Pain Control in Endodontics

Pain:

It is an unpleasant and distressful body sensation which could result from injury or disease.

Noxious stimuli in teeth are transmitted in primary afferent "nociceptive" (pain-detecting) sensory neurons located in the trigeminal ganglion, and their nerve axons terminate in the dental pulp. Regardless of the nature of the sensory stimulus (i.e., thermal, mechanical, chemical, or electric), almost all afferent impulses generated from pulp tissue result in the sensation of pain.

The tooth is innervated by a large number of myelinated (mainly Aδ (delta)) and unmyelinated (C fibers) sensory axons. Both fiber types are nociceptors. C fibers in the dental pulp are generally 3 times higher in numbers than A fibers.

Aδ fibers are myelinated and larger in diameter, principally concentrated near the pulp-dentin complex, and they can transmit a sharp, instant, and intermittent pain feeling.

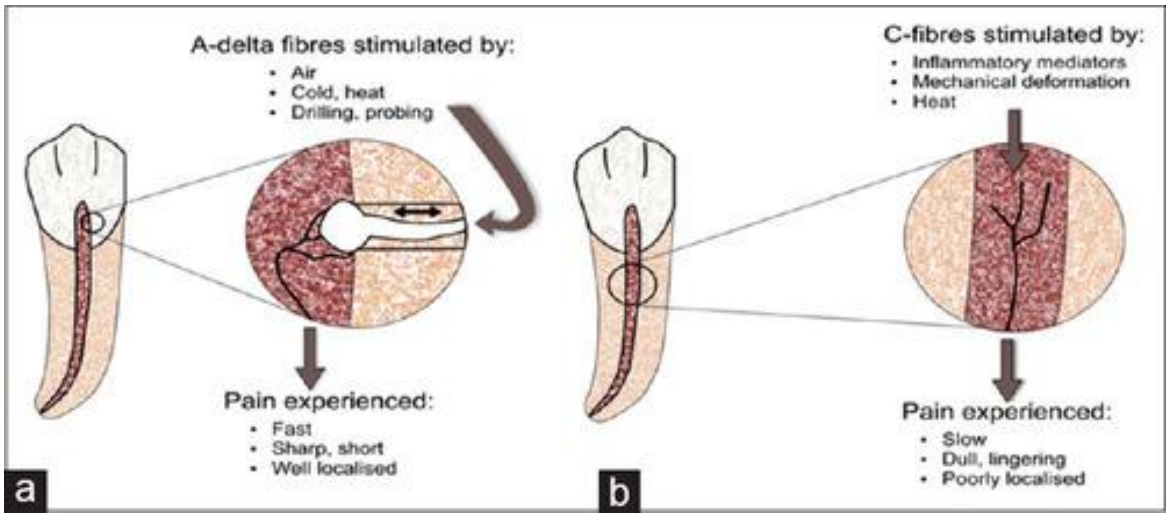
C-fibers are unmyelinated, smaller in diameter, and distributed within the pulp with a higher concentration near the pulp-dentin complex. Pain transmitted by these fibers is characterized by a burning and aching sensation.

Table 1: Excitation of A-δ and C-fibers to stimulus and intrapulpal conditions

Stimulus and intrapulpal conditions	*A-δ fibers response*	*C-fibers response*
Cold	√	×
Heat	√	√
Ion effect	√	×
EPT	√	×
Hypoxia	×	√
↑Pulpal pressure	×	√
Hyperosmotic solutions	√	×
Inflammatory mediators	×	√

In addition, these fibers can be stimulated during inflammation to release neurotransmitters such as substance P and calcitonin gene-related peptide (CGRP). These neurotransmitters can cause vasodilatation, which increases the pulp volume, and this can increase the pressure on the nerve ending, intensifying the pain sensation. This process is called neurogenic inflammation.

The pain reaction threshold (PRT) is the point at which a person can feel pain. With endodontic therapy, hyperresponse to stimulation is significantly increased.



