

Complications of Local Anesthesia

24-26

**Local
Complications**



**Systemic
Complications**

Complications of local anesthesia

The widespread use of local anesthesia in dental practice is in itself attributed to both the effectiveness and safety of the method. Nevertheless, complications occasionally occur and it is essential that the dental surgeon should know how to minimize its incidence.

The complications associated with the administration of local anesthesia classified into **local** and **systemic**.

Local complications of the local anesthesia:

- 1. Pain on injection*
- 2. Burning on injection*
- 3. Failure to obtain anesthesia*
- 4. Persistent anaesthesia*
- 5. Needle breakage*
- 6. Facial nerve paralysis*
- 7. Trismus*
- 8. Soft tissue injury*
- 9. Hematoma*
- 10. Infection*
- 11. Ocular Complications*
- 12. Oro-Facial blanching :*



1. Pain on injection:

Pain on injection increases patient anxiety and may lead to sudden unexpected movement, increasing the risk of needle breakage.

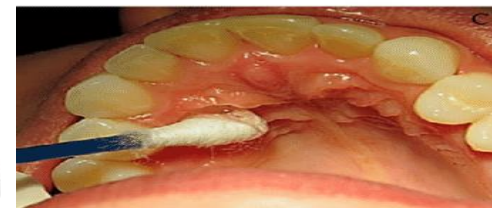
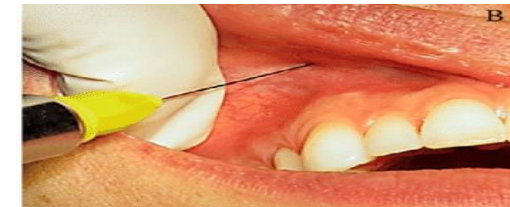
Causes:

- 1) Careless injection technique.
- 2) Rapid deposition of the solution may cause tissue damage and pain.
- 3) Low PH of the solution could irritate the tissue.
- 4) The temperature of the solution: a warmer solution is more comfortable for the patient than the cold one.
- 5) Aggressive insertion of the needle can tear the soft tissue, blood vessel, nerve and periosteum, causing more pain.



Prevention of the pain during injection could be achieved by avoiding the causes

1- Use topical anesthetic application, **2-**warming anesthetics to body temperature, **3-**using a smaller-gauge needle (27 gauge), **4-**switching to a fresh needle when you have to inject multiple times in the same lesion or when you have multiple injections sites, and **5-**injecting slowly and with low pressure which reduces pain are done. A rate of 30 seconds per mL of solution is recommended.



2. Burning on injection:

A burning sensation occurring during injection of local anesthesia is common. **The burning** is dependent on the **rate of injection** and **the acidity of the solution**. **Lidocaine** causes an intense burning sensation when injected locally. When the needle penetrates a nerve, the patient may also feel a sudden “**electric**” shock, suddenly moving the head, with the **risk of self-inflicted damage**

Causes:

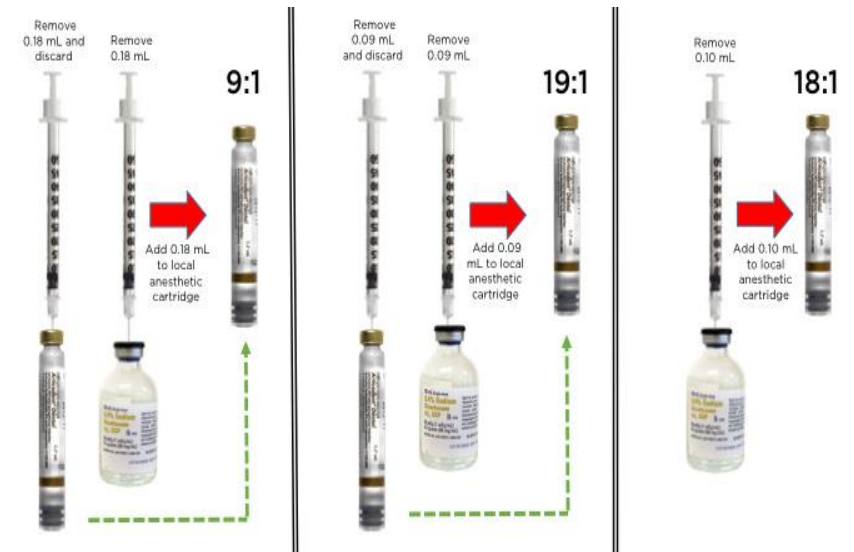
- 1) Low PH of the solution.
- 2) Rapid injection of local anesthesia, especially in the denser more adherent tissues of the palate.
- 3) Contamination of the local anesthetic cartridge that can result when they are stored in alcohol or other sterilizing solutions (diffusion of these solutions into the cartridge).

Management: Because most instances of burning on injection are transient and do not lead to prolonged tissue involvement no treatment is indicated.

prevention:

1-A smaller diameter of needle and warming the injection solution can reduce pain

The choice of needle diameter can influence the pain felt by the patient. A well-designed randomized controlled trial (RCT) showed an inverse relation between needle diameter and pain. A smaller needle also forces the injector to slow down, minimizing pain from volume expansion.



A longer needle (e.g., 1 1/4" v. 5/8") is useful for injecting larger areas. In addition, warming the solution results in a less painful injection.

2-Buffering lidocaine with sodium bicarbonate can minimize the typical burning sensation

With an acidic pH of 4.7, lidocaine can cause an unpleasant burning sensation. A Cochrane meta-analysis of several RCTs determined that the addition of sodium bicarbonate (10:1 lidocaine: sodium bicarbonate [8.4% NaHCO₃]) can considerably minimize pain.³ When buffering the anesthetic, it is worth noting that syringes can hold a higher volume than indicated (e.g., a 10-millilitre syringe can actually hold 11 millilitres).

3. Failure to obtain anesthesia:

Although the incidence of this difficulty tends to decrease as the experience of the operator increases it is still probably the most common problem seen during the use of local anesthesia. The problem is most common with block anesthesia, especially in the lower jaw.

Causes:

1) Poor technique: it is the most common cause of insufficient anesthesia in inferior alveolar nerve block and common mistakes are:

- Injection of anesthesia too soon on the anterior ascending ramus.
- Giving the solution inferior to the mandibular foramen.

