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Local-regional anesthesia in dentistry and OMF surgery

Course notes

**for students and residents
Dentistry Faculty**

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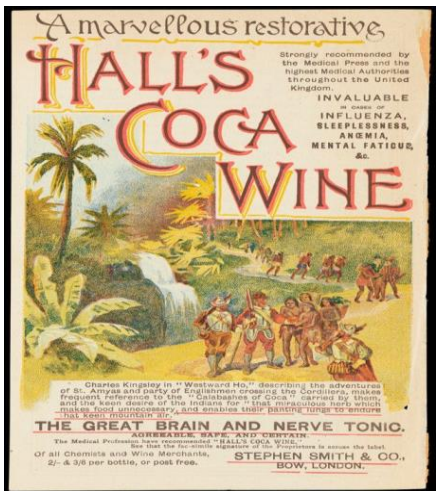
INTRODUCTION: A SHORT HISTORICAL BACKGROUND OF LOCAL ANESTHESIA

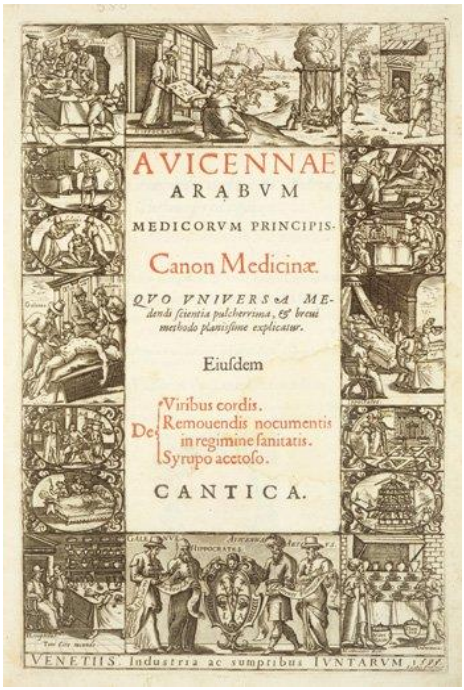
Prior to the 1840s, patients were not only anxious, but even terrified while entering the operating room. Why? Because there was no anesthesia. In his book *"We Have Conquered Pain"*, Dennis Fradin states: "Surgeons were known to enter the operating room keeping a bottle of whiskey in each hand: one for the patient and the other for the doctor to help him bear the patient's cries."

Inebriating or drugging the patient



Doctors, dentists and patients were able to do almost everything to minimize the pain caused during the surgery. In this way, Chinese and Indian doctors had been using marijuana and hashish. Also, in various parts of the world, opium and alcohol were often used in this concern. Dioscorides, a Greek doctor from ancient times and the first person known to use the term "anesthesia", had attributed anesthetic properties to mandrake and wine portions. Later, some doctors had even used it for hypnotism.

However, pain relief was unsatisfactory. Therefore, surgeons and dentists were working as quickly as possible. In fact, they were rated according to their working speed. But even the fastest doctor could cause enormous pain. That's the reason people usually found better to suffer from all possible diseases, starting from tumors till all teeth decay, rather than to deal with a surgery or dental extraction agony.



Sweet vitriol and laughing gas

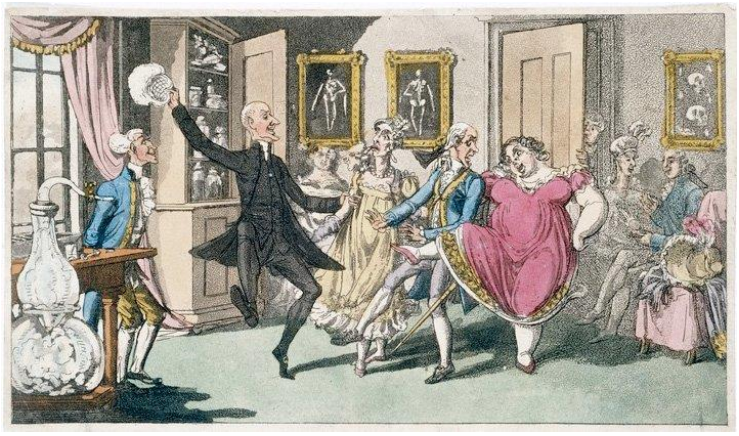
In 1275, the Spanish doctor Ramón Llull, while experimenting with chemicals, had obtained a volatile, flammable liquid, that he called sweet vitriol. In the sixteenth century, a Swiss doctor, Paracelsus gave chickens to inhale sweet vitriol and noticed they hadn't only fallen asleep, but even felt no pain. Neither Llull nor Paracelsus had tested this substance on humans. In 1730, the German chemist Frobenius gave this substance its

current name, ether, whose Greek meaning is "heavenly". But it would have taken 112 years until the anesthetic properties of the ether had been completely understood.

Meanwhile, in 1772, the English scientist Joseph Priestley discovered nitrous oxide. At first, people thought it was a lethal gas, whenever taking it in small doses. However, in 1799, the British chemist and inventor Humphry Davy decided to find out the truth by having tested it on himself. He was very surprised to discover the nitrous oxide had made him laugh, so, he called it laughing gas. Davy wrote about the possible anesthetic properties of nitrous oxide, but at the time, no one had continued to do the research.

Ethereal and laughing gas parties

Davy's buffoons presented under the influence of the laughing gas, he had been depending on for a while, became famous. Soon, the inhalation of the entertainment gas began to take its toll on the public. As special part of the program, even the travelling comedians were asking the volunteers from the audience to get on the stage and inhale nitrogen oxide. The gas dispelled any embarrassment, and soon, the sudden buffoons of the volunteers made the audience burst into laugh.



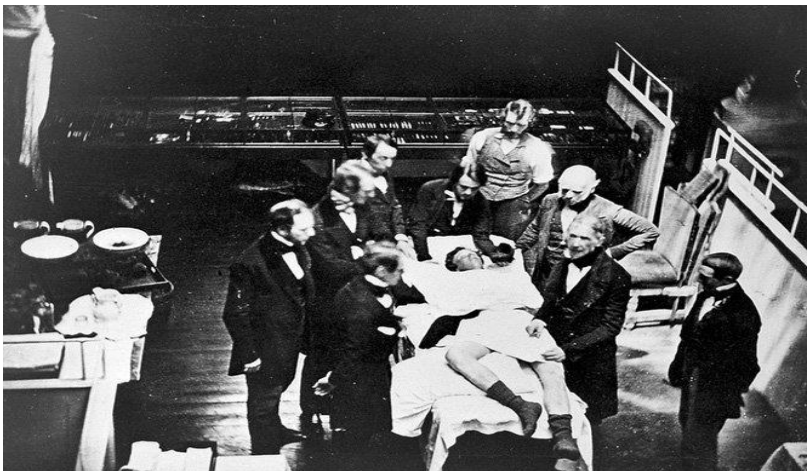
Nearly that time, the use of ether to entertain the audience was spreading. One day, a young American doctor, Crawford Long, noticed his friends did not feel any pain when they got wounded as they were wandering under the influence of ether. At once he realized the advantage of using it in surgery. A participant to one of these "ethereal parties", James Venable, a student who was having two tumors he wanted to remove, was the right person for that situation. However, due to the fear of the pain caused during the surgery, Venable continued to delay undergoing the surgery. Therefore, Dr. Long proposed to him a surgery hold under the etheric influence. Venable agreed, and on March the 30th, 1842, he had a pain free surgery. However, Dr. Long had not issued his discovery until 1849.

Dentists also discover anesthesia

In December 1844, the American dentist Horace Wells attended a performance held by a traveling band, during which some Gardner Colton proved the effects of nitrous oxide. As Wells

offered to test the gas, he remained aware enough to notice another participant to have hit a hard material bank and feel no pain, though he was bleeding. That evening, Wells decided to try the nitrous oxide in dental practice, but only after having tested it on himself. Then he asked Colton to bring him the gas, and John Riggs, a fellow dentist, to extract a wisdom tooth that hurt him. Extraction was a real success.

Wells decided to make his discovery public by showing it to his colleagues. However, as he was very excited during the demonstration, he turned out to administer the patient too little gas, who shouted after having his tooth extracted. Thereupon, the people attending the extraction demonstration began to kick Wells. Yet they should have asked the patient about the way he felt, as he told Wells later that besides the shouts it ached him very little.



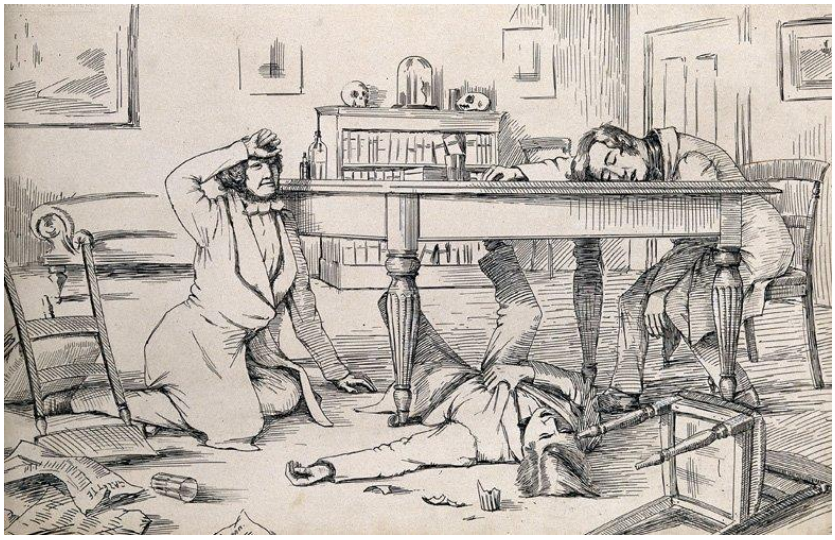
On September 30th, 1846, another American dentist, William Morton, performed a pain-free dental extraction to a patient who was willing to inhale ether, the same substance used by Long in 1842. Morton prepared the ether in assistance of the remarkable chemist Charles Thomas Jackson. Unlike Long, Morton organized a public demonstration concerning the anesthetic properties of ether on a patient who was undergoing a surgery. Morton anesthetized the patient on October 16, 1846 in Boston (Massachusetts). Then, the surgeon Dr. Warren performed the surgery by removing an excess growth from the lower part of the patient's jaw. The surgery was a great success. Not long after, the news had spread at the lightning flash speed throughout the United States and Europe.

Extra discoveries

As the result of these promising discoveries, all gases were still applied in different experiments. In 1847 chloroform had been successfully used, discovered earlier in 1831. Soon, in some areas, it had got required as an anesthetic. Not long after, chloroform was administered to women while giving birth, including even Queen Victoria of England in April 1853.

Unfortunately, the history of general anesthesia has somewhat got darken. A strong dispute broke out concerning the scientist who should be credited with anesthesia discovering (without considering its chemical compounds), Long, Wells, Morton or Jackson, the remarkable chemist to have assisted Morton. Although it has never got to a consensus, many people recognize all four men's contribution in discovering anesthesia.

Meanwhile, progress has been made in local anesthesia, often called regional anesthesia. Anesthetics have been given patients to allow them remain glossy while a certain part of the body is anesthetized or insensitive. Nowadays, dental surgeons use properly local anesthetics before performing dental and gum work, while doctors use them to minor or repairing surgeries resulting from different injures. Anesthesiologists tend to administer local anesthetics to the women giving birth.



During the time, anesthesiology has become an independent medical discipline. Actually, anesthesiologists take part in preparing patients for surgery. They perform anesthetizing procedures using intelligent equipment and complex anesthetics containing different chemical agents combined with oxygen. In fact, many patients may not even know their doctor has used anesthetic gases, as they are often given only after the first intravenous anesthesia administration. The anesthesiologist also plays an important role in relieving the postoperative pain.

THE PAIN AND THE NERVOUS IMPULSE CONDUCTION

Pain is an experience commonly encountered during the life of a being. For humans, simultaneously with their evolution and, subsequently, their human society transformation, the concept of pain origin and development has passed an interesting trace. At first, the notion involved a much more complex sphere of suffering. Besides the physical pain, the concept included the meaning of mental depression, poverty, loss of social and/or political status along with other deprivations. According to religious and socio-philosophical theories, suffering signified either the expression of divine discipline for human sins, the way to punish the violation of the laws of society, or a coercive-educational measure. During the nineteenth century social philosophers and biologists realized the boundaries between suffering and actual physical pain, the doctors understanding their duty to treat pain that represent the body's reaction to numerous aggressive, harmful stimuli.

For a long time, the necessity of preventing pain has not been clearly defined. Within the medical and, especially the surgical environment, pain was considered as a "physiological" component, indissolubly related to the disease and / or the surgical act. Thus, the words addressed by the surgeons to their patients are well known: "suffer or die". The attitude of the medical staff towards pain begins to change from the second half of the 19th century, while discovering the analgesic effects of opium, nitrous oxide, sulfuric ether, as well as their use to minimize the pain intensity during the surgery.

1846 is considered the year of anesthesia appearance as a future specialty, once the first "pain - free" surgeries have been performed (Wells, Morton).

During the last 150 years, remarkable discoveries in neurophysiology, neurobiochemistry and neuropharmacology have contributed to getting the in-depth knowledge regarding the mechanisms of painful phenomena, as well as, to the radical change in the attitude towards pain and the spectacular evolution of its therapeutic modalities. In addition, the cultural-educational deveopment determines, in most cases, today's the patients to tell the doctor: "no matter what you would do to me, it just didn't hurt!".

Definition

Pain is ***"an unpleasant sensory and emotional experience associated with a real, potential tissue injury or described in terms of such injury."*** The injuries causing this complex body response defined as pain can be real (trauma, burn, surgical incision, etc.), perceived as real (reflected pain: pain coming from the right scapulo-humeral region to the biliary colic), or potential (pain inside the "phantom" limb, after removal of some anatomical regions: lower amputated limb, radical mastectomy, etc.). Firstly, it should be specified the role of the consciousness state in the

pain onset. According to the definition, this unpleasant experience accompanied by a complex response of the body can occur only when the consciousness state is present. To the anesthetized patient, for example, we will record a series of reactions to an intense or incomplete painful surgical stimulus blocked in its transmission, but not the per se pain, as defined by the IASP. Regarding current, experimental and clinical data, pain is, in fact, the expression of the complex sum between the initial painful sensation, onset by a painful stimulus action with neurophysiological and neuro-biochemical basis, and the "algic" behavior, comprising the total response reactions of the body towards this painful sensation.

Terminology:

Allodynia: pain caused by a stimulus that under normal conditions is not painful.

Analgesia: absence of pain whenether the painful stimuli are present.

Painful anesthesia: the presence of pain in an anesthetized area or region.

Causalgia: persistent, burn-like pain occur after the traumatic injury of a nerve.

Central pain: pain generated by lesions to the central nervous system.

Hyperalgesia: increased sensitivity to painful stimuli of ordinary / low intensity.

Hyperesthesia: increased sensitivity to any type of stimulus.

Hyperpathy: painful syndrome characterized by hyperreaction and postsensation to painful stimuli.

Hypoalgesia: low sensitivity to painful stimuli.

Hypoesthesia: low sensitivity to any type of stimulus.

Modulation: a lesional stimulus processing (the painful informational input) to diminish or amplify it.

Neuralgia: pain within the distribution area of a nerve.

Neuropathy: functional or anatomical impairment of a nerve.

Nociceptor: sensitive preferential receptor to a painful or potentially painful stimulus action.

Nociception: the complex of the electrochemical phenomena occuring along the painful informational input path starting from the situs of an active tissue injury (peripheral) to the level pain perception (central) level.The electrical and chemical phenomena of nociception are: capture, transduction, transmission and complex processing of painful informational input.

Pain threshold: the minimum intensity required to a stimulus to initiate nociception.

Pain tolerance threshold: the maximum intensity value of a painful stimulus to be tolerated without initiating nociception.

Damage/ harmful stimulus: stimulus that causes tissue lesions having pain as final perception.

Painful sensations

Painful sensations are caused by the free nerve endings of A and C fibers, also called nociceptors. The peripheral nervous system contains 12 pairs of cranial nerves and 31 pairs of spinal nerves. The peripheral nerve consists of the nerve fibers of a neuron cluster, anchored in connective tissue. Each fiber may be surrounded by a myelin sheath - the Schwann sheath, called myelinated fiber, or may be devoid of it - non-myelinated fiber. The peripheral nerve pathways, formed by these nerves, have afferent fibers (sensitive - from the receptor to the CNS) and efferent ones (motor - from the CNS to the effector organ). Sensitive fibers contain dendritic extensions, the motor ones - axons. Each axon is surrounded by connective tissue called endoneurium, the nerve bundle consisting of several axons is covered by perineurium and several bundles form the fiber coated by the epineurium (figure 1).

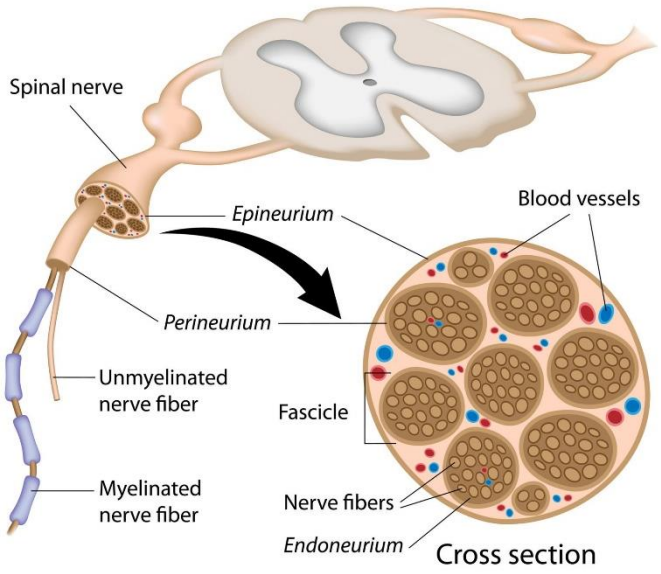


Figure 1. The structure of the nerve fiber

The painful sensations are generated by the free nerve endings of A and C fibers, also called nociceptors (Table 1).

Table 1. The type and functions of nerve fibers

The type of nerve fiber	**Function**
A-α (alfa)	Engine
A-β (beta)	Tactile, thermal, pressure sensitivity
A-γ (gamma)	Proprioceptive sensitivity
A-δ (delta)	Painful and thermal sensitivity
B	Sympathetic preganglionic fibers
C	Painful sensitivity

There are several types of nociceptors:

A. Unimodal nociceptors - respond to a single stimulus and have the adaptation property so that their response to the stimulus disappear in case of prolonged or permanent stimulation. They include mechanonociceptors, chemoreceptors, thermonociceptors.
B. Polymodal nociceptors - respond to a variety of mechanical, thermal and chemical stimuli and do not have adaptation phenomena, therefore, the response to the stimulus is present only during the excitation period.

Regarding nociceptor location the following types are detected:

1. Skin/superficial nociceptors (dermis, hypodermis and superficial fascia)
2. Joint/ deep nociceptors (muscle, tendon, fascia, periosteum, pericondrum, joint capsules)
3. Visceral nociceptor (subserousal, subepithelial, intra-adventitional, chorianic and inside the medium tunic of the blood vessels).

The hepatic, splenic, renal, bone tissues and cerebral cortex have no nociceptors.The nociceptive sensitivity of these organs is provided by the capsule or meningeal nociceptors.

Nerve impulse propagation

The non-myelinated nerve fiber structure includes axons surrounded by Schwann cell sheath (Figure 2). The specialized Schwann cells covering the axon of the myelinated nerve fiber produce multiple myelin layers to merge between the axon and the cytoplasm of the Schwann cell. The myelin sheath periodically interrupts inthe Ranvier nodes, where the axon comes in direct contact with the extracellular fluid. Ranvier nodes contain channels for ions to constantly run through.

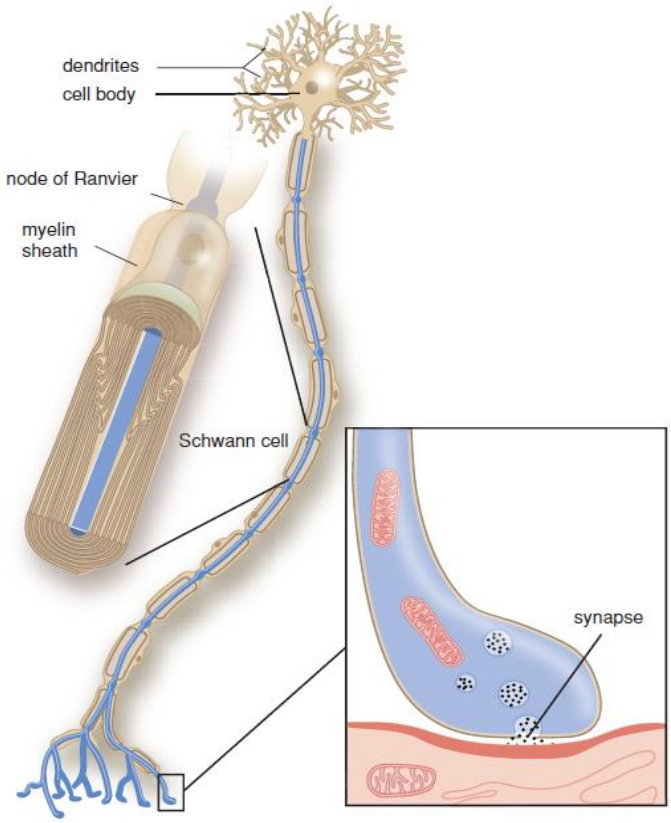


Figure 2. The nerve cell

The axon runs the electrical activity. The electric impulses, also called action potentials ripple along the axon.

The nerve impulse formation and propagation occurs due to the potential difference between the internal and external surface of the cell membrane, called resting potential. It is caused by Na + and K + ions distribution along the cell membrane.

Moreover, during the resting potential there is a high concentration of potassium (K +) inside the axon and a high concentration of sodium (Na +) outside it. As a result, the ion concentration difference produces an electric charge difference of about -70 mV between the inside and outside axon surface representing the membrane potential.

The ion concentration shift triggers the action potential to propagate along the axon (Figure 3). Under the influence of electrical, mechanical, physical or chemical stimulus, the membrane depolarization occurs with the Na + entry into the axon and a membrane potential of + 20 mV. The process is followed by the repolarization phase with the K + exit outside the axon. The normal concentration of Na + and K + is then restored through an active, energy-consuming process, that is, the membrane potential restoration by Na-K pump. Through successive depolarizations and repolarizations, the action potential propagates distally at a speed determined by the axon diameter and the myelin sheath presence.

Myelinated large diameter axons have a higher propagation speed than small diameter non-myelinated axons. Inside myelinated axons the action potential "jumps" from one Ranvier node to another, while inside non-myelinated axons it continuously propagates.

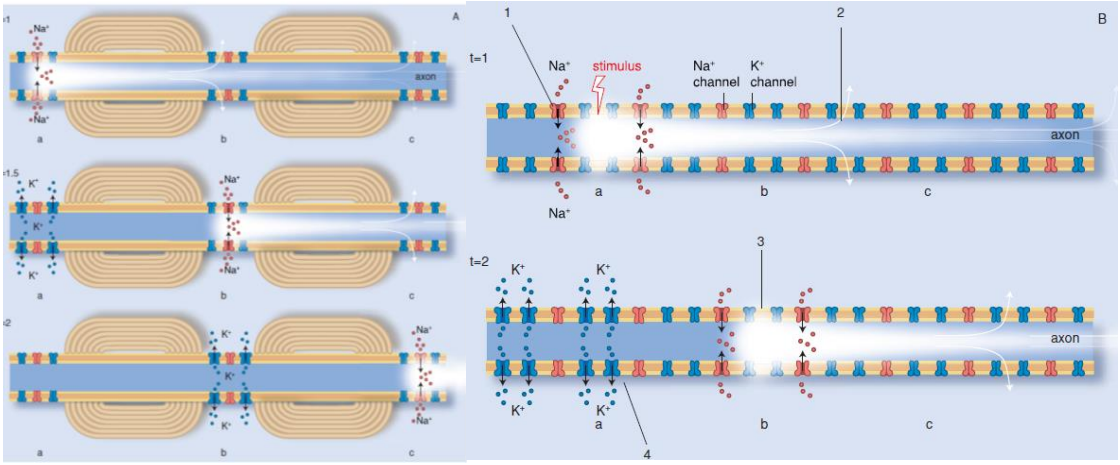


Figure 3. Types of nerve impulse conduction

The pain sensation is caused by the nociceptors stimulation - free nerve endings - activated by a wide variety of stimuli (mechanical, electrical, chemical, thermal) (figure 4). These stimuli must be strong enough to exceed the stimulation threshold. From nocireceptors, the painful stimulus is delivered along the axons of the sensory neurons to the posterior horns of the marrow, where it synapses with the second neuron.

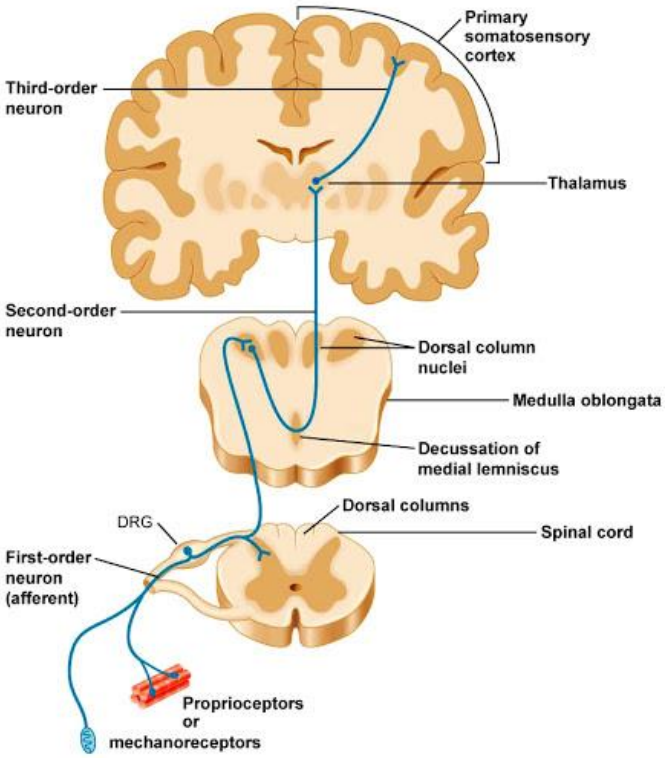


Figure 4. The afferent sensory pathways

Its axons cross the midline and raise to the thalamus, forming the lateral spinothalamic tract. The 3rd order neuron in the thalamus sends its axon into the sensory cortex, responsible for conscious perception of pain. Outside the spino-thalamo-cortical pathway, the painful stimuli information is conveyed to other parts of the brain as: bulb centers designated to regulate respiratory and cardiovascular functions; hypothalamus attributed to the regulation of autonomic and endocrine functions and "stress" response to pain; the limbic system, responsible for emotional states.

Pain intensity assessment

Acute pain assessment (eg. postoperative) is required for the correct management of the Algagille syndrome. The principle of self-assessment is applied, so the patient appreciates himself, within the "maximal - absent" limit, imagined by him. Pain intensity assessment include the following scores:

♣SVS (simple verbal score): no pain, minimal pain, medium pain, strong pain, unbearable pain (score 1-5);

♣SNS (simple numerical score): the patient is asked to "rate" the intensity of his pain, from 0 (no pain) to 10 (maximum imaginary pain). Maximum score: 11 points. Note: "the maximum imaginary pain" is the maximum pain intensity, that may exist in the patient's imagination, but not the one felt and caused by the surgery or the audience's "whispers".

♣VNS (visual-numerical score): the patient is given a "pain ruler", numbered from 0 to 10 and asked to position the cursor over the figure that best corresponds with the pain intensity felt by him within the limits "no pain = 0 points - maximum imaginary pain = 10 points".

Maximum score: 11 (0-10) (figure 5).