Factors that lower the PRT include:

1. Presence of pain at the beginning of treatment.
2. Fatigue.
3. Fear and anxiety.

By increasing pain sensation, the blood level of catecholamine suddenly elevates with an increase in blood pressure and heart rate. This might induce fainting, angina pectoris, asthma, and psychiatric reactions. To reduce the possibility of such conditions happening, anesthesia has to be introduced slowly and in a supine position.

Correct diagnosis of the etiological factors of pain and proper treatment of the problem relieves pain in many cases.

Differential diagnosis of dental pain

Pain in the facial region may be of different origins as:

1. Dental: This type is due to reversible pulpitis, irreversible pulpitis, and symptomatic apical infection.
2. Musculoskeletal: As temporomandibular joint disease.
3. Neuropathic: Pain may be due to trigeminal neuralgia or herpes infection.
4. Neurovascular conditions: As migraine.
5. Inflammatory conditions, Such as sinusitis.
6. Systemic disorders: As cardiac pain.
7. Psychogenic.

Local anesthesia

It is the temporary loss of sensation or pain in a certain part of the body produced by a topically applied or injected agent without depressing the level of consciousness. Prevention of pain during dental procedures eliminates fear and anxiety.

Knowledge of the anatomy prevents problems during anesthetic injection, such as muscle trismus, hematoma, and intravascular injection.

There are 2 general types of local anesthetic chemical formulations:

1. Esters such as procaine, benzocaine.
2. Amides such as lidocaine, mepivacaine, prilocaine, and articaine.

Mechanism of action:

The primary action of the local anesthetic agent is to decrease the nerve permeability to sodium (Na^+) ions, thus preventing the inflow of Na^+ ions into the nerve. This interferes with sodium conductance and inhibits the propagation of impulses along the nerve fibers. Movement of Na^+ ions is permitted through Na channels, which are called voltage-gated sodium channels (VGSCs). Therefore, the local anaesthesia molecules block VGNCs on the nerve membrane and prevent the generation and conduction of nerve action potential.

Local anesthetics are vasodilators, absorbed in the circulation, and have a systemic effect directly to the blood plasma level. Vasoconstrictors in the

local anesthetic constrict the blood vessels to lower the local absorption into the bloodstream to prolong the anesthetic effect in the working area and decrease the possibility of toxicity. It is also used to decrease bleeding by infiltration of a few drops in the bleeding area.
The condition of patients with hyperthyroidism, cardiovascular disease, diabetes, and having drugs such as tricyclic antidepressants and MAO inhibitors requires a consultation with the physician before injection with a local anesthetic having vasoconstrictor.
If the local anesthetic is injected in an infected area, its onset will be delayed. The inflammatory process in an area of infection lowers the pH of the extracellular tissue from its normal value to 5-6 or lower. This low pH inhibits anesthetic action because little of the free base form of the anesthetic is allowed to cross into the nerve sheath to prevent conduction of nerve impulses. In addition, inserting a needle into an active site of infection may spread the infection. Therefore, alternative nerve block injections can be used to prevent this complication.
Traditional methods of confirming anesthesia usually involve questioning the patient, probing the soft tissue, and/or starting treatment and waiting for the patient's reaction. These approaches may not be effective for determining pulpal anesthesia. The effective way to detect the onset of pulpal anesthesia is by doing pulp testing. The negative response means positive pulpal anesthesia.

Topical anesthetic

It is effective to minimize the surface discomfort of the injection of the needle (2-3 mm in depth). This anesthetic is composed of benzocaine (up to 20%) or lidocaine as a solution or ointment (5%) or spray (up to 10%).





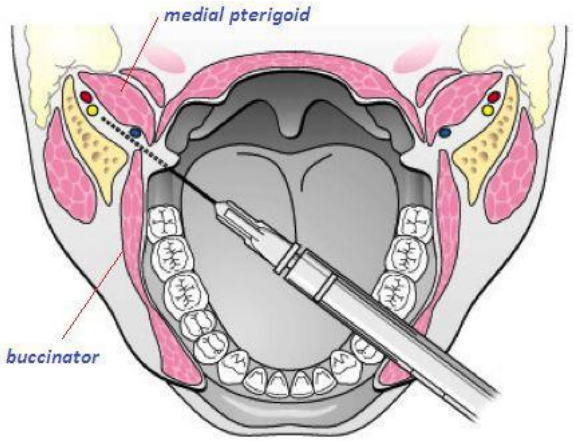
Techniques for mandibular anesthesia

1. Inferior alveolar nerve block.

The site of deposition is near the mandibular foramen before the entry of the inferior alveolar nerve. It anesthetizes the mandibular teeth on the side of injection. The lingual nerve can also be anesthetized in the same injection with its innervated regions (lingual mucosa and tongue). However, the buccal mucosa cannot be anesthetized.