2) Anatomical causes:

- Accessory nerve supply
- Abnormal course of the nerve
- Variation in the foramen location
- Sometimes the tooth is innervated by more than one nerve



3) Pathological causes:

- Trismus (limited mouth opening): in these cases, it is difficult to use the conventional technique of inferior nerve block.
- Infection and inflammation: if the pulp is inflamed the low PH may cause lack of effective anesthesia in that area. The inflammation makes the nerve more sensitive to pain; minimal stimulation can cause pain perception. In those patients, to obtain proper anesthesia more solution has to be injected, for example, by combining a block, infiltration and supplemental Intraligamentary injections.

4) Psychological causes:

Fear and anxiety can cause failure in local anesthesia, to enable successful anesthesia relaxation of the patient is sometimes needed. The use of a sedative like benzodiazepine may be helpful.

4. Persistent anesthesia or Paraesthesia, which can be defined as altered sensation **beyond the expected duration of anesthesia or it is the prolonged loss of sensation**. It is common in dental practice.

When anesthesia persists for days, weeks, or months, there is an increased potential for **self-inflicted injury**, **biting**, **thermal** or **chemical trauma** which can occur without **the patient awareness**.

Causes:

- 1) Trauma to any nerve during injection may lead to paresthesia. Patients report the sensation of an electric shock throughout the distribution of the involved nerve.
- 2) Injection of local anesthetic solution contaminated by a neurotoxic substance such as alcohol near a nerve.
- 3) Hemorrhage and infection in close proximity to a nerve may lead to transient paresthesia, due to pressure on the nerve, which resolves when the cause is removed. .



Prevention: Strict adherence to injection protocol and proper care and handling of dental cartridges help to minimize the risk of paresthesia

Management: Most paresthesia resolves within approximately 8 weeks without treatment. Reassure the patient that the condition is transient with strict follow up. **If the damage to the nerve is severe, the paresthesia will be permanent**

The severity of oral paraesthesia is related to the length of the altered sensations; despite in most of the cases, a nerve, affected abnormally by local anaesthetic, spontaneously recovers in an **8-week period** and in some cases, this adverse event could be **prolonged and persist for 6–18 months** or **even make the nerve unable to fully recover**.

Nerve injury was reported especially after **mandibular block analgesia**, and lingual nerve was affected in **64–79%** of all reported cases

A direct trauma is a first possible explanation about association **between paraesthesia and local anaesthesia**.

When mandibular block analgesia was performed, the **patient's mouth is wide open**; this could stretch the lingual nerve and deflect the needle used for the anaesthetic administration.

Therefore, the needle can penetrate the **nerve sheath** and **consequently could cause**

- (1) **direct damage** of nerve fibres; or
- (2) **damage of small blood** vessels located within the nerve, leading to intraneural haemorrhage; or
- (3) **damage of connective** tissues within the nerve, **producing oedema** within the nerve sheath.

All 3 events can lead to paraesthesia, in a transient or permanent way

However, this phenomenon does not completely explain the association between **oral paraesthesia** and local **anaesthetic administration**, especially **articaine** and *prilocaine*.

Indeed, a toxic effect of local anaesthetics may be related with increased risk of paraesthesia. Notably, articaine and prilocaine are available in dental cartridges as solutions in concentrations of 3–4% (articaine 4%, prilocaine 3–4%).

These concentrations are the **highest among injectable local anaesthetics** marketed for dentistry practice (e.g. **lidocaine used in dentistry is concentrated 2%**).

The **local anaesthetic concentration seems to play an important role for nerve injury**:, Researches by performing a **histopathological analysis**, this work **found a tissue damage with articaine 4% and not with articaine 2%**

This mechanism could explain the **high rate of oral paraesthesia**. Around the area of mandibular block injection, the **lingual nerve typically has fewer fascicles than the inferior alveolar nerve and may be unifascicular in about a third of patients** .

Therefore, the frequent occurrence of lingual nerve paraesthesia may be related to its **fascicular pattern**.

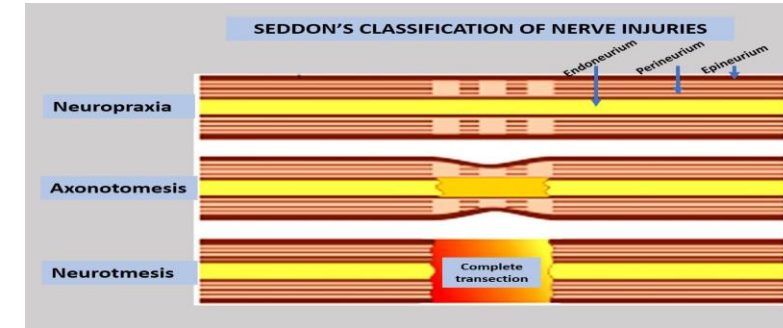
Clinicians, especially **dentists**, should consider risks and complications of **articaine** or **prilocaine** and the concentrations of the drug in the choice of local anaesthetic for their patients. Moreover, they should pay attention to any symptoms suggesting a possible **paraesthesia** after **anaesthetic administration**, and they should report this **adverse event to their pharmacovigilance service**

Classification of Nerve Injuries

Neurapraxia

Neurapraxia is seen as **motor paralysis**, and it is the **mildest injury type that is transient**. There is no effect on **nerve continuity**. The transient nature of this injury is believed to be caused by a **temporary disturbance in the conduction pathway that blocks neural transmission but does not damage the axon**. Symptoms include motor paralysis (for motor nerves), numbness, tingling, and loss of vibration and postural sensation.