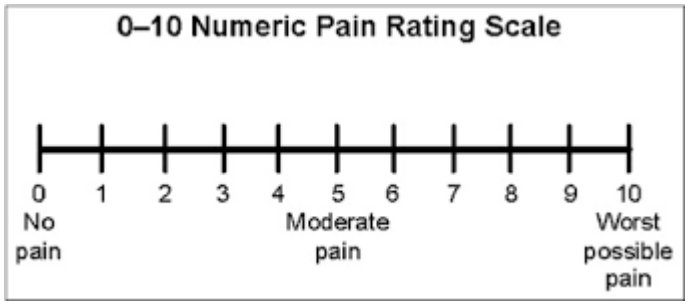


Figure 5. Visual-numerical score

♣VAS (visual-analogue score): similar to VNS. The difference is the ruler has two faces: one is numbered in millimeter (0-100), for the person measuring the pain and the other face is empty. The patient is asked to postion the cursor between the „no pain" and "the maximum imaginary pain" indicators on the empty face of the ruler. The person measuring the pain "sees" the result in millimeters on the opposite side of the ruler (Figure 6).

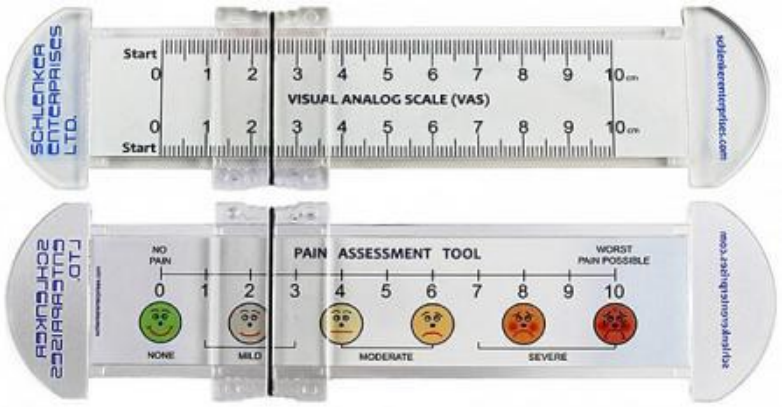


Figure 6. Visual-analogue score

♣ "Pain faces"score. The principle of use is identical to SVN (figure 7).

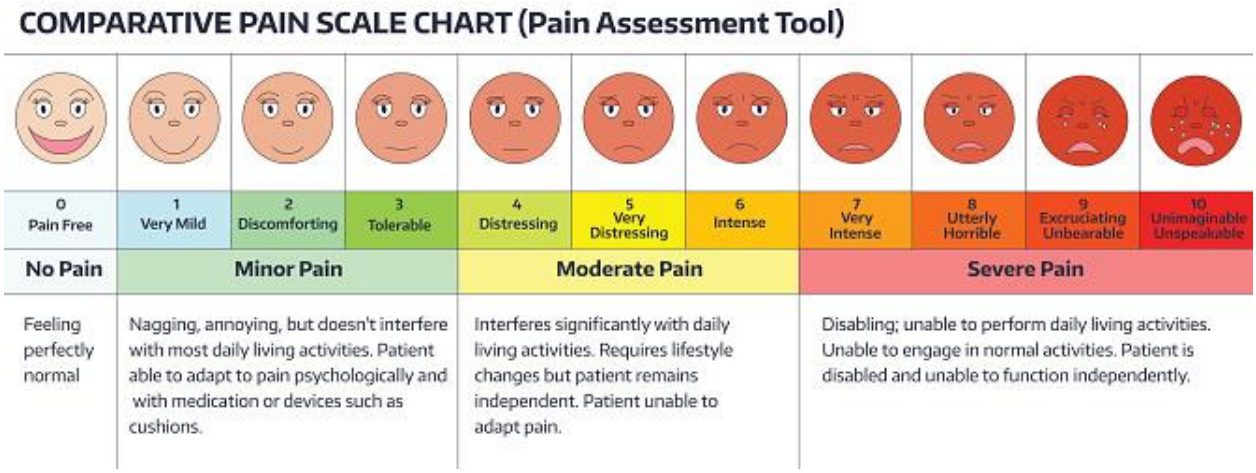


Figure 7. "Pain faces" score

ANATOMY OF TRIGEMINAL NERVE

The trigeminal nerve is the fifth paired and the largest cranial nerve, considered a mixed nerve due to its three terminal branches:

1. Ophthalmic nerve;
2. Maxillary nerve;
3. Mandibular nerve.

The trigeminal nerve includes three types of fibers:

- Motor fibers attributed to masticatory muscles;
- Sensory fibers for tactile, thermal, painful sensitivity to the skin of the face, the oral cavity mucosa, nasal fossa, paranasal sinuses and teeth;
- Vegetative secretory fibers.

The real origin of the trigeminal nerve

The trigeminal system includes four nuclei: one motor nucleus - the motor nucleus of the trigeminal nerve and three sensory nuclei - the main/chief trigeminal sensory nucleus, the trigeminal mesencephalic nucleus and the trigeminal spinal nucleus (figure 8).

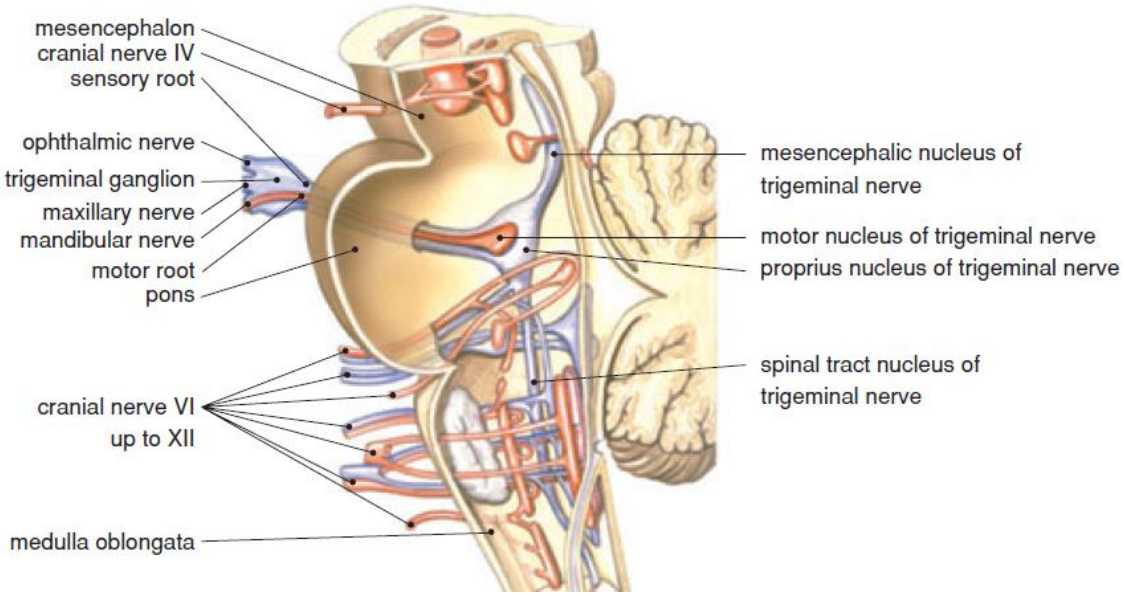


Figure 8. The nuclei of the trigeminal nerve

❖ **The trigeminal motor nucleus** is located in the midpons, medially positioned by the main sensory nucleus. It contains interneurons and bodies of alpha and gama multipolar motor neurons (branhiomotors), whose axons form the motor root of the trigeminal nerve at their exit from the pons. Branchiomotor fibers join the mandibular division of the trigeminal nerve. They are distributed to the masticatory muscles, as well as to the mylohyoid, digastric anterior belly, tensor tympani and tensor veli palatini muscles.

❖ **The sensitive trigeminal nuclei** consist of a cylindrical cell cluster extending between the mesencephalic and the first cervical spinal cord levels. Two of the nuclei - the main trigeminal sensory nucleus and the spinal trigeminal nucleus - receive the afferent terminals of the first-order pseudounipolar neurons, whose bodies are situated into the trigeminal ganglion. These nuclei serve as the first sensory relay station of the trigeminal system.

- ✓ **The chief sensory nucleus of the trigeminal nerve** is set in the mid pons. It receives the information about the fine touch and pressure sensations from mechanoreceptors.
- ✓ **The mesencephalic nucleus of the trigeminal nerve** is actually a true sensory ganglion (but not a nucleus) containg cells that are structurally and functionally similar to the ganglionic ones. The peripheral processes of these neurons are relatively larger in diameter, coated by a myelin sheath and responsible for conveying general proprioceptive information from the innervated and extraocular muscles, as well as the teeth periodontal ligament.
- ✓ **The spinal nucleus of the trigeminal nerves** is the largest of all three sensitive nuclei. It expands from the mid pon to the C3 level of the spinal cord continuing to the gelatinous substance of the spinal cord posterior horn. The nucleus includes three subnuclei: the oral subnucleus (pars oralis), the interpolar subnucleus (pars interpolaris) and the caudal subnucleus (pars caudals).

  Subnucleus oralis is responsible for carrying the light tactile sensation from the oro-facial region. Subnucleus interpolaris is also associated with the tactile sensation transmission, as well as dental pain, while subnucleus caudalis provides the delivery of nociceptive and thermal sensations from the head.

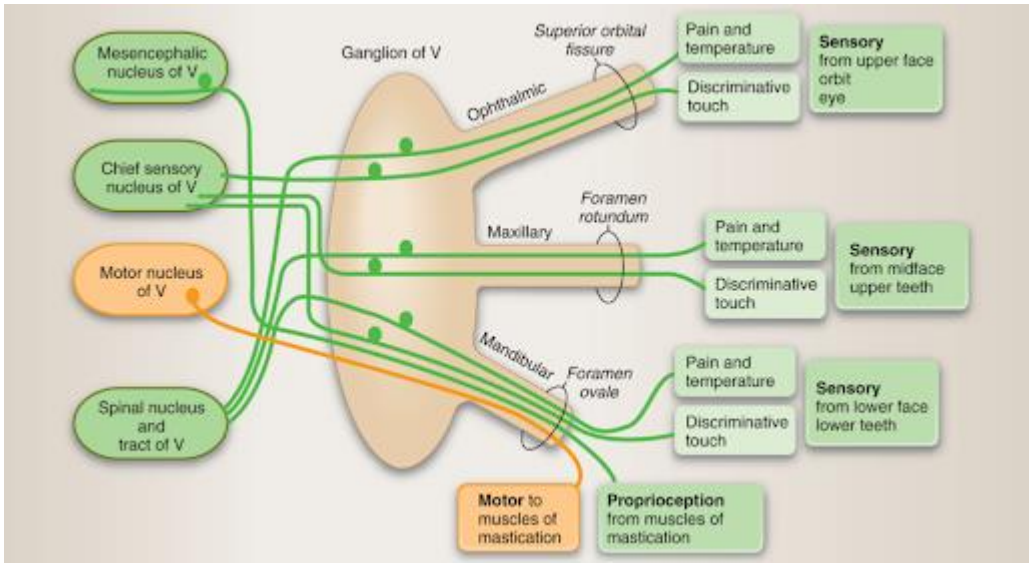


Figure 9. Information transmission from the trigeminal nuclei

The apparent origin of the trigeminal nerve

The apparent origin of the trigeminal is found on the anterior face of pons, at the boundary between the pontine pyramid and the middle cerebellar peduncle. The emergence of the trigeminal nerve has two roots: a sensitive voluminous root and another motor thin one.

The trigeminal nerve pathway and connections

Coming out from the origin, the two roots (sensory and motor) (figure 10) of the trigeminal nerve are directed obliquely upward, passing anteriorly and laterally to join the semilunar ganglion of Gasser. On their path, they go between the middle cerebellar peduncle and the postero-superior face of the petrous temporal bone apex. Then they cross the upper edge of apex, moving over the trigeminal cave and below the petrosal superior sinus.

At first, **the sensory root** is round, then it flattens from top to bottom as it approaches the semilunar ganglion. The sensitive root continues to travel towards the posterior edge of the semilunar ganglion through a plexiform formation called the trigeminal triangular plexus.

Initially, **the motor root** is medially positioned to the sensory root, then passing below it, the root gets a lateral and anterior direction. The motor root crosses the lower face of the semilunar ganglion, goes straight to and connects the mandibular branch of the trigeminal nerve. With this, the root joins at the oval hole (the foramen ovale) level placed to its base.

The trigeminal roots are coated in their origins by the pia mater membrane travelling towards the subarachnoid, the arachnoid and the subdural space to reach Meckel's cave.

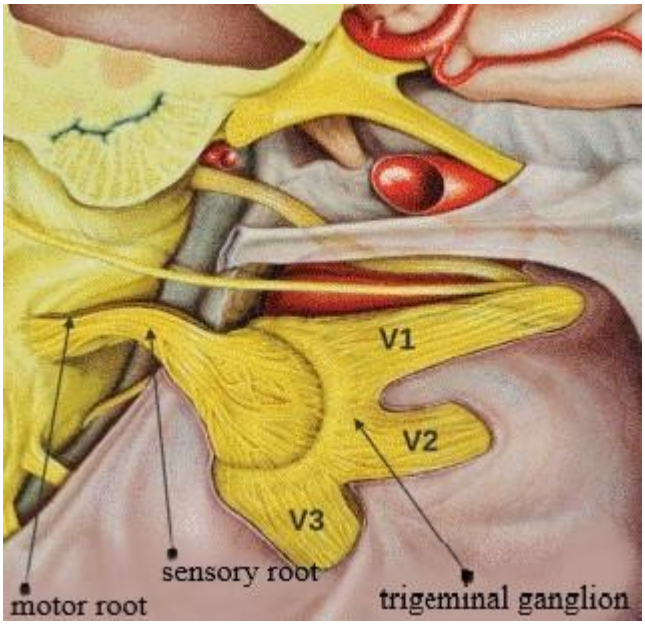


Figure 10. The roots of the trigeminal nerve

Meckel's cave (Meckel's cavity)

Meckel's cavity is a deduplication of the dura mater membrane. It represents a lodge depression to house the Gasserian semilunar ganglion. Meckel's cavity presents posteriorly an orifice the two trigeminal roots pass through, while its anterior face includes three extensions called tunnels. The three branches of the trigemen go through the tunnels. The medial tunnel is crossed by the ophthalmic nerve, the maxillary nerve passes through the middle tunnel, as well as the mandibular nerve emerge the lateral one.

Gasserian semilunar ganglion

Gasserian semilunar ganglion is attached to the trigeminal sensory root. It (Figure 11) occupies the trigeminal impression covering the anterior superior surface of the petrous temporal bone apex inside Meckel's cavity. The ganglion has a semilunar shape containing two margins, two terminals and two surfaces. Its posterior margin is concave to comprise the sensory root of the trigeminal nerve, while its convex anterior margin gives rise to the three branches of the trigeminal: medial ophthalmic, mid-maxillary and lateral mandibular divisions.

The semilunar ganglion is similar to a spinal ganglion, as it consists of pseudounipolar neurons with peripheral and central extensions. Peripheral extension is a constitutive part of the sensory branches of the trigeminal nerve. Central extension of neurons contributes in forming its sensory root.

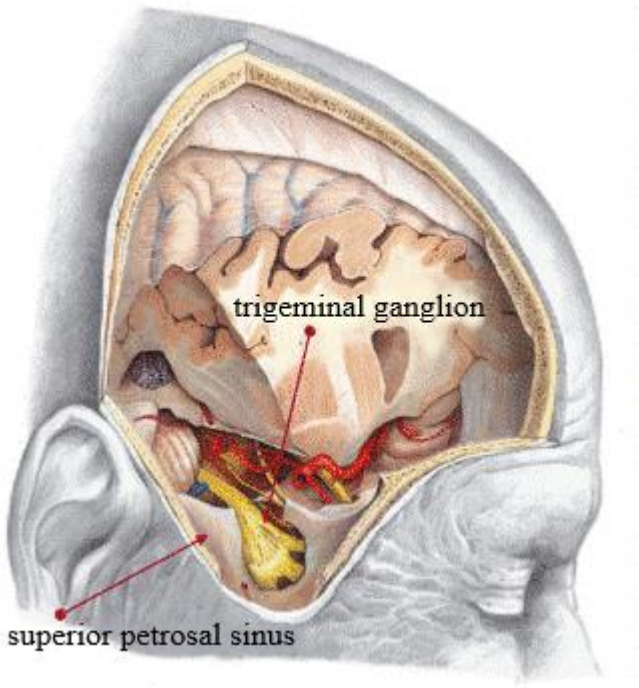


Figure 11. The trigeminal ganglion

The trigeminal nerve branches

I. Willis' ophthalmic nerve

Willis' ophthalmic nerve (Figure 12) is a sensitive nerve influencing the sensitivity of the upper eyelid, the teguments of the frontal and dorsal nose region, the eyeball, the tear ducts and the nasal fossa. The ophthalmic nerve originates from the medial portion of the semilunar ganglion.

Then, coming out of Meckel's cavity, the ophthalmic nerve crosses the lateral wall of the cavernous sinus. Initially the ophthalmic nerve sets below the oculomotor and trochlear nerves. Then it ascends and gets a lateral position to the trochlear nerve. As the ophthalmic nerve exits the cavernous sinus, it descences into three terminal branches: nasal, frontal and lacrimal nerves.

Collateral branches of the ophthalmic nerve

The collateral branches of the ophthalmic nerve comprise the collateral meningeal and the anastomotic branches to the pericarotid sympathetic plexus, the trochlear and oculomotor nerves.

The terminal branches of the ophthalmic nerve

The ophthalmic nerve has three terminal branches:

- **The nasal or nasocilliary nerve** contains the folowing collateral branches: the ciliary ganglion branch, the long ciliary nerves branch and Luschka's ethmoid-sphenoidal nerve branch. The terminal branches include the internal nasal and the external nasal nerves.
- **The frontal nerve** is the largest terminal branch of the ophthalmic nerve. Its collateral branches are the upper periosteum threads and the supratrochlear nerve. The internal frontal and the external frontal nerves stand for its terminal branches.
- **The lacrimal nerve** is the smallest terminal branch. Its branches comprise: tear branches attributed to the lacrimal gland, conjunctive-eyelid branches for the lateral third of the upper eyelid and an anastomotic branch attached to the maxillary nerve, that entirely form the orbital lacrimal arch.

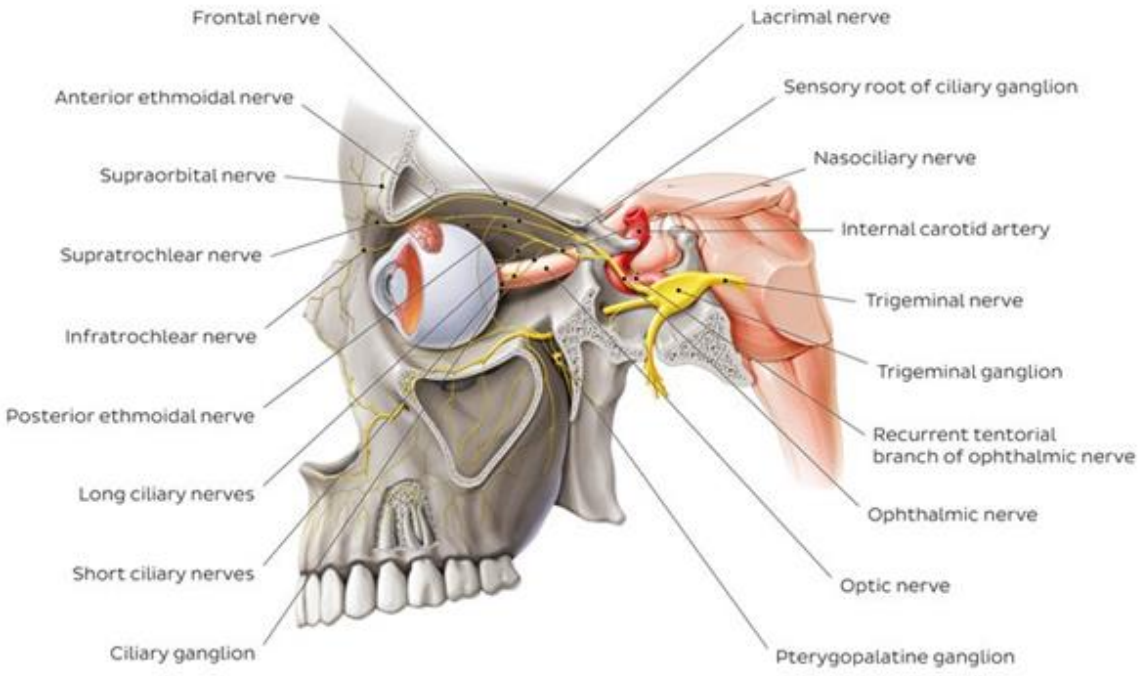


Figure 12. The branches of the ophthalmic nerve

II. The maxillary nerve

The maxillary nerve (figure 15) is a **sensitive nerve** having the following functional territory: the sensitivity to the dura mater in the temporal and parietal regions, the lower eyelid, the conjunctiva, the nose and cheek skin, the upper lip, the respiratory mucosa of the nostrils nasal fossa, the mucous membrane of the palate, palatal veil, pharyngeal orifice of Eustache's trunk, the maxillary bone, upper teeth and their corresponding gingival mucosa, as well as the innervation of the meningeal artery and the sphenoidal and maxillary sinuses mucosa.

The maxillary nerve arises from the middle portion of the semilunar ganglion, laterally placed from the ophthalmic nerve and medially positioned from the mandibular nerve. As the nerve exits Meckel's cave through the middle tunnel, it gets an anterior and lateral position. The maxillary nerve crosses the skull base through the round hole, penetrating the infratemporal fossa. Then, changing its direction, the nerve moves anteriorly, inferiorly and laterally towards the inferior orbital fissure. Within this level, it gets anteriorly and laterally redirected towards the suborbital trench. By passing it, the maxillary nerve enters the suborbital canal, crosses and then, exits it through the suborbital hole. As it emerges the suborbital hole, the maxillary nerve descends the numerous branches representing the suborbital bouquet.

The ratios of the maxillary nerve can be divided as follows (figure 13):

- **The origin portion** is located in Meckel's cave and comes in superior and medial relation with the origin of the ophthalmic nerve, while inferiorly and laterally it connects with the origin of the mandibular nerve.
- **The intracranial portion** is medially placed to the cavernous sinus and the ophthalmic nerve, laterally set to the oval hole and the mandibular nerve, superiorly located to the temporal lobe and inferiorly positioned to the anterior superior face of the temporal bone apex and the endocranial face of the great wing of the sphenoid bone.
- **The portion crossing the round hole** is attended by Nuhn's emissary veins. They make the connection between the cavernous sinus and the pterygoid plexus.
- **The infratemporal space** represents the "surgical section". It travels over the sphenopalatine or pterygopalatine ganglion, the internal maxillary artery and the pterygoid venous plexus.
- **The suborbital portion** moves through the suborbital trench and canal attended by the suborbital artery. It comes in anterior relation with a bone blade separating the nerve from the orbital cavity, while its inferior part connects with a thin bone placed between the nerve and the maxillary sinus. This explains the possibility to cause the trigeminal neuralgia during maxillary sinusitis.
- **Terminal portion** sets as the maxillary nerve leaves the suborbital canal through the suborbital hole and forms the suborbital bundle. It is covered by the upper lip and the nose wing lifting muscle.

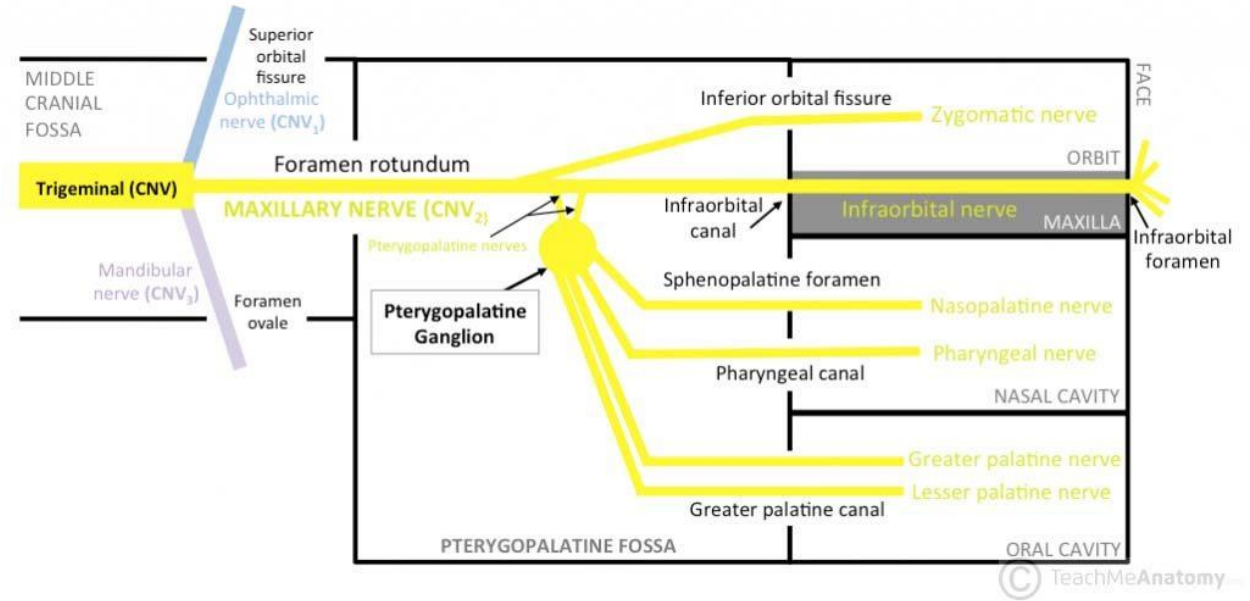


Figure 13. The maxillary nerve path

There are six collateral branches to the maxillary nerve:

1. Middle meningeal branch
2. Infraorbital branch

3. The sphenopalatine or pterygopalatine nerve
4. Dental or posterior superior alveolar nerves
5. Dental or middle superior alveolar nerve
6. Dental or anterior superior alveolar nerve.

