ORAL AND MAXILLOFACIAL SURERY:-

Is one of the dental specialties dealing with management of diseases, injuries and defects of human jaws and associated structures. Oral surgery forms the connecting link between medical and dental specialties.

Diagnosis in surgery:-

Oral diagnosis is the art of using the scientific knowledge to identify the oral diseases and also to distinguish one disease from another.

The diagnostic process classically involves the following steps:-

1- History taking.
2- Clinical examination.
3- Investigation.
4- Provisional diagnosis.
5- Definitive diagnosis and treatment plan.

In oral surgery practice, clinician is often faced with the diagnosis of the following conditions:-

1- Dental and facial pain.
2- Swelling (lump, mass).
3- Ulcers;
4- Injuries (dental, facial bones).
5- Tempromandibular joint problems.
6- Medically compromised patient.
7- Facial deformity.

History taking:-

The art of taking an accurate case history is probably the most important single step in the diagnosis of medical or surgical condition. History taking should be systematic, using special set or sequences. During history taking the clinician or the dental surgeon listen to the patient's story or talks and list the symptoms in order of severity or importance. By patient's words.

Symptoms:-

Means a subjective problem that the patient describes e.g. pain, parasthesia.

Signs:-

Means (objective) an abnormal presentation detectable by the clinician, e.g. swelling, ulcer.

So detection of signs and symptoms of a disease may aid in diagnosis of that disease.

Objectives of taking history:-

1- To provide the dentist with informationsthat may be necessary for making diagnosis.
2- To establish a good or positive professional relationship with the patient which affect cooperation and confidence.
3- To provide dentist with information concerning patient's past and present medical, dental and personal history.
4- To provide information about patient's systemic health which may greatly affect the treatment plan and prognosis and diseases that could be transmitted to the dentist, his staff or other patients.
5- It serves as a legal document.

How you take history:-

During history taking the dentist should encourage his patient to describe his symptoms in his own words, interrupting his story only to explain a point or stop a useless talk.

A clear and concise summary of patient's complaints should be recorded in the case sheet. The symptoms should be recorded or being listed in order of its importance (e.g. pain, swelling, bleeding).

During taking the history give your patient your whole attention and never take shortcuts.

You have to avoid speed in taking the history, so you have to give the patient a suitable time to give all information, because hurry in taking history may lead to many pitfalls that affect the accuracy or completeness.

You have to avoid the leading questions (e.g. does the pain comes on taking hot or cold?) it's better to ask him what is or what are the things that brings pain to you? Or anything hurt you?

During taking history don't depend on the patient diagnosis or the diagnosis of a previous doctor, so you have to ask the patient to describe his complaining-only-to establish your .diagnosis process.

Components of the patient history:-

The case history may include commonly the following sections or components:-

1- Biographic data (personal history).
2- Chief complaint (C.C).
3- History of the chief complaint (history of the present illness) H.P.I.
4- Past dental history.
5- Medical history and systems review.
6- Family history.

Biographic data:-

Includes the full name of the patient, age, sex, address and telephone number and occupation. *these*informationsmay aid or contribute to the diagnosis since some medical problems have a tendency to occur in a particular age group, sex or race. The patient occupation maybe associated with a particular disease or may influence the type of therapy.

Chief complaints (C.C):-

The chief complaint is usually the reason for the patient's visit. The chief complaint(s) is best stated in the patient's own words in a brief summary of the problems (e.g. pain, swelling, ulcer, parasthesia, numbness, clicking, haliatosis, bleeding, trismus). If the patient complaining of several symptoms in which case they should be listed, but with the major complaint first.

History of the present illness (H.P.I):-

This part of the story must be gone into complete details and get the patient to **tell** the story in his fashion, never ask the patient leading questions and you have **to see** if the patient in a condition able to give you a history which is reliable and his statement, can be relied upon.

It's best to start by asking the patient about:-

I- Duration (record the length of the complaint).

7,- Onset (date of onset, manner of onset).

3- Precipitating/predisposing factors, (e.g. hot, cold, sweet).

4- Characteristic, and this includes:-

a) Nature e.g. (continuous, intermittent, stabbing).
b) Severity e.g.(mild, sever, very sever).
c) Location.
d) Radiation (feeling of pain in site other than that of causative lesion, called referred pain).
e) Temperature features.
f) Aggravating- factors.
g) Relieving factors.
h) Associated constitutional symptoms and signs.

5- Course and progress.

6- Therapy:-

a) Type of therapy and dose.
b) Provider.
c) Effect of therapy.
d) Date of therapy.

7- Other informations.

So if a patient comes with a chief complaint (pain) very detailed history of the pain should be taken and particular attention paid to the following points:-

a) **The duration of pain**: Whether any incident which might have played some part in the etiology of the pain precede its onset (e.g. a blow on the jaw, dental treatment), duration record the length of the pain.

b) **Site of the pain**: The patient should be asked to point to the place where the pain is felt, using his finger.

c) **Any radiation of the pain**: If the pain radiates, the patient should be asked to demonstrate its course with the tip of his finger. On other occasions pain maybe felt in a site other than of the causative lesion or remote from the diseased area and this type called "referred pain", e.g. pain of pericoronitis radiates to the ear.

d) **The precise characteristic of the pain**: the pain maybe described as sharp, sever, dull, throbbing, excruciating, lancinating, mild, continuous, intermitted, all these objectives can be applied to the pain in different pathological process which may help you in the diagnosis. (In acute pulpitis, the pain is sharp and sever, in acute dental abscess the pain is dull, throbbing and sever and the tooth tender, in acute maxillary sinusitis the pain is dull, throbbing and continuous).

e) **Timing of pain**: Some pains are characteristically worse at particular time in the day e.g. pulpal pain often wakens the patient at night and tend to keep him awake, in-acute periodontitis the pain is worse at meal' time.

f) **Any factors which precipitate the pain**: Pulpal pain is often precipitated by thermal and osmotic stimuli (hot, cold, sweet). Periodontal pain often precipitated by biting and chewing.

g) **factors or Any drugs which relieve pain**: This will give you an idea about the nature and duration of severity of the pain.

h) **The presence of other symptoms**: Like the patient that says that, the pain started for two days, then a swelling appeared after that or discharging sinus appeared or a discharge of pus, or pain, swelling then Parasthesia of the lower lip ...etc.

i) **The patient also may be asked about relevant past medical history** which may assist you in the diagnosis of the pain like patient with facial pain of vascular origin like migraine, or chronic psychosomatic origin or angina (angina pectoris) pain. In addition to that the patient asked about his opinion of the cause of the pain.

Another example of complaint is:-

Patient presented with a "lump or mass":-.

The oral surgeon must be ascertain by asking some questions: -

1- How long the swelling has been present.
2- Whether it is getting larger or smaller or fluctuated in size.
3- What are the symptoms of the lump: The lump maybe painful or not. If the lump is associated with Parasthesia or numbness of the lower lip for example.
4- Whether there is any possible cause for the swelling e.g. trauma, injuries, or systemic illness known to the patient.
5- What made the patient notice the lump? By feeling or because it is painful or someone else noticed it and told him.

Past dental history (P.D.H):-

The past dental history includes:-

1-The frequency of previous visits (e.g. previous extractions or oral surgical procedures).

2- Any difficulties or complications (e.g. excessive bleeding or fainting).

3-Determination of the availability of past dental or oral radiographs. In other words, it is important to ask the patient about any type of dental or oral treatment received before, and if there is any complications or unsatisfaction arise and his impression about the type of treatment.

Medical history and systems review (M.H):-

The patient's medical history includes review, the past and the present illness or diseases because:-

1- These informations(M.H) may aid in the diagnosis of various conditions occurring or has oral manifestation that are related to specific systemic disease (e.g. aids, leukemia).
2- The presence of many diseases may lead or need modification for the treatment plan, and affect the manner in which therapy is provided.
3-Drugs used in treatment of some systemic diseases can also have effects on the mouth (have oral manifestation), or dictate some modification to the dental or surgical treatment (e.g. anticoagulant drugs, chemotherapy).

The past medical history includes:-

1-Previous serious illness or diseases.
2- Childhood diseases.
3- Hospitalization.
4- Operations.
5- Injuries to the head and neck.
6- Allergy to drugs or general allergy.
7- Listing of medication taken in the last six months.

Some examples of serious illness:-

♦Heart attack or diseases (e.g. myocardial infarction, angina pectoris).

♦Stroke (cerebrovascular accident C.V.A).

♦ Hypertension.

♦ Heart failure.

♦ Bleeding

disorders.Diabetes.

♦ Rheumatic fever or disease.

♦ Hospitalizations may indicate past disease and how it was treated.

♦ Aids (acquired immune-deficiency syndrome).

♦ Viral hepatitis.

♦ Neoplasm and the method of treatment (surgical, cytotoxic drugs) especially if the growth in the head and neck region **or** previous radiation (radiotherapy).

♦ Allergic reaction to drugs.

Review of the systems: Is that part of medical history covering each major system of the body. Review of systems lead to concentration on the signs and symptoms related to that system disorders, which dictate us to more investigations or referring of the patient for medical evaluation and preparation. The review of systems includes:-

Cardio vascular system, respiratory system, central nervous system, genitourinary system, musculoskeletal system, endocrine system, ears, eye, vital signs (blood pressure, pulse, temperature, respiratory rate)

Oral surgery

Components of medical history

Any patient come to you should be asked certain concise questions that aids you to have medical history, and these includes :

l. If he is currently receiving any medical care or under supervision of any clinician.

2 . Whether he has been hospitalized and Why ?

3 . If you have any serious illness remembered by the patient ?

4 . If you have any surgical operation before ?

5 . If your patient takes any type of drugs before in the past or present time ?

Family History: (F. H.)

Details of (F.H.) may reveal valuable information about diseases that are occurring in families (e.g. Tuberculosis , Hemophilia , Psychiatric or neurotic disorders , Breast cancer) Congenital Anomalies such as lip clefts or palate clefts

.Clinical Examination

Careful history taking should be followed by a thorough and systematic clinical examination.

Diagnostic instruments includes:

1. Dental Mirror.
2. Dental Prone.
3. Tweezer.

The clinical examination in Oral Surgery should be systematic and should be always includes the vital signs like Body Temperature , Pulse Rate , Blood Pressure , respiratory rate , also should have always Comparative Observations of both sides .

Clinical Examination includes:

1 . Extra oral Examination .
2 . Intra oral Examination :

In extra oral examination we consider the general evaluation e.g. Observation the patient Posture , Gait, Facial Form, Nutrition Status , Speech , Body movement, Skin, Hair , Vital Signs . In addition to that we examine the area of the head and the neck thoroughly and this includes :

-Examination of the Tempromandibular Joint.
-Lymph Nodes.
-Salivary Glands.
-Bones of the Skull.
-Sinuses (Maxillary Sinus)
-Ear , Eye& Perioral Tissues .
-Neck examined for enlarged Thyroid Gland , Lymph Nodes of the neck, & swelling.

Methods of Clinical Examination:

In Clinical practice , examination of patient involves FOUR ROUTINE PROCEDURES

1.INSPECTION.
2.PALPATI0N.
3.PERCUSSION.
4.AUSCULATI0N.

•**INSPECTION (VISUAL)** :- At the start of every examination you must begin by looking at patient as a whole before looking at the region in question for signs that may provide clue for a Diagnosis any changes in the color , or asymmetry of the face , any growth , ulceration, Scar , Defect, Loss of tissue should be inspected by your eye .

PALPATION:- Next use your fingertips to feel for tender spots , Lump , Fluctuant Swelling , & Mobile teeth . Palpation gives information about texture , Dimension , consistency , Temperature& Functional Events

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PROBING:- Is the palpation with an instrument & is one of the most important diagnostic techniques used in Dentistry. The teeth are probed for caries with the dental probe & periodontal probe is used to measure the periodontal sulcus depth . Lacrimal probe used for examination of parotid &submandibular salivary gland ducts . Fistulous tracts can be probed with GuttaPercha points to determine the origin of the Fistula .

PERCUSSION :- Is the technique of striking the tissue with fingers or an instrument (e.g. Handle of the mirror) . The examiner listen to the resulting sounds & observes the response of the patient. Extra orally , percussion is often used to detect tenderness in the frontal and maxillary sinuses by tapping the finger tips against a finger placed over the sinuses . Intra orally, percussion is used to evaluate the teeth by tapping the teeth with mirror handle , this technique may induce pain in the area of inflammation from periodontal diseases or periapical abscess.

AUSCULATION- Is the act or process of listening for sounds within the body. e.g.Auscultation to the clicking in the Tempromandibular Joint (T.M.J.) by the use of stethoscope . Auscultation technique is rarely used in Dentistry .

Extra oral Examination

Objectives: -

l.To evaluate any general abnormalities & in particular those of the head & neck region .

2.To look for signs & symptoms of the patient that could influence diagnosis & treatment. This examination includes :-

*General examination of the patient including his Posture, Gait, Facial Form , Nutritional, Status ,Habits ,Speech ,Skin ,Hair, nail, & all exposed parts of the body.

*Examination of head include T.M.J., Lymph Nodes (Submandibular, Sub mental, etc..), Salivary gland Parotid & Submandibular gland etc.), Bones of skull, Sinuses (Maxillary Sinus), ear , eyes,&peri oral tissues .

Examination of neck include Thyroid gland, Lymph Nodes (Cervical node anterior & posterior)& other midline structures & muscle (The neck should be inspected for midline or lateral swelling, scar, or any inflammatory lesions palpated for Thyroid enlargement or Cervical lymph node enlargement.

The T.M.J, palpated for any clicking or pain , & asking the patient to open and close the mouth to see if there is any limitation of opening (Trismus), or deviation of occlusion .

The eyes should be examined for Exophthalmos or proptosis , pallor of Conjunctiva may indicate Anemia. Sclera of the eye should be also examined, Yellow discoloration may indicate Hepatitis or Obstructive Jaundice (Liver Diseases).

IntraoralExamination:-

Objectives

1.To detect soft tissue abnormalities .

2.To evaluate the status of teeth and other hard tissues .

Intraoral examination consists of evaluation of the following areas in systematic ways : Lips, Labial & Buccal Mucosa, Muco buccal folds, floor of the mouth, Tongue, Hard & Soft Palate, Oropharynx, Muscle of mastication(Lateral & Medial muscles.), Teeth, Gingiva, Orifice of the ducts of the Parotid and Sub*ma*ndibular Glands .

intra oral examination should begin with the observation of the mouth for extent or deviation. The extent of the opening usually described in terms of the of the width of the patient's fingers e.g. 2, 3 or 4 fingers opening then we look for the oral Hygieneweather is good, fair poor, or very poor.

We use the mouth mirror to reflect or retract the cheek & the lips with good light, to evaluate the condition of the vestibules, floor of the mouth, ventral surface of the tongue avoid any overlooking of these hidden areas, also the opening of the salivary glands ducts examined for enlargement, redness, & discharge.

The ventral, lateral, dorsal, aspects of the tongue should be examined for the presence or absence of papillae, fishuring, ulceration, growth, indurations, limitation in extraction, & lateral movement.

Hard & soft tissue examined for swelling, ulcers, sinuses, & perforation .

Mucosal changes may be observed in association with Leukoplakia, Tobacco irritation, Pigmentation .

The gingiva examined for the slipping, the color & the size of interdentally papillae, any cause of food impaction, the presence of calculus, sinuses or retained roots, pocket etc...

Teeth Examination:

The presence, absence, appearance, mobility, retained roots, retained deciduous teeth, Malposed teeth, mobility of teeth classified as nil, marked or gross Attrition (Exposed dentin), Exposed roots, Carious Lesions, Vitality test (hot & cold application , Pulp tester , etc...). The teeth might be percussed or probed with our instrument to see any tenderness or sensitivity of the teeth. Any edentulous area should be dried with a piece of cotton and examined for the presence of retained roots or discharging sinuses . Occlusion should be examined in closed and rest position the presence of open bite, type of occlusion (Neutro occlusion class I, or disto occlusion class II , or mesio occlusion class III)

Investigations

Sometime the clinician determines that additional tests are needed to clarify some aspects of the diagnosis such tests include radiographic examination, Biopsy(Histological Study), Cytology, Aspiration, Clinical Laboratory studies .

*Radiographic examination:

Is one of the special methods of examination which mostly used in the Oral Surgery. It provide information about hard & soft tissues that are hidden for eye which aid in diagnosis & to evaluate the progress of the disease . For example Peri apical, occlusal, & extra oral views like lateral oblique of the Mandible radiograph. CT Scan, MRI

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•VTTALITYTEST:-

1 .Hot application (e.g. Hot instrument)

2.Cold application (e.g. Ethyl Chloride Application)

3.Electrical pulp Tester. Used to check the vitality or response of teeth.

BIOPSY:-

Small pieces of tissue taken from the lesion submitted to microscopical examination (Histopathology examination). Biopsy could be incisional or Excisional, Exfoliate Cytology. It is used to confirm the diagnosis of the lesion .

•ASPIRATION :-

Is the withdrawal of fluid from the lesion may aid in diagnosis . For example aspiration of pus indicates an inflammatory process like abscess or in infected cyst. Aspiration of yellow fluid may indicate cystic lesion , aspiration of blood may indicate Vascular lesion like Hemangioma , etc.... Aspiration is one of the methods used to aspirate fluid from swelling for evaluation the nature of that swelling which may assist in Diagnosis .

•LABORATORY TEST:- LIKE

1.bacteriological examination.

2.Hematological examination

3.Urine analysis (GUE)

4.Blood Chemistry &Serological examination .

5.Culture 8 sensitivity test.

All these tests or any one of these tests might be ordered to aid as in confirmingour Diagnosis.

So collection of all information taken from the history & clinical examination &accessory information (Special tests) must be evaluated, analyzed to reach thefinal Diagnosis.

•PATIENT RECORD (MEDICAL) RECORD :-

It consist of :-

1.case sheet.

2.All radiographs.

3.all investigation papers.

4.Reffering papers.

Objectives & Benefits :-

l.It assist in Diagnosis of the diseases.

2.For follow up & future checking.

3.For statistical analysis.

4.For studies & educations.

5.For Medico legal purposes.

EXTRACTION OF TEETH (EXODONT1A):-

Extraction of teeth is the most important part of minor oral surgery and the most common procedures to general dental practitioner(dentist).

Definition:-

Exodontia is a painless removal of teeth from their bony alveolar socket with relatively minimal amount of trauma to the investing or surrounding tissues, so that the wound may heal without postoperative problems.

Basically, we have two methods of extraction:-

1- Intra-alveolar extraction (forceps extraction).
2- Trans-alveolar extraction (surgical extraction).

In forceps extraction which is enough for extraction in most of cases consist of removing the tooth or root by the use of forceps or elevators or both. While in trans-alveolar extraction we dissect the tooth or root form its bony attachment by rising a flap and removal of some of the bone surrounding the roots, which are then removed by the use of elevators and/or forceps.

INDICATIONS OF Extraction:-

There are many indications for tooth extraction, if conservative treatment has either failed or is not indicated, a tooth may have to be extracted. The reasons for extraction of teeth are based on the presence of local pathology, the feasibility of restorative procedure(function of the dentition as a whole and the patient's attitude and education). Although the modern dentistry tries and takes all measures to preserve and maintain teeth in the oral cavity, there are still a variety of general indications for removing teeth.

Indications of teeth extraction.

1-Severe caries:-

This is the most common reason to remove a tooth. Badly carious teeth that are beyond restoration should be removed.

Badly carious teeth result in deterioration of the oral hygiene resulted in bad oral hygiene and bad smell in addition to that sharp edges of the carious teeth leads to repeated trauma and ulceration to the mucosa and the tongue, in addition to pain during eating and drinking. Untreated teeth with caries may end with pulpitis, periapical pathology etc...

2-Severe periodontal disease:-

Severe and extensive periodontal disease is a common reason for tooth removal, in chronic periodontitis there is excessive bone loss and mobility in the tooth. As a rough guide, loss of about half of the normal alveolar bone or extension of pockets to the bifurcation of the roots of posterior teeth and hypermobility of the teeth means that extraction of the involved tooth is necessary.

Patient with advanced periodontitis may complains of mild to severe throbbing pain in case of development of Paradontal abscess.

3- pulp pathology:-

For example acute pulpitis or chronic pulpitis, non-treatable pulpal lesion. if endodontic therapy wasn't possible or if the tooth isn’t amenable for endodontic treatment, extraction is indicated.

4- Apical pathology:-

Periapical lesions like periapical abscess, periapical granuloma, cyst. If the teeth fail to respond to all conservative treatment to resolve apical pathology due to technical reasons or other causes such teeth are indicated for extraction.

5- Orthodontic reasons:-

During the course of orthodontic treatment, tooth or teeth maybe extracted for:-

a) **Therapeutic extraction** e.g. extraction of upper first premolar for treatment of malposed upper canine and extraction of teeth to provide space for teeth alignment.

b) **Malposed teeth**: teeth which erupted out of the line of arch are difficult to clean and not amenable for orthodontic treatment are indicated for extraction.

c) **Preventive extraction**, means that during mixed dentition period (permanent and deciduous teeth), dental surgeon may extract few deciduous teeth to prevent malocclusion and all these extractions should be done after proper evaluation by specialist orthodontic.

6-Prosthetic considerations:-

Extraction of teeth are indicated for:-

1) Providing efficient dental prosthesis.
2) To provide better design for success of partial denture, few teeth maybe extracted.
3) Solitary tooth or non-strategic tooth to enable the patient to have complete denture e.g. full mouth clearance.

7- Impacted teeth.:-

Retention of un erupted teeth beyond the normal time of eruption may sometimes be responsible for: -

a) Vague facial pain.
b) Periodontal problems of the adjoining teeth.
c) Tempromandibular joint problems.
d) Bony pathology e.g. cyst (dentisgerous cyst), tumor, pathological fracture.
e) May predispose to anterior teeth crowding
f) Significant infection (pericornoitis) e.g. partially erupted third molar.

8-Supernumerary teeth

The teeth maybe impacted or malposed and such teeth may predispose to malocclusion, periodontal disturbances, facial pain, bony pathology (cyst), aesthetic problems and preventing the eruption of adjacent teeth.

9- Tooth in the line of fracture of the jaws:-

This tooth maybe extracted if:-

a) It is a source of infection at the site of the fracture.

b) The tooth itself is fractured.
c) Interfere with fracture reduction.
d) Interfere with healing of fracture.

10- Teeth in relation with pathological conditions:-

They are indicated for extraction if they are involved in:-

a) Cyst formation.
b) Neoplasm (tumor).
c) Osteomyelitis (Infection of bone).
d) Pyogenic granuloma

And the tooth interfere with complete surgical removal of the lesion the tooth should be extracted.

11-Retained roots

Retained roots may remain embedded in the bone without problems for a long period, but sometimes removal of such roots maybe necessary, for example, root maybe at the sub mucosal level producing recurrent ulceration under the denture, sometimes root fragments may be involved in initiation of bony lesions like osteomyelitis, cystic lesion or neoplasm, if such fragments are in close relation to the neurovascular bundle (e.g. inferior dental nerve of the mandible) the patient may complain of facial pain or numbness in the area supplied by that nerve. As a general rule, very small fragments maybe left alone and that patient should be kept under periodic observation, and all other root fragments are indicated for removal.

12- prior to irradiation:-(before radiotherapy)

Irradiation is one of the methods of treating oral carcinomas and teeth which cannot be kept in a sound condition should be removed before irradiation, trauma (extraction) with superadded infection will lead to development of osteoradionecrosis of the jaw bone which is unpleasant complication and difficult to be treated.

13-Focal sepsis:-

Sometimes teeth or a tooth may appear sound clinically, but on radiographic examination the tooth may appear to be considered as a foci of infection (teeth associated with periapical pathology or periodontal problems), these teeth or tooth should be extracted in certain conditions e.g. heart surgery; heart valve replacement, kidney transplant, eye Surgery

14- Aesthetics:-

Poor aesthetic, severely stained (tetracycline, fluorosis) attrition or hypoplastic (hypoplasia) of enamel or dentine and they cannot be restored may be indicated for extraction.

Contra-indications of teeth extraction.

In general, the contra-indications are subdivided into ;

1- Local contra-indications.

2- Systemic contra-indications.

Local contra-indications:- (L.C s)

There are several L.C s to extractions of indicated teeth:-

1- Acute and uncontrolled infection:-

Extraction in the presence of acute and uncontrolled infection may lead to spread of infection locally or systemically leading to many complications some of them are dangerous and life threatening (e.g. cavernous sinus thrombosis, mediastinitis, Ludwig's angina)and acute periapical abscess and facial abscess especially in medically compromised patient. In addition to that limitation of mouth opening especially in lower wisdom tooth infection.

2- Previous radiotherapy:-

Previous therapeutic radiation in oral and maxillofacial region for treatment of cancer lead to fibrosis and decreased vascularity of the tissue or area of extraction and end with a condition in the bone called osteoradionecrosis

3- Teeth located within area of tumor:-

Especially in vascular lesion or malignant tumor should not be extracted because extraction may lead to dissemination of the tumor, unhealed socket and postoperative complications, for example, bleeding postoperatively and intraoperative.

Systemic contra-indications:-

Systemic contra-indications preclude extraction because the patient's systemic health is such that the ability to withstand the surgical work may be compromised. So extraction should be postponed until the severity of the problem has been resolved and maybe arranged after consultation with the physician to perform extraction safely without complications so caution is advised in the following conditions:-

1- Severe uncontrolled metabolic disease, e.g. uncontrolled diabetes, end-stage renal disease.

2- Uncontrolled leukaemia and lymphomas.

3- Sever uncontrolled cardiac disease, e.g. myocardial infarction, unstable angina pectoris, dysrhythmias.

4- Sever uncontrolled hypertension.

5-Pregnancy.

6- Bleeding disorder e.g. haemophilia, platelet disorder, patient on anticoagulants.

7- Patients who take a variety of medicationse.g. patient on steroid and immunosuppressive drugs, cancer and chemotherapy.

8- Uncontrolled epilepsy.

Pre-extraction evaluation:-

P.E.E is very valuable and necessary for successful

extraction practice. Hurry and inadequate P.E.E of the case may lead to many embarrassing intra-operative problem for the operator, in addition to the postoperative problems to the patient,

P.E.E include:-

1- Clinical preoperative evaluation.

2- Radiological evaluation

1-Clinical P.E.E also includes:-

a) **General evaluation.**

b) **Local evaluation.**

a-General evaluation includes:-

- General impression of the patient.
- History of general diseases, Nervousness and orientation
- General oral hygiene.
- Gingival inflammation, calculus, neglected mouth.

b-Local factors evaluation Includes:-

- Clinical examination of the accused tooth.
- Adjoining structures.
- Access to the tooth. This includes the mouth opening, location of the tooth (e.g. buccally malposed, in standing) may present difficulty in positioning the dental forceps for extraction, so you may remove such a tooth surgically.
- Tooth mobility: The mobility of the tooth to be extractedshould be assessed preoperatively, greater than normal mobility is frequently seen with severe periodontal disease, but sometimes it maybe because of the underlying pathology like neoplasm

- **Condition of the tooth:- e.g.**

a) Carious destruction.

b) The presence of large restoration.

c) Presence or absence of the adjoining teeth.

d) Non-vital tooth.

e) State of the supporting tissue.

f) Shape, position, long axis and size of the crown.

g) Attrition.

Good P.O.E resulted from correction of data collected from history, clinical examination, radiographs and laboratory aids in addition to that P.E.E need good knowledge and experience in the basic sciences e.g. anatomy, physiology, pathology.

In general, P.O.E may help you in:-

a) Determine the method of extraction and type of anaesthesia.
b) Reduce the time spend for extraction.
c) Reduce the intra and post-operative crisis and complications.

2- Radiological evaluation:-

Preoperative clinical assessment maybe supplemented some times by preoperative radiographs, and the positive indications for preoperative radiograph are:-

1) History of difficult or unsuccessful extraction.
2) Crown with extensive caries, large restorations, non-vital tooth when diagnosis is not certain and tooth is malposed.
3) A tooth which is abnormally resistant to forceps extraction.
4) If after clinical examination you decide to remove the tooth surgically.
5) Any tooth which is in close relation to important or vital structures like neurovascular canal, maxillary sinus, mental nerve, nasal cavity.
6) Attrition teeth in elderly patient (maybe associated with hypercementosis).
7) If a tooth is partially erupted or completely unerupted or retained root.
8) Any tooth which has been subjected to trauma,

fracture of the root and/or alveolar bone maybe

present.

9) An isolated maxillary molar especially if it is unopposed and over erupted. The bony support of such a tooth is often weakened by the presence of maxillary sinus and this may predispose to certain of oro-anrtal communication or fracture of the maxillary tuberosity.

10) Whenever, underlying bony pathology is suspected e.g. cystic lesion, tumor.

11)Any systemic condition which may predispose to dental or alveolar abnormality like:-

a) Osteitis deformans (Paget's disease), in which the roots are hypercementosed andankylosed leading to difficult extraction, infection of the socket.
b) Cleido-cranial dysostosis, for pseudo-anodontia (multiple impactions, hooked roots occur, supernumerary teeth).
c) Patient who have received therapeutic irradiation to the jaw which may have to predispose to osteoradionecrosis.
d) Osteopetrosis (marble bone disease), which cause extraction very difficult and predispose to chronic osteomyelitis.

- **A good radiograph and careful interpretation may give or aid the operator to many factors that may cause difficult extraction, e.g.:-**
  1- Abnormal number and shape of roots.
  2- An unfavourable root pattern.
  3- Caries extending to the root mass.
  4- Fracture or resorption of the root.
  5- Hypercementosis of roots.
  6- Ankylosis (there is no space in periodontal ligament), and sclerosis of the bone.
  7- Gemination (the development of two teeth from one bud).
  8- Impaction.
  9- Bony sclerosis and pathological lesions.

Also careful interpretation of the radiograph may also reveal or show the possibility of the following complications:-

1) Involvement of, and damage to inferior dental nerve and mental nerve e.g. on extraction of impacted lowerthird molar
2) The creation of oro-anrtal fistula or or-nasal communication.
3) The retention of intra-bony pathology e.g. cyst.
4) The displacement of root or tooth into maxillary sinus.
5) Fracture of maxillary tuberosity.

Oral Surgery
Exodontia

General Arrangement or considerations for Extraction

1. Light:

Dentist or oral surgeon work in a limited accessible area (oral cavity) , then he is going to do his work (extraction site) in a relatively inaccessible area in addition to that the shadow of the hand & the instrument he use , so good illumination of the operative field is very important & necessary to perform your work efficiently & safely .

2. Position of the operator:

When extracting any tooth in the oral cavity except the right mandibular molars, premolars, canines, the operator stand on the right-hand side of the patient, in other words in front of the patient. For removal of right mandibular teeth (molars, premolars & canines) the operator stands behind the patient in order to achieve the good working position. For left-handed dentists, he should stand behind the patient when extracting lower left teeth.

3.Position of the patient:

Correct position of the patient is very important to avoid any Occupational **Postural** problems. The patient is seated comfortably in the dental chair with head rest adjusted to fit the shape of the neck & support of the head. Patients undergoing extraction of maxillary teeth should be positioned where the dental chair back support reclined so that the upper arch lies at an angle of 60 degrees to the floor (inclination of about 60 degrees). For extraction of mandibular teeth, the dental chair should be reclined slightly less (about 30-45 degrees) so that when the patient opens his mouth the occlusal plane parallel to the floor.

4.Height of the dental chair:

This is very important. If the site of the operation is either too high too low in relation to the operator he works in mechanical disadvantage & in tiring & uncomfortable position.

When maxillary teeth are being extracted the chair should be adjusted so that the site of the operation is levelled at the operators' elbow. During extraction of mandibular teeth the chair height should be adjusted so that the tooth to be extracted about 16cm (6 inches) below the level of the operators elbow. When the operator is standing behind the patient the chair should be lowered sufficiently to enable him to have a clear view of the field of the operation.

* The dental surgeon should attempt to have: -

1. Quick, efficient, unhurried & methodical approach to his work.
2. Sympathetic encouragement, to gain confidence & cooperation of the patient
3. You have to avoid to increase the misgivings of the patient by displaying instruments.
4. The dressing of operator should be suitable.
5. Try to do your work in a septic environment reduce the chance of contamination by using gloves, mask, trimming of the nails, tied the hair,…etc.

Instruments used in simple uncomplicated teeth extraction includes the followings:

- Diagnostic instruments :(Dental mirror, probe, Tweezer in kidney dish).
- Dental forceps.
- Dental elevators.

The instrument required for extraction are selected, sterilized & placed in a sterile dish at the side of the patient.

Dental forceps:

The most widely used instrument employed in extraction of the teeth are dental forceps. Dental forceps are designed in a large number of patterns & configuration which adapt to different teeth & techniques used to extract teeth. It is composed of three parts

1. Handle.
2. Hinge joint.
3. Blades (Beaks)

The more desirable properties & requirements of dental forceps

1. That must be made of strong metal so that it can resist the forces exerted during extraction movements.

2. It must be constructed of non -corrosive metal so that it can resist rusting during sterilization by boiling water and autoclaving.

3. The handles of forceps should be serrated in order to prevent slippage & good grip during extraction movements. The handles must of such design so that they can give the operator a chance to use maximum leverage force. Also it must be of suitable shape & size so that they can applied to area of extraction without injury to the opposing teeth & surrounding tissues.

4. The blades

The blades (beaks) are the source of the greatest variation among forceps . The beaks is designed to adapt to the root of the teeth at the junction of the crown & root & to adapt to the root surface & not to the crown . Also beaks designed for single rooted , two rooted , & three rooted teeth so that the tips of the blades will adapt closely to various root formation decreasing the chance for the root fracture . Other variation is the width of the beaks , some forceps are narrow (fine) & others are wider (heavy) . The edges of the blades is sharp enough so they cut through the periodontal fibres without causing injury to the gingiva . The blades should fit the surface of the extracted tooth . The space between the blades should be enough to accommodate the crown of the extracted tooth without making crushing of the crown . The design of the blades should be suitable to be applied to the surface of the root of the tooth so that the blades are parallel to the long axis of the tooth to be extracted.

5. The hinge joint:

Is that part of the dental forceps which transfers & concentrate the force applied to the handle of the dental forceps & then to the beaks . It is the part which connecting the handle to the beaks . The joint must be

. Bevelled so that it will not cause pinching of the lip or injury to the lip .

. Heavy & strong allow free movement without rocking .

• Lubricated with oil after sterilization to prevent rusting which lead to limitation & difficulty during application of the forceps blade to the tooth to be extracted .

Oral surgery

TYPES OF DENTAL FORCEPS

THE FORCEPS FOR UPPER TEETH

1- The upper straight forceps:-

The blades, joint and handle are in one long straight line. We have two types, one with broad blades that is we call heavy blades and this is used for extraction of upper central incisors and upper canines, left and right.

The second type of straight forceps has narrow blades or we call it fine blades for extraction of upper lateral incisors (left and right) and upper anterior retained roots.

2- The upper premolar forceps:-

Here we have two bends in the design of the forceps, one where the beaks (blades) bend in relation to the joint of the forceps to apply the forceps parallel to long axis of premolar, the 2nd bend or curvature is of the handle to avoid injury to the lower lip and apposing teeth (mandibular). The upper premolars teeth has either one root or two roots (one buccal and one palatal), so there is no difference in the anatomy of the tooth root of the premolar on the buccal and palatal surface so the two blades of the premolars forceps are mirror image to each other.

3- The upper molar forceps (full crown upper molar forceps):-

Since upper molar teeth have three roots, two buccal and one palatal, the blade of palatal side is round to conform or fit on palatal root, while blades on buccal has pointed tip or projection so it can enter or fit the bifurcation between the two buccal roots (mesial and distal) on the buccal side of the tooth. So we have two forceps; one for the right molars and one for the left molars and these forceps also double bend for the same requirement as mentioned for premolar teeth.

The Bayonet forceps, the blades of the forceps are off set to the long axis of the handles, used for extraction of upper 3rd molars right and left. In addition, there is another bayonet with fine curved blades for extraction of upper posterior roots.

Bayonet forceps for crowned maxillary 3rd molars

THE FORCEPS OF LOWER TEETH;-

Here we have the long axis of the blades is in right angle to the long axis of the handle so the blades can be applied apical to the cemento-enamel junction (on the root) of the tooth surface parallel to the long axis of the tooth and the handle not to cause injury to the upper lip. The forceps for the lower teeth are:-

1- Forceps for extraction of lower central and lateral incisors and canine:-

We have fine blades for extraction of the lower central and lateral incisors and lower anterior retained roots which have fine roots with flattened sides (mesiodistally) and heavy blades used for extraction of canines.

2- Premolar forceps:-

Because the bucco-lingual width of the crown in the premolar teeth is larger than that of lower incisors and canines we use forceps with heavy blades but partially away from each other when close to accommodate the crowns of these teeth without crushing for the crown.

Lower premolar tooth forceps Lower molar tooth forceps

3- Full crown lower molar forceps: -

Since the lower molar teeth have two roots, one mesial and one distal root so the buccal and lingual blades of the forceps designed with projected tapered tip to fit the bifurcation of these teeth on the buccal and lingual sides, so the buccal and lingual blades are identical so the same forceps can be used on the right and left sides on opposite to that in upper molar teeth.

In addition to that we have two Bayonet forceps for lower 3rd molars; one for left side and the other for right side.

Mechanical principles of extraction:-

The removal of teeth from the alveolar process employs the use of the following mechanical principles:-

I- Expansion of the bony socket:-

This is achieved by using the tooth itself as a dilating instrument, and this is the most important factor in forceps extraction, and this principle need:-

1- Sufficient tooth substance be present to be firmly grasped by the forceps.

2- The root pattern of the tooth in such that it is possible to dilate the socket to permit the complete dislocation of the tooth from its socket, e.g. dilacerated, divergent, converge roots.

3- Nature of the bone, elastic bone especially in young patients is maximal and decreased with age, older patients usually have denser, more highly calcified bone that is less likely to provide adequate expansion during extraction of the teeth.

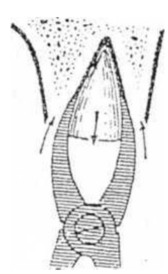
4- Thickness of the bone. Thick bone expansion is less likely to occur by using normal force.

II- The use of a lever and fulcrum

This is used to force a tooth or root out of the socket along the path of least resistance and the principle is the basic factor governing the use of elevators to extract teeth or roots

III-The insertion of a wedge or wedges:-

Between the tooth-root and the bony socket wall, thus causing the tooth to rise in its socket and this explains why some conically rooted mandibular premolar and molars sometimes shoot out of their socket when forceps blades are applied to it.



Physics forceps:

The Physics Forceps uses first-class lever mechanics. One handle of the device is connected to a “bumper,” which acts as a fulcrum during the extraction and stabilizes the beak during wrist movements. The beak of the extractor is positioned most often on the lingual or palatal root of the tooth and into the gingival sulcus

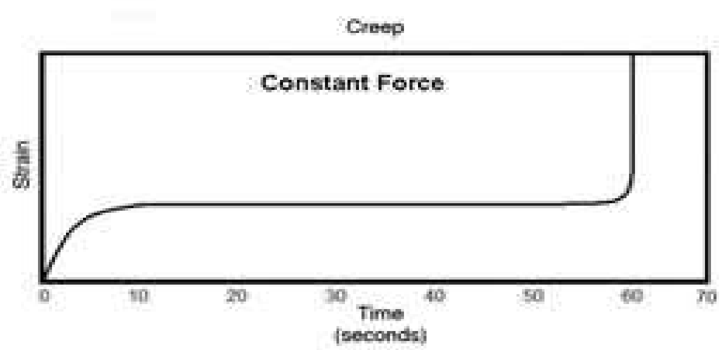
Standard Physics forceps set.

Bumper guards

The bumper is most often placed on the facial aspect of the dental alveolus, typically at the mucogingival junction. Unlike conventional forceps, only one point of contact is made on the tooth being extracted. No squeezing pressure is applied to the handles or to the tooth. Instead, the handles (once in position) are rotated as one unit for a few degrees, and then the action is stopped for approximately 1 minute. The torque force generated on the tooth, periodontal ligament, and bone is related to the length of the handle to the bumper (8 cm), divided by the distance from the bumper to the forceps beak (1 cm). As a result, a force on the handle connected to the bumper will increase the force on the tooth, periodontal ligament, and bone by 8 times. No force is required to be placed on the beak, which is only on the lingual aspect of the tooth root. Therefore, the tooth does not split, crush or fracture.

Moment of force in physics represents the magnitude of force applied to a rotational system at a distance from the axis of rotation. The principle of moment is derived from Archimedes' operating principles of the lever and is defined as M=rF, where "F" is the applied force and "r" is the distance from the applied force to the object. This is referred to as the moment arm. The length of the moment arm (or lever arm) is the key to the operation of the lever, pulley, and most other simple machines capable of generating mechanical advantage. This means that if the force applied to generate work cannot be increased, it is still possible to gain a greater amount of work by increasing the moment arm of the lever.

"Creep" is a phenomenon whereby a material continues to change shape over time under a constant load. In a tooth extraction, creep may occur in bone and the periodontal ligament. Reilly established the creep curve of bone, whereby under a constant load of 60 Mpa, the bone over time changes shape (strain) in 3 different stages



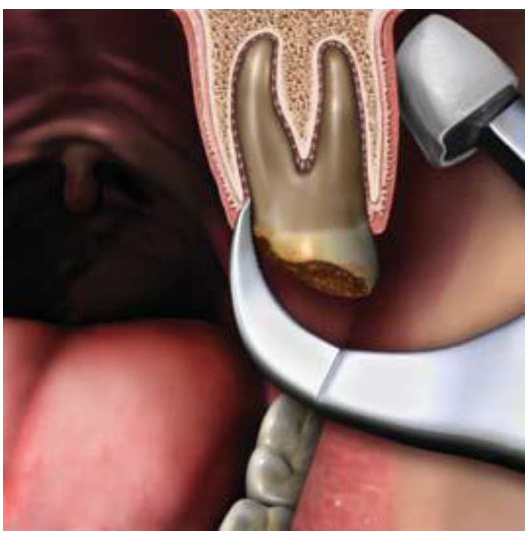
A creep curve demonstrates that a constant force applied to bone or a periodontal ligament results in initial changes in shape, with a prolonged period (horizontal line) necessary before the material fractures or releases (the vertical aspect of the line on the right at 60 seconds).

The majority of bone changes occur within the first minute, whereby the strain of bone (the change of length divided by the original length) is modified. The higher the force that is applied, the greater the deformation of the bone. This process allows the tooth socket to expand and permits the tooth to exit the socket.

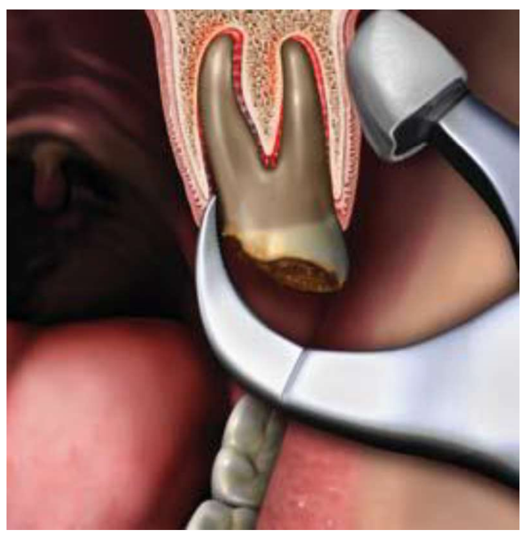
A secondary creep action occurs over time and allows the bone to further deform when the force is applied during a 1- to 5-minute period. The longer the time, the greater the deformation; however, it expresses only a 10% to 20% difference compared to the initial one-minute strain. Eventually, the third phase of the curve causes the bone to fracture if the load is applied over a long time frame, representing creep rupture. A similar phenomenon occurs in the periodontal complex.

Mechanical forces shift lateral force to a tooth, causing primary movement to the periodontal ligament and space. A greater force overtime causes a slight additional tooth movement. Therefore, the creep of the periodontal complex is similar to the creep of the bone, whereby the constant load weakens the periodontal ligament. Thus, a constant load on the tooth over time increases the tooth socket dimension and decreases the strength of the periodontal complex.

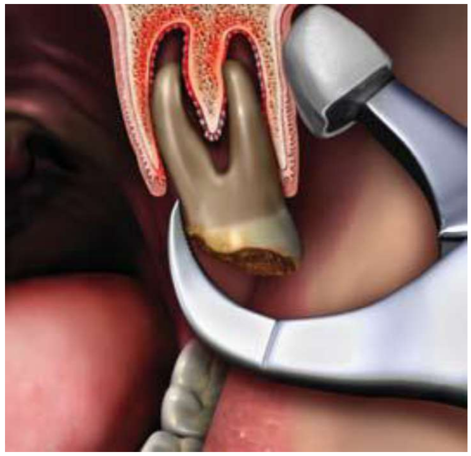
Once creep has expanded and weakened the periodontal ligament and bone, the handle of the extraction device may be slowly rotated another few degrees for 10 to 30 seconds. This action contributes to the creep rupture of the ligament and usually elevates the tooth a few millimeters from the socket. At this point the tooth is loose and ready to be removed from the socket using any pincer-like device, i.e, pickups, extraction forceps, or hemostats.



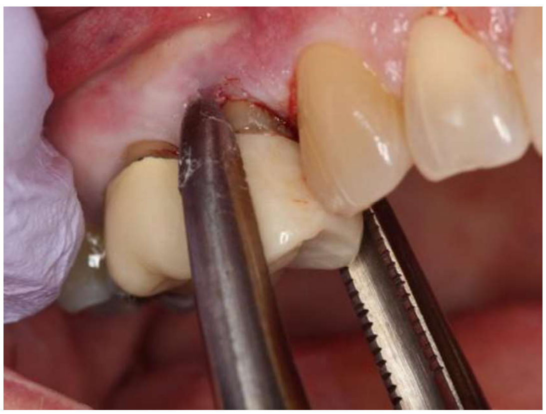
The Physics Forceps is in position, and constant pressure is applied.



Creep is expanding the bone and rupturing the periodontal ligament.



The tooth is rotated slightly and elevated from the socket.



The tooth was delivered from the socket with a pincer-type instrument as the Physics Forceps are not designed to remove the tooth all the way from the socket but simply to elevate the tooth or release the periodontal ligaments.

The tooth was easily removed from the socket.

The extraction of a tooth using the Physics Forceps is similar to the removal of a nail from wood using a hammer versus a pair of pliers (Figures below). The handle of the hammer is a lever, and the beaks of the hammer's claw fit under the head of a nail. The hammer's head acts as a fulcrum. A rotational force applied to the hammer handle magnifies the force by the length of the handle, and the nail is elevated from the wood. Unlike a nail in wood with parallel sides and friction along its full length, a tooth is tapered. After being elevated a few millimeters, the periodontal ligament fibers are broken and the tooth may then be easily removed without additional rotational force. This is important to note, since further rotational force on the tooth may fracture the facial plate of bone.

A “traditional” dental forceps removes a tooth similar to how a pair of pliers removes a nail.

A claw hammer uses class I lever mechanics, with the handle one lever, the head of the hammer as the fulcrum, and the claw as the short lever applied to the nail. The Physics Forceps uses a similar action to remove a tooth.

Stress is the internal distribution of force per unit area that balances and reacts to external loads applied to a body. Stress can be broken down into its shear, tensile, and compressive components. Materials in general are weakest to shear forces and strongest to compressive loads. For example, bone is strongest to force in compression, 30% weaker to tension, and 65% weaker to shear forces ***(Reilly DT., 1975)***. When a rotating force is applied to the Physics forceps on a tooth, the stress to the tooth and the periodontal complex is a shear component of force. The force applied to the gums and bone by the bumper of the Physics Forceps is over a greater surface area and is a compressive force, thus bracing the buccal bone. This permits the lingual plate to expand more and protects the facial plate from fracture.

The instrument is uniquely designed to allow tension to be placed of the periodontal ligament and to achieve excellent leverage. This tension results in the physiologic release of an enzyme by the body that breaks down the periodontal ligament over a short period of time which is called hyaluronidase (hyluronate glycanohydrolase). This is an enzyme that catalyzes the hydrolysis of the interstitial barrier, hyaluronan (hyaluronic acid), which is the cement substance (extracellular matrix) of all human tissues ***(KUMAR DM., 2015)***. This process may take 20 seconds or up to 4 minutes depending on the tooth and surrounding bone structure of the patient. The clinician must be patient, as the technique will feel like nothing is happening since none of the usual operator movements are employed. With the breakdown of the periodontal ligament, the tooth will release or "pop" out of the socket in an upward and outward motion, mirroring the arch form of the head of the instrument. This innovative instrument allows tooth dislodgment with little or no pressure, simply utilizing leverage. The handles are never squeezed like a conventional forceps; rather they are held lightly in the hand, and the wrist is rotated to simply create tension on the palatal aspect of the root. There is no forearm, bicep, or shoulder pressure used. The handles simply allow the beak to engage the root structure without slipping off. During a short time of constant tension, the root will disengage or pop from the socket incisally and facially. Although the facial bone may expand slightly, the movement of the tooth out of the socket is not straight facial, but rather up and out of the socket. This allows for maintenance of the facial plate of bone, when the instrument is used correctly. One of the biggest misconceptions of this innovative technique is that it is a "forceps" as the names implies, when it fact it is a lingual elevator ***(Kosinski T, Golden R., 2015).***

Atraumatic extraction with the Physics Forceps (Golden Dental Solutions). Using the proper hand position and hold are a must when utilizing the Physics Forceps technique.






Oral Surgery

Elevators:-

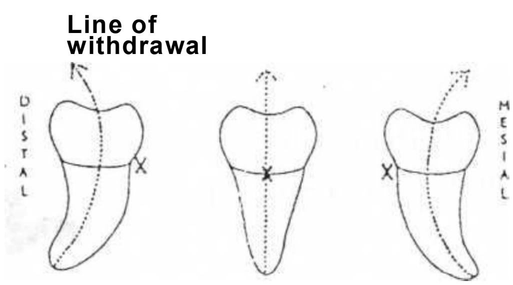
Are exo-levers, instrument designed to elevate or luxate the teeth or roots from their bony socket in close or surgical method of extraction to force a tooth or root along the line of withdrawal.

Line of withdrawal:-

Is the path along which the tooth or root will move out of its socket when minimal force is applied to it, and this line is primarily determined by root pattern (long axis of the tooth).

Point of application:-

Is the site on the root at which force must be applied to effect delivery, it is determined by the line- of withdrawal. We have buccal point of application, distal point of application, and mesial point of application.



X point of applic ation

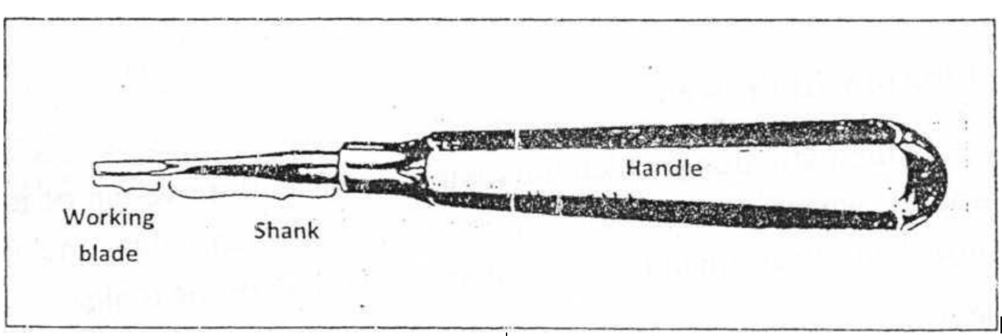
Parts of elevators:-

All elevators have the following parts:-

I-Handle: This maybe a continuation of shank or at right angle to it.

II- **Shank**.

III- Blade: This part engages the crown or root and transmit force to the tooth, bone or both. The working side of the blade is either concave or flat.



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Mechanical principles of uses of elevators:-

The work principles as applied to the use of elevators maybe that of:-

1- Lever principle.
2- Wedge principle.
3- Wheel and axle principle.
4- Combination of these principles.

Clinical uses of elevators:-

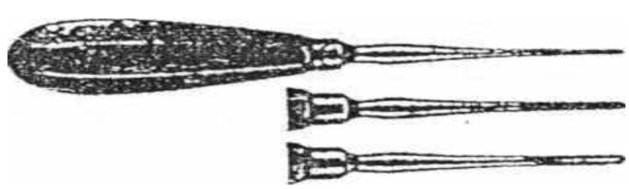
1- Elevators are used to luxate and remove teeth which cannot be engaged or grasped by the beaks of forceps (e.g. impacted teeth, malposed teeth), also badly carious teeth, teeth with heavy filling.
2- To remove old roots and fractured roots and sectioned roots.
3- To loosen teeth prior to use of forceps.
4- To split teeth which have had grooves cut in them, as in separation of roots.
5- To remove small amounts of bone to create point of . application for the beaks of forceps, or removal of interseptal bone.
6- Any tooth resisting normal extraction force by extracting forceps.

Elevators commonly used:-

There are so many elevators available but few are widely used because of their efficiency.

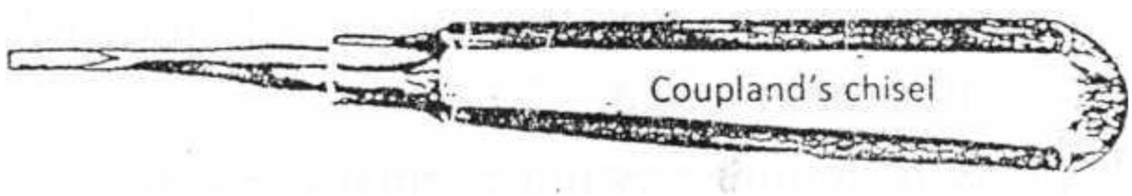
1-straight elevator:-

Elevator in which the blade, shank, and the handle are straight. The working blade or end is blind and round, there are many types and sizes of straight elevators,



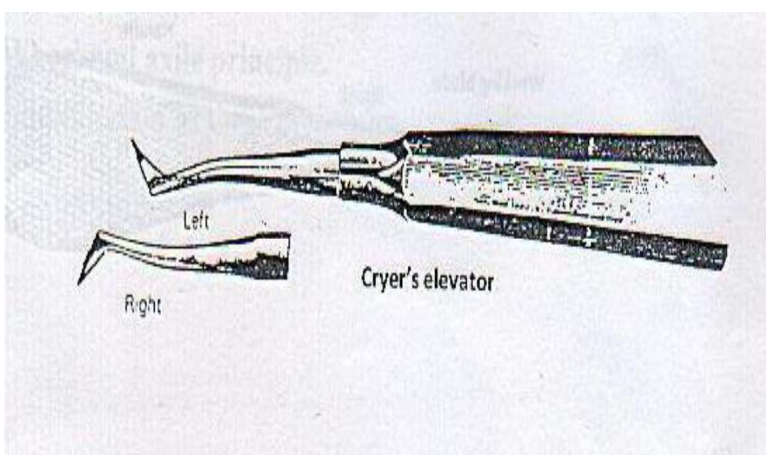
2-Coupland's chisel (elevator);-

It is similar to straight elevator but the working end is sharp and straight cut, used for chiselling of bone to create point of application or to split of teeth. It's of different sizes, size 1, size 2, size 3. Depending on the width of the working end.



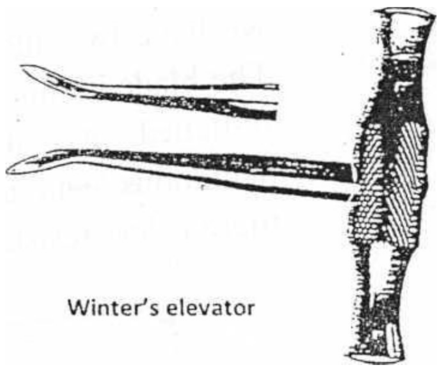
3-Cryer's elevators:-

In this type the working blades are sharp, pointed and triangular in shape just like a claw, forming an angle with the shank of the elevators. These are pair instrument mesial and distal (right & left) designed to fit the root surface on mesial and distal surface. It's mostly used for removal of retained root of the lower molar and for elevation for impacted teeth after surgical exposure of the bifurcation of the tooth.



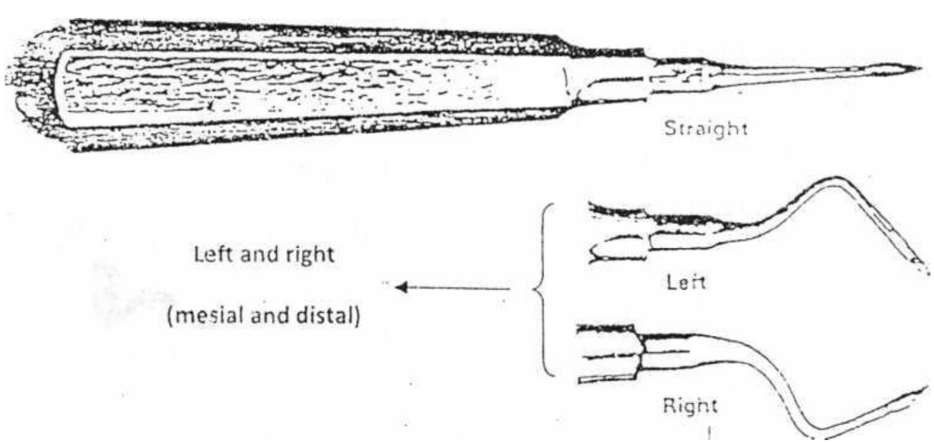
4- W inter's elevator:-

In which the working end is the same that of Cryer's elevator but the handle is in right angle to the shank so it is called winter's (T-bar) cross-bar handle elevator. Winter's elevators are very powerful and great force maybe applied or generated (sufficient to fracture the mandible) so the use of this elevator should be with great care to avoid fracture of the jaw.



5- Apexo elevators:-

The working blade is long, the margins are sharp, we have 3 Apexo, 2 angled and 1 straight (mesial, distal, straight). The blade forming an angle with the shank, this elevator is used mainly for removal of apical fragments of root deeply present in the socket of the lower jaw especially morals. We push it between the socket and the root to loosen the fractured tip and remove it from the socket.



6-War-wick James elevators:-

It is a light duty elevator. It's like Cryer's elevator, also we have two angled (mesial and distal) and one straight. The blade is short and the end is rounded and the handle is flattened, it's used for extraction of retained roots, deciduous teeth, anterior lower teeth extraction, and where there is less resistance area. e.g. extraction of upper wisdom tooth.

Guiding principles for use of elevators:-

The following rules should be observed when using elevators in general:-

1-Never use an adjacent tooth as a fulcrum, unless that tooth to be extracted itself in the same visit, and the fulcrum should always be bony one (alveolar bone).

2- An elevator should always be supported to avoid slippage and injury to the patient.

3- Avoid the use of excessive force if the tooth/root is resist luxation, by gentle rotation, then stop, look for the obstruction for elevation and deal with it.

4- The direction of force should be such that the roots are not directed toward major structures such as the maxillary antrum.

5-An elevator should never be used "blindly" in the socket

.

6- If an application point is not present, then this should be created by careful removal of bone.

7-Elevators should always be sterile and sharp.

8-- The sharp edges of the working blades are placed between the alveolus and the root surface and gently rotated apically along the long axis of the elevator to luxate or displacing the tooth or root.

Complications of use of elevators:-

Although elevators are very useful instrument for facilitating extraction of teeth, but misuse or miss-judgement may lead to some complications, part of it may be serious:-

1- Injury to the soft tissues, like injury to the tongue, floor of the mouth, soft and hard palate, caused by slipping of elevator during its use
.

2- Wrong application of force or excessive force may lead to fracture e of jaw especially the lower jaw at the angle of the mandible, also excessive force may lead to crushing of the alveolar bone and fragmentation

3- Fracture of maxillary tuberosity especially in extraction of upper third molars.

4- Uncontrolled force may lead to displacement of roots into maxillary sinus, infratemporal fossa, buccal soft tissue, submandibular space or inferior dental canal.

5- Use of elevator in periapical area of abscessed tooth may cause spread of infection to the surrounding tissue.

6- Tip of instrument (working blade) may be fractured and remain In the socket causing postoperative infection or delay healing, so always check the tip of instrument after use.

So most problems with elevators arise from:-

a) Miss-judgement of amount of force exerted.
b) Improper positioning of the elevators.

INSTRUMENTS FOR BASIC ORAL SURGERY

These lectures will introduce the basic and main instruments required to perform routine oral surgical procedures ,these instruments are used for many purposes including both soft and hard tissues (e.g./ Bone , dental procedures):-

The main instruments include the followings:-

1- Instruments to incise the tissues .
2- Instruments for elevating mucoperiosteum.
3- Instruments for controlling haemorrhage .
4- Instruments to grasp tissues.
5- Instruments for removing bone includes:-
a. Rounger forceps(bone cutter, bone nibbler)
b. Chisel and mallet.
c. Bone file.
d. Burs and hand piece.
e. Instruments to remove soft tissues from bony defects

6-Instruments for suturing mucosa:
a. Needle holder.
b. Needle.
c. Suture material.
d. Scissors.

7- Instruments for retraction of soft tissues.
8. Instruments for irrigation and for providing suction.

1-Instruments to incise tissues:

These instruments used to do incision in the soft tissues and this procedure needs scalpel and scalpel handle, the most commonly used handle is number 3 and number 7 . The tip of the scalpel handle is prepared to receive a variety of different scalpel blades that can be inserted into a slotted receiver, the most commonly used scalpel blade for intra oral incisions is number 15 , its relatively small and can be used to make incision around teeth and through mucoperiosteum.

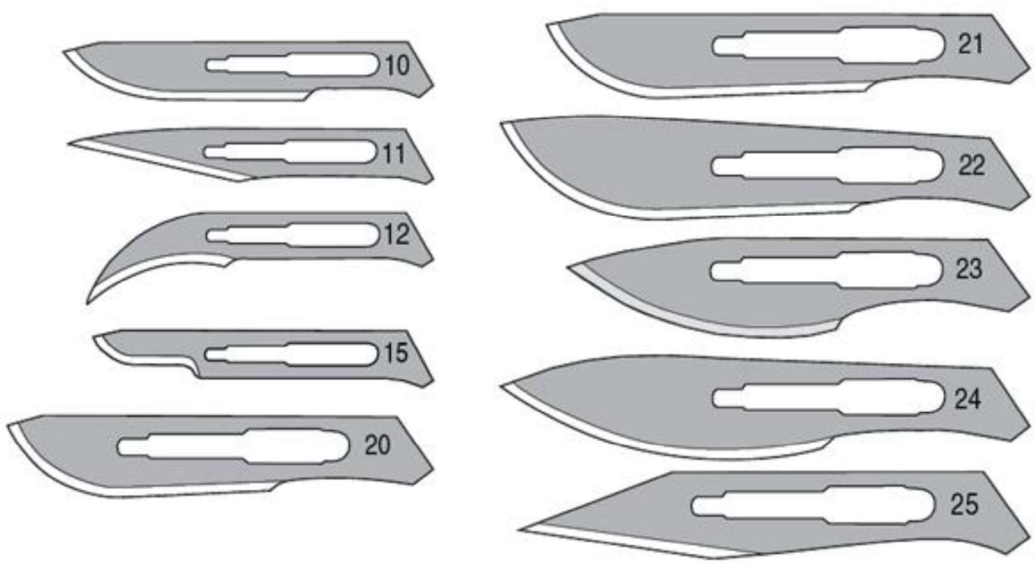
Another scalpel used to make incisions on the skin extra orally is number 10..

Other commonly used blades for intra oral surgery are number 11 and 12.

Blade no. 11 mostly used for making stab incision such as for incising abscess , its pointed triangular in shape , the hooked no. 12 blade is useful for mucogingival procedures especially in the posterior area of the oral cavity , for example maxillary tuberosity or posterior aspects of the upper molar region on the buccal or lingual aspects.

Scalpel handle no.3

Dental scalpel blades:



2-Instruments for mucoperiosteum elevation

After an incision is made through the mucoperiosteum (mucosa +sub mucosa+ periosteum=mucoperiosteum)

The mucosa and periosteum should be reflected from the underlying bone with periosteal elevator . The instrument that is mostly used in our department is Howarth periosteal elevator, this instrument has a sharp pointed end and a broad flat round end .

The broad flat round end is inserted into the incision beneath the cut edge of the periosteum to elevate or strip it off the bone , and the sharp end of the elevator used for detachment of muscle insertion or sometimes to complete the incision done by the blade if there is some resistance for the elevation by the flap, many types and sizes of the periosteal elevators are available like Mitchell trimmer

Mitchell trimmer is useful in separating through the fibres from around the crown of the unerupted teeth and the neck of erupted teeth.

The periosteal elevator can be used to reflect soft tissues by push stroke in which the broad end of the instrument slides underneath the flap separating through the periosteum from the underlying bone and sometimes periosteal elevators are used by a pull stroke or scrap stroke in special locations.

Howarth periosteal elevator:

Mitchell trimmer:

3-Instruments for controlling haemorrhage:

When incision is made through the tissues , small vessels may be cut leading to bleeding , to control this bleeding we have instruments Which aid in the stoppage of bleeding called haemostat or artery forceps , this comes in a variety of shapes and sizes , it’s a delicate instrument which may have straight or curved beaks , this instrument has a locking device assisting in the clamping of the blood vessels

In addition to its use as an instrument for controlling bleeding its used in oral surgery to evacuate pus from abscesses by inserting the haemostat inside the abscess after an incision is made , also haemostat may be useful in grasping tissues from the tooth socket and pick up small root tips , pieces of calculus, fragments of amalgam or any other small particles that have been dropped in the mouth or wound area.

Haemostat:

4-Instruments used to grasp tissues:

Here we have :

A-Toothed -tissue forceps.

B- Allis tissue forceps.

These instruments used to hold and stabilize flap during suturing to assist in passing needle through the mucoperiosteal flap , also these instruments, used to hold tissues or grasping tissues in some types of surgical procedures .e.g.\ Taking biopsy.

It should be used gently to prevent crushing of the tissues especially in the case of Allis tissue forceps

Allis tissue forceps:

<u>5-Instruments for removing bone includes</u>:

A-rounger forceps (bone cutter , bone nibbler)

B-chisel and mallet.

C-surgical burs and hand piece.

D-bone file.

E-instruments used to remove soft tissues from bony defects

sometimes we need to remove bone to gain access into a lesion like intra-bony cystic lesion, or bony biopsy or to correct an existing bony defect or abnormal bony exostosis or even sometimes to remove a retained root or a sharp bony projection ..etc.

So we have many tools and instruments to perform such a duty. the use of any one of these instruments depends on the case and preference of the operator, so we have many modalities to perform bone removal.

<u>A- Bone rounger</u>:

It's also called (bone nibbler) or (bone cutter) it's used to cut bone quickly and efficiently this instrument has sharp blades that are squeezed together by the handles cutting or pinching through the bone , it also has a leaf spring or spring loaded handles to control the opening of the blades, some of these are side cutting others are end cutting.

End cutting types are used to cut bone in less accessible areas like inside the tooth socket , these instruments are very useful for trimming of sharp bony projections or removing of a thin plate of compact bone during saucerization of bone cavity.

Bone Cutter (rounger forceps):

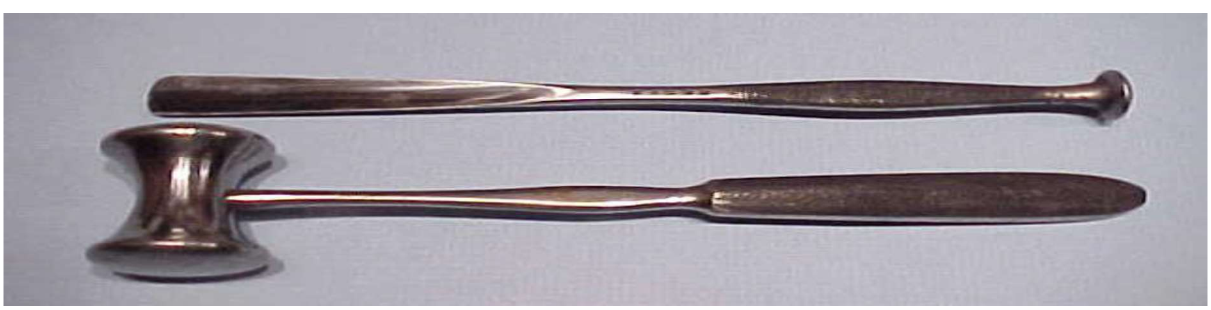
B-Chisel and mallet:

This is another method for removing bone especially when large piece of bone need to be removed ,we have many sizes and lengths of chisels ..chisel have a sharp monobeveled working end used to cut bone , bibevelede called ***osteotome*** , may be used for splitting bone or teeth.

cutting of bone by chisel need to be used by applying force and by the use of mallet,. A mallet with a nylon cover or facing will exert less shock to the patient, also its less noisy ..

Removal of bone using chisel and mallet mostly done under general anaesthesia.

Chisel & Mallet:



C- Surgical burs and hand piece:

It's a useful method and commonly used to remove bone by rotary means (hand piece) under local anaesthesia, burs are either used to remove bone or to cut a window in the bone , it's also useful in dividing teeth , we have dental steel burs and tungsten carbide burs carbide burs cut bone more efficiently than steel burs, these burs are-either-round or fissure burs...

* large diameter bone burs may be used for removal of wide area of bone or for smoothening of the margins of bony defect, in most cases the use of either size 6 or 8 bone burs will enable the production of deep narrow slots in the bone ,burs can be used with angle or straight hand piece which should have an accepted speed and torque to remove bone efficiently and should always be used with irrigation by distilled water or saline to prevent damage to the bone by heat generation during cutting.

*when large amount of bone need to be removed , large bur that resembles an acrylic bur is used .

High speed turbine which is used in restorative dentistry must not be used because the air exhausted into the wound may produce tissue emphysema.

Dental surgical burs:

D- *Bone file:*

This instrument is used for smoothening of bone before suturing of the flap , it's usually double ended instrument with a small and large ends , the working end of the bone file have grooves or slots to work in a ***pull stroke*** only, bone file is not used to remove bone

its used only for final smoothening ..
Notice that its used in a pull stroke one direction only , if pushing movement is used it will lead to crushing of bone and this should be avoided.

Bone file :

E-Instruments to remove soft tissues from bony defects:

It is called Curette : it's just like excavator , angled , double ended ,of different sizes,the working end is like a spoon, round -oval in shape, its mostly used to remove soft tissues from a bony cavity like :periapical granuloma or cystic lesion from the bone, it also may be used to remove foreign bodies or small pieces or spicules from the bony socket.

Curette:

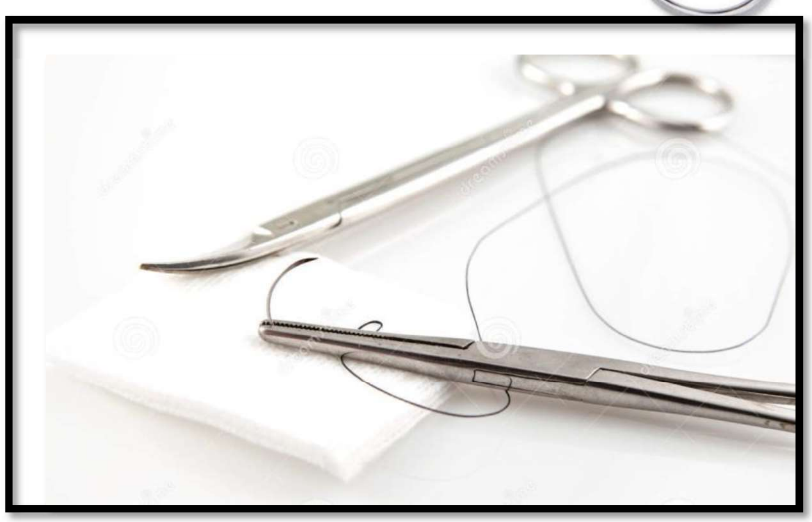
INSTRUMENTS FOR BASIC ORAL SURGERY II

6-Instruments for suturing:

At the end of the procedure the mucoperiosteal flap must be returned back to its original place or position by sutures, the instruments used to do any suturing may include the followings:

A-Needle holder

the needle holder is an instrument with a locking handle device the handle is long and the beaks are short ,stout for intraoral suturing, a 15 cm handle length needle holder is often recommended the beaks of the needle holder are shorter and stronger than the beaks of the haemostat, the inner surface of the beaks is crosshatched to provide good surface for grasping of the needle and to prevent its rotation or movement during suturing while in haemostat the inner surface has parallel grooves which differentiates it from the needle holder, so haemostat should not be used for suturing but preserved for its original work to control bleeding during surgery.



