of 1-2%. It is associated with cardiovascular and respiratory depression.

5. Methoxyflurane, Enflurane, and Isoflurane are additional members of the halogenated hydrocarbon family having many properties and complications similar to those of halothane. Methoxyflurane has been implicated in the production of a dose-related, high-output renal failure.

Intravenous Anesthetics

Thesxe anesthetics are administered via an intravenous route.

1. Thiopental is an ultrashort-acting thiobarbiturate. It has become one of the most widely used agents for the induction of anesthesia. Its use may be associated with profound cardiovascular and respiratory depression.

2. Ketamine is a newer intravenous anesthetic of the phencyclidine class of drugs. Ketamine induces a peculiar state, called dissociative anesthesia, in which patients are unresponsive to noxious stimuli but may appear to be awake with open eyes and spontaneous movement. Of significance, the pharyngeal and laryngeal reflexes remain intact until very deep levels of ketamine anesthesia are attained.

3. Innovar is a mixture of two drugs, droperidol (a major tranquilizer) and fentanyl (a potent narcotic). It acts much like other so-called "neuroleptic coctails" (morphine and chlorpromazine, meperidine and diazepam) and usually is administered in combination with

nitrous oxide and occasionally a short-acting barbiturate drug.

4. Butorphanol and Nalbuphine are examples of the narcotic-related drugs with mixed agonist-antagonist properties. These are being introduced into clinical practice because of their promise of providing potent analgesia with fewer of the undesired narcotic side effects (respiratory depression, addiction potential).

5. Diazepam is a major tranquilizer of the benzodiazepine family. In large intravenous doses in combination with an inhalation agent such as nitrous oxide it is useful for the induction and maintenance of general anesthesia.

Neuromuscular Blockers

Neuromuscular blocking agents act at the neuromuscular junction to induce a state of muscle paralysis. These are subclassified into the depolarizing agents (succinylcholine) and the nondepolarizing drugs (d-tubocurarine).

Complications

The discussion of the complications of general anesthetics will be confined to those of particular interest to otolaryngologists.

1. Aspiration pneumonitis may occur during general anesthesia as a consequence of the obtundation of laryngeal protective reflexes. Foreign matter which is permitted to accumulate in the pharynx (blood, gastric contents) will gain ready access to the pulmonary parenchyma. The result is the clinical syndrome of aspiration pneumonitis.

2. Cardiac arrhythmias: Most of the inhalation anesthetics are associated with a sensitization of the myocardium to catecholamines. In the presence of excess catecholamines, endogenously or exogenously produced, patients may develop ventricular arrhythmias and ventricular fibrillation. It is recommended that the exogenous administration of epinephrine be limited to a concentration of 1:100.000 and a total dose of 10 mL in any given 10 minute period when given in the presence of these anesthetics.

3. Hepatitis has been demonstrated to be a potential complication of virtually any anesthetic and surgical technique. A well publicized but poorly documented entity, so called "halothane hepatitis" has gained widespread notoriety. This type of hepatitis may be associated with an unusual metabolite of halothane to which certain sensitive individuals develop an allergic reaction. If this does exist as a unique entity it is extremely uncommon.

4. Malignant hyperpyrexia is a rare adverse reaction to anesthetics (occurring in approximately 1:15.000 anesthetic administrations). It is associated with a rapid rise in temperature to as high as 112°F and cardiovascular collapse and shock. Treatment consists of rapid termination of surgery and anesthesia, submersion of the patient into an ice bath, and general supportive measures. The mortality of this complication of anesthesia remains at greater than 50%.

5. Nitrous oxide-induced elevation in middle ear pressure: Because of its large blood solubility relative to nitrogen, nitrous oxide is well known for diffusing into closed gas spaces and producing an elevation in intraluminal pressures. In the case of tympanic surgery, diffusion of nitrous oxide into the middle ear may produce bulging of the tympanic graft. The distention is readily reversible if the nitrous oxide is discontinued and 100% oxygen is substituted.