Administration of a conventional IAN block to asymptomatic patients using 1.8 mL of 2% lidocaine with 1:100,000 epinephrine would provide pulpal anesthesia within 15 minutes that lasts for 1 hour.

Research also found that administration of a two-cartridge volume was significantly better for avoiding a missed block than a one-cartridge volume in asymptomatic subjects.



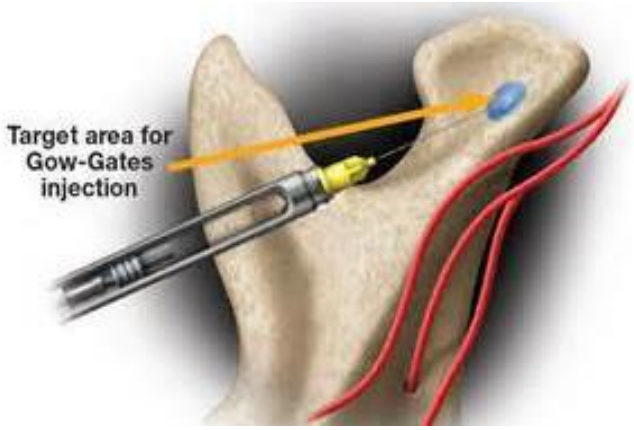


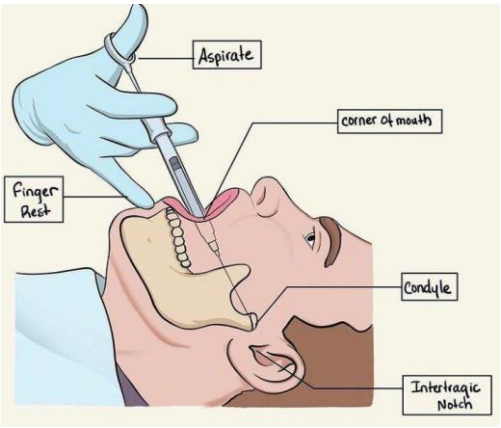
2. Mandibular nerve block.

It is a V3 (mandibular) nerve block anesthetizing all the mandibular teeth in the region with the buccal and lingual soft tissues. It also provides sensory anesthesia of the buccal and mylohyoid nerves. In most of the cases V3 block should be combined with the inferior alveolar nerve block to ensure pulpal anasthesis.

There are two injection techniques for the (V3) nerve block;

A. The Gow-Gates mandibular nerve block:
can be performed by laying the patient in a supine position and opening the mouth wide. The dentist aims to administer local anesthetic just anterior to the neck of the condyle in proximity to the mandibular branch of the trigeminal nerve after its exit from the foramen ovale.





B. The Akinosi-Vazirani nerve block:
The site is the height of the mucogingival junction of the maxillary third molar near the maxillary tuberosity. This is used where there is limited mouth opening.





3. **The Incisive nerve block:**
   The site is buccally between the mandibular two premolars. It provides anesthesia to the lower lip, buccal mucosa, and skin of the chin ventral to the mental foramen, in addition to premolars and anterior teeth in the region.