B-Needles:

Most sutures come fused to needles in a pre-sterilized package, suture needles differ according to the shape of their cross section and their length, there are generally two types:

1-cutting needle, have a triangular cross section

2-taper point, which are round or oval in cross section.

According to the shape of the needles, straight needles are only used where tissues or areas of surgery are easily accessible and therefore they are not used in oral cavity because of the limitation of space in oral cavity, so curved needles are mostly used of a length of 21-25 mm or 3/8 or 1/2 (half circle) and this design allows the needle to pass through the limited area of suturing easily, the cutting needle(triangular cross section) also called *traumatic needle* but it has a good advantage of being able to enter the tissues easily during suturing specially in tout areas like mucoperiosteum of the hard palate(keratinized mucosa) or skin where the tissue is difficult to penetrate the tapered needle is generally used for closing muscle or fascia that are easily penetrated.

C-Suture material:

many types of suture materials are available and could be classified by size , resorb ability ,and whether or not they are monofilament or poly filament .Sutures are made of wide variety of materials and come in several sizes, each designed for a particular purpose

1-size

Sutures are available in various sizes that range from one zero (1/0) to (1 l/0)eleven zero, increasing number of zeros means decreasing the suture diameter, the most size used in oral and maxillofacial surgery is size 3/0 and 4/0 ,the size of the suture usually expressed on the package e.g. 3/0 ,4/0, 2/0....the size 3/0 used most commonly in suturing oral mucosa, which is enough to prevent tearing of the oral mucosa and strong to withstand the tension placed on them intraorally.

2.Resorbability:

Sutures may be resorbable or non-resorbable, resorbable sutures are made of materials that the body is capable of easily breaking them down, like catgut sutures and this is used for suturing deep structures like muscles, fascia, so these sutures does not require removal, while non resorbable sutures like black silk sutures ,nylon, stainless steel, those need removal of the suture postoperatively..
For the resorbable sutures we have two types. Plain gut and chromic gut ,the plain gut is subjected to rapid resorption or digestion by the proteolytic enzymes produced by the inflammatory cells ,while the chromic catgut is treated with special chromic salts ,to provide resistance for the proteolytic enzymes.

D-Scissors:-

the final instrument necessary for placing sutures are suture scissors ,the suture scissor usually have long handles, short cutting edge or beaks because main purpose is to cut suture during suturing or on the removal of sutures postoperatively, other types of scissors used in oral surgery are called soft tissue scissors or operating scissors: these scissors are used for cutting of tissues and for dissection or undermining of tissues during surgery , we mainly have two types of operating scissors..

1-small,sharp,delicate ones used for fine work

2-blunt nosed scissors: used for undermining soft tissues as well as for

Blunt tissue scissors

notice that these operating scissors should not be used for cutting sutures or other material because these materials make them blunt ,and it became less effective for cutting tissues.

7-instruments for retraction of soft tissues:

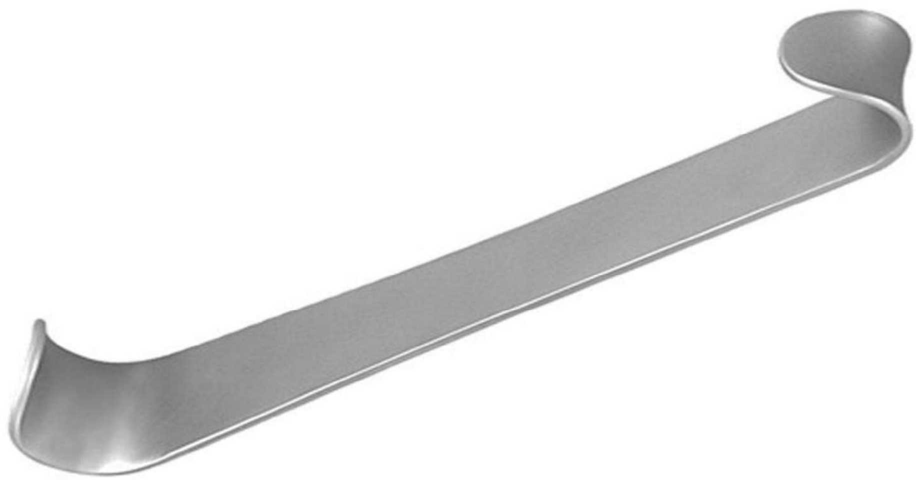
These instruments may be held by the surgeon or his assistant to help the surgeon to have a clear field or area during his work so that it will provide accessibility to the working field and in addition to that it will help in the protection of surrounding tissues from trauma or injury during surgery

There are many types and varieties of retractors ,that have been designed to retract the cheek ,tongue ,lips and mucoperiosteal flaps ,so we have ..

A-Cheek retractor:

from its name its used to retract the cheek ,the most popular one is the *kilner retractor,* its double ended with different sizes and widths ,and it's held against or at the angle of the mouth ,to retract the cheek away from the operative area .

cheek retractors



B-Mucoperiosteal flap retractor:

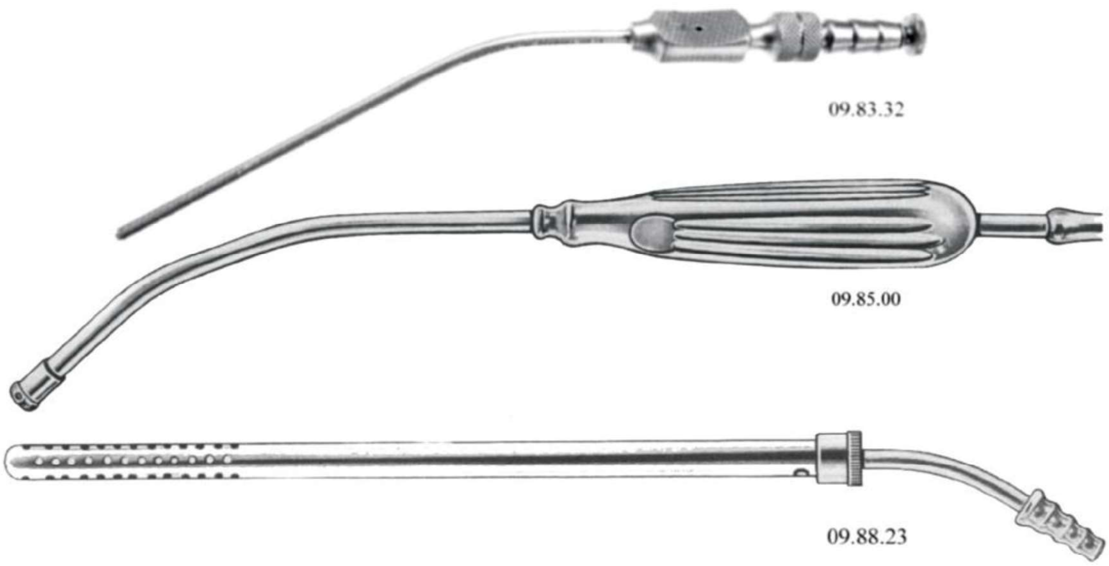
it is used to retract the flap away from the operative area ,to provide good vision for the surgeon and to help protect the flap from trauma ,it should be held against the bone and not on the flap ,there are many retractors of different shapes and designs especially made for flap retraction and even *periosteal elevator (Howarth 's)* may be used as a retractor for the flap ..

There is also special retractors for the tongue but usually the mouth mirror is the most common instrument used for tongue retraction and lip retraction

8-insrument for irrigation and suction

A-instrument for suction:

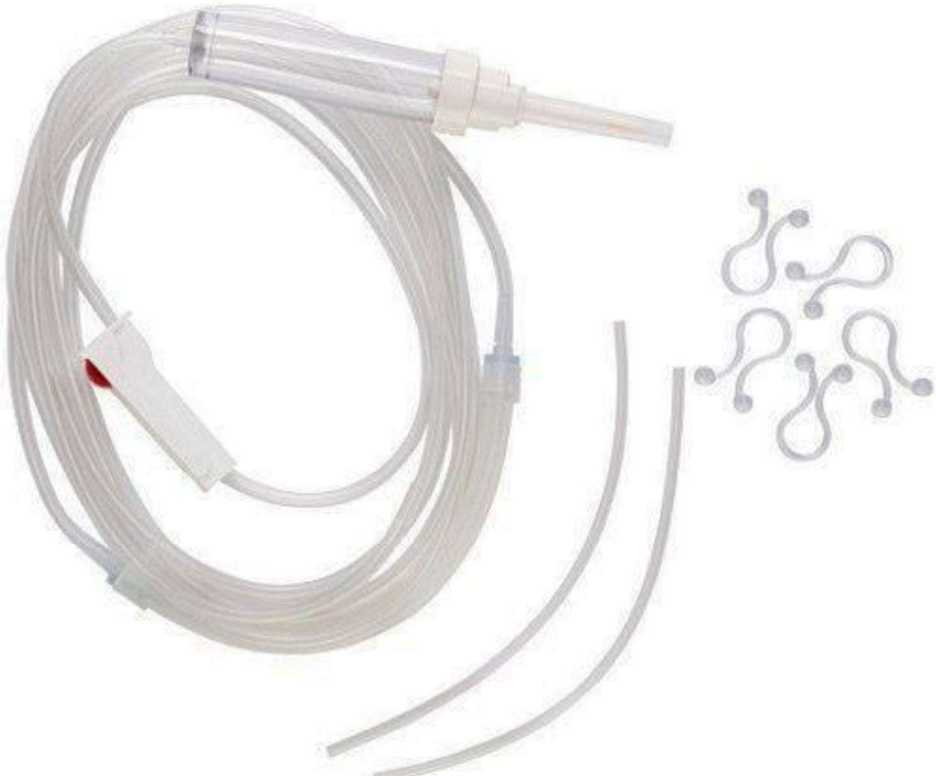
These instruments used to provide good visualization of the operative field by aspirating the blood ,saliva and the irrigating solution (e.g. saline , distilled water) used during operation . in oral surgery a fine metal suction tips on vacuum pressure most used to perform such duty . the use of suction also is very important and most be used when we are working under general anaesthesia to prevent the possibility of aspiration of blood and other fluids by the patient.



B-Instruments for irrigation:

When we use hand piece and bur to remove bone its necessary to use irrigating solutions like normal saline to cool the bur and prevent bone damage by the heat generated during cutting of bone ,also irrigation will clean the area of surgery from small debris and chips during the surgical procedure and after finishing before suturing the area ,large plastic syringe with blunt 18 gauge needle is used for irrigation purposes ,the needle should be blunt and smooth so that no damage to the vital organs or soft tissues can occur ,the needle also preferable to be angled to have good and efficient direction of the irrigation fluid .

Irrigation set:



Oral surgery
Technique of forceps extraction

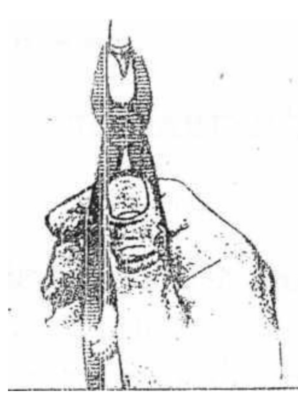
Regardless of the tooth to be extracted in the oral cavity, some common principles are applied to all dental extraction. The general steps in the closed-extraction (forceps extraction) procedure are: -

1- Soft tissue retraction: -

Before starting the application of the dental forceps, the gingival tissue surrounding the tooth should be reflected with blunt probe or tweezers, the neck of the tooth freed labially and lingually as far as the bony alveolar margin, so that no laceration or tearing of the gingival occur on extraction. So, care should be exercised to avoid application of the beaks over the gingival tissue. Reflection of the gingival tissue allows the surgeon to ensure that profound anesthesia is secured before starting extraction. Also, gingival retraction allows the beaks of forceps to be positioned more apically without interference or impingement of the gingival tissue.

2- Handling of the forceps: -

The forceps should be grasped by the palm of the right hand and the thumb finger is placed below the joint. The little finger is placed inside the two handles of the forceps so that it can control the opening and closure of the handle and guide the forceps beaks on the root surface, and when the tooth is grasped the little finger is placed outside the handle.



Handling of dental forceps while closing and grasping the tooth

Handling of dental forceps while opening

3- Retraction and support (the use of the left hand):

When using the forceps and elevators to luxate and remove teeth. It's important that the surgeon's opposite hand play an active role in the procedure. For the right-handed operator (dentist), the left hand has a variety of functions:

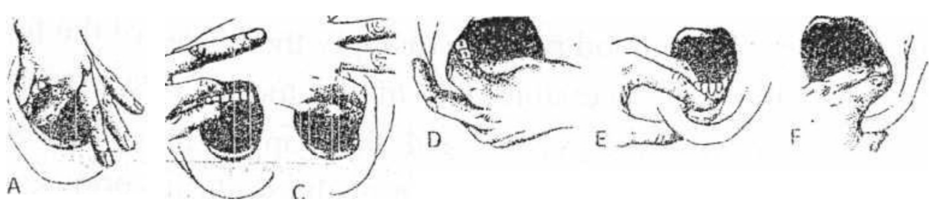
a) It's used to reflect the soft tissues of the cheeks, lips and tongue to provide adequate visualization of the area of surgery.

b) It helps to provide protection for the soft tissue structures and protect other teeth from forceps, if they release suddenly from the tooth socket.

c) It helps to stabilize the patient's head during extraction process.

d) The opposite hand plays an important role in supporting and stabilizing the lower jaw when mandibular teeth are being extracted to prevent injury and post-operative pain in the tempromandibular joint (T.M.J). And also, to prevent dislocation of the mandible during extraction especially when extraction is performed under general anesthesia

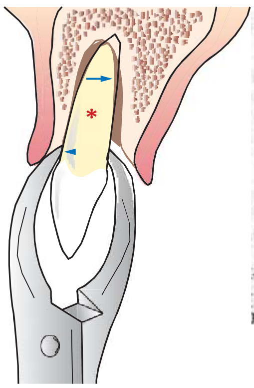
e) The opposite hand supports the alveolar process and provide tactile information to the operator concerning the expansion of the alveolar process during luxation process.

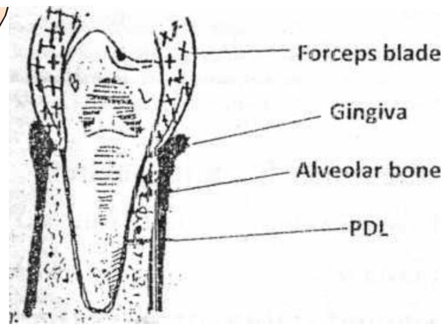


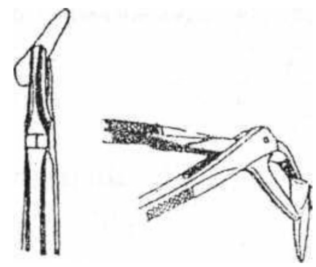
Correct use of left hand during extraction of: A, Right maxillary premolars, B, Maxillary anterior teeth. C, Left maxillary premolars and molars. D , Right mandibular cheek teeth (N.B. The operator stands behind the patient when extracting these teeth) E, Mandibular anterior teeth. F. Left mandibular molars and premolars

4- The application of forceps blades to the tooth (tooth grasp):-

After you select the proper forceps for the extraction of particular tooth. The forceps blades are applied on both labial (buccal) and lingual (palatal) surface of the tooth, so that the blades are parallel to the long axis of the tooth to be extracted





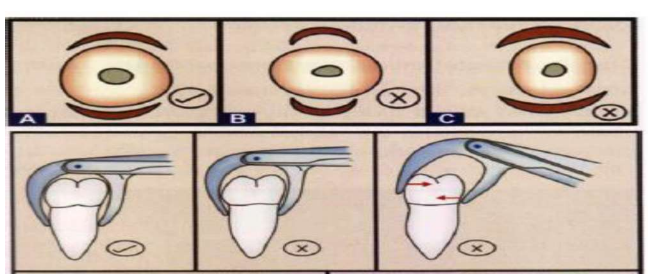


Incorrect alignment of upper premolar (left) and lower premolar (right)

The blades of the forceps are moved on the tooth surface apically and are allowed to move apically cutting through periodontal and gingival fibres to grasp the tooth-root surface below the cemento-enamel junction. The tooth to be extracted is grasped firmly, the blades are not allowed to slide on the surface of the root during extraction movement.

It's a good practice to apply the blades of the forceps to the less accessible side of the tooth to be extracted first under direct vision and then apply the other blade on opposite side. If one side of the tooth is carious, then the forceps blades applied to the carious side first and extraction movement should be started toward the carious side.

The surface of the beaks of the forceps should lie as close as possible to the surface of the tooth grasped tightly without slipping during extraction



5- The displacement of the tooth from its socket: -

This is performed by using the extraction movements.

The extraction movements are: -

a- Outward movement (labial or buccal)

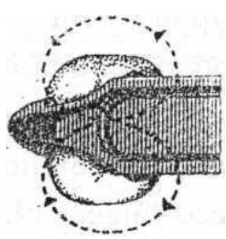
b- Inward movement (lingual or palatal)

c- Rotatory movement (rotation movement).

The purpose of extraction movements is:

1-Cutting the tooth attachment.
2- Separating the tooth from the wall of the socket.
3- Dilatation of the bony wall of the socket utilizing the resiliency of the living bone.
4- The removal of the tooth from the socket.

When the blades applied to the root surface a firm grip of the root taken by the forceps and buccolingual and lingobuccal movements are made in that order. This pressure should be firm but not crushing, smooth and controlled not jerking. By this movements normally after few lateral movements the tooth is felt to be loosen and begin to rise out of the socket, when this loosening occurs, rotatory or figure 8 movement will help to delivery of the tooth



. **Occlusal view of mandibular molar illustrating the figure 8 of movement**

Rotatory movement:

Can be used as primary movement for extraction of teeth with conical straight roots such as the upper central incisors and lower second premolars, also rotatory movements are useful in completing the removal of teeth previously loosened by other means and this is called secondary rotatory movement.

The gross distortion and laceration of the buccal plate and mucosa are happened if excessive lateral movement is done. The final movement by which the tooth is removed from its socket should be always directed outwards, so reducing the trauma to the opposing teeth and preventing slipping of the tooth in the mouth which may be swallowed or aspirated by the patient.

If the tooth dose not yield (no movement) after applying a reasonable force, excessive force should not be used, stop and re-evaluate the case clinically and/or radiographically because the tooth may be need surgical extraction.

Post-operative care of extraction wound: -

1- Examine the extracted tooth to be sure that there is no fracture in the roots (retained root), keeping in mind the anatomical variations in number of roots of each tooth, because some teeth have accessory roots.

2- Always examine the socket for any loose fragments of bone or roots (pieces of enamel), foreign materials (e.g. calculus, amalgam particles, or pathology (e.g. periapical granuloma, polyp) all these should be removed by using curette or suction tip or tweezer.

3- You have to remove or smooth any sharp bone or projections of inter-radicular bone, then apply suture if necessary, e.g. if there is laceration in the soft tissue.

4- The expanded bucco-lingual plate should be compressed or squeezed back to their original configuration or shape in order to reduce any distortion of the supporting tissue to re-establish the normal contour before extraction

5- Make sure that the socket is full of blood, (to form blood clot)

6- Place a properly shaped and size (2X2 Cm) gauze piece over the socket (and never put it into socket) in such a way that the patient close his mouth and it is not visible to gain initial control of haemorrhage. It is important to place the gauze directly over the extraction site in the space occupied by the crown of the extracted tooth and not over the occlusal table of teeth to ensure that the pressure is correctly transmitted over the socket to achieve haemostasis.

Instructions to the patient:

1- Keep biting over the gauze for at least (0.5-1) hours and discharge after that

but if haemostasis is not established, you can use another.

2- Do not spit during first *½* hour and use minimal talking and avoid violent exercise or activity to assist the formation of a firm clot in the socket, so rest for the first few hours following extraction are recommended ,avoid insertion of the tongue tip inside the socket which lead to dislodgement of clot or disturb its formation

3- use only cold fluid and soft diet for the few postoperative hours and not take solid or hard food for the rest of the day, and chew on the opposite side of extraction.

4- in case of continuous bleeding. Place a sterile gauze in place over the wound and keep it in place for an hour, if bleeding not stopped contact your dentist.

5- always prescribe the proper analgesic and antibiotic if necessary (e.g. The presence of residual infection, diabetic patient, patient on immune-suppressive drugs, aids...etc.

Oral surgery

Complications of exodontia

Complications can arise during the procedure of extraction or may manifest themselves sometime following the extraction ,so we have immediate complications and post-operative one.
All these complications arise from error in judgment, misuse of instruments, exertion of extensive force or from anatomic causes or factors.

By careful diagnosis and planning of the procedures many complications can be avoided but some of these complications may occur even when utmost, care is exercised , so that the dentist or the oral surgeon should be qualified to deal with each complications successfu1y.
So the possible complications are:-

1- Failure to secure anesthesia.

Failure to secure profound or good anesthesia may be due to:-

a- Faulty technique, or Insufficient dosage of anesthesia.

b- Expired anesthesia.

c- The presence of acute infection.

2- Failure to remove the tooth with either forceps or elevator.

failure to remove the tooth after applying a reasonable amount of force without movement or yielding of the accused tooth need further clinical and radiological evaluation, because the tooth may be need surgical extraction.

3- Fracture (#)of:-

A. Crowns and roots.
B. Alveolar bone.
C. Maxillary tuberosity.
D. Adjacent or apposing tooth. ,
E. Mandible.

a-Fracture of crowns and roots: -

The most common complication during tooth extraction is fracture of the tooth crown or roots.
The factors that may lead to fracture of crown or roots may be classified into three groups:

1. Factors related to the tooth itself.
2. Factors related to the bone investing that tooth.
3. Factors related to the operator (dentist).

1- factors related to the tooth itself

means that the tooth may be badly carious, or heavily filled, brittleness of the tooth due to age, or non-vitality, root canal filled tooth. Also peculiar root or crown formation like dilacerated tooth, geminated tooth, severely curved root, divergent roots, convergent roots, hyper-cementosis, accessory root and complex root shape, malposed tooth, insufficient space for the application of the extraction instrument, internal & external! resorption.

2-Factors related to the investing bone

means the surrounding bone might be excessively dense or sclerotic due to localized or systemic causes.

3-factors related to the operators

includes improper application of the beaks of the dental forceps or elevator on the tooth to be extracted; like the placement of the beaks of the dental forceps on the crown instead of the root or below the cemento-enamel junction, also the beaks are not parallel to the long axis of the tooth, also the use of wrong type of forceps.

Incorrect application of force during extraction by wrong direction in addition to that the use of twisting or rotational movement when not indicated like the use of twisting movement in extraction of upper 1^{st} premolar or upper 1^{st} and 2^{nd} molar for example.

b- Alveolar bone fracture: -

Fracture of alveolar bone frequently occurs when extraction is difficult. The fractured bone may be removed with tooth to which it is firmly attached or it may be remain attached to the periosteum or it may be completely detached in the socket or wound.

It is a common complication that especially occurs on labial(buccal) area during extraction of upper canine and upper and lower molar teeth.

This complication might be due to: -

1. The alveolar bone is very thin.
2. Accidental inclusion of the alveolar bone within forceps blades
3. Configuration of the roots.
4. The shape of the alveolus.
5. Pathological or physiological changes in the bone itself like Ankylosis (bony connection between the tooth and bone), the presence of destruction in the alveolar bone due to the presence of discharging sinus.

c- Maxillary tuberosity fracture: -

Sometime the tuberosity is completely fractured when we try to remove maxillary 3rd or 2nd molar.

Fracture of maxillary tuberosity may lead to a wide opening into the antrum called Oro-antrum communication with irregular tearing in the covering soft tissue lead to profuse bleeding and post- operatively may lead to difficulties in the retention of upper denture.

This complication might occur if the molar tooth to be extracted is isolated and subjected to full force of bite leading to sclerosis of the surrounding bone, or due to downward extension of the maxillary sinus to the nearby edentulous alveolar bone or due to large abnormal size of the maxillary sinus extended to involve the tuberosity; in addition to that, the use of excessive force or wrong positioning of the elevator in the extraction of upper 3rd molars

.

d-Fracture of the adjacent and opposing tooth; -

Adjacent teeth occasionally may be damaged during extraction procedures, this may include loosening or dislocation or fracture of the adjacent teeth.

This misshapes occur mostly due to careless use of the dental forceps or elevator by wrongfully using the adjacent tooth as a fulcrum during the use of elevator or the application of the beaks of dental forceps, also fracture of the crown of adjacent tooth or fracture and dislodgment of its filling.

In addition to that opposing teeth may be chipped or fractured if the tooth being extracted yield suddenly to uncontrolled force of the forceps striking the opposing tooth leads to this complication.

e-Mandible fracture: -

This is a rare complication, but it might occur almost exclusively with the surgical removal of impacted lower third molar tooth.

A mandibular fracture is usually the result of the application of a force exceeding that needed to remove a tooth and often occurs during the use of dental elevators(winters elevator), but sometimes pathological or physiological changes may lead to weakened mandible like: -

1.Senile atrophy and osteoporosis of the bone.

2. Osteomyelitis e.g. osteoradionecrosis.

3. cystic lesion.

4. Impacted teeth.

5. Tumor, benign or malignant..

So, preoperative clinical and radiographic evaluation is very important to avoid such complication or preventing it.

4. Dislocation of the tempro-mandibular joint (T.M.J.): - Exertion of high amount of force during extraction of lower teeth especially posterior teeth may lead to dislocation of the condyle of the mandible and the patient becomes unable to close his/her mouth, especially in patient who had a history of recurrent dislocations in TMJ.

if this dislocation occur it should be reduced immediately by the operator by standing in front of the patient and his thumbs placed intra-orally on the external oblique ridge lateral to the molar teeth and other fingers outside the mouth under the lower border of the mandible, downward pressure with the thumbs and upward pressure with the other fingers may reduce the dislocation, if reduction is delayed it become difficult to reduce it because of muscle spasm and the patient may need general anesthesia to reduce the dislocation, also the patient may complain of traumatic arthritis of the TMJ. Post-operatively due to high pressure applied to the joint during extraction, so supporting the mandible during extraction prevents such complication.

5. Displacement of a root into the soft tissue and tissue spaces and the maxillary antrum: -

During extraction especially on use of elevator, a root or piece of root may be dislodged into the soft tissue through a very thin bony plate overlying the socket and disappear buccally or lingually into the soft tissue between periosteum and bone in the vestibule, but sometimes a root or even a tooth may be displaced into the tissue spaces surrounding the jaws e.g. a retained root in the lower molar teeth may be displaced into the sublingual or submandibular space or e.g. upper third molar may displaced into the infratemporal space.

So the extraction with high force without direct vision on the retained root may lead to such complications, also retained root may be displaced into the maxillary antrum during the extraction of upper molar or sometimes premolar teeth especially palatal root of upper molar teeth.

The presence of large antrum or the use of excessive force during extraction or due to pathological conditions like periapical pathology. All these factors may assist or predispose to such complication, so pre-operative radiograph and clinical evaluation may assist in the prevention of such complication.

6- Excessive bleeding after extraction:-

At the beginning one must understand that some slight oozing of blood for several hours following tooth extraction is considered normal. But sometime excessive or abnormal bleeding may occur following tooth extraction.

<u>The causes of excessive bleeding may be due to</u>:-

A. Local factors

The local causes which are the commonest causes for prolonged bleeding as in usual, due to gross tissue damage, when there is severe bone injury and tearing of the periosteum many vessels are opened also severe gingival lacerations, also damage to large arteries like inferior dental vessel or greater palatine vessels may lead to profuse bleeding, also the presence of Hemangioma (central) and other vascular abnormalities may lead to such complication
Also post-operative infection of the extraction wound causing erosion of the blood vessel leading to secondary haemorrhage, also the working in acutely inflamed area may assist in the prolonged bleeding.

B .systemic factors

For the systemic causes like systemic haematological disorders like thrombocytopenia, reduction in the clotting factors, anticoagulant drugs, hereditary blood disease like haemophilia , all these factors may lead to severe bleeding; so good history and clinical examination and blood investigation is very important and essential before any extraction especially if the patient gives you a history of bleeding on previous extractions or trauma.

7-damage to the surrounding soft tissues.

a. Damage to the gum or lip.

like laceration of the gum during extraction occurs if the gingival tissue not reflected before extraction so gum adhere to the tooth to be extracted from its socket should be carefully dissected before any further attempts to deliver the tooth are made, also the inclusion of the gum by forceps beaks or by blind application of the forceps may lead to crushing of the soft tissue, also the lower lip may be pressed or crushed between the handles of the forceps and the lower lip on extraction of upper teeth if sufficient care is not taken .

b. damage to the tongue and floor of the mouth

Also slipping of elevator during extraction may lead to damage or wounds in the floor of the oral cavity, there are many vital structures in the floor of the oral cavity which might be damage like [sublingual gland , submandibular duct, lingual nerve & tongue]. So the operator should always keep in his mind that supporting of elevator during extraction is very important.

C. Damage to nerves

occur mostly on surgical extraction of teeth rather than simple extraction but one must always be aware of the risk when operating in the region of the (inferior dental nerve, lingual nerve & mental nerve). Inferior alveolar nerve injury is an uncommon occurrence in extraction of erupted mandibular teeth. In rare cases third molar roots may *encircle the nerve* so that extraction of the tooth will cause nerve injury also curration or improper use of elevators to remove root apices may cause tearing or displace bone fragments so that will impinging or pressing the nerve in the canal "inferior dental canal" result in *Parasthesia or anesthesia* of half of lower lip.

The mental branch of the alveolar nerve also may be injured during surgical procedures in the premolar region. The lingual nerve may be damaged during exodontias of the lower molar teeth especially the lower wisdom tooth by trapping the lingual soft tissue in the forceps beaks or by direct trauma from misusing of elevator or by using surgical extraction to remove impacted wisdom tooth.

8-post -operative pain:

Post-operative pain and discomfort after extraction due to traumatized hard tissue may be from bruising of bone during instrumentation or from using burs for removal of bone also damage and rough handling of soft tissue during extraction is another cause for postoperative pain.

The most common cause for the moderate to severe continuous pain after extraction is related to a well-known cause called ***dry socket*** *or acute localized alveolar Osteitis.* -The patient presented with continuous moderate to severe pain after 24-72 hours after extraction which may last for 7 to 10 days clinically the patient may presented with empty socket (there is no clot in the socket) , exposed bone or empty socket with some evidence of broken-down blood clot and food debris within it with intense bad odour. The aetiology of this condition is incompletely understood but many predisposing factors exist like infection, trauma, blood supply, site, smoking, sex, vasoconstrictors or systemic factors.

9-post- operative swelling:

After extensive surgical interference and exodontias some time may be associated with post-operative swelling ,this swelling may be related to one or more of the following causes: (A-Oedema , B-Infection, C-Hematoma.)

a. Oedema*:*

oedema occurs after surgery as a result of tissue injury *(it is normal response)*when there is great damage to the tissue by using blunt instrument. And rough handling of tissue may Increase the chance of production large oedema.

So laceration of tissue during extraction, trauma to the bone or periosteum are some of the most common causes of oedema and in other words *post-operative swelling,* persistent post-extraction swelling or the development of swelling several days after surgery is usually due to infection.

b. infection

swelling due to infection can be distinguished from postoperative oedema by the increased skin temperature ,greater redness of the overlying tissues, the usual presence of fever and sometime fluctuation is present due to presence of pus. The infection should be always considered a serious complication and need urgent management.

c.hematoma

means a collection of blood in the extra-vascular spaces of the tissues. It is rare complication following extraction of the teeth, but sometimes hematoma or ecchymosis may develop postoperatively if haemostasis is not developed and persistent bleeding from either the socket or adjacent alveolar bone.

10-The creation of an oro-anrtal communication.

On extraction of upper molar teeth and sometimes upper premolars a communication between the oral cavity and maxillary antrum may be created. This communication if not healed or closed after few days a chronic condition occurs called *Oro- antrum fistula.*

Close proximity of the maxillary cheek teeth to the maxillary antrum which are separated only by little amount of bone and sometime even the soft-tissue lining of the maxillary sinus, the presence of periapical infection, the antrum itself may be abnormal in size, misjudgement of force used in extraction or the presence of pathological lesions .all these factors may assist in the production of this complication.

to confirm the presence of this complication ,the patient is asked to pinch or close the nostrils together and blow air gently into the nose , the operator may see blood bubbling, or shooting of air through the communication is heard or a piece of cotton on tweezer may be defected. The *presence of* this complication need surgical correction by well-trained oral surgeon and surgical unit in which all instruments and qualified staff present.

11-Trismus:

Means inability to open the mouth, trismus is one of common complication following extraction of teeth especially the surgical removal of teeth especially the surgical removal of wisdom teeth. Trismus may be caused by post-operative *oedema, hematoma, inflammation of the soft tissue. Trauma and arthritis* of the tempromandibular joint, it may be related to the use *of inferior dental block* local anesthesia so the management of the trismus depend on diagnosis of the cause of this complication

12-syncope(fainting):-

Collapse on the dental chair is a common complication during extraction. The patient may often complaining of feeling dizzy, weak & nauseated ,and the skin is seen to be pale ,cold and sweating, these complains may be accompanied by loss of consciousness, and the patient if not noticed at the beginning of the fainting may shows episode of convulsion.

The primary pathophysiological component of this situation is *cerebral ischemia* secondary to an inability of the heart to supply the brain with an adequate volume of oxygenated blood. In the presence of anxiety ,blood flow is increasingly directed toward the skeletal muscles at the expense of other organ systems such as the gastrointestinal tract ,in the absence of muscular movement ,the increased volume of blood in the skeletal muscle remains there, decreasing venous return to the heart and decreasing the volume of blood available to be pumped by the heart (uphill) to the brain.

A slight decreased in cerebral blood flow is evidenced by the signs and symptoms of vasodepressor syncope(i.e. light headedness ,dizziness, tachycardia, palpitation) if this situation continues cerebral blood flow declines still further and the patient loses consciousness.

When the operator notice these signs and symptoms a first aids treatment should be started by lowering the head of the patient by putting him in supine position by lowering the back of the dental chair. Care should be taken to maintain the airway and you have to notice the condition of the patient .if consciousness is not returned within 1-2 minutes otherwise one should consider that something serious like *respiratory arrest* or *cardiac arrest* may happen and the patient need medical emergency.

LOCAL ANAESTHESIA IN ORAL SURGERY

Definition:-

Local anesthesia (LA) is defined as a loss of sensation in a circumscribed area of the body caused by a depression of excitation in nerve endings or inhibition of the conduction process in peripheral nerves. An important feature of LA is that it produces this loss of sensation without loss of consciousness, in this point LA differs dramatically from general anesthesia.

Pain:- unpleasant physical sensation Experienced following the application of noxious stimuli:

Although the supporting tissues of the teeth can them selves give rise to pain most of the nerve endings in the periodontal membrane are properoicptive

Pain threshold:- a point at which the patient feels discomfort when exposed to painful stimuli.
The patient who feels minimal discomfort from painful stimuli is having high pain threshold

Many factors influence this response such as fear, apprehension and fatigue, all of which lower the pain threshold.

Anesthesia:- means loss or abolition of all modalities of sensation which includes pain and touch

Analgesia:- means loss of pain sensation only.

Paraesthesia:- means altered sensation (tingling sensation) and this may occur when a damaged nerve is regeneration or when a local anesthesia is either starting to work or its effect is wearing off. the effect of a local anesthetics on nerve fiber has been shown to be dependent on
1-the duration of exposure of local anesthesia
2- Concentration
3- Volume of the solution

Premedication:- is the use of drugs to reduce a patient's apprehension prior to operative treatment.

Sedation techniques:- involve the administration of a sedative to reduce anxiety in the conscious patient, usually, given by I.V route .

Relative analgesia:- is a sedation technique in which the patient remains conscious but mental relaxation is induced by inhalation of a mixture nitrous oxide, oxygen and air. Local anaesthesia used when necessary.

Classification of nerve fibers:

The fibers of peripheral nerves classified according to the basis of electrophysiological and morphological differences:

<u>Type A ; fiber</u>: largest fibers further divided into four groups :-
1) A- alpha (α): responsible for motor action & muscle properioception.
2) A-beta (β): responsible for motor action & muscle properioception.
3) A-gamma (γ): responsible for motor action & muscle properioception.
4) A- delta (δ): responsible for fast sharp pain, temperatures, touch, & pressure

<u>Type B fibers</u> : preganglionic responsible for sympathetic activity.

<u>Type C fibers</u> : unmyelinated , the most numerous in the peripheral nervous system. Responsible for conduction of dull or burning pain.

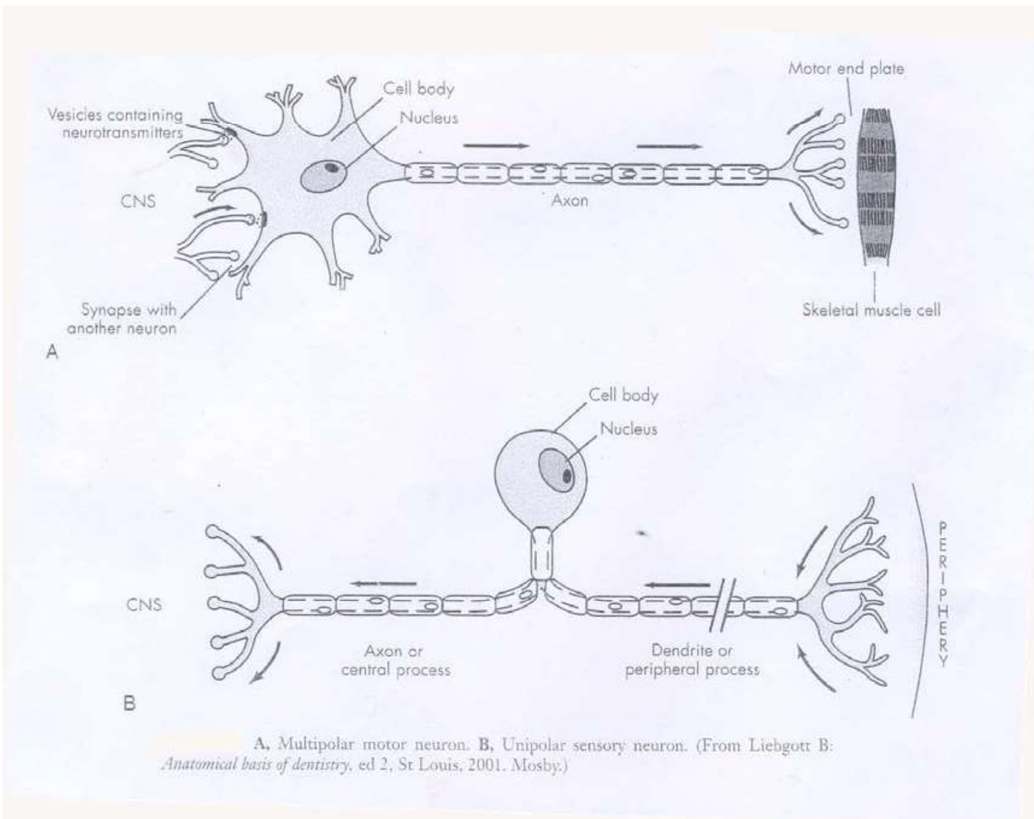
Noxious stimuli are transmitted to the CNS by way of Aδ &C fibers. The lightly myelinated Aδ fibers are responsible for conduction of sharp, bright pain while unmyelinated C fibers conduct dull or burning pain.

Requirements of local anesthetic agents:-

- It should not be irritating to the tissue to which it is applied
- It should not, cause any permanent alteration of nerve structure
- Its systemic toxicity should by low
- It must be effective regardless of whether it is injected into the tissue or applied locally to mucous membranes.
- The time of onset of anesthesia should be as short as possible
- The duration of action, must be long enough to permit completion of the procedure.
- It should have potency sufficient to give anesthesia without using harmful concentration
- It should be relatively free from producing allergic reactions
- It should be stable in solution and readily undergo biotransformation in the body
- It should either be sterile or be capable of being sterilized by the heat without deterioration.

Review of the nervous system and impulse generation:-

The basic structural unit of the nervous system is the neuron (fig: 1) which is able to transmit messages between the central nervous system and all parts of the body, it consists of three parts: cell body axon and dendrites. The dendrites which end in the free nerve endings respond to stimulation produced in the tissue they lie, provoking an impulse that is transmitted centrally along the axon. The axon which is the single nerve fiber, is a cable like structure composed of neural cytoplasm and covered by thin sheath which is the nerve membrane; in some nerves an insulating lipid rich layer of myelin covers this membrane. Current theories stated that nerve excitability and conduction are both attributable to changes developing within the nerve membrane.



A, Multipolar motor neuron. B, Unipolar sensory neuron. (From Liebgott B: *Anatomical basis of dentistry*, ed 2, St Louis, 2001, Mosby.)

Fig. 1 A.motor neuron. B.sensory neuron.

Nerve membrane:-

The nerve membrane consists of two layers of lipid molecules (phospholipids) and associated proteins, lipids and carbohydrates (fig:2). All biologic membranes are organized to (1) block the diffusion of water soluble molecules. (2) be selectively permeable to certain molecules via specialized channels and (3) transduce information by protein receptors responsive to chemical or physical stimulation. Since the nerve membrane exhibits selective permeability, therefore significant differences exist for ions between the intracellular and the extracellular concentrations. Accordingly high concentration of K inside while high concentration of Na and CI outside the nerve membrane.

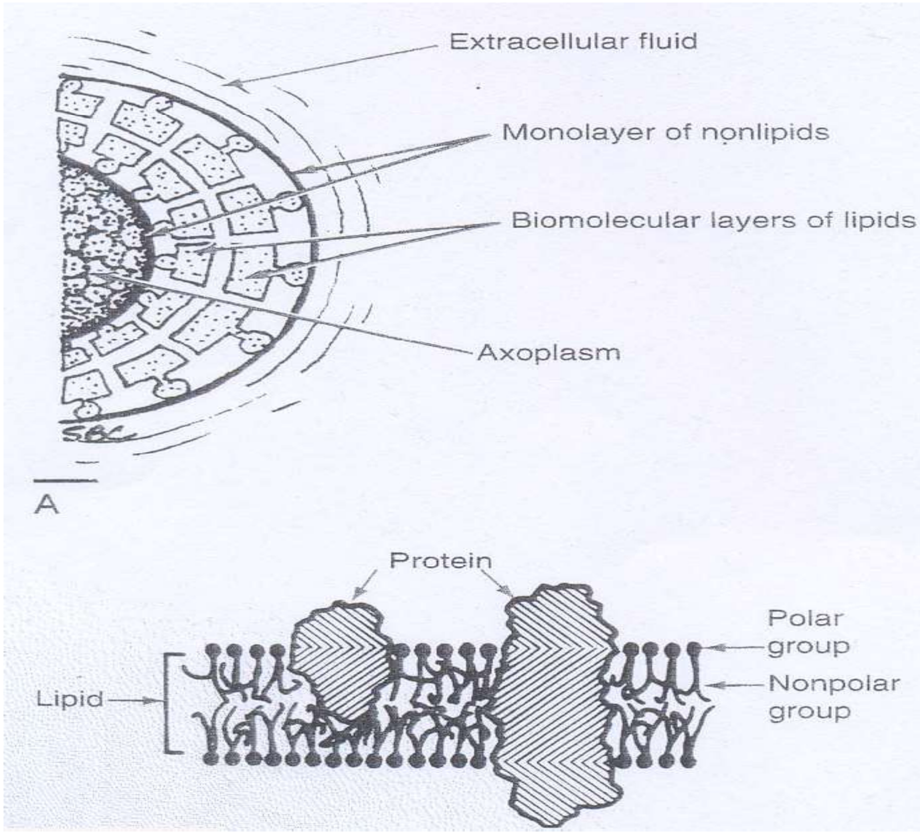


Fig.2 Configuration of the biological membrane.

In some nerves an insulating lipid rich layer of myelin covers this membrane (fig: 3). The outer most layer of myelin consists of the schwann cell cytoplasm and its nucleus. A gap between two adjoining schwann cells and their myelin spirals called node of ranvier at these nodes the nerve membrane is exposed directly to the extracellular medium.

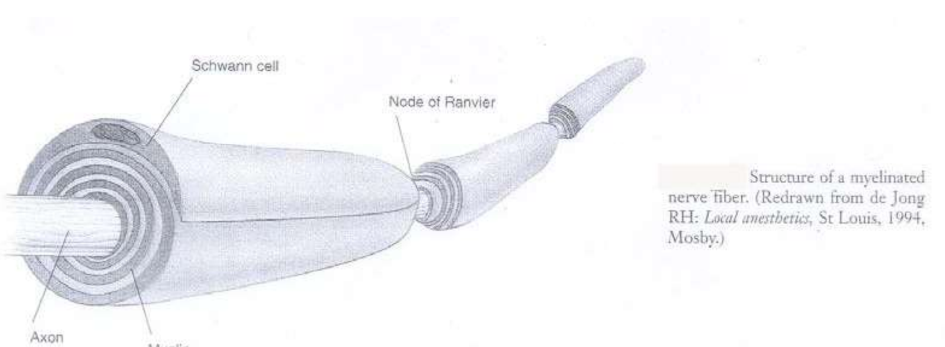


Fig. 3 The structure of a myelinated nerve fiber.

Impulse generation:-

The function of a nerve is to carry messages form one part of the body to another. These messages in the form of electrical action potentials are called impulses. Impulses are initiated by chemical, thermal, mechanical or electrical stimuli.

Resting state:-

(fig 4 step1) In the resting state the nerve membranes possess negative resting potential (-70mv) that comes from different concentrations of ions on either side of the membrane (due to the selective permeability property).

Depolarization:-

(Fig 4 step 2) when a stimulus excites a nerve this will lead to an increase in permeability of the membrane to Na ions, the rapid influx of Na ions to the interior of the nerve will cause depolarization of the nerve membrane from the resting level to its firing threshold of approximately (-50) to (-60) mv. The firing threshold is actually the magnitude of the decrease in the negative membrane potential that is required to initiate an action potential (impulse). When firing threshold is reached permeability of the membrane to sodium increases dramatically and at the end of depolarization the electrical potential of the nerve is reversed an electrical potential of + 40 mv exists. This process takes 0.3 millisecond.

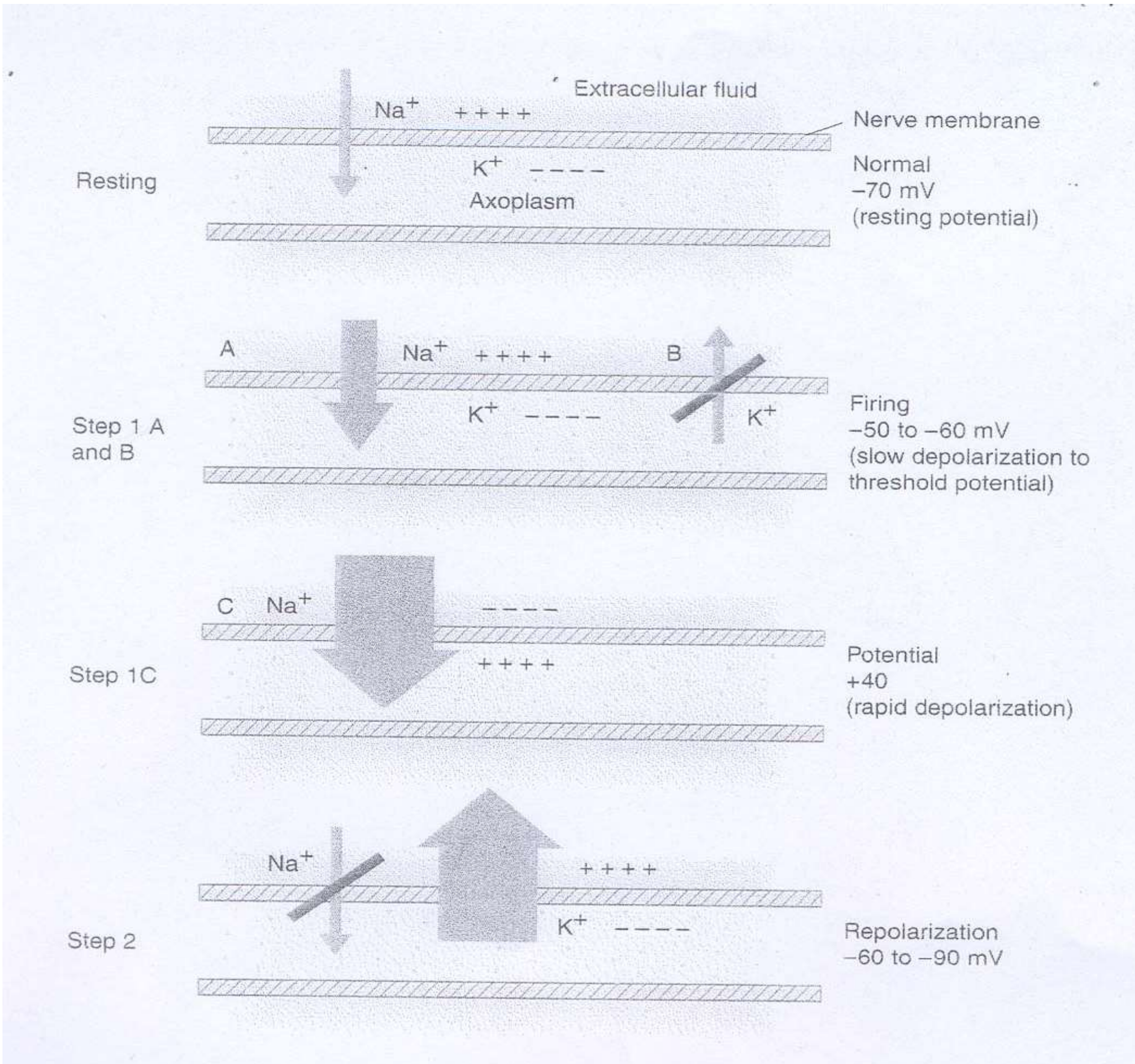
Repolarization:-

The action potential is terminated when the membrane repolarizes (fig 4 step 3) and this is achieved by increase permeability to K ions, resulting in the efflux of K ions (movement to the outside) leading to membrane repolarization and return to its resting potential (-70mv).This process takes 0.7 milliseconds.

The movement of Na ions to the inside of the nerve during depolarization and the subsequent movement of K ions out of the nerve during repolarization are passive process (not requiring energy) since each ion moves along its concentration gradient. After repolarizatino when the nerve return to its resting state a slight excess of Na ions exist within the nerve cell and a slight excess of potassium exists extracellularly, accordingly a period of metabolic activity begins called sodium pump leading to the movement of Na to the outside as well as movement of K to the inside, this pumping mechanism require energy that is coming from oxidative metabolism of adenosine triphosphate (ATP).

Accordingly action potential could be defined as a transient membrane depolarization that result from a brief increase in the permeability of the membrane to sodium and also from delayed increase in the permeability to potassium.

Once an impulse has been initiated it moves along the surface of the axon to the CNS.



Top, Resting potential. *Step 1, A* and *B*, Slow depolarization to threshold. *Step 1, C*, Rapid depolarization. *Step 2*, Repolarization.

Fig.4 Steps of the action potential.

Mechanism of action of local anesthesia:-

The concept behind the action of L.A. is that it prevents both the generation and the conduction of a never impulse thereby they act like a roadblock between the source of the impulse (ex; surgical incision in the soft tissue) and the brain. The aborted impulse prevented from reaching the brain is not therefore interpreted as pain by the patient.

Many theories have been suggested to explain the mechanism of action of local anesthesia, In general the nerve membrane is the site at which local anesthetic agents exert their pharmacological actions, the most popular theories are:

1. **<u>membrane expansion theory</u>**:- this theory states that local anesthetic molecules diffuse through the nerve membrane producing a general disturbance of the bulk membrane structure

expanding some critical regions in the membrane and thus preventing an increase in the permeability to sodium ions, thus inhibiting both conduction and nerve excitation.

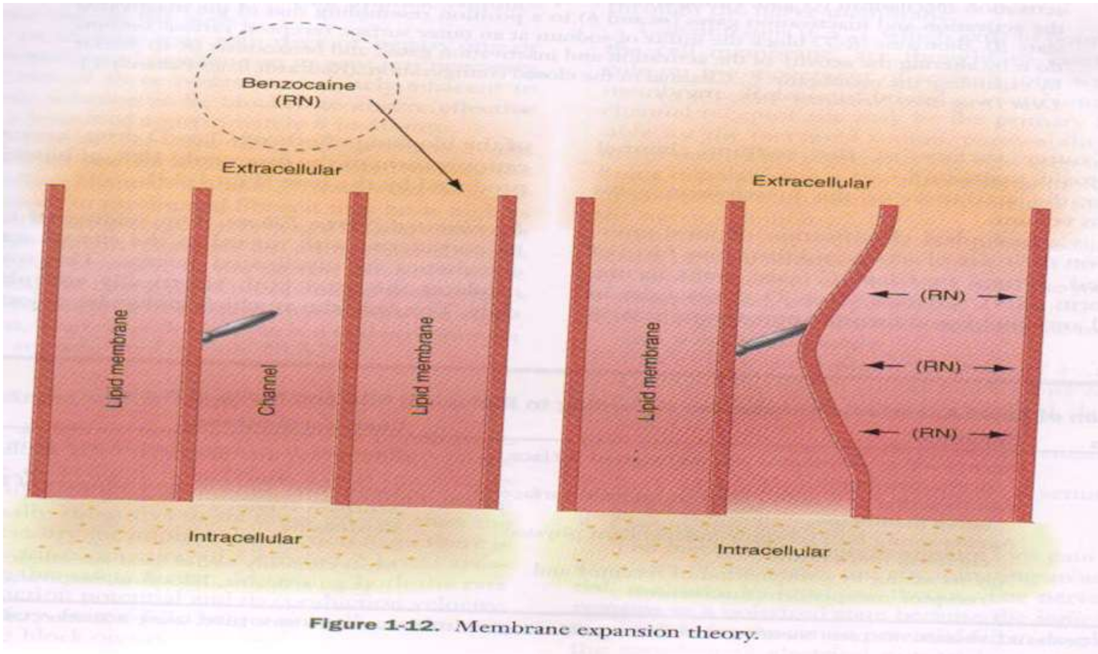


Figure 1-12. Membrane expansion theory.

Fig.5 Membrane expansion theory.

2. **<u>specific receptor theory</u>**:- this is most favored one, it proposes that local anesthetics act by binding to specific receptors in the sodium channel (protein channel along the membrane) and the action of the drug is direct not mediated by change in the general properties of the cell membrane. Once the local anesthetic has gained access to these receptors, permeability to Na ions is decreased or eliminated and nerve conduction is interrupted.

<u>Note</u>**:-**

A peripheral nerve is composed of hundreds to thousands of tightly packed axons (fig: 6) these axons are covered by several layers of fibrous and elastic tissues in which blood vessels and lymphtics course throughout these layers.\

Individual nerve fibers (axons) are covered with perinurium and separated form each other by the endoneurium. The perineurium then binds these nerve fibers together into bundles called fascicule.

The inner most layer of perineurium is the perilemma which represent the main barrier to diffusion of local anesthetic into a nerve. The whole fasciculi are contained within a loose connective tissue called the epineurium which represent 30% to 75% of the total cross section of a nerve.

Local anesthetics are readily able to diffuse through this tissue because of its loose consistency. Nutrient blood vessels and lymphatics traverse the epineurium. These vessels absorb local anesthetic molecules thus removing them from the nerve.

Induction of local anesthesia

Following the administration of a L.A into a tissue near a nerve molecules of the L.A will move from on site to anther according to their concentration gradient so it will move from site of deposition toward the nerve (this process termed diffusion).

Fasiculi that are located near the surface of the nerve are termed mantle bundles (fig 6) they are first ones reached by the local anesthetic and they are exposed to a higher concentration of it. These bundles will be blocked completely shortly after the injection of a local anesthetic.

Fascisuli that are located near the center of the nerve are called core bundles these bundles are contacted by a local anesthetic only after a much delay and by a lower anesthetic concentration because of the greater distance that the solution must move and the greater number of the barriers it must cross.

As the local anesthetic diffuses into the nerve it becomes increasingly diluted by tissue fluids and is absorbed by capillaries and lymphatics thus the core fibers are exposed to a decreased concentration of local anesthetic a fact that may explain the clinical situation of inadequate pulpal anesthesia developing in the presence of subjective sympotoms of adequate soft tissue anesthesia. Complete block of all nerve fibres in a peripheral nerve requires an adequate volume as well as an adequate concentration of the local anesthetic be deposited.

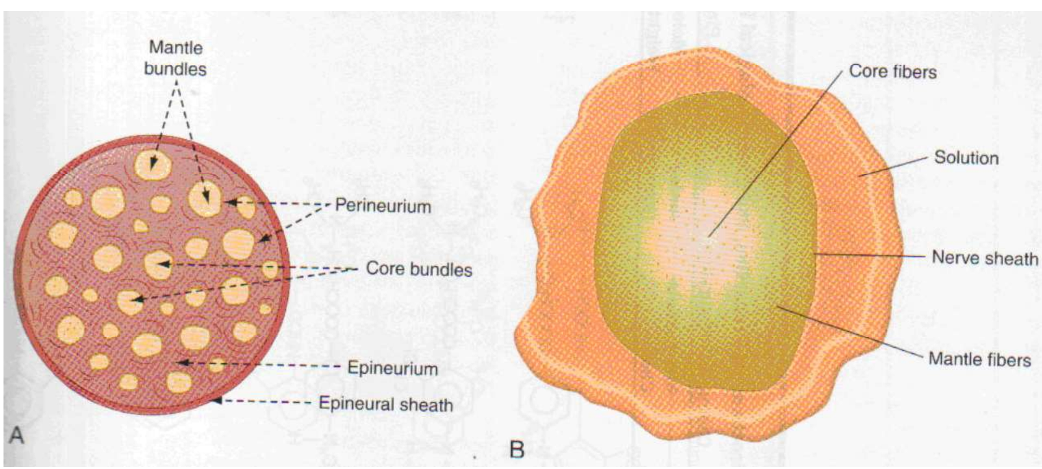


fig. 6 Cross section of a peripheral nerve.

Academic Year 3

[Pharmacology of local anesthesia]

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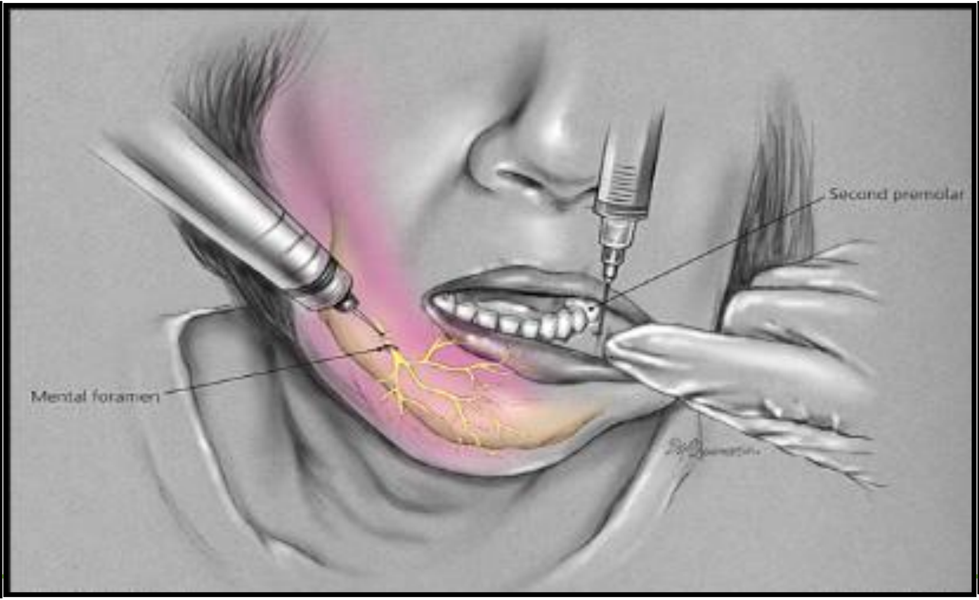
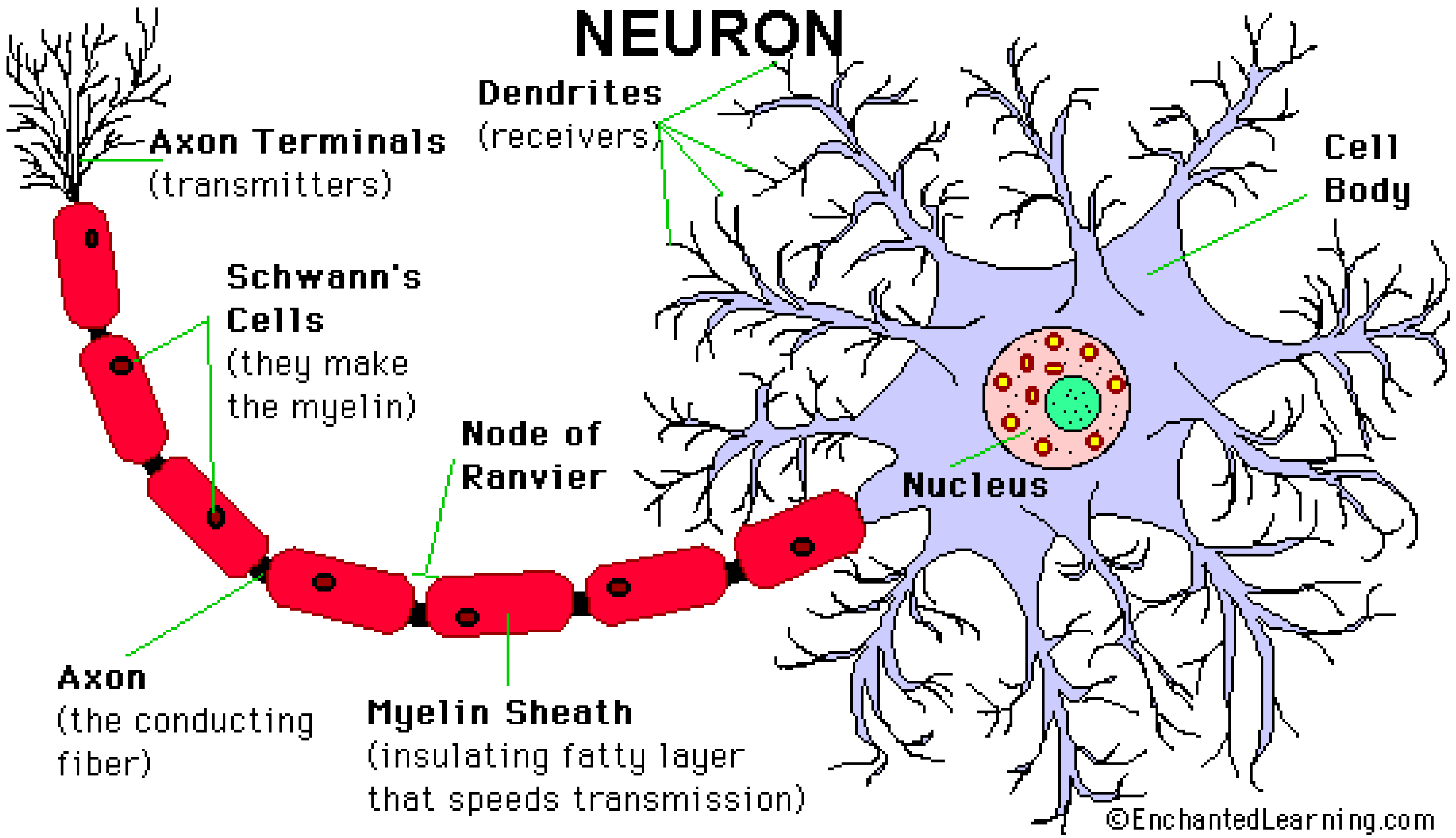


Pharmacology of local anesthesia

- Pharmacokinetics of local anesthetics
- Metabolism
- Systemic actions of local anesthetics
- Vasoconstrictors
- Mode of action
- Dilutions of vasoconstrictors
- Specific agents

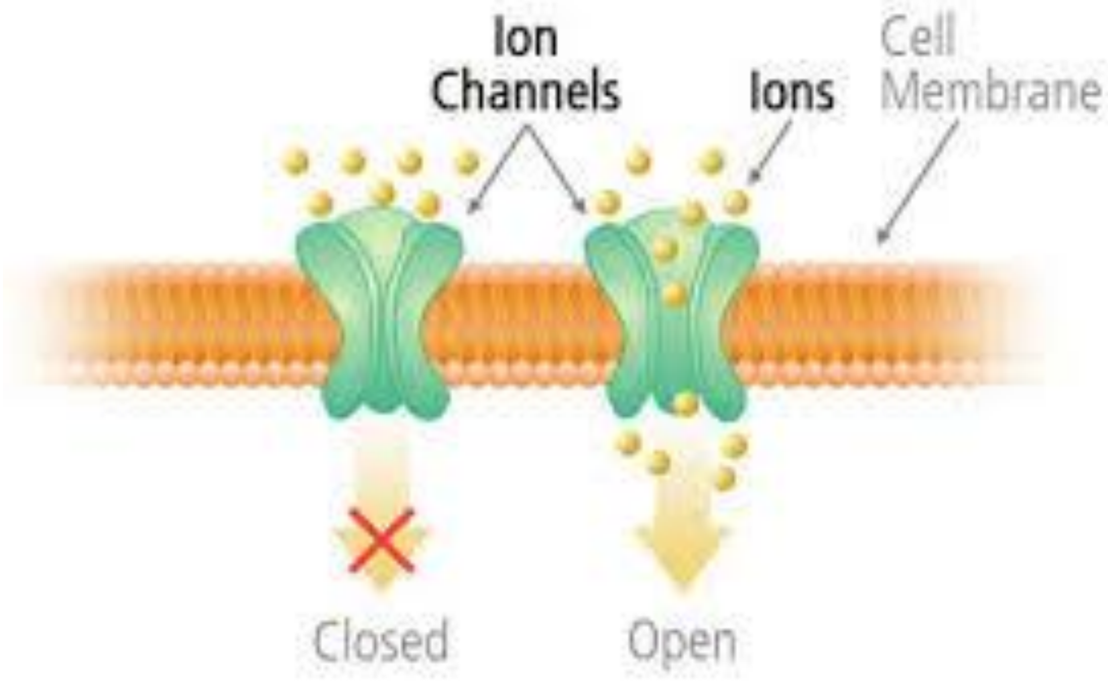
➢ Neurophysiology

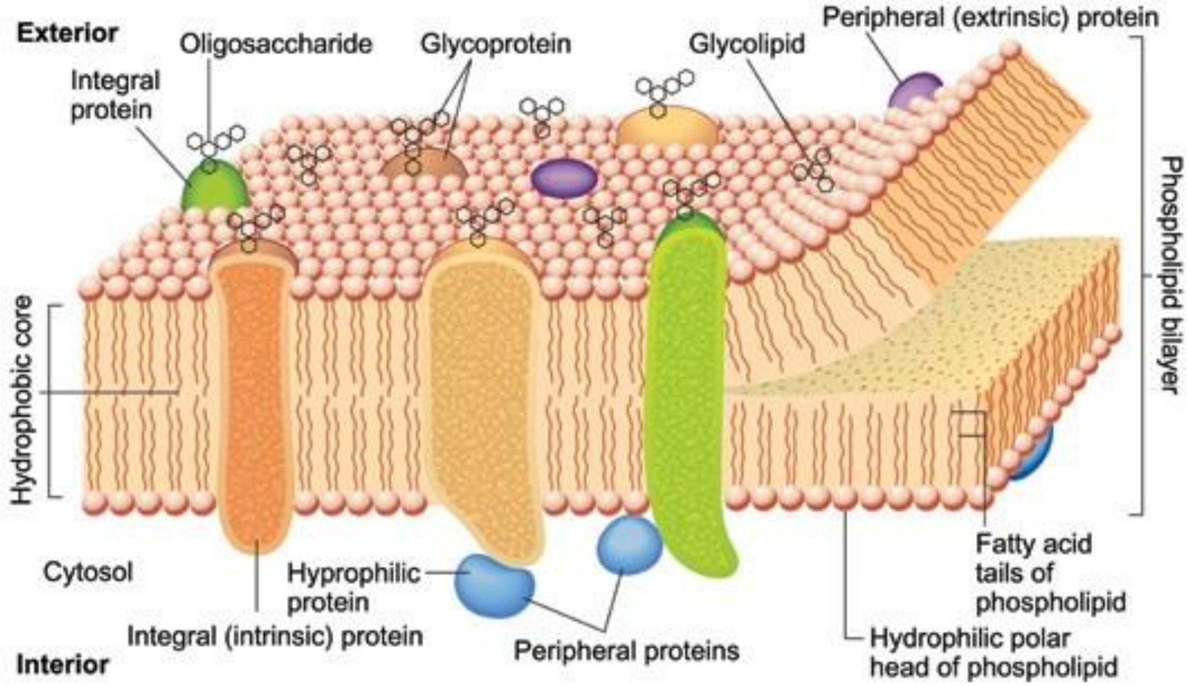
- The neuron or the nerve cell is the structural unit of the nervous system it is able to transmit messages between the central nervous and all part of the body . There are two types of neuron : the sensory neuron and motor neuron .

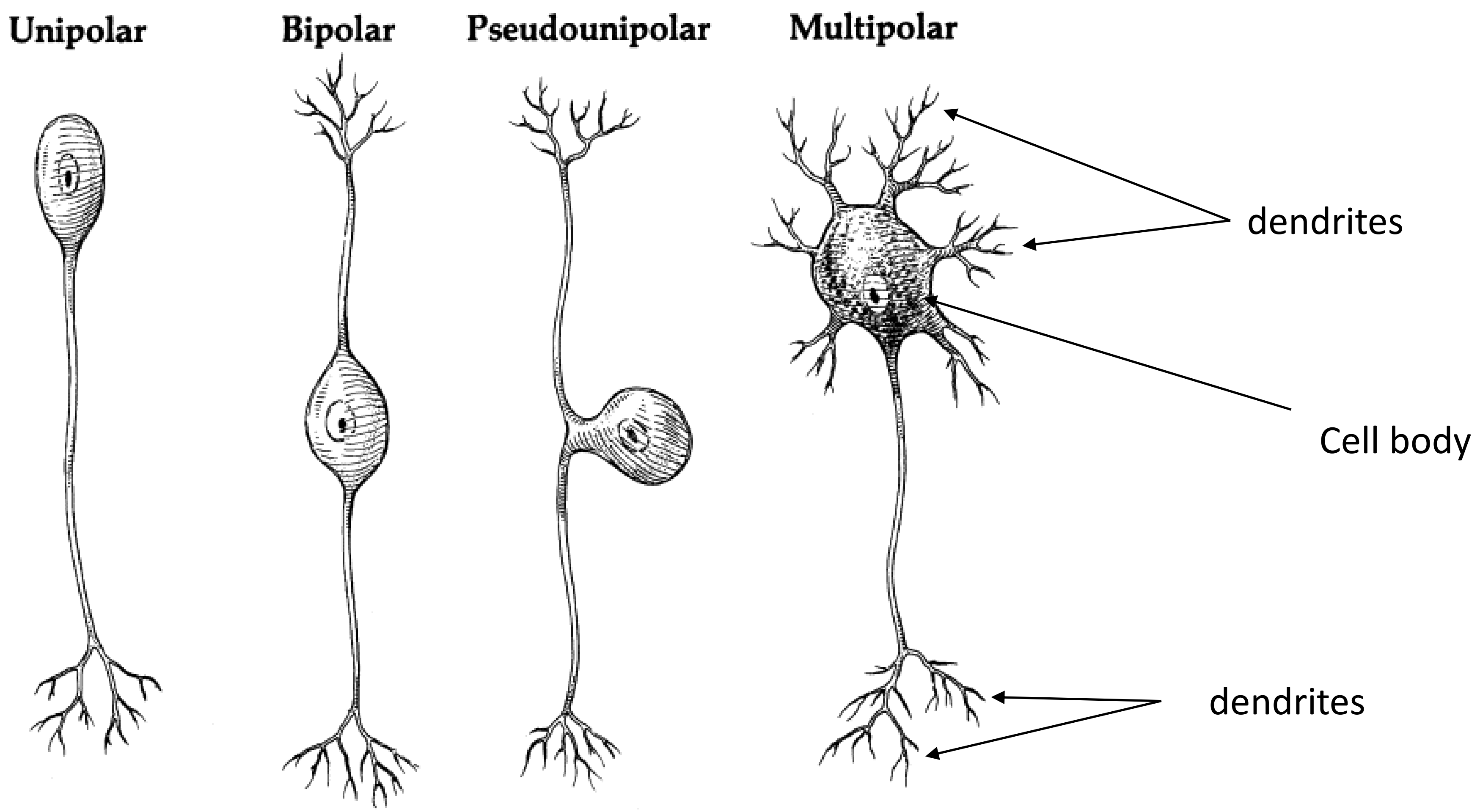


- Sensory neuron that are capable of transmitting the sensation of pain. consist of three major part which are *cell body* , the *axon* , and *dendrites* .
- The *dendrites* which ends in the free nerve endings and is the most distal segment of sensory neuron which respond to stimulation produced in tissue in which they lies provoking an impulse that is transmitted centrally along the axon .
- The *axon* is a thin cable – like structure that may be quite long (the giant axon has been measured as 100-200 cm) it is composed from neural cytoplasm and covered by thin sheath which is the nerve membrane, in some nerves a lipid rich layer of myelin covers this membrane and the membrane is known as myelinated nerve fiber , current theories stated that nerve excitability and conduction are both attributed to the changes developing within the nerve membrane

- The *nerve membrane* consist of two layers of lipid molecules (phospholipids) and associated proteins , lipid and carbohydrates . since the nerve membrane has selective permeability , therefore significant differences exist for ions between the intracellular and the extracellular concentrations .