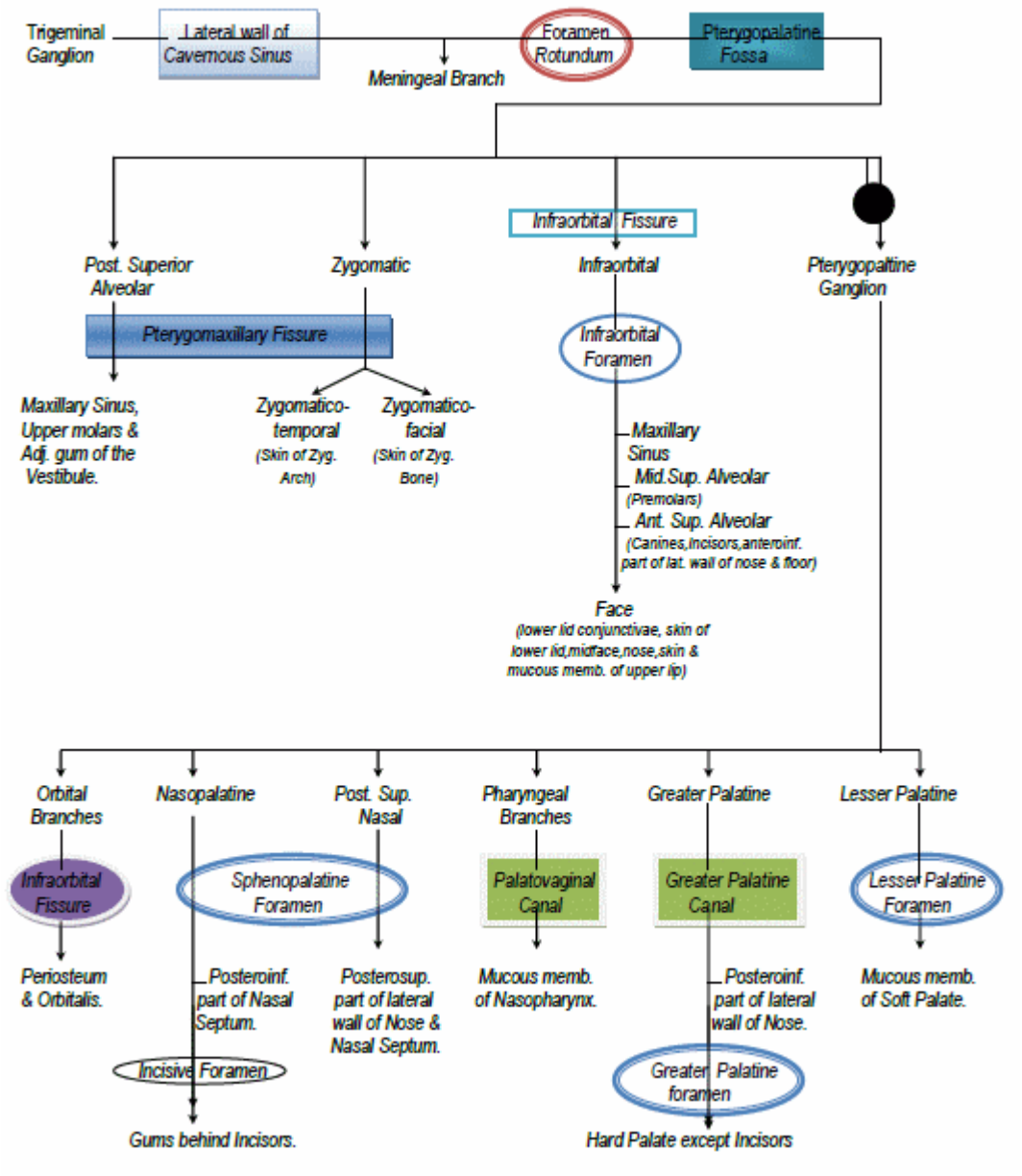


Figure 14. The branches of the maxillary nerve

Terminal branches

These are branches forming the suborbital bouquet.

- **superior or conjunctive-palpebrale branches** have an ascending path and are designated to the lower eyelid and the conjunctiva.
- **lower or genie-labial branches** have a descending path and attribute the lower eyelid and conjunction.

- **medial or nasal branches**: have a medial path and impute the nasal wing teguments.

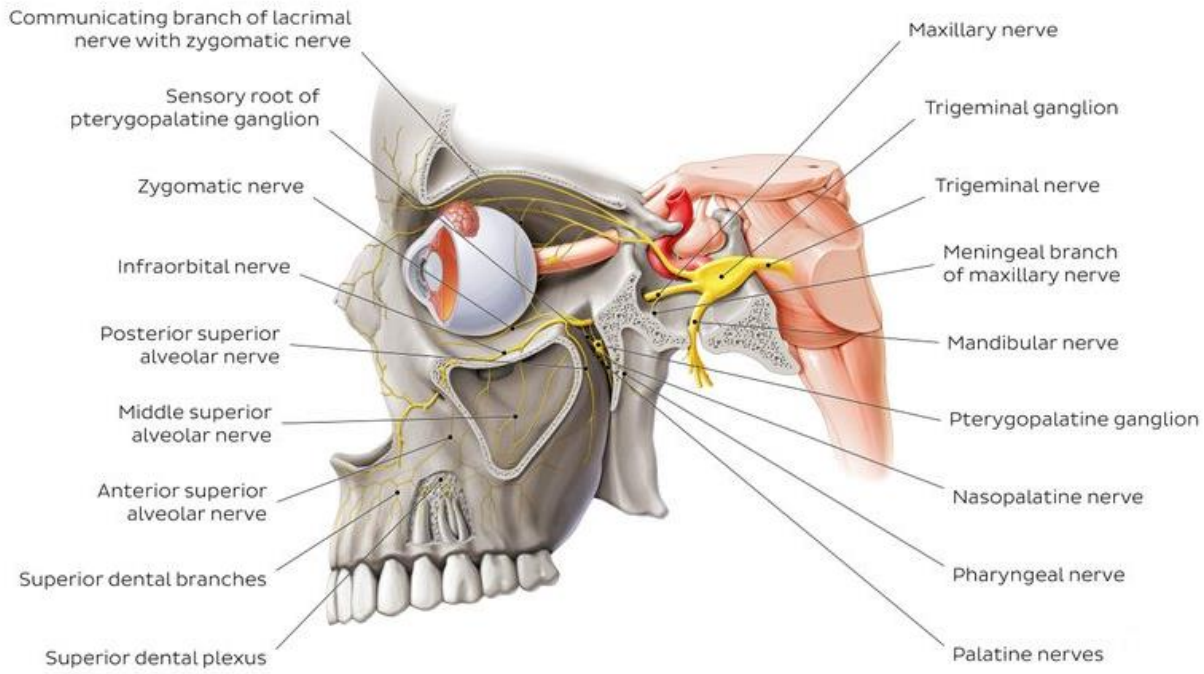


Figure 15. The maxillary nerve

III. The mandibular nerve

The mandibular nerve is a **mixed nerve** to provide the sensitivity of the hard matter corresponding to the middle meningeal artery portion, mucosal mastoid cells and the temporal-mandibular joint, the teguments in the lateral region of the head, the outer ear flag, the external auditory canal and the tympanic membrane. The mandibular nerve also supplies the innervation of the middle meningeal and superficial temporal vessels, the sensitivity to the skin and mucosa of the cheek and the lower lip, as well as to the skin in the mental region. It produces the sensitivity to the mucosa in the lingual "V" portion, the mandible, the lower teeth, the vegetative innervation to the parotid gland, the gustatory innervation on the top and edges of the tongue through the Wrisberg intermediate nerve.

The mandibular nerve originates from the joint of two roots: one sensory and another motor. The sensory root emerges from the lateral part of the semilunar ganglion, and the motor root is placed under the sensitive root. Arising from the origin, the mandibular nerve moves laterally and anteriorly crossing the skull base, vertically descends and ends in two nerve trunks, the two terminal branches come off from. The mandibular pathway is for about 2 cm long and it forms a right angle to the oval hole with a posteriorly oriented opening.

The mandibular nerve connections can be divided as follows (figure 16):

- **Intracranial portion** is superior to the temporal lobe, inferior to the large wing of the sphenoid nerves, large and small superficial petrous bone, medially set to the maxillary nerve and laterally located from the middle meningeal artery.
- **The portion crossing the oval hole** is attended by the small meningeal artery and Trolard small emissary vein.
- **The extracranial portion** is situated in the upper part of the mandibular-pharyngeal space and is surrounded by the tributary veins of the pterygoid plexuses. It gets a medial and posterior position from inter pterygoid aponeurosis, medial pterygoid muscle, tensor muscle of the palatine veil and pharynx. Laterally and anteriorly it is related with the lateral pterygoid muscle. Laterally the extracranial portion of the mandibular nerve, it attaches the optic ganglion.

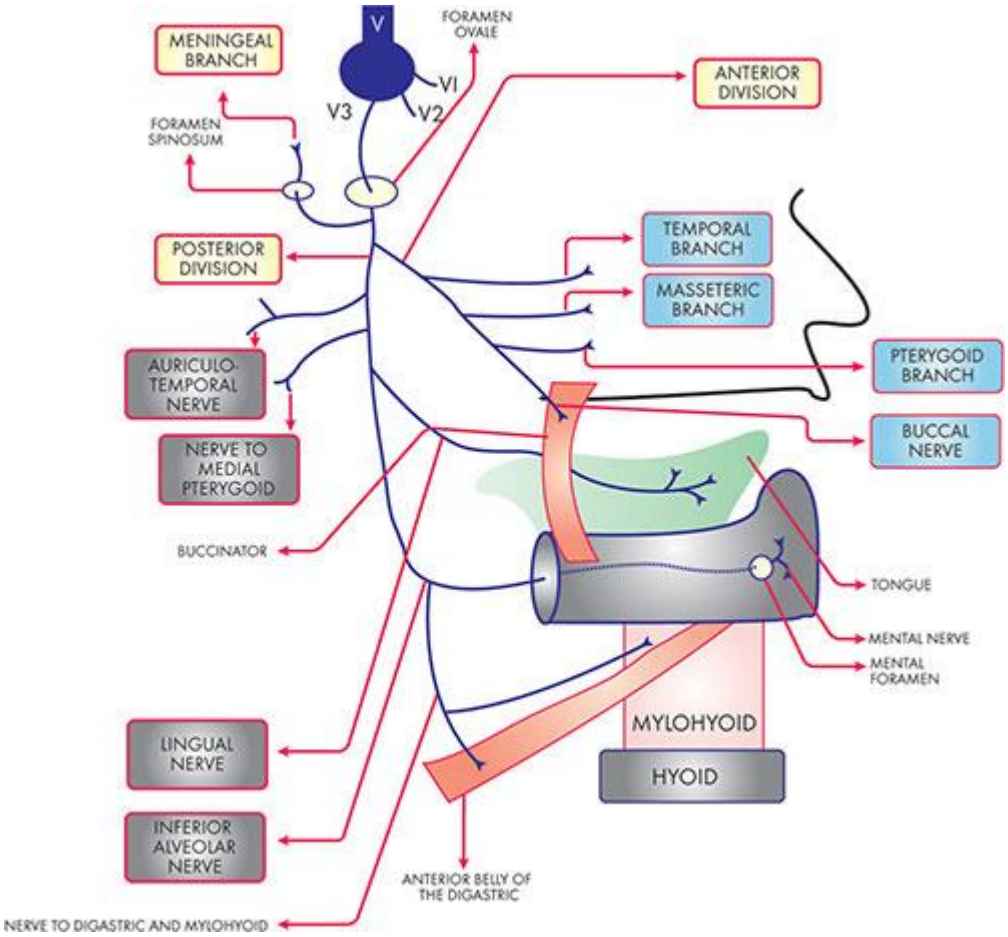


Figure 16. The pathway of the mandibular nerve

Collateral branches of the mandibular nerve

The mandibular nerve has **a single collateral branch** called the **recurrent meningeal branch.** It arises from the extra-cranial portion and moves through the foramen spinosum just to enter the cranial cavity, attended by the middle meningeal artery. The recurrent meningeal branch gives off two threads: one anterior to the middle meningeal artery and one posterior to the mastoid cells.

The terminal branches of the mandibular nerve

There are **seven** terminal branches of the mandibular nerve grouped into **two nervous trunks:** the **anterior and posterior ones.**

The anterior trunk is divided into **three** branches:

- Middle deep temporal nerve;
- Temporal-masseteric (masseteric) nerve;
- The temporal-buccal (buccal) nerve.

The posterior trunk gives rise to **four** branches:

- The common trunk of the nerves for the medial pterygoid muscle, tensor muscle of the palatine veil and the hammer muscle (tensor muscle of the eardrum);
- Auriculotemporal nerve;
- Dental (inferior alveolar) nerve;
- Lingual nerve.

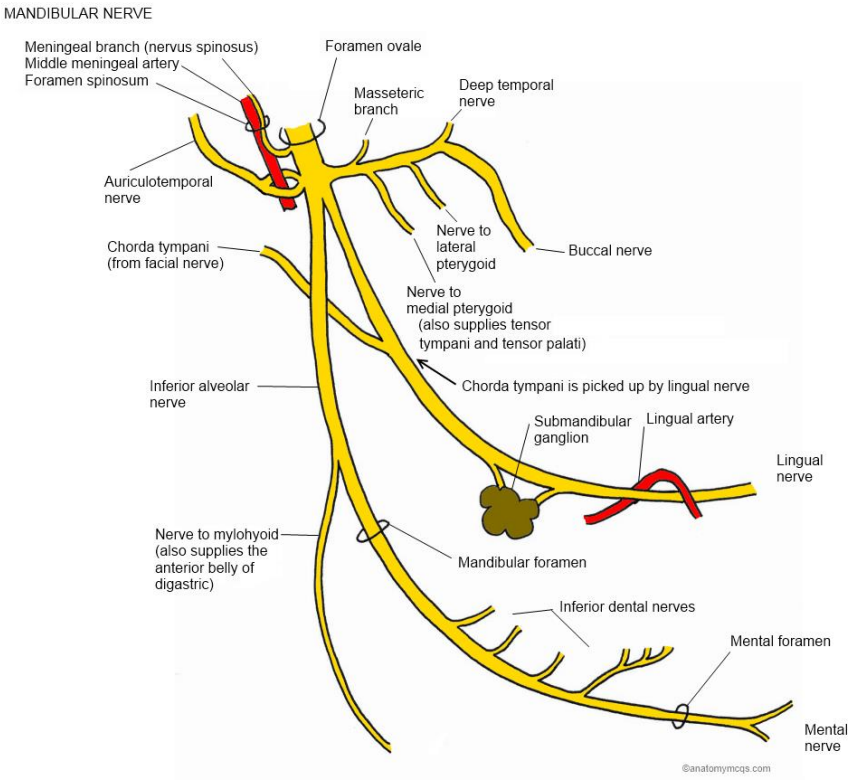


Figure 17. The mandibular nerve

THE LOCAL AND REGIONAL ANESTHESIA

The pharmacology of anesthetic drugs

All common local anesthetics have a 3-part structure (Figure 18): the aromatic ring, the intermediate chain and the amino (functional) group. The intermediate chain, comprising either an ester or an amide (peptide) bond, determines the classification of local anesthetics into esters and amides.

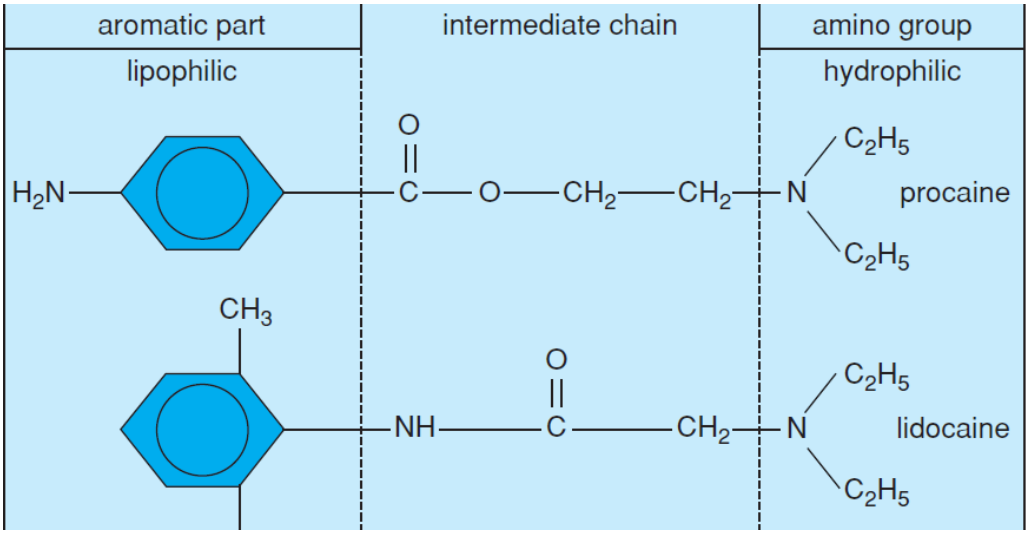


Figure 18. The molecular structure of an ester and an amide

The ester group (-COO -) - the ester bond is relatively unstable and these anesthetics are easily hydrolyzed, both, in solution and after the injection, into plasma by pseudo-cholinesterase. These anesthetics have a relatively short life and are difficult to sterilize as the heat is improperly to use. As they get rapidly dissolved into plasma, they are relatively non-toxic, but the duration of action, in this case, is also shorter.

Amide group (-NHCO-) - the amide bond is much more stable than the ester bond, and solutions support heat sterilization and pH shifts (that may be required in adding a vasoconstrictor). Also, they still hold an active potency whenever getting into the plasma and undergo biotransformation into the liver, so that just a very little or even no quantity at all is eliminated from the anesthetic. Local anesthetics do not influence the resting potential of the membrane, but determine the electrochemical shifts. Inside the nerve membrane preventing depolarization, they just block the nerve influx propagation. This phenomenon is called "membrane stabilization". It is produced by blocking the sodium channel opening and maintaining cell polarization. Most local anesthetics are relatively water insoluble and prepared as hydrochloric acid salt.

While injecting the substance (salt) dissociates into positive anions of local anesthetic and negative ions of chlorine: AHCl = AH + and Cl-, where A is the local anesthetic. The ionic form

must re-dissociate to the body pH: AH + = A (base) + H +. After the hydrochloric acid salt injection, both the positively charged (ionized) anionic form, that is hydrophilic and the basic form, electrically charged (non-ionized), that is lipophilic, appear rapidly. The ratio between the two forms, the electrically charged and the neutral one (AH+/A) depends on the anesthetic pKa. Anesthetics 10- have a pKa around 7.4 and the higher pKa is, the smaller the amount of basic (non-ionized) form. Only the liposoluble neutral form of the local anesthetic can penetrate the epineurium and nerve membrane. The membrane consists of a double lipid layer and protein molecules containing sodium channels. The intracellular medium (axoplasma) is aqueous and, after crossing the nerve membrane, the neutral, non-electrically charged base form must again dissociate and form a combination between the electrically charged and neutral forms. The electrically charged forms (anions) of local anesthetics get access to the sodium channels and block them, making Na + ions unable to cross the axolemma (nerve membrane). Due to this mechanism, the nerve impulse can no longer propagate. As the blockage develops, depolarization is initially slowed down and finally prevented. This process is common to most local anesthetics. However, some anesthetics, such as benzocaine, do not ionize at body pH and therefore exist only in the basic form. They can penetrate the nerve membrane, but do not reach the axoplasm. They seem to act through the expanding the nerve membrane that mechanically closes the Na + channels, an action mechanism similar to that of general anesthetics on the brain. The chemical structure largely determines the local anesthetics properties.

Pharmacodynamics and pharmacokinetics

Local anesthetics differ considerably depending on their action beginning, duration and power of the anesthetic effect.

Their **power (potency)** depends on their liposolubility. Bupivacaine and etidocaine are extremely fat-soluble and therefore have high anesthetic power, followed by those with a medium power, such as lidocaine, mepivacaine, prilocaine, chlorprocaine and finally those with low power - procaine.

The intensity of the effect depends on the concentration of the anesthetic.

The installation duration of the anesthesia depends on the anesthetic liposolubility. The basic form (salt) of the anesthetic is dominant in the nerve pH. The salt form goes inside the conjunctive barrier to reach the nerve, this property depending on its liposolubility.

The action duration of the anesthetic depends on the ability of the substance to bind to proteins. The free, non-protein bound fraction determines the intensity and duration of the anesthetic action. This fraction increases as the concentration of the anesthetic gets higher, but so does the risk of general accidents. The action duration also depends on the cationic forms

concentration around the axon, while their concentration depends on the anesthetic diffusibility and elimination rate. The elimination is the consequence of the anesthetic passive diffusion along the concentration gradient from the nerve to the extraneural space and the absorption inside the blood vessels in and around the nerve. The vasodilator effect is almost universal for local anesthetics. Clinically, as the anesthesia is installed, local anesthetics progressively cancel the transmission of pain, thermal variation, proprioception and finally the muscle tone.

Probably, pharmacokinetic aspects are of a particular importance for the analgesic effect power. A general overview of the factors that may affect the intrinsic activity of local anesthetics is presented in Table 2:

Table 2. Factors that may influence the intrisic activity of the local anesthetics

Factor	*Mechanism*
Pregnancy	Progesterone may increase the potency of local anesthetic over the nerve blocking effect
PH alteration	PH alteration inflammatory processes and uremia decrease the tissues pH. This reduces the pH percentage of the basic neutral form. PH alteration can also influence the connection between plasma and tissue proteins that is relevant for the rapid tolerance onset in case of repeated injection.
Vasodilation	Repeated vasodilation causes rapid removal from the injection site (bupivacaine is a vasodilator).
Vasoconstriction	A vasoconstrictor masks the inherent vasodilator properties of local anesthetics and causes an increased effect to enhance the anesthetics action duration.

To onset the action after injecting an aqueous solution of local anesthetic, four steps are required:

1) Diffusion inside the tissues around the injection site, that occurs during a variable period of time, depending on the chemical structure and vascular action of the substance.

2) Salt hydrolysis within the weakly alkaline intracellular environment, succeeding in the liposoluble base onset, able to penetrate the nerve fiber. This phase does not occur in the acid environment that exist, for example, inside the inflammatory outbreaks and therefore local anesthetics are weakly active inside such outbreaks.

3) The anesthetic penetration into the nerve with it’s the basic, liposoluble form;

4) Stabilization of the nerve fiber membrane and preventing depolarization with the loss of the impulse conduction capacity.

Local anesthetics are substances that applied in certain concentrations to peripheral nerve endings or nerves reversibly block the influx formation or transmission, causing the decrease or total lack of sensitivity and / or muscular paralysis. Local ester anesthetics get metabolized inside the plasma by plasma pseudo-cholinesterase, while the amide ones are bio-transformed to the liver. This particularity causes the first group of substances to get metabolized faster, as they have a shorter action duration, and the second group undergoing a slower biotransformation, to have a longer action duration (table 1). In therapeutic concentrations of local anesthetic the different types of nerve fibers are blocked successively, according to the concentration of the anesthetic solution, the diameter of the fibers and the degree of myelination. The damage includes the following order: the vegetative fibers, the painful, thermal, tactile, pressure and motor fibers. Damage to the motor fibers with the common striated muscles relaxation or paralysis that occurs in higher concentrations of local anesthetic is sometimes useful in surgery, sometimes undesirable or even dangerous (e.g. it may cause respiratory depression).

Local and regional anesthesia

Local and regional anesthesia (LRA) is the method by using chemical and physical substances or electrical current, to set a temporary desensitization within the worked on anatomical region, the patient's consciousness remaining intact.

Local and regional anesthetics are substances with localized, temporary and reversible action on nerve cells and fibers. Clinically, it progressively cancels the transmission of pain, thermal variation, proprioception and finally muscle tone.

The safe and effective local anesthetic must meet the following qualities:

1. Strong anesthetic effect
2. Short induction
3. Reduced systemic toxicity
4. Not to cause local irritation
5. Low incidence of adverse effects
6. Good efficacy / toxicity ratio

The anesthetic solution is injected within the immediate vicinity of the nerve trunk, but part of it is bound to nonspecific tissues (fat, muscle fibers), and part is absorbed into the blood, so only a small amount gets to the nerve fiber.

Inside the blood runs:

⇒ a circulating protein bond form- glycoprotein

⇒ a free (active) form - it has anesthetic and toxic effect

- Glycoprotein also transports other medicinal substances: beta-blockers (propranolol), calcium blockers (verapamil) antiarrhythmic (quinidine)
- To cardiovascular patients, the free fraction of local anesthetic will increase and may cause general overdose accidents, interpreted as intolerance accidents, allergy, etc.

NB The chemical structure determines the properties of local anesthetics:

- Power - depends on liposolubility
- Intensity - depends on concentration
- Onset time - depends on liposolubility
- Duration of action - depends on the ability to bind to proteins

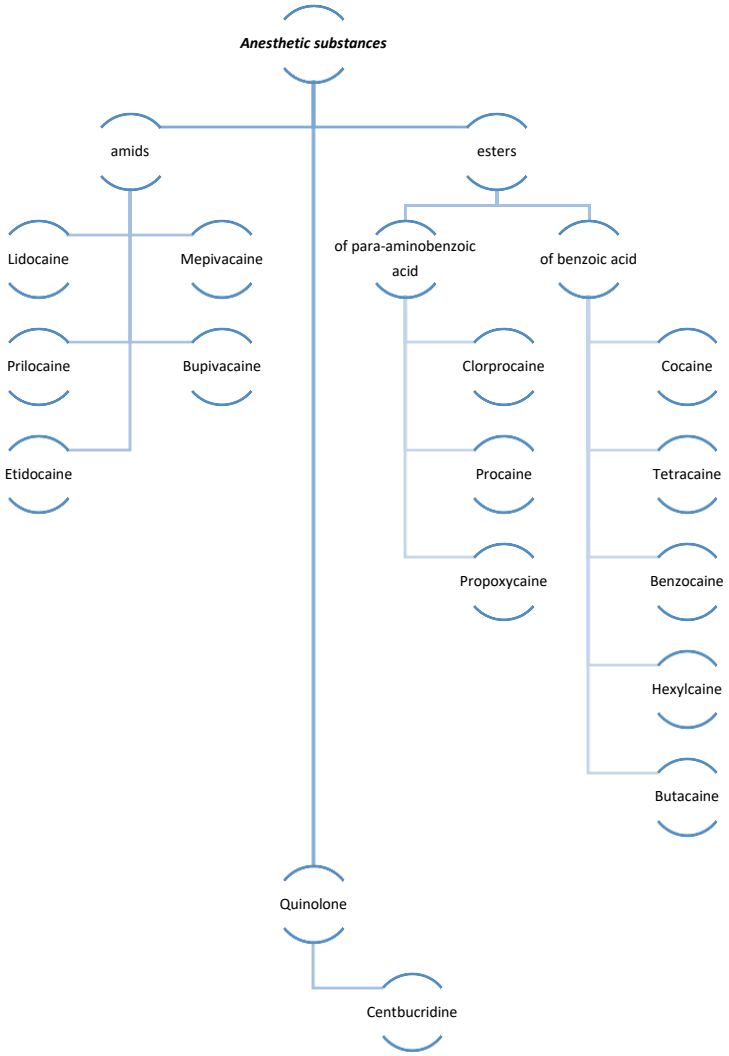
NB The qualities of anesthetic solutions:

- Not to influence the cells, irritate the tissues and cause structural shifts.
- Not to cause undesirable general effects, be toxic in admissible concentrations for geting the anesthetic effect or cause habitual phenomena, sensitization or allergy reactions.
- Be water soluble and stable in solutions.
- Not to alter during the heat sterilization process.
- Diffuse easily into tissues, but not to re-settle too fast in order to ensure a sufficient long-lasting action.
- The anesthetic action should be installed quickly and be sufficiently intense.

NB High quality anesthetic solutions should include:

- Anesthetic agent
- Vasoconstrictive agent
- Adjuvant agent Diluent solution

Local and regional anesthetic agents



Common features:

- Synthetic and have the same mechanism of action
- Contain "amino" groups
- Form salts in react with strong, water-soluble acids
- Have reversible action
- Are compatible with adrenaline or related drugs
- To a certain plasma concentration they produce typical general effects
- In anesthetic concentrations they have no irritating effect on the tissues

NB

THE NAME PREFIXES OF THE ANESTHETIC AMIDS INCLUDE "I" LETTER + "CAINE". (e.g. L*I*docaine, Bup*I*vacaine)

Side and adverse effects:

- Allergies - quite common.
- Action over the central nervous system presented by: shivers, anxiety, convulsions, death.
- Cardiac effects including extra-systoles, decreased contraction force of the heart, ventricular fibrillation

LIDOCAIN HYDROCHLORATE (Xylene)

Lidocaine was synthesized in 1943 by Nils Löfgren and went on sale in 1948 to substitute procaine (Novocaine). Compared with it, lidocaine has a much faster action onset of 3-5 minutes, and results in a longer, deeper anesthesia (90 minutes without adrenaline). Thus, it has an anesthetic power 3-4 times greater than procaine, a fast induction time of 0.8-2 minutes and a pKa equal to 7.9. It runs bound to plasma proteins in amount of 65% (especially to acid alpha-1 glycoprotein).

Lidocaine has a toxicity twice as high as procaine and thus the amount for injection has been limited. Also, following an accidental intravascular injection, it can cause acute toxic effects if systemic layers get grown. Neurotoxic reactions have an excitatory or depressive character leading to nervousness, tinnitus, nervous tics, euphoria, drowsiness, vision disorders, dizziness, convulsions, unconsciousness and possibly respiratory arrest.