All of these effects resemble the **common effects of local anesthesia**



Axonotmesis

Axonotmesis occurs when there is complete interruption of the nerve fibers, but the connective tissues (endoneurium, perineurium, and epineurium) remain intact. It is a disturbance of nerve cell axon, with Wallerian degeneration occurring near the site of injury. This type of nerve injury is caused by a crush or pressure damage. Spontaneous regeneration is likely to occur following this type of injury. The nerve as a mass is still in continuity

Neurotmesis

Neurotmesis involves complete severance of the nerve. Functional loss is complete and recovery without surgical intervention is unlikely. **There is a complete loss of motor and sensory function.**

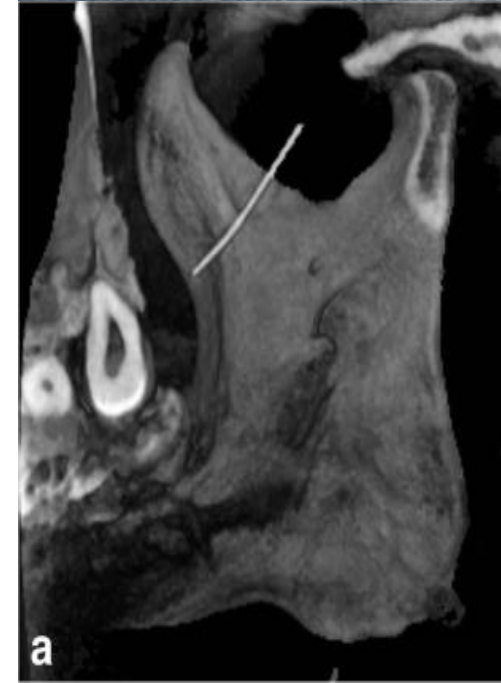
If there is recovery, it is **usually incomplete**. It is important to note that clinically, there may **no difference between axonotmesis and neurotmesis**. Discerning the **differences between the two entities includes:**

5. Needle breakage:

Breakage and retention of needles within the tissue have become an extremely rare occurrence because of the introduction of disposable needles. However, reports of needle breakage still appear.

Causes:

- 1) The primary cause of needle breakage is weakening of the dental needle by bending it before its insertion into the patient's mouth.
- 2) The sudden unexpected movement of the patient.
- 3) Smaller needles (such as gauge 30) are far more likely to break than larger needles (such as gauge 25).
- 4) Re-use of the needle (repeated injection cause fatigue of the needle structure and increases the risk of needle breakage).
- 5) Incorrect use of the needle:
 - Aggressive insertion of the needle into the tissue.
 - A sudden change in the direction inside the tissue.
 - Too deep penetration as the needle goes up to its hub inside the tissue and might fracture at this point. The hub is considered the most common point of needle fracture.
- 6) Needles may be defective in manufacture.



Prevention:

- The dentist should check the needles before using them. If there is any suspicion of inadequate product quality a new one should be used.
- Use larger gauge and long needle for techniques needing penetration of significant depths of soft tissue, gauge 25 is appropriate for an inferior alveolar nerve block.
- Do not redirect a needle once it is inserted into the tissue.

Management:

- Stay calm and try to localize broken part in the tissue.
- Tell the patient what has happened and try to relax and comfort him.
- Stabilize the patient's jaws in order that the needle stays in place, if the patient moves his jaw the tension from the muscle of the masticatory system help the needle to penetrate the tissues.
- If a portion of the needle is visible, grasp it firmly with a hemostat and remove it.
- If you cannot remove the broken part by yourself, refer the patient to an oral and maxillofacial surgeon.

6. Facial nerve paralysis:

The facial nerve is the 7th cranial nerve and it emerges from the skull via the **stylomastoid foramen**.

Before the facial nerve **emerges from the skull**, the **chorda tympani branches off from it at the level of the petrous bone**.

It supplies **preganglionic secretomotor fibers** to the submandibular as well as sublingual salivary glands and **carries efferent taste fibers from the front two thirds of the tongue with the exception of the lingual papillae**.

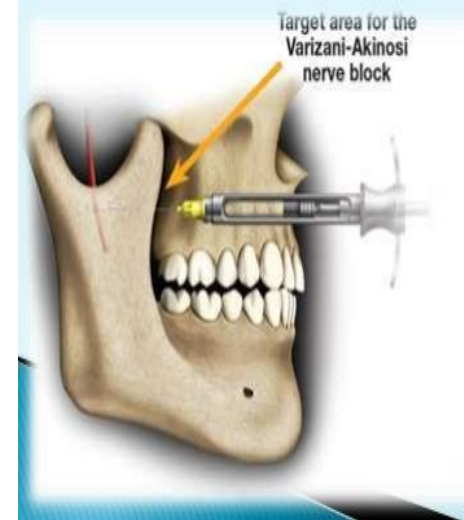
After emerging out of the skull, the facial nerve **divides into 2 main branches** – the **temporal** and **cervical** – before it enters the **parotid gland**, where it continues to **divide into the temporal, zygomatic, buccal, mandibular and cervical branches**, which eventually supply the **mimic muscles** .

It is very rare neurological complication after routine dental procedures under local anesthesia is the loss of function of the facial nerve (facial palsy) .

Paralysis of the facial muscles on one side is an uncommon complication of the inferior alveolar nerve block and may be either partial or complete depending upon which branches of the nerve are affected.

Cause: This complication arises if the tip of the needle is inserted too far back and behind the ascending ramus. The solution is then deposited in the substance of the parotid gland, where it anesthetizes the branches of facial nerve causing paralysis of the muscle they supply. Since a fascial sheath envelops the parotid gland there is also a failure in anesthesia of the inferior alveolar nerve.

Clinically the patient will immediately complain of transient paralysis of the muscles of the chin, lower lip, upper lip, eyelid (inability to close the eye) and inability to raise the eyebrow of the affected side.



Dental procedure could damage the nerve by three mechanisms;

A-direct trauma to facial nerve by a **needle**,

B-Intraneural hematoma formation, and

C-Toxic damage due to local anesthetics. Needle may damage the small blood vessels around the epineurium that causes hemorrhage within the nerve caused by **compression and fibrosis**.

This effect occurs quickly (within 20-30 min) that the damage has been more increased than expected. Therefore, increasing pressure on the nerve results with damage.

Facial palsy following local anaesthetic administration has been reported to have an **incidence between 1:42 and 1:750,000**, and can have **several causes**,

1-direct trauma,

2-injection into a lobe of the parotid gland near where the facial nerve branches,

3-neurotoxic effects of the local anaesthetic, and also

4-type I (immediate) and type IV (delayed) **hypersensitivity reactions**,

with the onset of facial nerve paralysis sometimes delayed for over two hours.

Effects :

Loss of Motor Function to the Muscles of Facial Expressions.

Minimal or No Sensory Loss.

Unilateral Paralysis

Patient is unable to Voluntarily Close one eye.