Unipolar neurons have one axon. Bipolar neurons have an axon and one dendrite extending from the cell body toward opposite poles. Multipolar neurons have multiple dendrites and a single axon

- The function of a nerve is to carry messages from one part of the body to another , these messages in the form of electrical action potentials are called *impulses* , *action potentials* are transient depolarization of the membrane that result from a brief increase in the permeability of the membrane to sodium , and usually from a delayed increase in the permeability to potassium . Impulses are initiated by chemical , thermal, mechanical , or electrical stimuli .
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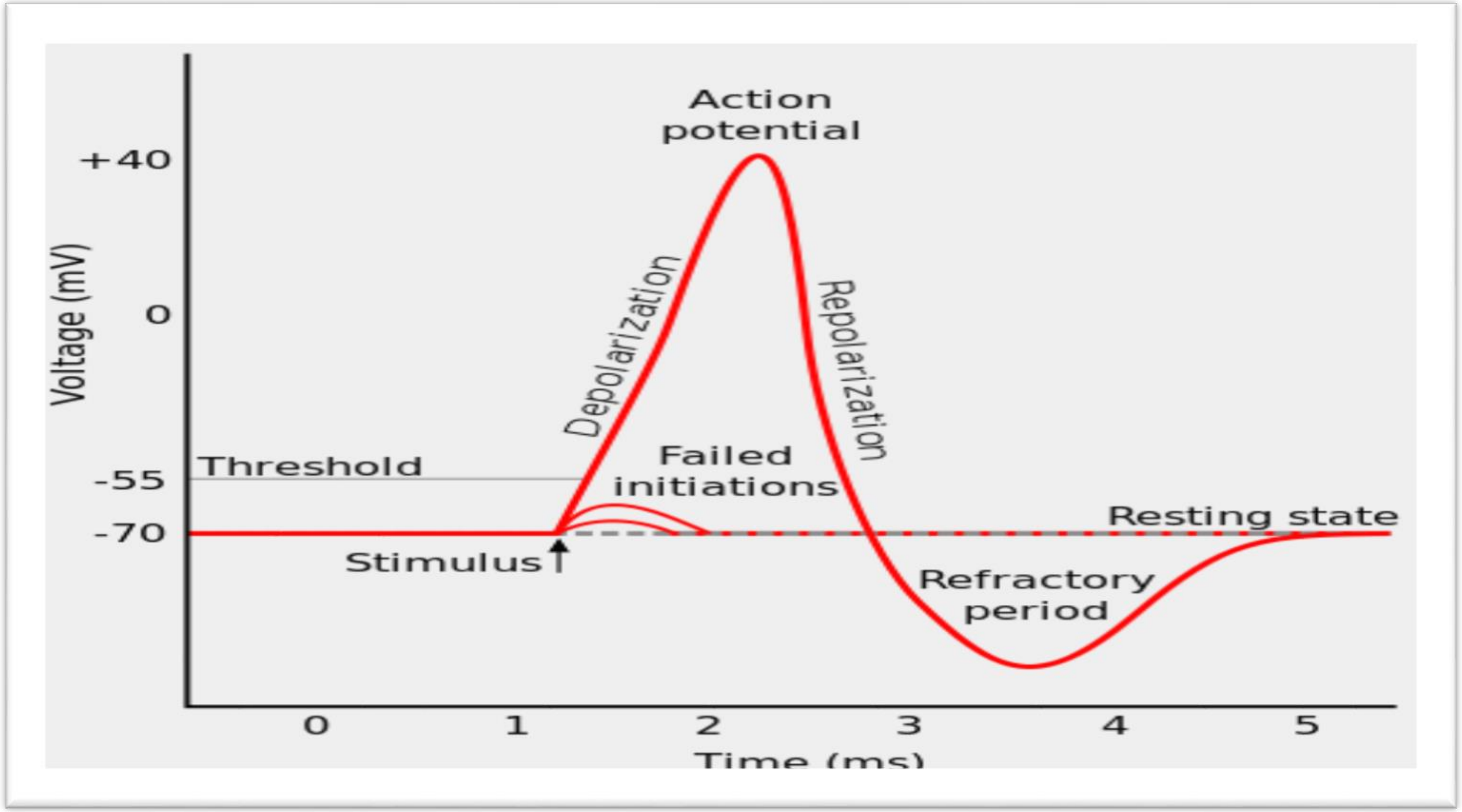
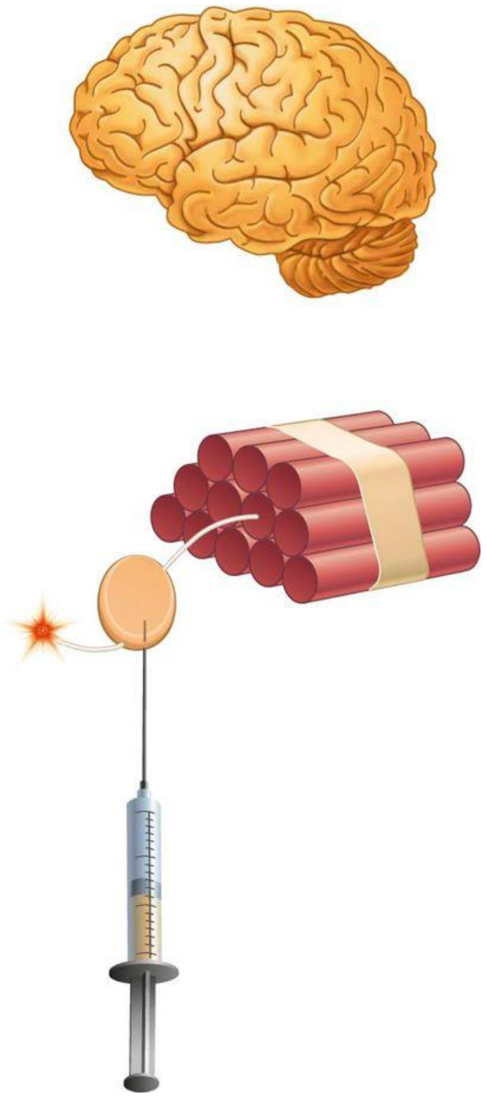
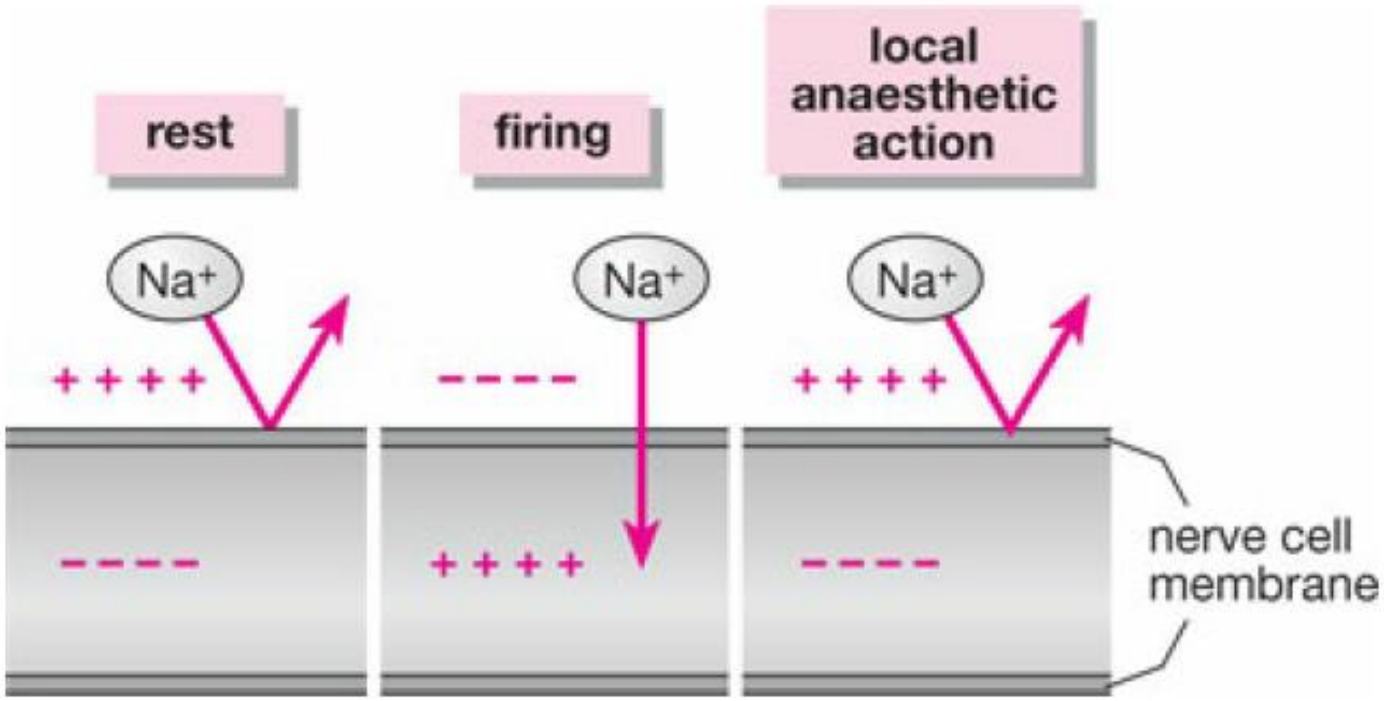


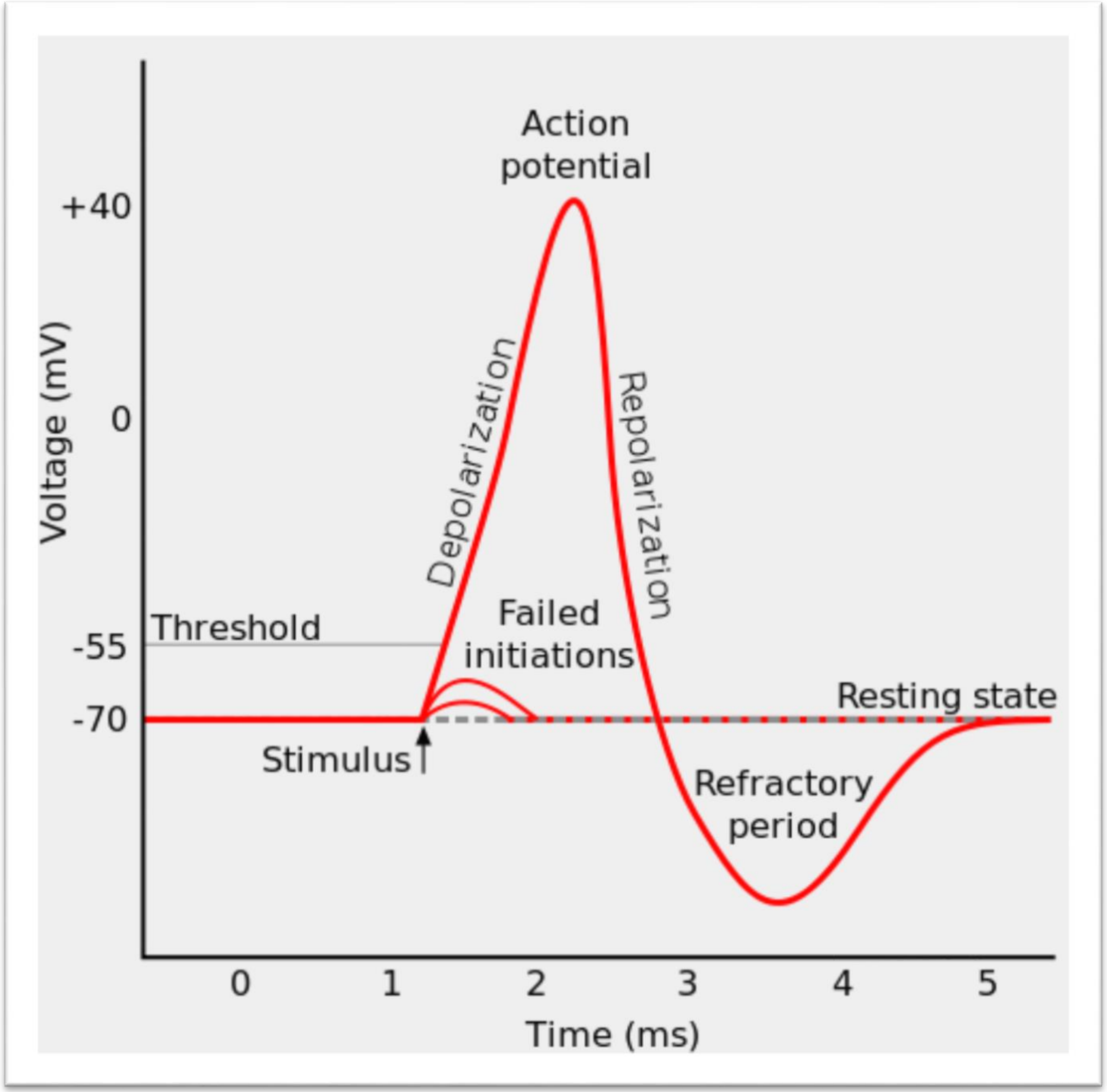


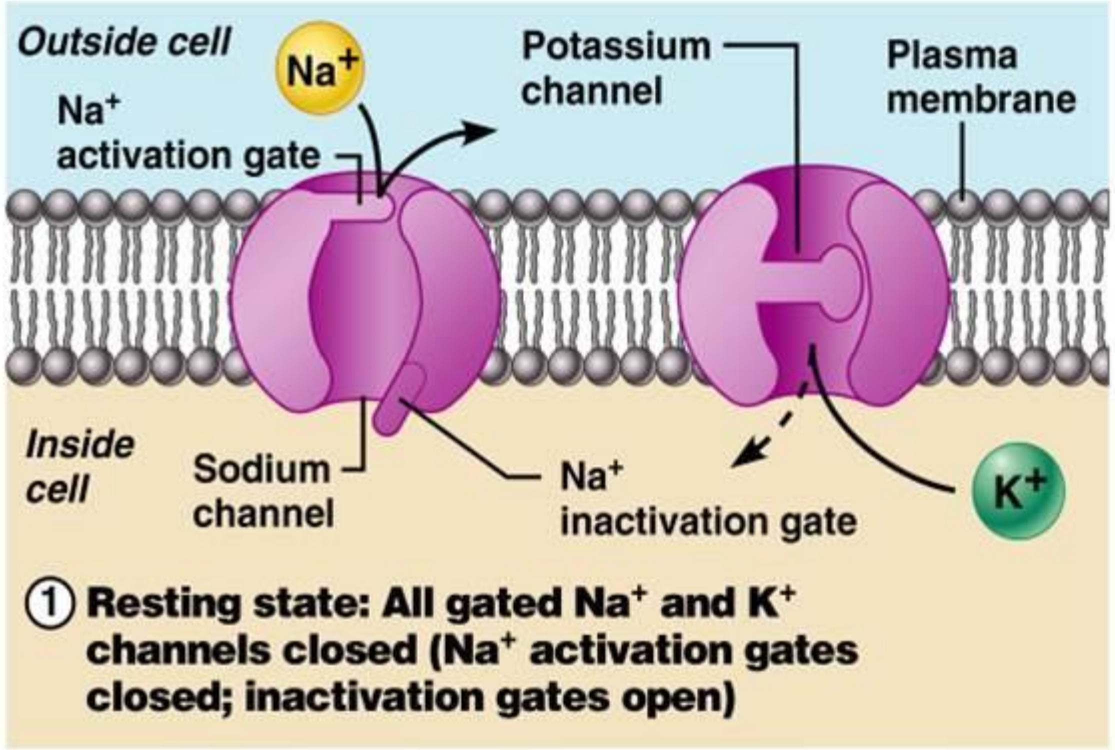
- Local anesthetic is placed at some point between the pain stimulus and the brain (dynamite).

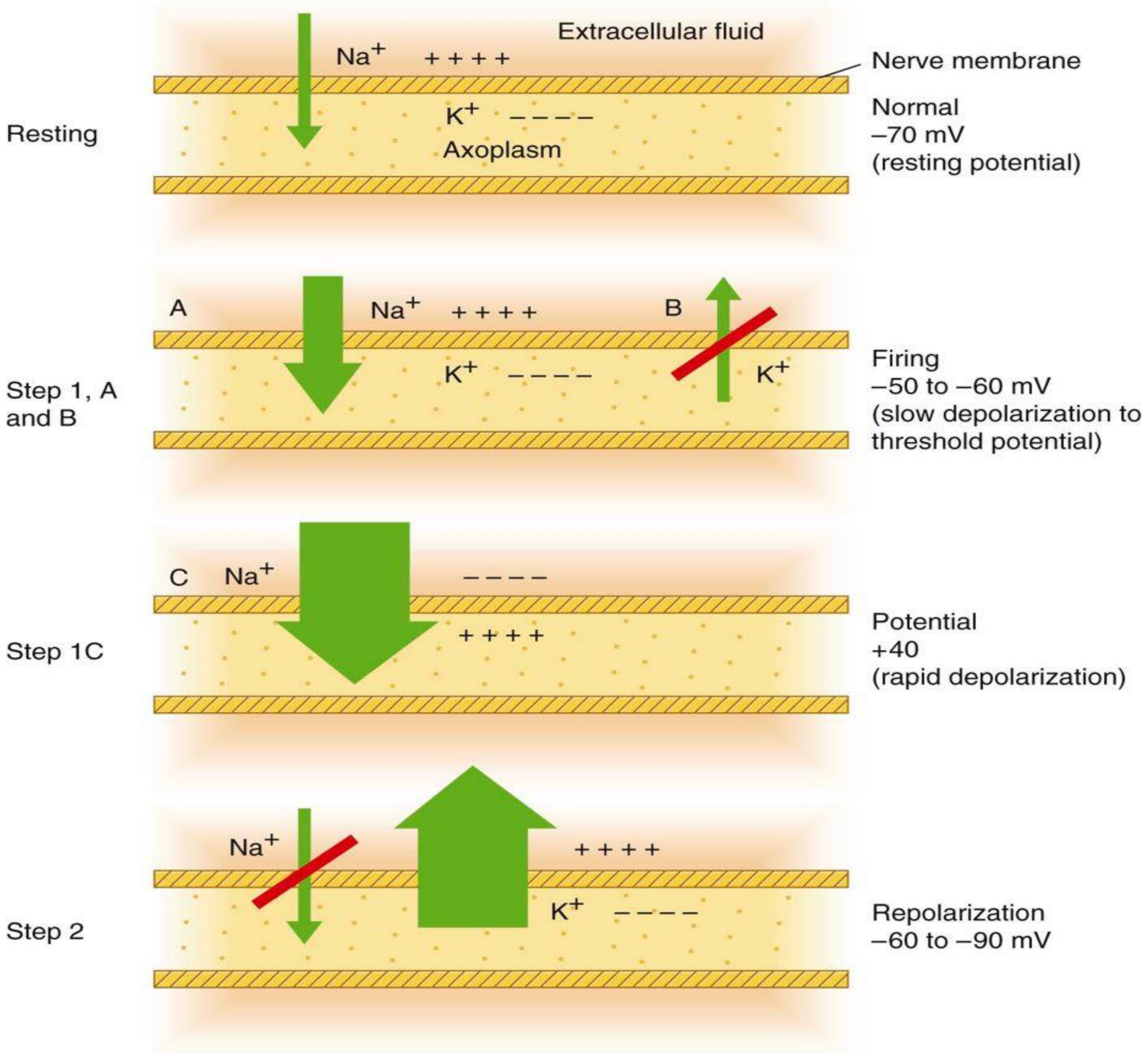
- The nerve impulse travels up to the point of local anesthetic application and then “dies,” never reaching the brain, and pain does not occur.

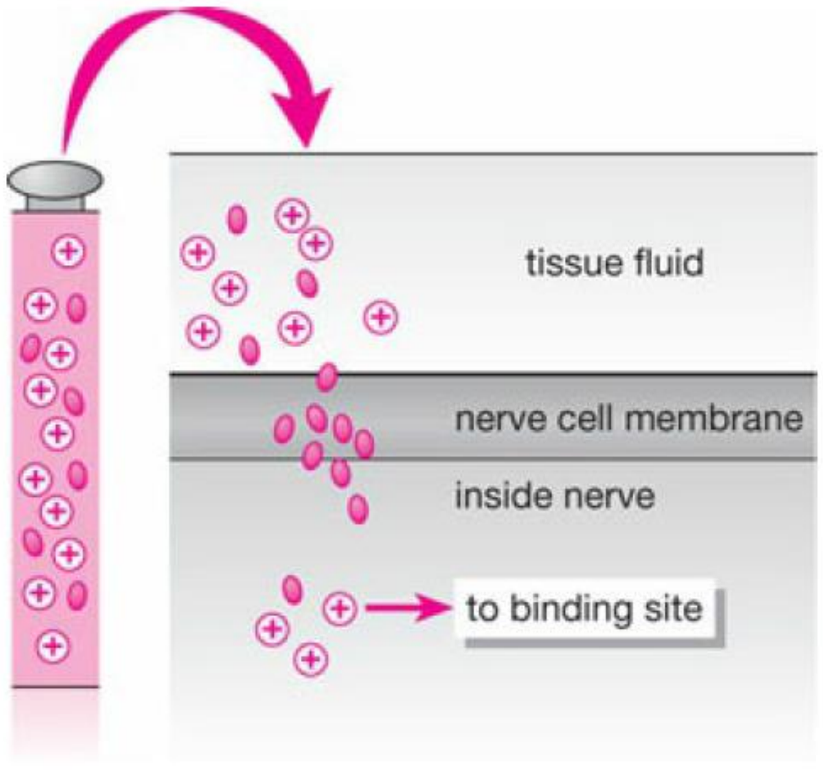


The major factor involved in nerve transmission is the differential concentration of sodium ions across the nerve membrane. Local anaesthetics block the entry of sodium into the cell and thus prevent "firing".


Action
potential
+40
0
-55
-70
Voltage (mV)
Threshold
Depolarization
Repolarization
Failed
initiations
Resting state
Stimulus
Refractory
period
0
1
2
3
4
5
Time (ms)


Outside cell
Na+
Potassium
channel
Plasma
membrane
Na+
activation gate
Inside
cell
Sodium
channel
Na+
inactivation gate
K+
1 Resting state: All gated Na+ and K+
channels closed (Na+ activation gates
closed; inactivation gates open)

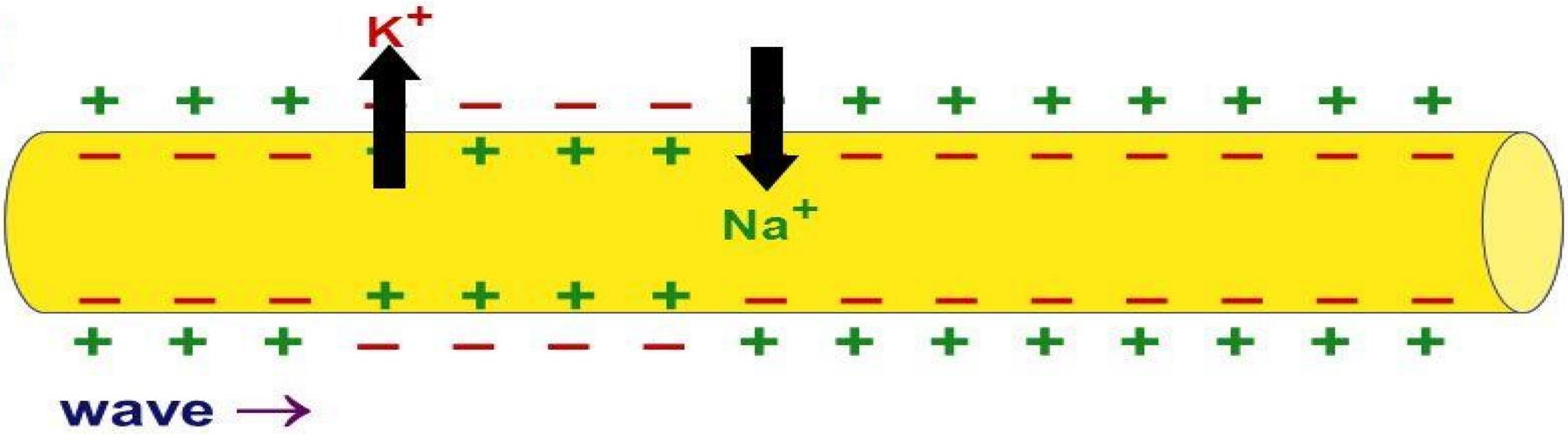

Extracellular fluid
Na+ ++++
Nerve membrane
K+ ––––
Axoplasm
Resting
Normal
–70 mV
(resting potential)
A
Na+ ++++
B
K+ ––––
K+
Step 1, A and B
Firing
–50 to –60 mV
(slow depolarization to threshold potential)
C Na+ ––––
++++
Step 1C
Potential
+40
(rapid depolarization)
Na+
++++
K+ ––––
Step 2
Repolarization
–60 to –90 mV



Local anaesthetics molecules are present in both charged and uncharged forms in solution. It is the uncharged moiety that enters the nerve cell. This then re-equilibrates to charged and uncharged forms and the charged portion binds to the specific receptor to block sodium entry.

How does a nerve impulse travel?

- Combined waves travel down neuron
    - wave of opening ion channels moves down neuron
    - signal moves in one direction → → → → →
        - flow of K^+ out of cell stops activation of Na^+ channels in wrong direction



Pharmacology of Local Anesthetics

Local anaesthetics are the most commonly used pharmaceuticals in dentistry.

Local anesthetics produce **anesthesia by inhibiting excitation of nerve endings or by blocking conduction in peripheral nerves .**

They **interfere reversibly** with the generation of **the action potential** and with **cellular impulse conduction** by **blockading the sodium channels** in the nerve cell. This results in a **local insensibility to pain stimuli**.

Local anesthetics, when used for the **management of pain**, differ from most other drugs commonly used in medicine and dentistry in one important manner. Virtually all **other drugs**, regardless of the route through which they are administered, must ultimately enter the **circulatory system in sufficiently high concentrations** (e.g., attain therapeutic blood levels in their target organ[s]) **before they can exert a clinical act**ion.

Local anesthetics, however, when used for **pain control**, *cease* to **provide a clinical effect** when they are absorbed from the **site of administration into the circulat**ion.

One prime factor involved in the termination of action of local anesthetics used for pain control is their redistribution from the nerve fiber into the **cardiovascular system (CVS)**. The presence of a local anesthetic in the circulatory system means that the drug will be **transported to every part of the body**.

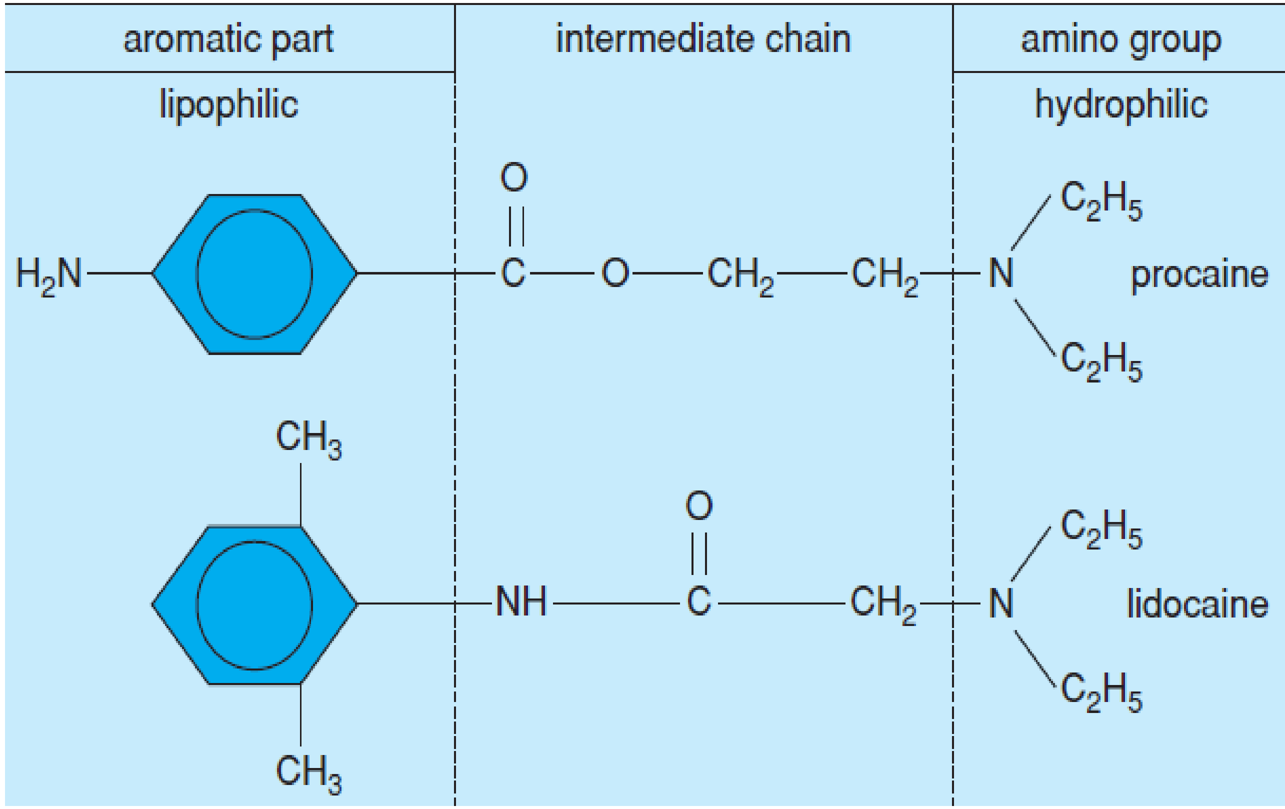
Local anesthetics have the ability to alter the functioning of some of these cells.

Pharmacokinetics of Local Anesthetics

Local anaesthetics share several common characteristics in their molecular structure . **A lipophilic group** can be identified which determines **lipid solubility**.
Another part contains a **hydrophilic group** that determines the **degree of water solubility**.

Usually the lipophilic part of the molecule is an aromatic structure that contains a **benzene ring**.

The **hydrophilic part** contains **a secondary or a tertiary amine**. Both parts, present at the **opposite ends of the molecule**, are connected by an **intermediate section**. This intermediate section consists of an **ester** or an **amide** group and a relatively short chain of **four to five carbon atoms**



Molecular structure of an ester-linked (procaine) and amide-linked local anaesthetic (lidocaine).

Local anaesthetics are distributed throughout all body tissues. In general, **tissues with a high vascular perfusion (e.g. lung) absorb local anaesthetics most rapidly while tissues with a low vascular perfusion show a slower rate of uptake**.

Plasma protein binding influences the rate of tissue uptake, the blood concentration of local anaesthetic agents following regional anaesthesia is **determined by the absorption**, **tissue redistribution**, **metabolism** and **excretion characteristics of the various agents.**

Absorption is related to the site of injection, dosage employed, use of vasoconstrictors and physicochemical profile of the specific agent.

Local anaesthetics are distributed throughout all body tissues. In general, tissues with a high vascular perfusion (e.g. lung) absorb local anaesthetics most rapidly while tissues with a low vascular perfusion show a slower rate of uptake. Plasma protein binding influences the rate of tissue uptake. Local anaesthetics are primarily bound to α-1-acid glycoproteins.

The metabolism of local anaesthetics varies according to their chemical classification. The amino esters are hydrolysed in plasma by the enzyme cholinesterase while the amino amides are primarily metabolized in the liver by microsomal enzymes.

Local anaesthetics and their metabolites are excreted primarily by the kidneys. Less than 5% of the parent compound is excreted in an unchanged form.

concomitant administration of other drugs. **The elimination** of local anaesthetics is influenced by a variety of factors such as age, clinical status of the patient, and general, neonates and geriatric patients eliminate local anaesthetics more slowly.

A disturbance of the calcium binding, with the **closure of the sodium channel as a result**. The strength of the effect of **local anaesthetics** on sodium channels is dependent on the **frequency of the action potential**.
On the one hand, when the **sodium channel is open** it is more accessible for the local anaesthetic.
On the other hand, **local anaesthetics have a higher affinity with open sodium channels**.
The total process is called **conduction blockade** or **membrane stabilisation** and **consists of a decrease in sodium conduction** and a **reduction in the rate of depolarisation**.
In addition, the threshold potential level will no longer be reached, so an action potential cannot occur anymore.
However, the resting potential, the threshold potential and the repolarisation of the cell membrane will not or hardly be affected.

Pharmacokinetics of local anesthetics :

Pharmacokinetics (PK) is defined as the **movement of drugs through the body**, whereas **Pharmacodynamics (PD)** is defined as the **body's biological response to drugs**. In other words, PK describes a **drug's absorption, distribution, metabolism, and excretion (also known as ADME)** and **PD** describes **how biological processes in the body respond to or are impacted by a drug**. Put in the simplest terms, pharmacokinetics is what the body does to the drug and pharmacodynamics is what the drug does to the body
Local anesthetics are distributed throughout all body tissues.
In general, **tissues with a high vascular perfusion (e.g. lung) absorb local anaesthetics most rapidly while tissues with a low vascular perfusion show a slower rate of uptake**.

Classification of Local Anesthetics
Esters
Esters of benzoic acid
Butacaine
Cocaine
Ethyl aminobenzoate (benzocaine)
Hexylcaine
Piperocaine
Tetracaine
Esters of *p*-aminobenzoic acid
Chloroprocaine
Procaine
Propoxycaine
Amides
Articaine
Bupivacaine
Dibucaine
Etidocaine
Lidocaine
Mepivacaine
Prilocaine
Ropivacaine
Quinoline
Centbucridine

A ccidental intra-arterial injection of a drug (e.g.,thiopental) or injection of e**pinephrine or norepinephrine** into a fingertip or toe. Intra-arterial administration of an irritating drug such as thiopental may produce **arteriospasm,** with an attendant decrease in tissue perfusion that if **prolonged could lead to tissue death**, **gangrene**, and loss of the affected limb. In this situation, procaine is administered intra-arterially in an attempt to break the arteriospasm and reestablish blood flow to the affected limb. Tetracaine, chloroprocaine, and propoxycaine also possess vasodilating properties to differing degrees but not to the degree of procaine.

Cocaine is the only local anesthetic that consistently produces vasoconstriction..

The initial action of cocaine is vasodilation, followed by an intense and prolonged vasoconstriction.

It is produced by inhibition of the uptake of catecholamines (especially norepinephrine) into tissue binding sites.

This results in an excess of free norepinephrine,leading to a prolonged and intense state of vasoconstriction.

This inhibition of the reuptake of norepinephrine has not been demonstrated with other local anesthetics, such as **lidocaine and bupivacaine**.

A significant clinical effect of vasodilation is an increasein the rate of absorption of the local anesthetic into the blood, thus decreasing the duration and quality (e.g., depth) of pain control, while increasing the anesthetic blood (or plasma) concentration and its potential for overdose (toxicreaction). The rates at which local anesthetics are absorbed into the bloodstream and reach their peak blood level vary according to the route of administration

Plasma protein binding influences the rate of tissue uptake .When injected into soft tissues, local anesthetics exert pharmacologic action on blood vessels in the area.

All local anesthetics possess a **degree of vasoactivity**, most producing **dilation of the vascular bed** into which they are deposited, although the **degree of vasodilation may differ**, and some (e.g., **cocaine**). may produce **vasoconstriction.** To some degree, these effects may be **concentration dependent**.

Relative **vasodilating va**lues of **amide local anesthetics .Ester local anesthetics** are also potent **vasodilating drugs**.

Procaine, the most potent vasodilator among local anesthetics is, on rare occasion, injected clinically to induce **vasodilation** when peripheral blood flow has been compromised.

Oral Route OF LA

With the exception of cocaine, local anesthetics are absorbed poorly, if at all, from the gastrointestinal tract after oral administration. In addition, most local anesthetics (especially lidocaine) undergo a significant hepatic first-pass effect after oral administration. After absorption of lidocaine from the gastrointestinal tract into the enterohepatic circulation, a fraction of the drug dose is carried to the liver, where approximately 72% of the dose is biotransformed into inactive metabolites.

This has seriously hampered the use of lidocaine as an oral antidysrhythmic drug. In 1984 Astra Pharmaceuticals and Merck Sharp & Dohme introducedan analogue of lidocaine, tocainide hydrochloride, that is effective orally.

Topical Route OF LA

Local anesthetics are absorbed at differing rates after application to mucous membrane: in the tracheal mucosa, absorption

Distribution:

Once **absorbed into the blood**, local anesthetics are **distributed throughout the body to all tissues** .

Highly perfused organs (and areas), such as the **brain, head**, liver, **kidneys**, **lungs,** and **spleen**, initially will have **higher anesthetic blood levels than less highly perfused organs.**

Skeletal muscle, although not as highly perfused as these areas, contains the greatest percentage of local anesthetic of any tissue or organ in the body because it constitutes the largest mass of tissue in the body The plasma concentration of a local anesthetic in certain target organs has a significant bearing on the potential toxicity of the drug.

The blood level of the local anesthetic is influenced by:

1. the rate at which the drug is absorbed into the **CVS**
2. the rate of distribution of the drug from the vascular compartment to the tissues (more rapid in healthy patients than in those who are medically compromised [e.g., congestive heart failure], thus leading to lower blood levels in healthier patients).
3. elimination of the drug through metabolic or excretory pathways .**The latter two factors serve to decrease the blood level of the local anesthetic.**

Time to Achieve Peak Blood Level

Route	Time (min)
Intravenous	1
Topical	5 (approximately)
Intramuscular	5–10
Subcutaneous	30–90

Percentages of Cardiac Output Distributed to Different Organ Systems

Region	Percentage of Cardiac Output Received
Kidney	22
Gastrointestinal system, spleen	21
Skeletal muscle	15
Brain	14
Skin	6
Liver	6
Bone	5
Heart muscle	3
Other	8

Modified from Mohrman DE, Heller LJ. *Cardiovascular Physiology*. 7th ed. New York: Lange Medical Books/McGraw-Hill; 2010.)

The rate at which a local anesthetic is removed from the blood is described as its *elimination half-life*.

Simply stated, the elimination half-life is the time necessary for a 50% reduction in the blood level (one half-life is equivalent to 50% reduction; two half-lives are equivalent to 75% reduction; three half-lives are equivalent to 87.5% reduction; four half-lives are equivalent to 94% reduction; five half-lives are equivalent to 97% reduction; six half-lives are equivalent to 98.5% reduction) .

All local anesthetics readily cross the blood-brain barrier.They also readily cross the **placenta and enter the circulatory system of the developing fetus**

Half-Life of Local Anesthetics

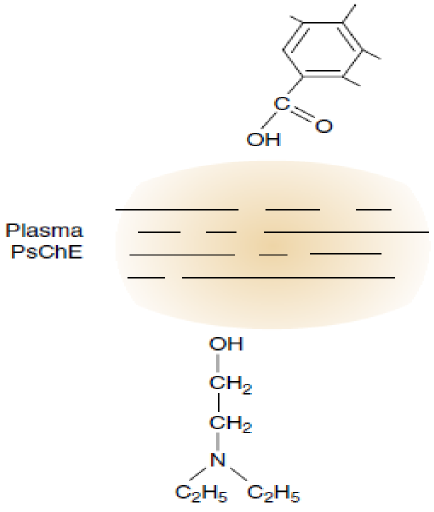
Drug	Half-Life (h)
Chloroprocaine[a]	0.1
Procaine[a]	0.1
Tetracaine[a]	0.3
Articaine[b]	0.5
Cocaine[a]	0.7
Prilocaine[b]	1.6
Lidocaine[b]	1.6
Mepivacaine[b]	1.9
Ropivacaine[b]	1.9
Etidocaine[b]	2.6
Bupivacaine[b]	3.5
Propoxycaine[a]	NA

[a]Ester.
[b]Amide.
NA, Not available.

Metabolism (Biotransformation,Detoxification)

A significant difference between the two major groups of local anesthetics, the esters and the amides, is the means by which the body biologically transforms the active drug into one that is pharmacologically inactive.

Metabolism (alsoknown as *biotransformation* or *detoxification*) of local anesthetics is important because the overall toxicity of a drug depends on a balance between its rate of absorption into the bloodstream at the site of injection and its rate of removal from the blood through the processes of tissue uptake and metabolism.



Metabolic hydrolysis of procaine. *PsChE*, Pseudocholinesterase. (From Tucker GT. Biotransformation and toxicity of local anesthetics.,

The aromatic part of the ester-type local anaesthetics is derived from **para amino benzoic acid (PABA**).
Ester anaesthetics are **metabolised in plasma** by the enzyme **pseudo cholinesterase**, which generates PABA **analogues and amino alcohol**.
The PABA analogues are excreted in the **urine mainly unaltered**; the **amino alcohol** is further **metabolised** in the **liver**.
Approximately 2% of ester anaesthetics are excreted unchanged by the kidneys. The PABA analogues are responsible for the **allergic reactions** that frequently occur with the use of **local anaesthetics of the ester type**.

Anaesthetics of the amide type are **metabolised in the liver** first by the **cytochrome P450 system**. This reaction is followed by **conjugation, resulting in highly water-soluble metabolites** that are excreted by the kidneys.
Between 70 and 90% of the amide anaesthetic is metabolised, and **10–30% is excreted by the kidneys unchanged**.

The velocity of **degradation in the liver** is reciprocally related to **the toxicity**.

Prilocaine is metabolised fastest and is **consequently the least toxic amide-type local anaesthetic**.

Patients with severe liver insufficiency degrade amide-type local anaesthetics at a delayed rate, which increases the **risk of a toxic effect and spontaneous respiration resumes**.
Persons with **atypical pseudocholinesterase are unable to hydrolyze succinylcholine at a normal rate, resulting in the duration of apnea being prolonged.**

Atypical pseudocholinesterase is a **hereditary trait**. Any familial history of adverse events during general anesthesia should be **carefully evaluated** by the doctor before **dental care commences**.

A confirmed or strongly suspected history, in the patient or biological family, of atypical pseudocholinesterase represents a relative contraindication to **administration of ester-type local anesthetics.**

There are **absolute** and **relative contraindications to the administration** of drugs.
An *absolute contraindication* implies that under no circumstance should the drug in question be administered to the patient as the possibility of **potentially toxic** or **lethal reactions is increased**, whereas a *relative contraindication* means that the drug in question may be administered to the patient after careful weighing of the **risk associated** with use of the drug versus the potential benefit to be gained, and if an acceptable alternative drug is
not available. **However, the smallest clinically effective dose should always be used because the likelihood of adverse reaction to this drug is increased in the patient.**

Amide Local Anesthetics

The biotransformation of amide local anesthetics is more complex than that of ester local anesthetics. The primary site of biotransformation of amide local anesthetics is the liver. Virtually the entire metabolic process occurs in the liver for lidocaine, mepivacaine, etidocaine, and bupivacaine.
Prilocaine undergoes primary metabolism in the liver, with some also possibly occurring in the lung.
Articaine, a hybrid molecule containing **both ester and amide components**, undergoes metabolism in **both the blood (primarily)** and the **liver.** The rates of biotransformation of **lidocaine, mepivacaine, etidocaine, and bupivacaine are similar.**

Therefore liver function and hepatic perfusion significantly influence the rate of biotransformation of an amide local anesthetic. Approximately 70% of a dose of injected lidocaine undergoes biotransformation in patients with normal liver function.
Patients with lower-than-usual hepatic blood flow (hypotension, congestive heart failure). or poor liver function (cirrhosis) are **unable to biotransform amide local anesthetics at a normal rate**. This slower-than-normal biotransformation results in **higher anesthetic blood levels** and **increased risk of toxicity**.

Significant liver dysfunction (American Society of Anesthesiologists [ASA] physical status classification system class 4 or 5) or **heart failure (ASA class 4 or 5)** represents a **relative contraindication to the administration of amide local anesthetic drugs**.

Articaine has a shorter half-life than other **amides (27 minutes vs. 90 minutes)** because a significant portion of its biotransformation occurs in the blood by the **enzyme plasma cholinesterase**. The biotransformation products of certain local anesthetics can possess significant clinical activity if they are permitted to accumulate in the blood.

This may be seen in renal or cardiac failure and during periods of prolonged drug administration. A clinical example is the production of methemoglobinemia in patients receiving large doses of prilocaine

Prilocaine, the parent compound, does not produce methemoglobinemia, but orthotoluidine, a primary metabolite of prilocaine, does induce the formation of methemoglobin, which is responsible for methemoglobinemia.
When methemoglobin blood levels become elevated, clinical signs and symptoms are observed.

Another example of **pharmacologically active meta**bolites is the sedative effect occasionally observed after **lidocaine** administration.
Lidocaine does not **produce sedation**; however, **two metabolites**— **monoethylglycinexylidide** and **glycinexylidide**— are thought to be responsible for this clinical action. The metabolic pathways of lidocaine and prilocaine are shown in

Hydrolysis Rate of Esters

Drug	Rate of Hydrolysis (μmol/mL/h)
Chloroprocaine	4.7
Procaine	1.1
Tetracaine	0.3

Lidocaine Disposition in Various Groups of Patients

Group	Lidocaine Half-Life (h)	Mean Total Body Clearance (mL/kg/min)
Normal	1.8	10
Heart failure	1.9	6.3
Hepatic disease	4.9	6
Renal disease	1.3	13.7

Data from Thomson PD, Melmon KL; Richardson JA, et al. Lidocaine pharmacokinetics in advanced heart failure, liver disease, and renal failure in humans. *Ann Intern Med*. 1973;78:499–513.

Excretion

The kidneys are the primary excretory organ for both the local anesthetic and its metabolites. A proportion of a given dose of local anesthetic is excreted unchanged in the urine. This proportion varies according to the drug. Esters appear only in very small concentrations as the parent compound in the urine because they are hydrolyzed almost completely in the plasma. Procaine appears in the urine as PABA (90%) with 2% unchanged. Ten percent of a cocaine dose is found in the urine unchanged.

Amides are usually present in the **urine as the parentcompound** in a greater percentage than the esters, primarily because of their more complex process of biotransformation.
Although the percentages of parent drug found in urine differ from study to study, **less than 3% lidocaine**, 1% mepivacaine, and **1% etidocaine** is found **unchanged in the urine**.
Patients with significant **renal impairment may be unable to eliminate the parent local anesthetic compound** or its major metabolites from the blood, resulting in **slightly elevated blood levels** and therefore **increased potential for toxicity**. This may occur with the **esters or amides**, and is especially likely **with cocaine**. Thus significant renal disease (ASA class 4 or 5) represents a relative contraindication to the administration of local anesthetics. This includes patients undergoing **renal dialysis** and those with **chronic glomerulonephritis** or pyelonephritis

Systemic Actions of Local Anesthetics:

Local anesthetics are chemicals that reversibly block action potentials in all excitable membranes .

The central nervous system (CNS) and the CVS therefore are especially susceptible to their actions. Most of the systemic actions of local anesthetics are related to their blood or plasma level in a target organ (CNS, CVS). The higher the

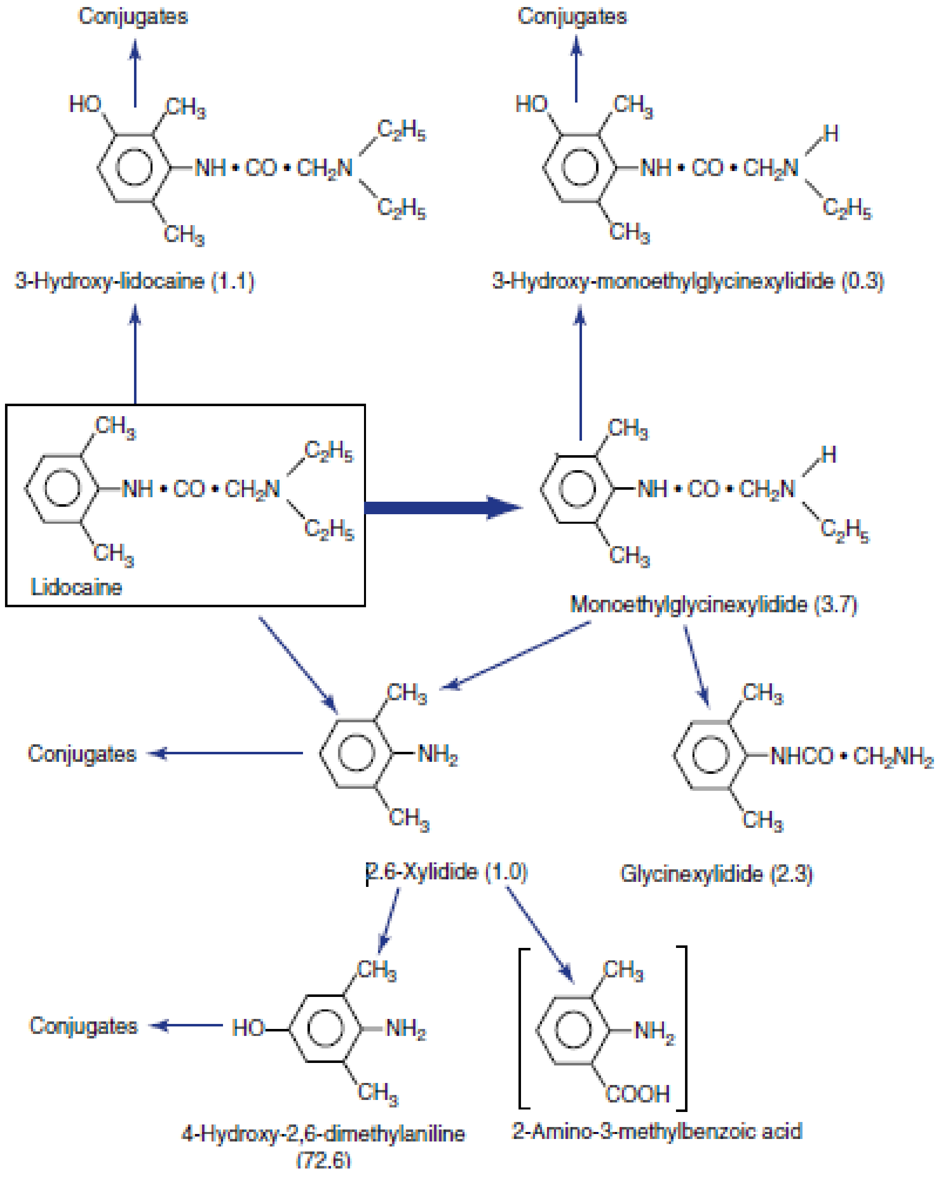
blood level, the greater will be the clinical action.

Centbucridine (a quinoline derivative) has proved to be five to eigh times as potent a local anesthetic as lidocaine,with an equally rapid onset of action and an equivalent duration.

Centbucridine works by reducing the **excitations in the nerve ending** as well as **prohibiting any further electrical conduction in the peripheral nerves**, in and around the **area where the anesthetic is applied**.

The drug is able to achieve this by **reducing the influx of sodium ions through cell membranes of neuron cells by closing the voltage gated sodium channels** and other **sodium specific ion channels present in these cells.**

Temporary side effects like **dizziness**, **headache**, **pins** and **needles**, twitchiness of **the muscles** and **continued numbness** in the area may occur upon usage of **Centbucridine**



Metabolic pathways of lidocaine. Percentages of the dose found in urine are indicated in parentheses.

It is great importance is the finding that it does not adversely affect the CNS or CVS, except in very high doses. It has been used both by injection and by topical application in ophthalmic surgery but not, as yet, in dentistry. Local anesthetics are absorbed from their site of administration into the circulatory system, which effectively dilutes them and carries them to all cells of the body.
The resulting **blood level of the local anesthetic** depends on **its rate of uptake** from the site of administration into the circulatory system (**increasing the blood level**), and on the rates of distribution in tissue and biotransformation (in **the liver**), processes that remove the drug from the blood (**decreasing the blood level**).

Central Nervous System:

Local anesthetics **readily cross the blood-brain barrier**. Their pharmacologic action on the CNS is **one of depression**.
At low (**therapeutic, nontoxic**) blood levels, no CNS effects of any clinical significance l
At higher (**toxic,overdose**) levels the primary clinical manifestation is **a generalized tonic-clonic convulsion.** Between these two extremes exists a spectrum of other clinical signs and symptoms.
It is important to note that individual patients may respond either **more positively** or **more negatively** to **doses** or **blood levels of a drug** that are considered to be **"normal" (e.g., within an acceptable range)**.
However, approximately 15% of persons are "**hyperresponders**"to an "average" dose of a given drug.

Preconvulsive Signs and Symptoms of Central Nervous System Toxicity

Signs (Objectively Observable)	Symptoms (Subjectively Felt)
Slurred speech	Numbness of tongue and circumoral region
Shivering	Warm, flushed feeling of skin
Muscular twitching	Pleasant dreamlike state
Tremor of muscles of face and distal extremities	
Generalized lightheadedness	
Dizziness	
Visual disturbances (inability to focus)	
Auditory disturbance (tinnitus)	
Drowsiness	
Disorientation	

Within this 15% there is a normal distribution curve, so yet another 15% of these persons would be considered as "**extreme hyperresponders**." In such an individual an "**average**" or "**normal**" dose of a drug could result in the manifestation of **significant signs and symptoms of toxicity (overdose).**

Anticonvulsant Properties

The "classic" overdose reaction to a local anesthetic is a **generalized tonic-clonic convulsion**. Some local anesthetics (e.g., procaine, lidocaine, mepivacaine, prilocaine, and even cocaine) have demonstrated anticonvulsant properties.

These occur at a blood level considerably below that at which the same drugs produce seizure activity. Values for anticonvulsive blood levels of **lidocaine** are shown in **Procaine**, **mepivacaine**, and **lidocaine** have been used intravenously to **terminate or decrease the duration of both grand mal and petit mal seizures**.
Of the local anesthetics tested, ***lidocaine appeared to be the most promising anticonvulsant***
as it exhibits the **widest therapeutic range:** a threefold
margin between **seizure-protecting** and seizure-inducing doses. **The anticonvulsant blood level of lidocaine (about 0.5 to 4 µg/mL) is very close to its cardio therapeutic range**
It has been demonstrated to be effective in temporarily arresting seizure activity in most human epileptic patients.
It was especially effective in **interrupting status epilepticus at therapeutic doses** of 2 to 3 mg/kg when given intravenously at a rate of 40 to 50 mg/min.
This use of local anesthetics has been essentially dormant since then as more effective anticonvulsants have been introduced into clinical practice.

Mechanism of Anticonvulsant Properties of Local Anesthetics

Epileptic patients possess hyperexcitable cortical neurons at a site within the brain where the convulsive episode originates
(called the *epileptic focus*). Local anesthetics, by virtue of their depressant actions on the CNS, raise the seizure threshold by decreasing the excitability of these neurons, thereby preventing or terminating seizures.

Preconvulsive Signs and Symptoms

With a further increase in the blood level of the local anesthetic to above its therapeutic level, adverse reactions may be observed. **Because the CNS is much more susceptible than other systems to the actions of local anesthetics**, it is not surprising that the initial **clinical signs and symptoms of overdose (toxicity) are of CNS origin**.

With lidocaine, this second phase is observed at a level between **4.5 and 7 µg/ mL** in the average normal **healthy patient**.

Initial clinical signs and symptoms of **CNS toxicity are usually excitatory in nature** .

Lidocaine and procaine differ somewhat from other local anesthetics in that the usual progression of signs and symptoms just noted may not be seen.

Lidocaine and procaine frequently produce an initial mild sedation or drowsiness (more common with lidocaine). Because of this potential, “air crew/SOD (special operational duty) members cannot fly for at least 8 hours after receiving a **local or regional anesthetic agent**.” Sedation may develop in place of the excitatory signs. If excitation or sedation is observed during the **first 5 to 10 minutes after intraoral administration of a local anesthetic**, it **should serve as a warning to the clinician of a rising local anesthetic blood level** and the **possibility (if the blood level continues to rise) of a more serious reaction, including a generalized convulsive episode**.

Convulsive Phase

Further increase in local anesthetic blood level leads to signs and symptoms consistent with a generalized tonic-clonic convulsive episode.

Signs & Symptoms of Local Anesthetic Overdose

Minimal to Moderate Overdose Levels

Signs
- Talkativeness
- Apprehension
- Excitability
- Slurred speech
- Generalized stutter, leading to muscular twitching and tremor in the face and distal extremities
- Euphoria
- Dysarthria
- Nystagmus
- Sweating
- Vomiting
- Failure to follow commands or be reasoned with
- Elevated blood pressure
- Elevated heart rate
- Elevated respiratory rate

Moderate to High Overdose Levels

Tonic-clonic seizure activity followed by
- Generalized central nervous system depression
- Depressed blood pressure, heart rate, and respiratory rate

Symptoms (Progressive With Increasing Blood Levels)

- Lightheadedness and dizziness
- Restlessness
- Nervousness
- Sensation of twitching before actual twitching is observed (see "Generalized stutter" under "Signs")
- Metallic taste
- Visual disturbances (inability to focus)
- Auditory disturbances (tinnitus)
- Drowsiness and disorientation
- Loss of consciousness

From Malamed SF. *Medical Emergencies in the Dental Office*. 7th ed. St Louis: Mosby; 2015.

Vasoconstrictors

Vasoconstrictors like adrenaline in local anaesthetics are associated with more drug interactions than any other drug in Dentistry with an incidence of adverse reactions ranging from 2.5%-11%. Therefore, understanding the **physiological** and **pharmacological effects**, **interactions with other drugs**, and **dosages are important in day to day dental practice**.

Local anaesthetics are vasodilators, hence the addition of a **vasoconstrictor like adrenaline provides the following advantages: improves the anaesthetic onset** and **duration**, **reduces bleeding**, and **decreases the systemic absorption rate of local anaesthetics by reducing the plasma concentration**.

However, **adrenaline** is unstable and therefore an **antioxidant is added to prevent it oxidizing**. Sodium bisulphite is the **preservative most commonly added to local anaesthetics**.

Of course, **patients allergic to sulphites will now react to a local anaesthetic containing sodium bisulphites**

Vasoconstrictors are extremely important in the clinical use of local anaesthetics. Without these compounds, the clinical use of local anaesthetics in dentistry would be **hampered by their limited duration of action**, as most anaesthetics **produce vasodilatation**. Exceptions are **mepivacaine and prilocaine**.
All clinically effective injectable local anesthetics are vasodilators.

The degree of vasodilation ranges from significant (**procaine)** to minimal (**prilocaine, mepivacaine**) as well as with both the injection site and individual patient response

After deposition of a local anesthetic into tissues, blood vessels (arterioles and capillaries primarily) in the **area dilate**, resulting in increased perfusion at the site, leading to the following reactions:
1. an increased rate of absorption of the local anesthetic into the cardiovascular system, which in turn removes it from the injection site (redistribution of the drug);
2. higher plasma levels of the local anesthetic, with an attendant increase in the risk of local anesthetic toxicity (overdose);
3. decrease in both the depth and the duration of anesthesia because the local anesthetic is removed from the site of injection more rapidly;
4. increased bleeding at the site of treatment as a result of increased perfusion.

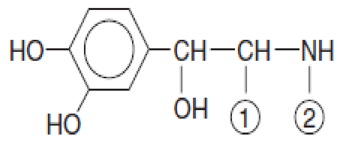
Vasoconstrictors are drugs that **constrict blood vessels** and **thereby control tissue perfusion**. They are added to local anesthetic solutions to oppose the inherent vasodilatory actions of the local anesthetics.
Vasoconstrictors are important additions to a local anesthetic solution for the following reasons:
1. By constricting blood vessels, vasoconstrictors decrease blood flow (perfusion) to the site of drug administration.
2. Absorption of the local anesthetic into the cardiovascular system is slowed, resulting in lower anesthetic blood levels. compares blood levels of local anesthetic both with and without a vasoconstrictor.
3. Local anesthetic blood levels are lowered, thereby decreasing the risk of local anesthetic toxicity from over administration of the drug. (**Overdose from rapid intravascularinjection can still occur.**)
4. More local anesthetic diffuses into the nerve, where it remains longer, thereby increasing (in some cases significantly,

Effects of a Vasoconstrictor (Epinephrine 1:200,000) on Peak Local Anesthetic Levels in Blood

		Peak Level (µg/mL)	
Local Anesthetic	Dose (mg)	Without Vasoconstrictor	With Vasoconstrictor
Mepivacaine	500	4.7	3
Lidocaine	400	4.3	3
Prilocaine	400	2.8	2.6
Etidocaine	300	1.4	1.3

Data from Cannall H, Walters H, Beckett AH, Saunders A. Circulating blood levels of lignocaine after peri-oral injections. *Br Dent J.* 1975;138:87–93.

in others minimally4) the duration of action of most local anesthetics.
5. Vasoconstrictors decrease bleeding at the site of administration.
Their inclusion in the local anesthetic solution is useful when increased bleeding is anticipated (e.g., during a surgical procedure).
The vasoconstrictors commonly used in conjunction with injected local anesthetics are chemically identical or similar to the sympathetic nervous system mediators epinephrine and norepinephrine.
The actions of the vasoconstrictors so resemble the response of adrenergic nerves to stimulation that they are classified as sympathomimetic, or adrenergic, drugs. These drugs have many clinical actions besides vasoconstriction.Sympathomimetic drugs may also be classified **according to their chemical structure and mode of action**



	1	2
Epinephrine	H	CH_3
Levonordefrin	CH_3	H
Norepinephrine	H	H

Chemical Structure

Classification of sympathomimetic drugs by chemical structure is related to the presence or absence of a catechol nucleus. Catechol is also known as ***o*-dihydroxybenzene**. Sympathomimetic drugs that have hydroxyl (OH) substitutions at the third and fourth positions of the aromatic ring are termed ***catechols.***
If they also contain an amine group (NH_2) attached to the aliphatic side chain, they are called ***catecholamines.***
Epinephrine, **norepinephrine**, and **dopamine** are the naturally **occurring catecholamines of the sympathetic nervous system.**
Isoproterenol and **levonordefrin** are **synthetic catecholamines**.

Vasoconstrictors that do not possess OH groups at the third and fourth positions of the aromatic molecule are not catechols but are amines because they have an NH_2 group attached to the aliphatic side chain.

Therefore, the vasoconstrictors adrenaline (also known as **epinephrine**) and **felypressin** are added to the local anaesthetics used in dentistry.

Adrenaline is an **endogenous compound**, released into the blood by the **adrenal medulla**, with a half-life of only a **few minutes**.
Nervous or anxious people may have increased blood levels of adrenaline from fear of dental treatment. Vasoconstriction by adrenaline is achieved by **stimulation of α1-adrenergic receptors of the smooth muscles of the vessel wall**. **The maximum dose of adrenaline for adults is 200 µg**.

Felypressin (octapressin) is a synthetic analogue of poly peptide vasopressin vasoconstrictor , derived from **vasopressin (antidiuretic hormone).** The vasoconstrictive activity of **felypressin** mainly originates from inducing constriction of the venous part of the circulation.
The duration of action is longer than that of adrenaline For adults, **the maximum dose of felypressin is 5.4 µg**.

Modes of Action:

Three categories of sympathomimetic amines are known:
direct-acting drugs, which exert their action directly on **adrenergic receptors**; **indirect-acting drugs**, which act by releasing norepinephrine from adrenergic **nerve terminals**; and **mixed-acting drugs,** with both **direct** and **indirect actions**

Categories of Sympathomimetic Amines

Direct Acting	Indirect Acting	Mixed Actin
Epinephrine	Tyramine	Metaraminol
Norepinephrine	Amphetamine	Ephedrine
Levonordefrin	Methamphetamine	
Isoproterenol	Hydroxyamphetamine	
Dopamine		
Methoxamine		
Phenylephrine		

Catecholamines	Noncatecholamines
Epinephrine	Amphetamine
Norepinephrine	Methamphetamine
Levonordefrin	Ephedrine
Isoproterenol	Mephentermine
Dopamine	Hydroxyamphetamine
	Metaraminol
	Methoxamine
	Phenylephrine

Adrenergic Receptors

Adrenergic receptors are found in most tissues of the body.

The concept of adrenergic receptors was proposed by Ahlquist in 1948, and is well accepted today.

Ahlquist recognized **two types of adrenergic receptors**, termed ***alpha*** **(α)** and ***beta*** **(β)**, on the basis of **inhibitory** or **excitatory actions** of **catecholamines on smooth muscle**.

Stimulation of α receptors by a **sympathomimetic drug usually produces a response that includes contraction of smooth muscle in blood vessels (vasoconstriction).** On the basis of differences in their **function and location**, **α receptors** have been subcategorized.

Whereas **α1 receptors** are excitatory postsynaptic, **α2 receptors** are inhibitory postsynaptic.

Stimulation of **β receptors** produces smooth muscle relaxation (vasodilation and bronchodilation) and **cardiac stimulation (increased heart rate and strength of contraction).**

Beta receptors are further divided into β1 and β2 receptors:

β1 receptors are found in the heart and small intestine and are responsible for cardiac stimulation and lipolysis;

B2 receptors, found in the bronchi of the lungs, **vascular beds**, and **uterus**, produce **bronchodilation** and **vasodilation.** the differences in degrees of α and β receptor activity of three commonly used vasoconstrictors.

lists the systemic effects, based on α and β receptor activity, of **epinephrine** and **norepinephrine.**

Adrenergic Receptor Activity of Vasoconstrictors

Drug	α_1	α_2	β_1	β_2
Epinephrine	+++	+++	+++	+++
Norepinephrine	++	++	++	+
Levonordefrin	+	++	++	+

Relative potency of drugs is indicated as follows: +++, high; ++, intermediate; and +, low.

From Jastak JT, Yagiela JA, Donaldson D. *Local Anesthesia of the Oral Cavity*. Philadelphia: WB Saunders; 1995.

Release of Catecholamines

Other sympathomimetic drugs, such as **tyramine** and **amphetamine**, act indirectly by causing the release of the **catecholamine norepinephrine** from storage sites in

Adrenergic nerve terminals. In addition, these drugs may exert direct action on α and β receptors. The clinical actions of this group of drugs therefore are **quite similar** to the actions of norepinephrine. Successively repeated doses of these drugs will be less effective than those given previously because of depletion of **norepinephrine from storage sites**. This phenomenon is termed ***tachyphylaxis*** and is not seen with drugs that act directly on **adrenergic receptors**.

Dilutions of Vasoconstrictors

The use of vasoconstrictors can have **negative effects**, both **locally** and **systemically.** The dilution of vasoconstrictors is commonly referred to as a ***ratio*** **(e.g., 1:1000**). Because the maximum doses of vasoconstrictors are presented in **milligrams,** or more commonly today in **micrograms**, the following interpretations should enable the reader to convert these terms readily:

A concentration of 1:1000 means that 1 g (1000 mg) of drug is contained in 1000 mL of solution.

Therefore, a 1:1000 dilution contains 1000 mg in 1000 mL or 1.0 mg per milliliter of solution (1000 μg/mL). Vasoconstrictors, as used in dental local anesthetic solutions,are much more dilute than the 1:1000 concentration described in the preceding paragraph. To produce these more dilute, clinically safer, yet effective concentrations, the 1:1000 dilution must be diluted further.

This process is asfollows:

• To produce a 1:10,000 concentration, **1 mL of** a **1:1000** solution is added to 9 mL of solvent (e.g., **sterile water**); therefore 1:10,000 = 0.1 mg/mL (100 μg/mL).

• To produce a 1:100,000 concentration, 1 mL of a1:10,000 concentration is added to 9 mL of solvent; therefore 1:100,000 = 0.01 mg/mL (10 μg/mL).

Percent solution

- Different anesthetics come in various concentrations
- The concentrations are given as a percentage
  - 0.5% = 5 mg/cc
  - 1% = 10 mg/cc
  - 2% = 20 mg/cc
  - 3% = 30 mg/cc
  - 4% = 40mg/cc

Cartridge Components

- The prefilled 1.8ml dental cartridge consists of four parts:
  - Cylindrical glass tube
  - Stopper
  - Aluminum Cap
  - Diaphragm

- To determine the mg/cartridge, multiply the mg/cc by 1.8cc

- How many milligrams of mepivacaine are contained within cartridges of mepivacaine 3%?
  - 3% mepivacaine = 30mg/cc
  - 1 cartridge = 1.8 cc
  - 2 cartridges = 3.6 cc
  - (30 mg/cc)x(3.6 cc) = 108 mg

Concentrations of Vasoconstrictor in Local Anesthetics

1:50,000	0.02 mg/ml
1:100,000	0.01 mg/ml
1:200,000	0.005 mg/ml

Calculation

- **1:50,000 =**
- **1gram/50,000 ml =**
- **1000mg/50,000ml =**
- **1mg/50ml = 0.02mg/ml**
- **(0.02 mg/ml)x(1.8 ml/cartridge) =.036 mg/cartridge**
- -OR-
  - the way someone once explained it to me:
    - start with 1,000,000 (aka convert to µg) and divide the ml amount of the concentration, this give you the concentration in MICROGRAMS/ml
    - 1,000,000 µg/50,000 = 20µg/ml
    - If you are looking for mg, instead of µg, you can always divide by 1000 at this point (aka, move the decimal point to the left 3 places)

Calculating Maximum Dose of Epinephrine

- Normal, healthy patient: 0.2 mg per appointment
- From the slide above, we know that there is 0.018mg of epinephrine per cartridge containing epi 1/100,000
- Therefore: 0.2mg/0.018mg per cartridge = 11.1 cartridges contains the maximum dose of epinephrine

Example

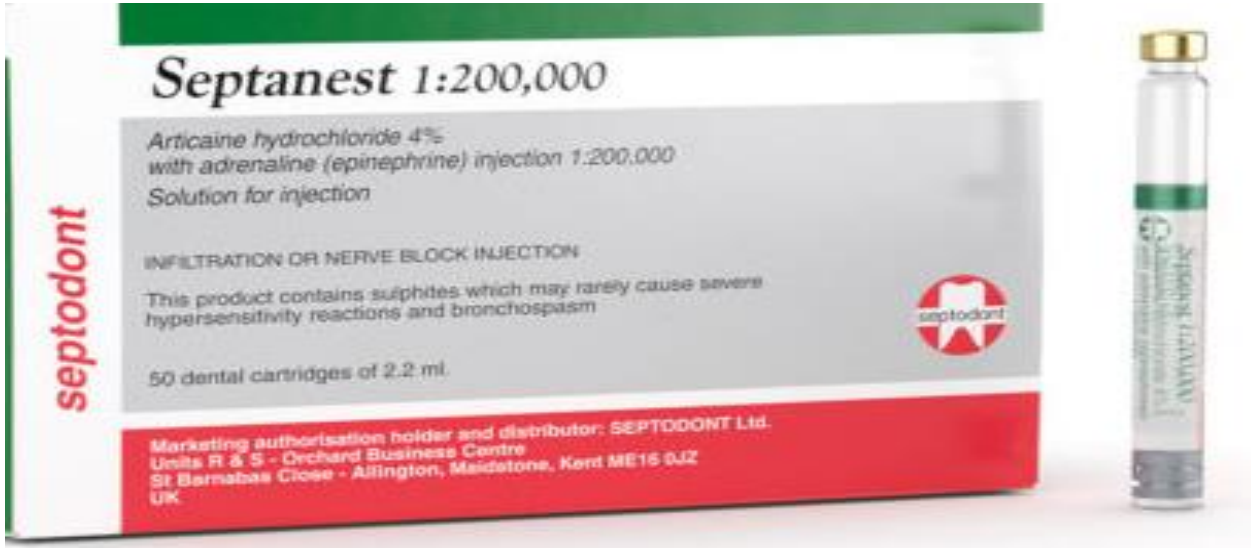
- How much lidocaine in a cartridge of lidocaine 2% with epinephrine 1/100,000? How much epi?
- 2% lidocaine = 20mg/ml
  - (20mg/ml)x(1.8 ml/cartridge)= 36mg lidocaine/cartridge
- Epi 1/100,000 = 0.01mg/ml
  - (0.01 mg/ml)x(1.8 ml/cartridge) =0.018mg epinephrine/cartridge

The milligram per milliliter and microgram per millilitervalues of the various vasoconstrictor dilutions used in medicine
and dentistry are shown in Table 3.4.The genesis of vasoconstrictor dilutions in local anesthetics
began with the discovery of epinephrine in 1897
by Abel. In 1903 Braun10 suggested using epinephrine as a
chemical tourniquet to prolong the duration of local anesthetics.
Braun recommended the use of a 1:10,000 dilution
of epinephrine, ranging to as dilute as **1:100,000**, with cocaine in nasal surgery **(a highly vascular area).**
It appears at present that an epinephrine concentration of **1:200,000** provides comparable results, with fewer systemic side effects.
The 1:200,000 dilution, which contains 5 µg/mL (or 0.005mg/mL), has become widely used in both **medicine** and **dentistry**, and is currently found for **articaine**, **prilocaine**,**lidocaine** (although not in North America), **etidocaine**, and **bupivacaine**.