It does not give allergic reactions, this is a major clinical advantage of lidocaine and all amides compared to esters. Anyway, whether allergic reactions occur, they are caused by the preservative introduced into the solution, called methyl-paraben. The xylene solution with adrenaline contains sodium methyl bisulfate or methyl parahydroxybenzoate that can cause severe allergic reactions.

Cardiovascular reactions are depressing and include hypotension, myocardial depression, bradycardia and possibly cardiac arrest. Cardiovascular effects occur only in severe situations and are generally preceded by signs of neurotoxicity. To avoid this inconvenience, the dose will be adjusted according to the age, body weight and general condition of the patient. To administer the most appropriate local anesthesia for dental procedures, the maximum dose of lidocaine with vasoconstrictor recommended by the U.S. The Food and Drug Administration (FDA) is 7mg / kgc for adults (approximately 3 carps) without exceeding 500 mg; the maximum dose of lidocaine without vasoconstrictor is 4.5 mg / kgc, without exceeding the absolute maximum dose of 300 mg. To children, MRD is 4.5 mg / kgc. For routine dental procedures, the recommended doses is 1-5 ml, without exceeding 10 ml lidocaine dose with adrenaline (200 mg lidocaine).

Lidocaine does not produce vasodilation, so it is not mandatory to add a vasoconstrictor. By combining with the vasoconstrictor, increases the active duration and the anesthetic potency.

Lidocaine is metabolized in the liver to 90% in two active metabolites, monoethylglycinexylidin (MEGX) and glycinexylidin (GX). Metabolism products and 10% of the non-metabolized substance are excreted renal.

Lidocaine should be used with caution by patients receiving antiarrhythmic medication (Nifedipin, Adalat, and Norvasc etc.), monoamine oxidase inhibitors (Nardil, Aurorix) and tricyclic antidepressants (Amitriptyline), as the toxic effects can cause prolonged hypertension.

It is prescribed in lower doses to patients undergoing treatment with beta-adrenergic blockers (Propranolol, Betaloc, and Atenolol) or calcium channel blockers (Verapamil, Isoptin).

It is contraindicated for **patients:**	**It is administered** with caution and in small doses **to patients:**
✓ **allergic to lidocaine or other amide anesthetics;**	✓ with severe hepatic impairment,
✓ **with malignant hyperthermia background;**	✓ acute myocardial infarction;
✓ **uncompensated heart failure;**	✓ respiratory failure;
✓ **with atrioventricular conduction disorders**	✓ with a convulsive history..

Lidocaine has a relatively low teratogenic effect (toxicity class B), but it is recommended to delay the administration for women during the first semester of pregnancy, the organogenesis process.

As 55% cross the fetal-placental barrier, cardiac monitoring of the fetus is recommended. It has not given clinical evidence regarding its remove through breast milk, but breast feeding is recommended to be delayed.

In dentistry, it is used for infiltration anesthesia and peripheral trunk anesthesia in concentrations of 0.5%, 1%, 2% with or without vasoconstrictor, as well as for contact anesthesia in concentration of 10%.

The most commonly used commercial products in dentistry are: Xylene 1%, Xylene 2%, Xylene 2% with adrenaline, Xylene 4% in 2 ml vials by Sicomed; Lignospan with adrenaline 1:

100000, special Lignospan with adrenaline 1: 80000, strong Lignospan with adrenaline 1: 50,000 in 1.8 ml cartridges produced by Septodont; Xylestesin-A with adrenaline 1: 80000 produced by 3M ESPE in 1.7ml carps.

MEPIVACAIN CHLORHYDRATE

It is a local amide anesthetic, prepared by A.F. Ekenstam in 1957 and introduced in dentistry in 1960 as a 2% solution combined with a synthetic vasopressor, the levonordefrin. In 1961 a 3% solution without vasoconstrictor was synthesized.

It has a potency of 2, a pKa of 7.6 and a toxicity of 1.5-2 to procaine. The anesthesia has a short outset action time (2-3min). It has a mild vasodilator and intrinsic vasoconstrictor effect, a longer action duration (2-3 hours) and does not require the addition of vasoconstrictive agents (adrenaline or noradrenaline).

Side effects are rare, the signs and symptoms of mepivacaine overdose include CNS stimulation and depression. Sometimes, its overdose can induce the lack of stimulation leading to the immediate depression of the CNS clinically manifested by drowsiness and loss of consciousness.

It binds 78% to the plasma proteins, undergoes a rapid biotransformation to the liver and only 5-10% is excreted renal without being metabolized. The rate of systemic absorption depends on the anesthetic amount, concentration and the presence or lack of the vasoconstrictor.

As it crosses 70% of the fetal-placental barrier, it falls into the toxicity class C and is not available to use during the first semester pregnancy.

The maximum recommended dose for children over 6 years and adults is 6.6 mg / kg body weight without exceeding 500 mg. To a 20 kg child can be given 2 cartridges, but to an adult 7.5 anesthetic cartridges without any vasoconstrictor. The maximum number of anesthetic cartridges with vasoconstrictor is 3 for a 20 kilo child and 11 for a normoponderal adult.

✓ **It** is contraindicated **for patients with:**
✓ **allergy or hypersensitivity to local amide anesthetics;**
✓ **II and III degree bundle branch block, severe bradycardia;**
✓ **acute decompensated heart failure;**
✓ **severe hypotension.**

In dentistry, 0.5%, 1%, 2% or 3% mepivacaine solution is used. Mepivacaine is compatible with levonordefrin concentration of 1: 20000 and extends the anesthesia active duration to approximately 60 minutes for dental pulp and 3-5 hours for soft tissue. When hemostasis is desired, adrenaline is added to the wound instead of levonordefrin.

The commercial products frequently used in dentistry are: Mepivastezin cartridge 1,7ml produced by 3M ESPE; Scandonest 3% plain cartridges 1.8ml, Scandonest 2% noradrenaline cartridges 1.8ml, special Scandonest 2% with adrenaline 1: 100000 produced by Septodont; Carbocaine 3% cartridge 1,7ml, Carbocaine, 2% Neo-Cobefrin Carbocaine (levonordefrin 1: 20000) cartridges 1,7ml produced by Zeneca Astra.

ARTICAINE CHLORHYDRATE

Articaine is a local hybrid amide anesthetic containing an amide and an ester group. It was prepared by H. Rusching in 1969 and put on for sale in Germany and Switzerland in 1976, in Canada in 1983, and in the USA in 2000. Since its introduction into medical practice, articaine has become the most used anesthetic agent, so it got the second place in 2011. The articaine is considered to diffuse much better in hard and soft tissues than other local anesthetics.

It has a vasodilator effect similar to lidocaine and is sold only in combination with a 1: 200000 single form or 1: 100000 strong form adrenaline vasoconstrictor.

The anesthetic agent has an anesthetic potency of 1.5 against lidocaine and 1.9 against procaine, but the toxicity is similar to lidocaine and procaine. Anesthetic onset time is short taking 1-2 minutes, pKa equal to 7.8 and the action duration lasts 2-3 hours.

Biotransformation of the articaine is carried out in plasma by 90% plasma cholinesterase and 8% in liver by liver microsomal enzymes. Approximately 90% metabolized and 5-10% non-metabolized articaine is renally removed from the body.

As 95% is protein-bound and exclusively excreted renal, the election anesthetic is prescribed to pregnant women.

The maximum recommended dose is 7 mg/kg+- to a normoponderal adult without exceeding 500 mg (equivalent to 12.5 ml injection)/ session. To children, the minimum volume needed to get anesthesia should be used.

The amount of injection should be individually adjusted to the child age and weight and the maximum dose of 7 mg of articaine per body weight kg (0.175 ml / kgc) should not be exceed. The use of articaine for children under 4 years is contraindicated.

It is contraindicated **to the patients with:**
✓ **allergy to local amide anesthetics or sodium metabisulfite;**
✓ **severe disorders of the heart rhythm (atrioventricular block of I or II degree);**
✓ **severe disorders of the heart rhythm (atrioventricular block of I or II degree);**
✓ **acute heart failure;**
✓ **myocardial infarction;**
✓ **acute recurrent porphyria;**
✓ **bronchial asthma;**
✓ **treatment of beta-blockers such as propranolol;**
✓ **pheochromocytoma**
✓ **hypertension;**
✓ **following treatment with antidepressant or Parkinson's disease drugs (tricyclic antidepressants, MAO inhibitors), as these drugs may enhance the cardiovascular effects of adrenaline;**
✓ **cholinesterase deficiency;**
✓ **epilepsy.**

In the case of excessive administration, it may induce toxic reactions, due to the increased plasma concentration of articaine, manifested by nervousness, headache and nausea, tachypnea followed by bradypnea and finally apnea and hypotension.

Also, late adverse effects have been detected as: local necrosis, sensitivity disorders along the anesthetized nerve pathway.

The commercial products commonly used in dentistry are: Ubistezin with adrenaline 1: 200000, Ubistezin strong with adrenaline 1: 100,000, cartridges 1.7ml, produced by 3M ESPE; Septocaine with adrenaline 1: 100000, cartridges 1.7ml, Septanest 4% cartridges 1.7ml with adrenaline 1: 200000 or 1: 100,000 produced by Septodont; Ultracain D-S 1.7ml cartridges or 2ml vials with adrenaline 1: 200000, Ultracain forte 1.7ml cartridges with adrenaline 1: 100,000 produced by Sanofi Aventis.

BUPIVACAINE HYDROHYDRATE

Bupivacaine is an amino-amide local anesthetic prepared in 1957 by A. F. Ekenstam and introduced into medical practice in October 1972. It is used for epidural anesthesia, also to induct the local or regional anesthesia in the oro-maxillo-facial area. It has an anesthetic power 4 times higher than xylene, mepivacaine and prilocaine.

The anesthesia is onset within 6-10 minutes due to an increased pka of 8.1 and duration action takes about 90 minutes. Bupivacaine is metabolized to the liver by amidases and is renal excreted.

It is sold in 0.05%, 0.125%, 0.25%, 0.5% concentrations, while in dentistry, it is used in a concentration of 0.5% with 1: 200000 adrenaline. In addition to the local anesthetic effect, bupivacaine also has a postoperative analgesic effect.

The maximum recommended dose is 90 mg injected in divided doses. Bupivacaine doses will be reduced for young, elderly, patients with neurological disorders, heart or liver disease.

High doses or accidental intravascular injections may result in increased plasma levels, correlated with myocardial depression, decreased cardiac output, hypotension, bradycardia, ventricular arrhythmias and cardiac arrest. Bupivacaine is not recommended for children younger than 12 years. It induces increased cardiotoxicity producing hypoxia, acidosis and hypercapnia.

Bupivacaine should be used with caution by patients receiving antiarrhythmic medication because the toxic effects are additive.

Commercial products for dental use are: Marcaine, Sensorcaine or Vivacaine (Astra Zeneca).

PRILOCAINE HYDROCHLORIDE

It is a local amino-amide anesthetic, prepared by Löfgren and Tegnér in 1953 and introduced in practice in 1965.

Prilocaine has an anesthetic potency twice as high as procaine and equal to xylene. It is 40% less toxic than xylene and almost equal to prilocaine. The anesthetic effect is set slowly, during 3-5 minutes, because it has a pKa = 7.9 and lasts about 75-90 minutes.

Prilocaine is rapidly hydrolyzed by liver amylases into ortho-toluidine and N-propylalanine and is considered a low toxicity anesthetic. Ortho-toluidine may induce methemoglobin formation, a major disadvantage in case of overdose. Prilocaine and its metabolites are primarily excreted by the kidneys.

The maximum recommended dose for a healthy adult is 8 mg / kg body weight without exceeding 600 mg. It has a greater vasodilator effect than mepivacaine but smaller than lidocaine. In combination with a low dilution 1: 200,000 adrenaline, it provides long-term anesthesia.

It is relatively contraindicated **to the patients with:**
✓ **congenital or idiopathic methemoglobinemia;**
✓ **hemoglobinopathies;**
✓ **anemia**
✓ **cardio-respiratory diseases with severe hypoxia.**
✓ **patients receiving acetaminophen or phenacetin as they both increase the methemoglobin risk**

The commercial product is Citanest plain 4% without vasoconstrictor or Citanest forte 4% with adrenaline 1: 200000, produced by Astra Zeneca.

ADJUVANCING VASOCONSTRICTOR AGENTS

Vasoconstrictors are agents causing the blood vessel vasoconstriction and opposed to the vasodilator action of local anesthetic substances. Thus, by reducing the systemic absorption local anesthetics have the following effects:

- decrease the toxicity risk;
- reduce the overdose risk;
- minimize or neutralize the allergic effects;
- increase the onset speed of local anesthesia;
- increase the duration and intensity of local anesthesia;
- decrease intraoperative hemorrhage;
- stimulates the metabolism of local anesthetics.

The disadvantages of their action are determined by their misuse:

- in higher concentrations than needed;
- increasing the vasoconstrictor concentration, through repeated injections, up to almost toxic doses;
- accidental intravascular injection.

The use of vasoconstrictors in local anesthetic substances began with the adrenaline discovery in 1897 by Abel. In 1903, Brown proposed the use of adrenaline to extend the action duration of local anesthetics.

The most commonly used vasoconstrictive agents are chemically identical with the sympathetic nervous system mediators - adrenaline (epinephrine) and noradrenaline

(norepinephrine), as well as their actions are similar to the response adrenergic stimulation response.

They are classified as natural and synthetic sympathomimetics, acting on alpha and beta adrenergic receptors. Activation of alpha receptors causes smooth muscles constriction at the blood vessels level.

Activation of beta receptors results in smooth muscle relaxation (vasodilation and bronchodilation), cardiac stimulation, increased pulse and myocardial contraction force and lipolysis. Natural catecholamines are secreted by the adrenal medulla under stress or effort conditions.

ADRENALINE (Epinephrine)

Adrenaline or epinephrine is a hormone secreted in the blood by the medullary adrenal gland under stress conditions. It was discovered by Takamine and Aldrich in 1901 and was first synthesized in the laboratory by Stolz in 1904.

It comes as a white crystalline and very soluble in water powder. As adrenaline is rapidly altered by heat, light or air contact, the ampoules must be light-free and checked before use (changing color to pink or brown-red translates to altering the solution). To delay oxidation, a sodium bisulphite preservative is added to the adrenaline solution. The validity of a local anesthetic cartridge containing a vasoconstrictor is shorter (18 months) than a non-vasoconstrictive one (36 months). It is biotransformed in the liver by the catechol-O-methyltransferases (COMT) and monoamineoxidase enzymes. Only small amounts of adrenaline (about 1%) are non-metabolized excreted by urine.

Adrenaline acts on:

 Alpha 1 receptors in the skin, mucosa and the hepatic-renal territory producing vasoconstriction

 Cardiac β1 receptors having the following effects:

- intensifies the contraction force of the myocardium;
- rises the heart rate;
- increases the myocardium excitability causing arrhythmias;
- stimulates cardiac output;
- grows systolic blood pressure;
- coronary blood flow increase on the cardiac cord;
- cardiac pathology: the mechanical work of the heart increases with the myocardium oxygen consumption growth, clinically manifested by angina pains, arrhythmias and ischemiac-basal changes.

- ***β2 receptors*** cause vasodilation inside skeletal muscles and bronchodilation

They inhibit insulin release, stimulates hepatic glycogenolysis, leading to increased glicemia level. Adrenaline inhibits the chemical mediators (histamine) remove by β-antagonistic effect on mast cells.

As a result of these effects, adrenaline has the following clinical indications:

- bronchospasm treatment;
- allergic reaction treatment acting as symptomatic (see mechanism explained to allergic reactions);
- cardiac and respiratory resuscitation;
- low heart flow syndrome;
- to produce mydriasis;
- vasoconstrictor in anesthetic substances (reduces the systemic absorption rate of
- local anesthetics) to increase the active duration of anesthesia;
- reduces surgical bleeding.

Adrenaline is contraindicated to:

- hypertension;
- diabetic patients;
- hyperthyroidism;
- patients undergoing treatment with digitalis and tricyclic antidepressants (MAO inhibitors);
- pregnant women in the first trimester (teratogenic effect class C) and in the last trimester (risk of labor onset).

In dentistry, adrenaline is used in the following concentrations:

- for local regional anesthetics in concentrations of 1: 80000; 1: 100,000; 1: 200,000;
- for topical anesthetics in higher concentrations 1: 40000; 1: 50000.

The maximum dose of adrenaline in the anesthetic solution is 200-250µg.

NORADRENALINE (Norepinephrine, Levophed, Levarterenol)

Noradrenaline or norepinephrine, naturally, constitutes about 20% of the catechol amines produced by the adrenal gland.

To patients with pheochromocytoma, the adrenal gland tumor, norepinephrine may represent 80% of gland secretion. It is relatively stable in acidic solutions and alters under light

and air exposure. To increase noradrenaline stability, a preservative, such as sodium bisulphite acetate, should be added.

Noradrenaline is available in both natural and synthetic forms.

It influences exclusively α receptors (90%), producing arterial and venous vasoconstriction. Noradrenaline has a 4-time lower toxicity compared to adrenaline and a lower vasoconstrictive effect than that of adrenaline.

It has the following effects:

- acts on cardiac α1 and β1 receptors, causing a strong ionotropic effect (increases contractility);
- the heart rate decreases in contrast to the baroreceptor reflex activity increase;
- coronary blood flow grows;
- cardiac output falls due to systemic vasoconstriction;
- renal and hepatic blood flow reduces;
- produces metabolic acidosis.

It has no action on bronchial and peripheral vessel β2 receptors

The maximum admissible dose to a healthy patient is 0.34 mg / session; 10 ml of solution 1: 30000. In the case of cardiovascular disease only (ASA 3 or 4) 0.14 mg / session or approximately 4 ml of the 1: 30000 solution is allowed. Overdose causes mild hypertension and bradycardia.

It is recommended indications to hypertensive, hyper-thyroiditic, diabetic patients, as well as, to neuro-vegetative dystonia.

The concentrations used in dentistry are 1/30 000 - 1/20 000. The maximum admissible dose is 0.34 mg.

LEVONORDEFRIN (Neo-Cobefrin)

Levonordefrin is a non-catechol amine synthetic vasoconstrictor directly stimulating alpha (75%) and beta (25%) receptors. It has systemic actions similar to adrenaline, but is less active on the myocardium, respiratory tract, CNS and metabolism.

For all patients, the maximum dose should be 1 mg / session equivalent to 20 ml of the 1: 20000 solution (11 cartridges).

It comes in combination with mepivacaine dilution of 1: 20000.

PHENYLEPHRINE (Neo-Sinephrine)

Phenylephrine is the most stable and weakest vasoconstrictor used in dentistry. The cardiovascular actions of phenylephrine comprise:

- increased systolic and diastolic pressure;
- reflex bradycardia;
- slight decrease in cardiac output (by pressure growth and bradycardia);
- strong vasoconstriction without marked venous congestion;
- rarely cardiac arrhythmias.

It does not produce bronchodilation and is not neurotoxic. Phenylephrine is used as a vasoconstrictor in local anesthetics, to hypotension treatment, as a nasal decongestant and in ophthalmological solutions to produce mydriasis.

Phenylephrine was used in combination with procaine 4% in a dilution of 1: 2500.

To healthy patients, the maximum recommended dose is 2-5 mg / session or 10 ml of the 1: 2500 solution. To patients with cardiovascular disease (ASA 3 or 4) the maximum recommended dose is 1.6 mg / session similar to 4 ml of the 1: 2500 solution.

FELYPRESSIN (Octapresin)

Felypressin is a synthetic vasopressin analogue with slow induction and reduced toxicity.

It is an election vasoconstrictor used to patients with cardiovascular disease and thyroxytosis. Felypressin has antidiuretic and oocyte actions and is contraindicated to pregnant patients. The vasoconstrictor is available in dilution of 0.03 IU / ml with 3% prilocaine. It is not recommended to hemostasis as its predominant effect is reflected on the venous system.

For patients with major cardiovascular disease (ASA 3 or 4) the maximum recommended dose is 0.27 IU compatible to 9 ml of the 0.03 IU / ml solution.

In conclusion, while choosing a vasoconstrictive agent or adjunctive to the local anesthetic substance, it should be adjusted to:

- the patient's territory
- duration and complexity degree of treatment
- hemostasis necessity during treatment
- the need for postoperative pain control

For all patients, and especially those with pathological territory, the benefits and risks of including a vasoconstrictor in the local anesthetic solution should be weighed. When used, intravascular injection by repeated aspiration should be avoided and anesthetic administration should be slow.

GENERAL PRACTICAL ASPECTS IN LOCAL AND REGIONAL ANESTHESIA

In dentistry, local anesthesia is most often used to facilitate dental treatment, so comfort is maximum for both the patient and the physician who can work calmly, accurately and with concentration. Local selective anesthesia is often used for diagnostic purposes to identify the pain cause in the face region. It is also applied to prevent short or long-term postoperative pain.

Indications and contraindications

Before administering the anesthetic, the dentist must, firstly, explain the reason its use is absolutely necessary. The indications and contraindications of the anesthetic are determined according to the patient's condition (table 3). Therefore, each patient should be drawn attention to determine if a certain type of anesthetic is possible or appropriate to use. This includes obtaining a full medical history by completing surveys related to the patient's previous experiences with local anesthetics. The medical history must be well structured, systematized and preferably in written form. Even negative conclusions such as "no preparation", "non-allergic" and "no bleeding tendency" need to be documented.

After the doctor explains to the patient the type of anesthesia will be used, he should check if the explanations have been correctly understood. It is also important for the patient to consent to the use of the anesthetic (informative agreement), because, legally, if the patient does not protest against the use of anesthesia, it does not mean he gives his consent implicitly.

Table 3. General guide of indications

GENERAL GUIDE AND INSTRUCTIONS TO USE DIFFERENT TECHNIQUES IN LOCAL ANESTHESIA ADMINISTRATION		
Procedure	mandible	maxilla
Scaling		
• **Local**	IA	IA
• **General**	MB	IA
Filling/ orthopedic preparation	MB + IA	IA
Endodontic	MB (IL)	IA
Dental extraction	MB + IA	IA
Periodontal surgery	MB + IA	TA, INA, PN
Third molar extraction	MB + IA	NPN
Dental implantation	MB + IA	TA +PN

Pre-implant surgery	MB + MNB	TA +PN
Central or peripheral pain	MB	TA +PN+ IA
Odontogenic or not?	MB	INA, TA
Which tooth?	IL	IL sau IA

Abbreviations:

TA - tuberosity anesthesia

IL - intraligamentary anesthesia

INA - infraorbital nerve anesthesia

IA - infiltrative anesthesia

MB - mandibular nerve block

MNB - mental nerve block

NPN - nasopalatine nerve anesthesia

PN- palatal nerve block (large)

The instrumentation used to administer anesthesia

In order to perform the application techniques of local and regional anesthesia, it is necessary an instrumentation consisting of:

1. Examination kit: dental mirror, dental forceps, dental probe.

2. Instrumentation to apply the anesthesia technique including: syringe, puncture needle, anesthetic solution

The syringes can be:

- Made of disposable plastic material and used for vials, having a body, a piston and a needle (Figure 19).

Figure 19. Syringe for vials

- for cartridges, they are either plastic and disposable or metal and can be sterilized (Figure 20)

The plastic disposable syringes have a capacity of 2-5 ml and atraumatic needle with a diameter of 0.6-0.8 mm with a length of 25-50 mm. These syringes have the possibility of aspiration by withdrawing the piston before injecting the anesthetic substance.

Cartridges have an aspiration device to penetrate the rubber diaphragm and hold the piston. The cartridge is inserted either by posteriorly displacing the piston or by the opening the syringe body in two halves.

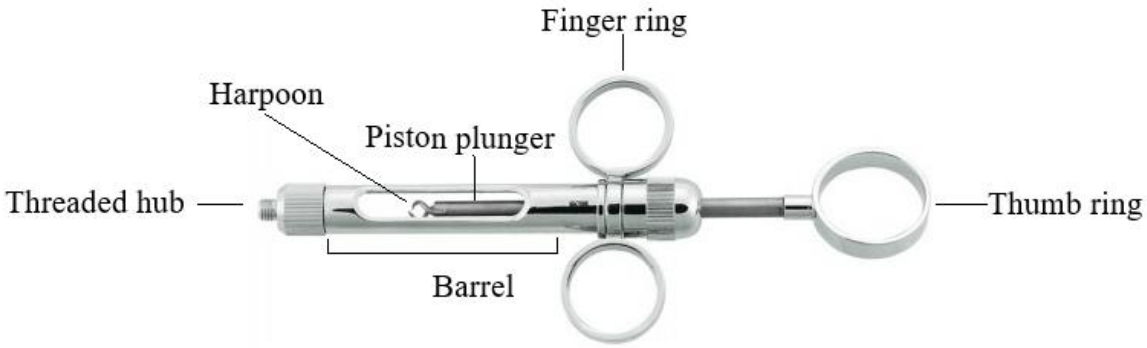


Figure 20. The cartridge syringe

A special type of syringes is those used for pressure injections (intraligamentary) having a "pen" shape and a strong arch that slightly pushes the anesthetic into the alveolar-dentary space, dosing it very well (Figure 20). To these syringes the cartridge is completely covered to minimize the risk of injury in case of vial rupture, considering the high pressure while injecting. The only disadvantage is the very rapid introduction of the anesthetic that can cause the patient's discomfort during the injection as well as the possibility of more frequent alveolitis by pressure ischemia.

Figure 21. Automatic syringe

- **The cartridges** composition consists of:

1. Anesthetic agent.
2. Vasoconstrictor agent (adrenaline or fenilpressine that increases the action duration of the anesthetic and its potency, but decreases the systemic toxicity and hemorrhage).
3. Reducing agent (prevents oxidation of the vasoconstrictor).
4. Fungicide.
5. Preservative agent (increases its validity. Most often this component of the anesthetic can cause allergic reactions).
6. Vehicle agent (must be isotonic, sterile, non-toxic, its pH compatible with the pH of the tissue).

The cartridges (Figure 22) are glass cylinders having at one end a rubber cap to represent the piston and at the other end a cap, usually aluminum, with an opening in the center to show the rubber diaphragm closing the vial and the needle penetration. All the cartridges have marked on them the anesthetic type and its concentration, as well as the added vasoconstrictor and its concentration, the substance volume and validity. On some cartridges, a scale is represented to mark the liquid volume to various levels, usually from 0.3 to 0.3 ml. Its standard size is 1.8-2.2 ml. Sometimes an air bubble appears in the cartridge. When the bubble has a small diameter, it is considered to be a normal nitrogen bubble to represent the medium, the anesthetic solution is loaded in, not to enter oxygen that can inactivate the vasoconstrictor. The larger bubbles are due either to manufacturing defects, the warranty period exceed or improper storage of cartridges (in the refrigerator and frozen). In all these cases, the cartridges can no longer be used.

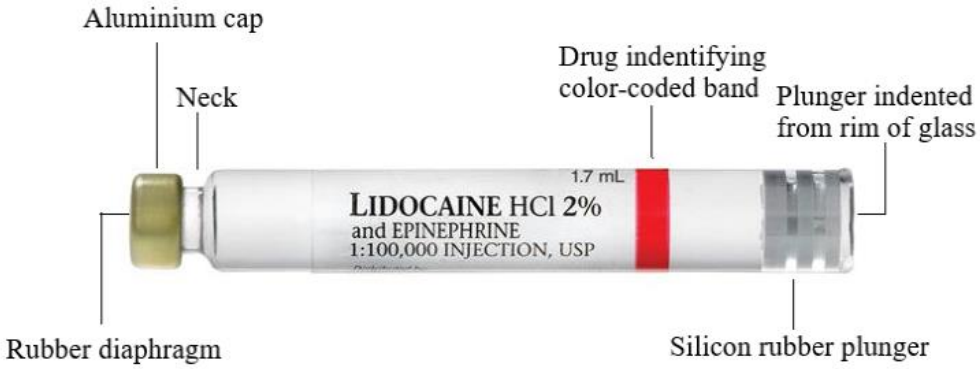


Figure 22. The cartridge components

Each cartridge has four components:

- Glass cylinder tube
- Rubber piston
- Aluminum cap
- Rubber diaphragm

- **The needle**

The needle allows the anesthetic agent move from cartridge inside tissues to be anesthetized. Most of the needles used in dentistry are made of stainless steel to have a single use only.