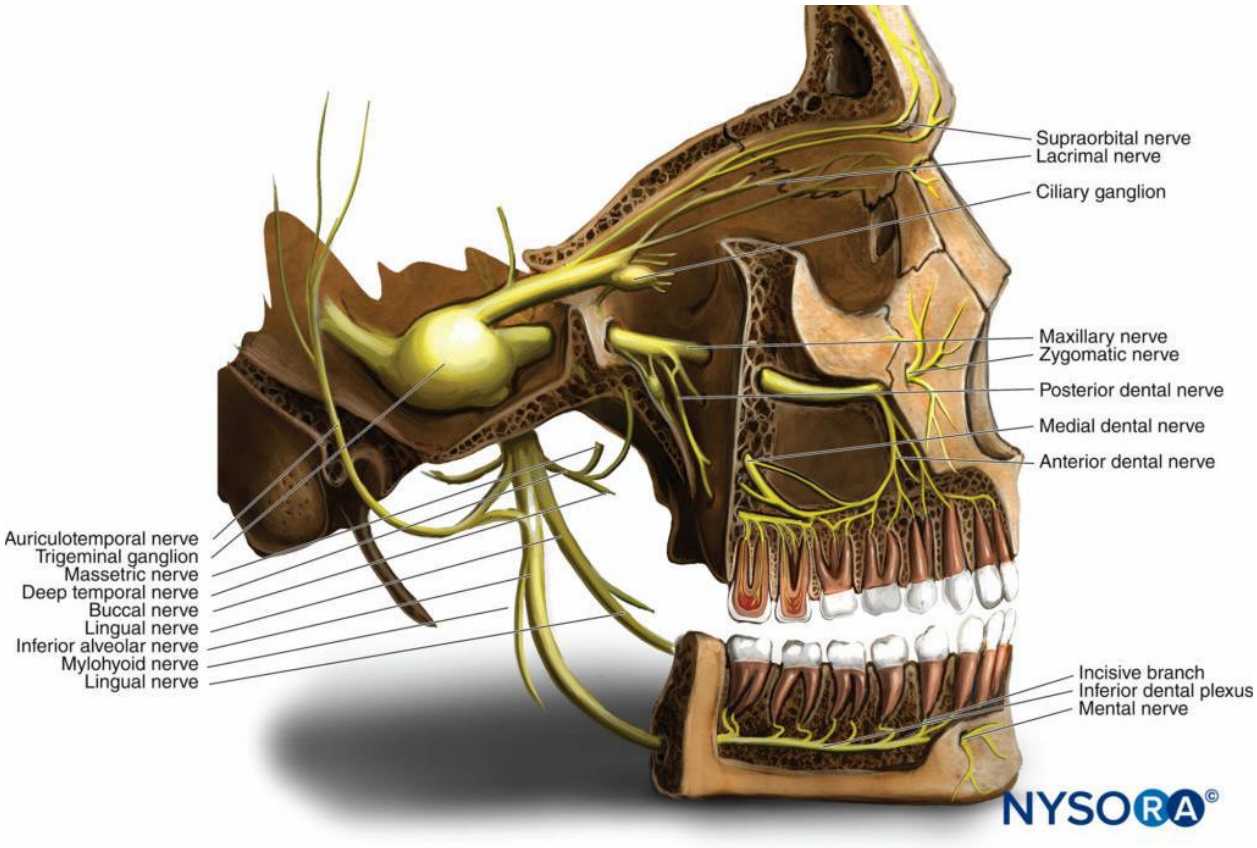




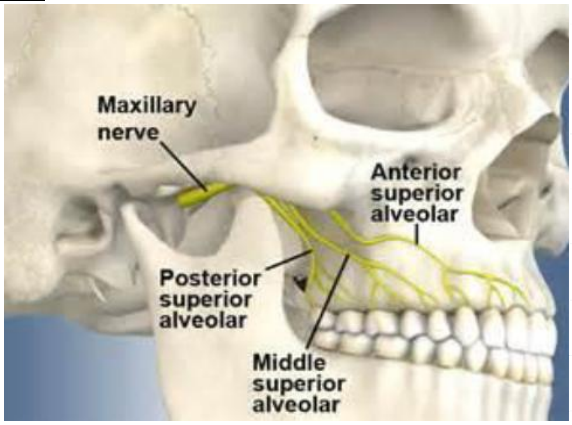
Techniques for maxillary anesthesia

The most commonly used injection for anesthetizing maxillary teeth is infiltration with a cartridge of 2% lidocaine with 1:100,000 epinephrine. Infiltration results in a fairly high incidence of successful pulpal anesthesia (around 87% to 92%). Pulpal anesthesia usually occurs within 3 to 5 minutes. However, the duration of pulpal anesthesia is a problem with maxillary infiltrations. Pulpal anesthesia of the anterior teeth may last for about 30 minutes, while in the premolar region, until about 40 to 45 minutes, and then it starts to decline. Therefore, additional local anesthetic should be administered depending on the duration of the procedure and the tooth group affected.