Concentrations of Clinically Used Vasoconstrictors

Dilution	Milligrams per Milliliter	Micrograms per Milliliter	Amount per 1.8-mL Cartridge (µg)	Therapeutic Use
1:1000	1.0	1000		Epinephrine—emergency medicine (IM/SC in anaphylaxis)
1:2500	0.4	400		Phenylephrine
1:10,000	0.1	100		Epinephrine—emergency medicine (IV/ET in cardiac arrest)
1:20,000	0.05	50	90	Levonordefrin—local anesthetic
1:30,000	0.033	33.3	73 (2.2-mL cartridge)	Norepinephrine—local anesthetic
1:50,000	0.02	20	36	Epinephrine—local anesthetic
1:80,000	0.0125	12.5	27.5 (2.2-mL cartridge)	Epinephrine—local anesthetic (United Kingdom)
1:100,000	0.01	10	18	Epinephrine—local anesthetic
1:200,000	0.005	5	9	Epinephrine—local anesthetic
1:400,000	0.0025	2.5	4.5	Epinephrine—local anesthetic

ET, Endotracheal; *IM*, intramuscular; *IV*, intravenous; *SC*, subcutaneous.



In several European and Asian countries, lidocaine with epinephrine concentrations of 1:300,000 and 1:400,000 is available in dental cartridges.

Although it is the most used vasoconstrictor in local anesthetics in both **medicine** and **dentistry**, epinephrine is *not* an ideal drug. The benefits to be gained from adding epinephrine (or any vasoconstrictor for that matter) to a local anesthetic solution must be weighed against any risks that might be present.

Epinephrine is absorbed from the **site of injection**, just as is the local anesthetic. Measurable epinephrine blood levels are obtained, influencing **the heart** and **blood vessels**. Resting plasma epinephrine levels (39 pg/mL) are doubled after administration of one cartridge of lidocaine with epinephrine 1:100,000.

Elevation of epinephrine plasma levels is **linearly dose dependent** and persists from several minutes to 30 minutes after administration.

Contrary to a **previously held position** that **intraoral administration of usual volumes of epinephrine produces no cardiovascular response**, and that patients are more at risk from endogenously released epinephrine than they are from exogenously administered epinephrine, evidence now demonstrates that epinephrine plasma levels equivalent to those achieved **during moderate to heavy exercise** may occur **after intraoral injection**.

These are associated with moderate increases in **cardiac output** and **stroke volume** (see the following section). Blood pressure and heart rate, however,are minimally affected at usual doses.

In patients with **preexisting cardiovascular or thyroid disease**, the side effects of absorbed epinephrine must be **weighed against those of elevated local anesthetic blood levels.**

It is currently thought that the cardiovascular effects of conventional epinephrine doses are of little practical concern,even in patients with heart disease.

However, even after usual **precautions** (e.g., aspiration, slow injection) have been taken, sufficient epinephrine can be absorbed to cause **sympathomimetic reactions** such as **apprehension, tachycardia**,**sweating**, and **pounding in the chest (palpitation):** the so-called **epinephrine reaction.**

Intravascular administration of vasoconstrictors and their administration to sensitive individuals (hyperresponders), or the occurrence of unanticipated drug-drug interactions,can, however, produce significant clinical manifestations. Intravenous administration of 0.015 mg of epinephrine with lidocaine results in an increase in the heart rate ranging from 25 to 70 beats per minute, with elevations in systolic blood pressure from 20 to 70 mmHg.

Occasional rhythm disturbances may be observed, and premature ventricular contractions are most often noted.

Other vasoconstrictors used in **medicine and dentistry** include **norepinephrine, phenylephrine**, **levonordefri**n, and **felypressin**.

Norepinephrine, lacking significant **β2 actions**, produces intense peripheral vasoconstriction with possible dramatic elevation of blood pressure, and is associated with a side effect **ratio nine times higher** than that of epinephrine.

Although it is currently available in some countries in local anesthetic solutions, the use of norepinephrine as a vasopressor in dentistry is diminishing and cannot be recommended.

The use of a **mixture of epinephrine and norepinephrine** is to be **absolutely avoided**.

Phenylephrine, a pure **α-adrenergic agonist**, theoretically possesses advantages over other vasoconstrictors. However, in clinical trials, peak blood levels of lidocaine were actually higher with **phenylephrine** 1:20,000 (lidocaine blood level 2.4 μg/mL) than with epinephrine 1:200,000 (1.4 μg/mL).

The cardiovascular effects of levonordefrin most closely resemble those of norepinephrine.24 Felypressin was shown to be about as effective as epinephrine in reducing cutaneous blood flow. **Epinephrine remains the most effective and the most used vasoconstrictor in medicine and dentistry.**

Epinephrine(Adrenalin).

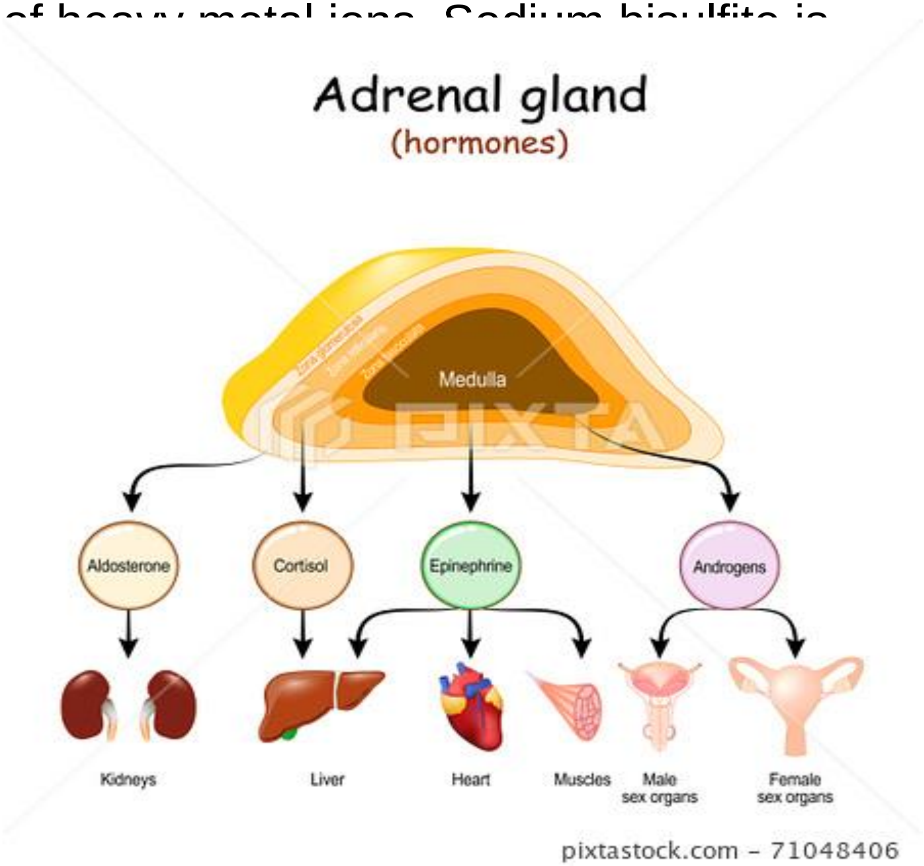
Chemical Structure

Epinephrine as the acid salt is highly soluble in water. Slightly acid solutions are relatively stable if they are protected from air. Deterioration (through oxidation) is hastened by heat and the presence of heavy metal ions. Sodium bisulfite is commonly added to epinephrine solutions to delay this deterioration.

Source

Epinephrine is available as a **synthetic** and is also **obtained** from the **adrenal medulla** of animals (epinephrine constitutes approximately **80% of adrenal medullary secretions**).

It exists in both **levorotatory** and **dextrorotatory** forms; the levorotatory form is approximately 15 times as potent as the dextrorotatory form



Mode of Action:

Epinephrine is a nonselective adrenergic agonist, stimulating **α1-, α2-, β1-, and β2-adrenergic receptors**. The degree of stimulation varies with the concentration of epinephrine at the specific receptor.

Systemic Actions

Myocardium :

Epinephrine stimulates **β1** receptors of the myocardium. There is a positive inotropic (**force of contraction**) and a positive chronotropic (**rate of contraction**) effect. Both **cardiac output** and **heart rate are increased**.
Pacemaker Cells .**Epinephrine stimulates β1 receptors** and **increases the irritability of pacemaker cells**, leading to an increased incidence of **dysrhythmias**. **Ventricular tachycardia** and **premature ventricular contractions** are common.

Coronary Arteries :

Epinephrine produces dilation of the coronary arteries,increasing coronary artery blood flow.

Blood Pressure:

Systolic blood pressure is increased.
Diastolic pressure is decreased with small doses of epinephrine because of the greater sensitivity to **epinephrine of β2 receptors compared** with **α receptors** in blood vessels supplying skeletal muscle.
Diastolic blood pressure is increased with larger doses of epinephrine because of constriction of blood vessels supplyingthe skeletal muscles caused by **α-receptor stimulation.**

Cardiovascular Dynamics

The overall action of epinephrine on the heart and cardiovascular system is one of direct stimulation:

- increased systolic and diastolic pressures
- increased cardiac output
- increased stroke volume
- increased heart rate
- increased strength of contraction
- increased myocardial oxygen consumption

These actions lead to an overall *decrease* in cardiac efficiency.

2% lidocaine with epinephrine 1:100,000 in hypertensive patients. A decrease in systolic blood pressure in patients with the diastolic blood pressure decreased after the injections.

2% mepivacaine with epinephrine 1:100,000 . There were no statistically significant differences in heart rate and blood pressure monitored before and after injection.

Articaine, either 1:100,000 or 1:200,000, via intraosseous injection in patients with irreversible pulpitis
Reported no significant changes in blood pressure, heart rate, or ECG.

Another study demonstrated that there were no significant changes in heart rate, systolic blood pressure, diastolic blood pressure, oxygen saturation (Spo2), and ECG in the patients studied.

The reduced blood flow decreases the **pH of the tissue**, which shifts the equilibrium reaction towards the ionised form of the **anaesthetic.** This reduces the penetration of the local anaesthetic in the **nerve** and **diminishes the anaesthetic effect**. In addition, **decreased blood flow may have a negative effect on wound healing**.

A third **local disadvantage** is that a ‘rebound’ effect may occur as soon as the vasoconstrictor has worn off, due to the accumulation of degradation products; because of increased blood flow, there is an increased risk of secondary hemorrhage.

After intravasal injection of an adrenaline-containing local anaesthetic, blood pressure and heart frequency will increase (dangerously).

Other adrenergic agonist vasoconstrictor

- Nor-adrenaline and phenylphrine have prominent alpha activity comparing to beta activity which may result in **severe vasoconstriction** (increase blood pressure) and ischemia.
- It is contraindicated in patients with cardiac problem.

It is contraindicated in terminal extremities

Metabolism of catecholamines

Catecholamines are metabolized by multiple pathways involving oxidative deamination catalyzed by **monoamine oxidase (MAO)**, O-methylation by **catechol O-methyltransferase** (COMT) and **conjugation** by **sulfotransferases** or **glucuronidases**

Side effects and overdose of adrenaline

CNS: Fear apprehension palpitation

CVS: Cardiac stimulant effects , increase blood pressure and rebound bleeding at prolonged dental procedure.

<u>Causes of rebound bleeding</u>:Adrenaline selectivity on receptor: Low concentration β effect. High concentration α effect

Felypressin:

Felypressin is a non-catecholamine vasoconstrictor that is chemically related to **vasopressin**, the posterior pituitary hormone

It is an **analogue** of the naturally occurring **Vasopressin(**Vasopressin is a hormone of the posterior pituitary that is secreted in response to **high serum osmolarity**.

Excitation of atrial stretch receptors inhibits vasopressin secretion.

Vasopressin is also released in **response to stress**, **inflammatory signals**, and **some medications**.

Hypotension, **morphine,** **nicotine**, **angiotensin II**, **glucocorticoids**, and **IL-6** **all** stimulate release of vasopressin)**.**

•Bind to **vasopressin V_1 receptor** in the vascular smooth muscle producing **vaso-constriction** and **reduce local blood flow.**

•**Less potent** than the **catecholamines** & **poorer control of bleeding** during operative procedures.

•Acts on the **venous side** rather than the **arterial side.**

Vasopressin (Felypressin)

Available concentration = 0.03 IU/ml in combination with prilocaine 2% or 3%

MRD= 0.27 IU Uses of small amount available in dental cartridge is better than exposing the patient to failure anesthesia which produce pain and bleeding that can stimulate fear and increase intrinsic adrenaline that may have more dangerous effect than extrinsic adrenaline

- **Dose: 0.03 IU/ml (0.54 μg/ml)** Synthetic analogue of **posterior pituitary hormone** (Octopressin)(Vasopressin is a hormone of the posterior pituitary that is secreted in response to high serum osmolarity. Excitation of atrial stretch receptors inhibits vasopressin secretion. Vasopressin is also released in response to stress, inflammatory signals, and some medications. Hypotension, morphine, nicotine, angiotensin II, glucocorticoids, and IL-6 all stimulate release of vasopressin)
- Act on **V1 receptor** that is found on **venous site of microcirculation**
- It **posses mild hemostatic effect** and used only when other **vasoconstrictor contraindicated**
- **Less potent** than the **catecholamines** & **poorer control of bleeding** during operative procedures.
- Acts on the **venous side** rather than the **arterial side.**

Consideration

- Maximum Recommended Dosage (MRD) for cardiovascular disease patient = 0.04 mg of adrenaline = 2 dental cartridge of 2ml 1:100000 concentration adrenaline

- Controversy still exists on using adrenaline in controlled cardiovascular diseased patient.

- **Uses of small amount available in dental cartridge** is better than exposing the patient to failure anesthesia which produce pain and bleeding that can **stimulate fear** and **increase intrinsic adrenaline** that may have **more dangerous** effect than **extrinsic adrenaline**

Preservative agent of LA

- Maintains sterility of the solution
- Caprylhydrocuprienotoxin used for this purpose

Methylparaben used in the past but nowadays omitted ?

Reducing agent (in vasoconstrictor containing solution)

- Antioxidant (reducing agent) used to prevent **oxidation of vasoconstrictor** that may deteriorate on exposure to sunlight (brown discoloration)
- **Sodium metabisulfite** used for this purpose
- On exposure to oxygen it will diffuse through the rubber of the cartridge where sodium metabisulfite will be converted to sodium metabisulfate (oxidized)
- Oxidized instead of vasoconstrictor
- Why is an old solution more acidic? Painful ? Irritant?

Selection of a Local Anesthetic agent

The decision to inject local anesthetic agents to achieve profound anesthesia is **dependent upon many factors**, **particularly the depth** and **duration of anesthesia required**, and **the possible need for hemostasis.**

To maximize the safety of local anesthetic injections, it is necessary to weigh the **risks against** the **benefits for each patient**, for each anesthetic agent, for **use of a vasoconstrictor**, and **for the delivery technique for the selected agent**

local anesthetics available are currently used in dentistry.

lidocaine, **mepivacaine**, **prilocain**e, and the combination of **procaine** and **propoxycaine**. **bupivacaine** and **etidocaine** .**The hybrid(ester/amide) molecule articaine** .**Articaine** is classified as an intermediate-duration local anesthetic..

With the availability of these local anesthetics, in various combinations with and without vasoconstrictors, it is possible for a doctor to select a local anesthetic solution that possesses the specific pain-controlling properties necessary to provide pain-free treatment for virtually all dental patients.

It is strongly suggested that the reader—the potential administrator of these drugs—become familiar with this material, including contraindications to the administration of certain local anesthetic combinations

Local Anesthetics Available in North America (February 2019)

Local Anesthetic (+ Vasoconstrictor)	Duration of Action[a]
Articaine hydrochloride	
4% + epinephrine 1:100,000	Intermediate
4% + epinephrine 1:200,000	Intermediate
Bupivacaine hydrochloride	
0.5% + epinephrine 1:200,000	Long
Lidocaine hydrochloride	
2% + epinephrine 1:50,000	Intermediate
2% + epinephrine 1:100,000	Intermediate
Mepivacaine hydrochloride	
3%	Short
2% + levonordefrin 1:20,000	Intermediate
2% + epinephrine 1:100,000	Intermediate
Prilocaine hydrochloride	
4%	Short (infiltration); intermediate (nerve block)
4% + epinephrine 1:200,000	Intermediate

[a]The classification of duration of action is approximate, for extreme variations may be noted among patients. Short-duration drugs provide pulpal or deep anesthesia for <30 min, intermediate-duration drugs provide it for about 60 min, and long-duration drugs provide it for longer than 90 min.

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Academic Year 3

[Instruments of local anesthesia]

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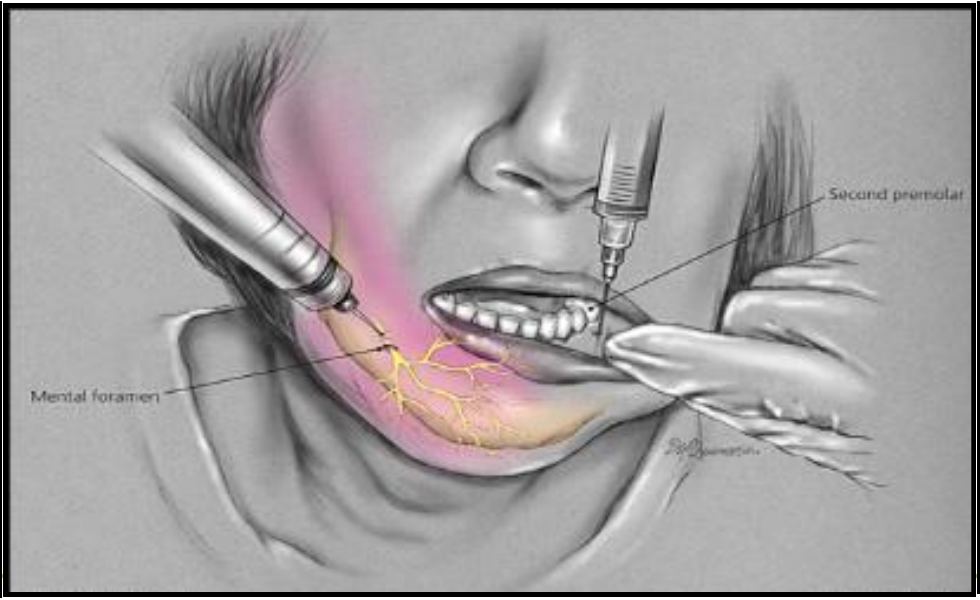


Instruments of local anesthesia

- The Syringe
- The Needle
- The Cartridge
- Additional Armamentarium
- Preparation of the Armamentarium

The necessary equipment's for LA:

Syringe
Needle
LA cartridge

The syringe is one of three essential components of the local anesthetic armamentarium (others include the needle and the cartridge). It is the vehicle whereby the content of the anesthetic cartridge is delivered through the needle to the patient.

Types of Syringes:

Eight types of syringes for local anesthetic administration are available for use in dentistry today. They represent a considerable improvement over the local anesthetic syringes formerly used.

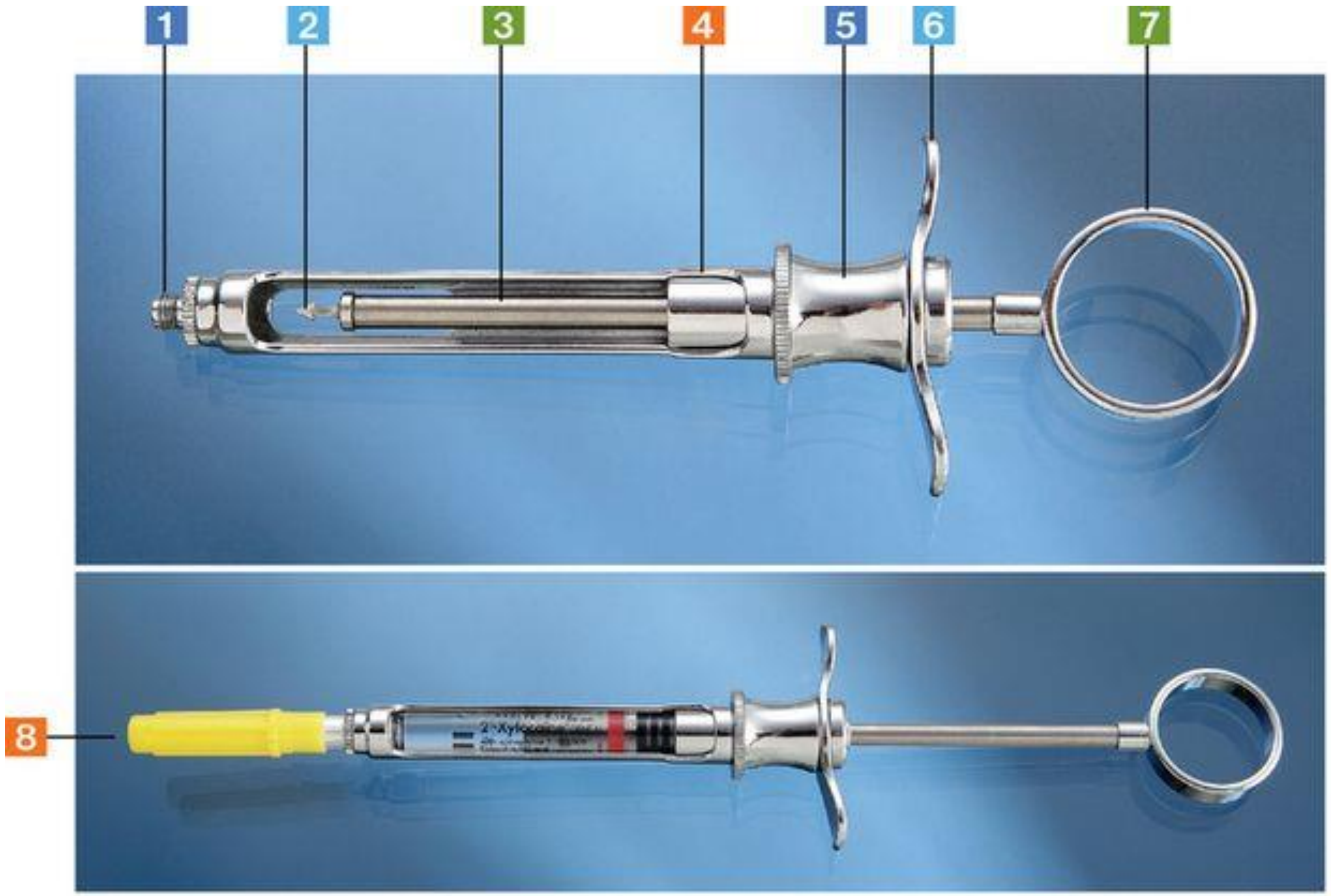
Non aspirating syringes are not discussed except to state that their use unacceptably **increases the risk of inadvertent intravascular drug administration**.

The use of aspirating dental syringes (capable of the aspiration of blood) represents the standard of care.

Syringe Types Available in Dentistry

1. Nondisposable syringes:
   a. Breech-loading, metallic, cartridge-type, aspirating
   b. Breech-loading, plastic, cartridge-type, aspirating
   c. Breech-loading, metallic, cartridge-type, self-aspirating
   d. Pressure syringe for periodontal ligament injection
   e. Jet injector ("needle-less" syringe)
2. Disposable syringes
3. "Safety" syringes
4. Computer-controlled local anesthetic delivery systems

1. Threaded tip
2. Harpoon
3. Piston rod
4. Barrel of syringe
5. Finger grip
6. Finger bar
7. Thumb ring
8. Syringe assembled with needle and anesthetic cartridge



Anesthetic aspirating syringe To administer a local anesthetic

The local anesthetic syringes include:

They must be durable and able to **withstand repeated sterilization** without damage. (If the unit is disposable, it
1-should be packaged in a sterile container.)
2-They should be capable of accepting a wide variety of cartridges and needles of different manufacture, and should permit repeated use.
3-They should be inexpensive, self-contained, light weight, and simple to use with one hand.
4. They should provide effective aspiration and be constructed so that blood may be easily observed in the cartridge.

Eight types of syringes for local anesthetic administration are available for use in dentistry today.

Two of the eight types are used in dental clinic. These two syringes are non-disposable, breech-loading (cartridge is inserted or loaded), metallic, and cartridge types.

1. Aspirating

Most common, Harpoon attached to the piston (penetrates rubber stopper at the end of the local anesthetic cartridge). Allows for aspiration testing

2. Self-Aspirating

Uses the elasticity of the rubber diaphragm in the anesthetic cartridge to obtain the required negative pressure for aspiration testing
Permits for multiple aspirations easily

With commonly used breech-loading, metallic or plastic, cartridge-type syringes, **an aspiration test must be conducted purposefully by the administrator before or during drug deposition**.

The key word here is ***purposefully***. However, as demonstrated in many dentists do not purposefully perform an aspiration test before injection of the anaesthetic drug.

To increase the ease of aspiration, self-aspirating syringes have been. These syringes use the elasticity of the rubber diaphragm in the anesthetic cartridge to obtain the necessary negative pressure for aspiration.
The diaphragm rests on a metal projection inside the syringe that directs the needle into the cartridge
Pressure acting indirectly on the cartridge through the plunger shaft distorts (stretches) the rubber diaphragm, producing positive pressure within the anesthetic cartridge.
When that pressure is released—by simply releasing thumb pressure on the plunger—sufficient negative pressure develops within the cartridge to permit aspiration. The use of a self-aspirating dental syringe permits easy performance of multiple aspirations throughout the period of local anesthetic deposition.

Advantages and Disadvantages of the Metallic, Breech-Loading, Aspirating Syringe

Advantages	Disadvantages
Visible cartridge	Weight (heavier than plastic syringe)
Aspiration with one hand	Syringe may be too big for small operators
Autoclavable	Possibility of infection with improper care
Rust resistant	
Long lasting with proper maintenance	

Advantages and Disadvantages of the Plastic, Reusable, Aspirating Syringe

Advantages	Disadvantages
Plastic eliminates metallic, clinical look	Size (may be too big for small operators)
Lightweight: provides better "feel" during injection	Possibility of infection with improper care
Cartridge is visible	Deterioration of plastic with repeated autoclaving
Aspiration with one hand	
Rust resistant	
Long lasting with proper maintenance	
Lower cost	

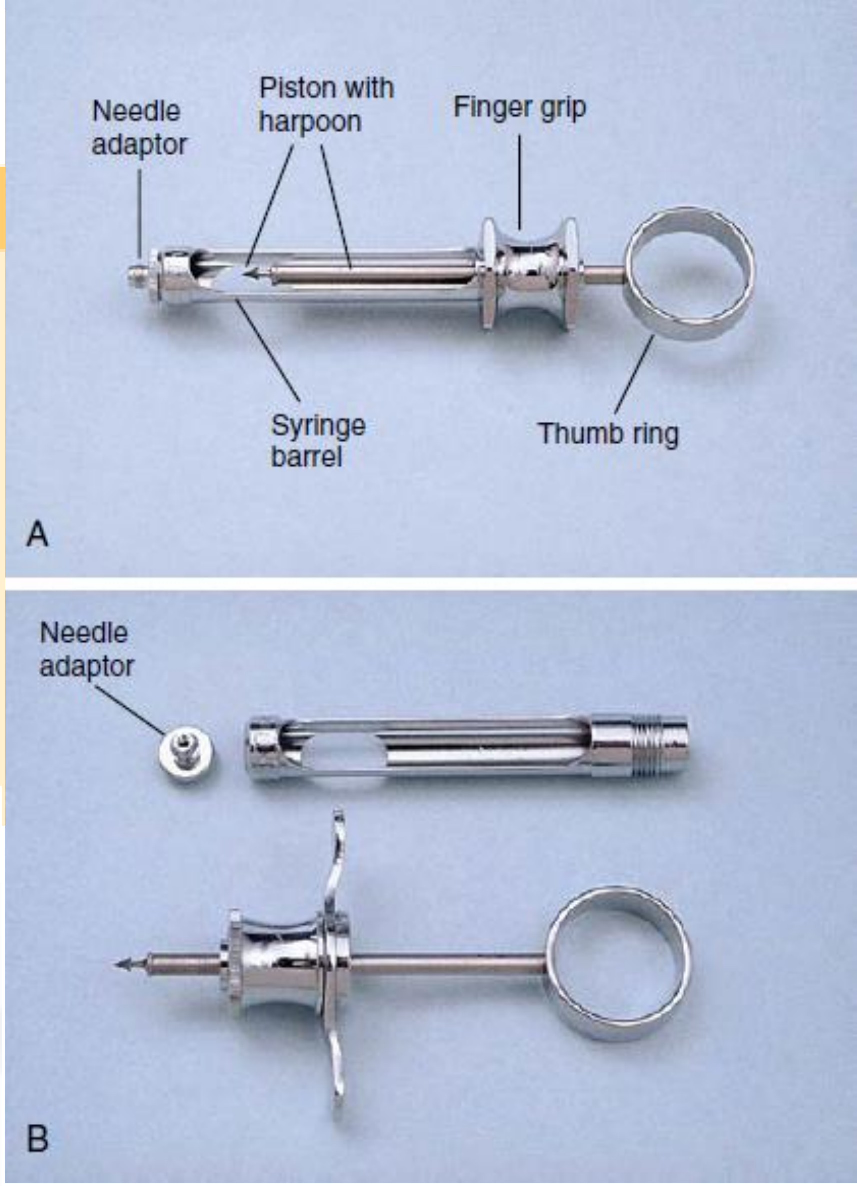
Some dentists believed that the self-aspirating syringe **did not provide the same reliability of aspiration** that was possible with the harpoon aspirating syringe.
It has been demonstrated, however, that this syringe does in fact aspirate blood as reliably as the harpoon-aspirating syringe.

In comparing two self-aspirating syringes with a non self-aspirating system during inferior alveolar nerve blocks, found the **non-self-aspirating system** to be more reliable positive aspiration than the two **self-aspirating syringes**

Advantages and Disadvantages of the Metallic, Self-Aspirating Syringe

Advantages	Disadvantages
Cartridge visible	Weight
Aspiration is easier with small hands	Feeling of "insecurity" for doctors accustomed to harpoon-type syringe
Autoclavable	Finger must be moved from thumb ring to thumb disk to aspirate blood
Rust resistant	Possibility of infection with improper care
Long lasting with proper maintenance	
Piston is scored (indicates volume of local anesthetic administered)	

Self-aspirating syringe. (Courtesy of Septodont



A) Breech-loading, metallic, cartridge-type syringe; assembled.
(B) Disassembled local anesthetic syringe

The major factor influencing ability to aspirate blood is not the syringe but the gauge of the needle being used.
In addition, most doctors using the harpoon-aspirating syringe tend to over aspirate, that is, they retract the thumb ring
back too far and with excessive force (and, on occasion, disengage the harpoon from the stopper). These doctors feel
more insecure with the self-aspirating syringe. Proper technique of aspiration is discussed in .Advantages and disadvantages of the metallic,

Advantages and Disadvantages of the Metallic, Self-Aspirating Syringe

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Autoclavable	Finger must be moved from thumb ring to thumb disk to aspirate blood
Rust resistant	Possibility of infection with improper care
Long lasting with proper maintenance	
Piston is scored (indicates volume of local anesthetic administered)	

3-Pressure Syringes

(Re)introduced in the late 1970s, pressure syringes brought about a renewed interest in the **periodontal ligament (PDL) injection** (also known as the *intraligamentary injection*).
Discussed
The PDL injection, although usable for any tooth, helped make it possible to achieve more reliable **pulpal anesthesia of one isolated mandibular tooth** where, in the past, nerve block anesthesia (e.g., inferior alveolar nerve block, Gow-Gates mandibular nerve block), with its attendant prolonged soft tissue (e.g., lingual, lip, chin) anesthesia, was necessary.

Although "special" syringes such as these are not necessary for a successful PDL injection, several advantages are associated with their use, not the least of which is the mechanical advantage they provide the administrator, making the local anesthetic easier to administer.

This same mechanical advantage, however, makes the injection somewhat "too easy" to administer, leading to "too rapid" injection of the anesthetic solution and patient discomfort both during the injection and later, when the anesthetic has worn off.

However, when used slowly, as recommended by manufacturers, pressure syringes are of some benefit in administration of this valuable technique of anesthesia.

Pressure syringes offer advantages over the conventional syringe when used for PDL injections because their trigger delivers ameasured dose of local anesthetic and enables a relatively physically weak administrator to overcome the significant tissue resistance encountered when the PDL injection is administered properly. This mechanical advantage may also prove to be detrimental if the administrator deposits the anesthetic solution too quickly (<20 seconds per 0.2-mL dose).

Advantages and Disadvantages of the Pressure Syringe

Advantages	Disadvantages
Measured dose	Easy to inject anesthetic too rapidly
Overcomes tissue resistance	Threatening (original devices)
Nonthreatening (new devices)	
Cartridges protected	

All pressure syringes completely encase the glass dental cartridge with plastic or metal, there by protecting the patient in the unlikely event that the glass cartridge cracks or shatters during injection. The original pressure syringes looked somewhat threatening, having the appearance of a gun .Newer devices are smaller and less intimidating.

lists the advantages and disadvantages of the pressure syringe.

4-Jet Injector:

The first report of the use of jet injections in dentistry was published in 1958.Jet injection is based on the principle that **liquids forced** through very **small openings**, called ***jets***, at very high pressure can penetrate intact skin or mucous membrane (visualize water flowing through a garden hose whose opening is being crimped).

The most frequently used jet injectors in dentistry are the **MadaJet** and the **Comfort-in needle-free injection system**.

Neither MadaJet nor Comfort-in supports the traditional dental local anesthetic cartridge. The primary purpose of the **jet injector** is to obtain **topical anesthesia before insertion of a needle**. In addition, it may be used to obtain **mucosal anesthesia** of the **palate**.

Regional nerve blocks or **supraperiosteal injections are still necessary** for complete anesthesia. The jet injector is not an adequate substitute for the more traditional needle and syringe in obtaining pulpal or regional block anesthesia.

Additionally, many patients dislike the feeling that accompanies the use of the jet injector, as well as the possible postinjection soreness of soft tissue that may develop even with proper use of thedevice.

5-Disposable Syringes:

Plastic disposable syringes are available in a variety of sizes with an assortment of needle gauges. Most often they are used for **intramuscular** or **intravenous** drug administration, but they may also be used for **intraoral injection .**These syringes contain a Luer-Lok screw-on needle attachment with no aspirating tip. **Aspiration can be accomplished by pulling back on the plunger of the syringe before or during injection**.

Because there is no thumb ring, aspiration with the plastic disposable syringe requires the use of both hands. In addition, these syringes do not accept dental cartridges. The needle, attached to the syringe, must be inserted into a **vial or cartridge of local anesthetic drug and an appropriate volume of solution must be withdrawn**.

Care must be taken to avoid contaminating the multiuse vial during this procedure. Two-milliliterand 3-mL syringes with **25- or 27-gauge** needles are recommended when the system is used for intraoral local anaesthetic administration. **The plastic, disposable, non–cartridge-containing syringe is not recommended for routine use.**

Advantages and Disadvantages of the Jet Injector

Advantages	Disadvantages
Does not require use of a needle (recommended for persons with needle phobia)	Inadequate for pulpal anesthesia or for regional block
Delivers very small volumes of local anesthetic (0.01–0.2 mL)	Some patients are disturbed by the jolt of the injection.
Used in place of topical anesthetic	Cost
	May damage periodontal tissues

Advantages and Disadvantages of the Disposable Syringe

Advantages	Disadvantages
Disposable, single use	Does not accept prefilled dental cartridges
Sterile until opened	Aspiration difficult (requires two hands)
Lightweight (may feel awkward to the first-time user; tactile sensation better)	

6-Safety Syringes:

There has been a movement toward the introduction of ***safety syringes*** in both medicine and dentistry. **Safety syringes minimize the risk of an accidental needle stick injury occurring to a dental health provider with a contaminated needle after administration of a local anesthetic**.

These syringes possess a sheath that "locks" over the needle when it is removed from the patient's tissues, minimizing the risk of accidental Needle stick.

The Ultra Safety Plus XL aspirating syringe system contains a syringe body assembly and a plunger assembly (see Once the syringe is properly assembled and the injection administered, the syringe may be made "safe" with one hand by gentle movement of the index and middle fingers against the front collar of the guard Once "guarded," the now contaminated needle is "safe," so it is virtually impossible for dental health providers to be injured with the needle.

On completion of the injection, the entire syringe is discarded into the proper receptacle (e.g., sharps container). **Dental safety syringes are designed as single-use item**s, although they permit reinjection.

Reloading the syringe with a second **anesthetic cartridge** and reinjection with the same syringe is **discouraged because this obviates the important safety aspect of the device**. Advantages and disadvantages of the safety syringe are listed.

Advantages and Disadvantages of the Safety Syringe

Advantages	Disadvantages
Disposable, single use	Requires additional training
Sterile until opened	May feel awkward to a first-time user
Lightweight (better tactile sensation)	

7-Computer-Controlled Local Anesthetic Delivery Systems

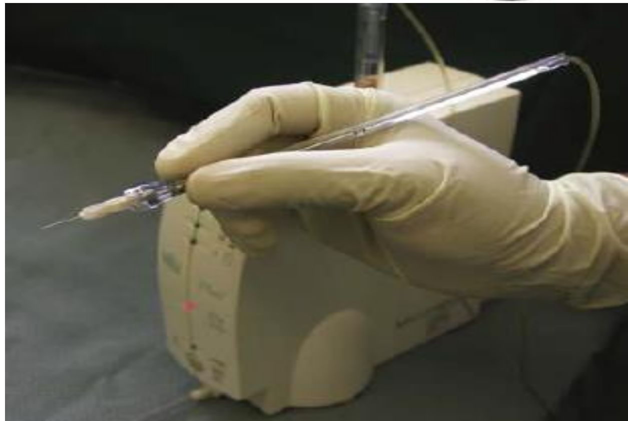
The standard dental syringe described previously is a simple mechanical instrument . The dental syringe is a drug-delivery device requiring that the operator simultaneously attempt to control the variables of drug infusion and the movement of a **penetrating needle**. The operator's inability to precisely control both of these activities during an injection can compromise injection technique. In addition, a traditional syringe is held with a palm-thumb grasp, which is not designed for ideal ergonomics or needle control during the injection.

For certain practitioners—those with small hands—just holding a syringe with a full cartridge of anesthetic may be difficult.

In 1997 the first computer-controlled local anesthetic delivery (C-CLAD) system was introduced into dentistry.

This system enabled a **dentist or hygienist to precisely manipulate needle placement** with fingertip accuracy and deliver the local anesthetic with a lightweight handpiece held in a pen-like grasp, provides increased tactile sensation and control compared with the traditional syringe.

The flow rate of local anesthetic delivery is computer controlled and thus remains consistent from one injection to the next.



C-CLAD systems represent a significant change in the manner in which a local anesthetic injection is administered.
The operator is now able to focus attention on needle positioning and insertion, allowing the motor in the device to administer the drug at a preprogrammed rate of flow.

It is likely that greater ergonomic control coupled with fixed flow rates is responsible for the improved injection experience demonstrated in many clinical studies conducted with C-CLAD devices in dentistry.
The investigators concluded that the new system provides comfortable anesthesia for patients and can be a good alternative for conventional manual syringe injection. The Wand STA system also has a training mode feature that provides clinicians with spoken instructional guidance on its use, thereby minimizing the learning curve when the system is used for the first time. Advantages and disadvantages of The Wand STA system are listed.

Advantages and Disadvantages of The Wand STA Single Tooth Anesthesia System

Advantages	Disadvantages
Dynamic pressure-sensing technology provides continuous real-time feedback when an injection is performed, resulting in a more predictable injection site location	Requires additional armamentarium
Allows the periodontal ligament injection to be used as a predictable primary injection	Requires additional training
Can be used for all traditional injection techniques	
Recommended device for newer injection techniques such as anterior middle superior alveolar nerve block, palatal anterior superior alveolar nerve block, and STA periodontal ligament injection	
Reduces pain-disruptive behavior in children and adults	
Reduces stress for patient	
Reduces stress for operator	

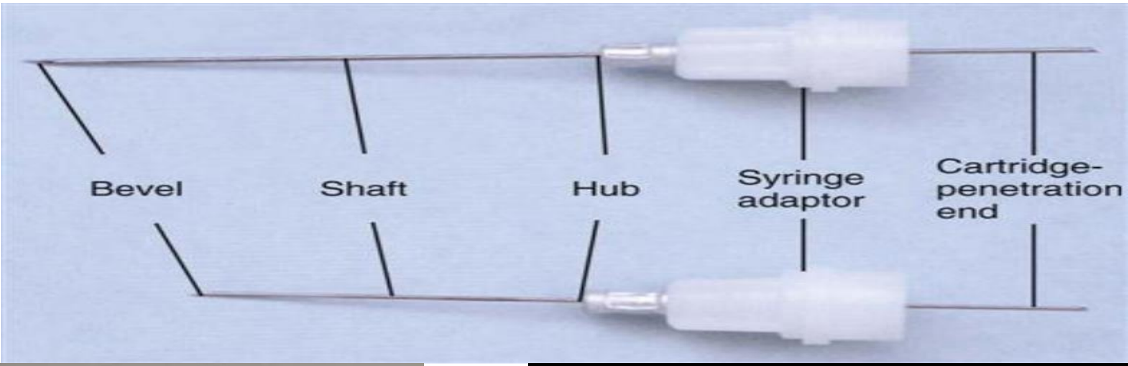
Dynamic pressure sensing on the STA Single Tooth Anesthesia System computer-controlled local anesthetic delivery device provides both visual and audible feedback regarding placement of the needle tip during the periodontal ligament (PDL) injection. Horizontal color bars (arrow) indicate pressure at the tip of the needle. Red means pressure is too low. Orange and dark yellow mean increasing pressure but not yet adequate. Light yellow means correct pressure for PDL injection. At this point the STA unit will also provide an audible clue “PDL, PDL, PDL” that the needle tip is properly situated.

diaphragm, allowing leakage of the anesthetic solution around the outside of the metal needle and into the patient's mouth (local anesthetics are unpleasant tasting)

Broken Cartridge

A badly worn syringe may damage the cartridge, leading to breakage. This can also result from a **bent harpoon**. A needle that is bent at its proximal end may not perforate the diaphragm on the cartridge. Positive pressure on the thumb ring increases pressure within the cartridge, which may cause the cartridge to break.

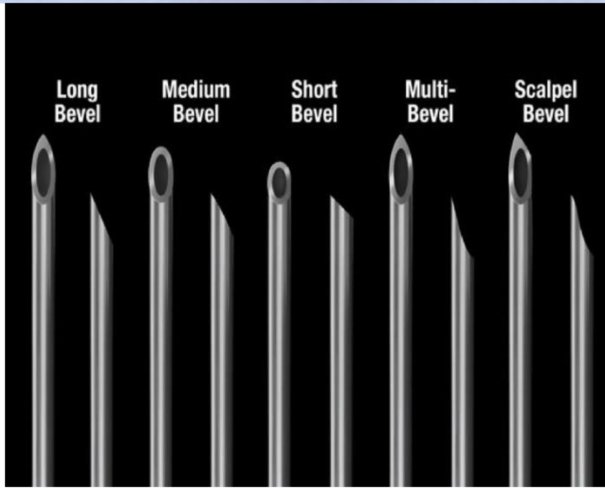
Needles



The main components of the dental anesthetic needle include the bevel, shank, hub, syringe adaptor, and cartridge penetration end. The syringe adaptor/hub complex is commonly referred to as the "hub

Needle selection factors: Needle selection should be based on three things: **A-injection type, B-depth of penetration**.

C-The needl's gauge

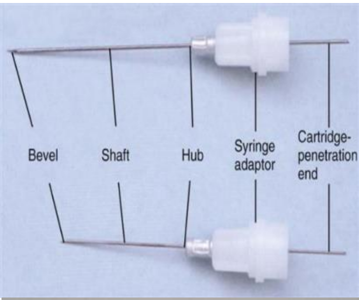


Needle length :

Dental needles are available in **three lengths**: **long**, **short**, and **ultra-short**. The two **most common lengths of needles used for intraoral injections with the traditional syringe are the "long" and the "short" .**

The length of the needle can vary some by manufacturer, but usually, from hub to tip, a long needle is about 32 mm (1.5 inches) and the short is about 20 mm (1.0 inch)

The long needle is required for mandibular blocks, because the depth of penetration ranges from **20-25** mm for an average adult **(20-25 mm for the inferior alveolar and 25 mm for Gow-Gates mandibular block techniques).**
It is extremely important to avoid insertion to the hub, the **weakest part of the needle**.
At the hub, the needle can break **more easily**. If the needle does break, and if there is not at **least 5 mm** of it exposed, it would likely be lost in the tissues and very difficult to retrieve.
The long needle is also recommended for **the anterior superior alveolar (ASA)/infraorbital approach injection**. A short needle is usually preferred for the posterior superior alveolar injection (PSA) to avoid overinsertion, for supraperiosteal injections, and also tissue infiltrations



Needle gauge :

The gauge of the needle represents the diameter of the lumen, the hollow tunnel within the needle. In dentistry, common gauges include:
25, 27, and 30 (the 30-gauge needle is no longer recommended)-the larger the number, the smaller and thinner the gauge.
As mentioned above, **patients are unable to distinguish between 25-, 27-, and 30-gauge needles**.
With the larger gauge (25 or 27) needles, because they are more rigid, there is resistance to needle breakage and less deflection as it advances through deeper tissues, which then results in greater accuracy for reaching the desired target. **More importantly, aspiration is more reliable since the larger lumen facilitates ease of aspiration.**
The 25-gauge needle has been recommended for all injections, but it is strongly recommended for all injections with a high risk of positive aspiration and/or with a significant depth of penetration. The 27-gauge needle is restricted for other injections where depth of penetration and risk of positive aspiration are minimal .
The 30-gauge needle is not recommended for supraperiosteal or block injections, but can be useful for localized infiltration (injecting the papillae, for example, to obtain hemostasis

Needle bevel

The bevel is the **slanted surface of a needle**, which creates the tip and facilitates nontraumatic entry into tissues.

Manufacturers often describe **bevels** as **short, medium**, or **long**.

Experts have discussed that the angle of the **bevel** as it relates to the long axis of the needle may affect the **degree of needle** deflection.

Septodont has developed a needle with an innovative scalpel designed bevel .

The purpose of the design is to allow for smoother penetrations, less tissue displacement, less deflection, and less force required of the clinician. They also have developed a needle with a larger inner bore design, with the intent to reduce pain during injections

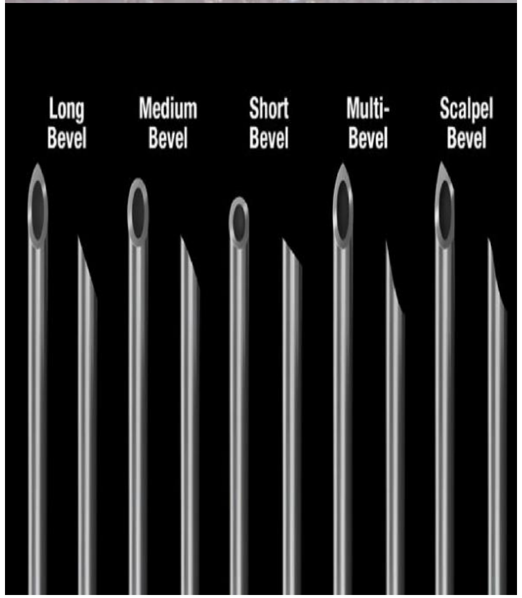
Although not critical to the success of injections, bevel design remains a topic of discussion and study.

The orientation of the bevel should be toward the bone during injections that are close to the periosteum.

Doing so **increases patient comfort and reduces trauma to the periosteum if bone is contacted**.

Some clinicians consider adjusting the bevel to facilitate placement of anesthetic closer to nerves.

Others assert that during deeper penetrations, **deflection can cause deposition away from a target if bevel orientation is not adhered to**



Metal versus plastic hubs

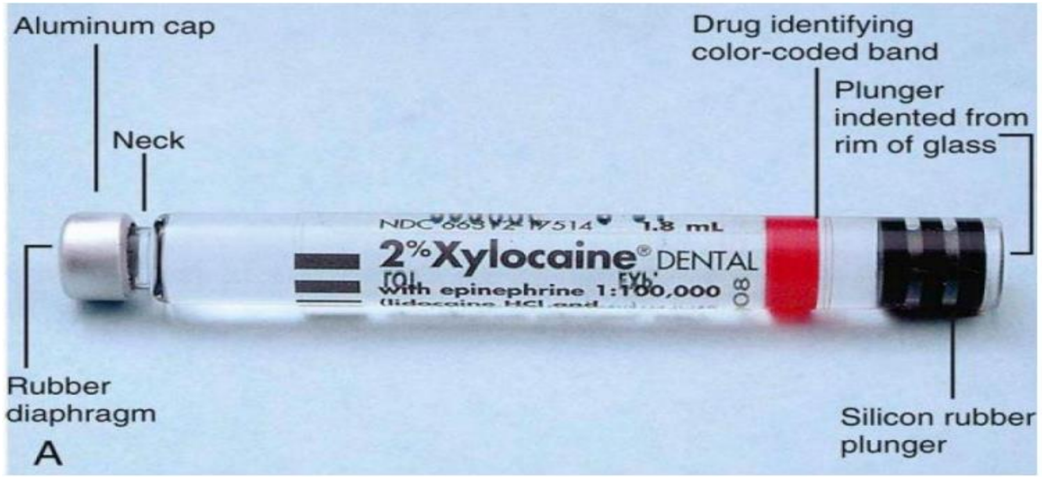
The metal or **plastic needle syringe adaptor/hub complex, the "hub,"** attaches **the needle to the syringe**.

Metal **hubs are pre-threaded** and **must screw** down tight to avoid loosening and often the bevel or syringe window will be out of position.

Once screwed down tight, they can then be more difficult to **remove**, removing the needle adaptor of the syringe (if present) with them.

The plastic hubs are usually **self-threading** and **usually fit all syringes well**, including syringes which have needle **adapters** that are **stripped or have defects**.

They are easy to rotate for bevel alignment and they are resistant to removing the needle adaptor. **Many manufacturers of plastic needle hubs include a dot or arrow indicating the location of the bevel**



Dental Cartridge (Carpule):

The dental cartridge is a **glass cylinder** containing the local anesthetic solution. It consists of:

1.Cylindrical glass tube.

2.Stopper located at the end of the cartridge that receives the hook of the aspirating syringe.

3.The aluminum cup is located at the opposite end of the cartridge from the stopper to hold the diaphragm in its position.

4.Diaphragm. Care and handling of the dental cartridge

Glass dental cartridges should not be autoclaved.

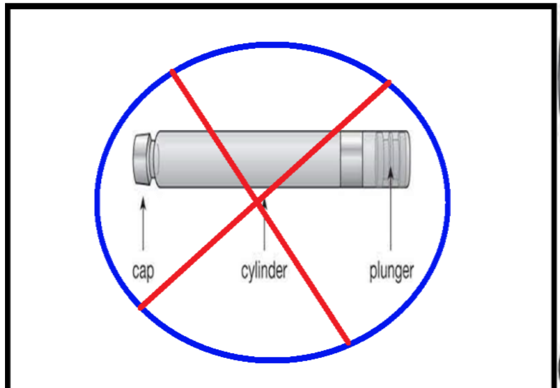
The **seal on the cartridge** cannot withstand extremes of temperature of autoclaving.

The dental cartridge should be stored **as aseptically as possible**; they should be stored dry in their original container and covered with a lid all the time, at room temperature and in dark place.

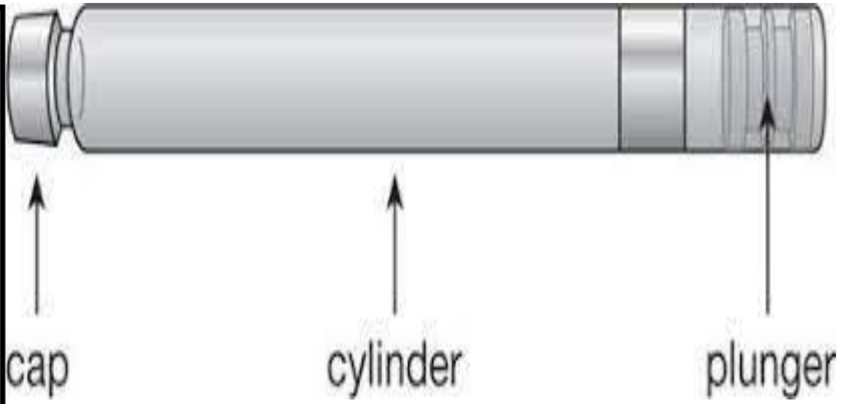
- The dental cartridges should not be left exposed to direct sunlight because some contents may undergo accelerated deterioration.

- Cartridges should not be permitted to **soak either in alcohol** or in **other cold sterilizing solutions** because the **permeable rubber plunger will allow diffusion of this solution into the dental cartridge.**

This leads to contamination of the **local anesthetic solution** resulting **in post-injection pain, edema and trismus.**



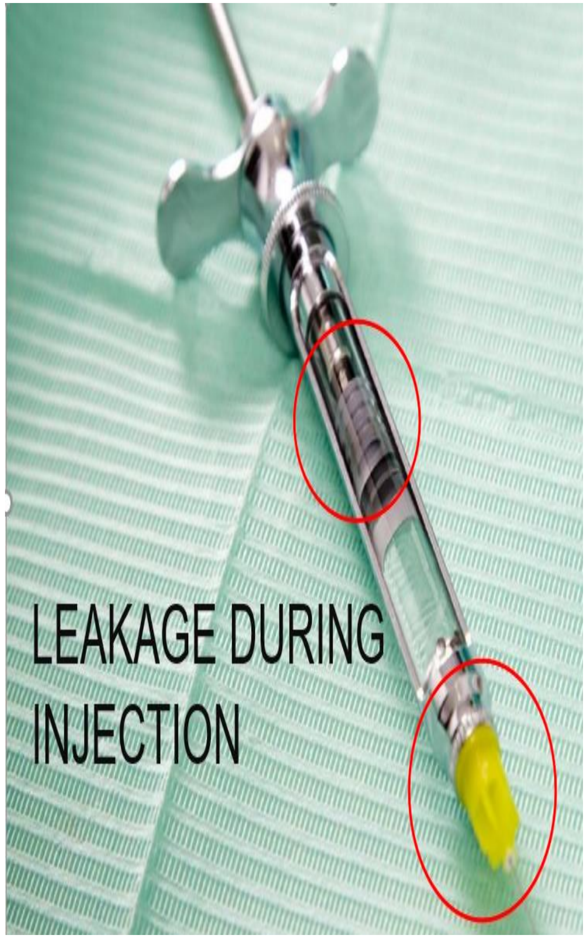
No soak either in alcohol or cold sterilizing solutions



Clinical problems associated with the equipment used in local anesthesia

A- Clinical problems related to the dental syringes

1- Leakage of the solution during injection:



The leakage of the anesthetic solution into the **patient's mouth during injection** will occur **if the cartridge** and **the needle are improperly mounted into the syringe**. When the needle is properly placed on the syringe after the cartridge is inserted; the needle produces a **centric perforation of the diaphragm that tightly seals itself around the needle**.

When pressure is applied **to the plunger during injection**, all of the solutions will be directed into the **lumen of the needle**.

When **reloading a syringe with a second cartridge** and the needle already in place an eccentric ovoid perforation may occur in the diaphragm and with pressure on the plunger some solution will be directed into the lumen of the needle and some may leak out of the cartridge between the needle and the diaphragm and runs into the patient’s mouth.

2- Broken cartridge The breaking of the cartridge may result from a **bent needle** at its proximal end, **which may not perforate the diaphragm of the cartridge**, positive pressure on the thumb ring increases **intracartridge pressure leading to breakage**.

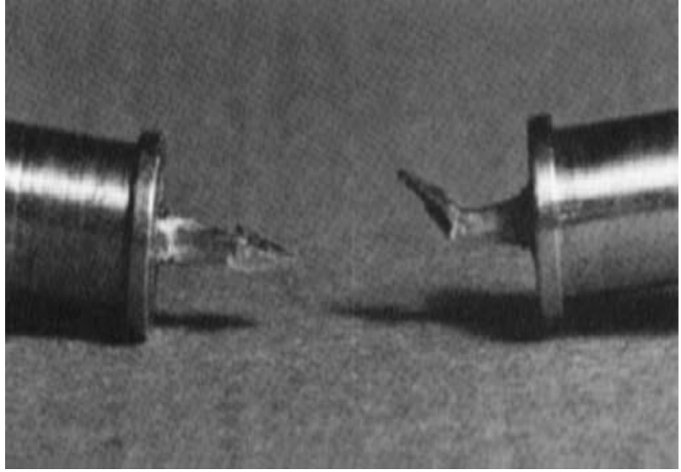
A broken cartridge may also result from a **bent hook of an aspirating syringe**.

Care and Handling of Syringes

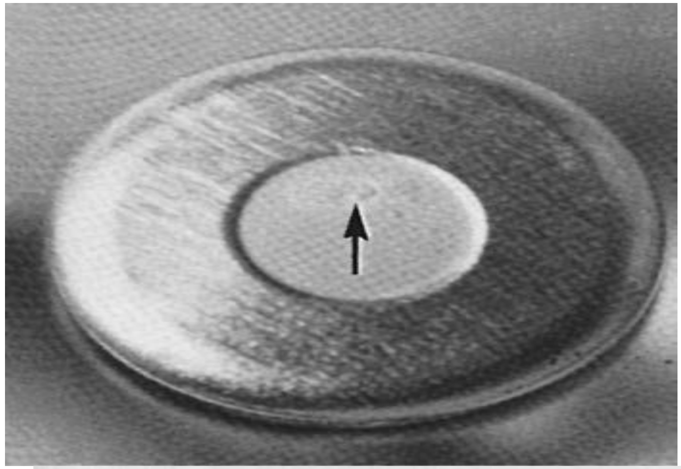
When properly maintained, metal and plastic reusable syringes are designed to provide long-term service. The following **is a summary of manufacturers' recommendations concerning care of these syringes:**

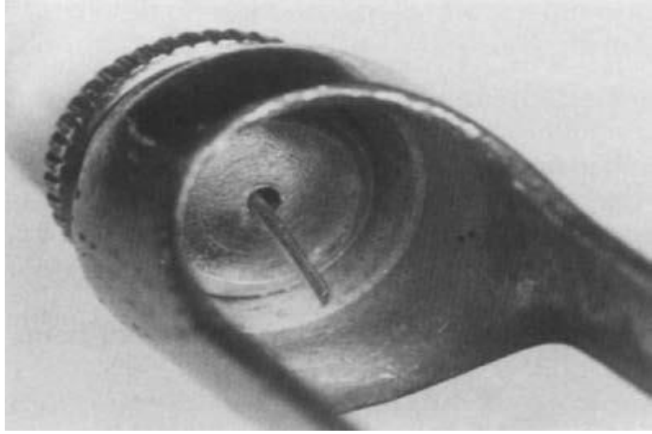
1. After each use, the syringe should be thoroughly washed and rinsed so as to be free of any local anesthetic solution,
saliva, or other foreign matter. **The syringe should be autoclaved in the same manner as other surgical instruments.**
2. After every **five autoclavings**, the syringe should be dismantled and all threaded joints and the area where the piston contacts the thumb ring and the guide bearing should be lightly lubricated.
3. The harpoon should be cleaned with a brush after each use.
4. Although the **harpoon is designed for long-term use**, prolonged use will result in **decreased sharpness** and failure to remain embedded within the stopper of the **cartridge**.

Replacement pistons and harpoons are readily available at low cost.



Bent Harpoon

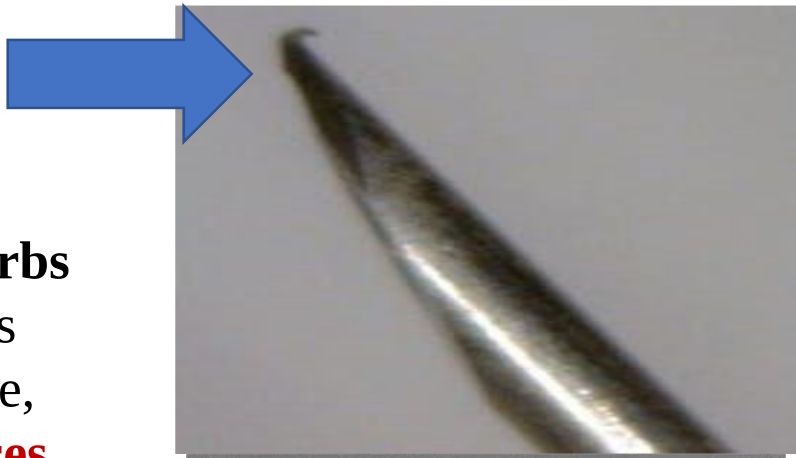




Leakage During Injection

When a syringe is reloaded with a second local anesthetic cartridge and a needle is already in place, care must be taken to ensure that the needle penetrates the center of the rubber diaphragm.

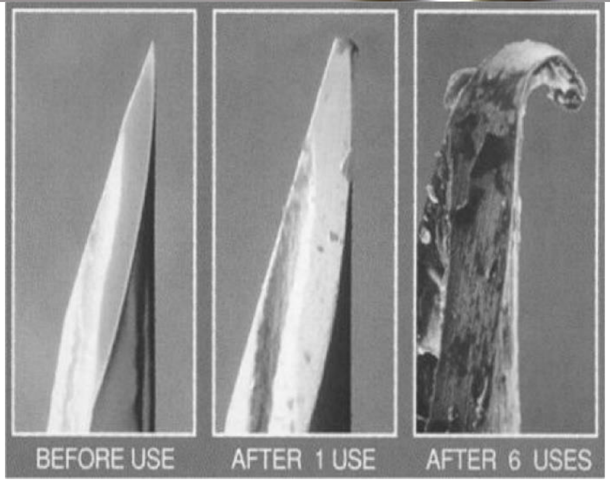
B- Clinical problems related to the dental needle



1 - Pain on withdrawal:

Pain on withdrawal of the needle from the tissue can be produced by **fishhook barbs on the tip**, these barbs may be produced during the manufacturing process but it is more likely that they occur when the needle tip forcefully contact a bone, therefore, needle should not be forced against resistance. **As a result, the patient experiences pain during withdrawal**.

Over time, **most of us adapt our techniques** so that, **regardless of the features** of **our armamentarium**, we can reach our targets for successful anesthesia. However, **needle selection** is an **important component for safe and effective provision of local anesthesia**. It is important that the main criteria for needle selection **include type of injection** to be administered, the distance to **target**, and the **vascularity of tissues.**



2 - Pain on insertion:

This may be **avoided by using sharp**, new disposable needles and the application of topical anesthetic at the penetrating site.

3 - Breakage of the needle:

In general, **bending of the needle during insertion weaken the needle** and **making them more likely to break on subsequent contact** with the hard tissue such as **bone**.

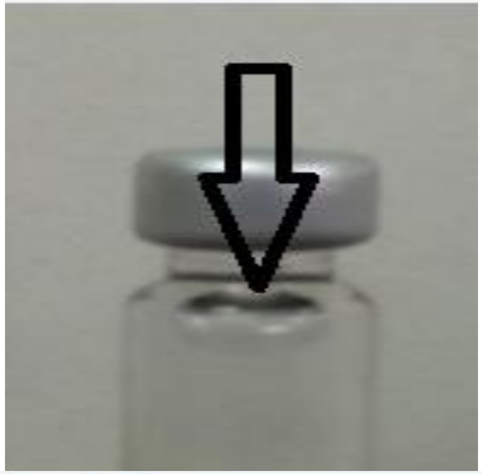
4 - Injury to the patient or the administrator:

Major cause of injury is carelessness by the operator, although sudden unexpected movement by the patient is also a frequent cause, therefore the needle should be capped until its use and should be recapped immediately after withdrawal from the patient's mouth.

C- Clinical problems related to dental cartridge

1 – Bubbles in the cartridge

A small bubble approximately 2 mm in diameter will frequently be found in the dental cartridge. It is composed of nitrogen gas which was bubbled into the local anesthetic solution during its **manufacture to prevent oxygen from being trapped in the cartridge and potentially destroying the vasopressor.**



2 – Extruded stopper

The stopper can be extruded when a cartridge is frozen and the liquid inside expands; in this case, the solution can no longer be considered sterile and should not be used for injection. Also, an extruded stopper may be due to prolonged storage in a chemical disinfecting solution and diffusion of the solution through the rubber diaphragm into the cartridge.

Extruded plunger

3-Damaged cap

A damaged cap (at the aluminum side or at the diaphragm rubber side) indicates that the seal of the cap is not guaranteed and the solution could exit from the neck of the cartridge into the mouth of the patient (Leakage effect).

It also means that air may have entered the cartridge causing contamination and/or degradation of the vasoconstrictor inside. In the case of a damaged cap, discard the cartridge and inform the manufacturer of the issue providing full details of the complaint (lot number, defect description, etc.)

5 – Burning on injection

A burning sensation on injection of the anesthetic solution may be the result of one of the following:

a. Normal response to the PH of the drug**:** The PH of the dental cartridge containing **vasopressor is (3.3- 4)** which is lower than that without **vasopressor (5.5 – 6)** because of this, plain anesthesia has somewhat more rapid onset of clinical action and **more comfortable (less burning on injection).**

b. Cartridge containing sterilizing solution:

This occurs when the cartridges are stored in a disinfectant solution for a long period so we get diffusion of the disinfecting solution into the cartridge upon injecting, it will cause a burning sensation.

c. Overheated cartridge:

Local anesthetic solution injected at room temperature **is well tolerated by the patients**, **overheated cartridge produces burning on injection**

NOTICE*:*

- **Topical antiseptic** may be used to prepare the tissue at the site of injection before the initial needle penetration, to minimize the risk of post-injection infection. Antiseptic like betadine is applied on an applicator stick and placed at the site of injection for 15 – 30 seconds. If a topical antiseptic is not available, a sterile gauze wipe serves to prepare the tissue adequately.
- **A topical anesthetic** could be used to reduce the discomfort during injection.
- **Applicator sticks:** they are wooden sticks with a cotton swab at one end. They can be used to apply topical antiseptic or topical anesthetic to the mucous membrane.
- **Hemostat** will be helpful to remove the needle from the soft tissue; in the event of needle breakage.
- **Finally, proper care and handling of the local anesthetic equipment can prevent or at least minimize the development of complications associated with needle, syringe and the cartridge.**

- Dental cartidge and needles are disposible and never used in more than one patient .
- Needles should be changed after several tissue penetration in the same patient .
- Needles should be coverd with a protective sheath when not being used .
- Administor should be aware the position of the needle tip ,whether inside or outside patient mouth to minimize the risk of injury to the patient or the operator .
- Cartridge should be stored at room tempreture and should never used after their expiry date .