All needles have the following components (Figure 23):

1. Needle bevel
2. Needle shaft
3. Needle hub
4. Needle adapter
5. Cartridge penetration end

There are 2 types of needles to cartridges: short gauging 20 mm and long gauging 35 mm. Needles can have 25-30 caliber.

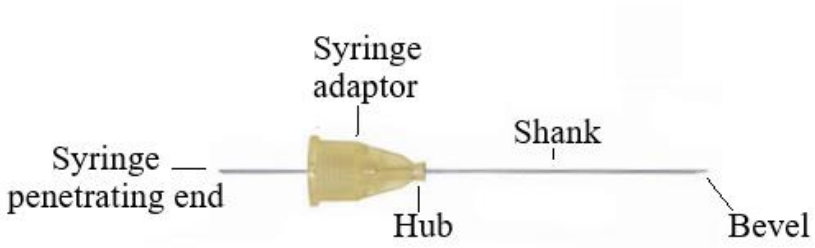


Figure 23. Anesthesia needle structure

Recommendations:

1. Only sterile disposable needles should be used.
2. In case of multiple injections, the needles should be changed every 3-4 injections.
3. Needles should NOT be used to few patients
4. They should not be inserted into the tissues to its hub unless it is absolutely essential to perform a certain anesthesia.
5. The needle direction should not be changed while it is inserted into the tissue.
6. It should never forced against resistance.
7. They must be disposed and destroyed after use, to prevent injury or reuse by unauthorized persons.

General stages of local anesthesia

Local infiltration anesthesia is performed according to a certain technique, strictly following a series of requirements:

1. Use sterile disposable needles.

The disposable sterile needles are sharp at the first use, but with each additional penetration their sharp tip is getting crushed. During the third, fourth injection, the patient feels an increased resistance of the injected tissues. Clinically, this is expressed by pain and post-anesthetic discomfort. Thus, it is recommended the sterile stainless steel disposable needle to be changed each third or fourth injection.

2. Check the permeability of the needle and move the piston (remove air)

3. The temperature determination of the cartridge with anesthetic.

This is not necessary if the cartridges with anesthetic solution are stored under room temperature conditions and the patient will not feel discomfort at injection temperature (too cold or too hot).

However, if stored in a refrigerator, they should be heated or better brought and kept at room temperature before use.

4. Position the patient correctly on the dental chair with the head adjusted to the anesthesia you intend to do.

During the anesthetic injection, the patient should be in a physiological position (the head and heart are parallel to the floor of the room, and the legs slightly raised above). This position minimizes the possibility of various complications that may occur during anesthesia, such as syncope (a symptom that results from cerebral ischemia and heart inability to pump oxygenated blood to the brain). However, depending on the type of anesthesia to be performed, the doctor adopts the patient a comfortable position for the procedure.

5. Dry the puncture site.

For this purpose, sterile gauze wraps are used, that in addition to drying the operating area, have the role of removing any residues.

6. Use a mucosal antiseptic (optional).

It is used to prevent puncture site infection. Iodine and chlorhexidine antiseptics can be used, but alcohol based ones should be avoided as they can cause burns.

7. a. Use a contact anesthetic to minimize puncture site pain.

A not too high anesthetic dose is applied on special applicators (to prevent an overdose- their absorption is high) and is kept for at least 1 minute.

7. b. Continuous communication with the patient

During topical anesthetic application it is advisable for the doctor to explain to the patient the reason he is using this type of anesthetic (to reduce his pain during the local anesthetic injection). This will decrease the patient's anxiety, thus reducing the risk of vaso-vagal reaction complications.

8. Make a firm contact of hands: the one with the syringe, support it by the patient, and with the other hand fix the mark sites.

At this stage, two essential things must be avoided: instability of the syringe (correct positioning the cartridge in the syringe, needle, and stability of all components of the syringe) and the support of the operator's hand by the patient's shoulder or hand.

9. Tune the tissues.

This allows the tissues "cutting", reducing the pain sensation. In the opposite case, when the tissues are free, they will be torn, broken by the needle bead. In the opposite case, when the tissues are free, they will be torn, broken by the needle bead.

10. The syringe is best not to be seen by the patient.

It reduces patient anxiety.

11. Do not touch the lips, cheeks, teeth, tongue and operating table with the needle, excepting only the puncture site.

12. The needle is inserted with the bevel towards the bone.

13. The needle is inserted firmly, slowly, a few drops of anesthetic to be injected as you go deeper inside.

14. Aspirate (required for peripheral trunk anesthesia).

15. Slowly inject the anesthetic, the optimal rate being 1 ml / minute.

16. Slowly retract the syringe until it exits the tissues.

17. Keep the patient under observation and record the use of the anesthetic on the observation sheet.

TECHNIQUES USED IN LOCAL-REGIONAL ANESTHESIA

I. Local anesthesia

1. Refrigeration
2. Contact
   a. *Topical application*
   b. *Imbibition*
   c. *Spraying*
3. Injection
   a. *Local infiltration*
      - *Plexus infiltration = anesthesia of the dental plexus (also called para-apical paraperiosteal)*
      - *Intrapapillary, intraligamentary, intraseptal, intraosseous, intrapulpular infiltration*
   b. *Field block*

II. Regional anesthesia

1. Peripheral trunk or nerve block
2. Basal trunk or basal anesthesia

Local anesthesia

1. REFRIGERATION ANESTHESIA

Local anesthesia is a type of anesthesia that acts directly on the receptors and nerve endings, the disappearance of sensitivity being strictly limited to the territory in which it is involved.

Indications to refrigeration anesthesia:

- Small area and superficial surgery.
- Temporary teeth extraction with increased root resorption action.
- Periodontal / mobile tooth extraction.
- Opening superficial abscesses to the mucosa or skin.

Anesthetic agents:

- Kelen (ethyl chloride) - produces a good freezing to the sprayed tissues, but has a number of disadvantages:

– Sudden cooling of the tissues is unpleasant and can cause pain;
– Strengthens tissues, making incision procedure difficult to undertake;
– Prolonged refrigeration anesthesia produces necrosis;

– Anesthesia projection on the vital teeth and especially decay ones causes intense pain;
– It is slightly flammable, hence the risk of accidents (it should be avoided any flame nearby, such as the use of thermo- or electric shock).

Refrigeration anesthesia technique

The patient's eyes are covered with a sterile field to avoid anesthesia projection on cornea that can get injured by Kelen. The intervention region is isolated from saliva with cotton and suction rollers. The Kelen jet is projected from 20 - 30 cm distance, walking on the area to be intervened. As the area is getting bleached, an immediate surgery has to be performed (incision, extraction, etc.).

Other substances: Ethyl bromide, Freon, Pharmaethyl- have a higher tolerability than Kelen.

2. TOPICAL ANESTHESIA (CONTACT ANESTHESIA)

For a number of anesthetic agents, topical anesthesia is based on the mucosa permeability having the property to determine the insensitivity to the superficial layer of mucosa and submucosal tissue (about 2-3 millimeters submucosa).

The concentration of the anesthetic is higher than the one used for the injection. A product (Figure 23) is used, having as active ingredient xylene in concentration of 5-10%, or sometimes tetracaine 2%, butacaine 4%, benzocaine 14-20%.

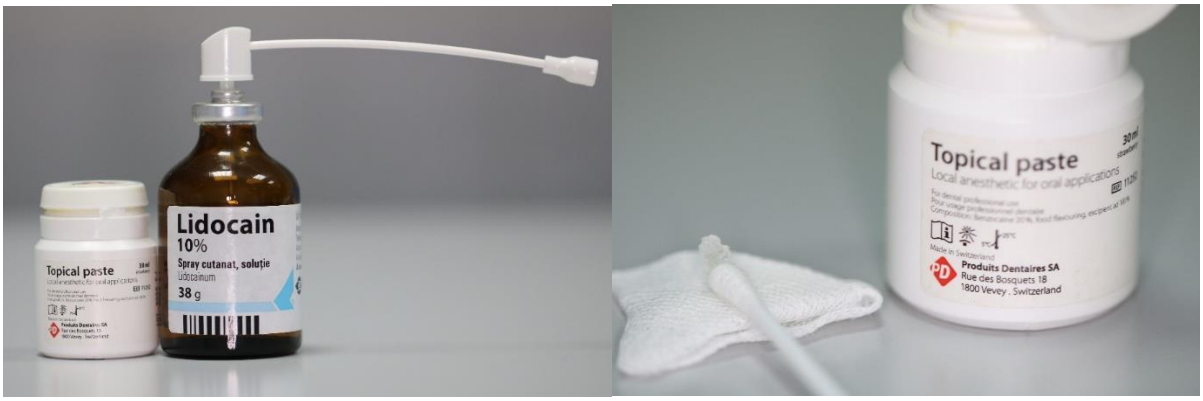


Figure 23. Local anesthetics

Indications for the local anesthesia:

- small interventions on the gingival fibromucosa (scaling, a crown adaptation , before the anesthetic puncture);
- suppressing the vomiting reflex in the case of the impressions;
- mobile temporary teeth extraction using increased root resorption;
- superficial abscesses opening;

- In the case of a relatively submucosal nerve, the technique is called "imbibition" and used for:
  1. anesthesia of the lingual nerve into the mandibular lingual groove, near the wisdom molar;
  2. Scarpo's nasopalatine nerve anesthesia on the nasal fossa floor, anteriorly positioned from the inferior cornet - The Escot Procedure.

Topical anesthesia technique:

1. roller insulation and pre-drying of the anesthetic application site;
2. the mixture application by using 2 methods - spraying and lubrifiation / (figure 24);
3. wait for the anesthesia output (2-3 minutes to 10-15 minutes) and then perform the necessary work.

The active duration of anesthesia lasts 10-15 minutes and can sometimes take up to 45-60 minutes. Although aerosol products have been widely used lately, it seems that pasta products are better as you can accurately determine the amount of anesthetic used.

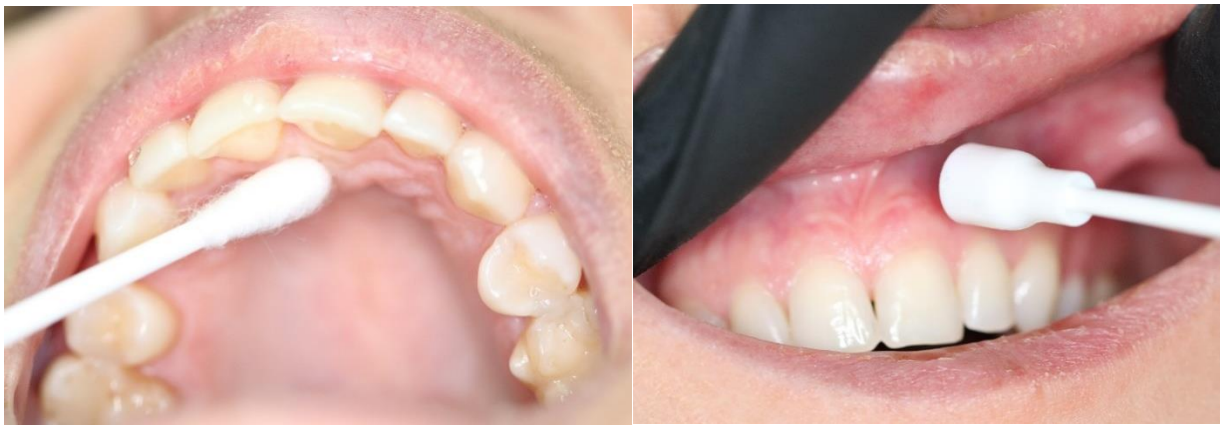


Fig. 24. Techniques of local anesthesia a. By batoning and b. By spraying

3. INFILTRATION LOCAL ANESTHESIA (INJECTION)

This technique involves the insertion of the anesthetic into the tissues by the syringe and distribution near the nerve endings or a nerve trunk (trunk). Local infiltration anesthesia can be applied to the mucosa of the oral cavity by submucosal puncture. For cervical-facial teguments the anesthetic puncture can be performed inter-dermally or subcutaneously, thus administering a superficial or deep anesthesia.

PLEXAL ANESTHESIA is the most commonly used anesthesia in the maxilla. It involves the anesthetic insertion between the mucosa and the periosteum, as well as, its diffusion through the Haversian channels into the thickness of the bone to anesthetize the dental branches before they enter the tooth apex. This anesthesia can only be used within regions with thin bone cortex so that the anesthesia can diffuse. The technique is applied to the maxilla throughout its length, except for

the six-year molar where the zygomatico alveolar crest obstructs the optimal diffusion of the anesthetic solution.

To the mandible, only the frontal area has a bony structure to make available the diffusion of the anesthetic by this technique. Plexus anesthesia is more effective to children and young people, having less dense bone cortex and a sponge with wider Haversian channels.

Indications for plexus anesthesia:

- dental extraction;
- apical resections;
- insertion of dental implants;
- periodontal surgery;
- removal of gingival tumors and small cysts.

The technique of performing plexus anesthesia:

1. The puncture is performed into the buccal vestibule and the mobile mucosa, above the tooth apex, the needle having the bevel oriented to the bone plane.
2. The direction of the needle is obliquely mesial and distally oriented above the tooth apex. The needle will be withdrawed leaving both mesially and distally a small amount of anesthetic solution, a procedure to allow the extension of the anesthetized territory (figure 25).
3. The amount of anesthetic commonly used for plexus anesthesia is 1.5-1.7 ml (1 cartridge).

This procedure provides anesthesia to1-2 teeth, the vestibular mucosa, the periosteum and the bone in the area the anesthetic substance has been inserted. Plexal anesthesia is mainly contraindicated to suppurative disorders located at the puncture site, the risk of dissemination as ulcerations or tumors.

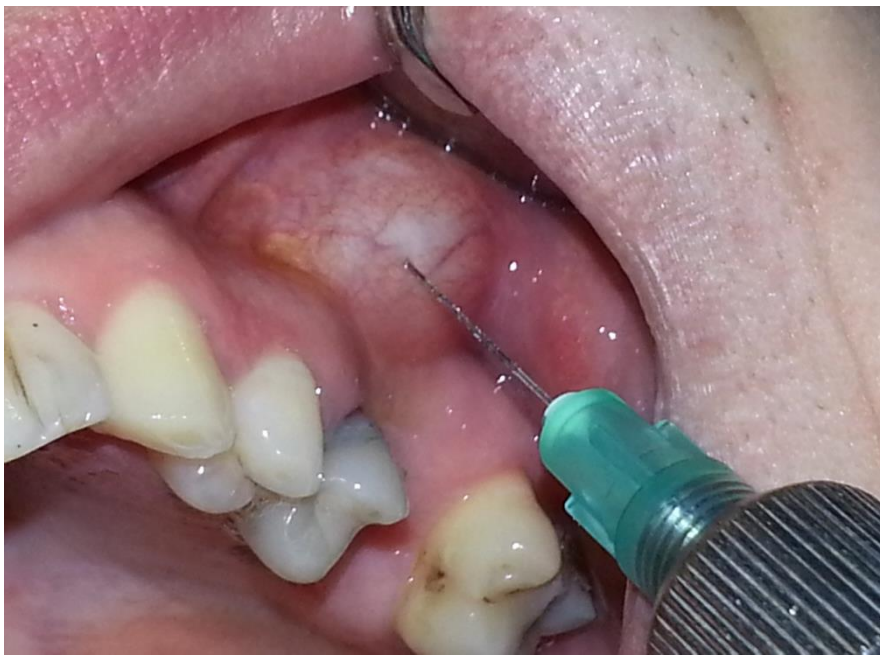


Figure 25. Plexus anesthesia technique

INTERLIGAMENTOUS ANESTHESIA is an anesthesia currently performed using special syringes (with automatic piston), to facilitate the technique.

Advantages:

- gives the possibility to administer anesthesia to a single tooth.
- has short anesthesia onset time (25-40 seconds).
- requires a small amount of anesthetic substance (0.15-0.20 ml).
- possibility to simultaneously anesthetize several teeth without the anesthetic overdosing.
- lack of anesthesia in the soft parts (the anesthetized area is reduced to the gingival fibromucosa, the alveolar bone and the dental vascular-nerve bundle).

Disadvantages:

- It requires special syringes.
- Post- extraction alveolitis frequently occurs due to the ischemia given by the pressure of the anesthetic substance inside the periodontal space, as well as, the presence of vasoconstrictors in the anesthetic.
- Post-anesthetic local pain is more common than in other anesthetic procedures.

Indications of intraligamentary anesthesia:

- patients with hemorrhage risk (hemophiliacs, patients undergoing anticoagulant treatment, patients with liver disorders, etc.), to whom intraligamentary anesthesia avoids the risk of deep puncture that may cause vascular or nerve damage.

The technique of performing intraligamentary anesthesia (Figure 26):

1. A contact anesthetic is used for the interdental papillae.
2. The needle is inserted through the dental papilla with the bevel oriented towards the tooth and penetrates the periodontal space, deposing 0.20 ml of anesthetic solution.
3. To the pluriradicular (maxillary, mandibular) teeth, intraligamentary anesthesia also involves vestibular and palatal / lingual puncture.

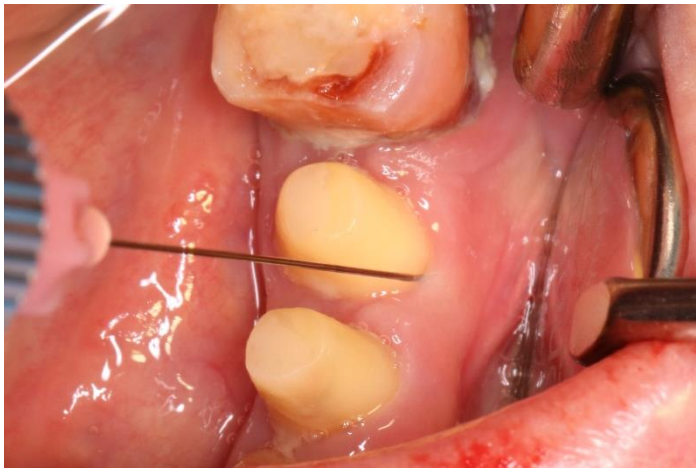


Figure 26. Intraligamentary anesthesia technique

INTRAOSOUS ANESTHESIA is a rarely used technique, usually performed in the mandible, to get the anesthetic solution infiltration inside the spongy bone by crossing through the cortical bone (Figure 27).

The indications and the anesthetized area are similar to the intraligamentary anesthesia. The onset time of anesthesia is short (about 30 seconds) and the duration of action varies from 15 to 45 minutes.

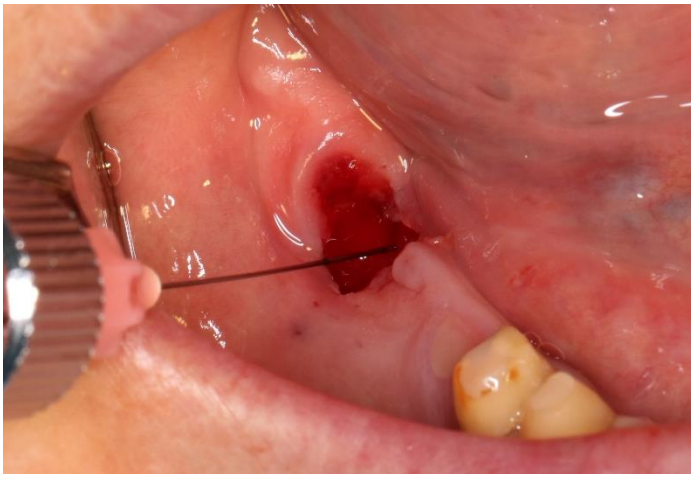


Figure 27. The technique of intraosseous anesthesia

INTRASEPTAL ANESTHESIA

The indications of this technique are very limited and are applied in: dental extractions to the hemophiliac patients having blood clotting problems.

The technique (figure 28):

- Operator territory processing.
- The puncture location is inside the papilla, 2 mm below the tip, equal distance from 2 adjacent teeth.
- The needle has a perpendicular direction on the vestibular surface of the papilla.
- A few drops of local anesthetic injected into the papillary mucosa, and proceeded with the needle to the bone, then the needle is pushed into the bone septum 1-3 mm, place 0.2 ml without pressure.

Advantages:

- It has quick onset (25-45 sec)
- It requires a small amount of anesthetic
- The soft parts have lack of anesthesia
- It is given to a single tooth

Disadvantages:

- It requires special syringes
- It produces postextractional alveolitis
- Post-anesthetic local pain is more common
- It can become risky, leading to necrosis of the bone septum

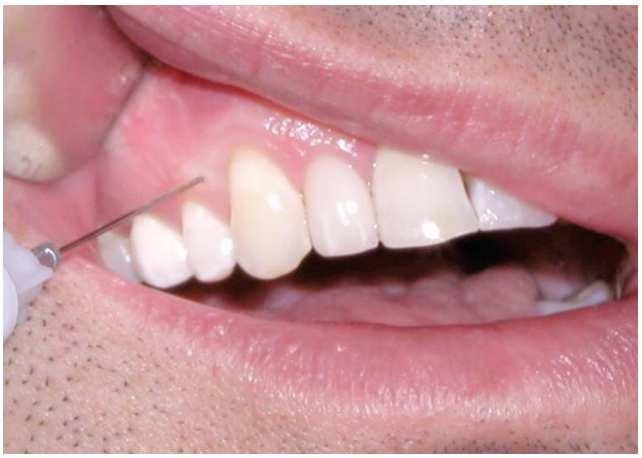


Figure 28. The technique of intraseptal anesthesia

INTRAPAPILLARY ANESTHESIA

It is an anesthesia performed by local infiltration to the dental papilla (figure 29).

Indications:

- It is rarely used, in the periodontal treatments, to get hemostasis during the treatment of periodontal diseases.

Contraindications:

- For patients having infections or tumors in injection puncture area.

Anesthetized areas:

- Neighboring bone, tooth or teeth and adjacent soft structures

Advantages:

- requires a low dose of local anesthetic (maximum 0.2 ml)
- needs a short time of onset
- very slightly traumatic
- avoids lip anesthesia
- reduces bleeding if vasoconstrictor is used

Disadvantages:

- Short duration of action
- Very limited area to anesthetized tissues

The technique:

- Operator field processing.
- The puncture location is inside the papillary triangle 2 mm below the tip of the interdental papilla, to an equal distance from the 2 adjacent teeth.
- The needle has a perpendicular direction on the vestibular face of the papilla.

Figure 29. Intrapappilar anesthesia technique

Regional anesthesia

1. PERIPHERAL TRUNK ANESTHESIA

Peripheral trunk anesthesia or nerve block (Table 4) is a local and regional infiltration anesthesia determined to act on a nerve pathway by interrupting the conductivity and anesthetizing the area of distribution (Figure 30). In contrast to the other types of anesthesia mentioned above focusing the terminal nerve threads, peripheral trunk anesthesia targets the nerve trunk and its branches. As it has a longer duration of action and does not deform the anesthetized region, peripheral trunk anesthesia allows therapeutic maneuvers to be performed on a larger surface and over a longer period of time.

Whenever trunk anesthesia is practiced along the branches of the maxillary or mandibular nerves, the techniques are called peripheral trunk anesthesia. Anesthesia of these nerves applied just at the very exit of the skull base (round hole for the maxillary nerve and oval hole for the mandibular nerve) constitutes ***the basal trunk anesthesia.*** Basal trunk anesthesia, widely used in oro-maxillo-facial surgery prior to the development and improvement of general anesthesia, are today techniques belonging to history.

Table 4. Peripheral trunk anesthesia techniques

Techniques	Anesthesia
The upper jaw	
Tuberosity anesthesia *Posterior superior alveolar nerve*	The molars of the maxilla (except for the mezio-vestibular root of the 1 maxillary molar), also the hard and soft tissues of the corresponding vestibular region.
Infraorbital nerve block *Anterior superior alveolar nerve and middle superior alveolar nerve*	The canine, the lateral incisors and laterally, with the hard and soft tissues of the corresponding vestibular region. The mesial vestibular root of the 1 upper molar, the premolars, the hard and soft tissues of the corresponding vestibular region.
Greater palatine nerve block *The greater palatine nerve*	The palate mucosa and the hard tissues from the first premolar towards posterior, to the midline of the palate.
Nasopalatine nerve block *The nasopalatine nerve*	The hard and soft tissues in the palatal region from one canine to another.
The lower jaw	
Spina Spix nerve block *The inferior alveolar nerve*	Mandibular teeth on the anesthetized hemiarch, soft vestibular tissues in the area of the incisors, canines and premolars, as well as the hemiarch on the injected site.
Buccal nerve anesthesia *The buccal nerve*	Soft vestibular tissues from the molar region
Mental foramen anesthesia *The mental and incisive nerves*	The soft vestibular tissues, towards the midline of the mental foramen, lower lip and chin. Premolars, canine, incisors, lower lip, chin teguments, soft vestibular tissues, towards the midline of the mental foramen.
Lingual nerve block *The lingual nerve*	The soft lingual tissues on the hemiarch, buccal floor and half tongue

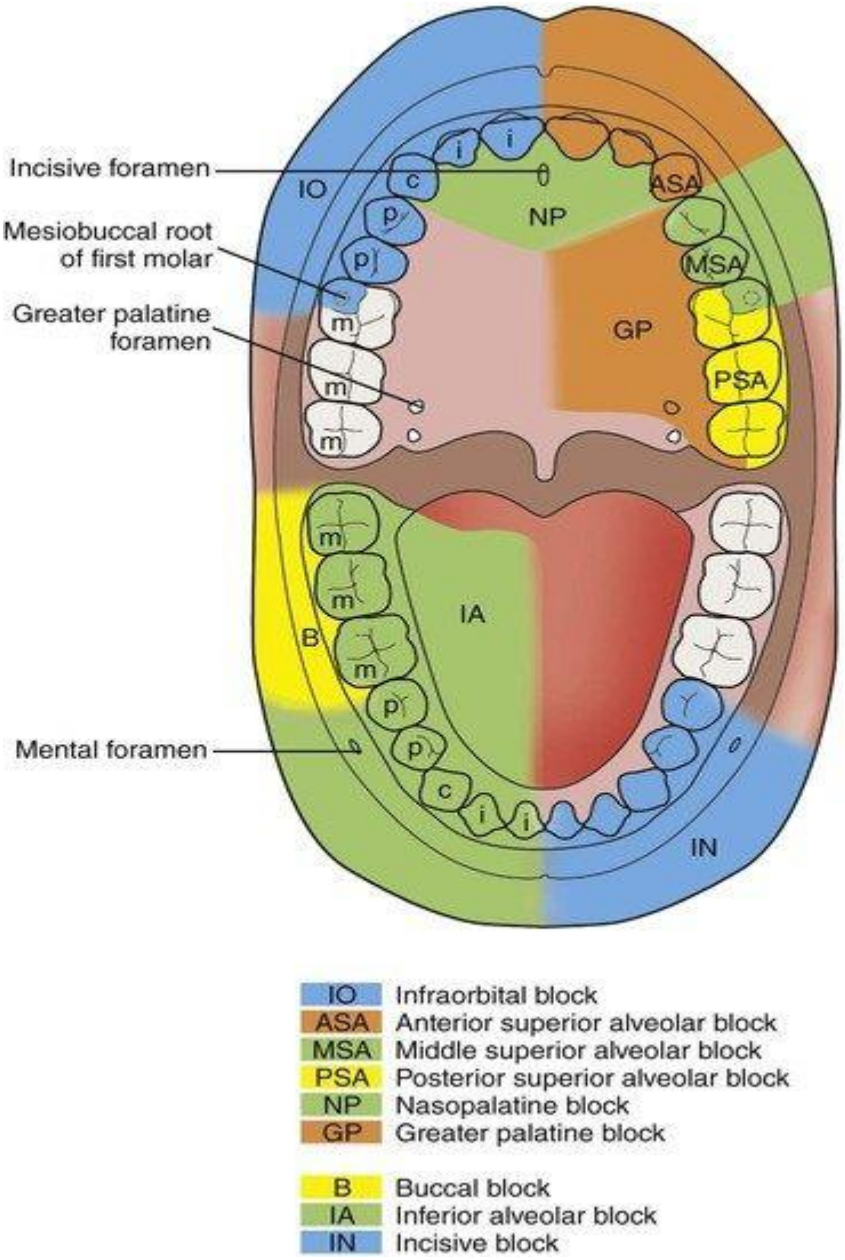


Figure 30. The anesthesia areas of peripheral trunk techniques

Peripheral trunk anesthesia (nerve block) of the upper jaw

1. Anesthesia of the posterior superior alveolar nerves

At the upper archway/arcade, the molar region is frequently anesthetized by a technique commonly defined in dentistry as "tuberosity" or zygomatic blockage, a procedure to include the posterior superior alveolar nerves (figure 31).