7. Trismus:

Trismus defined as a **prolonged spasm of the jaw muscles by which the normal opening of the mouth is restricted (locked jaw).**

Causes:

- 1) Trauma to the muscles due to the injection of local anesthetics which is the most common etiological factor in Trismus.
- 2) Muscles irritation by local anesthetic solution contaminated by alcohol.
- 3) Hematoma in or around the muscles, the blood is slowly resorbed over approximately 2 weeks.
- 4) Infection after injection can also cause trismus.
- 5) Excessive volume of local anesthetic solution deposited into a restricted area produce distention of tissues which may lead to post-injection trismus and this is more common after multiple missed inferior alveolar nerve block.

Prevention:

- Use sharp, sterile, disposable needle.
- Practice atraumatic insertion and injection technique.
- Avoid repeated injections and multiple insertions into the same area, by getting a good knowledge of anatomy and proper technique.
- Use the minimum effective volume of local anesthetic solution.



Management:

- Heat therapy, which consists of applying hot and moist towels to the affected area for approximately 20 min every hour or using warm saline rinse; a teaspoon of salt is added to a glass of warm water.
- Use analgesic and muscle relaxant.
- The patient is advised to initiate physiotherapy consisting of opening and closing the mouth as well as lateral excursions of the mandible, chewing gum is another means of providing lateral movement of the temporomandibular joint.



8. Soft tissue injury:

The soft tissue anesthesia lasts longer than pulpal anesthesia. Trauma to the anesthetized soft tissue can lead to swelling, pain and even infection.

Causes: Self-inflicted trauma to the lips and tongue frequently occurs when the patient bites or chews these tissues while still anesthetized. Trauma occurs most frequently in younger children and in mentally retarded patients.

Prevention:

- The local anesthetic of appropriate duration should be selected.
- A cotton roll can be placed between the lip and the teeth if they are still anesthetized.
- Warn the patient against drinking hot fluid, and biting the lips or tongue.

Management: Management is symptomatic:

- 1) Analgesics for pain.
- 2) Antibiotic if necessary.
- 3) Warm saline rinses to aid in decreasing any swelling present.
- 4) Use any lubricant to cover the lip lesion and minimize the irritation.



9. Hematoma:

Hematoma is a localized mass of extravasated blood that may become clinically noticeable following injection. It is caused by penetration of the blood vessel with the needle during injection. The patient will notice the development of swelling and discoloration (bruise). Intraorally the blood vessels most commonly associated with hematoma are:

- the pterygoid venous plexus
- the posterior superior alveolar vessels
- the inferior alveolar vessels in the pterygomandibular space
- the mental vessels
- the infraorbital vessels

Prevention:

- 1) Learn anatomical landmarks and injection technique.
- 2) Avoid relocating the needle to different sites inside the tissue.

Management:

- If it is visible immediately following injection, apply direct pressure if possible. Once bleeding has stopped, inform the patient of what was happened and reevaluate the possibilities of continuing the treatment. Instruct the patient to avoid application of heat over the area, prescribe analgesic and antibiotic if necessary.
- If it is invisible like in case of pterygomandibular space hematoma, the patient will come in the 2nd or 3rd day complaining of trismus, in this case, treat the case as trismus.



10. Infection:

Infection after local anesthesia has become rare since the introduction of sterile disposable needles.

Causes:

- Contamination of the needle by touching the mucous membrane in the oral cavity before the administration of local anesthesia.
- Improper technique in the handling of the local anesthetic equipment.
- Injecting the solution into an area of infection, which might transport bacteria into adjacent healthy tissues (spreading the infection).

Prevention:

- 1) Use sterile disposable needles.
- 2) Proper handling of the needle to avoid its contact with nonsterile surfaces.
- 3) Use cartridge only once and store it in their original container, covered at all times.

Management: If an infection does occur the patient will complain of pain and trismus, immediate treatment consists of those procedures used to manage trismus. A course of antibiotic should be prescribed to the patient for 7 days.

Summary: to give efficient local anesthesia, you should gather the following three elements:

- Thorough anatomical knowledge
- Mastering a good technique
- Sterile handling of the dental syringe assembly



11. Ocular Complications :

The most widely used method for controlling pain during dental procedures is the intraoral administration of local anesthetics in close proximity to a specific nerve or fiber to obtund nerve conduction.

The most commonly anesthetized nerves in dentistry are branches or nerve trunks associated with the maxillary and mandibular divisions of the trigeminal nerve (cranial nerve V).

However, other nerves may be inadvertently affected by intraoral local anesthesia injections, resulting in anesthetic complications of structures far from the oral cavity.

Practitioners should be aware of potential ocular complications following intraoral injections in dentistry. These complications **include oculomotor paralysis** and **vision loss**. The knowledge of these conditions and their potential cause should alert the dentist to the importance of appropriate injection techniques and an understanding of management protocol

Ocular Complications

▣ **Amaurosis** “temporary blindness“.

▣ **Mydriasis** “Pupillary dilation”

▣ **Ptosis** “droopy eyelid”

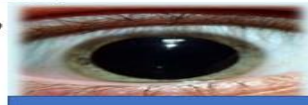
▣ **Diplopia** “double vision”

The most commonly reported **complication** was diplopia (39.8%), mostly resulting from paralysis of the lateral rectus muscle. Other relatively frequent **complications** included **ptosis** (16.7%), mydriasis (14.8%) and amaurosis (13%)

Causes

Inadvertent arterial injection with retrograde blood flow

▣ **Orbital injection** : Inadvertent injection into the orbit through the inferior orbital fissure.



An estimated **1 in 1,000** local anaesthetic injections in the **maxilla or the mandible** lead to unwanted effects on the **ipsilateral eye**

Ocular complications following local anaesthesia are uncommon and the frequency is estimated to be **1 in 1000**.

They can, however, cause considerable anxiety to both the patient and the clinician. From the patient's point of view, this is a totally unexpected event and may be extremely alarming.

The clinician, if not acquainted with the nature of these complications, may fail to diagnose such an incident, and may even attribute it to a more serious event, like a transient ischemic attack.

It is therefore essential that the **clinician understand the etiology and pathogenic mechanism of these complication**

• **Prevention** □ **Aspiration** before actual injection. □ **Inject slowly**.