Dr. Hamid Hammad Enezei

PhD

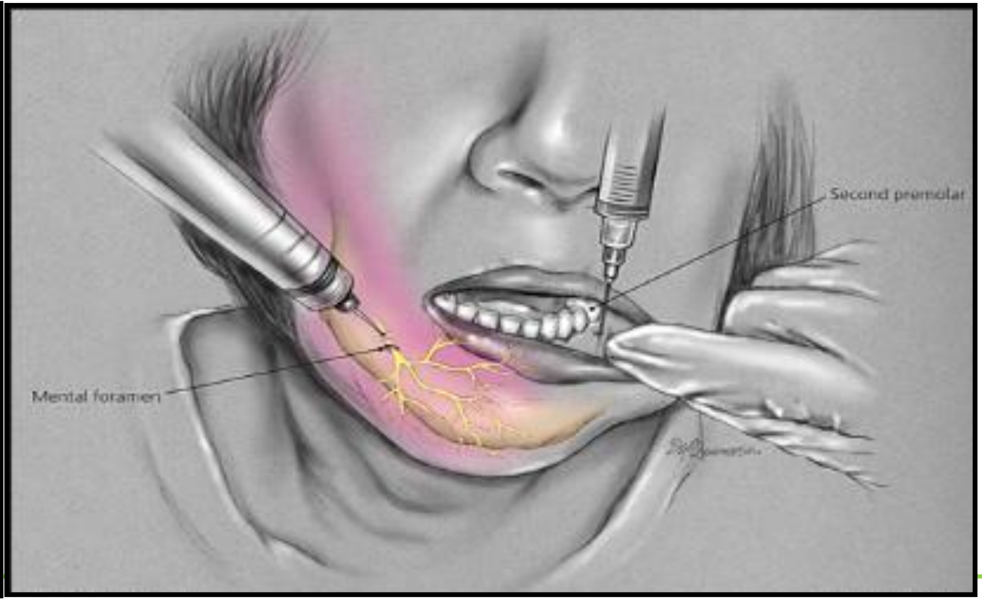
Oral & Maxillofacial Surgery

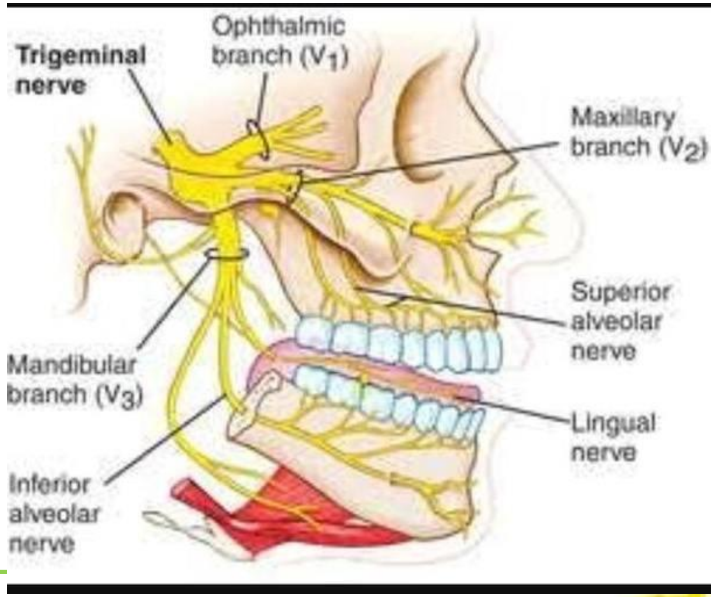
Academic Year 3

[Surgical Anatomy in Local Anesthesia]

Assistant Prof Dr. Hamid Hammad Enezei

Ph.D in Oral & Maxillofacial Surgery

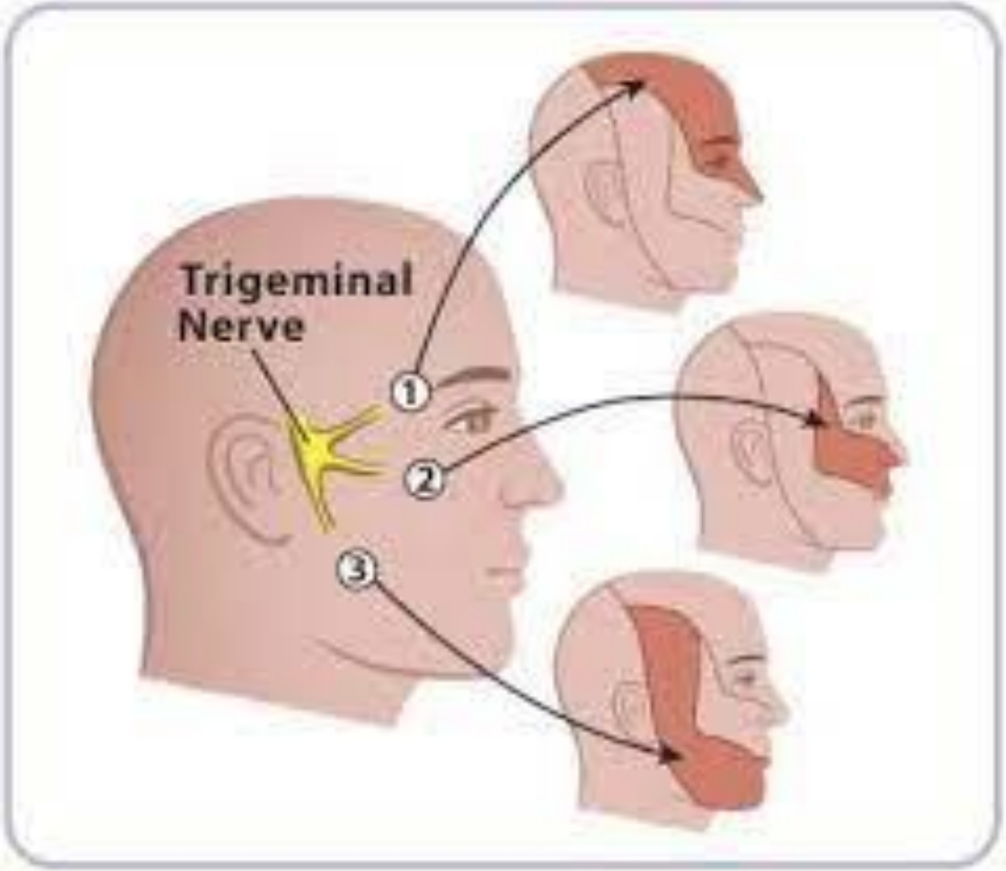




Surgical anatomy in local anesthesia

- Trigeminal nerve:
  - ✓ Ophthalmic branch
  - ✓ Maxillary branch
  - ✓ Mandibular branch

- Osteology of the maxilla
- Osteology of the mandible



The trigeminal nerve :

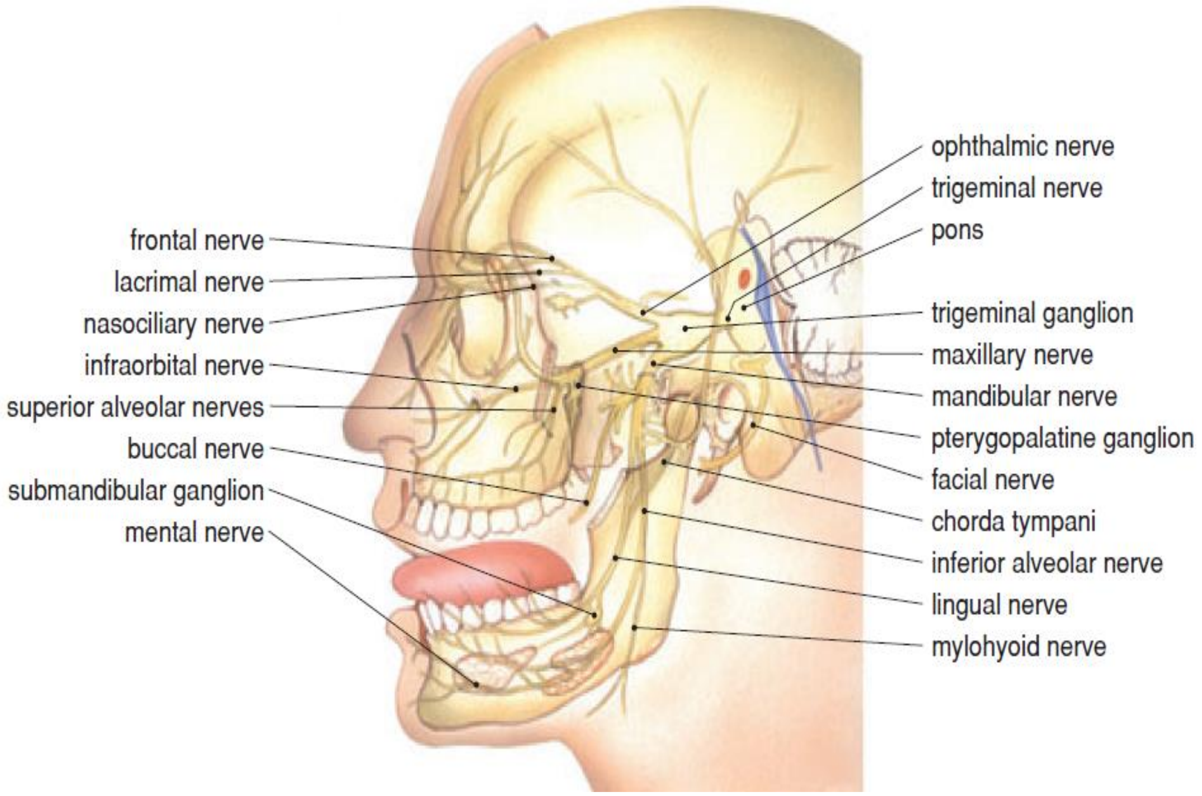
The trigeminal nerve is the largest and most complex of the 12 cranial nerves (CNs).

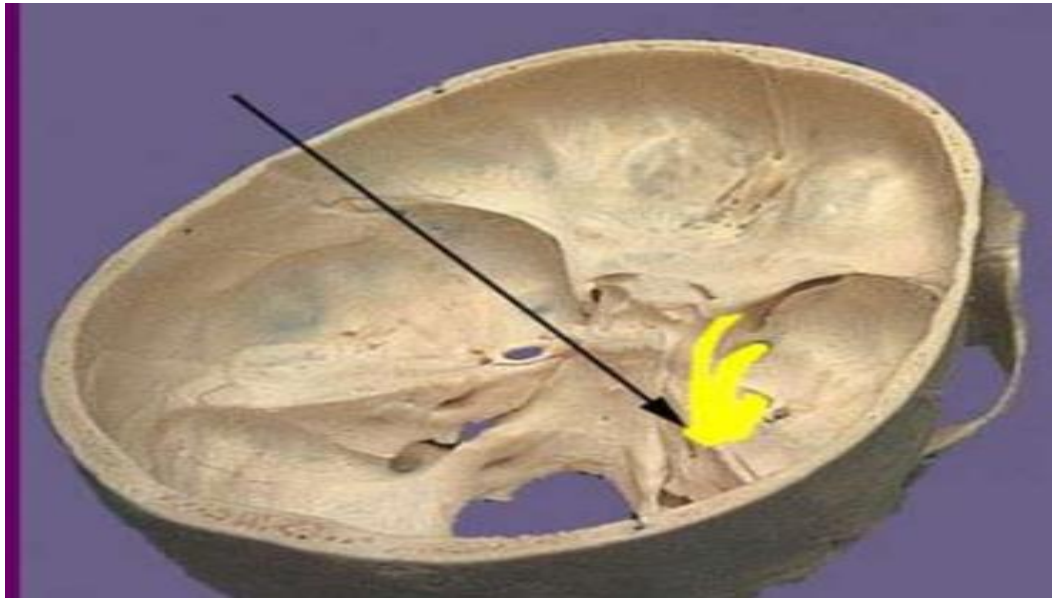
It supplies sensations to the face, mucous membranes, and other structures of the head. It is the **motor nerve for the muscles of mastication** and some other muscles .

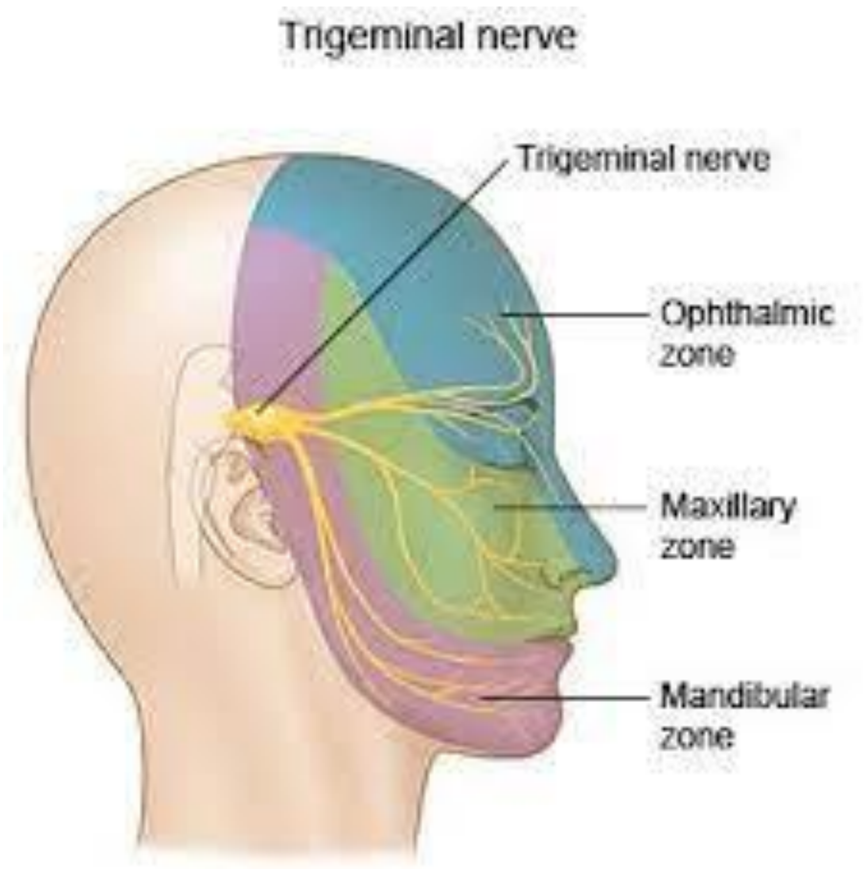
It **contains proprioceptive fibers**.

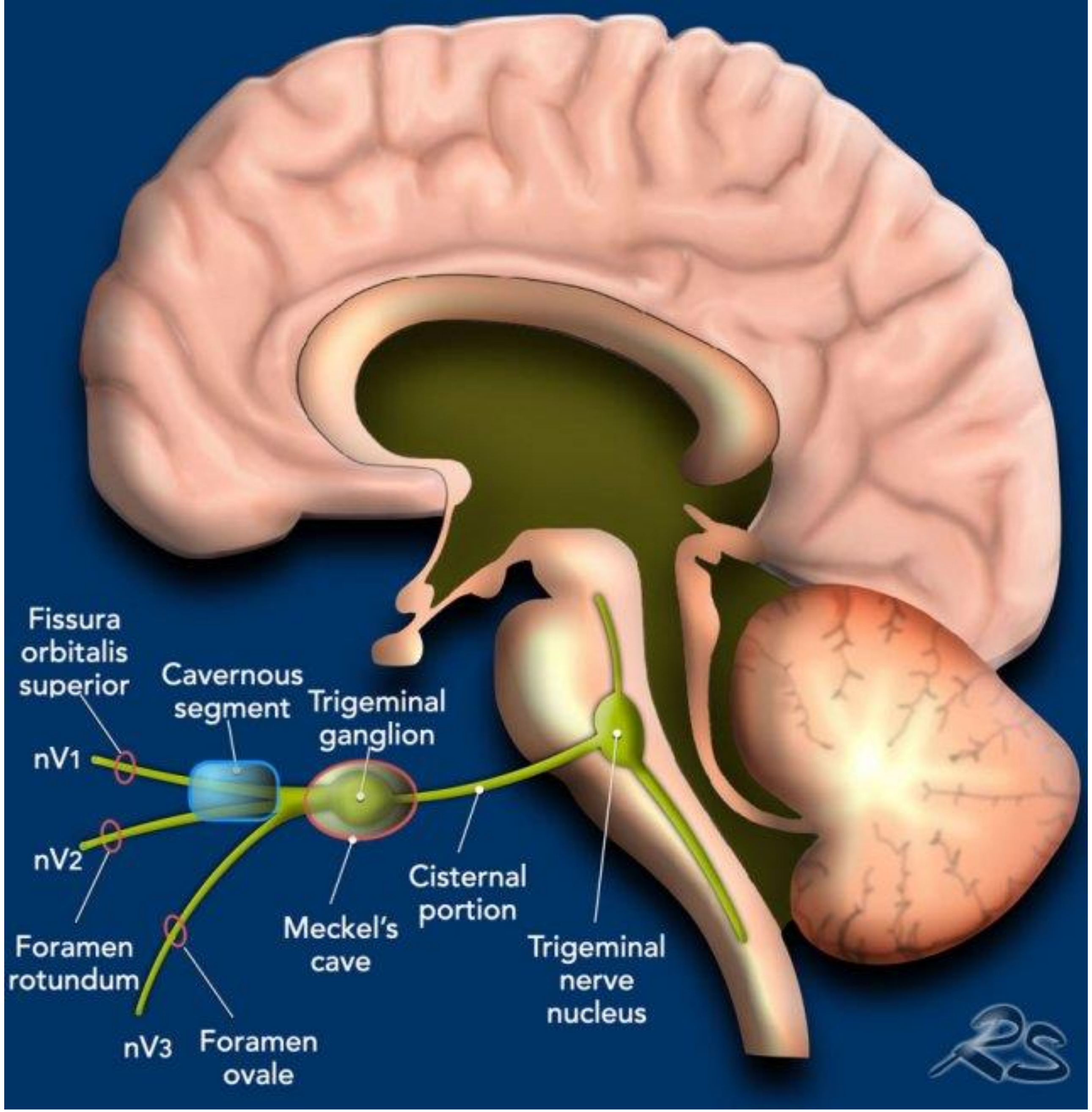
It exits the brain by a **large sensory root and a smaller motor root coming out of the pons at its junction with the middle cerebral peduncle**. **It passes laterally to join the gasserian (semilunar) ganglion in the Meckel cave.**

The Gasserian ganglion(semilunar ganglion), is a sensory ganglion of the trigeminal nerve that occupies a cavity (**Meckel's cave**) in the **dura mater**, covering the trigeminal impression near the apex of the petrous part of the temporal bone.



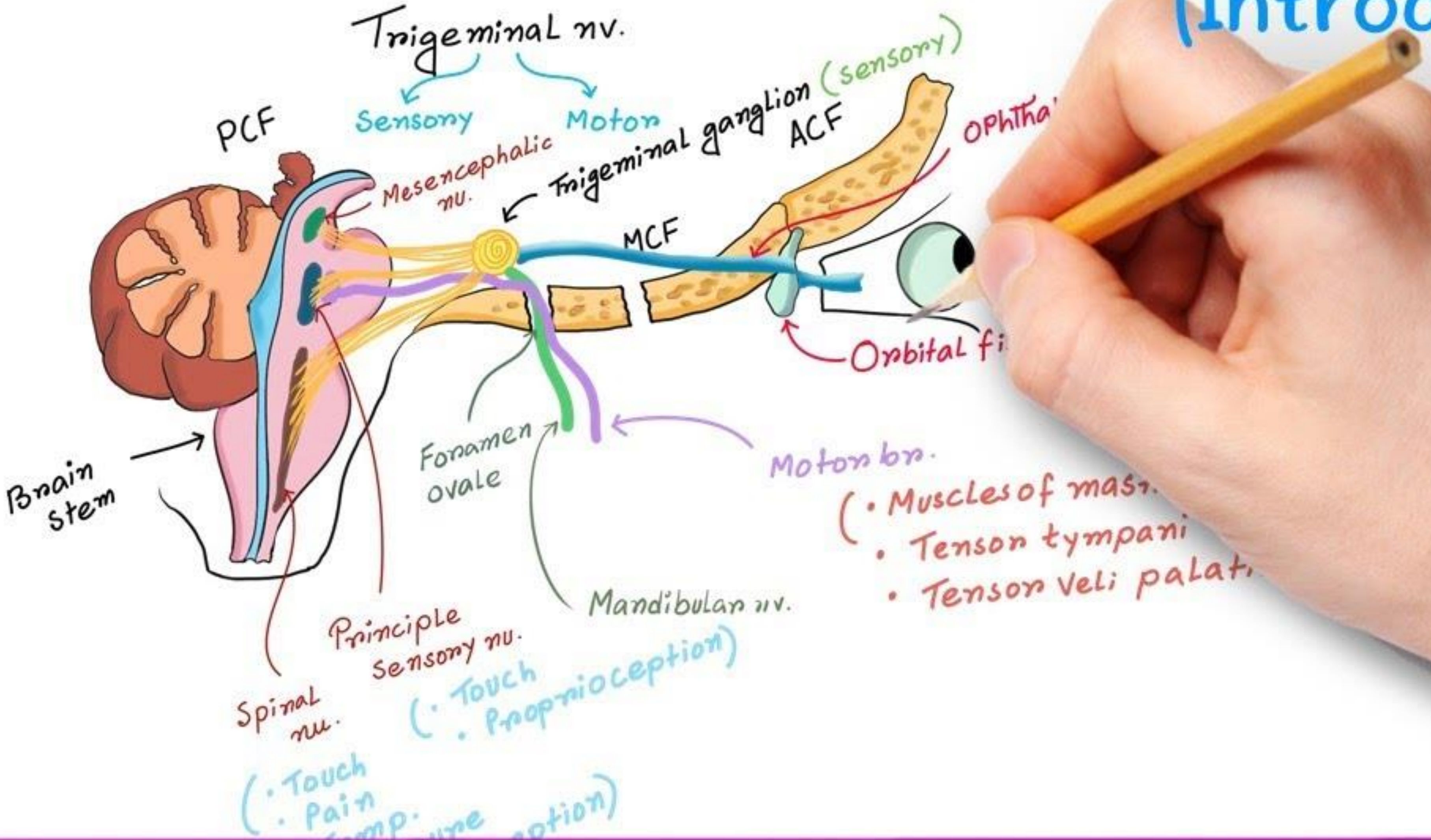



Trigeminal nerve
Trigeminal nerve
Ophthalmic zone
Maxillary zone
Mandibular zone

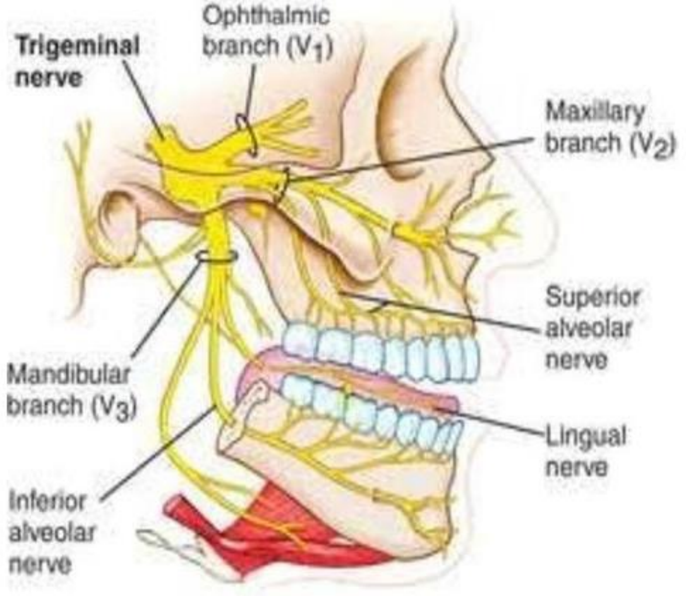

Fissura orbitalis superior
Cavernous segment
Trigeminal ganglion
nV1
nV2
Foramen rotundum
Meckel's cave
Cisternal portion
Trigeminal nerve nucleus
nV3
Foramen ovale

Trigeminal Nerve

(Introduction)



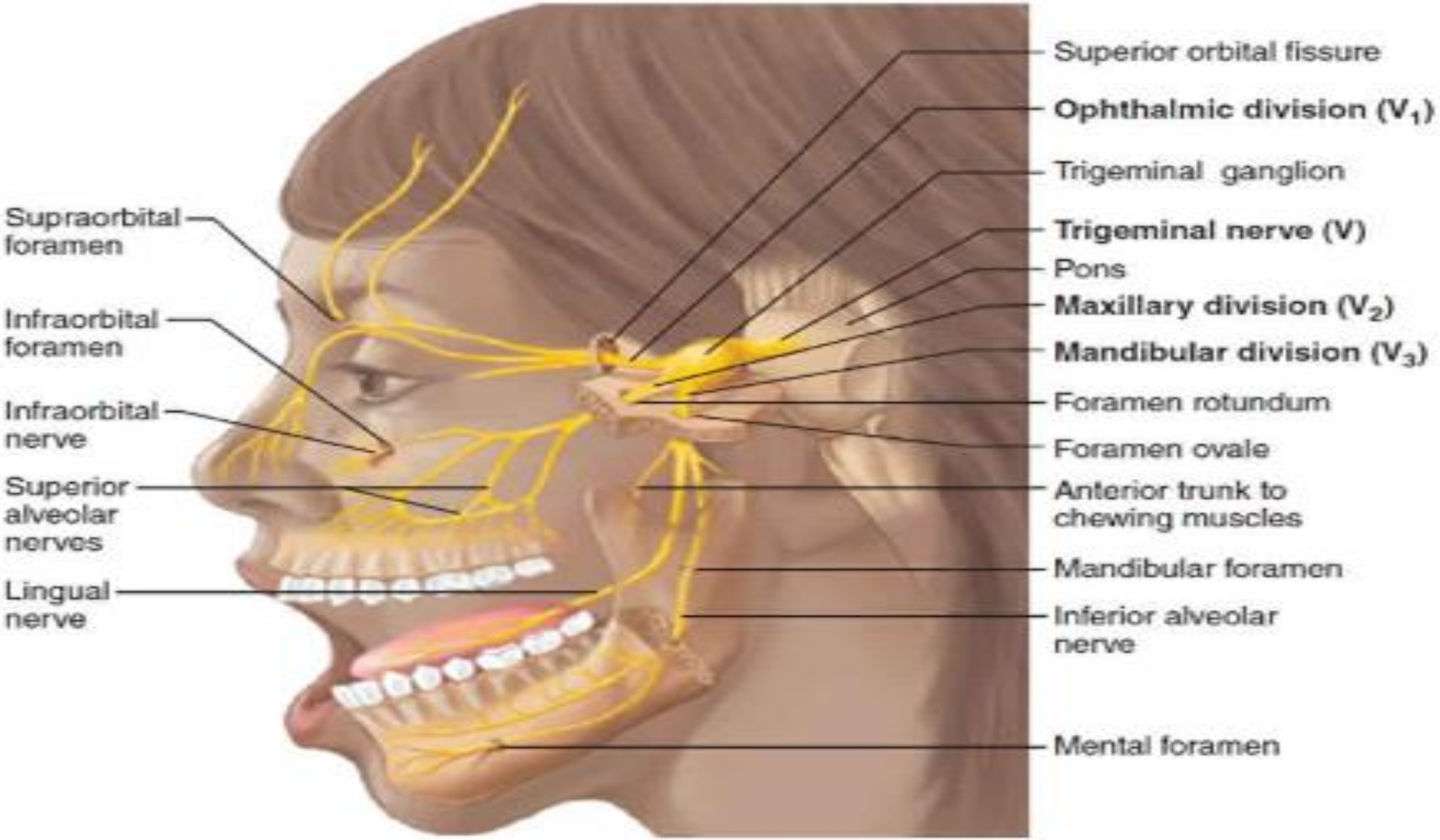
The trigeminal nerve contains a large number of **sensory (afferent)** and **motor (efferent) neurons**. The sensory fibres carry nerve impulses towards the **central nervous system**, while the motor **fibres carry impulses away from the central nervous system**.



The trigeminal nerve has a wide **innervation area** .The nerve provides the sensitivity of the **dentition**, the **mucosa of the mouth, nose and paranasal sinuses,** and the **facial skin**. The nerve also contains **motor fibres that innervate, among** others, the **masticatory muscles**.

Although the trigeminal nerve is the most important nerve for the **sensory and motor innervation of the oral system**, the facial (n. VII), glossopharyngeal (n. IX), vagus (n. X), accessory (n. XI) ,and hypoglossal (n. XII) nerves are also of significance.

The n. VII, n. IX and n. X, for example, take care of the taste sense, the n. IX and n. X provide the general sensation (pain, touch and temperature) to the pharynx, soft palate and the back of the tongue, whilst the n. XII is responsible for the motor innervation of the tongue. Although these latter nerves do play an important role in innervating the oral cavity.



Supraorbital foramen
Infraorbital foramen
Infraorbital nerve
Superior alveolar nerves
Lingual nerve
Superior orbital fissure
Ophthalmic division (V_1)
Trigeminal ganglion
Trigeminal nerve (V)
Pons
Maxillary division (V_2)
Mandibular division (V_3)
Foramen rotundum
Foramen ovale
Anterior trunk to chewing muscles
Mandibular foramen
Inferior alveolar nerve
Mental foramen

The trigeminal nerve emerges from the middle of the pons, at the lateral surface of the brainstem. The nerve consists of two parts here: the sensory fibers form a thick root and the motor fibers form the much thinner motor root. These two roots run to the front of the petrous part of the temporal bone where the large sensory trigeminal ganglion (semilunar or Gasserian ganglion) lies in a shallow groove surrounded by dura mater.

The trigeminal ganglion is formed by the aggregation of cell bodies of sensory neurons. Three main branches of the trigeminal nerve emerge from the ganglion: the ophthalmic nerve (n. V1), the maxillary nerve (n. V2) and the mandibular nerve (n. V3). The motor root joins the mandibular nerve only, once it has exited the skull via the foramen ovale.

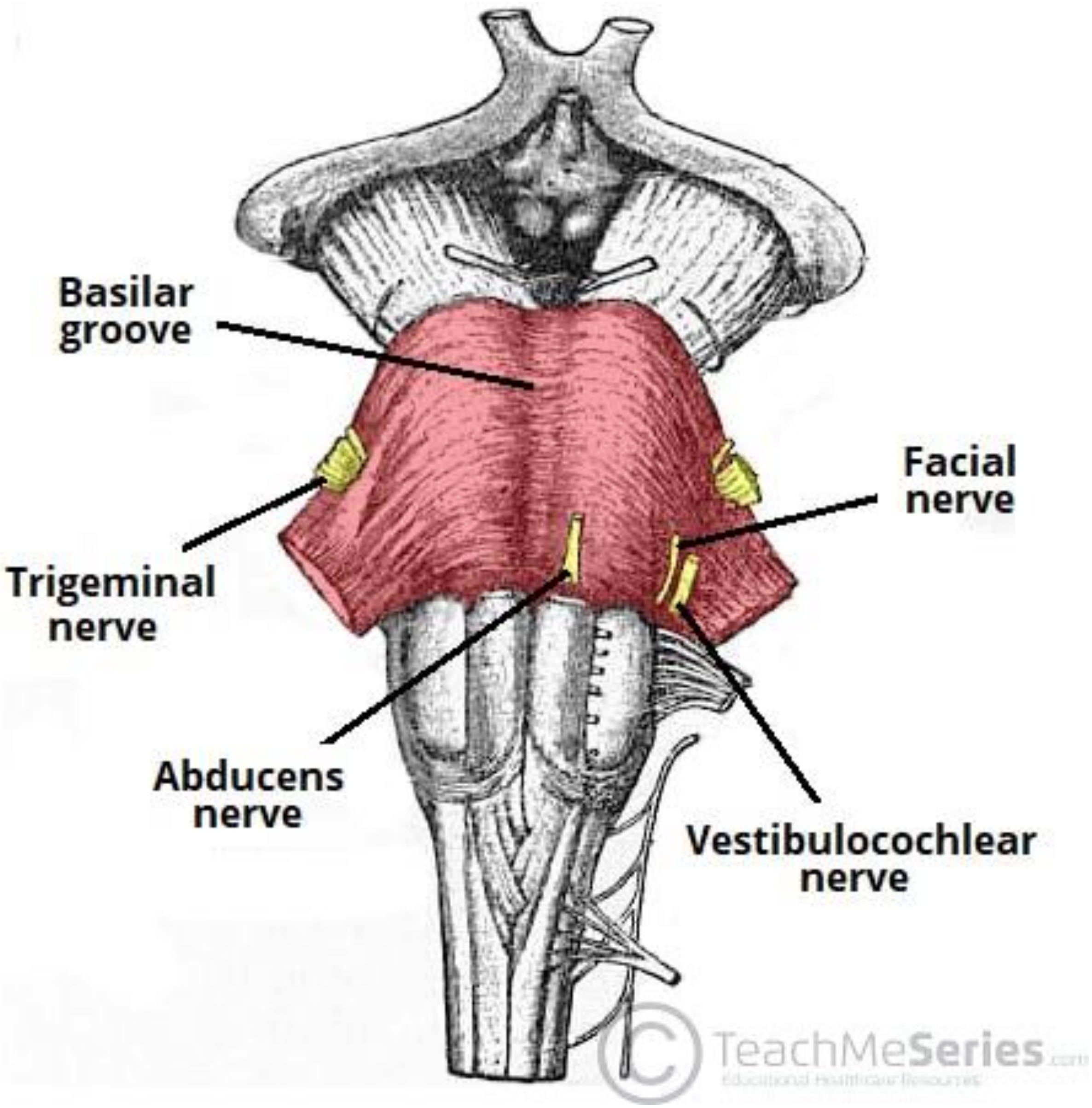
The sensory areas covered by the three main branches are generally as follows:

- **The ophthalmic nerve** carries sensory information from the skin of the forehead, the upper eyelids and the nose ridge, and part of the nasal mucosa.
- **The maxillary nerve** innervates the skin of the middle facial area, the side of the nose and the lower eyelids, the maxillary dentition, part of the nasal mucosa (including the maxillary sinus) and the palate.
- **The mandibular nerve innervates** the skin of the lower facial area, the mandibular dentition, the mucosa of the lower lip, cheeks and floor of the mouth, part of the tongue and part of the external ear.

Of all the areas that the trigeminal nerve innervates, the oral cavity is the most enriched with sensory neurons. The density of sensory neurons in the mouth is much larger than in any other area, e.g. the facial skin.

This density of sensory neurons increases from the back to the frontal area of the mouth.

Most of the trigeminal ganglion neurons are **pseudo-unipolar.** This means that each neuron in the ganglion has a peripheral and a central process. The peripheral process (axon) is relatively long and carries the impulses coming from **sensory receptors.**



Basilar groove
Facial nerve
Trigeminal nerve
Abducens nerve
Vestibulocochlear nerve
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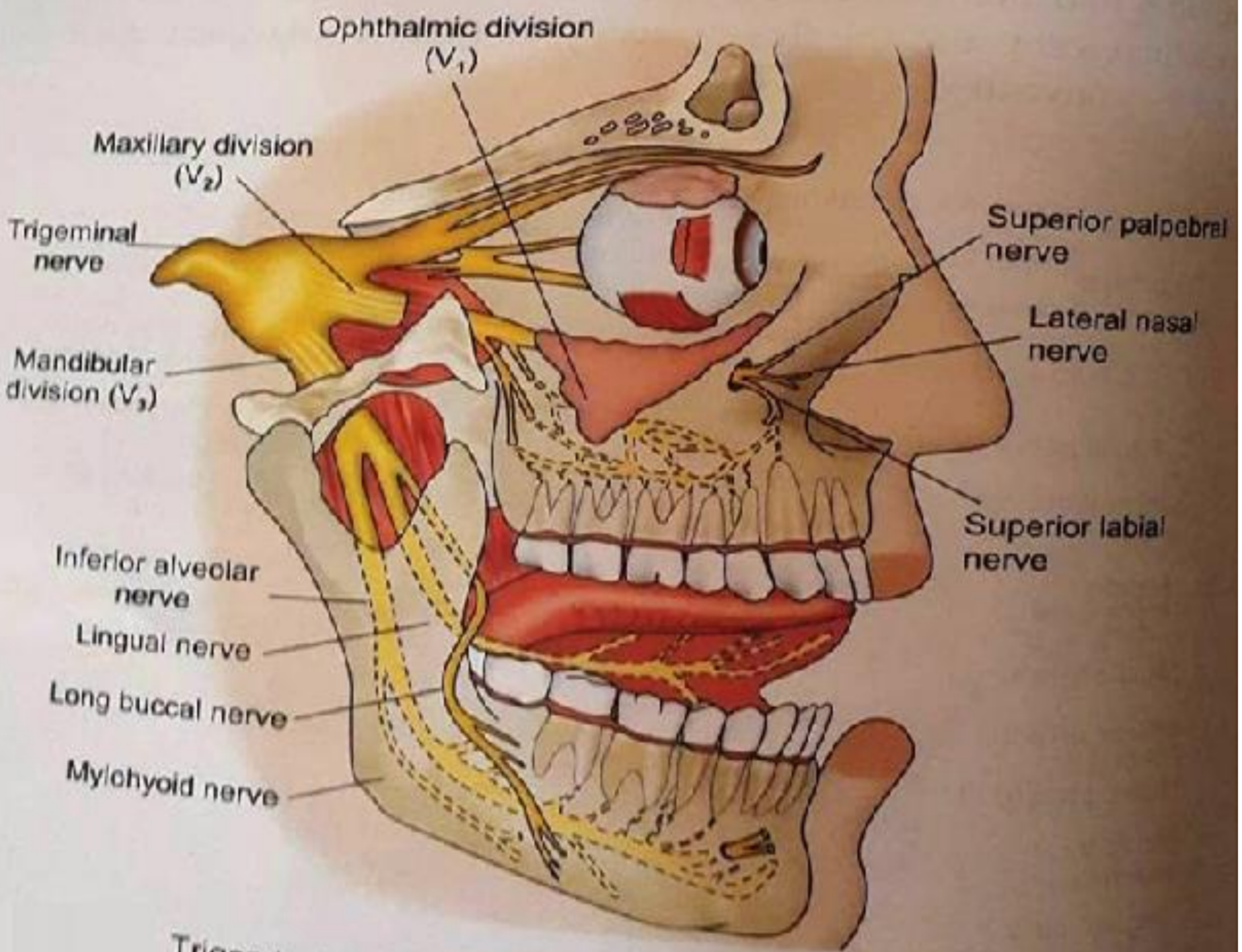


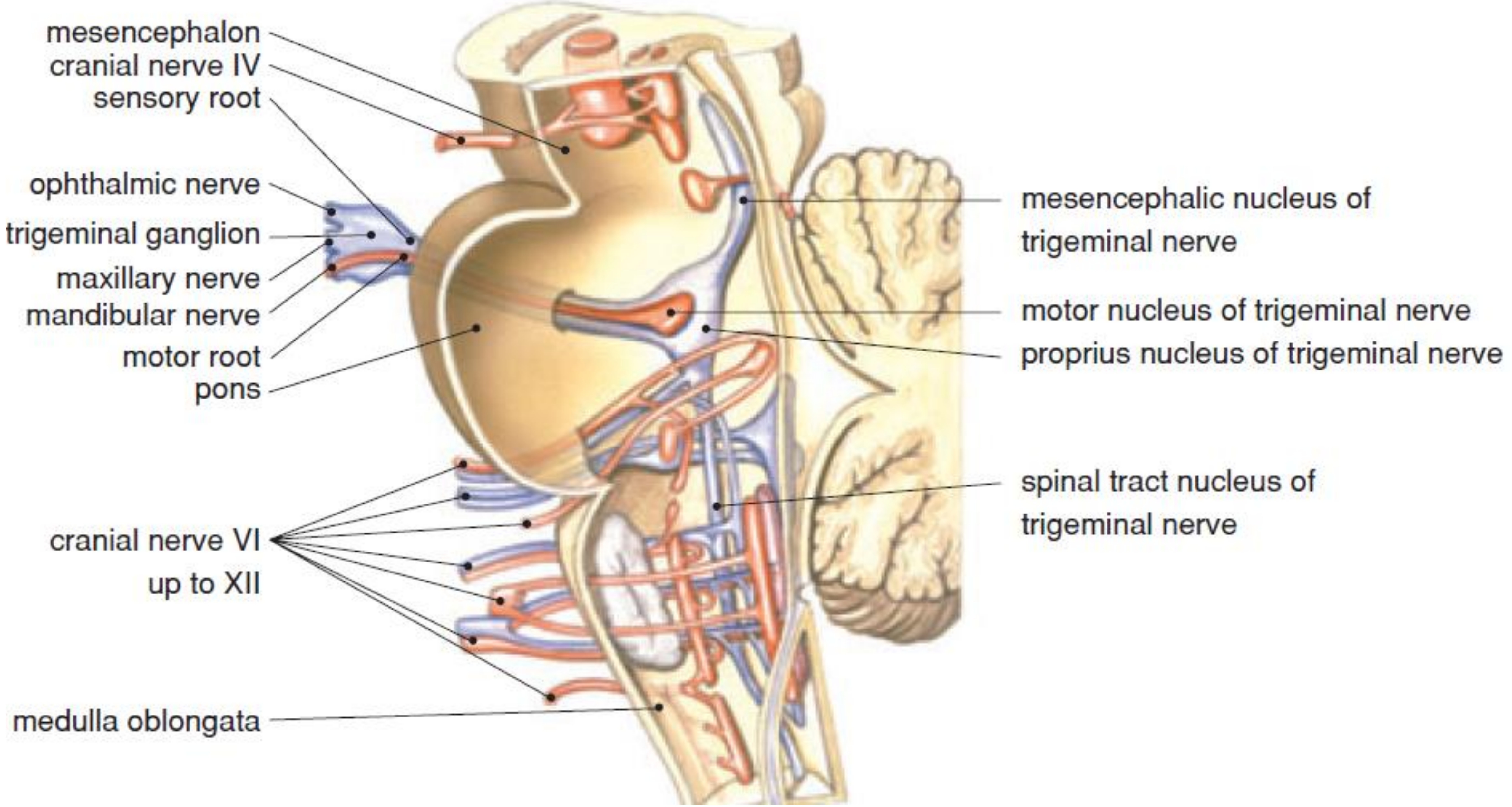
Trigeminal nerve and its branches in relation to maxilla, mandible and teeth (diagrammatic)

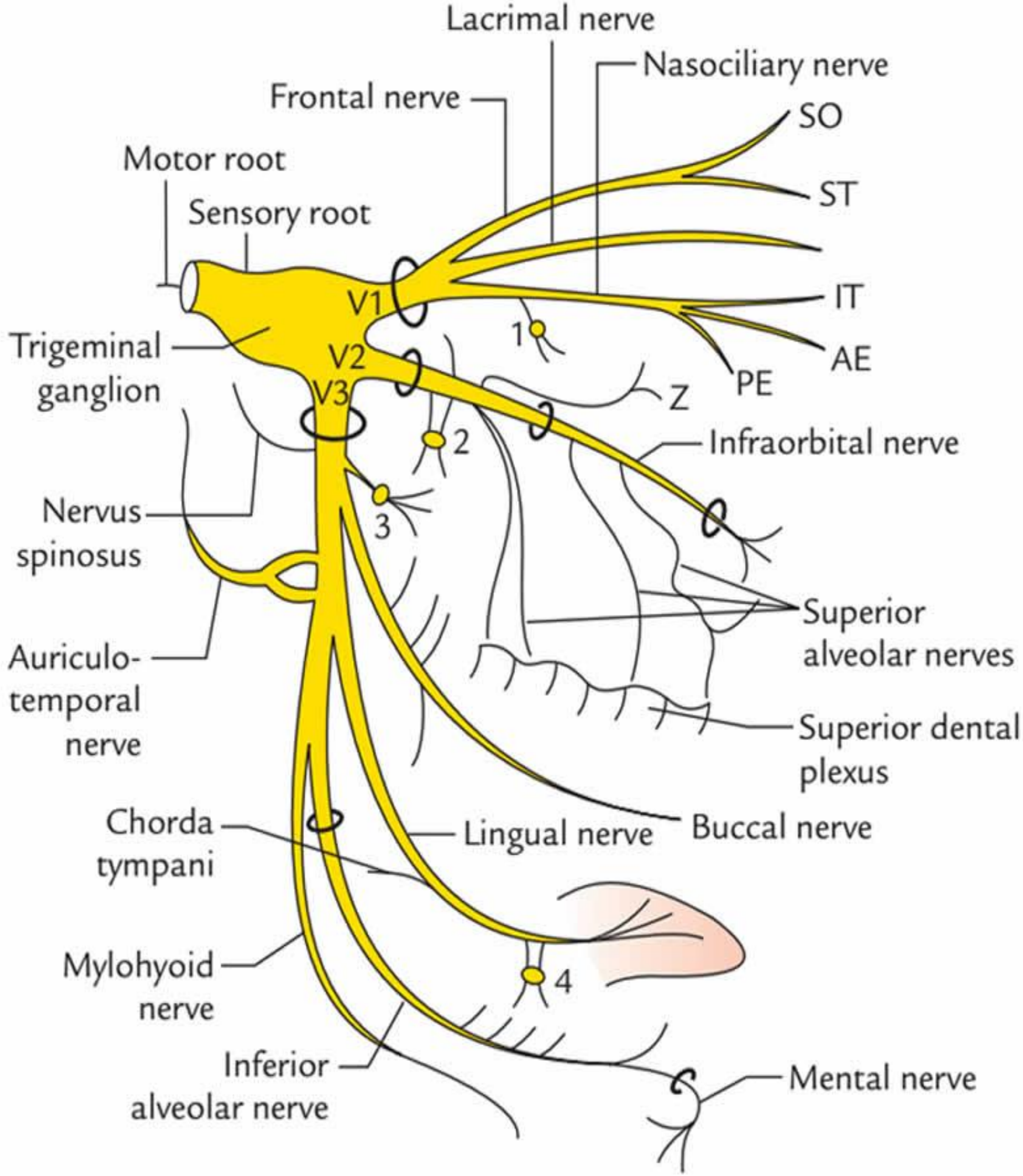
Sensory nerves are capable of picking up impulses from the external world and the body. The ends of the fibres themselves function as **receptors** or there are **special receptors** (e.g. **taste receptors**, **muscle spindles**).

Each receptor type is the most **sensitive to one specific sensation**. There are, for example, **mechano-receptors** (**reacting to touch and mild pressure**), **thermo-receptors** (reacting to temperature) and nociceptors (reacting to tissue damage). Nociceptors serve pain sensation. There are also so-called **proprioceptors**.

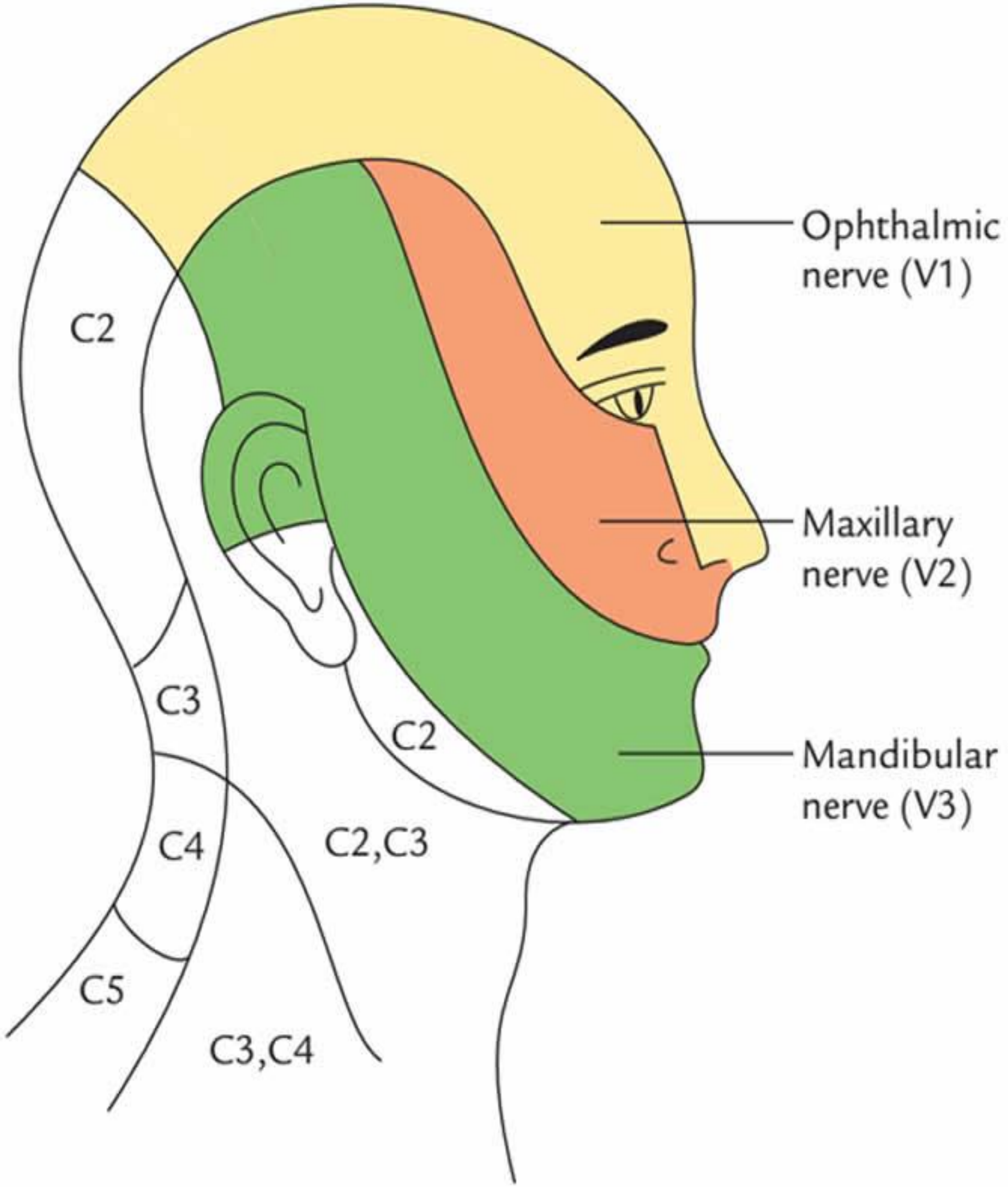
These are mostly found in muscles (muscle spindles and Golgi tendon organs) and in the joint capsules. They supply information on the position of the jaw and the speed and direction of movement. This form of sensation is called **proprioceptive sensibility or proprioception**. There are a great number of receptors present in the facial skin and lips, in the mucosa of the oral cavity and tongue, in the teeth and the periodontium, and in the masticatory muscles and temporomandibular joint.

The trigeminal nerve has a **sensory** and **motor nucleus within the brainstem** .
The sensory nucleus lies laterally and most of the sensory neurons of the trigeminal nerve contact (synapse) with the neurons in this nucleus.
The nucleus forms a **long column that extends from the midbrain to the spinal cord**. It consists of (from cranial to caudal) the mesencephalic, the principal and the spinal trigeminal nuclei.


mesencephalon
cranial nerve IV
sensory root
ophthalmic nerve
trigeminal ganglion
maxillary nerve
mandibular nerve
motor root
pons
cranial nerve VI
up to XII
medulla oblongata
mesencephalic nucleus of
trigeminal nerve
motor nucleus of trigeminal nerve
proprius nucleus of trigeminal nerve
spinal tract nucleus of
trigeminal nerve

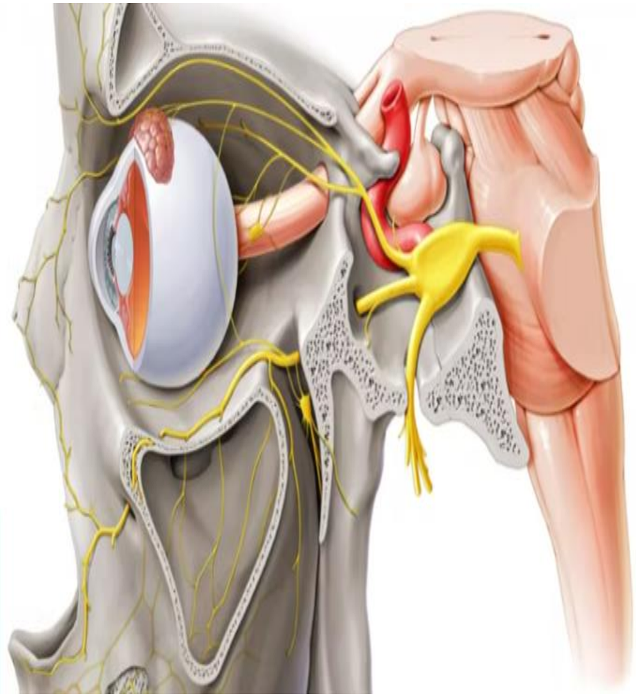


Lacrimal nerve
Nasociliary nerve
Frontal nerve
SO
Motor root
ST
Sensory root
V1
IT
Trigeminal ganglion
1
AE
V2
PE
V3
Z
2
Infraorbital nerve
3
Nervus spinosus
Superior alveolar nerves
Auriculo-temporal nerve
Superior dental plexus
Chorda tympani
Lingual nerve
Buccal nerve
4
Mylohyoid nerve
Inferior alveolar nerve
Mental nerve



C2
Ophthalmic nerve (V1)
Maxillary nerve (V2)
C3
C2
Mandibular nerve (V3)
C4
C2,C3
C5
C3,C4

The ophthalmic nerve:

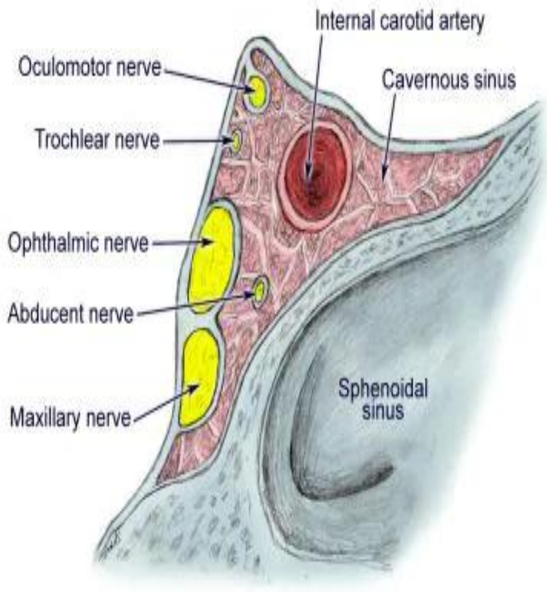


The ophthalmic nerve carries **sensory information** from the **scalp and forehead**, the upper **eyelid**, the **conjunctiva and cornea of the eye**, **the nose (including the tip of the nose, except alae nasi)**, **the nasal mucosa**, **the frontal sinuses**, and **parts of the meninges (the dura and blood vessels).**

The ophthalmic nerve **receives sympathetic filaments from the cavernous sinus** and **communicating branches from CN III and IV**.

Just before it exits the skull through the superior orbital fissure, **it gives off a dural branch**, and **then divides into 3 branches**: the **frontal**, **lacrimal**, and **nasociliary**

Frontal nerve:



This is the **largest branch** of the ophthalmic nerve It passes in the lateral part of the superior orbital fissure, below the lacrimal nerve and above CN IV, between the periorbita and levator palpebrae superioris.

It divides in the **middle of the orbit** **into the** **supraorbital** **(larger branch**) and **supratrochlear nerves.**

- The supraorbital nerve It supplies the upper lid.
- The supratrochlear nerve supplies the conjunctiva and the skin of the upper lid, as well as to the lower and medial parts of the forehead.

Lacrimal nerve: It supplies the lacrimal gland, conjunctiva, and upper lid. In the orbit, it receives a communication from the zygomatic branch of the maxillary nerve. This represents postganglionic parasympathetic secretory fibers from the sphenopalatine ganglion to the lacrimal gland. The preganglionic fibers reach the ganglion via the greater petrosal and vidian nerves from CN VII.

Nasociliary nerve

After passing through the superior orbital fissure to supply the **frontal and anterior ethmoid sinuses**.

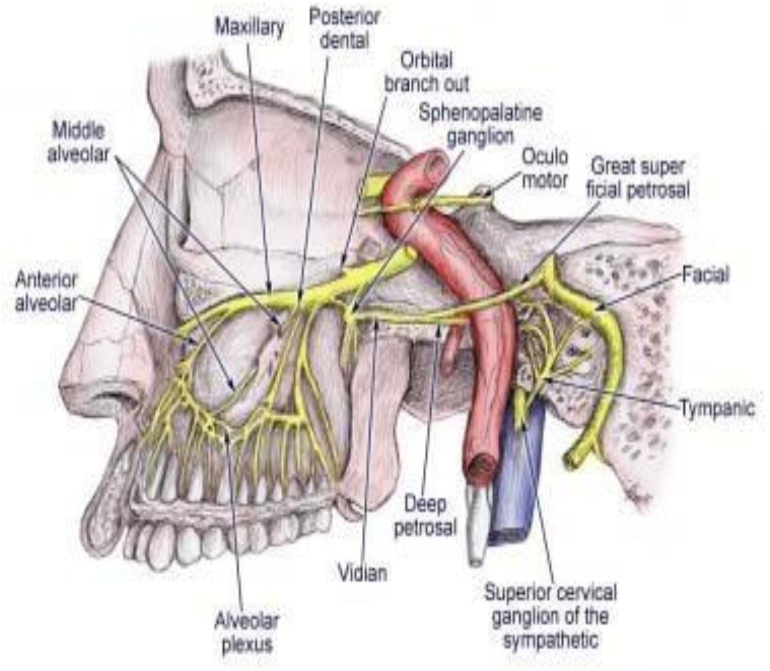
SUMMARY TABLE 14–9 Cranial Nerve Branches and Functions

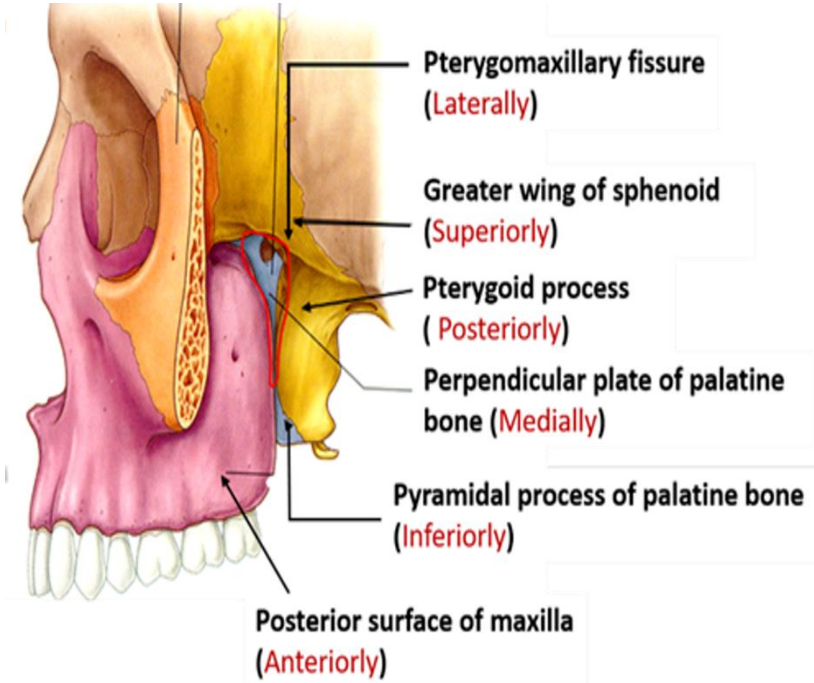
Cranial Nerve (Number)	Sensory Ganglion	Branch	Primary Function	Foramen	Innervation
Olfactory (N I)			Special sensory	Olfactory foramina of ethmoid	Olfactory epithelium
Optic (N II)			Special sensory	Optic canal	Retina of eye
Oculomotor (N III)			Motor	Superior orbital fissure	Inferior, medial, superior rectus, inferior oblique and levator palpebrae superioris muscles; intrinsic eye muscles
Trochlear (N IV)			Motor	Superior orbital fissure	Superior oblique muscle
Trigeminal (N V)	Semilunar		Mixed	Superior orbital fissure	Areas associated with the jaws
		Ophthalmic	Sensory	Superior orbital fissure	Orbital structures, nasal cavity, skin of forehead, upper eyelid, eyebrows, nose (part)
		Maxillary		Foramen rotundum	Lower eyelid; superior lip, gums, and teeth; cheek, nose (part), palate, and pharynx (part)
		Mandibular		Foramen ovale	*Sensory*: inferior gums, teeth, lips, palate (part), and tongue (part) *Motor*: muscles of mastication

Maxillary nerve:

The maxillary nerve (n. V2), too, is solely sensory.
It enters the pterygopalatine fossa via the **foramen rotundum**. Through the **inferior orbital fissure** it reaches the **floor of the orbit** and proceeds there as **the infraorbital nerve**, **first i**n the **infraorbital sulcus** and then in the **infraorbital canal**. It then reaches **the face** via the **infraorbital foramen.**
Within the **pterygopalatine fossa** the **maxillary nerve** is connected via a number of branches to the upper side of the **parasympathetic pterygopalatine ganglion**.
Sensory fibers run through these branches which exit on the lower side of the ganglion .
These sensory fibers form, among others, the **following nerves**:

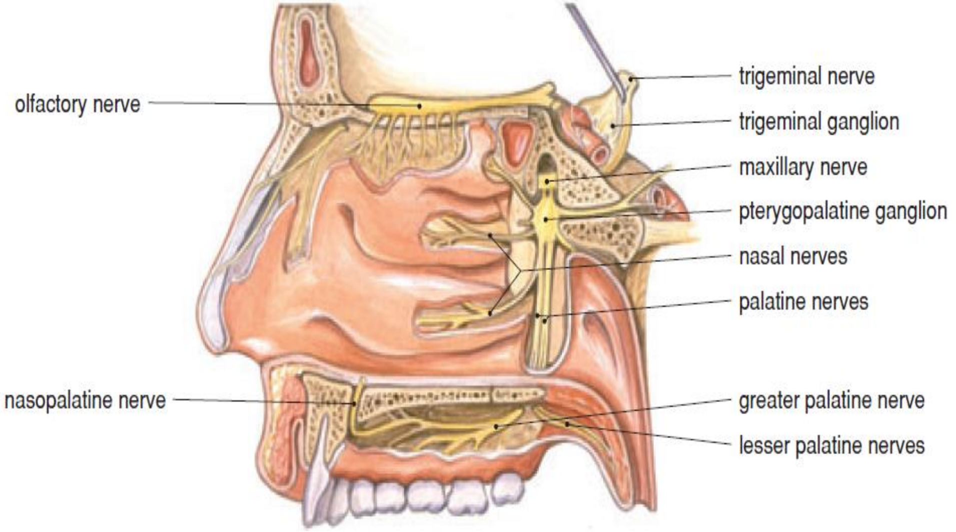
• **The nasal nerves** and nasopalatine nerve that run through the sphenopalatine foramen to the nasal mucosa. The nasal nerves innervate the back part of the nasal mucosa. The nasopalatine nerve, which runs forwards over the nasal septum and reaches the oral cavity through the incisive canal, innervates the frontal part of the palatal mucosa and the palatal gingiva of the maxillary incisors.



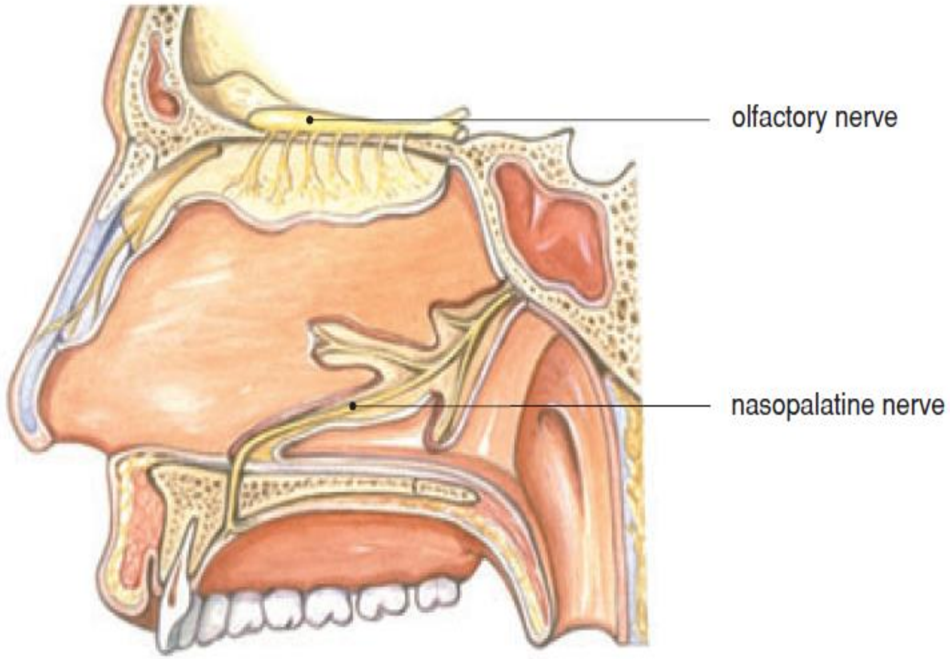


- **The greater palatine** nerve that runs via the greater palatine canal to the mucosa of the hard palate and to the palatal gingiva of the maxillary alveolar process.
- **The lesser palatine nerves** that run to the mucosa of the soft palate via the lesser palatine canals.

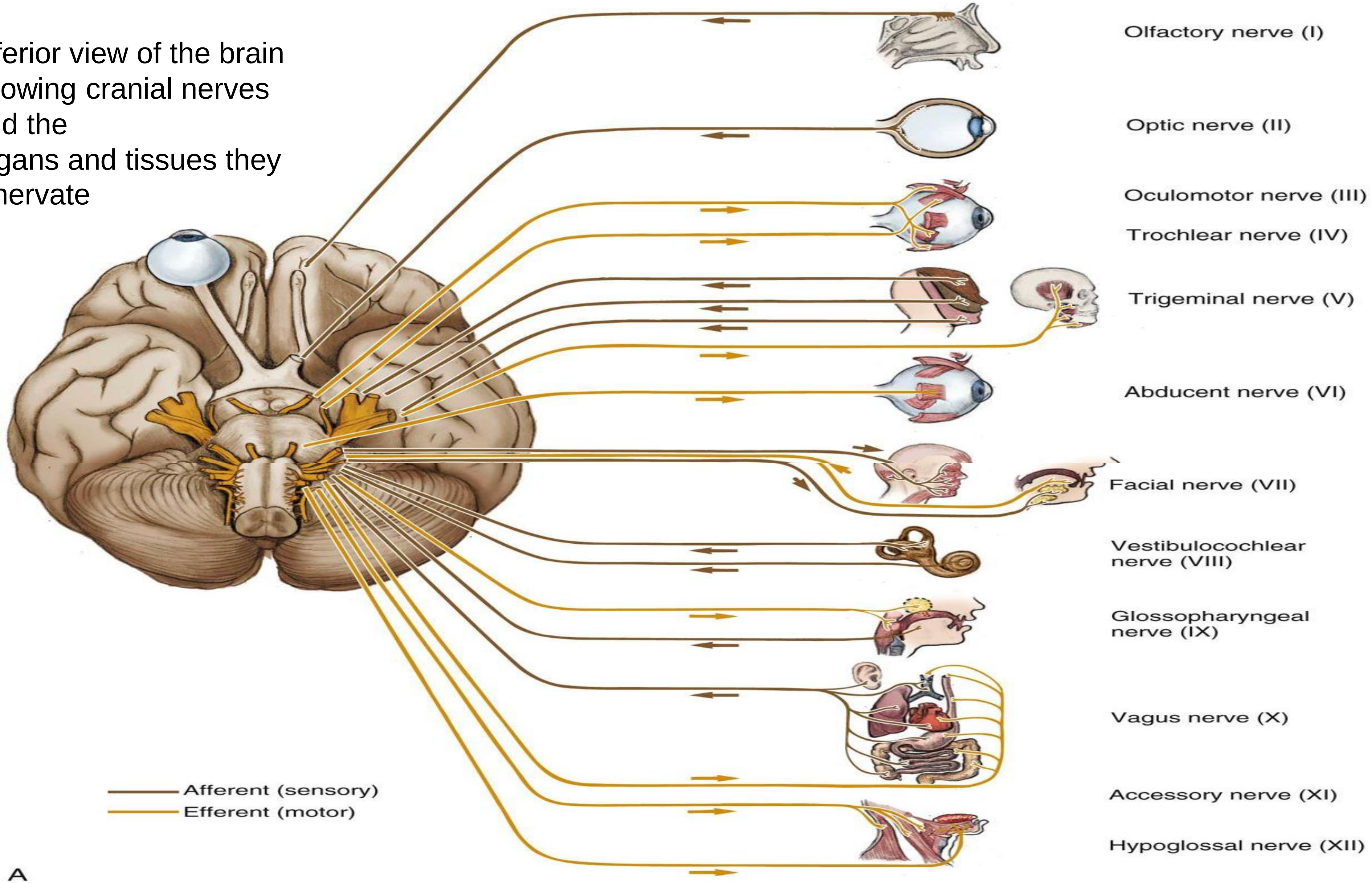
Together with the palatine nerves there are also **parasympathetic** and **orthosympathetic fibres** that run from the **pterygopalatine ganglion** to the **salivary glands in the palatal mucosa**.

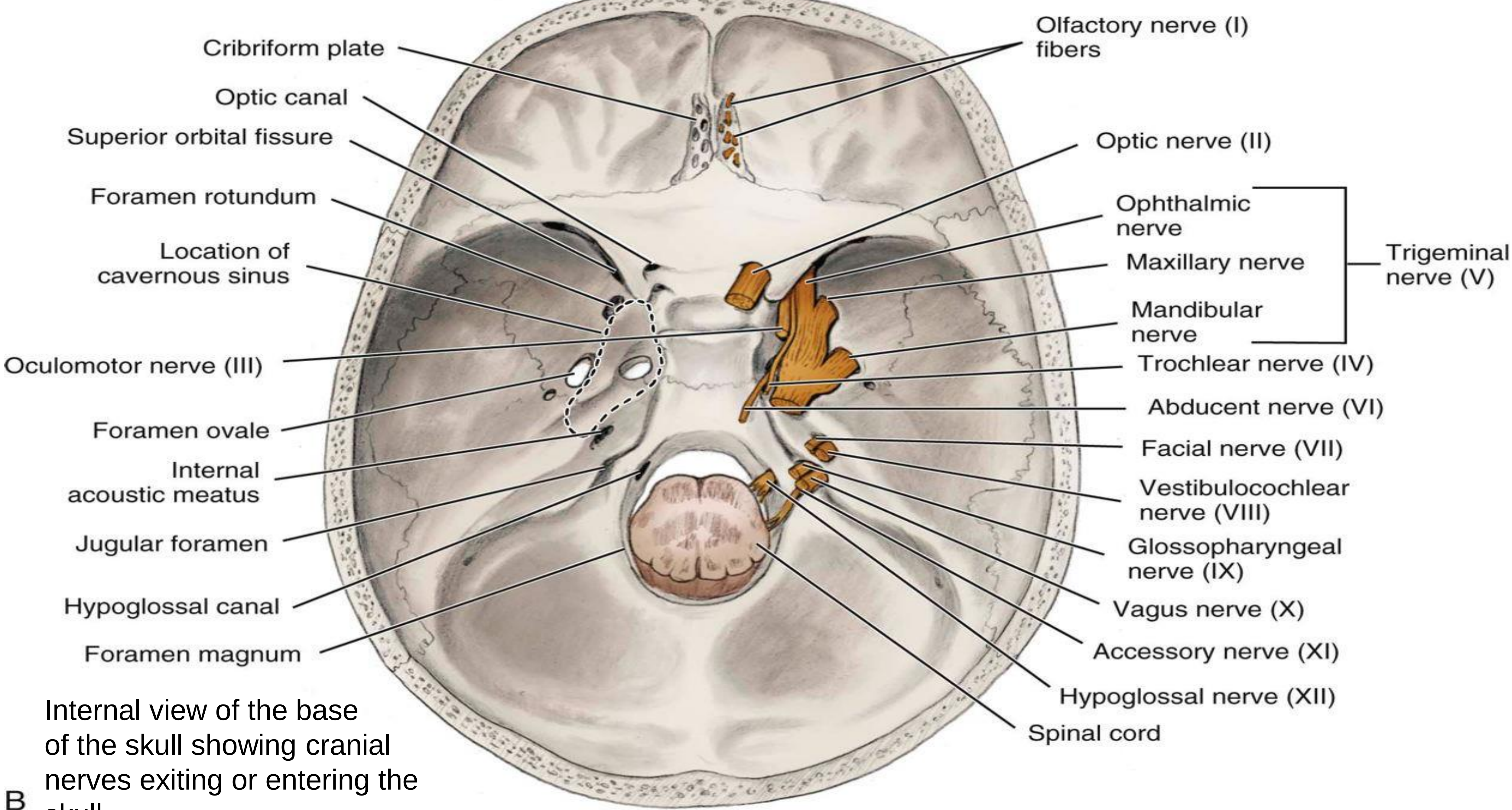


In the **pterygopalatine fossa** the maxillary nerve also branches into the **posterior superior alveolar nerve** and the **zygomatic nerve.**
The posterior superior alveolar nerve exits the pterygopalatine fossa through the **pterygomaxillary fissure** and runs over **the maxillary tuberosity**.
The nerve divides into a **large number of little branches**, **the posterior superior alveolar rami,** which enter the wall of the maxilla through small openings and innervate the **maxillary molars** and **corresponding buccal gingiva**.
The zygomatic nerve arrives in the **orbit** via the inferior orbital fissure and branches into the zygomaticotemporal and zygomaticofacial nerves.



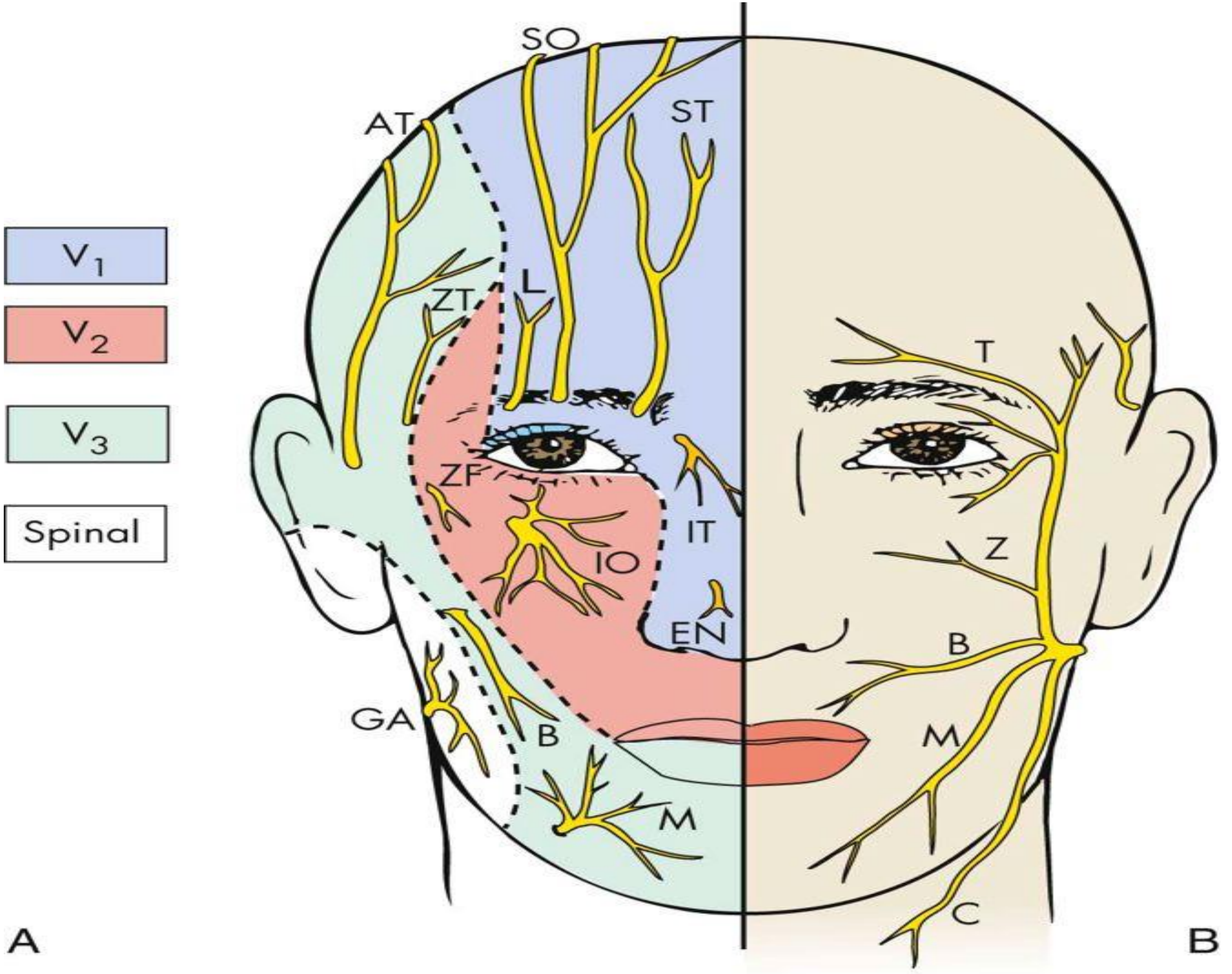
Inferior view of the brain showing cranial nerves and the
organs and tissues they innervate





Internal view of the base of the skull showing cranial nerves exiting or entering the skull.