Any technique of peripheral trunk anesthesia should be described by a few elements related to ***the landmarks, the puncture site, the needle direction and depth of insertion, the amount of anesthetic administered.*** The landmarks to guide the puncture site may be main or essential, represented by the bone elements at the skull and remain stable throughout life. The secondary landmarks consist of teeth and soft parts.

The anesthetized areas:

The anesthetized area comprises the upper molars with the alveolar bone, the vestibular fibrous mucosa, the posterior wall of the maxillary sinus and the adjacent sinus mucosa.

However, the mesial-vestibular root of the six-year-old molar is not anesthetized, but sometimes the anesthesia may partially or totally encompass the premolar area.

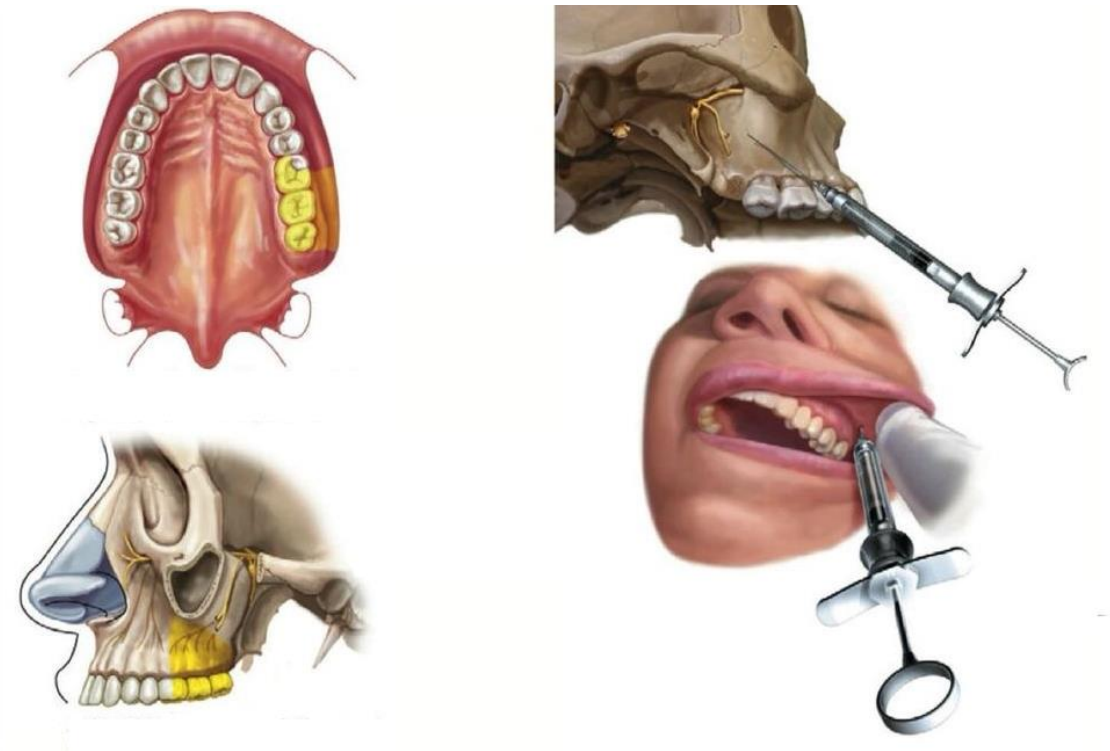


Figure 31. Posterior superior alveolar nerve anesthesia (*Atlas net* illustration)

Indications:

- "Tuberosity" anesthesia is administered for dental therapeutic or surgical procedures in the upper molar region (Figure 32), in cases when plexal anesthesia is inefficient or contraindicated.

Contraindications:

- inflammatory or tumor processes present in the distal third of the superior or retro-tuberosity vestibule.
- patients with hemorrhage risk (hemophiliacs, patients undergoing anticoagulant treatment, etc.), due to local anatomical conditions (pterygoid venous plexus).

Advantages:

1. It's atraumatic, if it technically undergoes a correct administration. The patient does not feel any strong pain, due to the large area of soft tissue, the anesthetic is stored and the direct contact with the bone.
2. It has a high success rate (> 95%)
3. A minimum number of injections is required (one tuberosity injection effect is equaled to that of 3 infiltrative injections)
4. It minimizes the total volume of injected anesthetic solution (equivalent volume of 3 plexus injections = 1.8ml)

Disadvantages:

1. The risk of hematoma, usually diffuse, creates discomfort and embarrassment to the patient.
2. It is somewhat an arbitrary technique without contacting the bone.
3. To about 28% of patients, a second injection is required to the treat the first molar (mesial vestibular root).

Alternatives:

1. Plexal anesthesia
2. Infiltrative anesthesia
3. The maxillary nerve block

Anesthesia of the superior-posterior alveolar nerves can be performed in two ways: orally (endo-buccal) and cutaneous (external-buccal). Cutaneous tuberosity anesthesia is performed extremely rarely in both dentistry and the current oro-maxillar facial surgery. The anesthesia puncture is performed inside the cheek before the masseter muscle, under the inferior edge of the zygomatic

bone, to the distal zygomatic alveolar ridge without perforating the oral mucosa. It may be prescribed in case the general status conditions contravene the general anesthesia having much higher risk than benefit. In the above mentioned context, the cutaneous procedure is indicated in case of any obstacles at the oral puncture site (abscesses, tumors, trismus). In current practice, oral procedures are most commonly used.

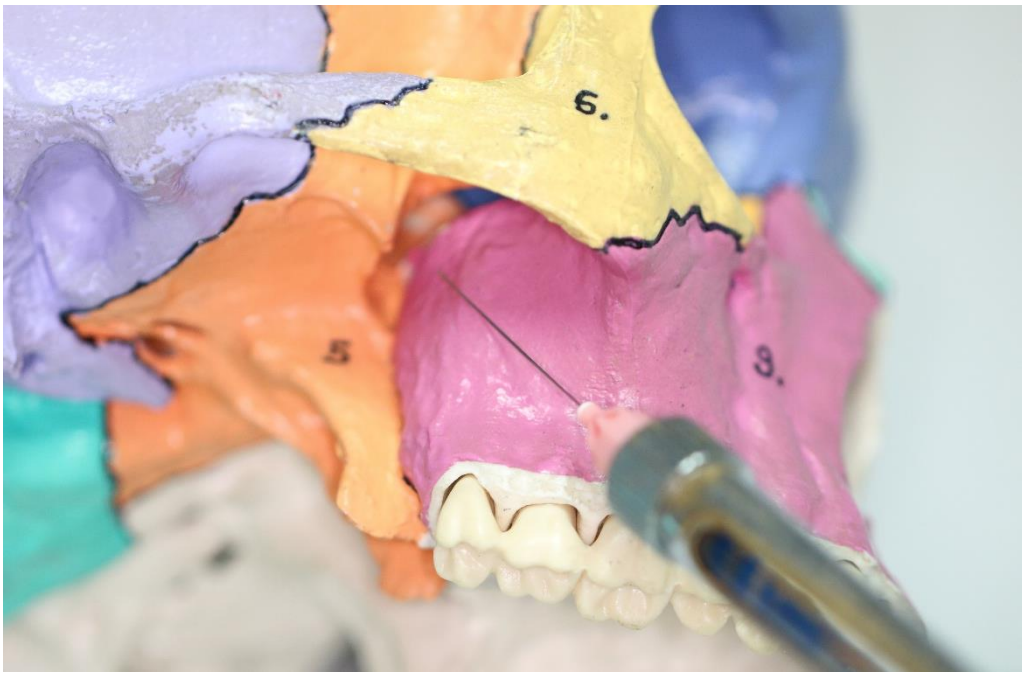


Figure 32. Anesthesia of the posterior superior alveolar nerves (illustration)

The landmarks for "tuberosity" anesthesia include:

- the zygomatic-alveolar ridge.
- the mesial root of the 12-year-old molar.
- the mobile mucosa.

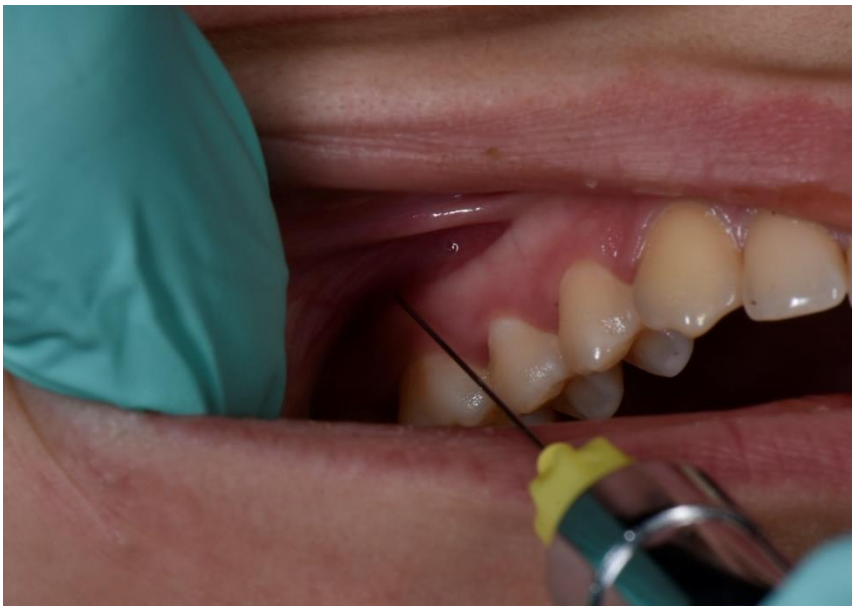


Figure 33. Anesthesia of the posterior superior alveolar nerves

Technique:

1. The patient is placed in the dental chair, the head is slightly in extension, opens slowly the mouth and the jaw deviates from the side intended for anesthetic puncture, so that the coronoid does not block access to the molar area.

2. The labial-genius soft parts are removed with the index of the left hand as the "tuberosity" anesthesia is performed on the right side and the left thumb fixes the bony puncture site (zygomatic-alveolar ridge) (figure 33).

3. The puncture is performed into the mobile mucosa above the medial root of the 12-year-old molar and distal to the zygomatic-alveolar ridge. The needle direction gets obliquely upward, backward and inward to form an angle of 45 ° with the occlusion plane of the upper molars. The needle is inserted with a single continuous movement combining all three different movements.

4. After the bone contacting, the needle penetrates along the tuberosity and maintains the bone contact to a depth of 2-2.5 cm. As the needle progresses penetrating the tissue, aspiration is required to check if it didn't get inside a vessel of the pterygoid plexus. The anesthetic solution will be injected progressively, continuously, as the branches of the posterior superior alveolar nerves enter the bone at different levels (Figure 34).

5. The amount of anesthetic administered is 1.7-2 ml.

6. Whenever bone or anatomical contact is not maintained, the pterygoid venous plexus gets a very low position determining a tuberosity hematoma risk. The resulting bleeding, caused by the needle pinching to pterygoid venous plexus, has a rapid extension character, with no strict bone limits to the pterygoid -maxillary space. Emergency treatment consists of measures to minimize the hematoma by compressing the cheek under the zygomatic bone with the palm bridge, a procedure associated with the oral compression by placing a tampon at the bottom of the upper vestibular sack. Antibiotic and anti-inflammatory drug treatment is also prescribed to prevent infection of the hematoma.

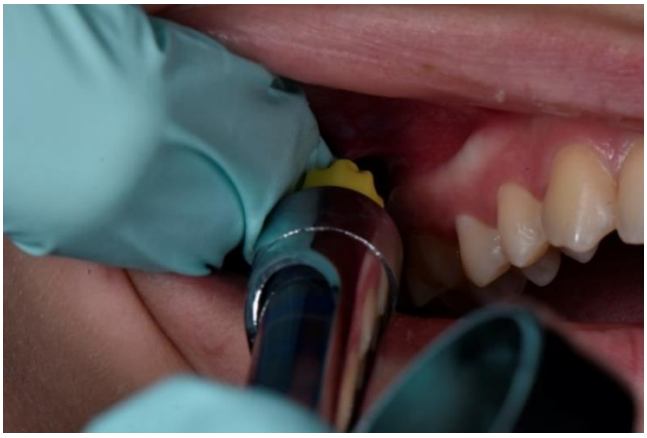


Figure 34. Anesthesia of the posterior superior alveolar nerves

Possible failures:

1. Needle is oriented too laterally. For correction: redirecting the needle towards the medial site.
2. The needle is not inserted deep enough. For correction: upper needle redirecting.
3. The needle is inserted too posteriorly. For correction: retracting needle to the required depth.

2. Anesthesia of the nasopalatine nerve

This technique provides the incisive nerve and the sphenopalatine nerve block (Figure 35).

The anesthetized area:

- The palatal gingival mucosa is innervated in the anterior area by the nasopalatine nerve. The nerve emerges the hard palate through the orifice of the nasopalatine channel, anteriorly positioned to the inter-incisive papilla, 1 cm posterior to the central inter-alveolar uvula.
- Anesthesia induces insensitivity to the palatal mucosa from the midline up to the canine of the respective side. The superior canine is innervated by two nerves: nasopalatine, or Scarpa nerve, and greater palatal nerve, so that a tooth extraction will require anesthesia to both nerves.
- In the oral cavity, the incisive papilla covers the incisive foramen, located on the midline, immediately behind the central incisors. The two incisal canals join at the incisive foramen level, then are separated by a thin bony blade and take an oblique direction upward, backward and forward, opening on the nasal fossa floor in both sides of the nasal septum, the front section image being "V" or "Y".
- The anesthetized region refers to the anterior third of the palatine fiber-mucosa, while the posterior demarcation is a transverse line from one canine to the other.

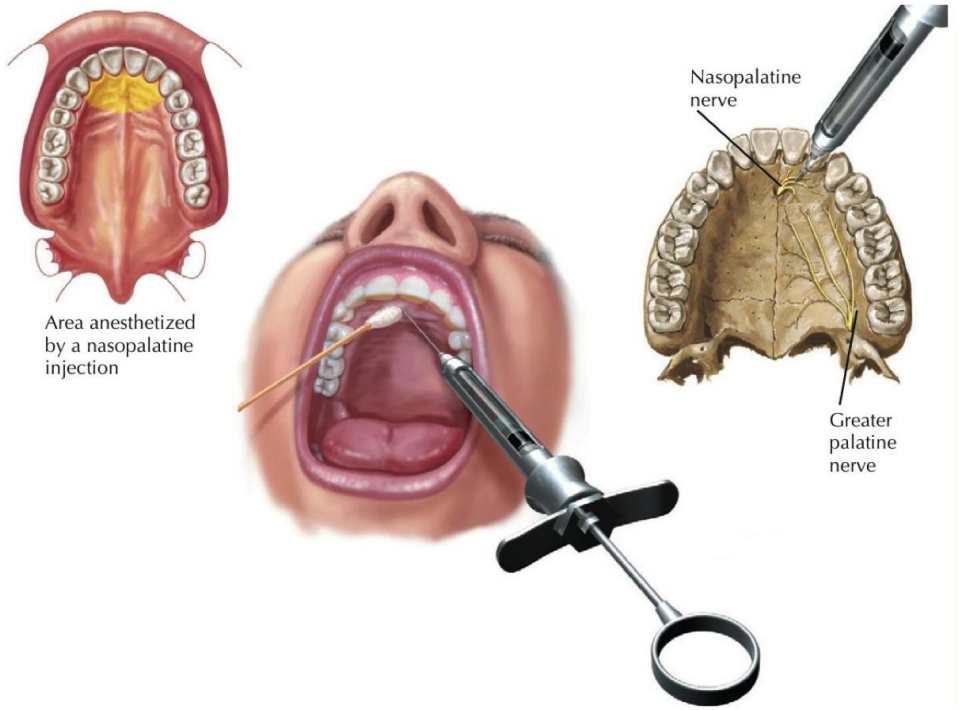


Figure 35. Nasopalatine nerve anesthesia (*Netter atlas* illustration)

Indications:

- It comes in combination with plexus or peripheral trunk anesthesia of the infraorbital nerves for surgeries undertaken in the group of upper front teeth region. In dentistry, the most common procedure used for anesthesia in the incisive foramen (anterior palatal foramen) is practiced orally.

Landmarks:

The anesthetic puncture is done at the incisor papilla level covering the incisive foramen and is located (figure 36):

– on the midline, palatally, between the upper central incisors.

– 0.5 cm backward and above the cingulum of the upper central incisors.

This anesthetic procedure is quite painful due to the rich innervation of the papilla, the adhesion of the palatine fiber mucosa and the lack of connective tissue at this level. In order to prevent the pain caused by the anesthetic puncture, contact anesthesia or the puncture on the edge of the papilla should be performed by injecting it as slow as possible.

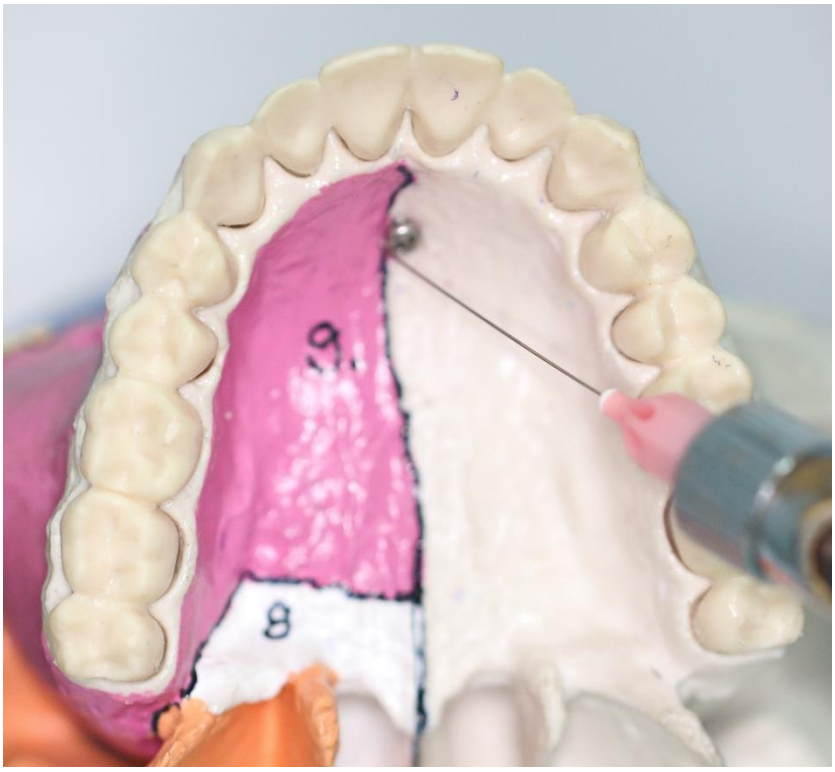


Figure 36. Nasopalatine nerve anesthesia (illustration)

Technique:

- The needle will be inserted laterally on the edge of the incisal papilla, 0.5 cm behind and above the gingival margin of the upper central incisors.
- The needle is directed upward, backward and outward, entering about 0.5 cm the incisor canal.
- It runs parallel to the axis of the central incisor and introduces 0.20-0.50 ml anesthetic solution.

The anesthesia technique of the nasopalatine nerve has been modified by some authors to make it less painful by avoiding itching around the incisive papilla (Figure 37). Malamed describes the anesthetic puncture in the upper lip frenulum as to introduce 0.2-0.3 ml anesthetic solution, wait for a few minutes, then make a puncture in the interdental papilla between the central maxillary incisors on the vestibular crest at the base of the lip frenulum and enter perpendicularly between the incisors towards the inter-incisive papilla inside the vault and slowly inject the anesthetic (0.3-0.4 ml).

After a few minutes, if the sensitivity of the area persists, a pain-free palatine puncture can be performed.

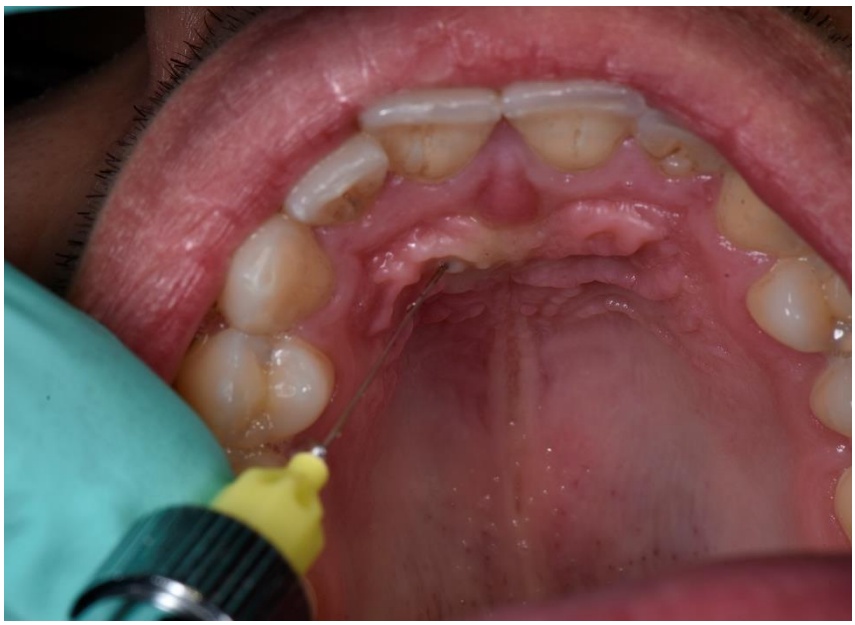


Figure 37. The nasopalatine nerve anesthesia

Contraindications:

1. Infections or inflammation on the territory to be anesthetized;
2. A smaller area of treatment (one or two teeth).

Advantages:

1. Minimizes the number of needle punctures and the volume of the anesthetic solution.
2. Reduces patient's discomfort caused by multiple needle penetrations.

Disadvantages:

1. An adequate hemostasis cannot be performed, except for a small area near the injection site.
2. It is probably the most traumatic intraoral injection.

Alternatives:

1. Plexal anesthesia.
2. Basal trunk anesthesia of the maxillary nerve.

3. Anesthesia of the anterior palatine nerve (large palatine nerve)

Palatal fibrous mucosa in the posterior region (distal to the canine) is innervated by the large palatine nerve, a branch of the maxillary nerve passing through the large palatine foramen (posterior palatine foramen) to enter the hard palate (figure 38).

Anesthetized areas:

The posterior part of the hard palate and the tissues covering it anteriorly to the first premolar and medially to the midline of the palate.

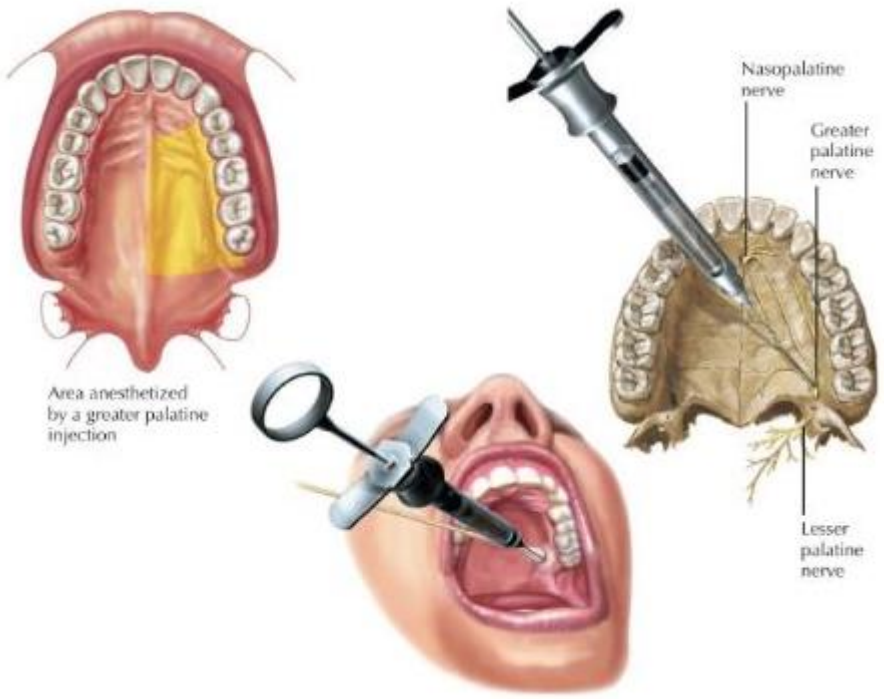


Figure 38. Anesthesia of the anterior palatine nerve (*Netter atlas* illustration)

Indications:

- to get the fibrous mucosa in the posterior 2/3, at premolar and molar level anesthetized.
- usually administered to complete a plexal or a tuberosity anesthesia, in case of therapeutic procedures performed in the posterior region of the maxilla.

Contraindications:

1. Presence of inflammation or infection in the area to be anesthetized.
2. A smaller area of treatment (one or two teeth).

Advantages:

1. Minimizes the number of the needle punctures and the volume of the anesthetic solution.
2. Provides minimal discomfort to the patient.

Disadvantages:

1. Impossibility to perform a good hemostasis.
2. Traumatic potential.

Alternatives:

1. Plexal anesthesia.
2. Basal trunk anesthesia of the maxillary nerve.

Landmarks:

- the last molar, 1 cm above the gingival festoon.
- 0.5 cm before the posterior edge of the hard palate, on the dihedral angle formed by the alveolar ridge and the horizontal blade of the palatal bone.
- 1 cm before the hook of the inner wing of the pterygoid apophysis.

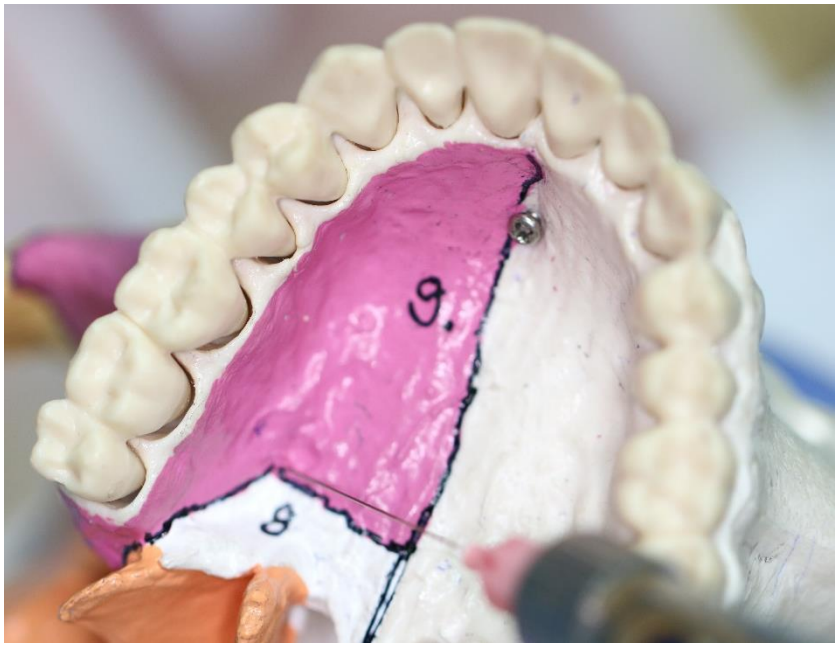


Figure 39. Anesthesia of the anterior palatine nerve (illustration)

Technique:

- The anesthetic puncture is made in the palatine channel near the second molar, where the mucosa is “plugged into the funnel” (Figure 40).
- The needle is directed upward, backward and slightly outside to let the syringe reach the commissure on the opposite side.
- The needle is not intended to enter the channel as 0.5 ml of anesthetic solution is enough for the area to get anesthetized.