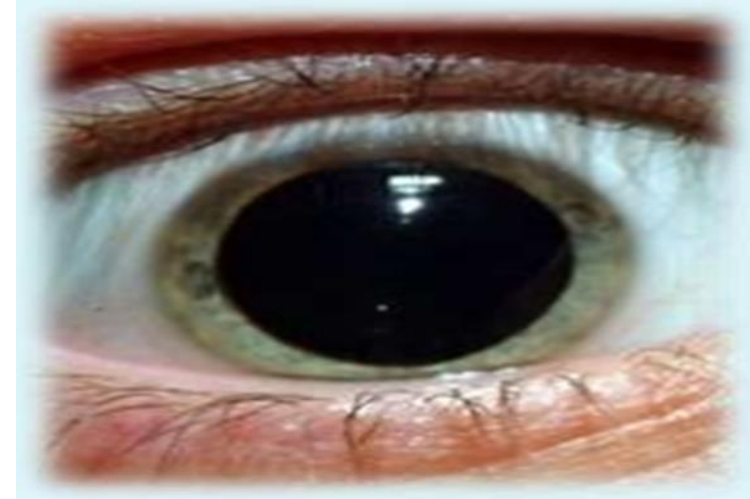
❖ **Treatment**

❖ Reassure the patient that is **transient**.

• □ **Cover** the affected eye with gauze dressing.

• □ Refer **patients to an ophthalmologist for evaluation** if it last **more than 6 hours**

• □ Regular **follow-up**



12-Oro-Facial blanching :

Facial blanching as a complication of local anesthesia is reported in dentistry. **Inadvertent arterial penetration and subsequent vasospasm has been accepted as the mechanism of this phenomenon.**

Inferior alveolar nerve block anesthesia (IANBA) is commonly used in dental anesthesia while performing minor mandibular surgeries, such as third molar extraction. It is also essential in dental conservative treatments, including endodontic treatment.

However, the widespread use of IANBA in dental treatment has resulted in various complications.

Delivering an anesthetic to the mandibular foramen is difficult when the injection site is further away from the mandibular foramen; therefore, IANBA can lead to failures and cause complications.

Facial blanching after IANBA can be caused by anesthetic injection into the **maxillary artery area**, affecting the **infraorbital artery**.

Studies have suggested that peripheral vasoconstriction of the facial arterioles supplied by the **infraorbital artery occurs** because of the effect of the **α -receptor agonist** .



Prevention:

The most important measures that can be taken in order to avoid such complications in dental anaesthesia are aspiration control before injecting anaesthetic solution and injecting anaesthetic agent at a slow rate after negative aspiration.

After the skin ischaemia associated with the severe and instant pain, the patient should be calmed and informed about the reversibility of the situation

13-Edema

Swelling of tissues can be due to:

1-trauma during injection, 2-infection, 3-allergy, 4-hemorrhage, and 5-injection of irritating solutions.

The management of edema is dependent on the cause.

Allergy-induced edema treatment consists of intramuscular epinephrine injection and, additionally, antihistamine and corticosteroid administration and consultation with an allergist to determine the precise cause of the edema. Trauma-induced edema should be managed as a hematoma. For the treatment of edema produced by infection, antibiotics should be prescribed



Sloughing of tissue

Causes

- **Epithelial desquamation**

1. Application of topical anesthetic for prolonged period
2. Heightened sensitivity of tissues to chemical agents
3. Reaction in an area where topical anesthetic is applied

- **Sterile abscess**

- 1-Secondary to prolonged ischemia, always occur in the tissue of hard palate

Prevention

- Use topical anesthetic as recommended
- Allow the solution to contact the mucous membrane for 1 to 2 min
- When using vasoconstrictor for homeostasis don't employ overly concentrated solutions

Management

- Management may be symptomatic
- Topically applied ointment such as Orabase to minimize irritation



Post anesthetic intraoral lesions

The regular dental practice involves various dental procedures which needs the application of local anesthetics. It is mainly given as **local infiltration** or **nerve blocks**.

In local infiltration, anesthetic solutions are injected close to the apex of the **involved tooth structures**.

Developed ulceration of palate after administration of local anesthetic containing a **vasoconstrictor along with its management**

CAUSES

- Recurrent aphthous stomatitis • Herpes simplex
- Trauma to the tissue by needle and LA solution

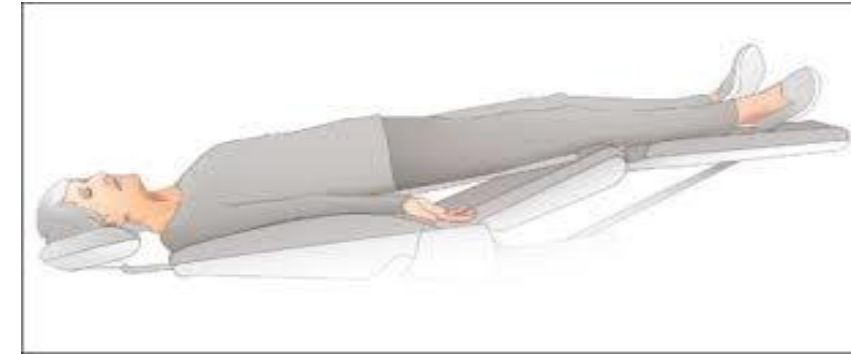
MANAGEMENT

- Primary management is symptomatic
- Viscous lidocaine can be applied
- A mixture of equal amount of diphenhydramine and milk of magnesia rinse in the mouth effectively coats the ulceration
- Orabase a protective paste provides a degree of pain relief.



Systemic Complications of Local Anesthesia

1. Fainting (vasovagal attack)
2. Hypersensitivity or allergy to local anesthesia
3. Overdosage and toxicity
4. Drug interaction



1. Fainting (Vasovagal Attack)

The nerve, which is the largest and most complex nerve in our body, exits the brain behind the ear and continues to innervate structures in our **throat**, through the chest (including heart, lungs and aorta) all the way down to our abdomen to our intestines

It is the most **common systemic complication that occurs with local anesthesia in the dental office**. It refers to a **sudden transient loss of consciousness** usually **secondary to cerebral ischemia**.

The cerebral ischemia is secondary to vasodilatation or an increase in peripheral vascular bed, with a corresponding drop in blood pressure.