171
172

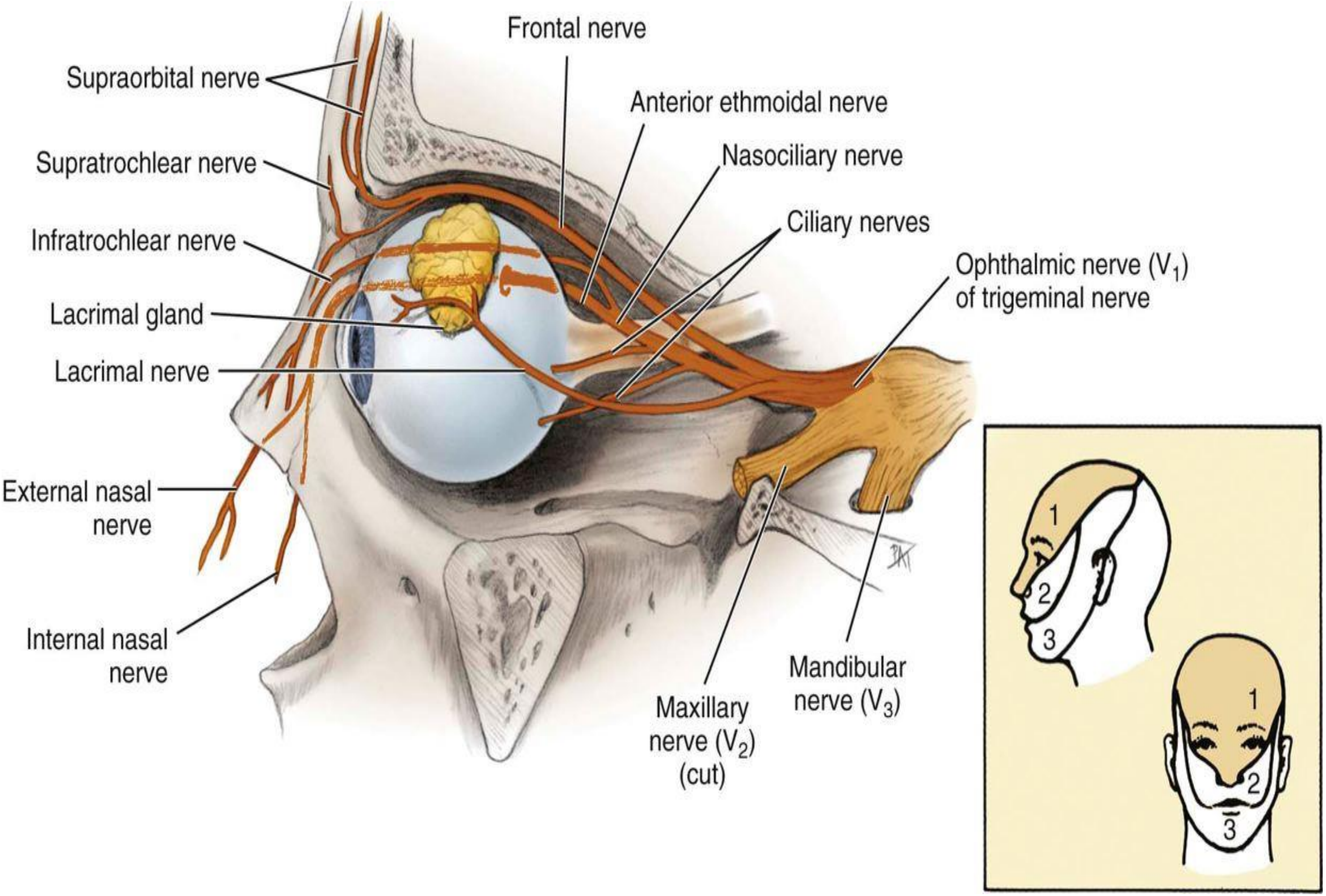


Cutaneous nerves of face.
V1 (ophthalmic nerve):
EN, External nasal nerve; *IT,* infratrochlear nerve; *L,* lacrimal nerve; *SO,* Supraorbital nerve; *ST,* supratrochlear nerve.

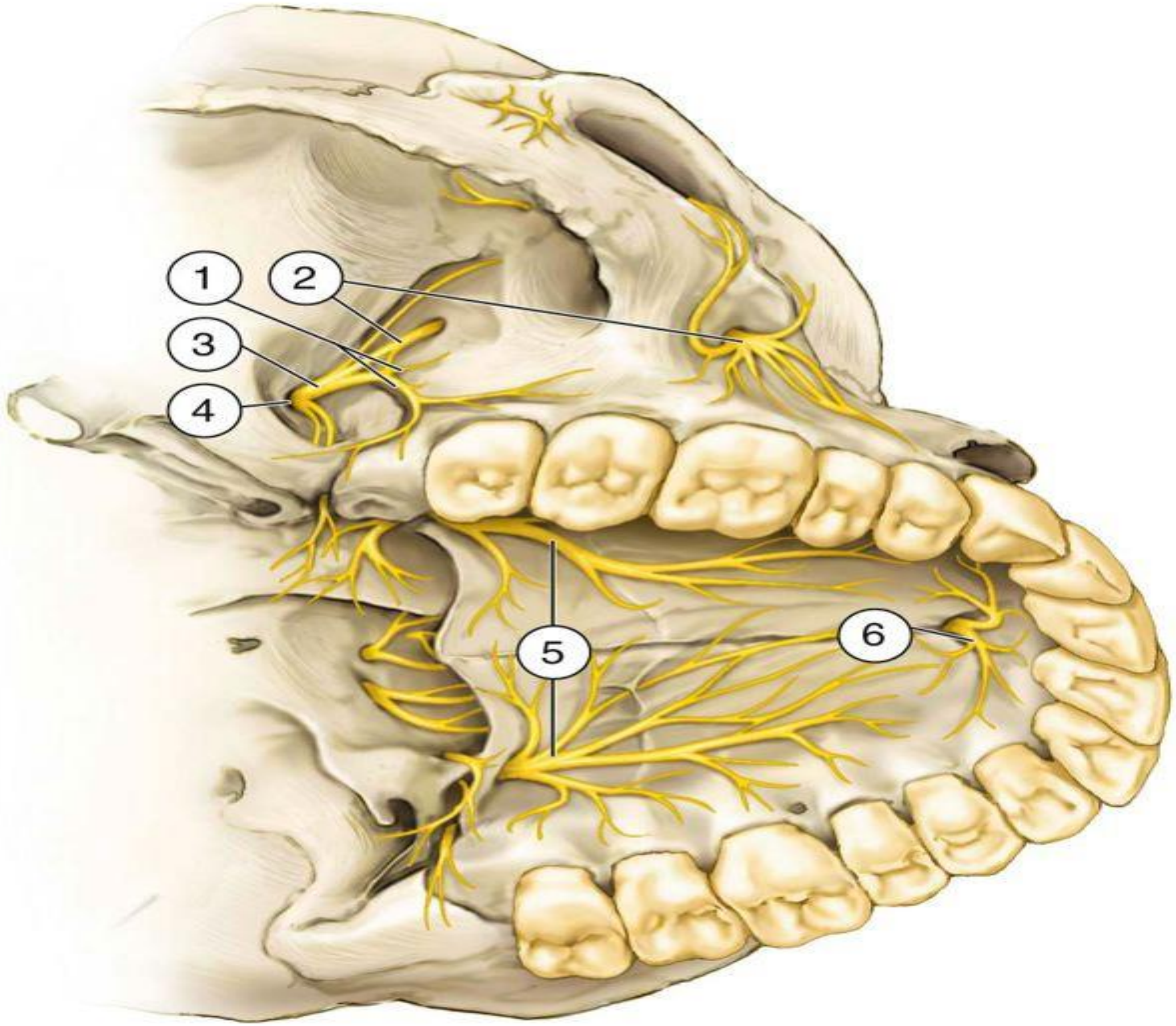
V2 (maxillary nerve): *IO,* Infraorbital nerve; *ZF,* zygomaticofacial nerve; *ZT,* zygomaticotemporal nerve.

V3 (mandibular nerve):
AT, Auriculotemporal nerve; *B,* buccal nerve; *M,* mental nerve.
Spinal nerve: *GA,* Great auricular nerve

V	Trigeminal	Mixed	
V_1	Ophthalmic	Sensory	V_1 Sensory from muscles of forehead; V_2 Sensory from lower eyelids, zygoma and upper lip; V_3 Sensory from lateral scalp, skin anterior to ears, lower cheeks, lower lips and anterior aspect of mandible; Motor to muscles of mastication (temporalis, masseter, medial and lateral pterygoids, tensor veli palatine and tensor tympani)
V_2	Maxillary	Sensory	
V_3	Mandibular	Sensory & motor	

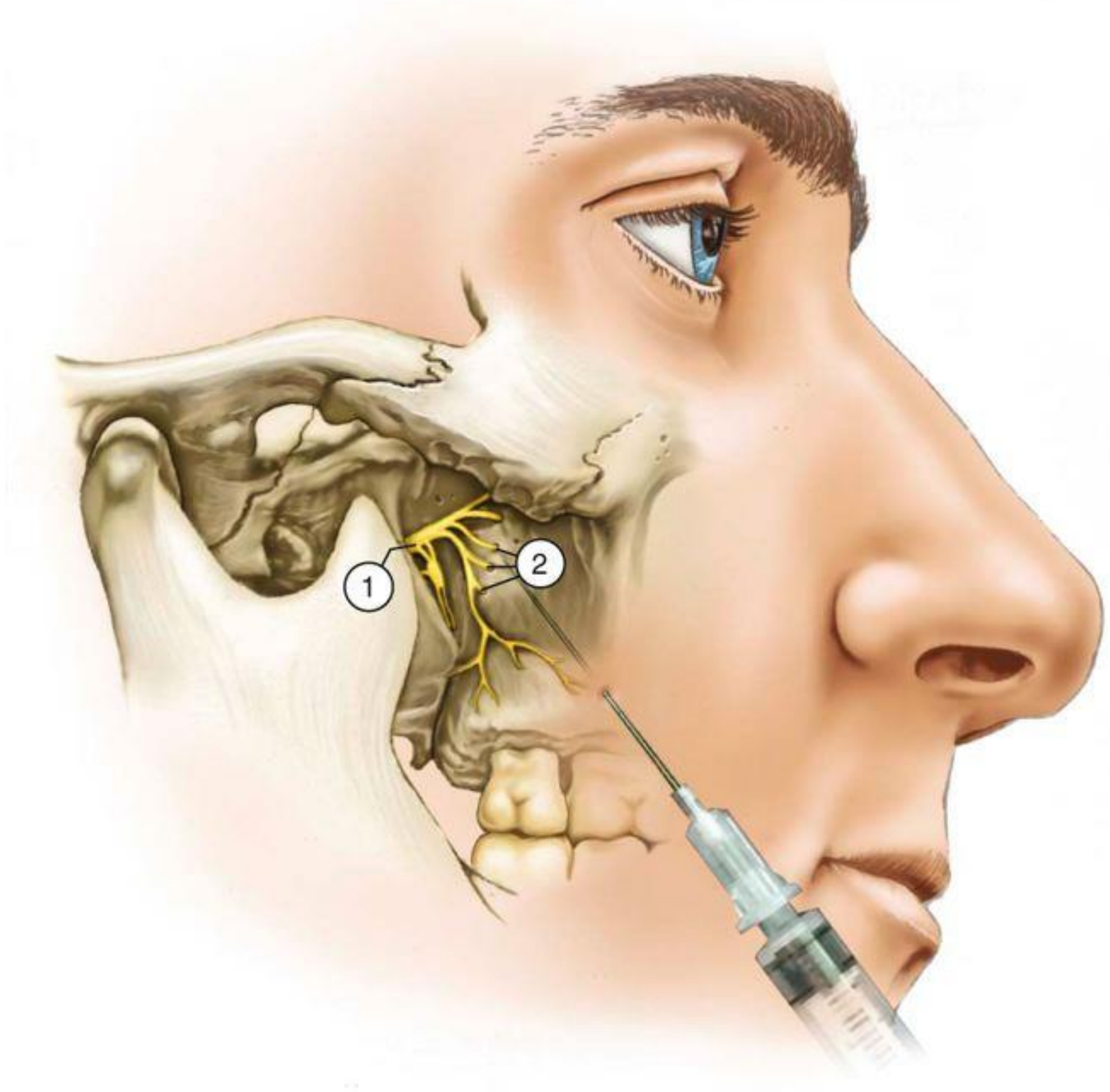


Frontal nerve
Supraorbital nerve
Anterior ethmoidal nerve
Supratrochlear nerve
Nasociliary nerve
Infratrochlear nerve
Ciliary nerves
Ophthalmic nerve (V_1) of trigeminal nerve
Lacrimal gland
Lacrimal nerve
External nasal nerve
Internal nasal nerve
Maxillary nerve (V_2) (cut)
Mandibular nerve (V_3)
1
2
3
1
2
3



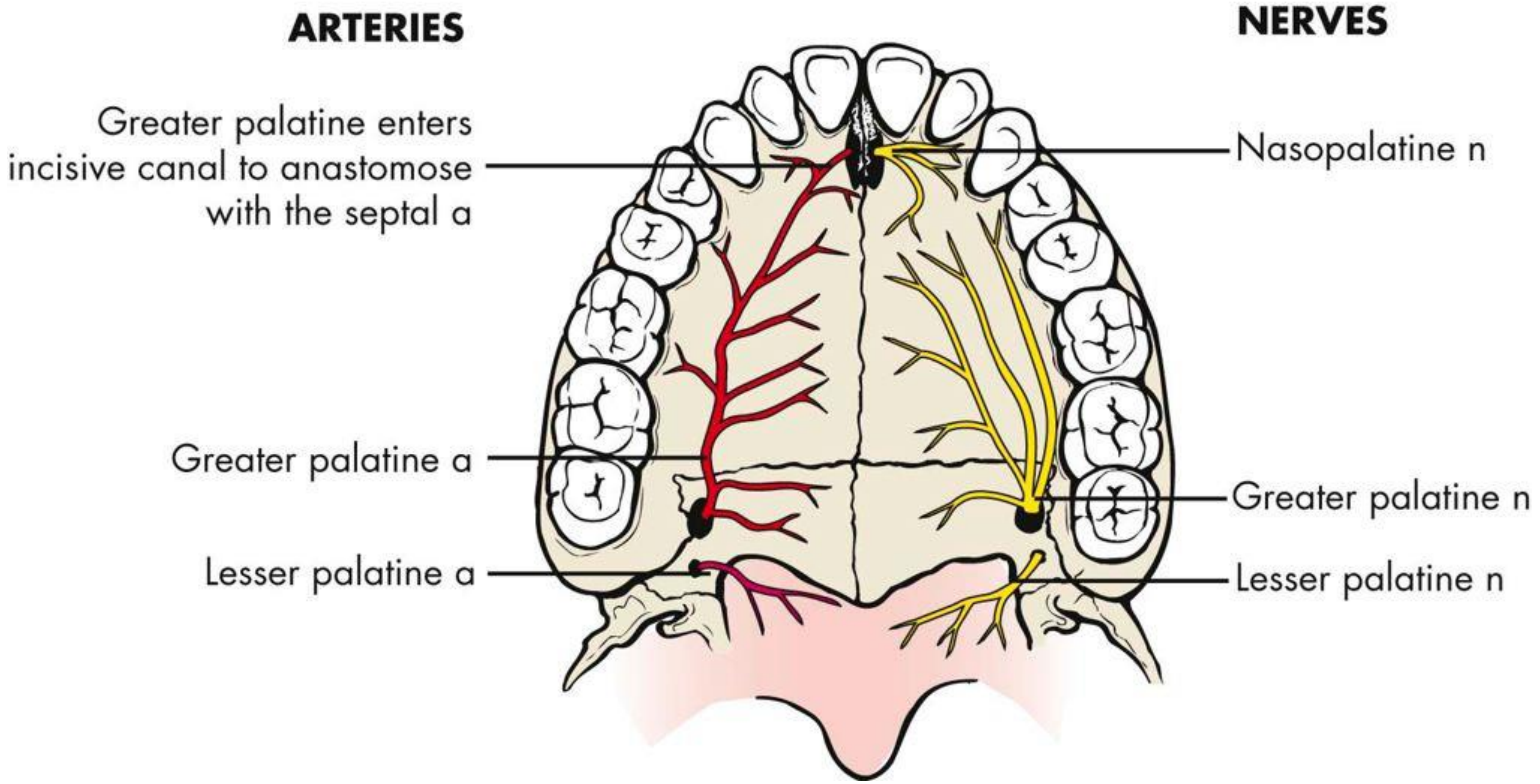
Distribution of the maxillary division (V_2).

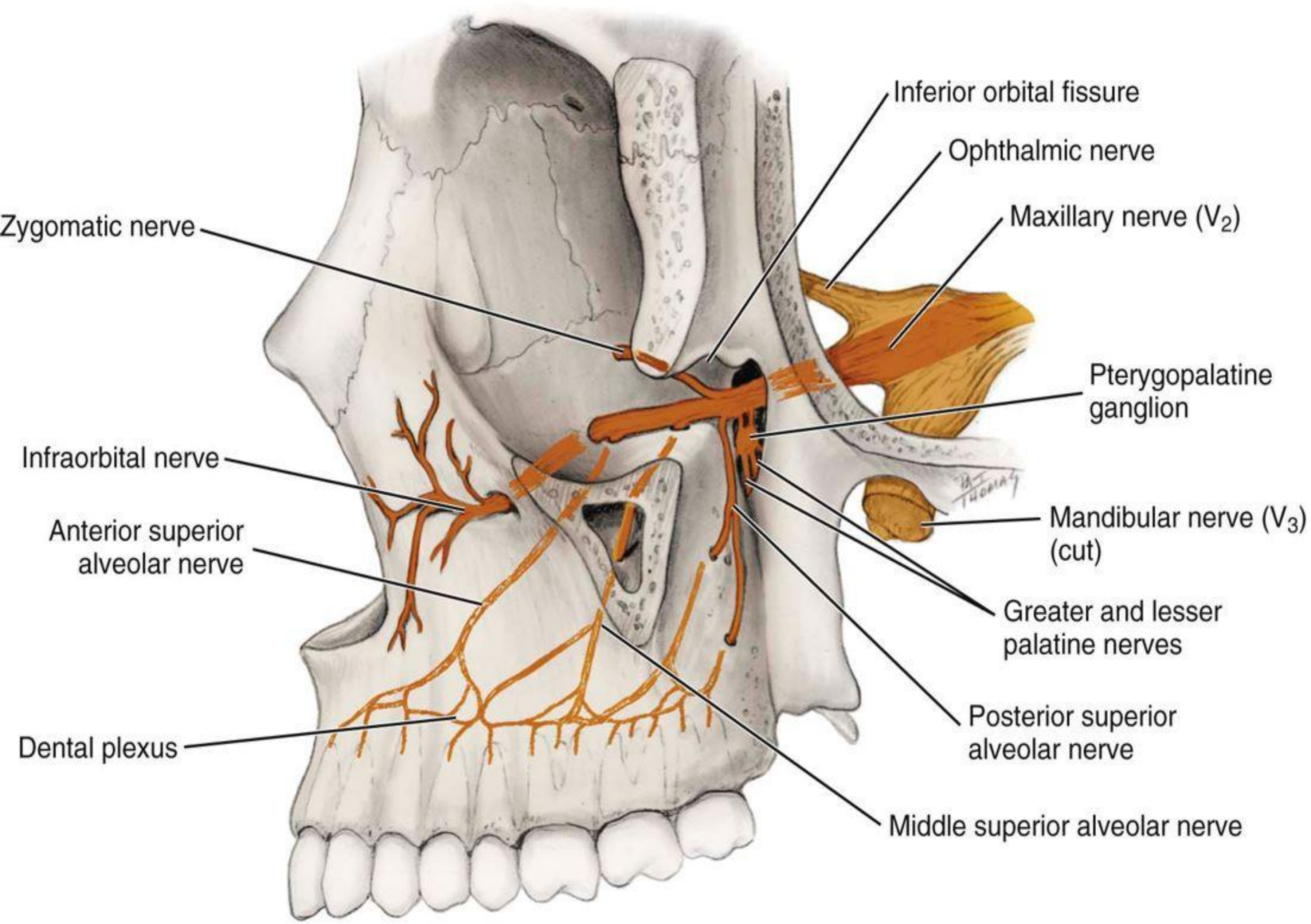
1, Posterior superior alveolar branches;
2, infraorbital nerve;
3, maxillary nerve;
4, foramen rotundum;
5, greater palatine nerve;
6, nasopalatine nerve.



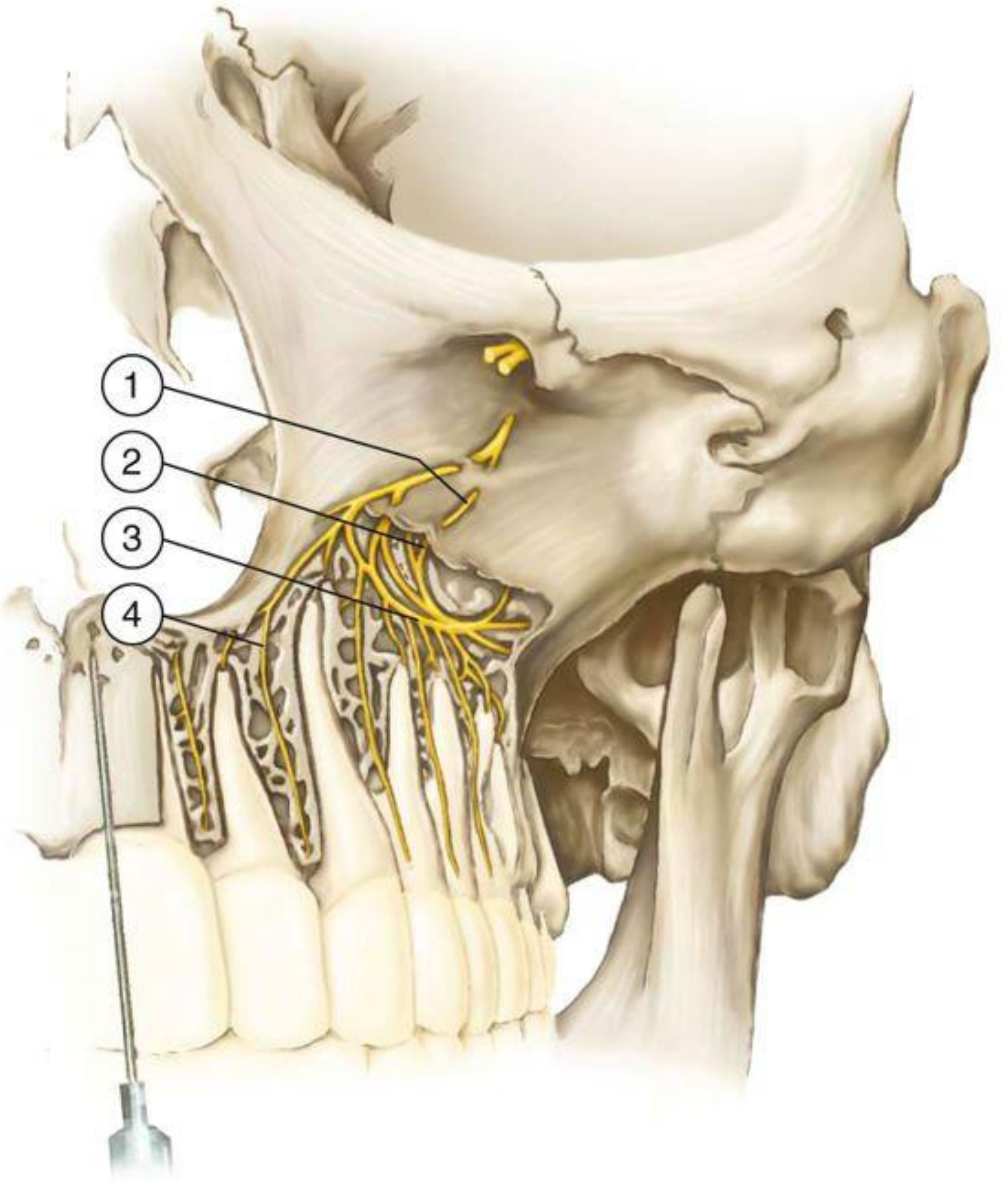
Branches of V2 in the pterygopalatine fossa

1, Maxillary nerve;
2, posterior superior alveolar branches.


ARTERIES
Greater palatine enters incisive canal to anastomose with the septal a
Greater palatine a
Lesser palatine a
NERVES
Nasopalatine n
Greater palatine n
Lesser palatine n

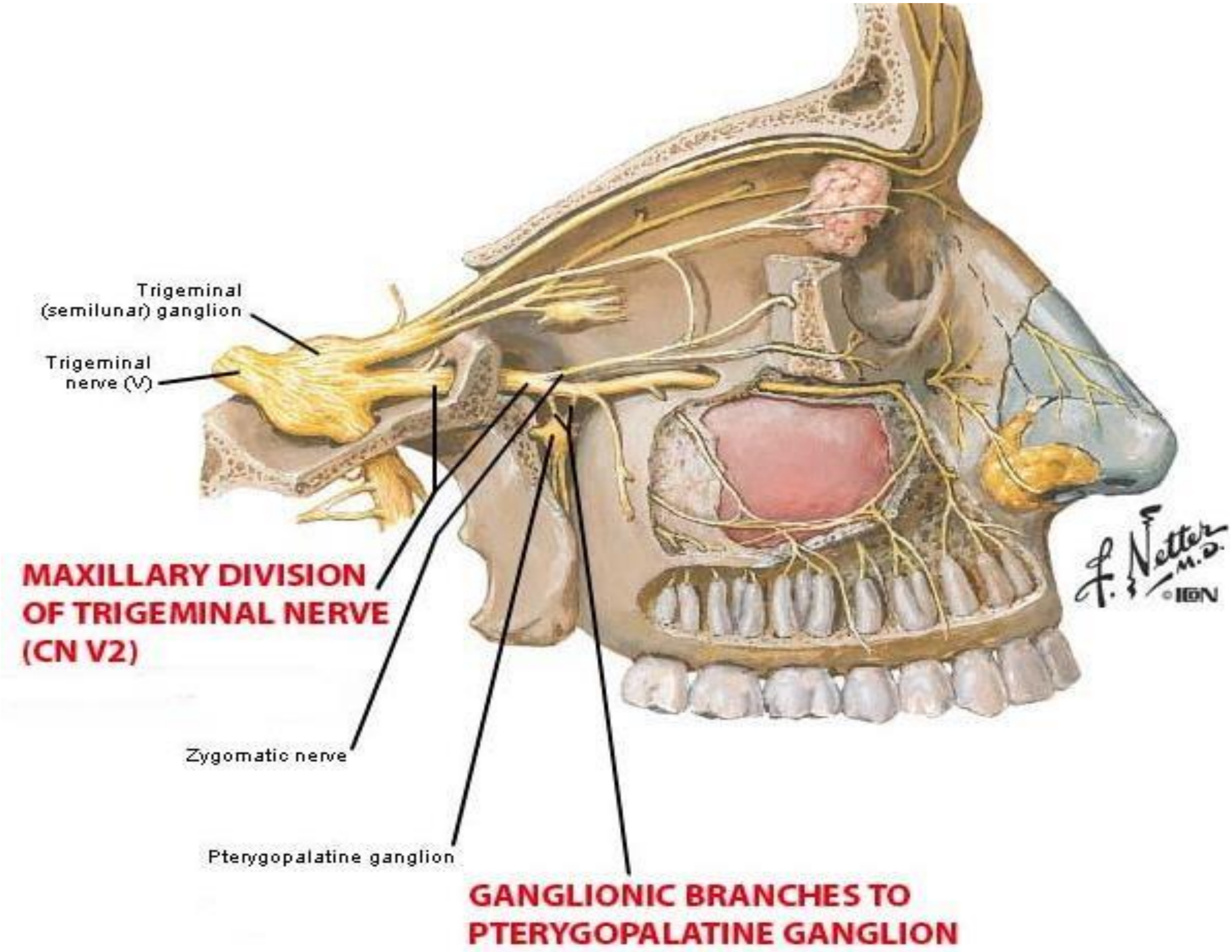


Lateral view of the skull-
(a portion of the lateral wall of the orbit has been removed) with the branches of the maxillary nerve highlighted



Anterior superior alveolar (ASA) nerve (bone over the nerves removed).

1, Branches of the ASA nerve;
2, superior dental plexus;
3, dental branches;
4, interdental and interradicular branches



Trigeminal (semilunar) ganglion
Trigeminal nerve (V)
MAXILLARY DIVISION OF TRIGEMINAL NERVE (CN V2)
Zygomatic nerve
Pterygopalatine ganglion
GANGLIONIC BRANCHES TO PTERYGOPALATINE GANGLION
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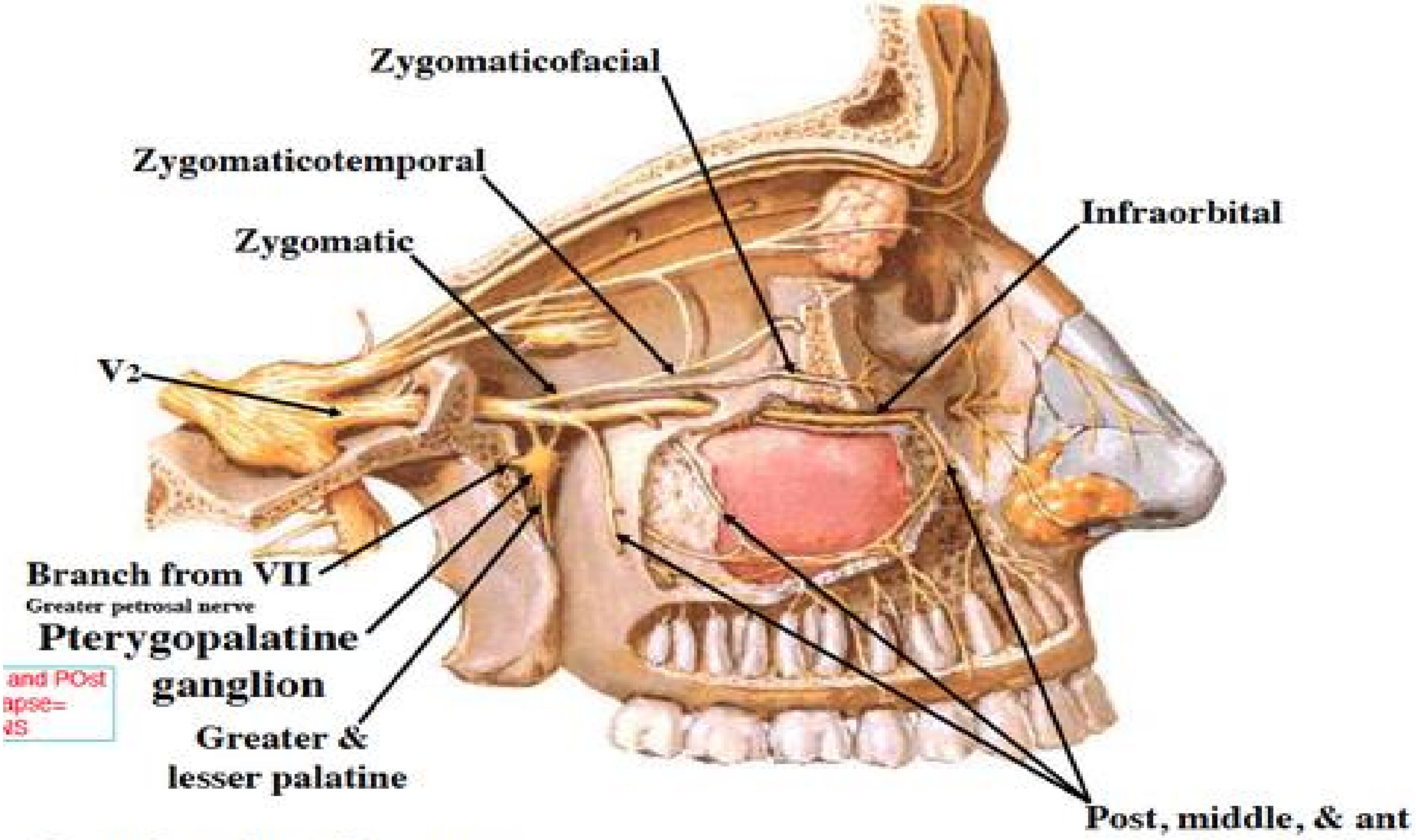
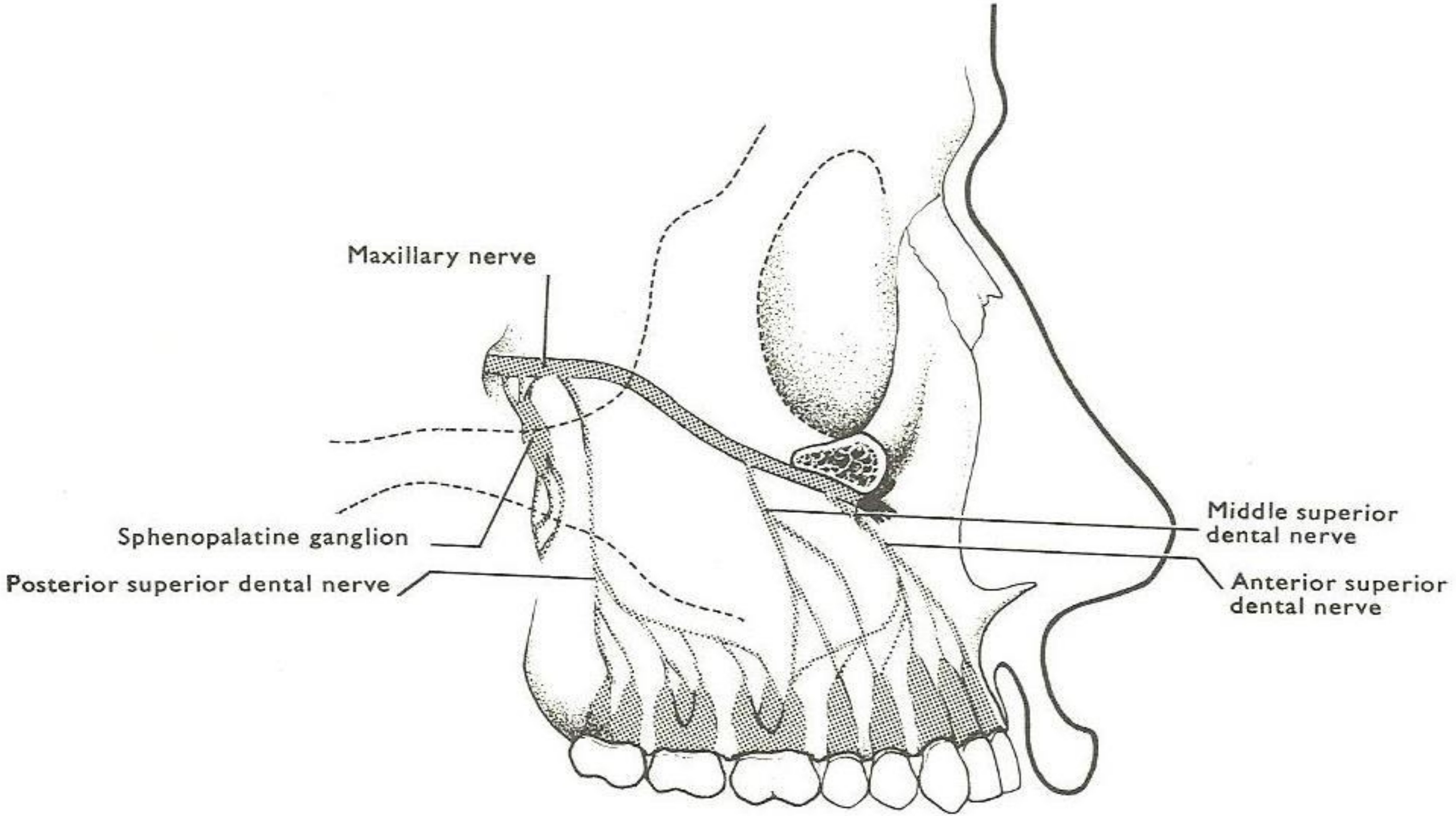
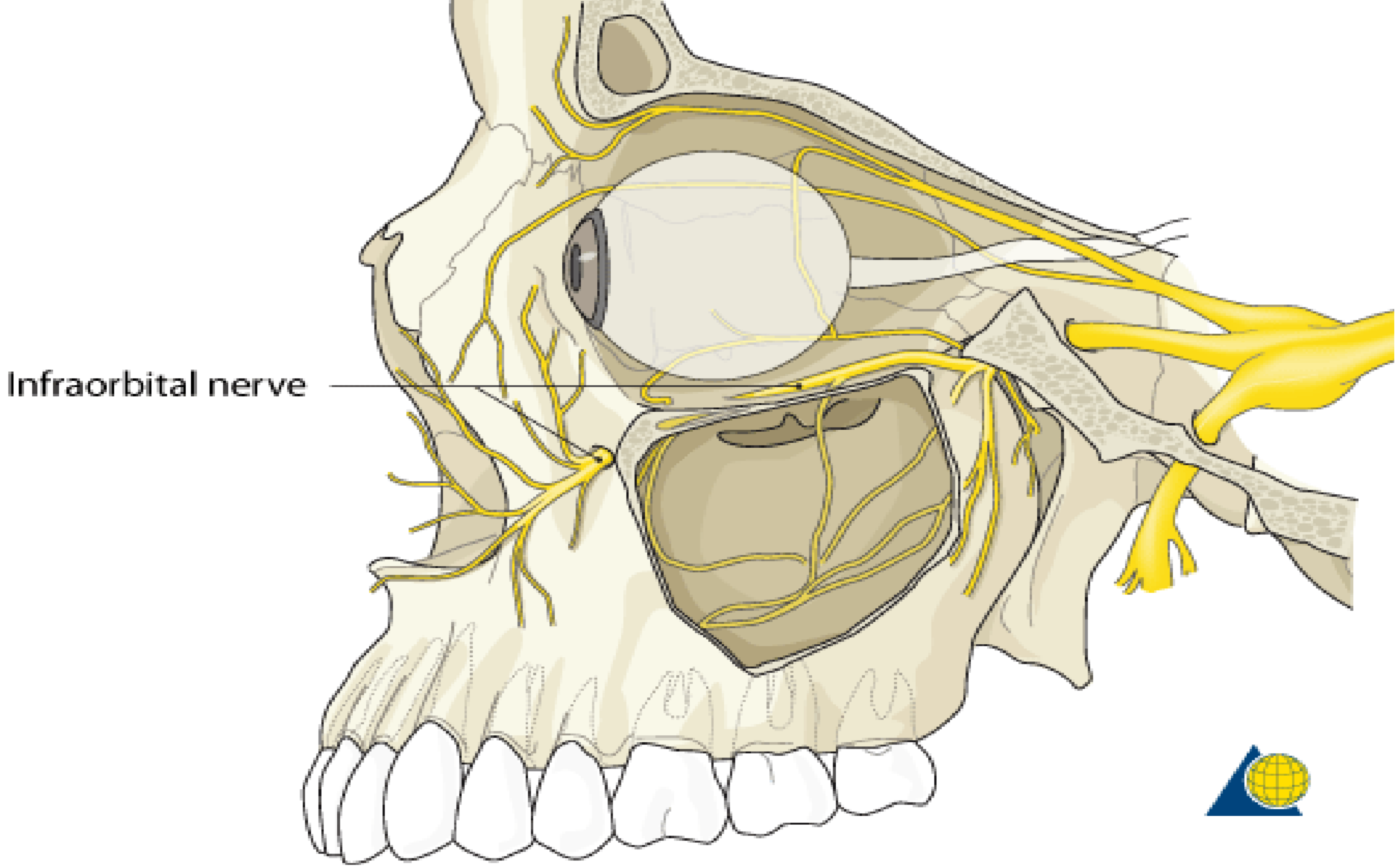


Branches of maxillary nerve



Maxillary nerve
Sphenopalatine ganglion
Posterior superior dental nerve
Middle superior dental nerve
Anterior superior dental nerve


Infraorbital nerve

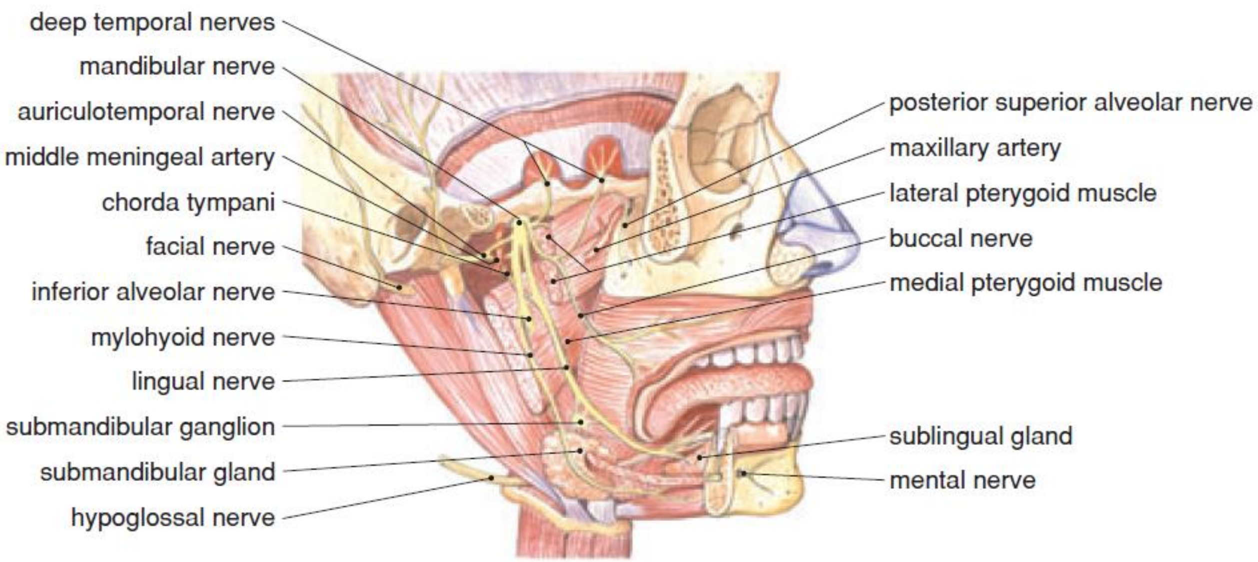
The mandibular nerve:

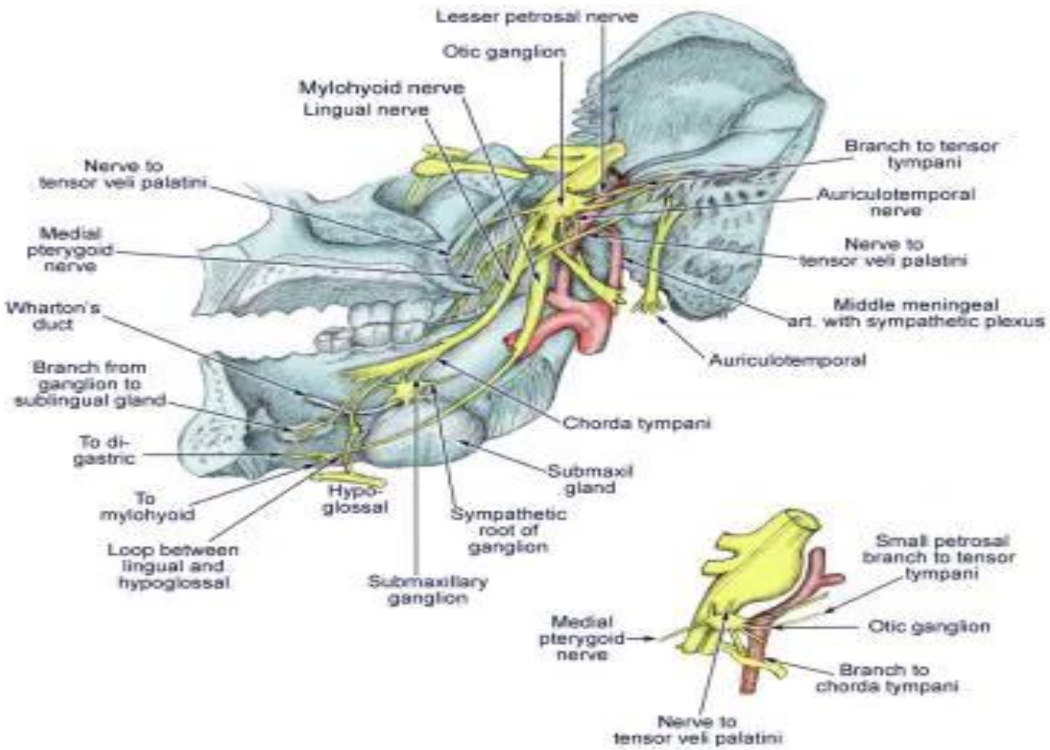
The mandibular nerve (n. V3) contains both **sensory** and **motor fibers**. **This nerve exits** the skull through the **foramen ovale** and ends in the **infratemporal fossa .**

The mandibular nerve runs between the **lateral pterygoid muscle** and the **tensor veli palatini muscle**. The nerve sends a motor branch to the **latter muscle**.

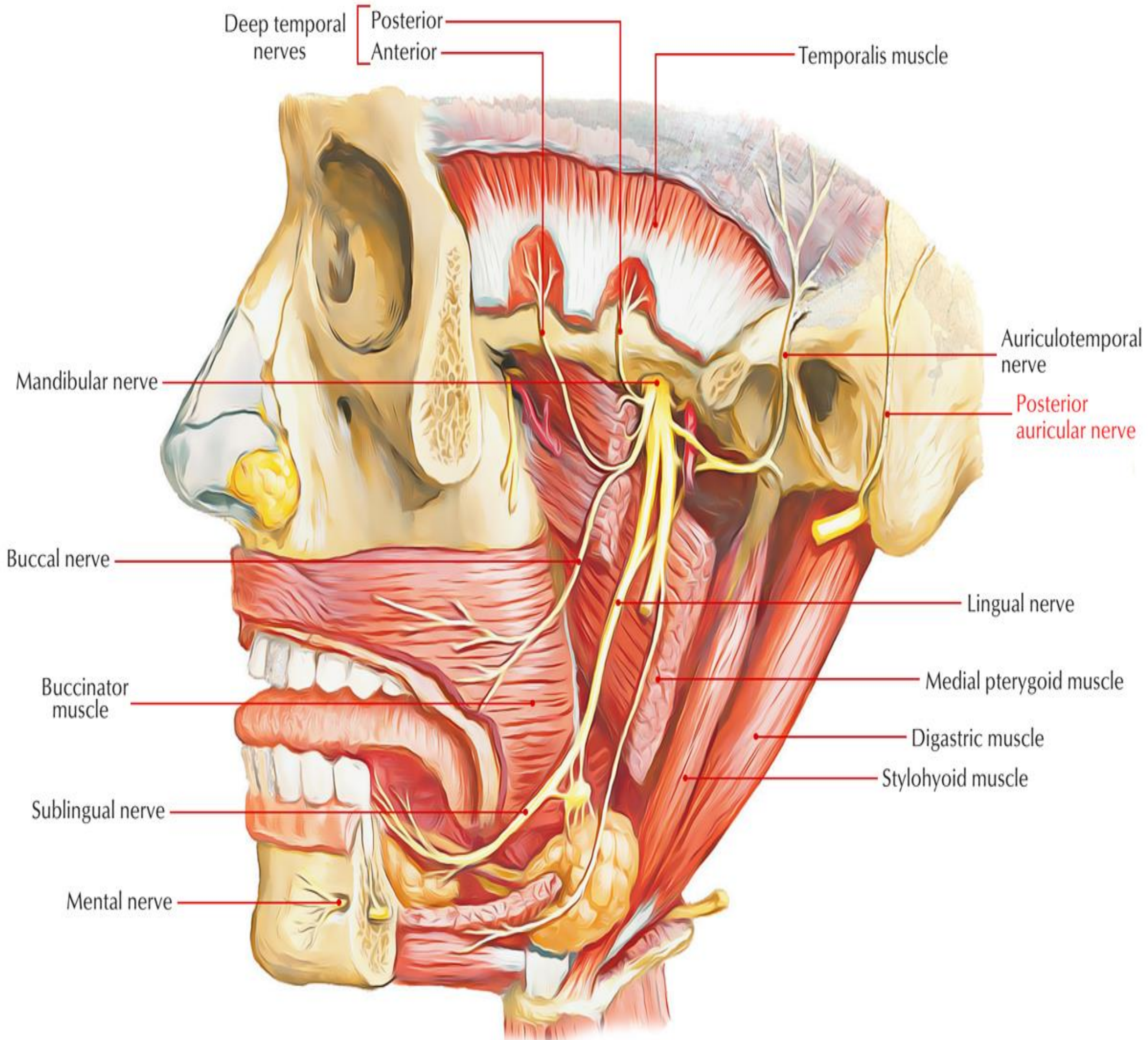
The mandibular **nerve splits into two main** branches, the **anterior** and **posterior trunks.**
From the **anterior trunk a sensory nerve emerges**, the buccal nerve, and a number of motor nerves, i.e. the **pterygoid nerves**, the **deep temporal nerves** and the **masseteric nerve**.

Three branches emerge from the **posterior trunk**: the **auriculotemporal nerve (sensory),** the **inferior alveolar nerve (mixed sensory and motor)** and the **lingual nerve(sensory).**





Motor branches of the trigeminal nerve are distributed in the mandibular nerve. These fibers originate in the motor nucleus of the fifth nerve, which is located near the main trigeminal nucleus in the pon.



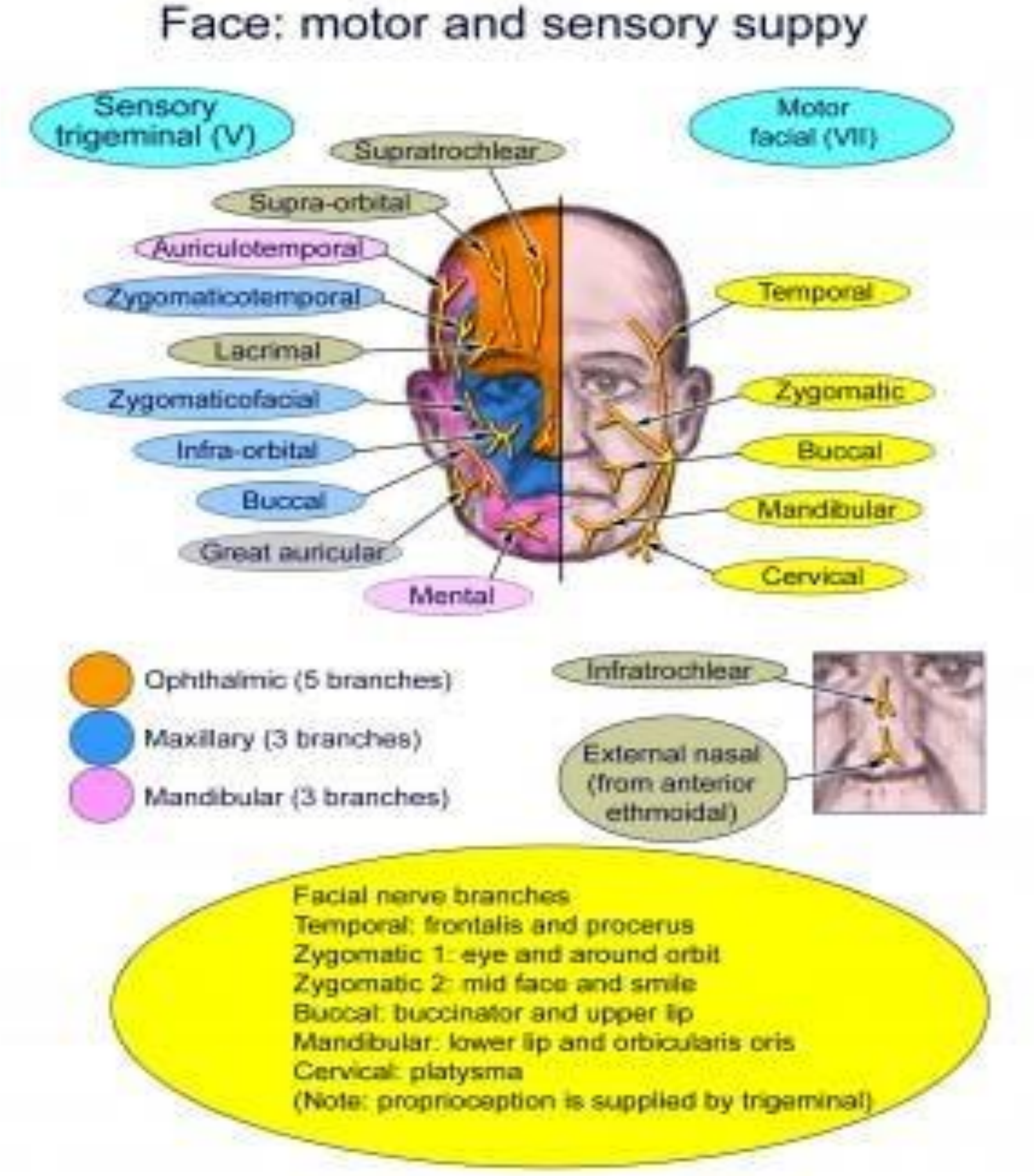


Diagram of the sensory and motor supply of the face.

The mandibular nerve has the following 9 branches:

- **Recurrent meningeal nerve -** This nerve enters the skull via the foramen spinosum with the meningeal artery
- **Medial pterygoid nerve** - After passing through the otic ganglion without synapsing, this nerve supplies the medial pterygoid, tensor veli palatini, and tensor tympani muscles
- **Masseteric nerve -** This nerve passes through the mandibular notch to innervate the masseter muscle and temporomandibular joint (TMJ)
- **Deep temporal nerves** - The anterior and posterior branches supply the temporal muscle
- Lateral pterygoid nerve
- **Buccal nerve -** This nerve divides into the temporal and buccinator branches
- **Auriculotemporal nerve -** This nerve begins as **2 roots** that encircle the middle meningeal artery, then forms a **single trunk medial to the neck of the mandible; it emerges superficially between the ear and the mandibular condyle** deep to the parotid gland and ends in **2 superficial temporal branches** (for autonomic supply to the parotid gland,
- **Lingual nerve** - This nerve runs parallel to the inferior alveolar nerve, is joined by the chorda tympani nerve of the facial nerve (CN VII) near the internal maxillary artery, courses forward between the **hyoglossus muscle** and **the deep part of the submandibular gland**, and, as it passes forward, crosses the submandibular (Wharton) duct; the lingual nerve could be injured in this location during surgery on the floor of mouth or during excision of the submandibular gland (for more details regarding the nerve supply of the salivary glands, see below)
- **Inferior alveolar nerve -** This nerve accompanies the inferior alveolar artery in the mandibular foramen and courses into the mandibular canal to exit through the mental foramen

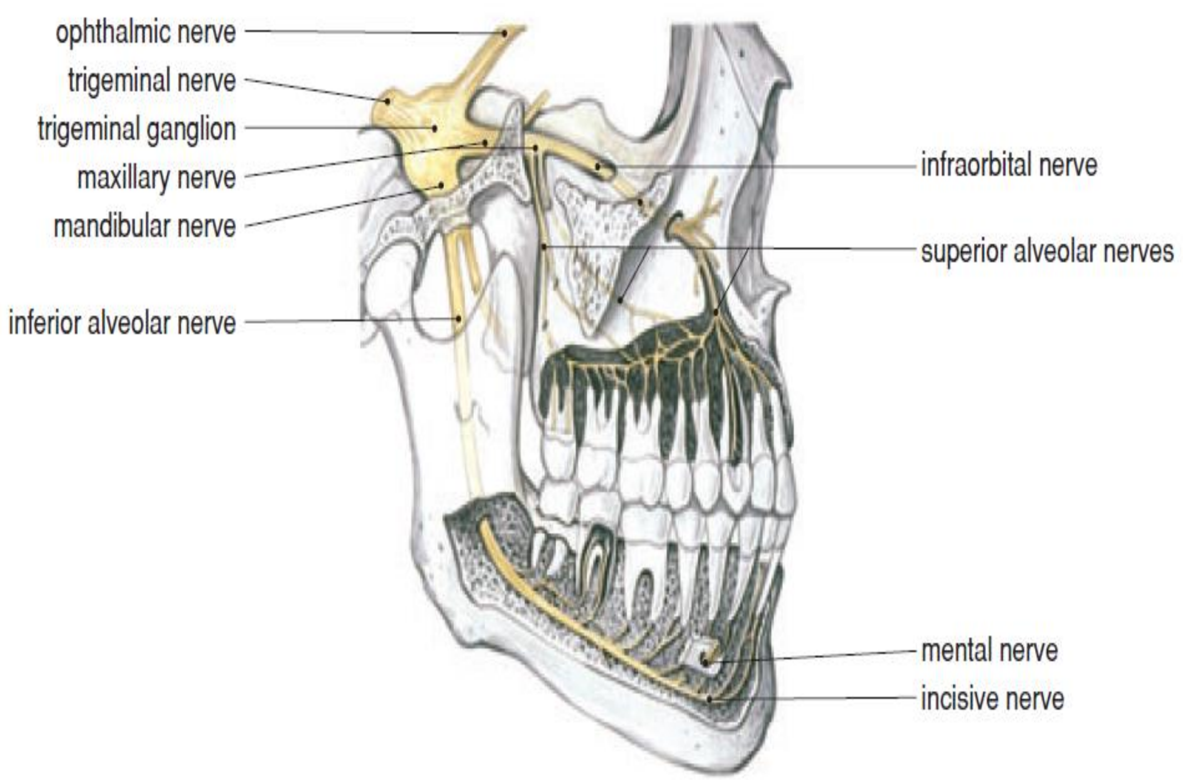
Motor Supply:

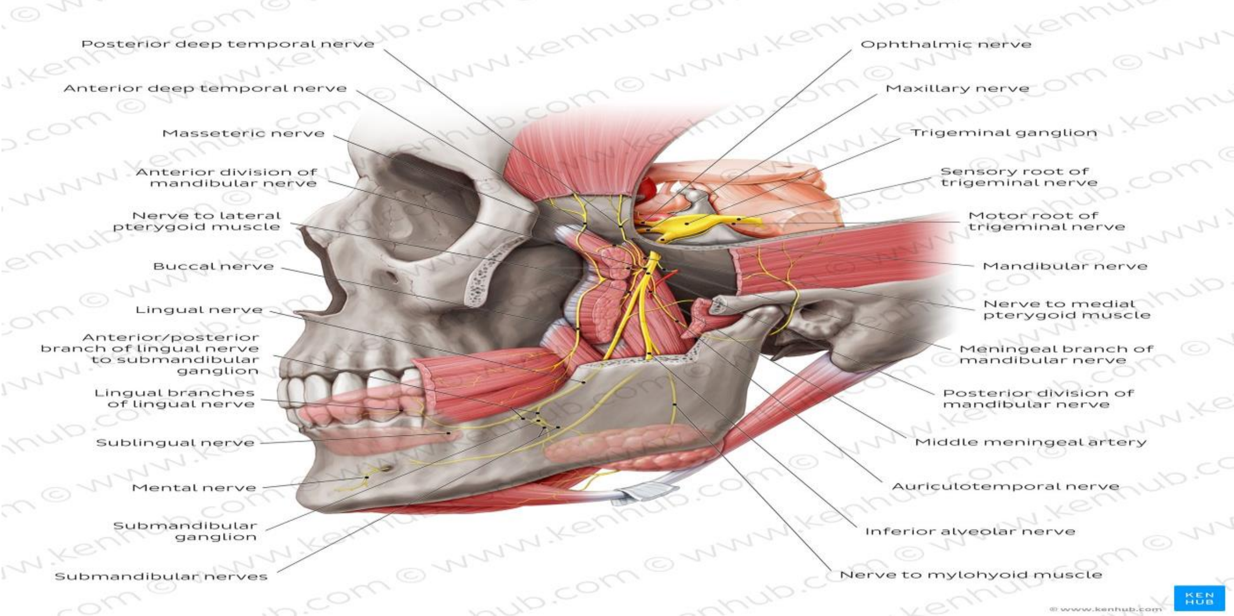
Muscles of mastication; medial pterygoid, lateral pterygoid, masseter, temporalis
Anterior belly of the digastric muscle and the mylohyoid muscle (these are suprahyoid muscles)**Tensor veli palatini ,Tensor tympani**

Parasympathetic Supply:

•**Submandibular and Sublingual glands:** Post-ganglionic fibers from the submandibular ganglion (derived from the facial nerve), travel with the lingual nerve to innervate these glands.

•**Parotid gland:** Post-ganglionic fibers from the otic ganglion (derived from the glossopharyngeal nerve, CN IX), travel with the auriculotemporal branch of the V3 to innervate the parotid gland





Tongue nerves :

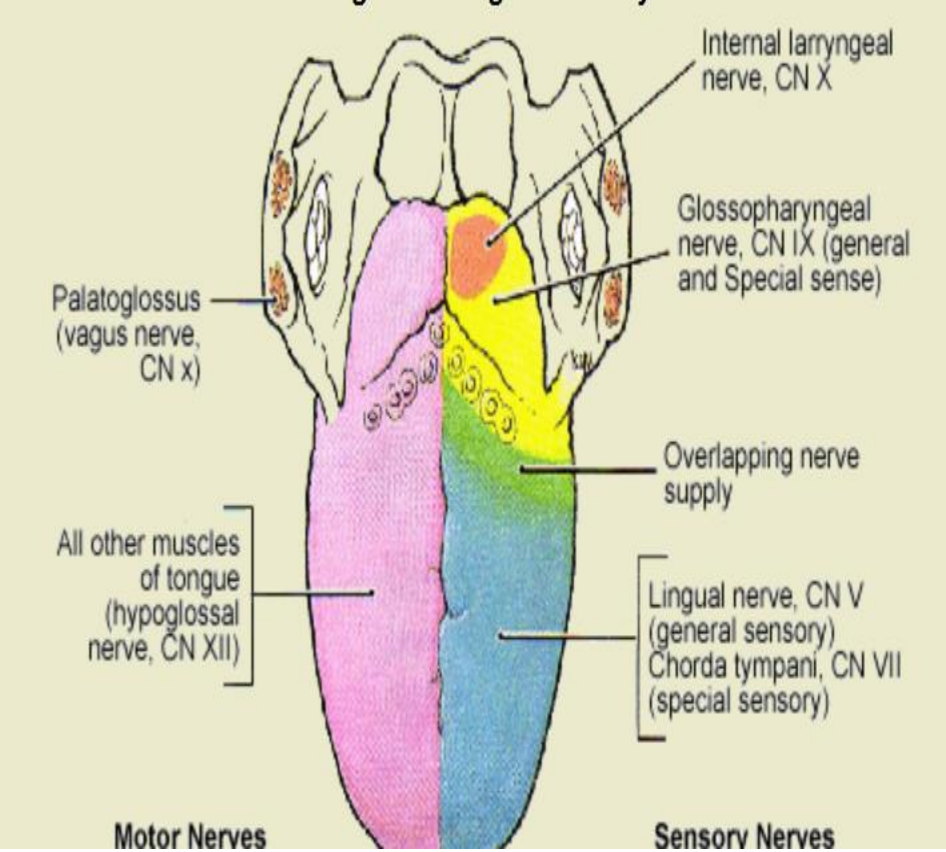
There is a lingual nerve for the right side of the tongue and one for the left side. The lingual nerve also carries a branch of the facial nerve called the chorda tympani which splits off the lingual nerve before the tongue is innervated and provides the sensation of taste to the anterior (front) two-thirds of the tongue. The mandibular nerve (V3) is the largest of the three branches of the trigeminal nerve.

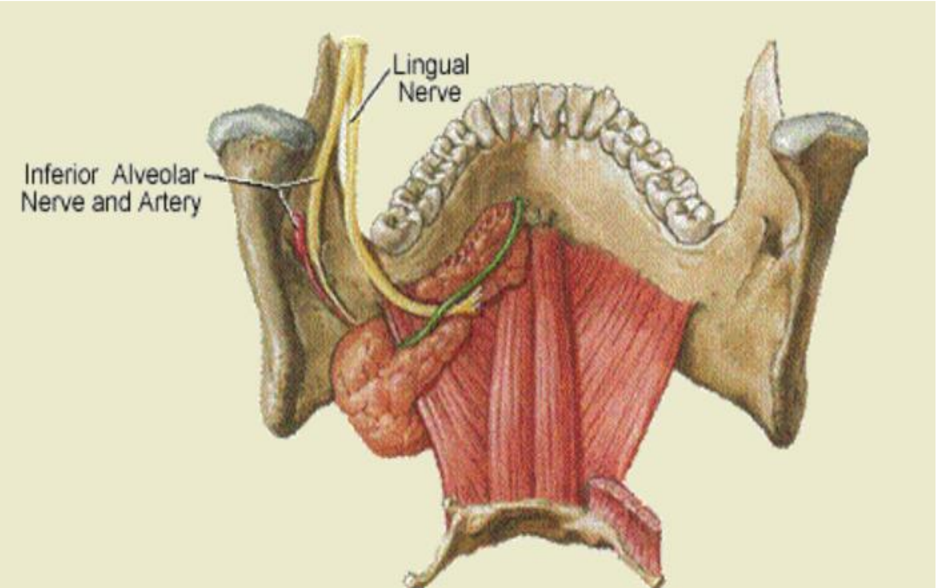
We are interested in the posterior division of the mandibular nerve which separates into the **lingual nerve** and the **inferior alveolar nerve**

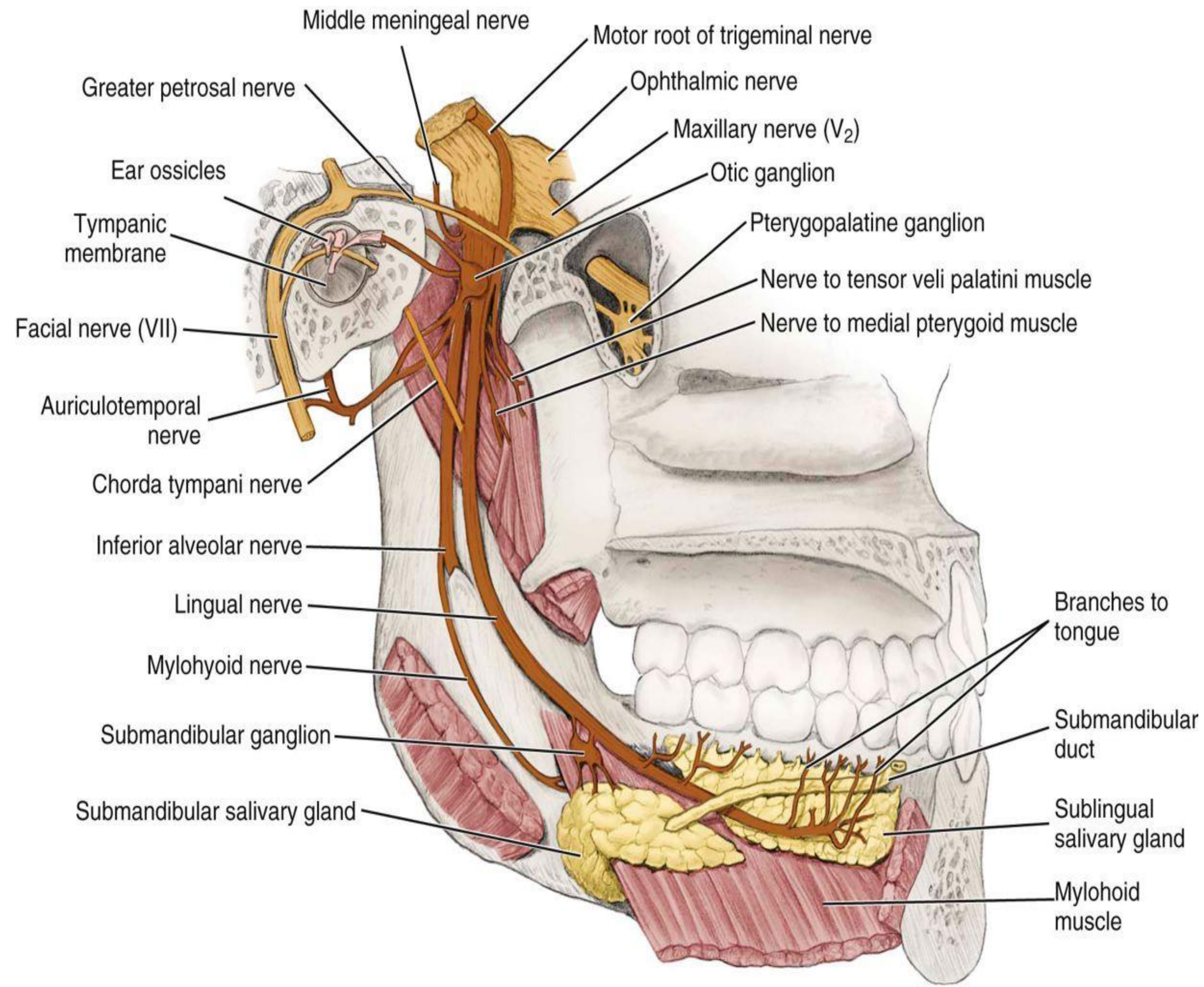
The lingual nerve runs to the medial (inside of the mandible or lower jaw) and next to the inferior alveolar nerve as they move towards the [ramus of the mandible]

The lingual nerve takes the inside track next to the mandible and runs next to the third molar (wisdom tooth on the lingual side (tongue side) of the third molar

There is a boney barrier between the third molar and the lingual nerve called the lingual plate which the lingual nerve runs along on the lingual side (tongue side).