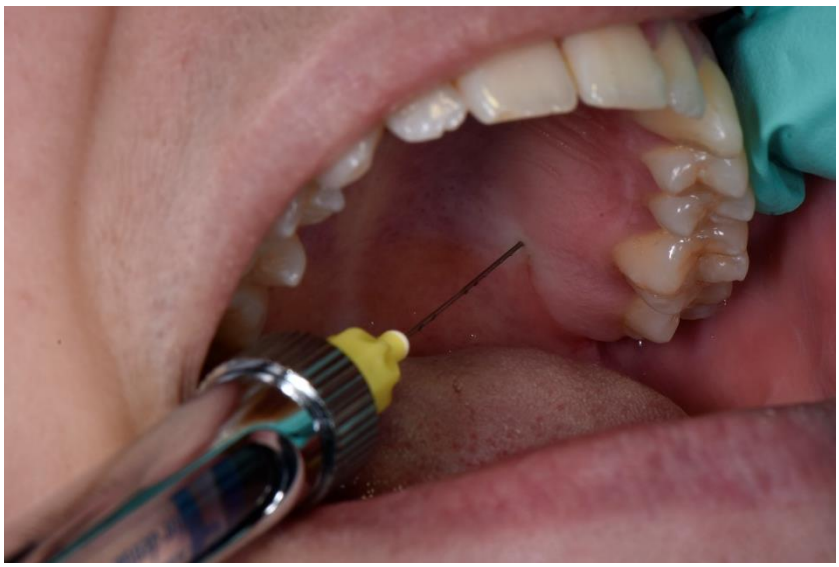


Figure 40. Anesthesia of the anterior palatine nerve

Possible complication effects:

- hemorrhage by pinching the palatal vessels, thus, the hemostasis is performed by digital compression for several minutes.
- sudden injecting a large amount of anesthetic results in mucous periosteum take-off with the risk to limited necrosis of palatal fibrous mucosa
- infiltration of the soft veil inducing a transient edema as the needle enters to the posterior part.

Infiltration anesthesia of palatal fibrous mucosa

Indications:

- for palatine fibrous mucosa, in case, the surgery targets a small area (1-2 teeth).

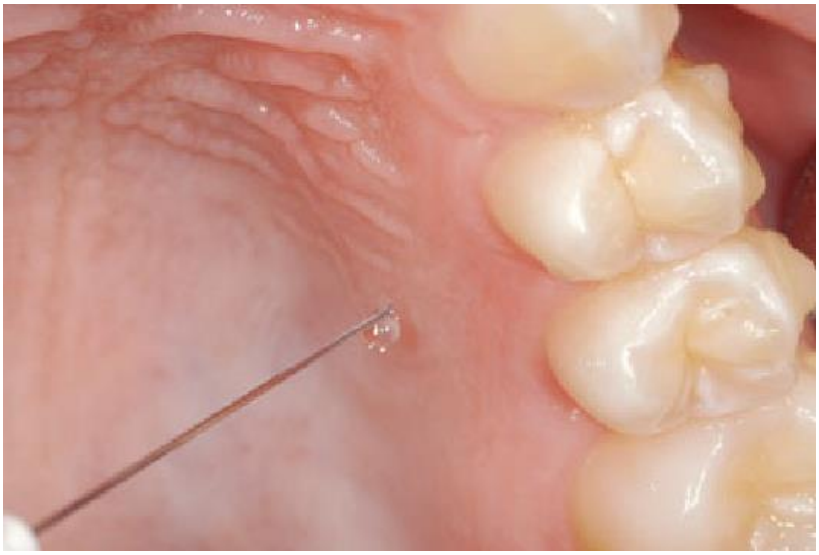


Figure. 41. Infiltration anesthesia of palatal fibrous mucosa

Technique:

- The anesthetic puncture is performed 1 cm from the gingival margin by holding the needle perpendicularly to the bone (figure 41).
- The amount of anesthetic solution recommended is 0.30-0.50 ml.

4. Anesthesia of the anterior superior alveolar nerves (infraorbital n.)

Anesthesia area (Figure 42):

- upper front teeth (central incisor, lateral, canine) on the anesthetized side.
- the alveolar process between the midline and the first premolar (in case, the mid-alveolar nerve doesn't exist)
- vestibular mucosa and periosteum in this area
- the anterior wall of the maxillary sinus and the mucosa lining it
- half of the upper lip
- the nose wing
- the lower eyelid

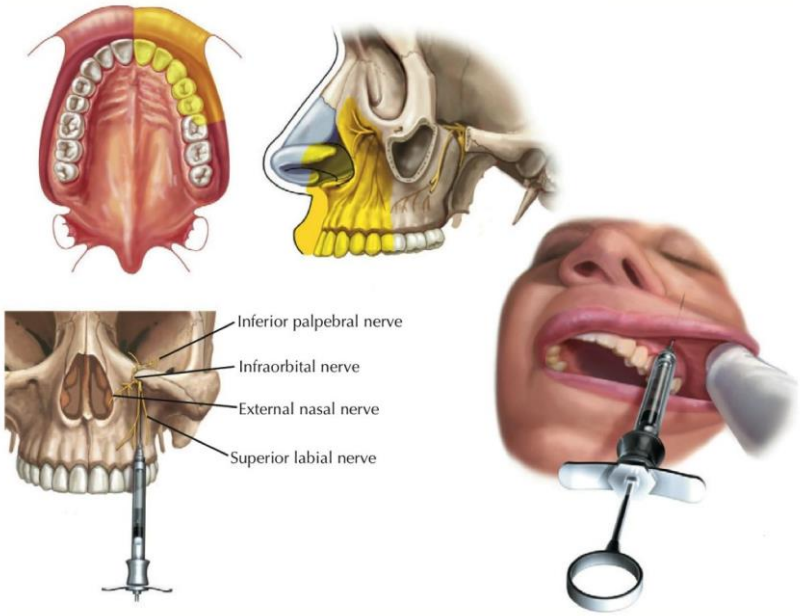


Figure 42. Anesthesia of the anterior superior alveolar nerves (Infraorbital n.)
(*Netter atlas* illustration)

Indications:

1. Therapeutic works targeting more than two maxilla teeth and their covering tissues.
2. Anesthesia of the vestibular mucosa and alveolar bone in the frontal area.
3. Anesthesia of the anterior wall to the maxillary sinus.

4. Anesthesia of the cheek, lower eyelid, nose wing, lower lip.
5. Inflammation or infection presence that does not allow the plexal anesthesia to be used.
6. As plexal anesthesia was ineffective due to the thickness of the cortical bone.

Contraindications:

1. Reduced area of treatment.
2. The necessity to induce a vasoconstriction in the infraorbital nerve-dependent area, as the infiltration of the anesthetic associated with the vasoconstrictor must be supra-periosteum.

Advantages:

It's a quite simple technique.

Quite safe, as it reduces the volume of anesthetic used and the number of needle pinches.

Disadvantages:

1. Psychological:
    a. Doctor: he may have fear not to traumatize the patient's eye.
    b. Patient: the needle can penetrate and disrupt the nerve while applying the oral technique.
2. Anatomical: difficulty in appreciating the anatomical marks.

Alternatives:

1. Plexus anesthesia to each tooth.
2. Infiltrative anesthesia to periodontium and hard tissues.
3. Basal trunk anesthesia of the maxillary nerve.

Landmarks:

The infraorbital hole is located (Figure 43):

- 6-8 mm below the lower orbital rim.
- at the junction site of the external 2/3 with the internal 1/3 of the infraorbital margin, under the zygomatic maxillary suture.
- 5 mm inside the medial pupillary vertical line.
- on the vertical line passing between the two upper premolars.
- on the same vertical line joining the supraorbital hole with the mental foramen.

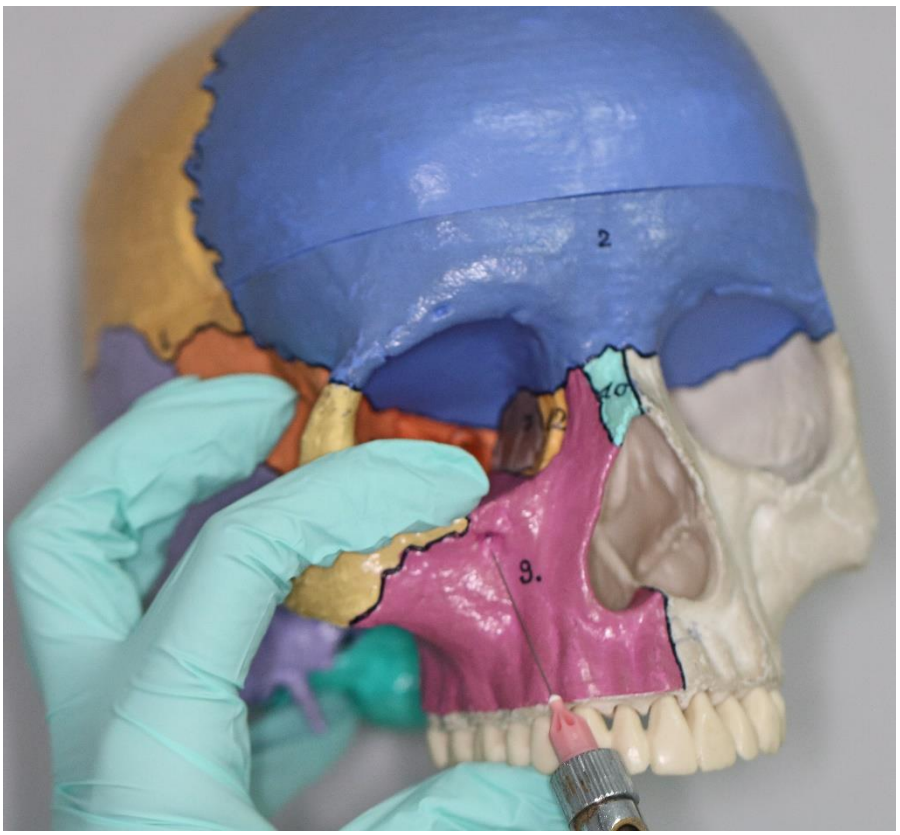


Figure 43. Anesthesia of the anterior superior alveolar nerves (Infraorbital n.)

Technique (Figure 44):

- Oral anesthetic puncture is practiced in the canine fossa, the mobile mucosa, above and laterally the top of the canine root.
- After contacting the bone, the needle passes along the canine fossa, pointing upward, backward and outward, just to enter the infraorbital hole.
- During this technique, the left hand index will be suborbital fixed to perceive the needle entering inside the channel.
- To anesthetize the front teeth, the needle is necessary to penetrate 6-10 mm inside the channel as the anterior superior and middle superior alveolar nerves detach inside the canal at this level.

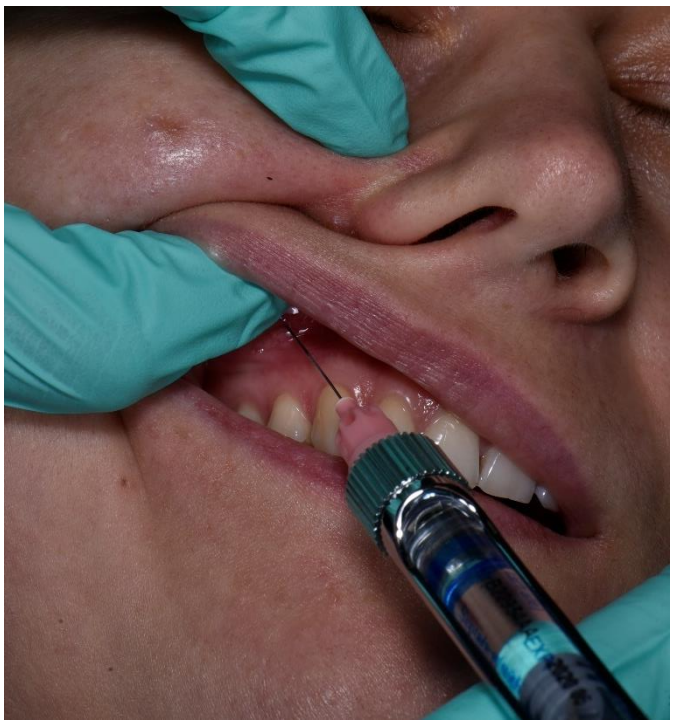


Figure 44. Anesthesia of the anterior superior alveolar nerves (infraorbital nerve)

Peripheral trunk anesthesia of the mandible

1. Anesthesia of the inferior alveolar nerve (Spina spix or inferior alveolar nerve block)

Anesthesia of the inferior alveolar nerve is one of the most used techniques in current practice both in dentistry and maxillofacial surgery.

Anesthetized areas (Figure 45):

The anesthetized area makes available surgeries to the bone, the teeth, the vestibular gingival mucosa (from the chin hole to the midline) on a hemi-arcade, as well as the labial and mental soft parts, excepting the area innervated by the buccal nerve (the vestibular mucosa distal to the chin hole). This anesthesia is commonly practiced orally in dentistry. But, it has rarely a cutaneous use in current oro-maxillofacial surgery, only in case inflammatory or tumor processes attended by trismus do not allow access to the puncture site.

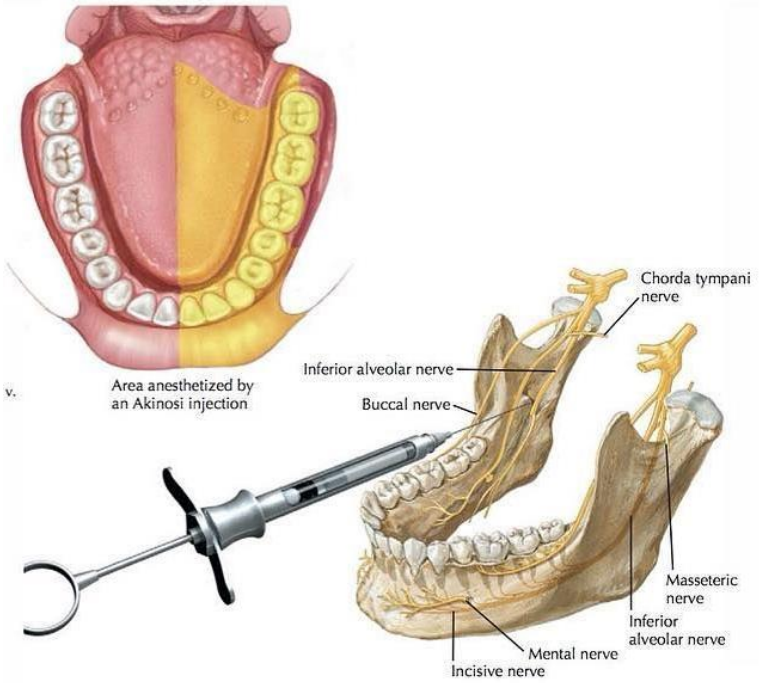


Figure 45. Inferior alveolar nerve anesthesia (*Netter atlas* illustration)

Indications:

1. Surgical interventions to several teeth in a hemi-arcade
2. In case the oral and lingual soft tissues are necessary to be anesthetized

Contraindications:

1. Inflammation or infections in the area intended for anesthesia practice
2. Patients who can bite their lips or tongue (very young children with disabilities)

Advantages:

1. A single injection provides a large area with anesthesia

Disadvantages:

1. Large anesthetized area (not required for localized procedures)
2. Rate of inadequate anesthesia (15-20%)
3. Intraoral anatomical landmarks are not quite accurate.
4. Anesthesia of the tongue and lower lip leads to discomfort for the patients and could induce post-treatment accidents.

Inferior alveolar nerve block has the following landmarks (Figure 46):

- temporal, medial and posterior ridge to the anterior margin of the mandibular branch
- pterygoid mandibular sac located along the anterior margin of the internal pterygoid muscle
- occlusion plane of the lower molars

Figure 46. Inferior alveolar nerve anesthesia (Spina spix anesthesia) (illustration)

Intraoral technique (Figure 47):

- The patient is placed in the dental chair keeping his head right and mouth widely open, to highlight the pterygoid raphe;
- The temporal ridge of the mandible is palpated with the left hand index, in case, the anesthesia is practiced on the right side or the left thumb, when injected on the left side, the arm going round the patient's head. The finger remains fixed to the puncture mark, along the cheek keeping the soft parts in tension.
- A long needle is inserted between the outside temporal ridge and the pterygoid-mandibular raphe, 1 cm above the occlusion plane of the lower molars in a dentate patient and 1.5 cm for the edentulous patient, the syringe body is parallel positioned to the dental arcade on the same side. In children, the puncture is made between the two landmarks, at the occlusion plane of the lower molars level.
- First, the needle direction is sagittal, anterior - posteriorly along the inner face of the ascending branch.
- 1 ml of anesthetic solution for the lingual nerve is aspirated and injected to 1 cm depth.

- As the needle goes deeper, the direction changes, due to the ascending branch oblique position, so that the syringe moves towards the midline, then towards the premolars or even the molars on the opposite side;
- To 1.5 cm depth the needle exceeds Spix spine, reaches the inferior alveolar nerve and deposit 1.5 ml anesthetic solution;

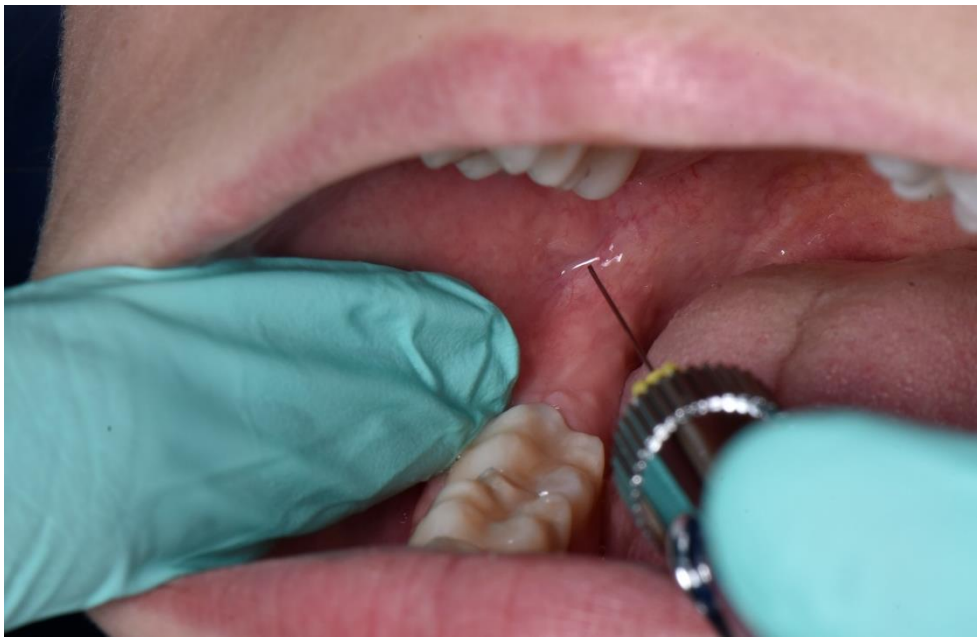


Figure 47. Inferior alveolar nerve anesthesia (Spix spinal anesthesia)

Extraoral techniques are recommended in patients dealing with trismus, infections or tumors.

Extraorally, the inferior alveolar nerve can be anesthetized using the submandibular, retromandibular and superior methods

❖ ***The submandibular method***

The patient's head is turned back and the opposite side of the anesthetic site to expose the mandibular angle. The posterior margin of the ascending branch and the mandibular angle are marked. The skin is used iodized alcohol antiseptic and a long needle (7-8cm) punctures about 1.5cm before the mandibular angle, below the basilar margin of the mandible.

The needle is directed upwards and slightly backwards, on the inner face of the ascending branch, parallel to the posterior margin, permanently maintaining contact with the bone. It penetrates a 4-4.5 cm depth to anesthetize the inferior alveolar nerve.

To anesthetize the lingual nerve, the needle is inserted 5.5 cm above and 6.5 cm to get the buccal nerve anesthetized.

❖ ***Sicher retromandibular method***

It is rarely used, only to weak patients and the ones having prominent ascending branches. The patient has his head obliquely turned to the opposite side. The puncture is made under the ear lobe, backward and inside the posterior margin of the ascending branch, at half its height. Insert the

needle 2-3cm depth with a lateral and forward direction, permanently maintaining contact with the bone.

Accidents: temporary paresis of the facial nerve, puncture of the external carotid artery, the external jugular vein and the facial vein.

The superior method is rarely used. A curved needle is inserted under the temporo-zygomatic arcade before the mandibular condyle. Going through the shallow planes and the masseter muscle, the needle reaches the sigmoid incision. Here, the needle retains the contact with the bone and takes a downward direction parallel to the posterior margin of the ascending branch getting a 2-3 cm depth to inject an anesthetic solution.

Currently, these techniques are no longer used and have been replaced with general anesthesia.

2. Peripheral trunk anesthesia of the lingual nerve

This anesthesia is applied to get the insensitization to:

- the mucosa of the ventral face and the margin of the tongue from the base towards the tip;
- the mucosa of the tongue dorsal face before the lingual V towards the midline;
- the mucosa of the buccal floor;
- lingual gingival mucosa from the midline to the last molar.

The lingual nerve can get anesthetized using several techniques:

a) peripheral trunk;
b) local infiltration;
c) contact.

a) Peripheral trunkular anesthesia:

Due to the anesthesia of the inferior alveolar nerve to the Spix spine, it is the most used technique in the dental practice.

The lingual nerve is anesthetized at 1 cm depth from the puncture site, described above (see anesthesia at Spina Spix) keeping the syringe parallel to the occlusion plane of the lower molars to deposit 0.5-1 ml of anesthetic solution.

Simultaneous anesthesia - see simultaneous anesthesia.

b) Local infiltration anesthesia

Separate anesthesia of the lingual nerve in the mandibular lingual groove (Figure 48).

The main ***landmarks*** are:

- the last lower molar;

- the mandibular-lingual groove to halfway between the tongue and the lingual slope of the alveolar ridge.

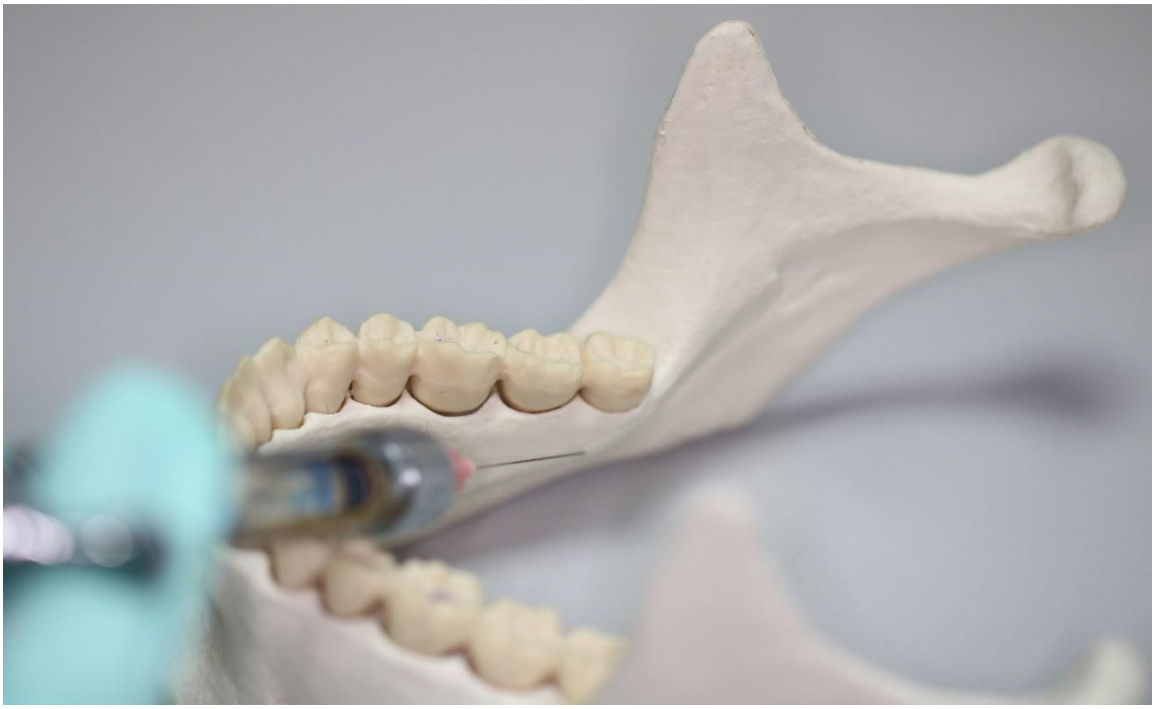


Figure 48. Lingual nerve anesthesia (illustration)

Technique: the patient is seated in the dental chair with his head straight and his mouth wide open. Then, the mirror is used to remove the tongue and highlight the mandibular-lingual groove and give the mucous membrane antiseptic.

The needle is inserted into the mucosa of the buccal floor in the lower molar right, to the equal distance between the gum and the tongue base (Figure 49).

It passes through the mucosa to a depth of about 1-2 mm without penetrating the milohioid muscle and injects 0.5-1 ml of anesthetic solution.

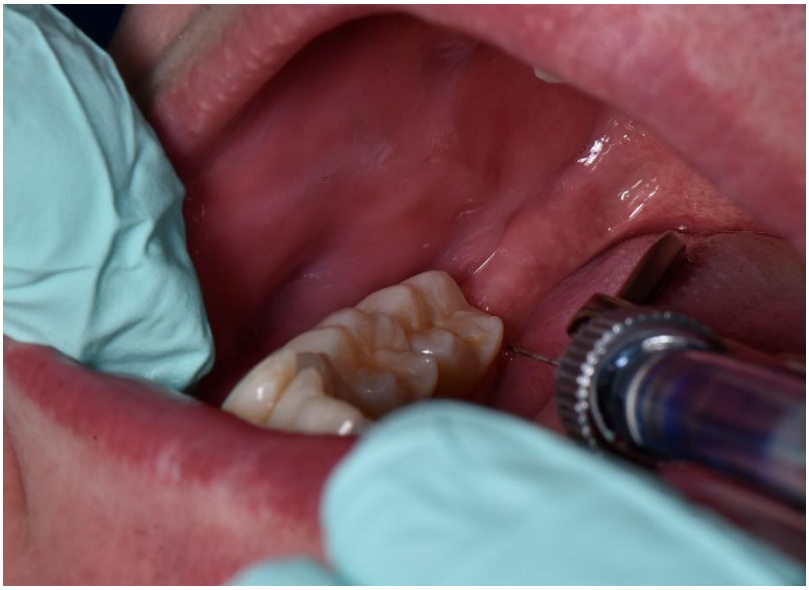


Figure 49. Anesthesia of the lingual nerve

c) ***Contact anesthesia:*** by applying a tampon soaked with anesthetic solution into the mandibular lingual groove, next to the last molar. This technique is based on the superficial lingual nerve and the permeable oral mucosa for anesthetic solution insert.

3. Peripheral truncular anesthesia of the buccal nerve

The buccal nerve, the terminal branch of the temporal buccal nerve innervates the jugal mucosa and vestibular gingiva from the right of the second premolar to the last molar through the internal branch and the skin of the cheek through the external branch.

Indications:

Surgical interventions on the soft tissues of the cheek

Complete the anesthesia of the inferior alveolar nerve while working on the inferior premolar and inferior molars.