The collapse in the dental chair may occur suddenly and may not be accompanied by loss of consciousness, in most instances, these episodes are vasovagal attack and spontaneous recovery is usual. The patient often complains of feeling dizzy, weak, and nauseated, the skin is pale, cold and slow pulse is noticed.



Predisposing Factors or Triggering Stimulus

The predisposing factors for this condition may be divided into two groups:

1) Psychogenic factors:

- Anxiety
- Emotional stress
- Pain of sudden and unexpected nature
- The sight of blood, surgical or other dental instrument such as a local anesthetic syringe, an injection needle, etc.

2) Non-psychogenic factors:

- Sitting in an upright position or standing for a prolonged period, it leads to pooling of the blood in the periphery, thereby decreasing cerebral blood flow.
- Hunger or starvation, which leads to a decrease in cerebral blood glucose level.
- Poor physical condition.
- Hot, humid and crowded environment.



Mechanism of Vasovagal Syncope

Regardless of the trigger, the mechanism of syncope is similar. The brainstem is activated directly or indirectly by the triggering stimulus, resulting in simultaneous enhancement of parasympathetic nervous system (vagal) tone and withdrawal of sympathetic nervous system tone.

This results in the following responses:

- 1) The cardioinhibitory response, characterized by a drop-in heart rate and in contractility leading to a decrease in cardiac output that is significant enough to result in a loss of consciousness.
- 2) Vasodepressor response: dilation of the blood vessels as a result of the withdrawal of sympathetic nervous system tone. The blood will pool in the dilated peripheral vessels, at the same time, the blood flow to the brain is reduced.

Management

The first aid treatment should **be started at once**, the head of the patient should be lowered, the legs elevated, tight belt and collar should be loosened, and respiration is stimulated. Spontaneous recovery is usual and it is often possible to complete the treatment at the same visit.

If signs of recovery are not apparent within 30 – 40 seconds, the collapse probably is not a vasovagal attack, the airway must be maintained, oxygen administered and, in this case, a medical emergency team should be asked immediately for help.



Cardiopulmonary Resuscitation (CPR)

An emergency procedure that combines chest compressions with artificial ventilation in an effort to manually preserve intact brain function until further measures are taken to restore spontaneous blood circulation and breathing in a person who is in cardiac arrest. It is recommended in those who are unresponsive with no breathing or abnormal breathing.

CPR involves chest compressions for adults of about 5 cm deep and at a rate of at least 100 per minute with a universal compression to ventilation ratio of 30:2 is recommended for adults. The rescuer may also provide artificial ventilation by either exhaling air into the subject's mouth (mouth-to-mouth resuscitation) or use a device that pushes air into the subject's lungs (mechanical ventilation).



2. Hypersensitivity or Allergy to Local Anesthesia

In general, **hypersensitivity reaction to local anesthesia is very rare and represent less than 1 % of all complications of local anesthesia.** True allergic response to local anesthesia may be **localized or generalized** and it may be **immediate or delayed in onset**, also the allergic reactions may vary from mild skin irritation or rashes to an anaphylactic reaction.

Local reactions are seen more frequently than systemic and usually resolve without active treatment. If any degree of allergic reaction is observed, it is very important to determine the actual cause (allergen).

Inadequate diagnosis and treatment can be life-threatening to the patient.

Clinical manifestations of allergy vary and include the following: fever, angioedema, urticaria, dermatitis, photosensitivity or anaphylaxis.

It is more commonly seen with **ester type agent than amide type.**

Hypersensitive state, acquired through exposure to a particular allergen.



Signs and Symptoms:

• **Fever** • **Edema** • **Urticaria** • **Dermatitis** • **Bronchospasm** • **Systemic Anaphylaxis**

Hypersensitivity reaction could be due to:

- local anesthetic agent
- vasoconstrictor
- additives like bisulfite which is used as a preservative



Allergic reactions cover a **broad spectrum of clinical manifestations ranging from mild and delayed response** occurring as long as **48 hours** after exposure to allergen, to **immediate and threatening reaction** develop within **seconds of exposure**.

1% of all reactions occurring during administration of LA are allergic in nature.

Caused by **antigen – antibody reaction leading to release of histamine or histamine like substances**. ◦ **Sodium Bisulfite**(Antioxidant in vasoconstrictor local anesthesia.1984 has been excluded.) , ◦ **Epinephrine.**, ◦ **Latex of the cartridge** . ◦ **Topical**

Anesthesia:

Mostly ester.

Preservatives containing such as **methylparaben**, **ethylparaben**, or **propylparaben**.

Prevention

Proper pre-anesthetic evaluation, which includes a **proper personal history** and **the past dental history**, particularly history of allergy to the **local anesthetic agent**, or **history of allergy** to any other drug.

Management

- Antihistamine injection
- Epinephrine 0.5 ml of 1:1000 IM (intramuscular)
- Administer O2 if necessary.



Steps of management :

- 1-The patient should **lie flat**, but also in the case of breath
 - 2-Adrenaline 1:1000 dilution (0.01 mg/kg up to 0.5 mg per dose) should be administered intramuscular **with 1-mL syringes**, 21 gauge needles, and **should be repeated every 5 minutes as needed**.
 - 3-Another recommendation for **epinephrine** is or **children and adults who weigh 30 kg or over is 0.3 mg**. For those weighing 15 to 30 kg, the epinephrine **dose is 0.15 mg**.
- The use of **adrenaline auto-injector** can also be chosen, which is carried mostly by heavy allergic patients themselves.

Adrenaline should be administered for anaphylaxis by intravenous (IV) route only in the case of profoundly hypotensive patients or **patients who develop a cardiopulmonary arrest** or those who fail to respond to multiple doses of IM adrenaline because of the potential cardiovascular adverse effects of IV administration of adrenaline.

Intramuscular injection is the **first line of treatment** in the treatment of anaphylaxis. But the use of **antihistamines** and **glucocorticoids** is controversial.

Substitution of the local anesthetic agent

The local anesthetic agent can be substituted with **another type of agent**. If the reaction is in response to **ester-type** then an **amide type** such as **lidocaine** could be used.

Anaphylactic Shock

It is a rare, life-threatening hypersensitivity reaction to an antigen. It develops fast causing death within a few minutes. It is characterized by:

- 1) profound fall in the blood pressure
- 2) dyspnea and respiratory embarrassment
- 3) facial and laryngeal edema
- 4) loss of consciousness

Management of anaphylaxis

If you suspect the anaphylactic reaction, immediately seek medical help.