Block anaesthesia for the maxillary teeth, including posterior superior alveolar, anterior middle superior alveolar, palatine-anterior superior alveolar, infraorbital, and second division nerve blocks. None of these are required for routine restorative and endodontic procedures.



1. <u>The posterior superior alveolar nerve block:</u>
   The site is in the buccal fold of the maxillary 2nd molar. It anesthetizes the maxillary molars and buccal soft tissues.
2. <u>The middle superior alveolar nerve block:</u>
   It anesthetizes the 2nd maxillary premolars, and the site of injection is in the buccal fold of the premolars.



3. <u>The anterior superior alveolar nerve block:</u>
   The site is the buccal fold of the first maxillary premolar and is aimed at the infraorbital foramen. The areas anesthetized are the anterior teeth and premolars with overlying soft tissues.

Supplemental injection techniques

1. Periodontal ligament (PDL) injection:

   This technique is used when no other technique can be used. It can induce local anesthesia in either maxillary or mandibular teeth. The needle is inserted between the tooth and PDL with the bevel of the needle toward the root. An anesthetic solution of 0.2 ml is placed per root. The onset of anesthesia is immediate, but the duration is variable. This technique required high injection pressure to force the local anesthetic solution through the PDL into the cancellous medullary bone surrounding a tooth. Therefore, high-pressure dental syringes such as the Peripress Pen (Panadent, Kent, England) and Ligmaject (Henke-Sass, Wolf, Tuttlingen, Germany) can be used. In addition, a special motor-driven anesthetic device, such as STA (single tooth anesthesia, Milestone Scientific, USA), has also been developed for painless PDL anesthesia.



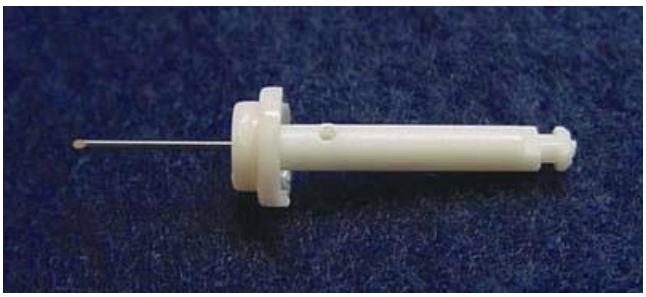


2. Intraosseous anesthesia:

   It is particularly useful in conjunction with an inferior dental block when it is likely that supplemental anesthesia will be necessary (e.g., in mandibular second molar teeth).

   The Stabident IO system (Fairfax Dental Inc., Miami, FL) utilizes a slow-speed, handpiece-driven perforator and a solid 27-gauge beveled wire that drills a small hole through the cortical plate. The anesthetic

solution is delivered into cancellous bone through a 27-gauge ultrashort needle placed into the perforation made by the perforator.





3. Intrapulpal anesthesia:
   When full anesthesia is not gained by other techniques, the intrapulpal approach is used, especially in cases of irreversible pulpitis. The needle is inserted directly into the pulp, and LA is introduced with force. This injection controls pain by both applying pressure and utilizing the pharmacologic action of a local anesthetic agent. A profound and rapid onset of anesthesia can be obtained.
   The major drawback of the IP injection technique is that the needle and injection are made directly into vital and very responsive pulp tissue; this injection is quite often moderately to severely painful.
   Another disadvantage of the technique is the duration of pulpal anesthesia (15 to 20 minutes). The pulpal tissue must be removed quickly and completely to prevent a recurrence of pain during instrumentation.



Dental and referred pain:

Most of the oral and dental pain can be traced to its source. There are cases where pain might be experienced away from its source on the same side but in the other jaw, ears, eyes, and sinus. Careful diagnosis reveals the affected tooth or related anatomic structure (in non-dental pain).