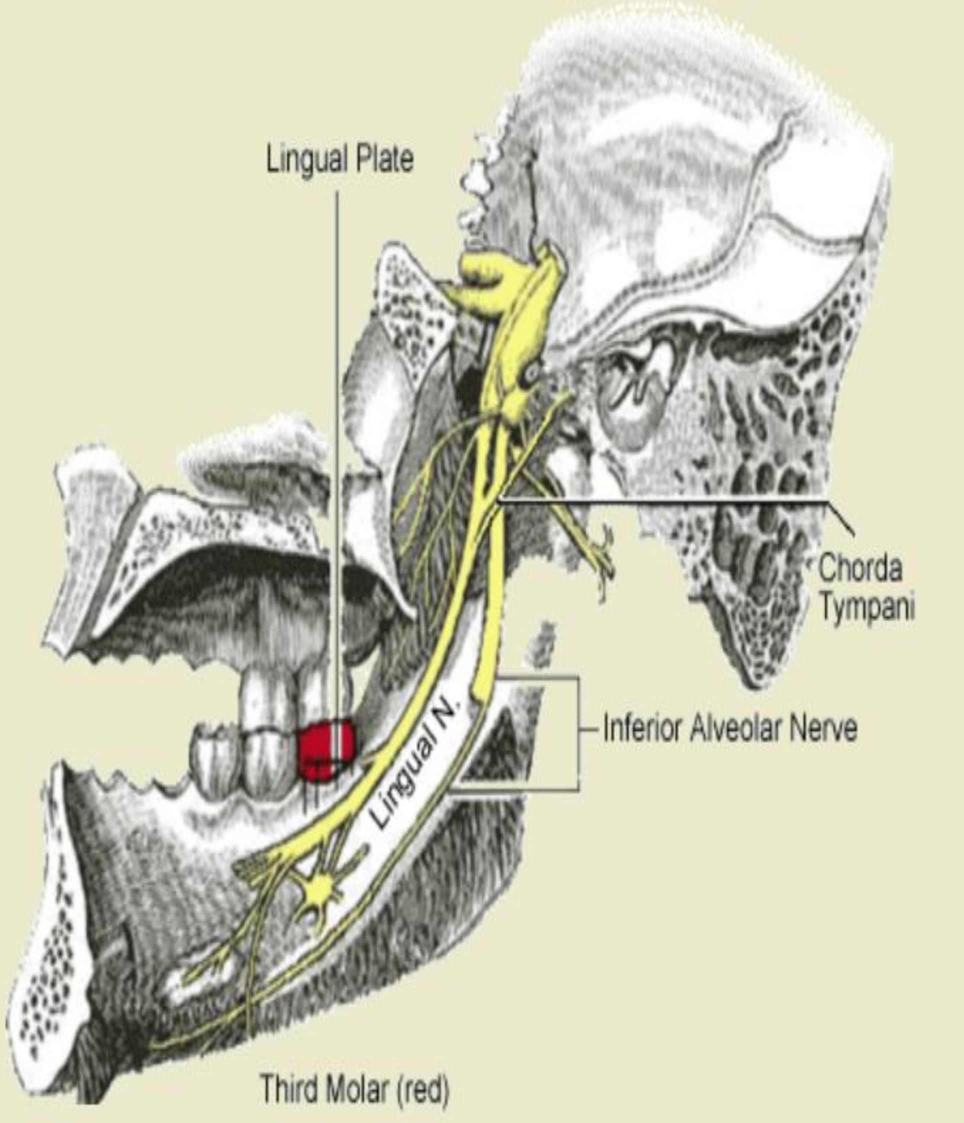

Middle meningeal nerve
Motor root of trigeminal nerve
Greater petrosal nerve
Ophthalmic nerve
Maxillary nerve (V_2)
Ear ossicles
Otic ganglion
Tympanic membrane
Pterygopalatine ganglion
Nerve to tensor veli palatini muscle
Facial nerve (VII)
Nerve to medial pterygoid muscle
Auriculotemporal nerve
Chorda tympani nerve
Inferior alveolar nerve
Lingual nerve
Branches to tongue
Mylohyoid nerve
Submandibular duct
Submandibular ganglion
Submandibular salivary gland
Sublingual salivary gland
Mylohoid muscle

After passing the third molar, the lingual nerve runs **below tongue**, sending nerve branches up to the **anterior (front) two thirds of the tongue** above which demonstrates the area of the tongue which the lingual nerve innervates in blue).

The lingual nerve also carries nerve fibers that are not part of the trigeminal nerve, including the chorda tympani nerve (a branch of the facial nerve or cranial nerve VII), which provides special sensation (taste) to the anterior 2/3 part of the tongue. It is also important to keep in mind the inferior alveolar bundle in relationship to the third molar which can also be injured during the third molar extraction .

A true close relationship between the third molars and the mandibular canal increases the risk of injury to the inferior alveolar nerve, and an accurate evaluation of the relationship is essential to avoid the risk of surgery.

Dentists should be aware of the limitations of the radiographic markers of panoramic radiography and should consider the more detailed imaging of CBCT in specific cases in which the radiographic findings raise concerns of potential injury to the inferior alveolar nerve due to the close relationship between the third molar and the mandibular canal



Lingual nerve in relation to the lingual plate and third molar.

Mandibular branch divided into:

a. anterior division

i. nerve to the lateral pterygoid muscle

ii. nerve to the masseter muscle

iii. nerve to the temporal muscle

iv. *buccal nerve*

b. posterior division

i. auriculotemporal nerve

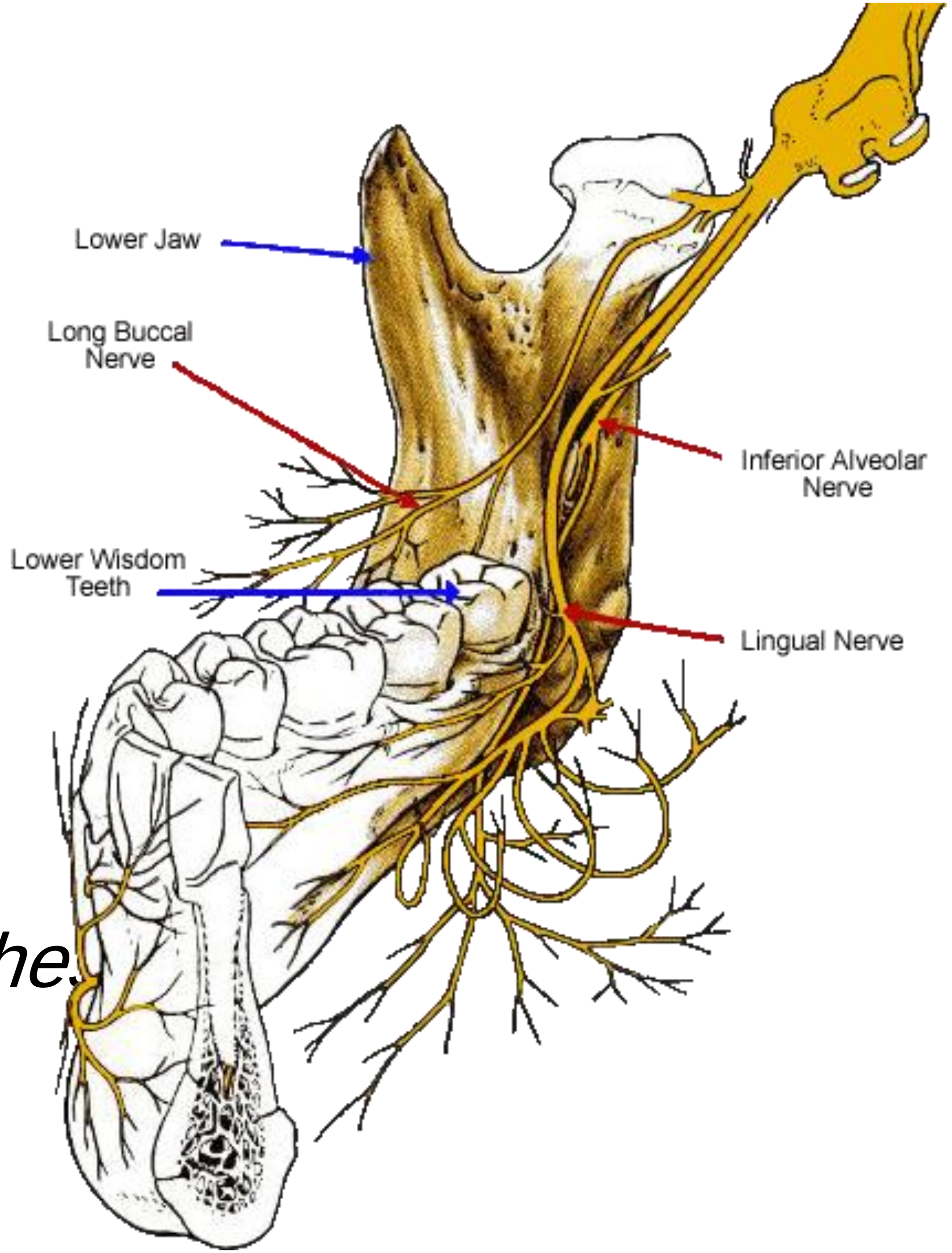
ii. *lingual nerve*

iii. *mylohyoid nerve*

iv. *inferior alveolar nerve: dental branches*

v. *incisive branch: dental branches*

vi. *mental nerve*



Nerves supplying the tongue:

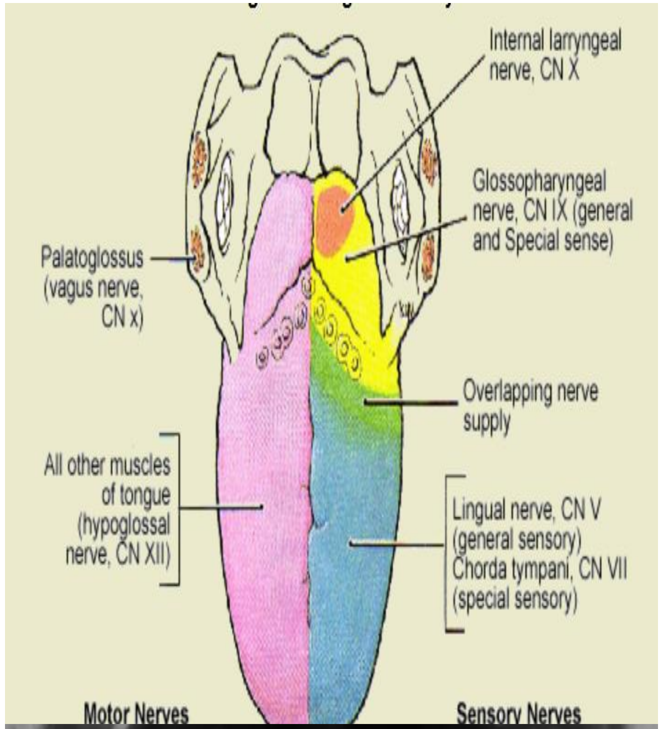
Fibe cranial nerve are innervated the tongue, normal sensation, special sensation, and motor

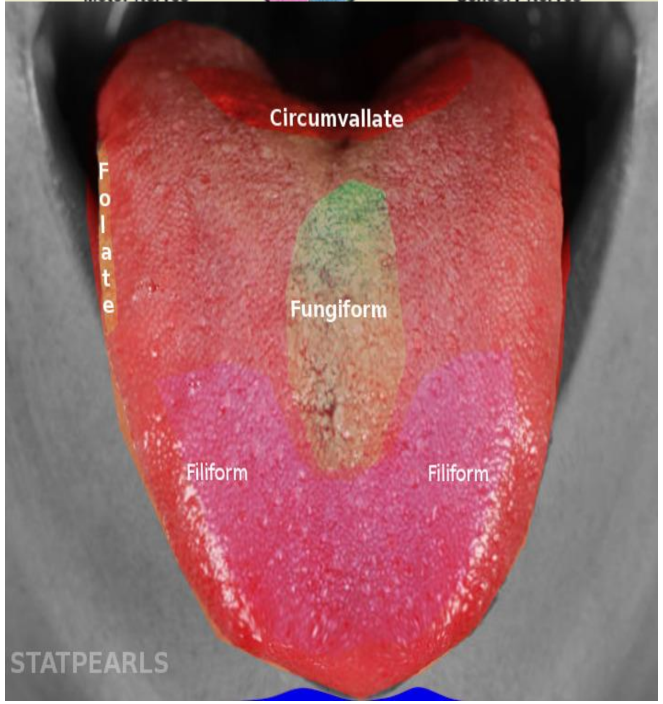
The **neural taste pathway will** undergo scrutiny from the perspective of starting within the tongue and moving away from it towards the brain.

The three nerves associated **with taste** are the **facial nerve (cranial nerve vii)**, which provides fibers to the anterior two-thirds of the tongue; **the glossopharyngeal nerve (cranial nerve ix), which provides fibers to the posterior third of the tongue**; and **the vagus nerve (cranial nerve x), which provides fibers to the epiglottis region.**

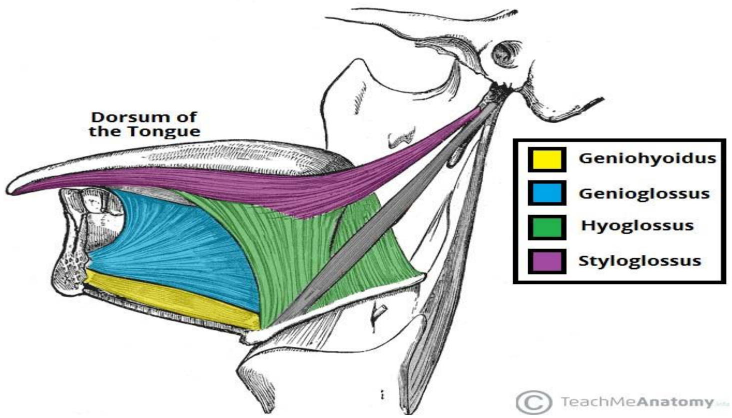
taste fibers categorize as special visceral afferent (sva)

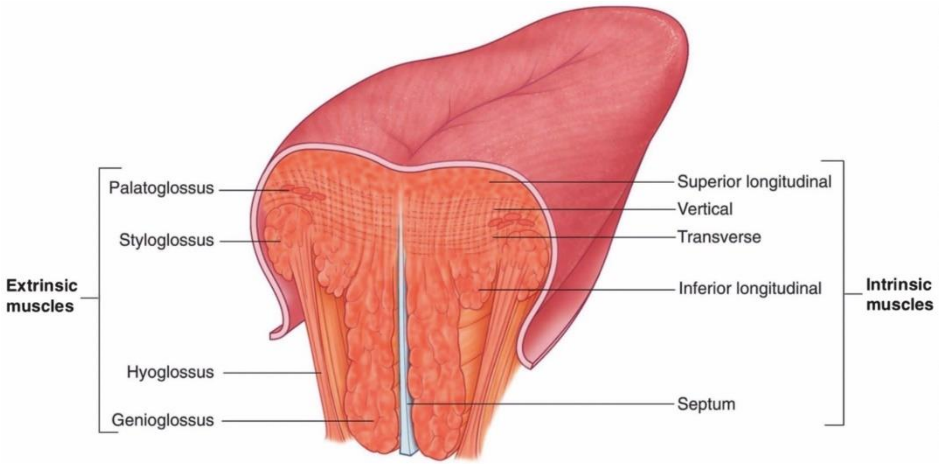
The normal sensation , anterior two thirs lingual nerve posterior two thirs glossopharngeal nerve

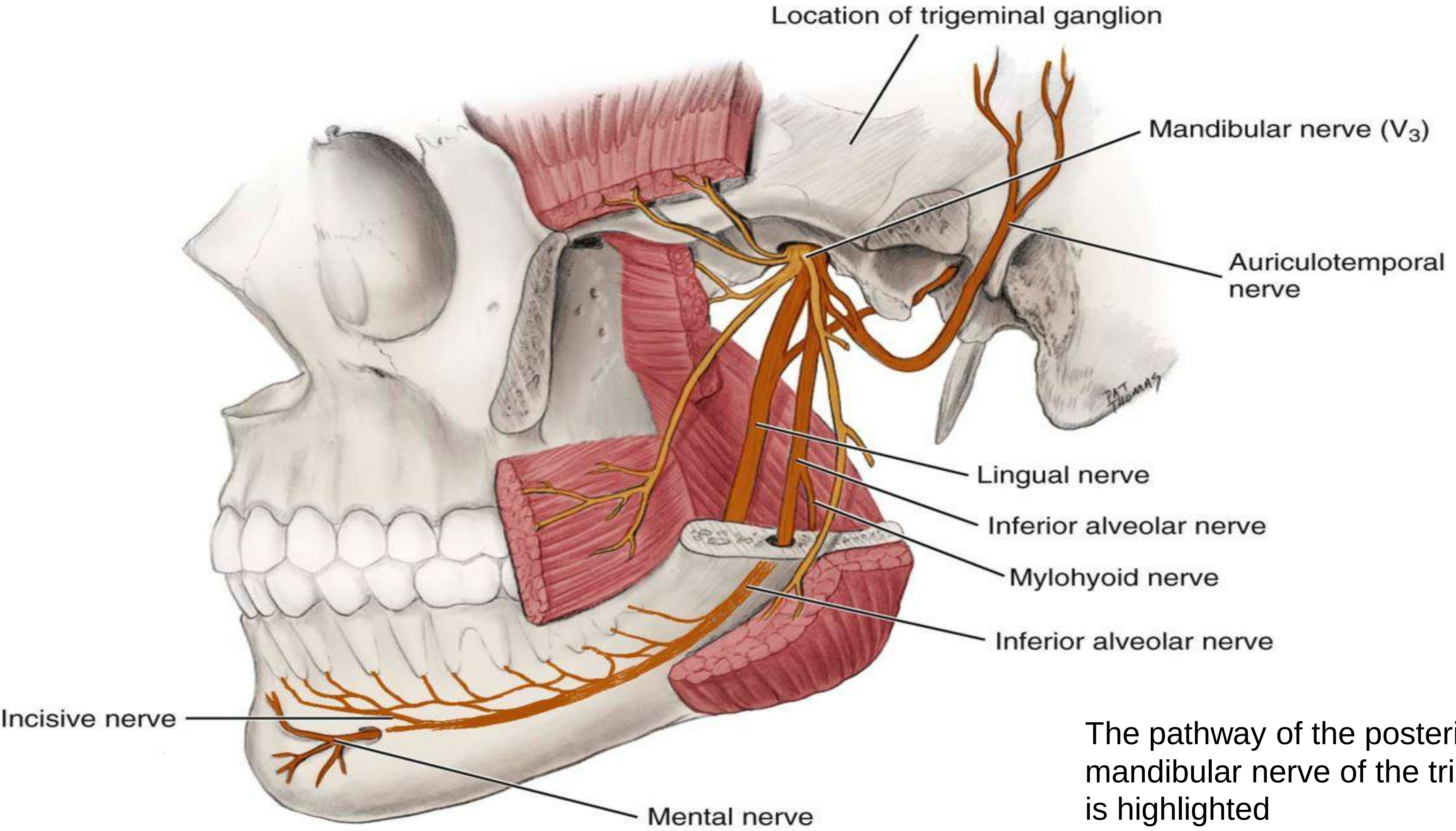




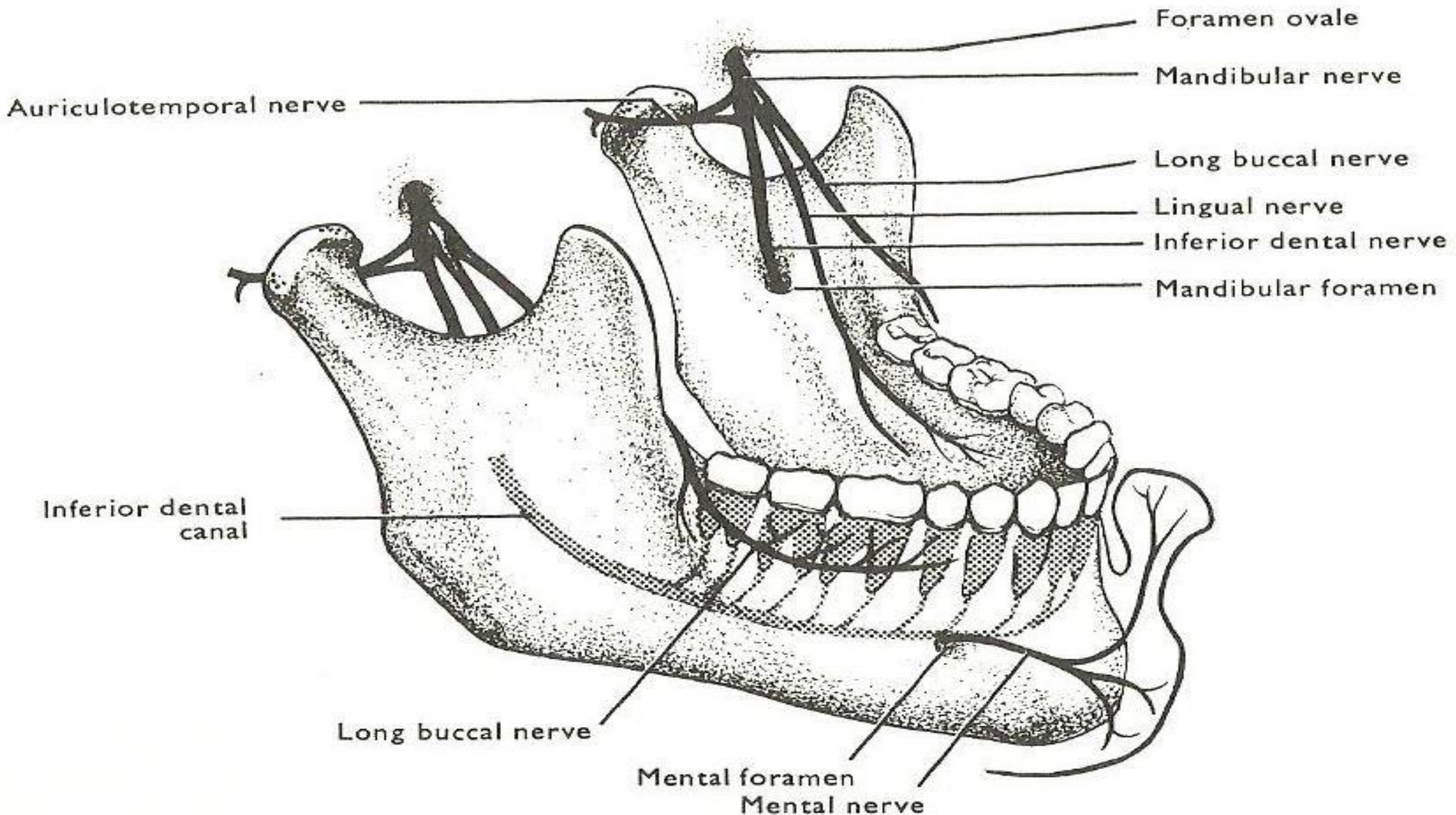
Extrinsic and intrinsic muscle of the tongue, hypoglossal nerve







The pathway of the posterior trunk of the mandibular nerve of the trigeminal nerve is highlighted



Foramen ovale
Mandibular nerve
Auriculotemporal nerve
Long buccal nerve
Lingual nerve
Inferior dental nerve
Mandibular foramen
Inferior dental canal
Long buccal nerve
Mental foramen
Mental nerve

Osteology: Maxilla:

In addition to the neuroanatomy of pain control in dentistry,one should be aware of the relationship of these nerves to the osseous and soft tissues through which they course. The maxilla (more properly, the right and left maxillae) is the largest bone of the face, excluding the mandible. Its
anterior (or facial) surface is directed both forwardand laterally.

At its inferior borders are a series of eminences that correspond to the roots of the maxillary teeth.The most prominent is usually found over the canine tooth
and is often referred to as the ***canine eminence.*** Superior to the canine fossa (located just distal to the canine eminence) is the infraorbital foramen, through which blood vessels and terminal branches of the infraorbital nerve emerge.
Bone in the region of the maxillary teeth is commonly of the more porous cancellous variety, leading to a significantly greater incidence of clinically adequate anesthesia than in areas where denser cortical bone is present, such as in the mandible. In many areas, bone over the apices of the maxillary teeth is tissue-paper thin or shows evidence of dehiscence.

Anterior View of the Skull. *1*, Anterior nasal spine; *2*, body of mandible; *3*, frontal bone; *4*, frontal notch; *5*, frontal process of maxilla; *6*, glabella; *7*, greater wing of sphenoid bone; *8*, infraorbital foramen;
9, infraorbital margin; *10*, inferior nasal concha; *11*, inferior orbital fissure; *12*, lacrimal bone; *13*, lesser wing of sphenoid bone; *14*, maxilla; *15*, mental foramen; *16*, mental protuberance; *17*, middle nasal concha; *18*, nasal bone; *19*, nasal septum; *20*, nasion; *21*, orbit (orbital cavity); *22*, ramus of mandible; *23*, superior orbital fissure; *24*, supraorbital foramen; *25*, supraorbital margin; *26*, zygomatic bone.

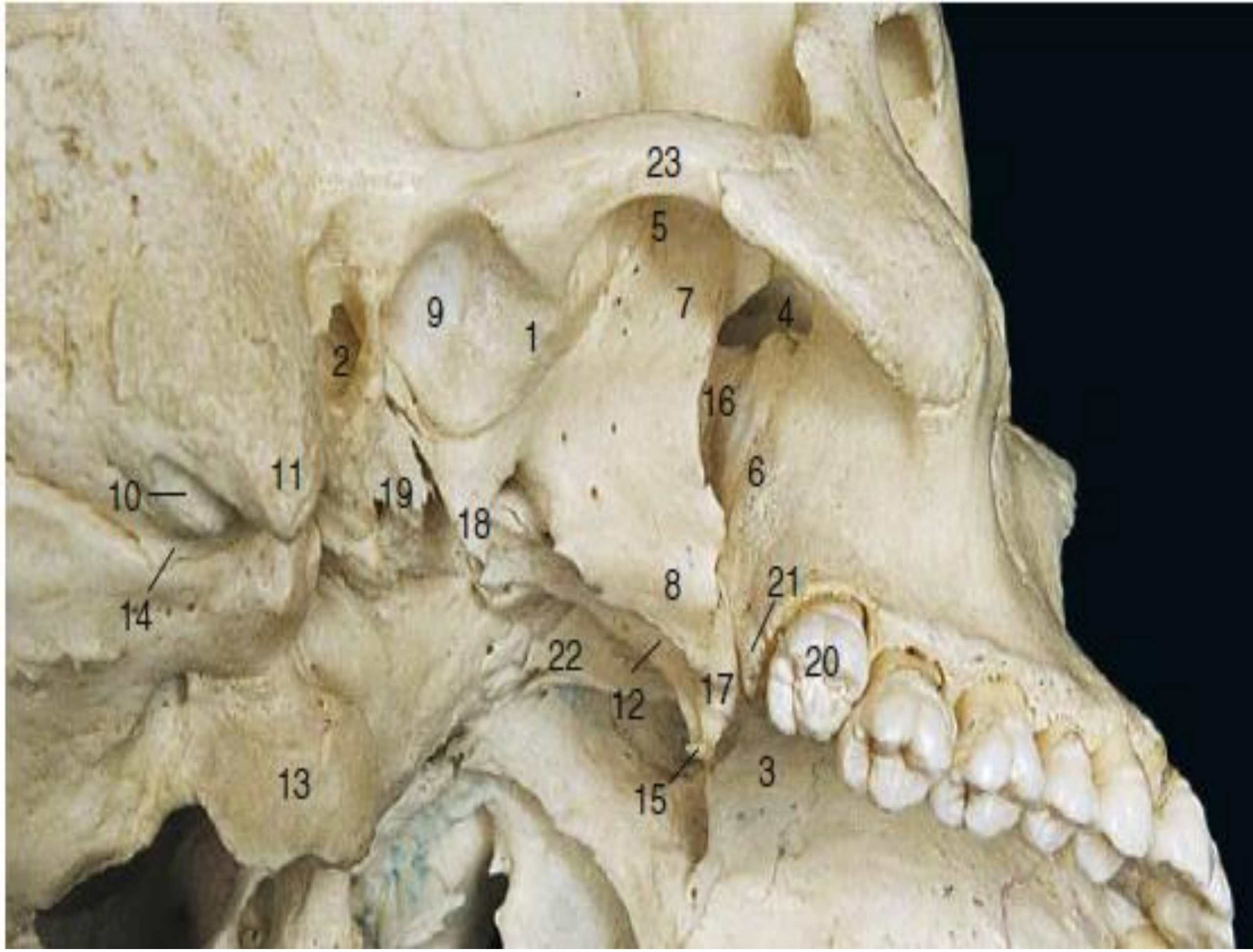
The inferior temporal surface of the maxilla is directed backward and laterally Its posterior surface is pierced by several alveolar canals that transmit the PSA nerves and blood vessels. The maxillary tuberosity, a rounded eminence, is found on the inferior posterior surface. On the superior surface is a groove, directed laterally and slightly superiorly, through which the maxillary nerve passes.

This groove is continuous with the infraorbital groove.

The palatal processes of the maxilla are thick horizontal projections that form a large portion of the floor of the nose and the roof of the mouth. The bone here is considerably

thicker anteriorly than posteriorly. Its inferior (or palatal) surface constitutes the anterior three-fourths of the hard palate

Many foramina (passages for nutrient blood vessels) perforate it. Along its lateral border, at the junction with the alveolar process, is a groove through which the anterior palatine nerve passes from the greater palatine foramen. In the midline in the anterior region is the funnel-shaped opening of the incisive foramen.

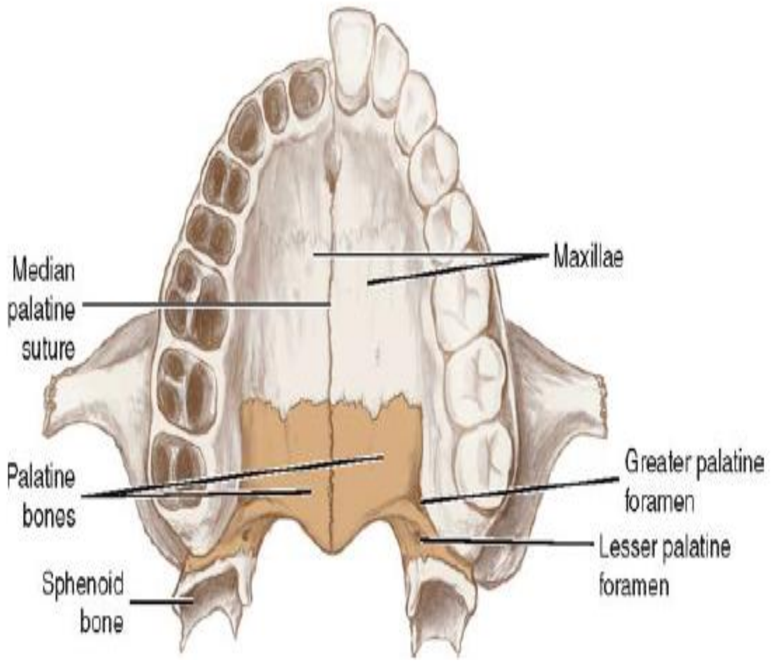


Infratemporal Aspect of the Maxilla. *1*, Articular tubercle; *2*, external acoustic meatus; *3*,horizontal plate of palatine bone; *4*, inferior orbital fissure; *5*, infratemporal crest; *6*, infratemporal (posterior) surface of maxilla; *7*, infratemporal surface of greater wing of sphenoid bone; *8*, lateral pterygoid plate; *9*,mandibular fossa; *10*, mastoid notch; *11*, mastoid process; *12*, medial pterygoid plate; *13*, occipital condyle; *14*, occipital groove; *15*, pterygoid hamulus; *16*, pterygomaxillary fissure and pterygopalatine fossa; *17*, pyramidal process of palatine bone; *18*, spine of sphenoid bone; *19*, styloid process and sheath; *20*, third molar tooth; *21*, tuberosity of maxilla; *22*, vomer; *23*, zygomatic arch

Four canals are **located in this opening**: two for the descending palatine arteries and two for the **nasopalatine nerves**. In many skulls,especially those of persons below 6 years of age, a fine suture line extends laterally from the incisive foramen to the border of the palatine process by the canine teeth. The small area anterior to this suture is termed the *premaxilla*.

The horizontal plate of the palatine bone forms the posterior fourth of the hard palate. Its anterior border articulates with the palatine process of the maxilla, and its posterior border serves as the attachment for the soft palate.

Foramina are present on its surface, representing the lower end of the pterygopalatine canal, through which descending palatine blood vessels and the anterior palatine nerve run

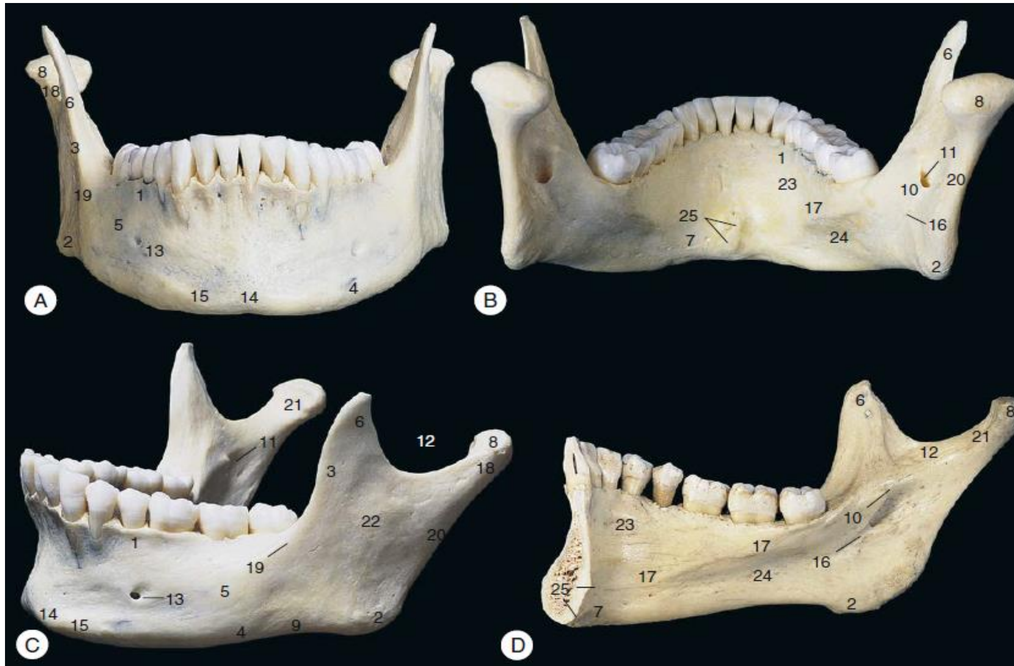


Inferior View of the Hard Palate

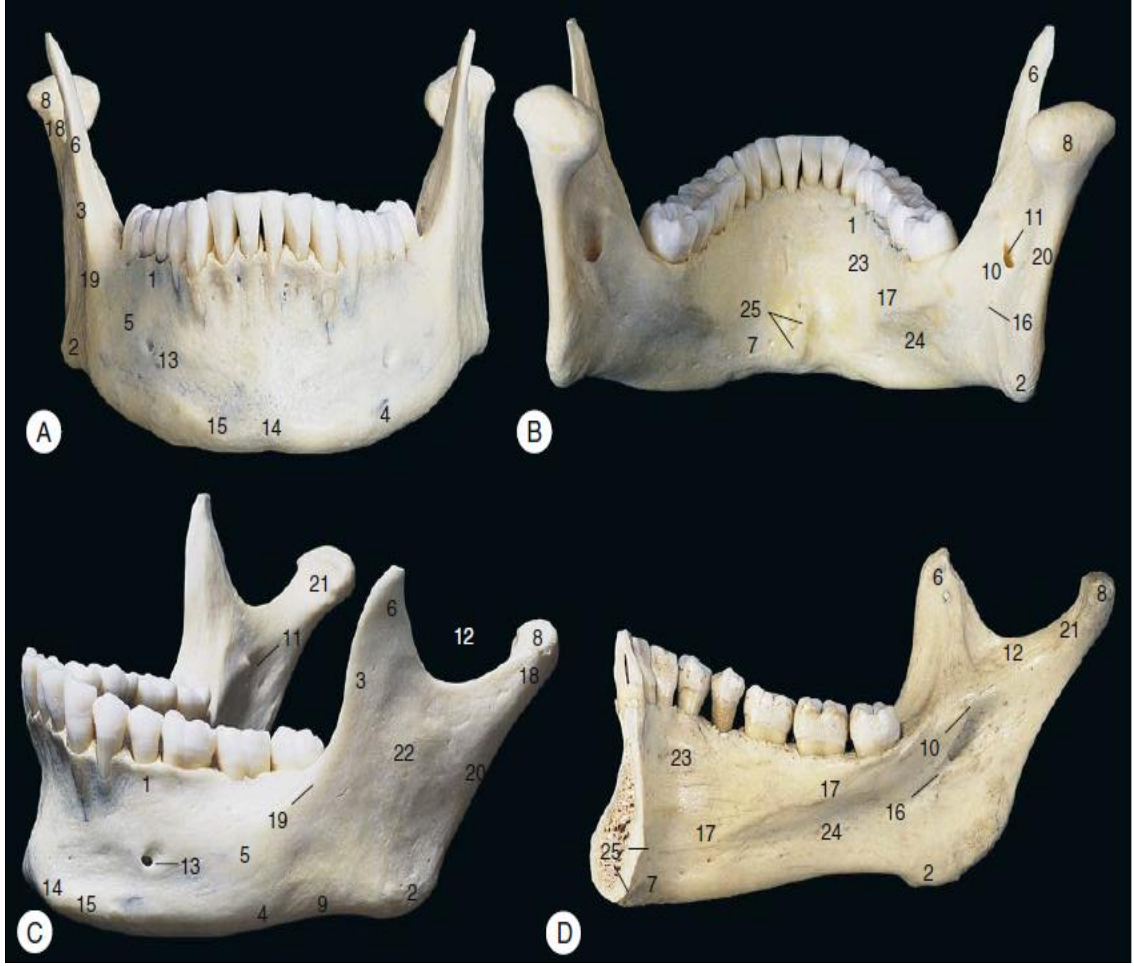
Osteology: Mandible

The mandible is **the largest** and **strongest bone of the face**. It consists of a curved **horizontal portion (the body**) and **two perpendicular portions (the rami).** The buccal cortical plate of the adult mandible most often is **sufficiently dense so as to** preclude effective infiltration of anesthesia in its vicinity.

The external (lateral) surface of the body of the mandible is marked in the midline by a faint ridge, an indication of the symphysis of the two pieces of bone from which the mandible is created



The bone that forms the buccal and lingual alveolar processes in the anterior region (incisors) is usually less dense than that over the
posterior teeth, permitting infiltration (supraperiosteal) anesthesia to be used with some expectation of success.
In the region of the second premolar on each side, midway between the upper and lower borders of the body, lies the mental foramen.
The mental foramen is below the crown of the second premolar. The mental **nerve**, **artery**, and **vein exi**t the mandibular canal here. Bone along this external surface of the mandible is commonly thick cortical bone.
The lingual border of the body of the mandible is concave from side to side
Extending upward and **backward** is the **mylohyoid line,** giving origin to the mylohyoid muscle.
Bone along the lingual aspect of the mandible is usually quite thick; however, in **approximately 68% of mandibles**, lingual foramina are located in the **posterior (molar) region**.
The function of these foramina is as yet unclear, but some may contain sensory fibers from the mylohyoid nerve that innervate portions of mandibular molars.



The mandible (A) from the front, (B) from behind and above, and (C) from the left and front, and (D) internal view from the left. *1*, Alveolar part; *2*, angle; *3*, anterior border of ramus; *4*, base; *5*, body; *6*, coronoid process; *7*, digastric fossa; *8*, head; *9*, inferior border of ramus; *10*, lingula; *11*, mandibular foramen; *12*, mandibular notch; *13*, mental foramen; *14*, mental protuberance; *15*, mental tubercle; *16*, mylohyoid groove; *17*, mylohyoid line; *18*, neck; *19*, oblique line; *20*, posterior border of ramus; *21*, pterygoid fovea; *22*, ramus; *23*, sublingual fossa; *24*, submandibular fossa; *25*, superior and inferior mental spines (genial tubercles).

In addition, bone on the lingual surface of the incisor teeth frequently demonstrates multiple small perforations, perhaps explaining recent clinical trials in which mandibular lingual infiltration had significant success in providing pulpal anesthesia.

The lateral surface of each ramus is flat, composed of dense cortical bone, and provides attachment for the masseter muscle along most of its surface The medial surface contains the mandibular foramen, located roughly halfway between the superior and inferior borders and two-thirds to three-fourths the distance from the anterior border of the ramus to its posterior border.

Other studies of the **anteroposterior location of the mandibular foramen have provided differing locations**.

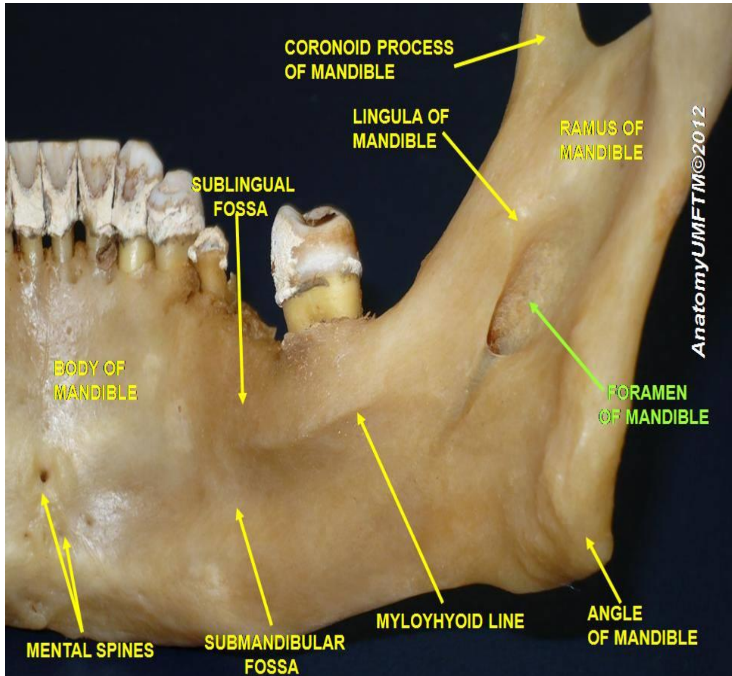
found the foramen most often in the third quadrant from the anterior part of the ramus .The mandibular canal extends obliquely downward and anteriorly within the ramus. It then courses horizontally forward in the body, distributing small dental branches to the mandibular teeth **posterior to the mental foramen.**

The mandibular foramen is the point through which the inferior alveolar nerve, artery, and vein enter the mandibular canal.

The height of this foramen varies greatly, ranging from 1 to 19 mm or more **above the level of the occlusal plane**.Aprominent ridge, **the lingula mandibulae**, lies on the **anterior margin of the foramen**. The lingula serves as an attachment for the **sphenomandibular ligament**.

At the **lower end of the mandibular foramen**, the mylohyoid groove begins, coursing obliquely downward and anteriorly. In this groove lie the mylohyoid nerve and vessels.

Bone along the lingual surface of the mandible is usually dense .On rare occasions, bone over the lingual aspect of the third molar roots is less dense, Permitting a greater chance that supraperiosteal anesthesia will be successful.



However, the proximity of the lingual nerve to this site leads to caution against attempting lingual infiltration in the area of the mandibular molars.

The superior border of the ramus has two processes:

the **coronoid anteriorly** and the **condylar posteriorly**.

Between these two processes is a deep concavity, the mandibular **(sigmoid) notch.**

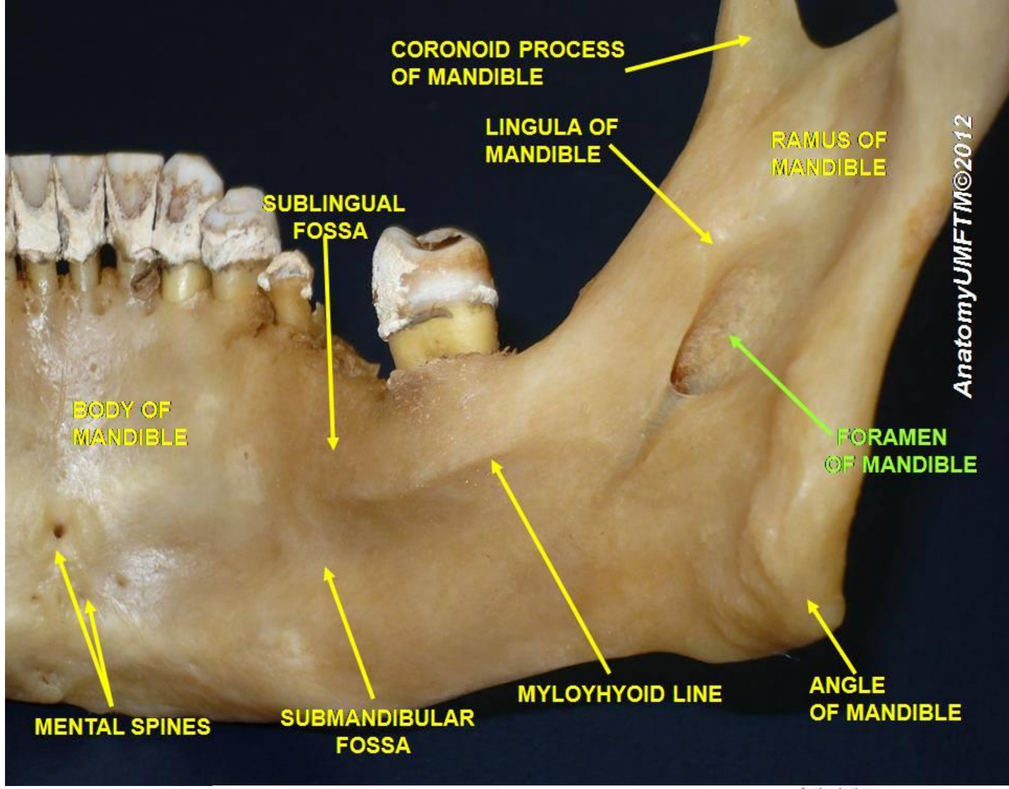
The **coronoid process** is thinner than the **condylar process**.

Its anterior border is concave—the coronoid notch.

The coronoid notch is a landmark for determining the height of needle penetration in the inferior alveolar nerve block technique.

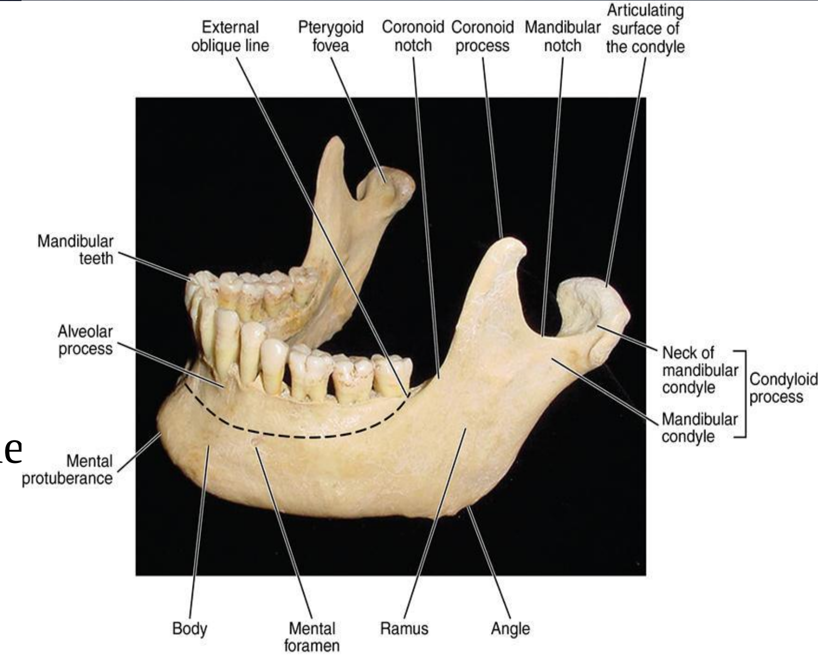
The condylar process is thicker than the coronoid process.

The condylar head, the thickened articular portion of the condyle, sits atop the constricted neck of the condyle.



The condylar neck is flattened front to back. The attachment for the external pterygoid muscle is on its anterior surface. When cut horizontally at the level of the mandibular foramen, the ramus of the mandible can be seen to be thicker in its anterior region than it is posteriorly. **This is of clinical importance during the inferior alveolar nerve block**. The thickness of soft tissues between needle penetration and the osseous tissues of the ramus at the **level of the mandibular foramen averages about 20 to 25 mm.** Because of increased thickness of bone in the anterior third of the ramus, the thickness of soft tissue is decreased accordingly (approximately 10 mm).

Knowing the depth of penetration of soft tissue before contacting osseous tissues can aid the administrator in determining correct positioning of theneedle tip.



The ligaments;

1. **lateral temporomandibular ligament**

This ligament limits the movement of the mandible in a posterior direction

2. Sphenomandibular ligament ; medial side of the joint

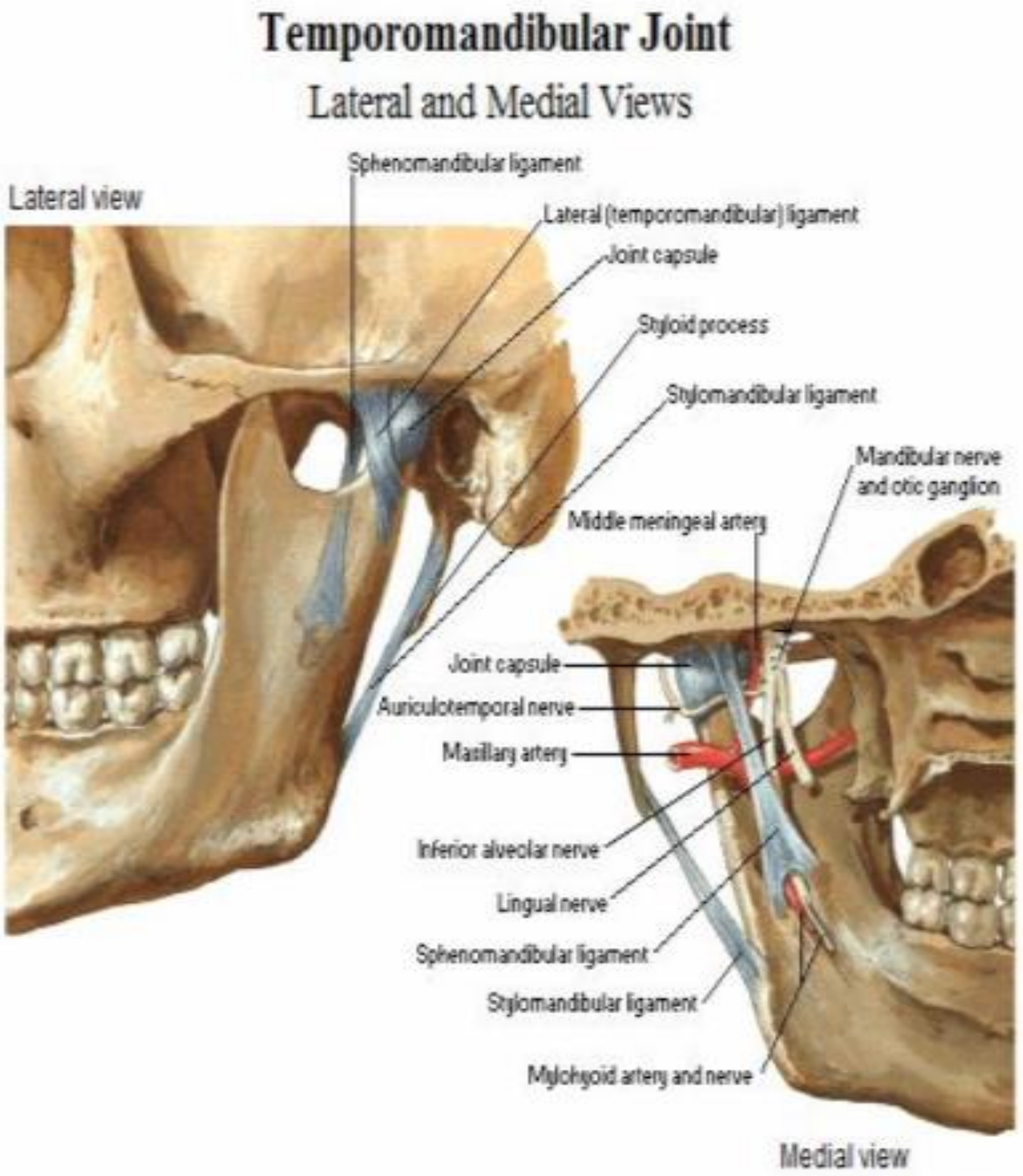
3. Stylomandibular ligament

Nerve Supply

Auriculotemporal and masseteric branches of the man- dibular nerve

Movements of mandible

1. Protraction: forward movement
2. Retraction: backward movement
3. Elevation: upward movement
4. Depression: downward movement
5. Rotation can also occur, during chewing.



Clinical Significance of the Temporomandibular Joint :

The great strength of the lateral temporomandibular ligament
prevents the head of the mandible from passing backward and fracturing the tympanic plate when a severe blow falls on the chin.

(c) Protraction and retraction
(d) Elevation and depression

Clinical Relevance:

Inferior Alveolar Nerve Block:

The **inferior alveolar nerve**, a branch of V3, travels through the mandibular foramen and mandibular canal. Within the mandibular canal, the inferior alveolar nerve forms the inferior dental plexus, which innervates the lower teeth. A major branch of this plexus, the **mental nerve**, supplies the skin and mucous membranes of the lower lip, skin of the chin, and the gingiva of the lower teeth. In some dental procedures which require a **local anaesthesia**, the inferior alveolar nerve is blocked before **it gives rise to the plexus.**

The anaesthetic solution is administered at the **mandibular foramen,** causing numbness of area supplied by the inferior alveolar nerve. The anaesthetic fluid also spreads to the **lingual nerve** which originates near the inferior alveolar nerve, causing numbness of the anterior 2/3 of the tongue

Clinical Relevance:

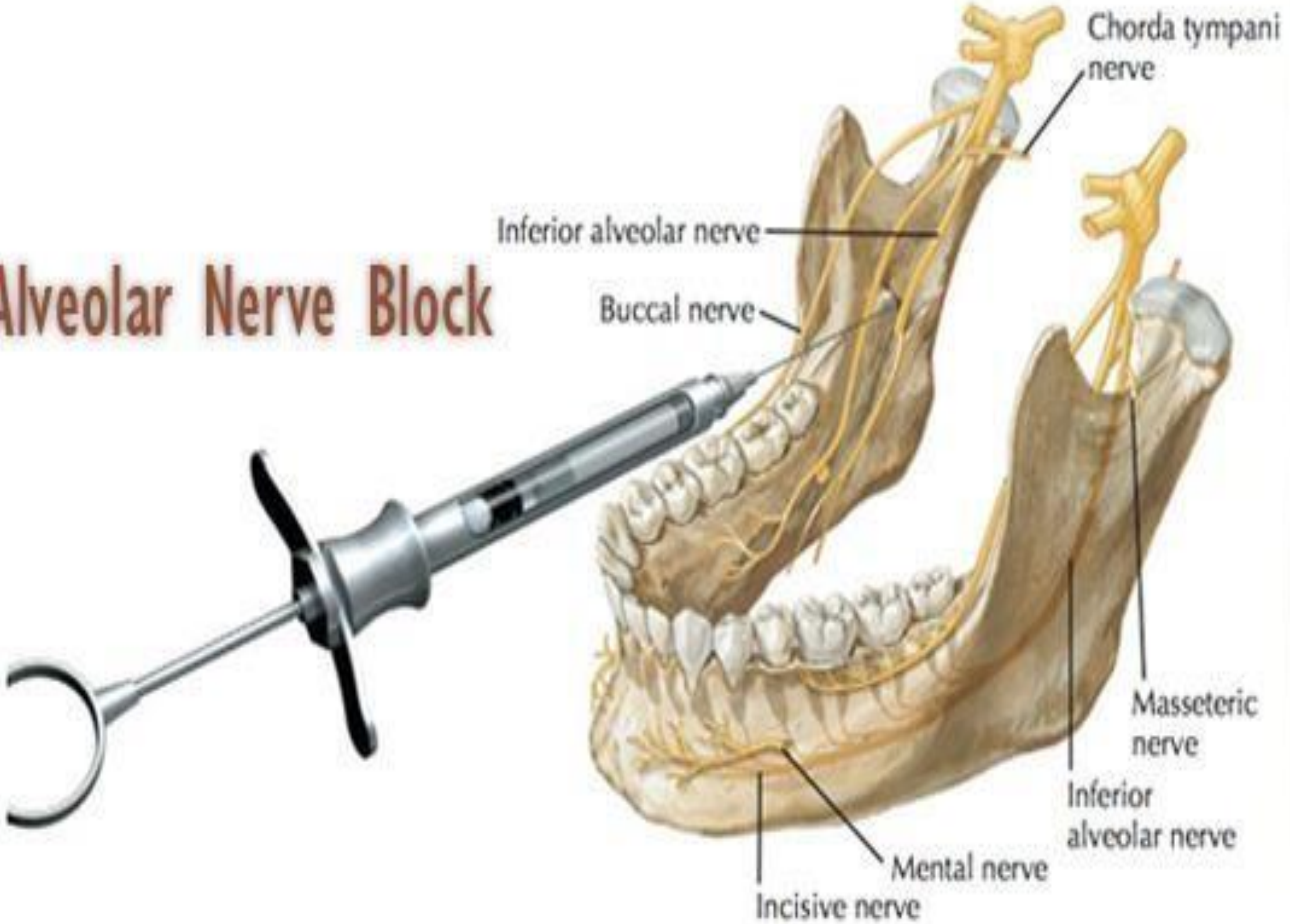
Examination of the Trigeminal Nerve

Testing sensory supply: ask the patient to close their eyes and introduce a cotton wisp to areas of the face supplied by the three divisions of the trigeminal nerve to detect tactile sensory competence.

Testing motor supply: ask the patient to **clench their jaw** as you palpate superior to the **zygomatic arch** to feel for contraction of the **temporalis** and then repeat **palpating inferiorly for the masseter**. Ask the patient to open their mouth and deviate their mandible to the right and left to check for competence of the medial and lateral pterygoid muscles.

Test for corneal reflex – note this requires a competent **ophthalmic** branch and **temporal** and **zygomatic branches** of the facial nerve.

Inferior Alveolar Nerve Block



- **BRANCHES of Mandibular, Or Third Division Of The Trigeminal Nerve**

• Meningeal branch (nervus spinosus)
• Nerve to medial pterygoid

• **Anterior trunk**:
 Masseteric nerve
 Deep temporal nerves
 Nerve to lateral pterygoid
 Buccal nerve

• **Posterior trunk:**
 Auriculotemporal nerve
 Lingual nerve
 Inferior alveolar nerve

Motor fibers of the trigeminal nerve supply the following muscles:
1- Masticatory
A- Masseter
B- Temporalis
C- Pterygoideus medialis
D- Pterygoideus lateralis

2- Mylohyoid
3- Anterior belly of the digastric
4- Tensor tympani
5- Tensor veli palatini

The areas innervated by V_3 are included in the following outline:

1 Sensory root

A- Skin

Temporal region

Auricula

External auditory meatus

Cheek

Lower lip

Lower part of the face (chin region)

B- Mucous membrane

Cheek

Tongue (anterior two thirds)

Mastoid cells

C- Mandibular teeth and periodontal tissues

D- Bone of the mandible

E- Temporomandibular joint

F-Parotid gland

2- Motor root

A- Masticatory muscles

Masseter

Temporalis

Pterygoideus medialis

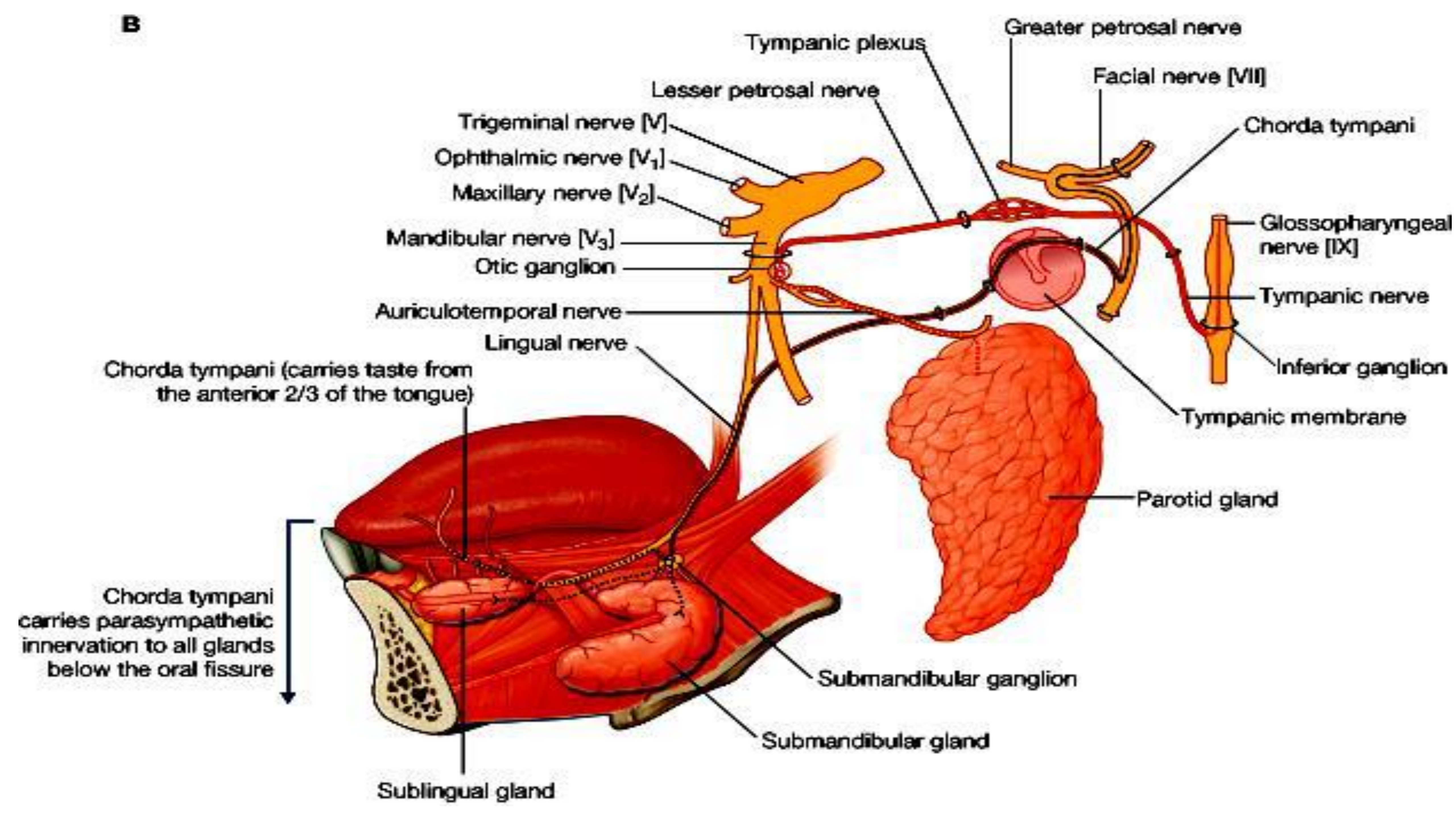
Pterygoideus lateralis

B- Mylohyoid

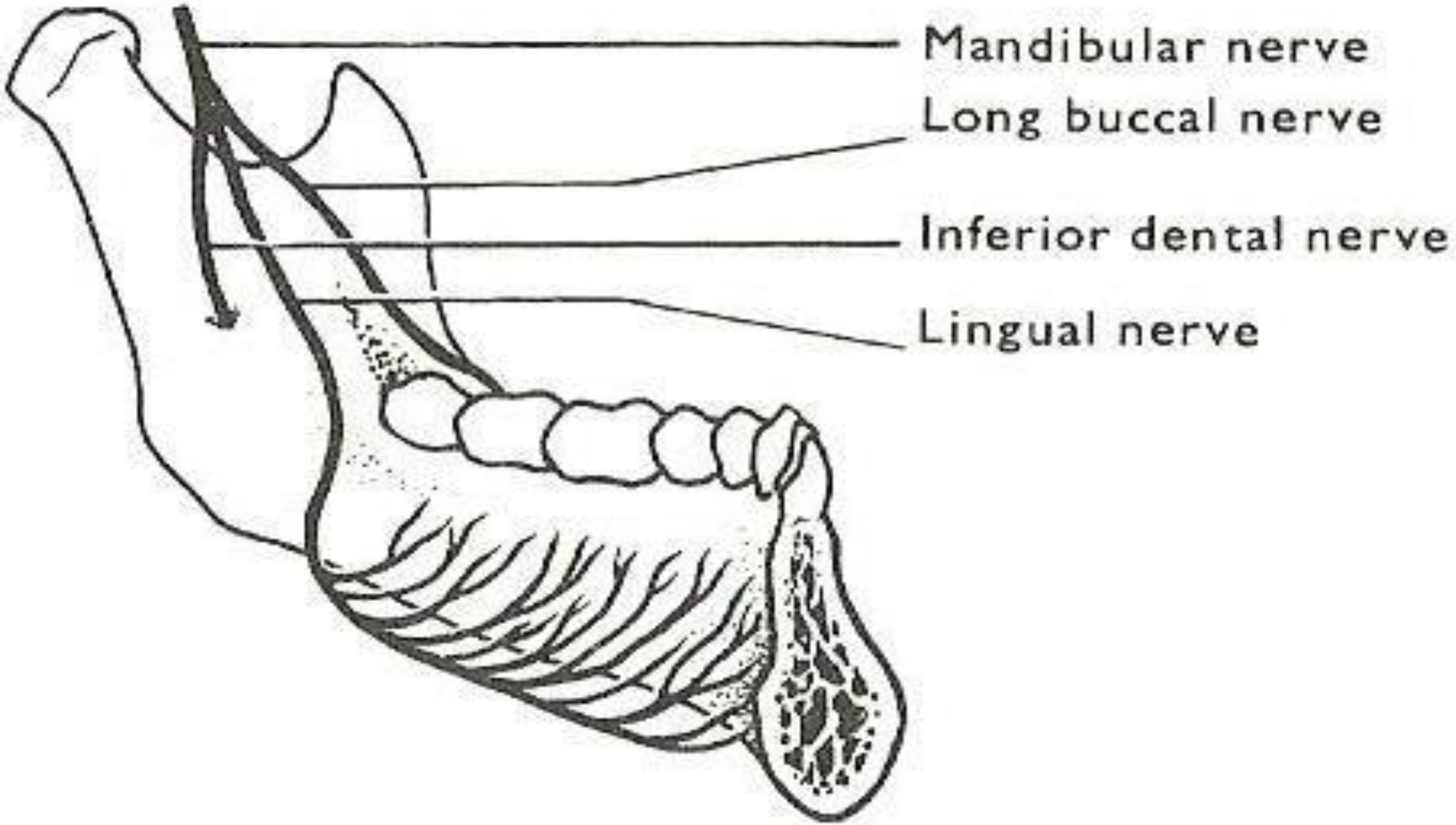
C-Anterior belly of the digastric

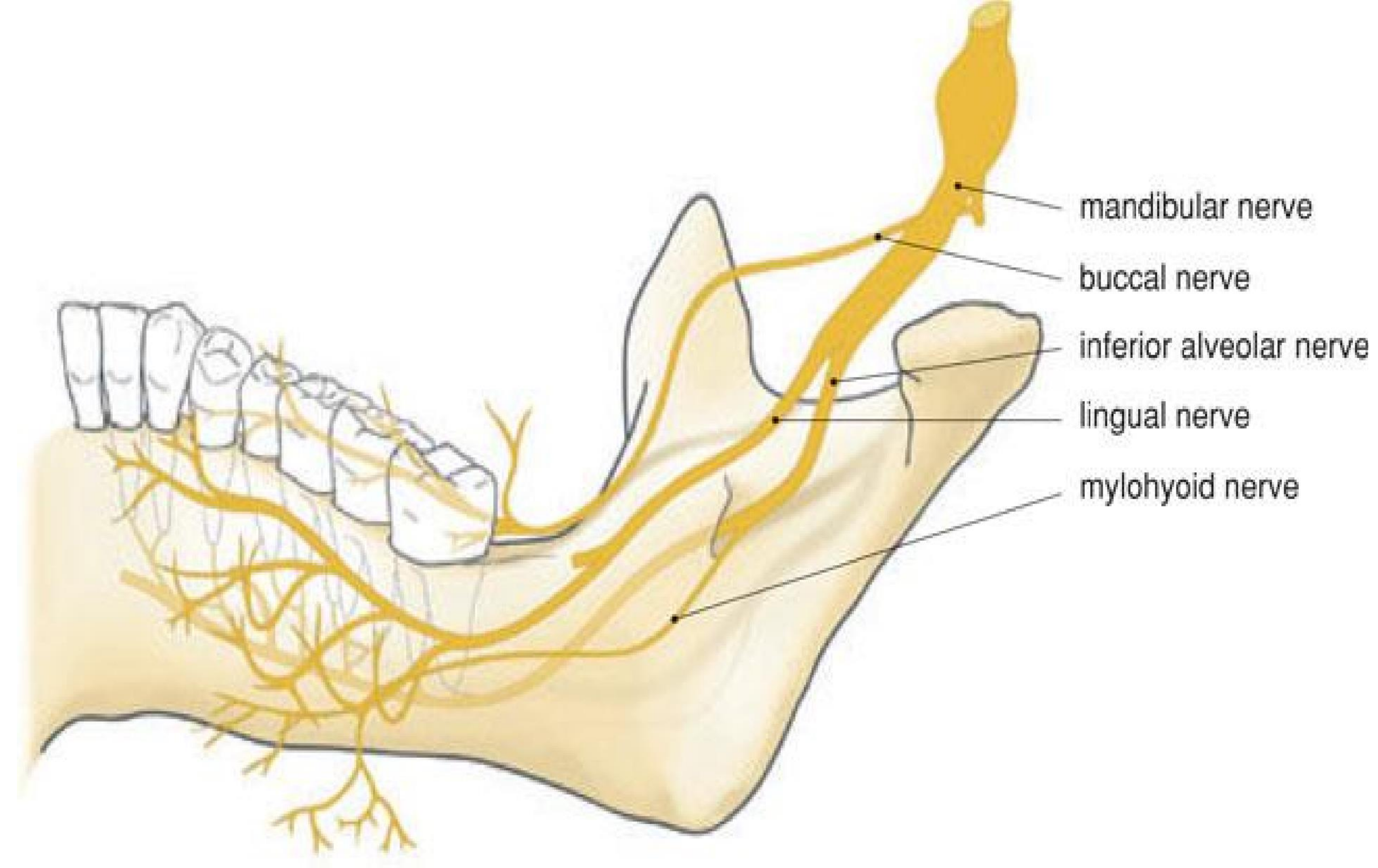
D- Tensor tympani

E- Tensor veli palatini

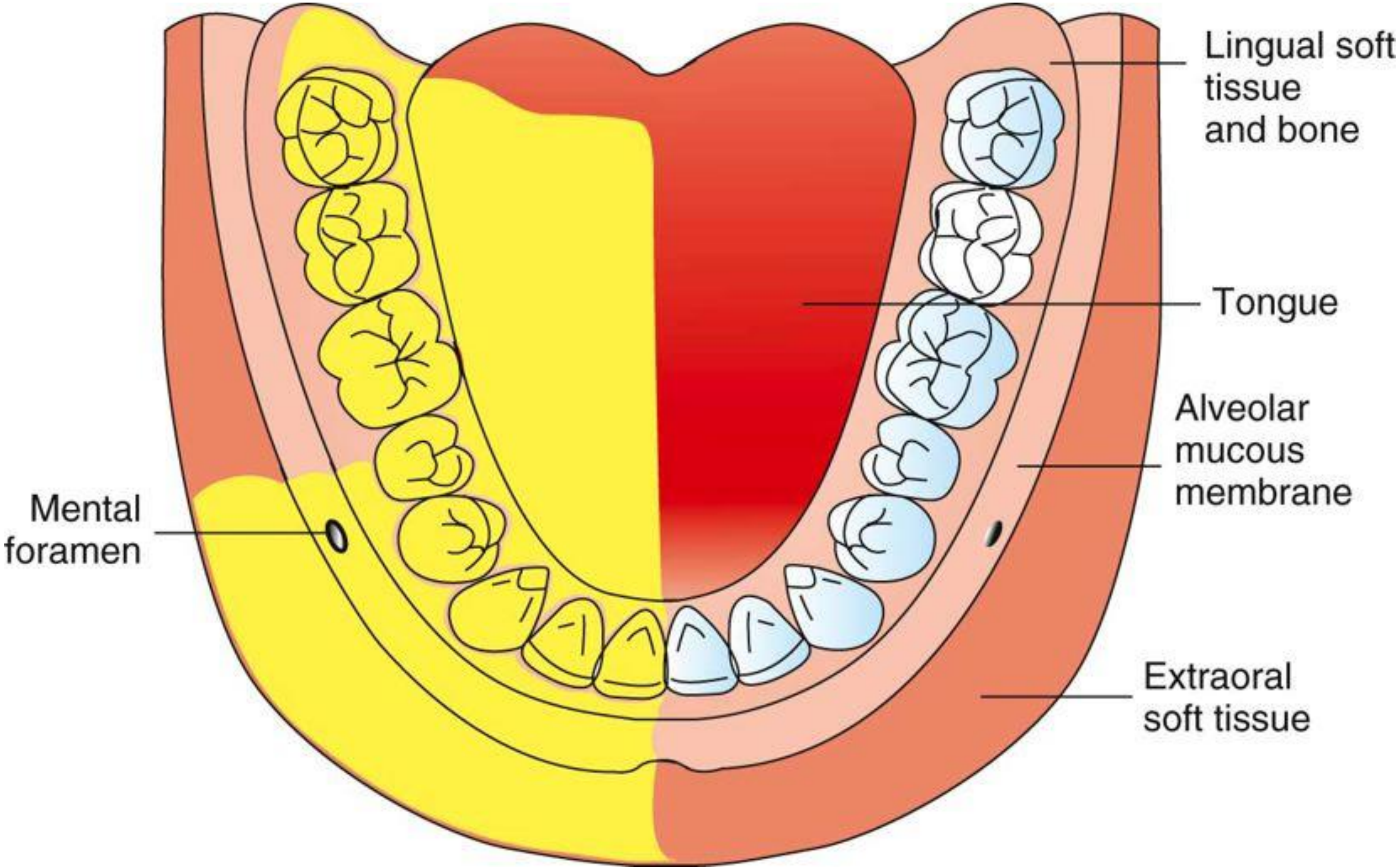


B
Tympanic plexus
Greater petrosal nerve
Facial nerve [VII]
Lesser petrosal nerve
Trigeminal nerve [V]
Chorda tympani
Ophthalmic nerve [V_1]
Maxillary nerve [V_2]
Glossopharyngeal nerve [IX]
Mandibular nerve [V_3]
Otic ganglion
Tympanic nerve
Auriculotemporal nerve
Lingual nerve
Inferior ganglion
Chorda tympani (carries taste from the anterior 2/3 of the tongue)
Tympanic membrane
Parotid gland
Chorda tympani carries parasympathetic innervation to all glands below the oral fissure
Submandibular ganglion
Submandibular gland
Sublingual gland

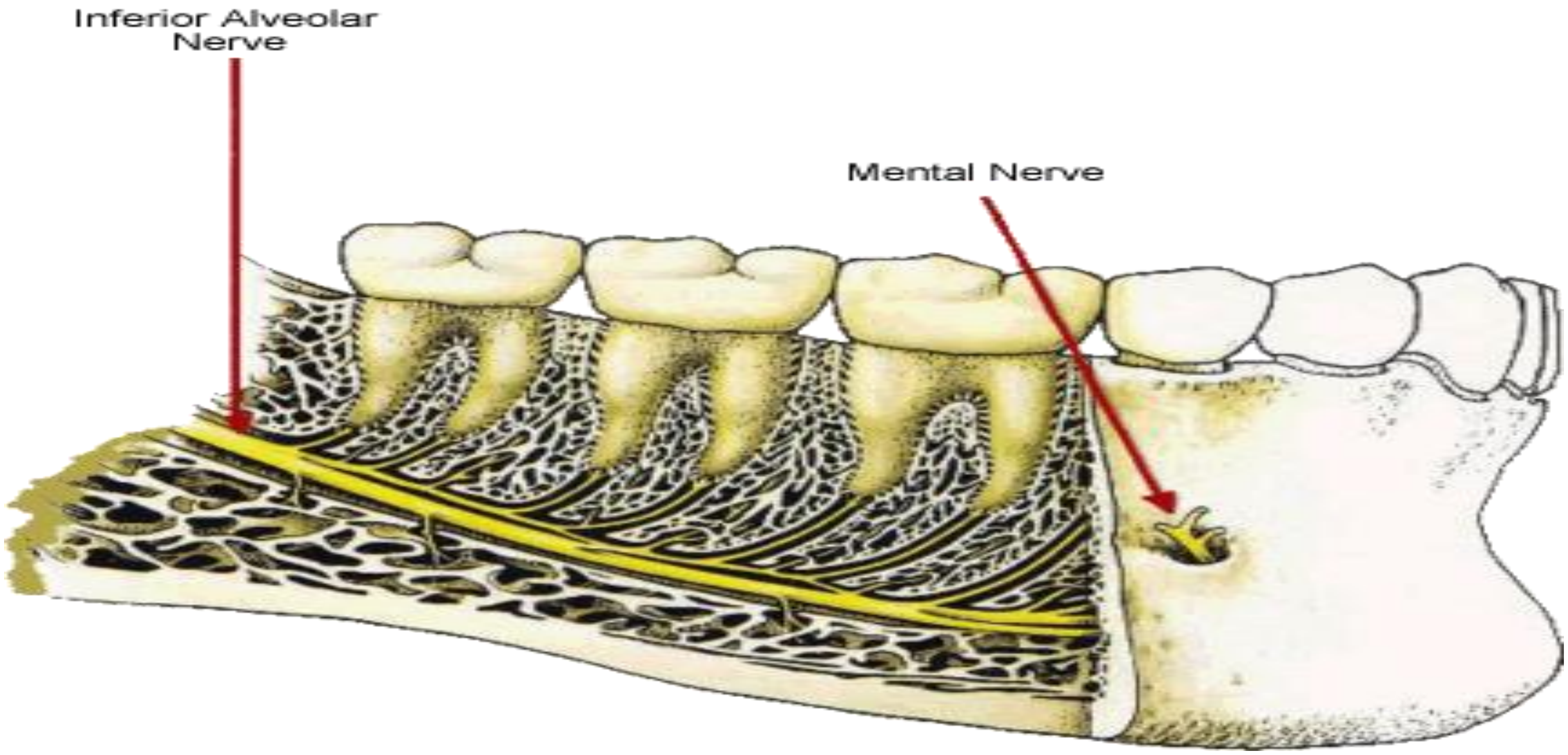

Mandibular nerve
Long buccal nerve
Inferior dental nerve
Lingual nerve


mandibular nerve
buccal nerve
inferior alveolar nerve
lingual nerve
mylohyoid nerve

Area anesthetized by an inferior alveolar nerve block



- Dental plexus represent the terminal branches of main fibers that innervate the roots , bone and periodontium



- Bone in the maxilla quite porous and cancellous leading to greater incidence of adequate anasthesia than mandible

- The labial cortical bone of anterior mandible usually less dense than that of posterior region so regional mental block or even infiltration sufficient to anasthetize the anterior teeth

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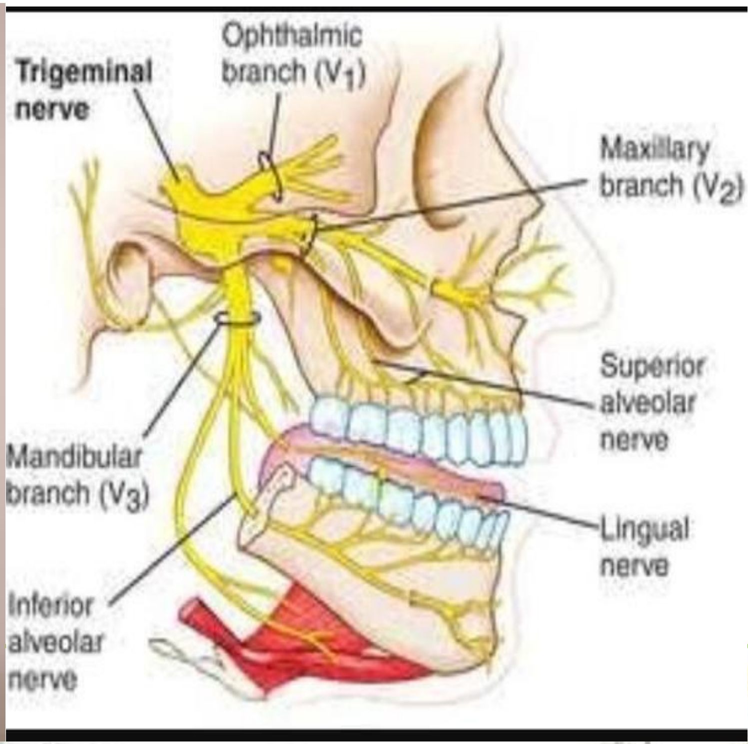
Academic Year 3
Techniques of Local Anesthesia
Assistant Prof Dr. Hamid Hammad Enezei

Ph.D in Oral & Maxillofacial Surgery



Techniques of local anesthesia

- Basic injection techniques
- Techniques of maxillary anesthesia

✓ Local infiltration.
✓ Posterior superior alveolar nerve block
✓ Middle superior alveolar nerve block
✓ Anterior superior alveolar nerve block (infraorbital nerve block)
✓ Greater palatine nerve block
✓ Nasopalatine nerve block
✓ Maxillary nerve block

Techniques of local anesthesia

- Techniques of local anesthesia

✓ Techniques of mandibular anesthesia
✓ Inferior alveolar nerve block
✓ Buccal nerve block
✓ Mandibular nerve block: The Gow-Gates technique
✓ Vazirani-Akinosi closed-mouth mandibular block
✓ Mental nerve block
✓ Incisive nerve block

Techniques of local anesthesia

- Supplemental injection techniques

✓ Intraosseous injection
✓ Periodontal ligament injection
✓ Intraseptal injection
✓ Intrapulpal injection

The dental cartridge:

Is a **glass cylinder containing** the local anesthetic drug, among other ingredients. the glass cylinder itself can hold 1.8, 2 ,2.2 mL of solution; however, as prepared today, the dental cartridge contains approximately 1.8 mL of local anesthetic solution. Local anesthetic cartridges list their volume
The dental local anesthetic cartridge is, by common usage, referred to by dental professionals as a ***carpule.***
The following is a brief history of the dental anesthetic "carpule." Local anesthesia was introduced in 1905 with
the synthesis of procaine hydrochloride by Alfred Einhorn
(1856–1917) in Germany. The drug was supplied in powder
form, so dentists had to mix up a new solution each
time they needed it.