- Epinephrine is the most important medication; it is given as intramuscular injection working rapidly to make the blood vessels contract. it also relaxes the airway, helping the individual breathe easier and stop itching.
- Even if the patient responds to the epinephrine, it is vitally important to go to an emergency room immediately.
- Oxygen should be given to improve breathing.
- Intravascular **(IV) fluid** may be necessary to **restore adequate blood pressure**.
- Antihistamine should be given to contract the effect of histamine.
- If the patient stops breathing, start cardiopulmonary **resuscitation (CPR) immediately** until the patient begins to breathe again.



3. Overdosage and Toxicity

It is relatively rare, a toxicity reaction can occur when the concentration of local anesthesia in circulation increases too rapidly within a short period of time as in injecting too rapidly into the highly vascular area or when giving IV injection.

The toxic effect is primarily directed to the central nervous system (CNS) and cardiovascular system (CVS). The dose necessary to induce toxicity varies among patients and is influenced by numerous factors which are:

- The patient general health, age and weight: in patients with dysfunction of the liver and kidneys, there is an increased level of local anesthetic in the bloodstream.
- Rapidity of injection.
- Rout of administration.
- Amount of local anesthesia administered.

• Clinical Manifestation overdosage toxicity

- Talkativeness , Apprehension.
- Excitability. Slurred speech. Stutter. Sweating.
- Vomiting.
- Failure to follow commands.
- Elevated blood pressure, heart and respiratory rate
- Tonic-clonic seizure in highly overdose.
- CNS depression, Myocardial Depression and cardiac arrest



Management :

- Stop the dental procedure.
- Position the patient supine with legs elevated.
- Reassurance of the patient.
- Administer O₂, IV anticonvulsant and monitor vital signs.
- Allow the patient to recover and then discharge.
- If the patient fails to recover then transfer him to the hospital.

Basic Emergency Management : **P-C-A-B-D**

1. Position.
2. Circulation.
3. Airway.
4. Breathing.
5. Definitive Care

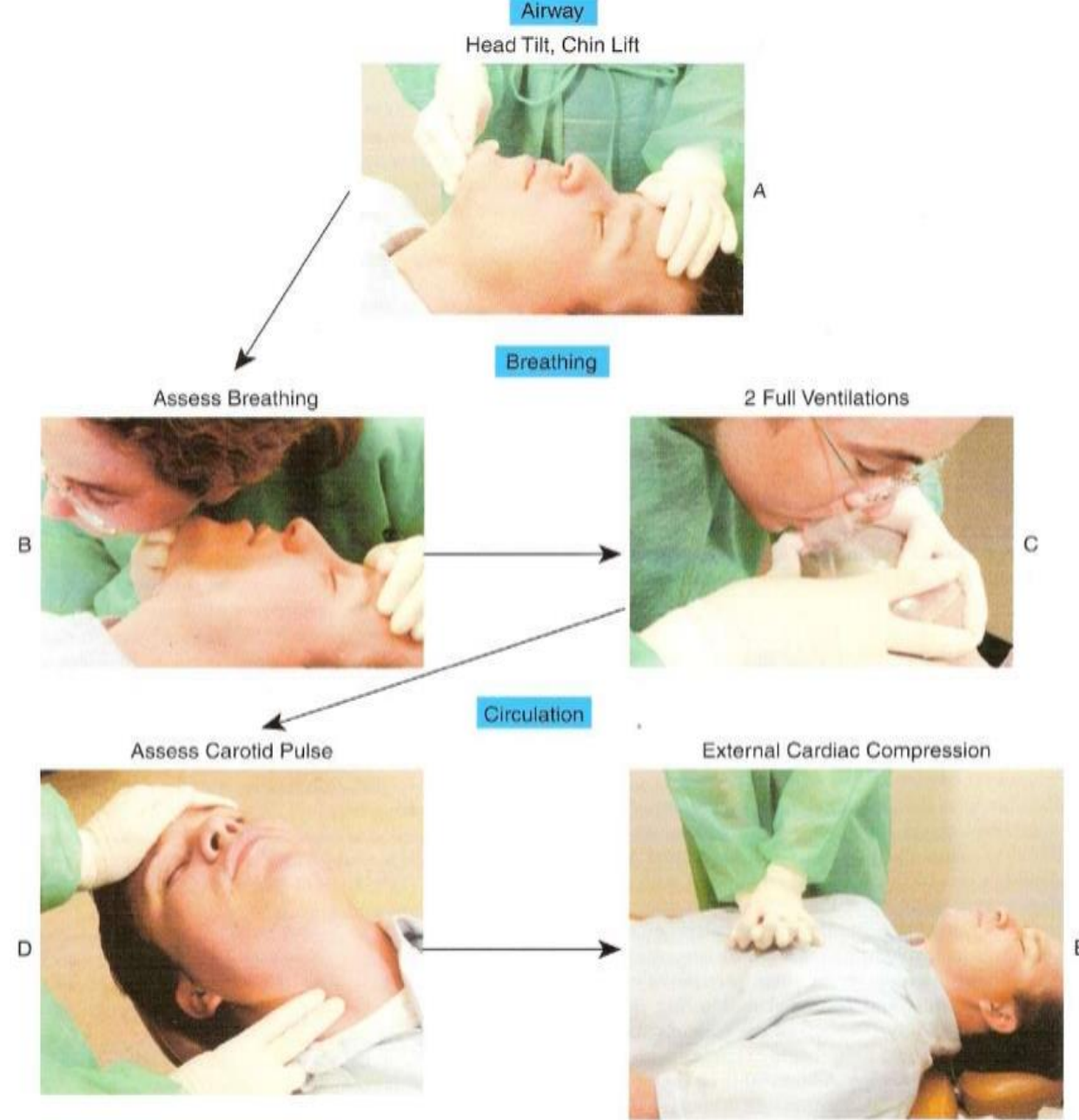


Figure 18-17. Summary of basic life support. A, Airway—head tilt, chin lift. B, Assess breathing. C, Two full ventilations. D, Assess carotid pulse. E, External chest compression—15 compressions; 2 ventilations.