Anesthesia of the buccal nerve can be performed intraorally and extraorally.

The following techniques can be used intraorally:

a) peripheral trunk anesthesia;
b) terminal anesthesia (Figure 50);
c) simultaneous anesthesia (see simultaneous anesthesia).

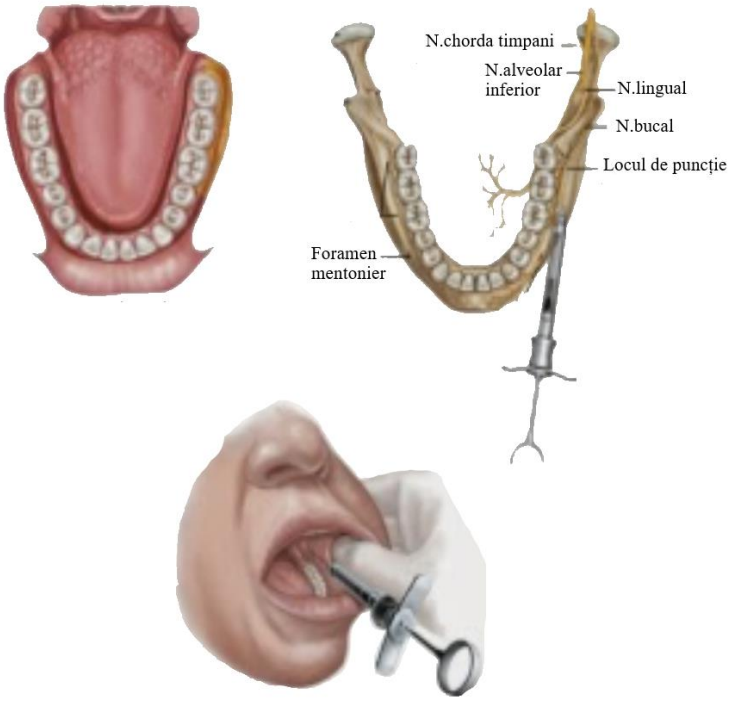


Figure 50. Oral nerve anesthesia (Netter atlas illustration)

a) Peripheral trunk anesthesia.

The peripheral trunk anesthetic ***landmarks*** of the buccal nerve are:

- the anterior margin of the coronoid apophysis;
- the occlusion plane of the upper molars (when the patient has a wide open mouth, the nerve surrounds the anterior margin of the coronoid at the occlusion plane of the upper molars);
- the hole of the Stenon channel (the deep branches cross the buccinator muscle at 1 cm behind and below the Stenon channel);
- the line to join the ear lobe and the labial commissure.

Technique:

1. the patient sits with his mouth wide open;
2. the left thumb of the left hand and the index of the left hand on the right side identify the anterior margin of the coronoid apophysis and apply antiseptic to the puncture site;
3. the puncture is made within the region the occlusion plane of the upper molars meets the anterior edge of the coronoid;
4. the needle has a horizontal direction from front to back and from the outside, and the syringe body is positioned at the oral commissure on the opposite side;
5. cross the mucosa and buccinator muscle, at a depth of 1 cm, and inject 1-2 ml of anesthetic solution.

b) Terminal anesthesia is the technique most commonly used in dentistry and in oral maxillofacial surgery (figure 51).

Landmarks:

- the apex of the second lower premolar;
- the mobile mucosa of the lower oral vestibule, distal to the chin hole.

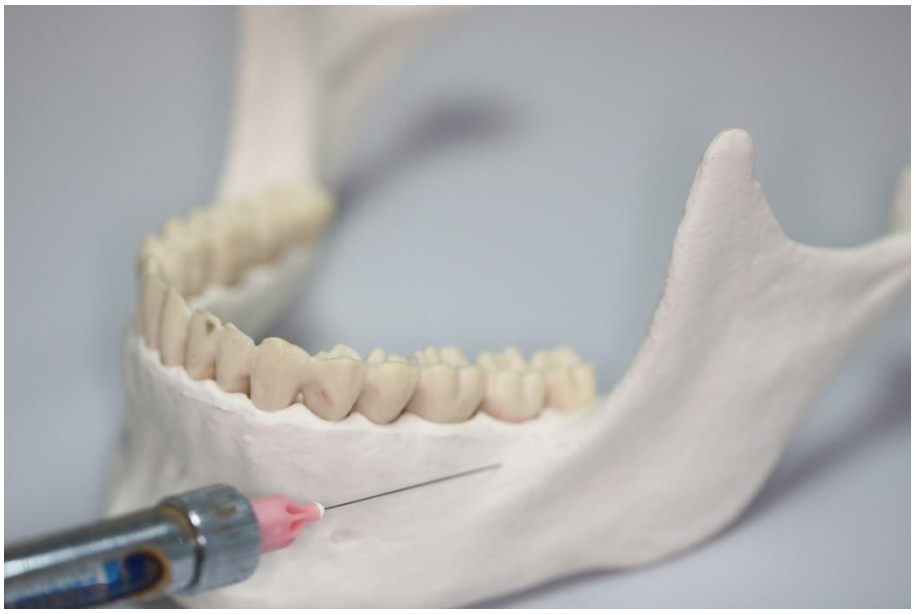


Figure 51. The bucal nerve anesthesia (illustration)

Technique:

1. the patient stands with his head straight and mouth slightly open;
2. between the index and the left hand thumb, the soft labio-jugal parts are removed and the lower buccal vestibule is highlighted;
3. the puncture is made in the mobile mucosa at the apex of the second premolar, with the needle bevel oriented towards the bone plane and parallel to the teeth; it gets posterior to distal direction to the wisdom molar;
4. Subcutaneously, 0.5 ml of anesthetic is injected.

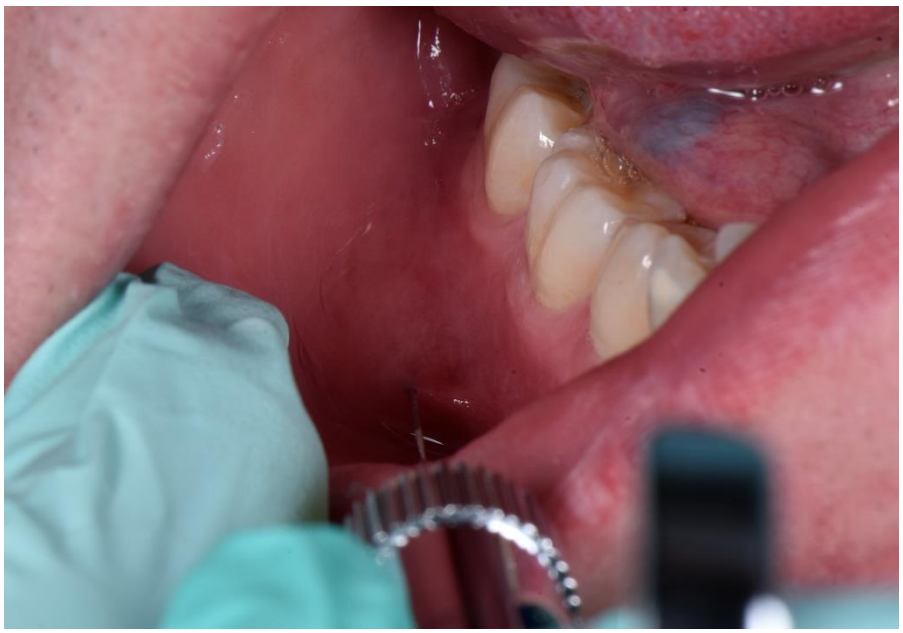


Figure 52. Terminal anesthesia of the buccal nerve

The extraoral method is rarely used. The patient has his head turned on the opposite side of the area to get anesthetized. The tegument is antisepticized with betadine and punctured near the anterior margin of the ascending branch, 2 cm below the zygomatic arch.

The needle has a horizontal direction, backwards and inwards perpendicular to the skin plane. The anesthetic is stored at the bone level. To protect the oral mucosa, it is advisable to insert the index into the oral cavity.

4. Peripheral truncular anesthesia of the mental nerve and incisival nerve

Anesthetized areas (Figure 53):

- inferior frontal teeth to the anesthetized part;
- vestibular fibrous mucosa and anterior alveolar process of the chin hole;
- the lower hemiarch and the tegument in the mental region.

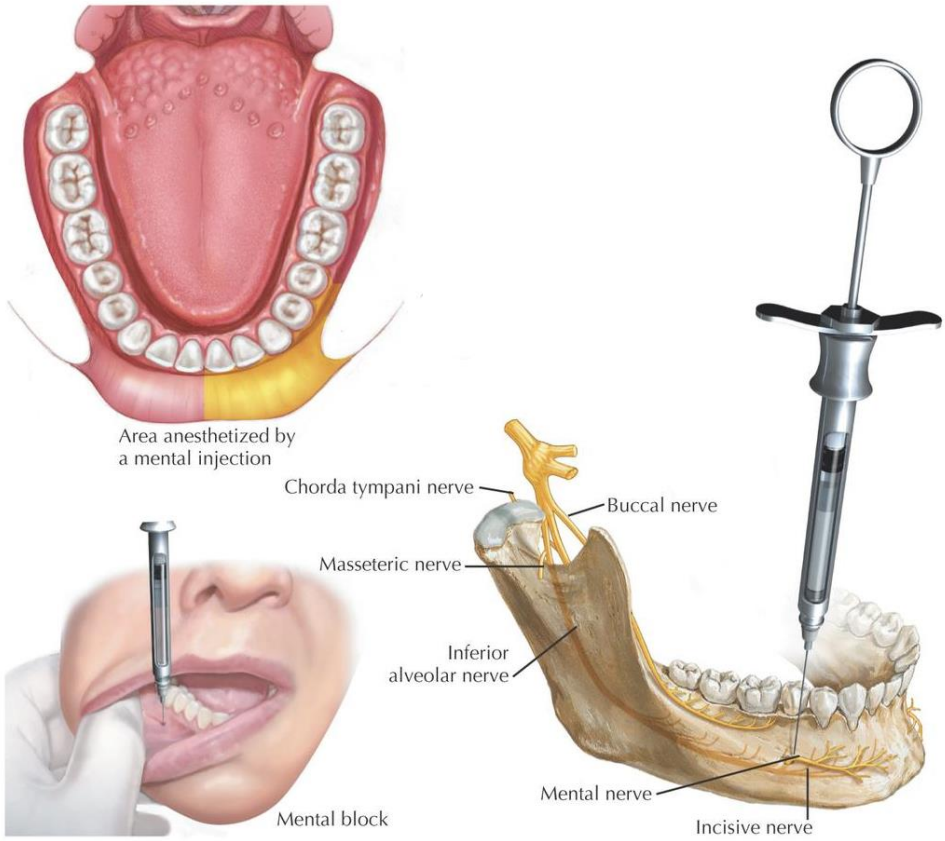


Figure 53. Mental nerve block (*Netter atlas* illustration)

Indications:

- Incisive-canine dental-alveolar interventions;
- Surgeries on the labial-mental soft parts;
- Completing the inferior alveolar nerve block on the opposite side when performing surgical interventions in the anterior region;
- treatment of trigeminal neuralgia, with the trigger area in this territory.

The mental foramem, the main puncture mark, (figure 54) is located on the external surface of the mandible horizontal branch, between the apexes of the two inferior premolars, at an equal distance between the basilar margin and the alveolar one. At the edentate patients, it is closer to the alveolar ridge and sometimes, due to the increased bone resorption, it can reach the ridge under the gingival mucosa.

The mental foramen is located halfway between the midline and the anterior edge of the masseter muscle.

This anesthesia can be practiced intraoral as well as extraoral.

The technique of intra-oral anesthesia

1. the patient is seated with his mouth open;
2. the index finger and the thumb remove the soft labial-jugal parts and highlight the vestibule next to the first molar;
3. the anesthetic puncture is made in the mobile mucosa near the mesial root of the 6 year molar;
4. the needle inserted with the bevel towards the bone has an oblique direction downwards, in and forward to an 15-20º angle with the axis of the second premolar.

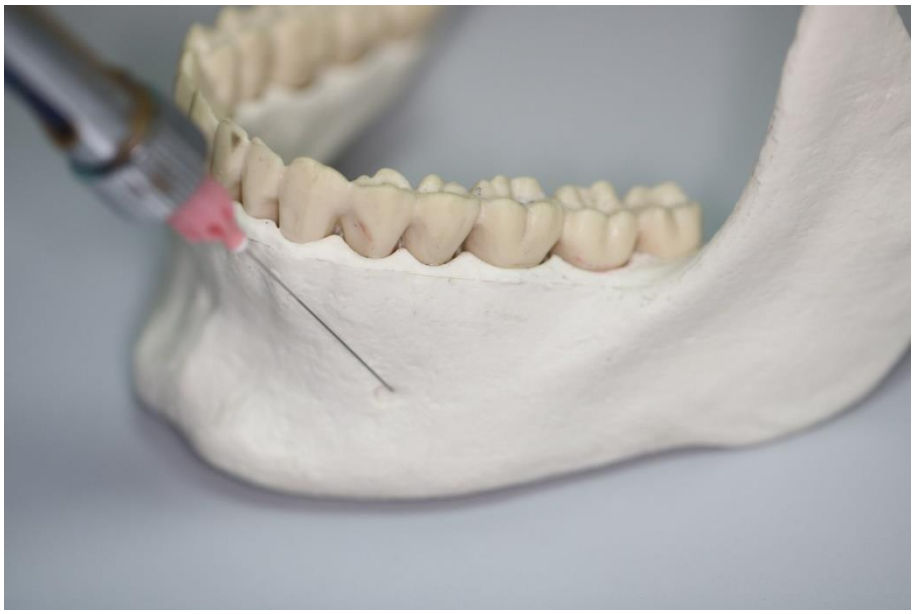


Figure 54. Peripheral truncular anesthesia of the mental and incisive nerve (illustration)

If we want to anesthetize only the mental nerve, it is not necessary to enter the mental foramen; if we want to anesthetize the incisive nerve, intracanalicular penetration of approximately 5mm is required and an amount of 0.5-1ml anesthetic solution is injected. Because the technique is difficult to penetrate intracanalicularly and there is a risk of traumatisation of the mental and incisive nerve, the anesthetic is deposited around the mental hole and diffuses intracanally through skin pressure (Figure 55).

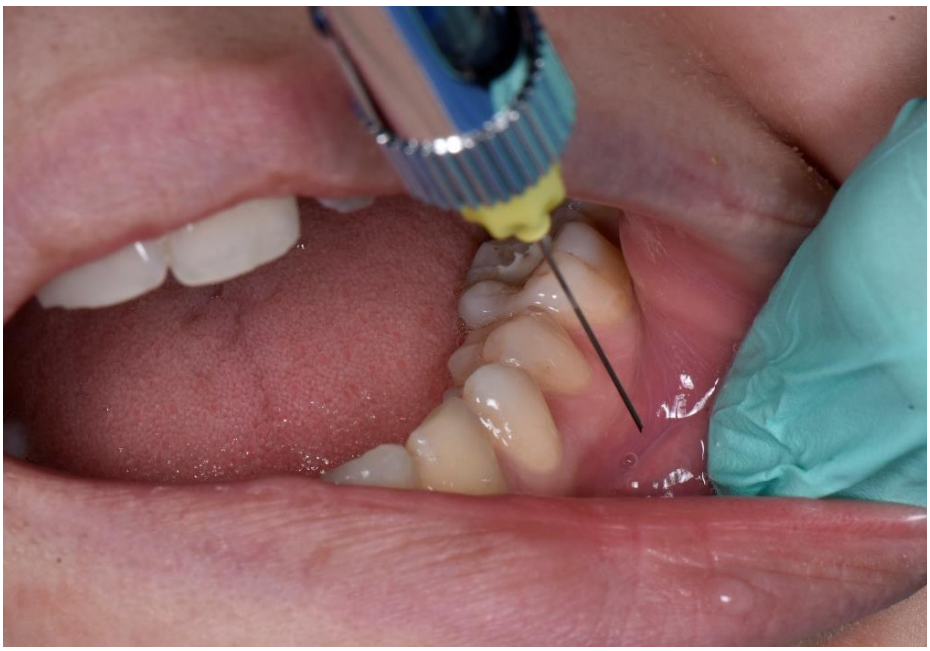


Figure 55. Peripheral truncular anesthesia of the mental and incisive nerve

The extraoral technique of anesthesia:

- the patient is placed in the dental chair with the head slightly extended and the mouth closed;
- the doctor is seated to the right of the patient and slightly to the back, and the left hand holds the chin;
- the skin is antisepticised with iodine and 70º alcohol;
- the puncture is made cutaneous, slightly above and behind the mental foramen, about 1.5-2 cm behind the buccal commissure;
- the needle has an oblique direction downwards, inwards and slightly forward, passing through the soft parts until it reaches the the bone plane;
- it penetrates into the mental foramen 0.5-1 cm and injects slowly the anesthetic sollution.

SIMULTANEOUS ANESTHESIA

Simultaneous anesthesia is the technique to anesthetize several nerves by a single procedure.

❖ ***Veisbrem technique*** is simultaneous anesthesia of the inferior alveolar, lingual and buccal nerves at the mandibular tuberosity level.

The mandibular tuberosity is located on the inner face of the ascending branch, halfway between the sigmoid notch and the Spina spix. It is formed by joining the ridge descending from the internal face of the coronoid with the ridge descending from the mandibular condyle level.

At this level are the buccal nerve, lingual nerve and inferior alveolar nerve have locations coming from anterior to posterior.

The landmarks are the same as those for Spina spix anesthesia, but the puncture site differs.

Technique:

- the patient is seated in the dental chair with his head straight and mouth wide open;
- the temporal ridge of the ascending branch of the mandible is observed with the index of the left hand for the right side and the thumb for the left side;
- the anesthetic puncture is made outside the pterygoid-mandibular raphe, 0.5 cm below the occlusion plane of the upper molars for dentate patients. In edentulous patients, the puncture is made outside the pterygoid-mandibular raphe, 1.5 cm below the maxillary alveolar ridge;
- the needle is perpendicular to the mucosa, and the body of the syringe is positioned towards the buccal commissure on the opposite side, opposite the premolars or inferior molars.
- The needle is inserted at a 1.5 cm depth, until contact with the bone at the mandibular tuberosity level.

Leave anesthetic for the inferior dental nerve and lingual nerve, then remove the needle 5 mm and leave the anesthetic for the buccal nerve.

❖ ***Gaw-Gates technique***

It was described in 1973 and targets the simultaneous anesthesia of the inferior alveolar, lingual, buccal and auriculo-temporal nerves (figure 56).

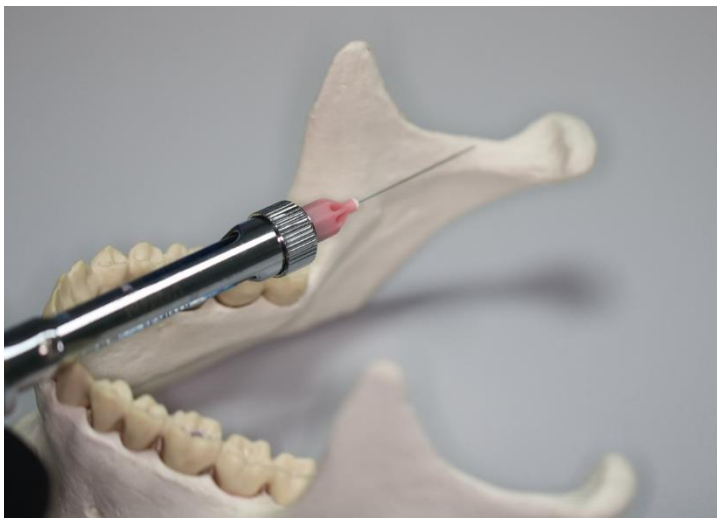


Figure 56. Simultaneous anesthesia of the mandibular nerve branches

Technique: the puncture is made in the jugal mucosa, at the junction of the trachea with the buccal commissure, the line that passes halfway between the pterigoid-mandibular raphe and insertion temporal tendon.

The needle is directed back and forth, and the syringe body is placed opposite the canine or premolar on the opposite side (Figure 57).

It penetrates to a 3-3.5 cm depth, aspiration is performed and the anesthetic is left under the insertion of the external pterygoid muscle. The aspiration is obligatory, because if it penetrates further, the internal maxillary artery can be punctured.

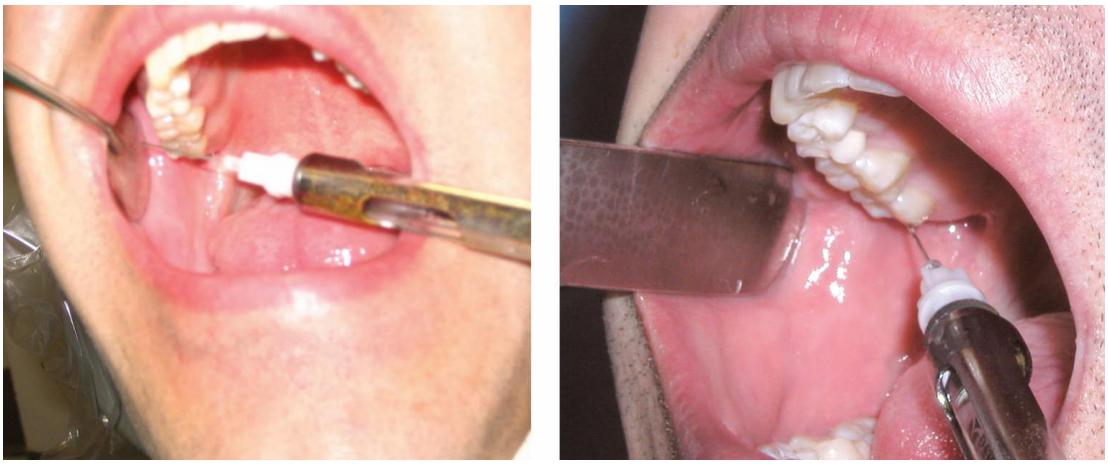


Figure 57. Simultaneous anesthesia of the mandibular nerve branches

Other simultaneous anesthesia techniques have been described:

- ***Ginestet procedure:*** inferior alveolar nerve, lingual nerve, buccal nerve, maseterin nerve
- ***Akinosis process:*** inferior alveolar nerve, lingual nerve, milohioid nerve.

KNOWLEDGE SELF-ASSESSMENT

1. Blockage of nerve conduction determined by local anesthetic:
   a. it will supress transmission of nervous impulse.
   b. causes reversible loss of painful sensitivity in an unlimited area.
   c. causes irreversible loss of painful sensitivity in a limited area.
   d. determines the reversible loss of painful sensitivity in a limited area.
   e. it is easier to interest the smaller diameter nerve fibers.:

2. Painful sensations are caused by free nerve endings of the type fibers:
   a. A-δ (delta) and C
   b. A-γ (gamma) and C
   c. B
   d. C
   e. A-α (alfa)

3. The stimuli that trigger the pain sensation must be:
   a. only mechanic
   b. only thermal or chemical
   c. strong enough to exceed the stimulation threshold
   d. to produce membrane depolarization
   e. to prevent depolarization of the membrane

4. The Gasser lymph node is:
   a. located in Meckel's dugout
   b. also called semicircular
   c. also called semilunar
   d. it is located on the anterior-upper portion of the temporal bone
   e. this engine

5. The trigeminal nerve has:
   a. 3 branches - facial, maxillary, mandibular
   b. 3 types of fibers
   c. 4 cores
   d. origin in the cortex
   e. pure motor function

6. The mandibular branch of the trigeminal is:
   a. strictly sensitive
   b. strictly motor
   c. mixed
   d. partially sensitive
   e. partially motor

7. The first modern local anesthetic, an amide derivative, is represented by:
   a. Mepivacaine
   b. Bupivacaine
   c. Prilocaine
   d. Articaine
   e. Lidocaine

8. The duration of the anesthetic

action depends on:

a. the ability of the substance to bind to proteins.
b. concentration of cationic forms around the axon.
c. the diffusibility of the anesthetic.
d. injection rate.
e. elimination rate of anesthetic.

9. Lidocaine:

a. chemically, it is an acetamide.
b. has a pH of about 4.5.
c. it is sold as aqueous, isotonic, sterile, apyrogenic solutions containing anesthetic agent.
d. is marketed with or without adrenaline
e. does not contain preservatives that can induce allergic reactions.

10. They will be monitored permanently after anesthesia with lidocaine:

a. heart rhythm.
b. respiratory rate.
c. the patient's state of consciousness.
d. the patient's blood sugar.
e. the body temperature of the patient.

11. Regarding the injection of the local anesthetic substance:

a. is attended by aspiration.
b. it is not attended by aspiration.
c. aspiration is done after injection.
d. aspiration should be performed prior to injection.
f. the injection will be performed as soon as possible.

11. Regarding the injection of the local anesthetic substance:

a. is attended by aspiration.
b. it is not attended by aspiration.
c. aspiration is done after injection.
d. aspiration should be performed prior to injection.
e. the injection will be performed as soon as possible.

12. Local amide anesthetics should be used with caution in patients with:

a. rheumatic disorders
b. femural neck fracture
c. severe liver disease
d. urinary lithiasis
e. prostate adenoma

13. The maximum dose of articainee in a normoponderal patient, without general conditions, in a session is:

a. 5 ml
b. 6 ml
c. 8 ml
d. 10 ml
e. 12.5 ml

14. Simultaneous injection of articainee with beta blockers leads to:

a. lower blood pressure
b. skin reactions

c. maintaining blood pressure
d. blood pressure rise
e. digestive disorders

15. What is the role of the vasoconstrictor associated with local anesthetics?

a. provides a slow resorption of the anesthetic in circulation
b. increases the potency and duration of local anesthetic
c. the potency and duration of the local anesthetic decreases
d. increases the risk of systemic toxicity
e. has no role.

16. What are the basic rules in local infiltration anesthesia?

a. contact anesthesia is applied at the puncture site
b. a firm hand contact is established
c. the syringe hand always rests on the patient, and with the other the tissues are put in tension
d. no anesthetics with vasoconstrictor are used
e. anesthesia should last at least 2 hours

17. Infiltration anesthetics are the following:

a. supraperiostal paraapical
b. intraligamentary
c. by spraying
d. intravenous
e. peripheral trunk

18. Nasopalatine nerve anesthesia (puncture at the incisor hole):

a. it is not painful, due to the precarious innervation of the incisive papilla
b. it is painful due to the adhesion of the fibrous mucous membrane of the palate
c. painful due to the lack of connective tissue at this level
d. also involves performing contact anesthesia
e. presents the injection of 1.7 ml anesthetic solution

19. Spix spine puncture markers are:

a. medial and posterior temporal ridges from the anterior margin of the mandibular branch
b. pterigoid mandibular raphe located along the anterior edge of the external pterygoid muscle
c. occlusion plane of the lower molars
d. pterigomandibular raphe located along the posterior edge of the internal pterygoid muscle
e. occlusion plan of the upper molars

20. In the hypertensive crisis that appeared as an accident of the local

regional anesthesia, the emergency treatment consists of the administration of:

a. factor VIII;
b. sublingual nitroglycerin;
c. aspirin
d. hydrocortisone hemisuccinate;
e. adrenaline injectable.

1. Patient X addressed the dentist with the following symptoms: 3.8. tooth pain. Following Clinical and paraclinical examination, a severe cavity deacy was detected. As a treatment alternative was proposed the dental extraction. What type of anesthesia is required for this surgery? Describe the landmarks, technique and give arguments to support your choice.
2. Patient Y will be recommended prosthetic treatment by undertaking a total prosthesis. As a preprotective treatment, it is necessary to extract the roots of the teeth 1.2, 2.4, 4.1, 4.2. Specify the type and technique of the of anesthesia to be performed.

Answer Key

1. ADE
2. A
3. CD
4. AC
5. BC
6. C
7. E
8. ABCE
9. ACD
10. ABC
11. AD
12. C
13. E
14. C
15. AB
16. ABC
17. ABDE
18 BCD
19. ACE
20. B

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