Preventing from a toxic dose complication,

Preventing from a toxic dose complication:

The best method to avoid toxic reactions is by:

- 1-Use the smallest possible volume and lowest effective concentration. Healthy adults, the suggested maximum safe dose of 2% lignocaine in 1:80,000 adrenaline is **four-and-a-half 2** or 2.2 mL cartridges (180–198 mg lignocaine); for 3% prilocaine and felypressin 0.03 i.u./mL, the maximum safe dose is 400 mg (**six 2 mL cartridges**)
Another strategy to reduce toxicity is using the **guideline of 1/10th cartridge per kilo as a rough guide to the maximum dose** .
- 2-The local anesthetic solution should be injected slowly.
- 3-Avoid intravascular administration by the use
- 4-Dentists should be aware that excessive doses of topical anesthetics while these agents are more concentrated to facilitate infiltration may lead to toxic effects, particularly in children
- 5-Treatment at the **office includes airway support**, administration of **100% oxygen**, supine positioning, and protection from injury in the event of seizure activity, treating convulsions (benzodiazepines or thiopental;

Factors adding to the increased risk of local anesthetic overdose in younger patients

- 1) Treatment plan where all four quadrants are treated with local anesthetic in one visit.
- 2) Use of plain local anesthetic.
- 3) Exceeding the maximum dosage based on patient's body weight.

4. Drug Interaction :

Unless the patient is on a concomitant local anaesthesia infusion for an ailment (**a potential risk for local anaesthetic systemic toxicity**), there are no **significant drug interactions** with **non-epinephrine-containing local anaesthetics**. **Drug interactions stemming** from the contents of a local anaesthetic cartridge are almost exclusively from the **included vasoconstrictor**. As such, epinephrine should be use with great caution when concomitant use of one of the drugs is present, and levonordefrin should be avoided altogether when the patient is taking a tricyclic antidepressant

In some patients, the administration of two drugs will **counteract** each other, while in others, potentiation occurs. In patients using a **tricyclic antidepressant**, variable degrees of potentiation of blood pressure response to adrenaline will occur even to **small doses**; therefore, precautions should be taken during the use of these vasoconstrictors with the patient taking a **tricyclic antidepressant**.

THE DRUG INTERACTION SIGNIFICANCE RATING SCALE.*		
SIGNIFICANCE RATING	SEVERITY RATING	DOCUMENTATION RATING
1	Major	Established, probable or suspected
2	Moderate	Established, probable or suspected
3	Minor	Established, probable or suspected
4	Major or moderate	Possible
5	Minor All	Possible Unlikely

ADVERSE DRUG INTERACTIONS IN DENTISTRY: VASOCONSTRICTORS.

POSSIBLE DRUG INTERACTION	CUMULATIVE RATING*	MECHANISM AND CLINICAL PRESENTATION
Vasoconstrictor with tricyclic antidepressant (levonordefrin with imipramine)	1	Sympathomimetic effects may be enhanced. Epinephrine should be used cautiously; use of levonordefrin should be avoided.
Vasoconstrictor with nonselective β -adrenoceptor antagonist (epinephrine with propranolol)	1	Hypertensive and/or cardiac reactions are possible. Vasoconstrictor should be used cautiously; blood pressure and heart rate should be monitored.
Vasoconstrictor with general anesthetic (epinephrine with halothane)	1	Increased possibility of cardiac arrhythmias exists with some general anesthetics. Consultation with anesthesiologist is recommended.
Vasoconstrictor with cocaine (epinephrine with cocaine)	1	Arrhythmias and hypertensive responses possible. Concurrent use should be avoided.
Vasoconstrictor with antipsychotic or other α -adrenoceptor blocker (epinephrine with chlorpromazine)	4	Hypotension resulting from overdose of antipsychotic agent may be worsened. Vasoconstrictor should be used cautiously.
Vasoconstrictor with adrenergic neuronal blocker (levonordefrin with guanadrel)	4	Sympathomimetic effects may be enhanced. Vasoconstrictor should be used cautiously.
Vasoconstrictor with local anesthetic (lidocaine with epinephrine)	4	Multiple effects on systemic toxicity, which may be self-limiting.
Vasoconstrictor with thyroid hormone (epinephrine with thyroxine)	4	Summation of effects possible when thyroid hormones are used in excess. Vasoconstrictor should be used cautiously if signs of hyperthyroidism are present.
Vasoconstrictor with monoamine oxidase inhibitor (epinephrine with phenelzine)	5	No substantial evidence of an interaction.

STOP AND REMEMBER:

Patients with complex medical and drug histories are becoming more commonplace in dental practice..

Nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit the renal excretion of lithium and lead to lithium toxicity.

Metronidazole and fluconazole inhibit the metabolism of warfarin by blocking cytochrome P-450 2C9 (CYP-2C9), the major metabolic pathway of warfarin, with the end result being dramatic increases in patients' international normalized ratios (INRs) and potentially fatal bleeding.

Propranolol and other nonselective beta-adrenergic blocking agents can inhibit the vasodilatory effect of epinephrine in dental local anesthetic solutions, leading to hypertensive reactions and a concomitant reflex bradycardia.

It is important for clinicians to recognize and avoid these serious drug interactions

Methemoglobinemia:

Methemoglobinemia is a unique dose-dependent reaction where the iron in hemoglobin is stabilized in the ferric (Fe³⁺) form, unable to attach oxygen, leading to tissue hypoxia and causing a varying degree of cyanosis.

Methemoglobinemia can be either inherited or acquired .

The risk of methemoglobinemia increased in infants and the elderly. Patients with underlying health problems; **liver cirrhosis**, with **underdeveloped hepatic and renal function**; **heart disease**; and **pulmonary disease (chronic obstructive pulmonary disease, pneumonia)** are under the risk of methemoglobinemia. When administered in **excessive doses**, the local anesthetics mostly prilocaine and benzocaine (90% of reported cases) and barely lidocaine and articaine may also lead to **methemoglobinemia**.

The complications related to dental **local anesthetics lidocaine, bupivacaine, cocaine, mepivacaine, prilocaine**, and **tetracaine** in children and adults.

Benzocaine should be out of usage. In a specific **patient group, in children younger than 6 months, in pregnant women, or in patients taking other oxidizing drugs, prilocaine should not be used**. The dose should be limited to 2.5 mg/kg .



THANK YOU