Plastic cartridges have several negative features, **primarily leakage of solution** during injection, the requirement for **considerable force to be applied** to the plunger of the syringe (e.g., periodontal ligament [PDL], nasopalatine), and the fact that the plunger does not "glide" down the plastic cartridge as smoothly **as it does down the glass cartridge**, leading to sudden **spurts of administration of local anesthetic** under increased pressure, which can produce pain in the patient.

Another problem with plastic cartridges is that they are permeable to air. Exposure to oxygen leads to more rapid degradation of the vasoconstrictor in the cartridge and to a shorter shelf life.

The Cartridge

Components

The prefilled 1.8-mL dental cartridge consists of four parts

1. cylindrical glass tube
2. stopper (plunger, bung)
3. aluminum cap
4. diaphragm

The stopper (plunger, bung) is located at the end of the cartridge that receives the harpoo of the aspirating syringe. The sharp harpoon is embedded into the silicone (non–latex-containing) rubber plunger with gentle finger pressure applied to the thumb ring of the syringe. The plunger occupies a little less than 0.2 mL of the volume of the entire cartridge. Today, local anesthetic stoppers are treated with **silicone**, **eliminating both the paraffin** and **the glycerin that were used in years past**. “Sticky stoppers” (stoppers that do not move smoothly down the glass cartridge) are rare today. Recent years have seen a move toward the use of a uniform black rubber stopper in all local anesthetic drug combinations. Virtually gone are the color-coded red, green, and blue stoppers that aided in identification of the drug.

Composition of Local Anesthetic agent

1) Local anaesthetic agent : eg Lignocaine HCL – 2% (20 mg/ml)
2) Vasoconstrictor : Adrenaline – 1:80,000 (0.012 mg) or Epinephrine
3) Reducing Agent: Sodium Metabisulphite – 0.5 mg
4) Preservative: Methylparaben – 0.1% (1mg)
5) Isotonic Solution: Sodium Chloride – 6 mg
6) Fungicide: Thymol
7) Vehicle: Ringer’s Solution
8) Diluting Agent: Distilled water
9) To adjust pH: Sodium Hydroxide
10) Nitrogen Bubble: 1-2mm in diameter and is present to prevent Oxygen from being trapped in the cartridge and potentially destroying the Vasopressor or vasoconstrictor.

Actions of each component of Local Anesthetic agent:

Vasoconstrictor function:

Decrease blood flow to the site of injection, absorption of local anesthetic into the cardiovascular system is solved, **decrease the risk of local toxicity**, **higher volume of local anesthetic agent remain in and around the nerve for longer period**, **thereby increasing the duration of action**, **vasoconstrictor decreases bleeding at the site of their administration**.

Preservative :

Stability of **modern local anesthetic solution** is maintained by adding c**aprylhydro-cuprienotoxin** which includes **xylotox** and **methyl paraben.**

Reducing agent :

These act as preservatives for vasoconstrictor agents. Vasoconstrictors are unstable in solution and may **oxidize,** especially on a **prolonged exposure to sunlight**. **Sodium metabisulphite** which competes for the available oxygen is added in the concentration **between 0.05% and 0.1%**

Vehicle :

All the above solutions and local anesthetic agent are **dissolved in a modified ringer solution**. This isotonic vehicle minimizes **discomfort during inje**ction

Function of Nitrogen bubble in LA cartridge

The bubble size 1-2mmin diameter and is present to prevent .Oxygen from being trapped in the cartridge and potentially destroying the **Vasopressor** or **vasoconstrictor,** so this is the function of Nitrogen Bubble in the LA cartridge.

We see many patients who ask "**what do dentists** use to **numb teeth or mouth**" before performing a procedure and the answer is dental anesthetics which help in making dental procedures painless be it related to the tooth or the gums and other soft tissue in the mouth or oral cavity. Having proper knowledge about the Allergic Reactions to LA and also we should have good knowledge about the amount of LA to be given to avoid

Dental Local Anesthesia Techniques

The most important skill required of all dental practitioners is the ability to **provide safe** and **effective local anesthesia** (LA).
The injection of local anesthetic is perhaps the **greatest source of patient fear and inability to obtain adequate pain control with minimal discomfort remains a significant concern of dental practitioners**.

The achievement of good local anesthesia requires knowledge of the agents being used, the neuroanatomy involved, and best techniques and devices available.

The agents and anesthetic delivery equipments available today provide the practitioner an array of options to effectively manage the pain associated with dental procedures.

Local anesthesia forms the backbone of pain control techniques in the dental profession.
Local anesthetics represent the safest and most effective method for managing pain associated with dental treatment.
They are the only drugs that prevent the nociceptive impulse from reaching the patient's brain.

Local anesthetics need to be deposited as close to the nerve as possible so that optimal diffusion of the drug may occur, providing profound anesthesia and a pain-free dental experience.

The importance of this is demonstrated by the fact that **when patients** are asked to list the most important factors used when selecting a dentist, the **2 most important are:**
(1)A dentist who does not hurt
(2)A painless injection.

Unfortunately, for painless dentistry to be accomplished, local anesthetics need to be injected using a cartridge, syringe, and needle. This leads to the major problem of fear of needles **(trypanophobia)** and its consequences, ie, the occurrence of syncope or other medical emergencies during injection of the local anesthetic.
More than 50% of medical emergencies occurring in dental offices happen during or immediately following administration of a local anesthetic.

Several highly efficacious and practical techniques can be used to achieve anesthesia of the dentition and surrounding the hard and soft tissues of the maxilla and mandible.
The type of procedure to be performed as well as the location of the procedure determine the technique of anesthesia to be used are:

Topical , Local infiltration, Field block, Nerve block., and supplement technique

Topical anesthesia:

Topical anesthetics act on the peripheral nerves and reduce the sensation of pain at the site of application. In dentistry, they are **used to control local pain caused by needling, placement of orthodontic bands, the vomiting reflex, oral mucositis, and rubber-dam clamp placemen**

Topical anesthetic reduces the slight discomfort associated with **insertion of the needle**.

It is affective to a depth of 2-3mm. Although its application is beneficial for reducing patient discomfort during the **initial phase of local anesthetic administration**, it may be a **disadvantage in children** if the **taste is disagreeable to the patient**.

Also, **excessive length of application** time may **increase apprehension of the approaching procedure**.

It is available in **gel**, **liquid**, **ointment**, **patch** and **pressurized spray forms**.

The most **common topical anesthetics used in dent**istry are those **containing benzocaine** or **lidocaine.**

Benzocaine (ethyl aminobenzoate) is an ester local anesthetic. It is available in up to **20% concentrations**. **It is not known to produce systemic toxicity** but can produce **local allergic reactions** especially **after prolonged** or

It exhibits **poor solubility in water** and **poor absorption into the cardiovascular system**, thus it **remains at the site of application longer**, providing a **prolonged duration of action**.

Systemic toxic (overdose) reactions are **virtually unknown**. Benzocaine is reported to **inhibit the antibacterial action** of **sulfonamides.**

Lidocaine is available as a **solution** or **ointment up to 5% concentration** and as a **spray up to 10% concentration**.
It has a **low incidence of allergic reactions** but is **absorbed systemically** and application of **excessive amounts of topical lidocaine may absorb rapidly into the cardiovascular system leading to higher local anesthetic blood levels with an increased risk, especially in the pediatric patient**, of **overdose reaction**. Thus, **a minimal amount of topical gel should be applied to the tissue** and a **metered spray** is suggested if an **aerosol preparation is selecte**

Tetracaine is also used for **endoscopic procedures** and **gag control** [

Topical anesthesia will not cause a completely painless injection and that depends more on the needle gauge and duration of the injection.

Topical anesthesia will be helpful for **periodontal examinations** and **very conservative** treatments.

Preparation of Patient

Preparation of the patient prior to injection consists of **two components**, **mental** and **physical**.

Mental preparation begins with explaining to the child, in terminology they can understand, the anesthesia administration process. **Example:**

*Today I'm going to put **your tooth asle**ep, wash some **germs out of your teeth** and **place a white star**. When your tooth falls asleep your **lip** and **tongue will feel fat and funny for a little while**.*

*First, you're going to **sit in my special chair** and then **I'm going to place some (goofy, cherry, bubble gum)** tooth **jelly next to your tooth**. Then I'll wash it away with the **sleepy water**. I'm going to show you everything I do so you can see **how easy this is**.*"

Administration protocol of topical LA

1-Positioning the Patient in the Dental Chair

The patient is positioned with the head and heart parallel to the floor and the feet slightly elevated. Positioning the patient in this manner **reduces the incidence of syncope** that can occur because of **increased anxiety**

2-Drying the Tissue

Use a 2 X 2 gauze to dry the tissue and remove any gross debris around the site of needle penetration. **Retract the lip to obtain adequate visibility during the injection**. Wipe and dry the lip to make retraction easier

3-Apply Topical Anesthetic

Topical anesthetic reduces the slight discomfort associated with insertion of the needle. It is effective to a depth of 2-3mm. It is applied only at the site of preparation. The clinician should avoid excessive amounts that can anesthetize the soft palate and pharynx. The topical anesthetic should remain in contact with the soft tissue 1-2 minutes.

4-Administration of the Anesthetic

There are **two important goals 1-** must accomplish during anesthetic administration; **control** and **limit movement of the patient's head and body** and 2-**communicate with the patient to draw their attention away from the minor discomfort that may be felt during the injection process**.

Most clinicians prefer to keep the **uncapped needle out of the patient's line of sight**. Do not ask the child to close his/her eyes as that is usually a sign to the child that something bad or painful is about to occur.

Instead, the assistant passes the **uncapped syringe behind the patient's head**

5-Stabilization

Before placement of the syringe in the mouth, the patient's head, hands and body should be stabilized. There are two basic positions for stabilizing the patient's head.

A behind the patient position is assumed for injecting the **contralateral quadrants to the clinician’s favored hand** and the anterior regions, i.e., right-handed clinicians injecting the left side, left-handed clinicians injecting the right side.

The clinician stabilizes the **patient’s head** by supporting the head against the clinician’s body with the less favored hand and arm. The clinician stabilizes the jaw by resting the fingers against the mandible for support and retraction of lips and cheek.

For injections on the same side as the clinician’s favored hand, i.e., right side for right-handed clinicians, left side for left-handed clinicians, the clinician assumes a more forward position, 8 o’clock for right-handed clinicians, 4 o’clock for left-handed clinicians.

The clinician stabilizes the patient’s head and retracts the soft tissues with the fingers of the weaker hand resting on the bones of the maxilla and mandible.

To prevent **unexpected movements** of the child’s hands during the injection, the assistant restrains the hands by asking the child to place them on their belly button and placing her hands over them.

6-Communication

The clinician initiates communication with the patient by speaking in a reassuring manner during anesthesia administration. The subject matter can range from describing the process in child friendly terminology, to praise, to storytelling, to singing, or, if the clinician is totally unimaginative, counting. **Avoid words like shot, pain, hurt and injection and substitute words like cold, warm, weird, fat and funny**

Basic Injection Technique

The anesthetic injection begins by stretching the tissue taut at the administration site. Insert the needle 1-2mm into the mucosa with the bevel oriented toward bone. Inject several drops of anesthetic before advancing the needle. Slowly advance the needle toward the target while injecting up to ¼ cartridge of anesthetic to anesthetize the soft tissue ahead of the advancing needle. **Aspirate**.

The anesthetic injection begins by stretching the tissue taut at the administration site. Insert the needle 1-2mm into the mucosa with the bevel oriented toward bone. Inject several drops of anesthetic before advancing the needle. Slowly advance the needle toward the target while injecting up to ¼ cartridge of anesthetic to anesthetize the soft tissue ahead of the advancing needle and then aspirate.

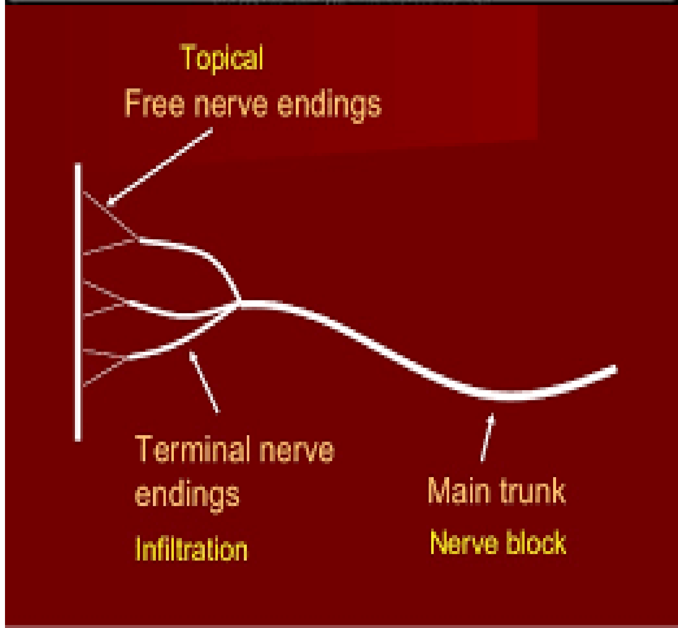
The depth of insertion will vary with the type of injection; however, one should never insert a needle in its entirety **to the hub**. Although a rare occurrence, **retrieving a broken needle fully embedded in soft tissue is extremely difficult.**

Infiltration Anaesthesia:

Small terminal nerve endings in the area of the dental treatment are flooded with local anesthetic solution.

Incision (or treatment) is then made into the same area in which the local anesthetic has been deposited.

An example of local infiltration is the administration of a local anesthetic into an **interproximal papilla before root planing**



FIELD BLOCK

Local anesthetic solution is deposited near the **larger terminal nerve branches** so the anesthetized area will be circumscribed, **preventing the passage of impulses from the tooth to the central nervous system (CNS**). Incision (or treatment) is then made into an area away from the site of injection of the anesthetic.

Maxillary injections administered above the apex of the tooth to be treated are properly termed field blocks **(although common usage identifies them as infiltration or supraperiosteal).**

FIELD BLOCK

NERVE BLOCK

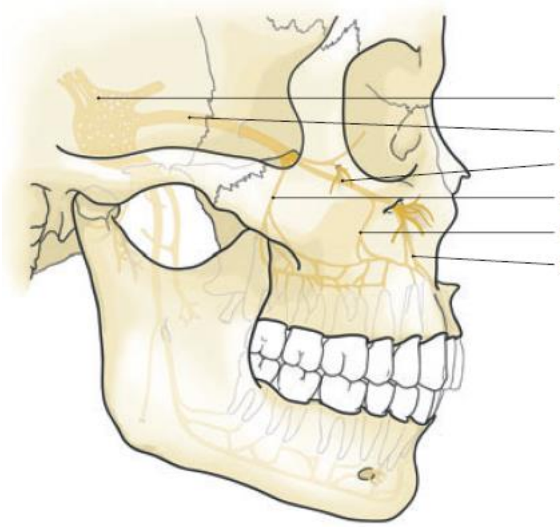
NERVE BLOCK

Local anesthetic is **deposited close to a main nerve trunk**, usually at a distance from the site of operative intervention. **Posterior superior alveolar**, **inferior alveolar**, and **nasopalatine injections** are **examples of nerve blocks.**

The type of injection administered for a given treatment is determined by the extent of the **operative area**. For management of **small,** isolated areas, infiltration **anesthesia may suffice**. When two or three teeth are being restored, **field block is indicated**, whereas for pain control in **quadrant dentistry, regional block anesthesia is recommended**

Techniques of local anaesthesia

- Basic injection techniques
- Techniques of maxillary anesthesia
- Local infiltration.
- Posterior superior alveolar nerve block
- Middle superior alveolar nerve block
- Anterior superior alveolar nerve block (infraorbital nerve block)
- Greater palatine nerve block
- Nasopalatine nerve block
- Maxillary nerve block



Sensory innervation of the upper jaw arises from the **second trunk of the trigeminal nerve**, the maxillary nerve. This main branch of the trigeminal nerve leaves the neurocranium via the foramen rotundum, reaches the pterygopalatine fossa and runs straight through as the infraorbital nerve, branching off many times along its course.

With regard to local anaesthesia in the upper jaw, the following branches are of importance:

- the greater and lesser palatine nerves;
- the posterior, middle and anterior superior alveolar nerves;
- the infraorbital nerve (Thus the main trunk of the maxillary nerve can be reached via the greater palatine foramen, via the infraorbital foramen as well as from high behind the maxillary tuberosity. In practice, high tuberosity anaesthesia is the only practical regional block anaesthesia for almost the entire maxillary nerve. Therefore this regional block anaesthesia technique is used for surgical procedures.

For everyday dental procedures in the upper jaw, infiltration anaesthesia is commonly used. The cortical bone of the outer surface of the upper jaw is **relatively thin**, which facilitates the diffusion of local anaesthetic fluid. All (buccal) roots of the upper teeth can be reached in this way.

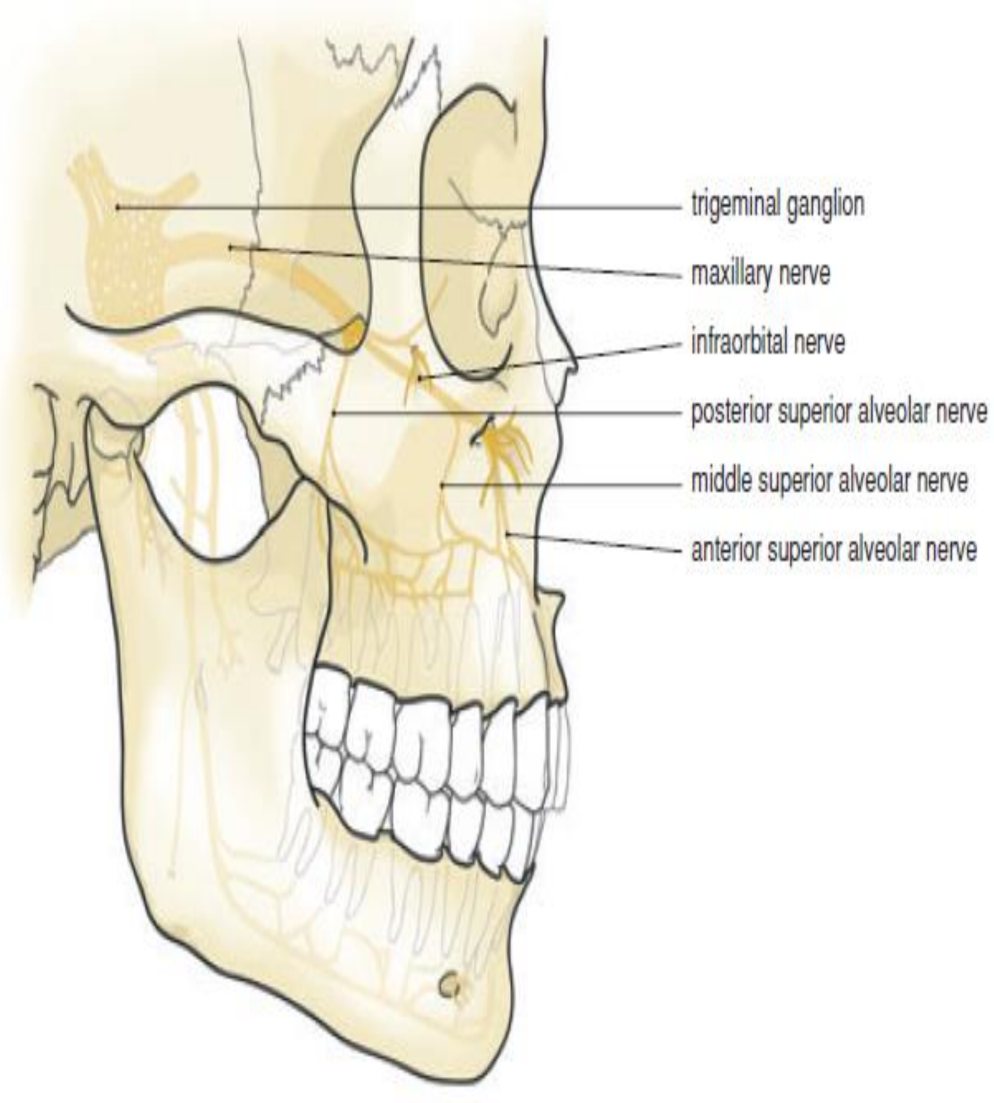
The palatine roots of the molars and possibly the **premolars** are anaesthetized by infiltration anaesthesia of the branches of the greater **palatine nerve** and those of the **nasopalatine nerve**.

Regional block anaesthesia is also **possible via** the **greater palatine foramen** and the **nasopalatine canal**.

Infiltration anaesthesia of the upper jaw is particularly effective, unless an injection is made into an **inflamed area**.

Regional **block anaesthesia** of the **greater pala**tine, **nasopalatine** and **infraorbital nerves** is equally effective.

In cases of regional block anaesthesia using a **high tuberosityblock**, it is usually only the **posterior superior alveolar** and **medial branches that are numbed**, but sometimes also the **palatin**e and infraorbital nerves



Incisors and canines:

Anatomical aspects

Before leaving the **infraorbital foramen** the infraorbital nerve branches off in the infraorbital canal towards the **incisors and canines,** the **anteriorsuperior alveolar nerves**. These nerve branches provide the sensory innervation of the **incisor and canine pulp**, as well as the **vestibular fold**, **the gingiva, the periosteum and the bone.**

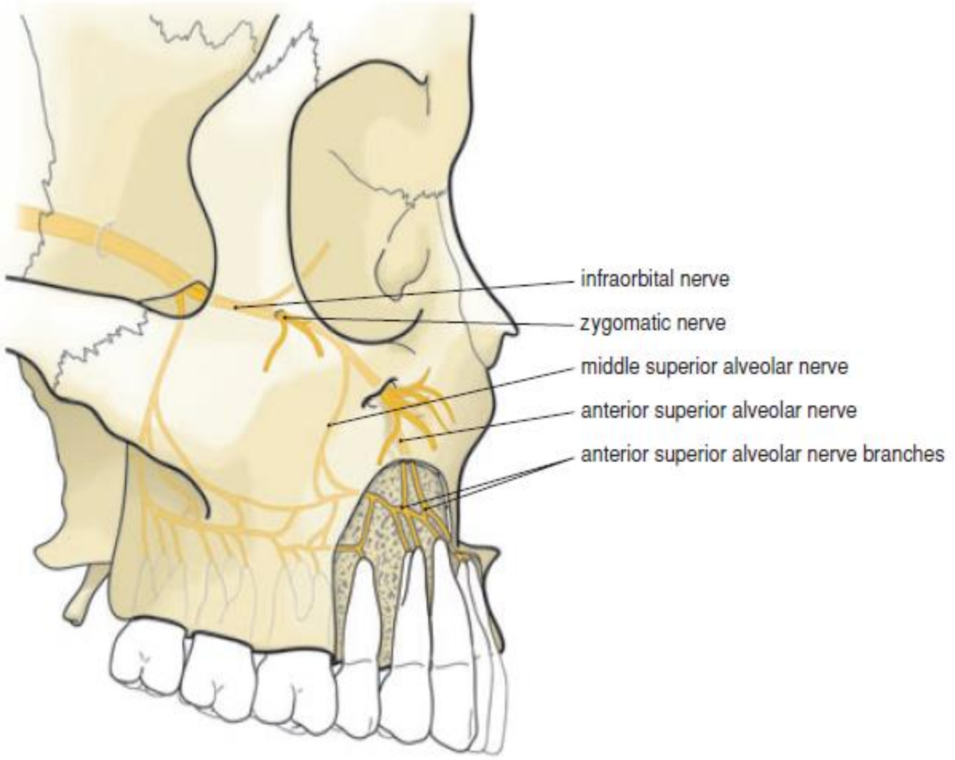
They anastomose with small branches of the other vestibular side .

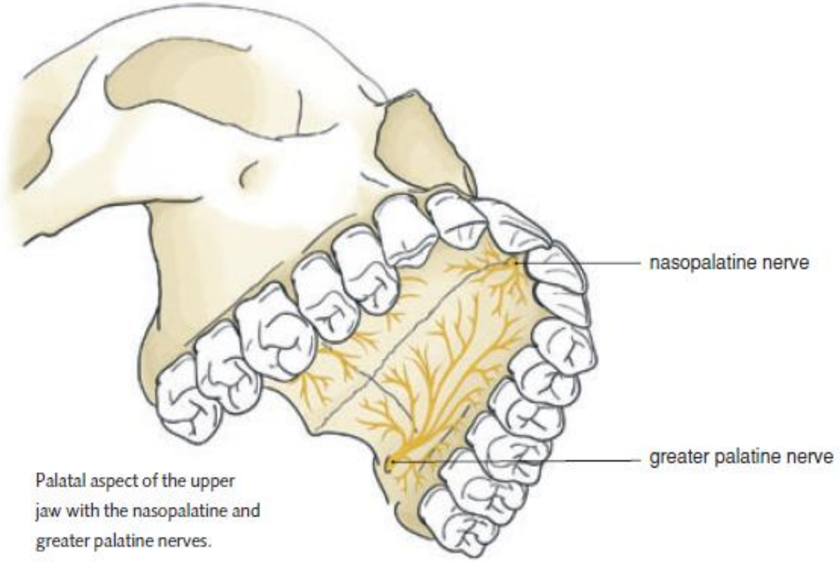
The nasopalatine nerve leaves the incisive foramen, and provides the sensory innervation of the palatine bone, periosteum and mucosa

Because of the relatively **thin** and **porous nature of the maxilla's cortical bone**, an **extraperiosteal (infiltration) anaesthetic can spread easily within the maxillary bone**.

The apices of the root of the central incisor and canine are found on the buccal side of the bone, **whilst the apex of the lateral incisor is found on the palatinal side**. This must be taken into consideration when infiltration anaesthetics are given, especially when used for an **apicoectomy**.

The anterior superior alveolar branches run from high lateral to low medial. For this reason, infiltration anaesthetics may best be applied laterally, just above the apex





Palatal aspect of the upper jaw with the nasopalatine and greater palatine nerves.

Indication:

For **cavity preparations** in the upper frontal teeth, buccal or labial infiltration anaesthesia is usually sufficient. T
he same applies to **endodontic treatments**. In cases where a **cofferdam** is used, or **wedges**,supplementary palatine anaesthesia is sometimes required.
For crown preparations, it is sensible to use **buccal and palatine infiltration anaesthesia**.
For surgical procedures in the upper frontal teeth area, such as **periodontal surgery**, **implants**, **extractions** and **apicoectomies**, it is advisable to anaesthetise a larger area using **regional block anaesthesiawith** supplementary **infiltration anaesthesia**.
Because regional block anaesthesia is **highly effective in this area**, it can be directly followed by infiltration anaesthesia.
The infraorbital and **nasopalatine nerves** can be **reached** via the **infraorbital foramen** and the **nasopalatine canal**.
Infiltration anaesthesia is given in the buccal area and, if necessary, in the **interdental (palatine) papillae**. Nevertheless, there are exceptions where **good anaesthesia** is not achieved.
The intraossal branches of the **nasopalatine nerve are responsible for this**. These smaller branches can be anaesthetised by an **injection or the application of a cotton bud with**
anaesthetic ointment in the respective nostril

A cotton bud, soaked with a topical anaesthetic, in the nose of a patient in order to numb the intraossal branches of the nasopalatine nerve.

Technique:

Buccal infiltration anaesthesia of the upper frontal teeth is performed by lifting the lip with the free hand, gently pinching the lip and then piercing the mucosa of the buccal fold with the needle, just above the apex of the respective tooth. The syringe is thereby held parallel to the longitudinal axis of the tooth. The needle is inserted no more than 3–5 mm. Any contact of the needle point with the periosteum or the bone must be avoided, and the fluid must be injected slowly.

Aspiration is recommended but **not really necessary**: there are no large blood vessels in this area

Palatine infiltration anaesthesia is applied in the palatal gingiva of the respective tooth.

This anaesthesia is particularly painful if the needle is moved up over the periosteum and when not injected extremely slowly.

It is, therefore, sensible to insert the needle tangentially and not to move it up, or to resort to palatine conduction anaesthesia for the central and lateral incisors There is usually enough space for an anaesthetic around the canines at the transition between the vertical and horizontal sections of the palate.

Here the space for injection fluid is maximum **sub- and supraperiostally.**

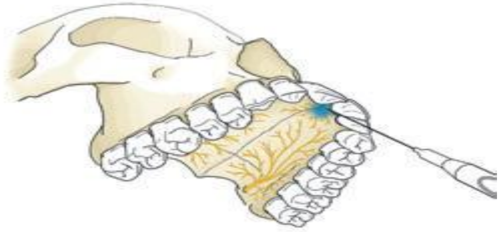
Here too it is necessary to inject extremely slowly.

The required amount of local anaesthetic is small, both for buccal and palatine anaesthesia.

For buccal anaesthesia a quarter of a cartridge per tooth is sufficient, whereas a maximum of an eighth of a cartridge is needed for palatine anaesthesia.

Infiltration anaesthesia for the I1 superior right.

Drawing (**A**) and photo (**B**) of regional block anaesthesia of the nasopalatine nerve. The needle is inserted at an angle into the incisive papilla to avoid damaging the nerve and bleeding from the vessels in the nasopalatine canal.

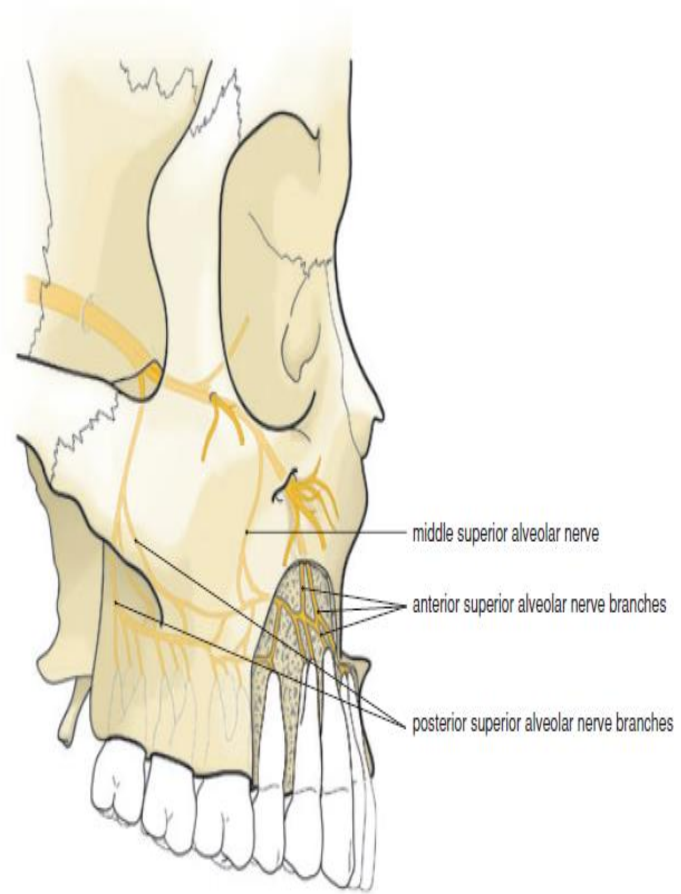


Sometimes it is necessary, in cases of **periodontal** or implant procedures, to inject anaesthetics into the interdental papillae, but this is painful for the patient.
The dentist
should, therefore, **wait until the vestibular and/or palatine infiltrationanaesthetic takes effec**t, before anaesthetising the interdental papillae.

Premolars:

Anatomical aspects

Sensory innervation of the **first and second premolars in the upper jaw** arises from the superior alveolar nerve, via the **middle and anterior superior alveolar branches**.
The middle superior alveolar branches run from high dorsal to low ventral, whereas the anterior superior alveolar branches come from the upper frontal area
Furthermore, because the lateral aspect of the upper jaw in this area is **concave**, the needle must be inserted a **fraction from out to in**, in order to **inject close to the periosteum**.



The facial artery runs near the first premolar, high in the buccal fold, so **aspiration prior to injection is indispensable**.

Innervation of the premolar area arises on the palatal side from branches of the **greater palatine nerve** and also from **smaller branches of the nasopalatine nerve that run dorsally**

Besides the branches of the **greater palatine nerve**, the greater palatine artery and vein also run along the palatinal side, at the transition from the vertical to the horizontal aspect. **An intravasal injection must be avoided by careful aspiration**.

Piercing the artery here also has the disadvantage of causing persistent bleeding from the needle hole. On the other hand, the injection must **not be too superficial**, since a high injective pressure is then required, which will result in a **lot of pain**, and **ischaemic necrosis of** the palatal mucosa may occur after treatment.

Indication:

Buccal infiltration anaesthesia is sufficient for cavity preparations and endodontic treatments. If the first premolar of the upper jaw (P1sup) has two **diverging roots**, additional **palatal anaesthesia may be needed**.

This supplementary **palatal anaesthesia** is also needed for crown preparations.

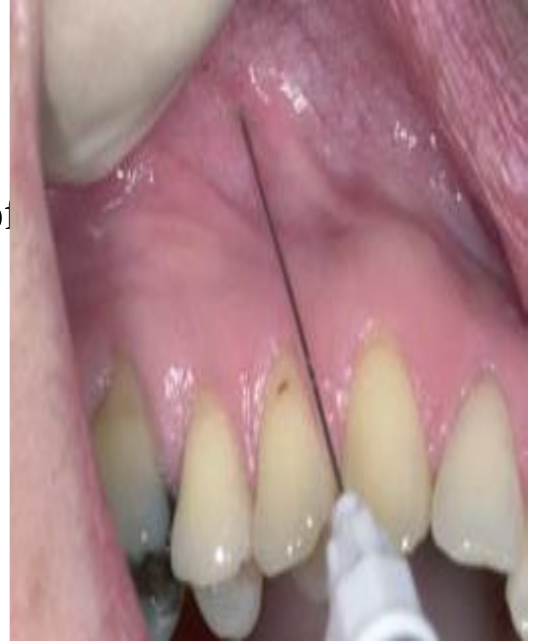
For surgical operations, such as periodontal surgery, implantology, extraction and apicoectomy, a larger anaesthetised area is required. For this reason, the anaesthetic is injected into the **vestibular area** at a point much higher than the apices, and in the **palatinal area the anaesthetic is injected near the apices at the point of transition from the horizontal to the vertical aspec**t.

Though palatine block anaesthesia is possible, it is discouraged as it requires two injections: one into the greater palatine foramen and another into the incisive papilla.

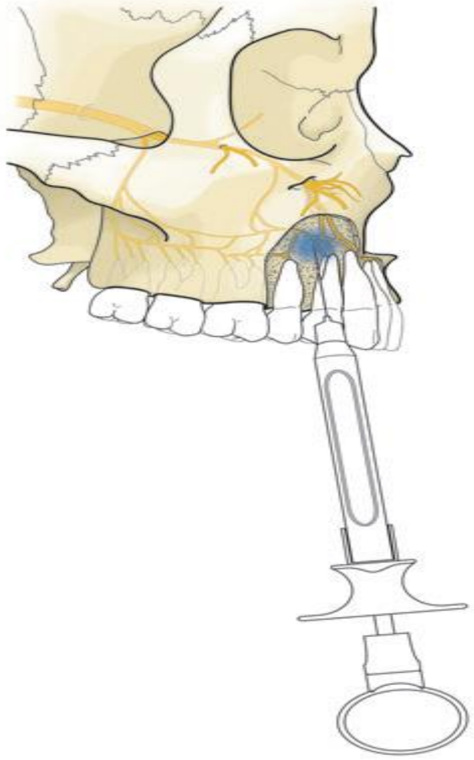
Technique:

In the upper jaw the transversal width of the alveolar process is narrowest in the area of the canines and rapidly increases in a dorsal direction. The apices of the roots of the first premolar lie, when they are bi-rooted and divergent, immediately below the buccal and palatal cortical bone, respectively.

The single-rooted second premolar has an apex that lies more centrally in the alveolar process.
This must be kept in mind when a local anaesthetic is applied here.
The corner of the patient's mouth is lifted and the free hand should pinch the lip carefully, so that the needle's penetration into the buccal mucosa is hardly felt. With the point of the needle a small amount of anaesthetic fluid is deposited just above and dorsal to the apex



Aspiration for injection near the first premolar must be carried out in order to **avoid an intravasal injection**.
If the fluid is **injected intravasally** the patient will **feel a short**, sharp shot of pain in the face, and the **skin of the cheek** and **lower eyelid will pale immediately (*blanching*).**
The needle must be **inserted from out to in**.
For restorative dental treatments the needle point **should be approx. 5 mm above the apex**.
For surgical procedures a more **cranial infiltration anaesthesia is required**.
On the palatal side, the needle is inserted counter-laterally and vertically at the transition of the horizontal to the vertical aspect of the palate
After aspiration, the fluid is **injected extremely slowly**. The amount of anaesthetic fluid used in the buccal area is approx. 1 ml and a maximum of 0.25 ml is used for the palatal side.



Molars:

Anatomical aspects

The posterior superior alveolar branches innervate the buccal side of the molar region of the upper jaw.
The source of these branches of the second trunk of the trigeminal nerve is high in the pterygopalatine fossa and they run along the maxillary tuberosity to the low ventral area.

The zygomatic buttress is found in the buccal area above the apices of the M1. The point of attachment can vary, however, so that an impermeable cortical layer of bone may sometimes be found lateral to the buccal roots of M1, depending on the length of the vestibulary radices and the crest's point of attachment.

Palatine infiltration anaesthesia of the P1 and P2 superior right. The needle is inserted from the left into the transitional area of the horizontal to vertical sections of the palate

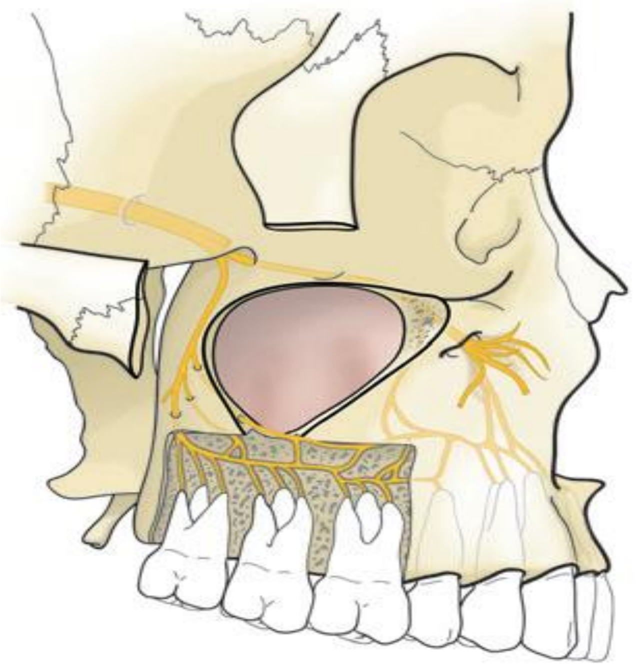
The position of the roots of the M2 and the erupted M3sup is more or less central in the bone, depending on the level of convergence. The transversal width of the alveolar process in the molar region is considerable, so that more anaesthetic fluid is needed for adequate numbing.

The pterygoid venous plexus is found laterally high to the maxillary tuberosity.

Indication:

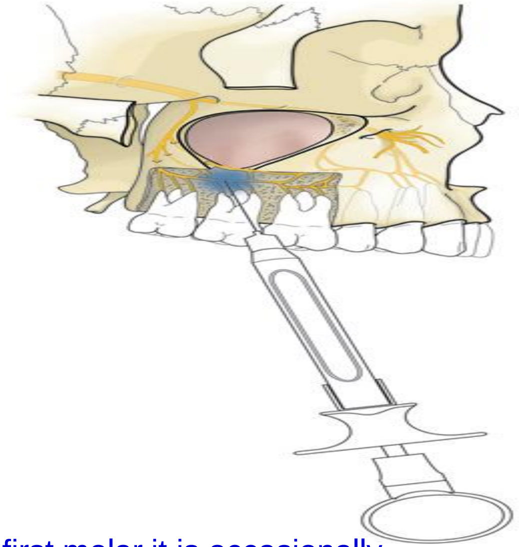
For cavity preparations in the M1sup both buccal and palatal infiltration anaesthesia is required. For the second and third molar in the upper jaw, vestibulary anaesthesia will suffice for these indications. The same applies with regard to endodontal treatment of this area. If the M1sup happens to have long buccal roots and/or a low-positioned zygomatic buttress, the infiltration anaesthesia must be applied behind the crest, i.e. higher and more dorsal.

For operative treatments such as **periodontal surgery**, **implantology**,**extraction** or **apicoectomy** (of the buccal roots), **regional block anaesthesia** is commonly used high above the maxillary tuberosity and at the position of the greater palatine foramen, supplemented with some buccal infiltration anaesthesia.



The course of the superior alveolar nerve and the posterior superior alveolar branches.

The method of high tuberosity anaesthesia is described in Section
Regional block anaesthesia of the major palatine nerve is administered vertically from the counter-lateral corner of the mouth.
The needle must not be inserted too deeply in the direction of the foramen in order to avoid damage to the nerve or piercing the artery. **Aspiration prior** to the actual injection is prescribed.
The amount of anaesthetic fluid for buccal infiltration anaesthesia should be approx. 1–1.5 ml. For palatine infiltration or block anaesthesia, no more than 0.25 ml is required.



Patients that are given an infiltration anaesthetic in the molar region of the upper jaw can have the impression that the anaesthetic has not worked. They compare the feeling they observe with the sensation that they know from an infiltration anaesthetic in the premolar region or the front teeth or from mandibular regional block anaesthesia. It is therefore wise to explain to patients this difference in sensation.

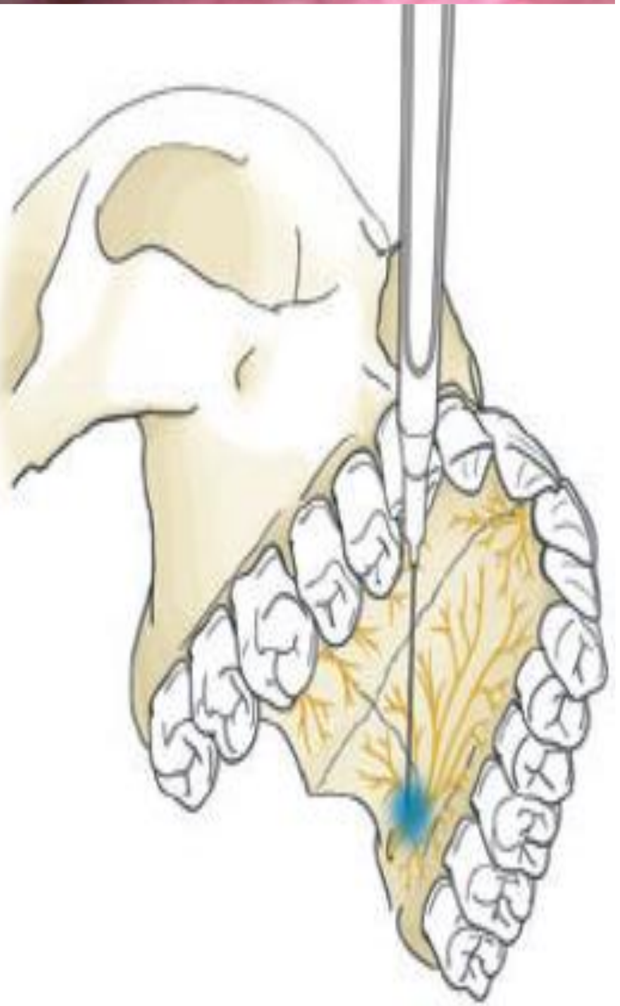
Technique

Infiltration anaesthesia on the buccal side of the upper molars is applied,at an angle from the front. The jaw here is flat to convex.
The zygomatic buttress sticks outwards and is found **near the first molar**. The point of the needle should be inserted right above and **dorsal to the apices**

For the first molar it is occasionally necessary to inject behind the crest and slightly higher, due to the inaccessibility of the buccal roots through the thick cortical bone. For the palatal side, infiltration anaesthesia of the major palatine nerve at the first molar, and regional block anaesthesia for the second and third molar, are sufficient

Drawing (**A**) and photo (**B**) of the palatal regional block anaesthesia of the greater palatine nerve. Careful aspiration prior to injection is required!

For an extraction of the second and erupted third molar, an injection of 0.25 ml fluid next to the gingival fold on the palatal,



Palatal infiltration anaesthesia of the gingiva on the level of the M2 superior right

The impacted third molar of the upper jaw

Anatomical aspects:

The impacted third molar of the upper jaw (M3sup) is usually found completely **in the maxillary tuberosity** with a slight **distovestibular inclination**.

Here, the buccal cortical bone is very thin. Slightly higher and in a more lateral position is the pterygoid venous plexus.

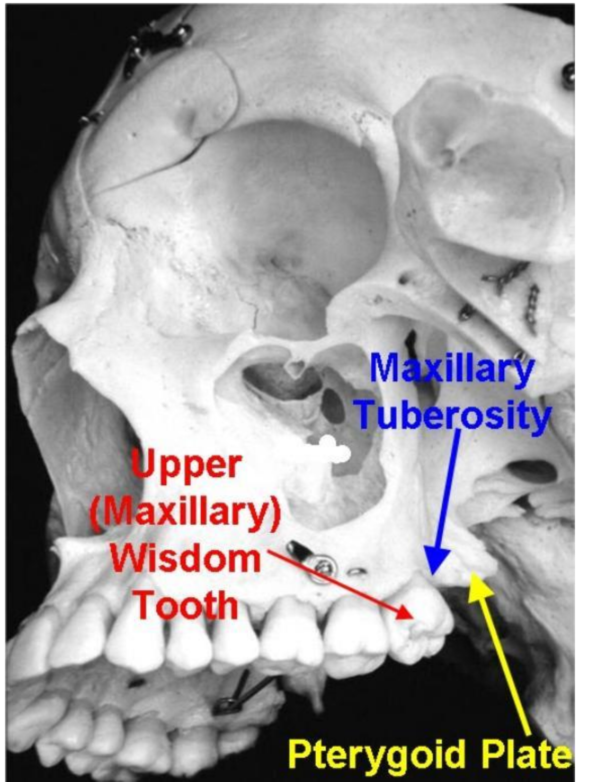
The branches of the second trunk of the trigeminal nerve, and the maxillary artery and vein, run **behind the tuberosity** and **higher in the pterygopalatine fossa**.

On the **palatine side** of the impacted M3sup are the greater palatine foramen and the lesser palatine foramina, from which the palatine nerves branch off that innervate the palatine gingiva and the soft palate. The greater palatine arteries and vein also come from the greater palatine foramen.

Therefore, the area **lateral**, **dorsal** and **medial** to the impacted M3sup is richly **innervated** and **vascularised**. **Here surgery outside of the periosteum is risky**.

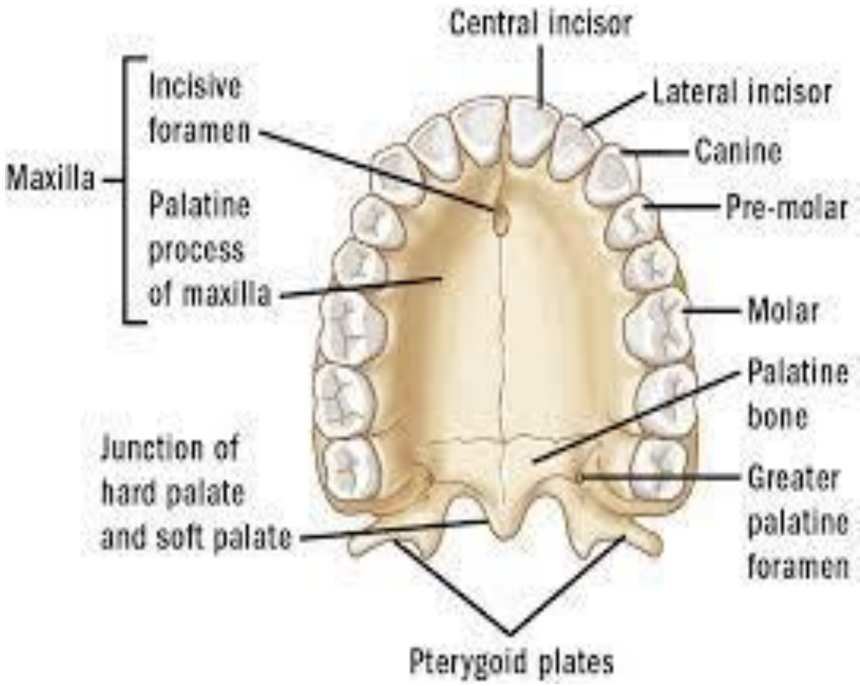
Indication:

In dental practice, local anaesthesia will only be used in this area for the removal of an impacted M3sup or for harvesting bone for pre-implant treatment elsewhere in the mouth.

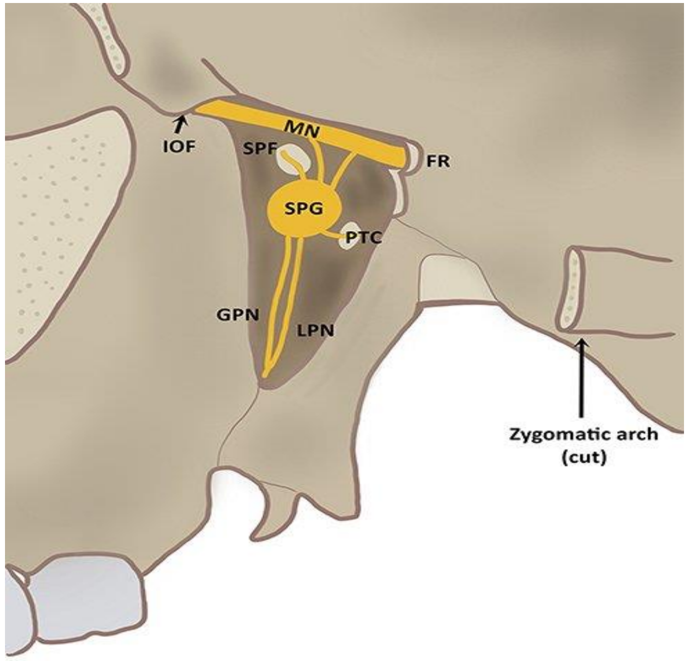


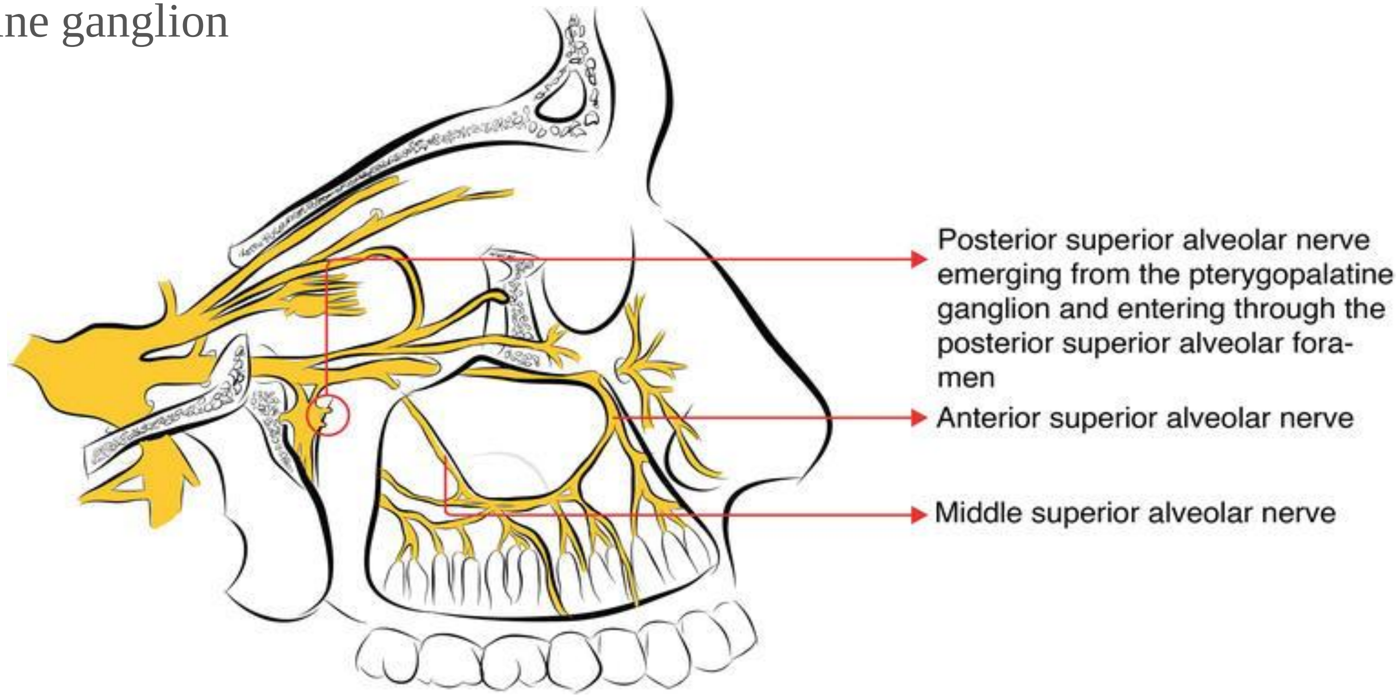
Technique:

Anaesthesia of the entire greater palatine nerve and, if necessary, of the lesser palatine nerves is performed with regional block anaesthesia.
The needle is brought forwards from the counter-lateral corner of the mouth in the direction of the **greater and lesser palatine foramens**.
Touching the nerve or piercing the artery must be avoided.
For this **reason aspiration** is necessary. Approximately 0.25 ml anaesthetic fluid is Sufficient in this area .



Schematic illustration of the pterygopalatine fossa.
Abbreviations: FR, **foramen rotundum**; GPN, **greater palatine nerve**; IOF, **inferior orbital fissure**; LPN, **lesser palatine nerve**; MN, **maxillary nerve**; PTC, **pterygoid canal**; SPF, **sphenopalatine foramen**; SPG, sphenopalatine ganglion





Maxillary nerve block

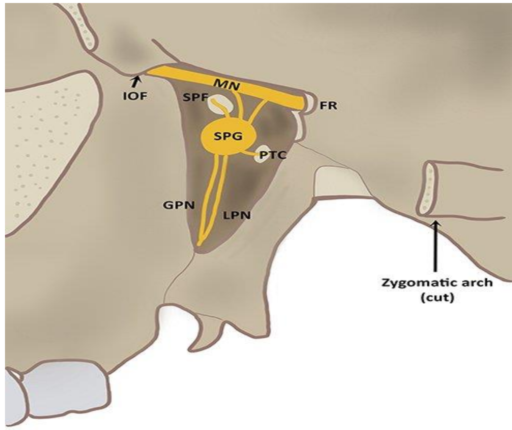
Blockade of the maxillary nerve **induces anaesthesia of half of the maxilla**, which enables **surgical treatment in the upper jaw** and **maxillary sinus under local anaesthesia.** This regional block can also be used to counteract pain in cases of **inexplicable pain complaints**. A maxillary nerve block can be achieved **via high tuberosity anaesthesia** or via the **greater palatine foramen**. A local anaesthetic with vasoconstrictor is **used**, applied with anaspirating syringe and a **25-gauge needle (bent at approximately 45 degrees).**

High tuberosity anaesthesia

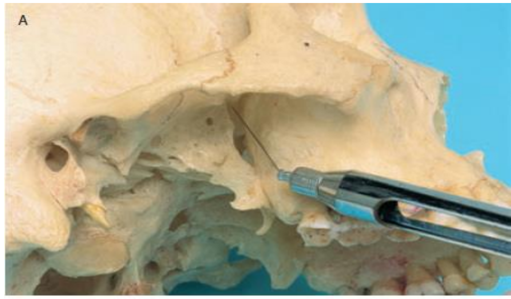
The maxillary nerve leaves the skull through the **foramen rotundum**.The nerve runs through the **pterygopalatine fossa** and then **on through the orbit as the infraorbital nerve**. The infraorbital nerve runs through a canal at the bottom of the orbit and leaves the canal again through the infraorbital foramen.

The pterygopalatine fossa is accessible from the mouth. This fossa can be reached with a **35-mm-long 25-gauge needle** if the needle is bent to **approximately 45 degrees**. This bent needle must **pass the mucosa behind the zygomaticoalveolar crest**, about 1 cm from the **alveolar proc**ess, and then be **directed dorso-medially .**

After aspiration, about one cartridge may be injected. **Two to three minutes later, half of the upper jaw** will be anaesthetised. In some cases the infraorbital nerve is insufficiently numbed by this technique. Furthermore, there is a chance of injecting intothe pterygoid plexus, so that there is a risk of intravasal injection and developing a haematoma.



Photos of skull (**A**) and patient (**B**) show a high tuberosity anaesthesia. The (bent) needle is inserted from out to in, high in the pterygopalatine fossa, just by the split of the maxillary nerve into the infraorbital and superior alveolar nerves. The injection is given after aspiration.



Greater palatine foramen block

A greater palatine foramen block is used less frequently than high **tuberosity anaesthesia**, but can be easily carried **out intraorally**. The greater palatine foramen lies approximately **1 cm palatinally to the M2–M3.** region and **approximately 0.5 cm in front of the pterygoid hamulus. The** direction of the canal is 45 degrees to dorsal in relation to the occlusion plane. The bent needle is carefully inserted into the foramen, and by inserting the needle slowly, the entire length of the needle can be used. **After aspiration**, a half to one cartridge may be injected. Within 2–3 minutes, **half of the maxilla will be anaesthetised.**

It is not always easy to find the **entrance to the foramen**. Moreover, inserting the needle roughly can lead to **long-term damage of the nerve**. **Finally,** if the patient has a small maxilla, the anaesthetic fluid may reach the **parasympathetic sphenopalatine ganglion** so that unintended side effects may occur, such as **diplopia (double vision)**

Infraorbital nerve block

The infraorbital nerve runs almost horizontally through the canal in the orbital floor until it leaves through the infraorbital foramen, approximately 5–10 mm caudally to the infraorbital rim. The nerve supplies sensibility to the nostril, cheek, lower eyelid, upper lip, gingiva and upper frontal teeth. An infraorbital nerve block is suitable for the dental treatment and surgery of frontal teeth.

Nasopalatine nerve block

The nasopalatine nerve, which runs over the floor of the nose through the incisor canal to the incisive papilla, provides sensibility to the anterior third of the palate. Anaesthesia of this nerve is appropriate for crown preparations in the entire upper front and for surgical treatment, such as ligating or removing an impacted canine, periodontal surgery and implantology.

An anaesthetic with a vasoconstrictor is administered with a regular cartridge syringe with a 25-gauge needle at least 20 mm long. With the mouth open, the needle point is placed right on the incisive papilla. The needle is introduced slowly, parallel to the direction of the buccal cortical bone contour .This is almost vertical in some patients; for others it is inclined dorsally. **The direction is important to avoid the needle getting stuck in the canal or having to be reinserted because it can no longer follow the canal. After approximately 1 cm a quarter cartridge is injected very slowly**

This injection technique is painful, even in expert hands. With explanation, precision and expertise, however, this anaesthesia can be used very successfully with children, e.g. for ligating an impacted canine or removing a mesiodens. **If the nerve is damaged, an anaesthetised area may arise at the anterior of the palate durum and last for 3–4 months**

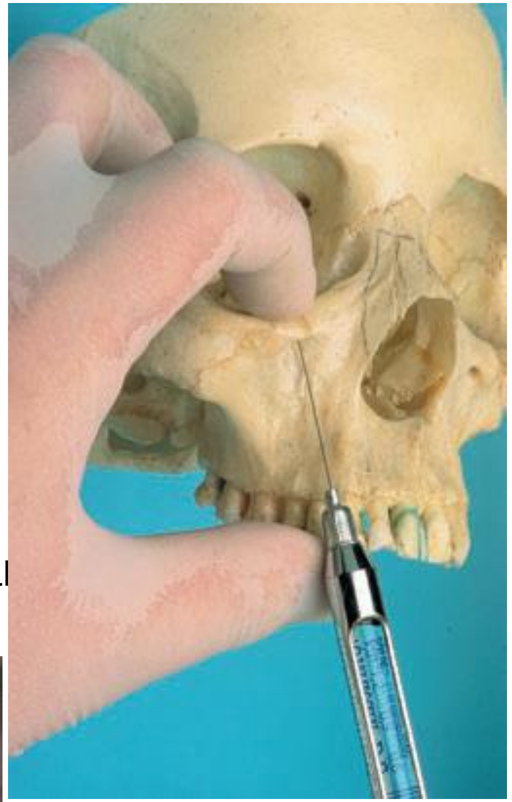

Photo of skull (**A**) and photo of patient (**B**) show block anaesthesia of the nasopalatine nerve. The needle is inserted upright into the incisive papilla and introduced carefully for approx. 1 cm into the nasopalatine canal. This runs parallel to the axis direction of the central incisor. The injection is given after aspiration.

A vasoconstrictor containing anaesthetic is used for this block, applied with a customary cartridge syringe with a 25-gauge needle of 25–35 mm. There are two intraoral methods for **blocking the infraorbital nerve**. The first involves the needle being positioned approximately 0.5 cm laterally from P2sup, whilst the other method involves the needle being inserted approximately 1 cm from the alveolar process of Csup. The lip is lifted with the thumb, and the index finger of the same hand feels the infraorbital rim extraorally. The needle is then moved in the direction of the finger. With the method in which the needle is inserted in the buccal sulcus at the level of the Csup, the needle is directed towards the pupil of the eye .With the 'P2sup method', the needle is inserted in the direction of the longitudinal axis of this tooth. After about 2 cm the needle will make contact with the bone at the level of the infraorbital foramen.

The unaltered position of the index finger prevents the needle from being fed in so far that it touches the eyelid or eyeball. **A depot of half a cartridge is enough.**

The method is simple, effective and safe. However, if a vein or small artery is damaged, a hematoma may occur directly under the eyelid, and touching the nerve with the needle leads to prolonged anesthesia and paraesthesia. bone contour .

This is almost vertical in some patients; for others it is inclined dorsally. The direction is important to avoid the needle getting stuck in the canal or having to be reinserted because it car no longer follow the canal. After approximately 1 cm a quarter cartridge is injected very slowly.



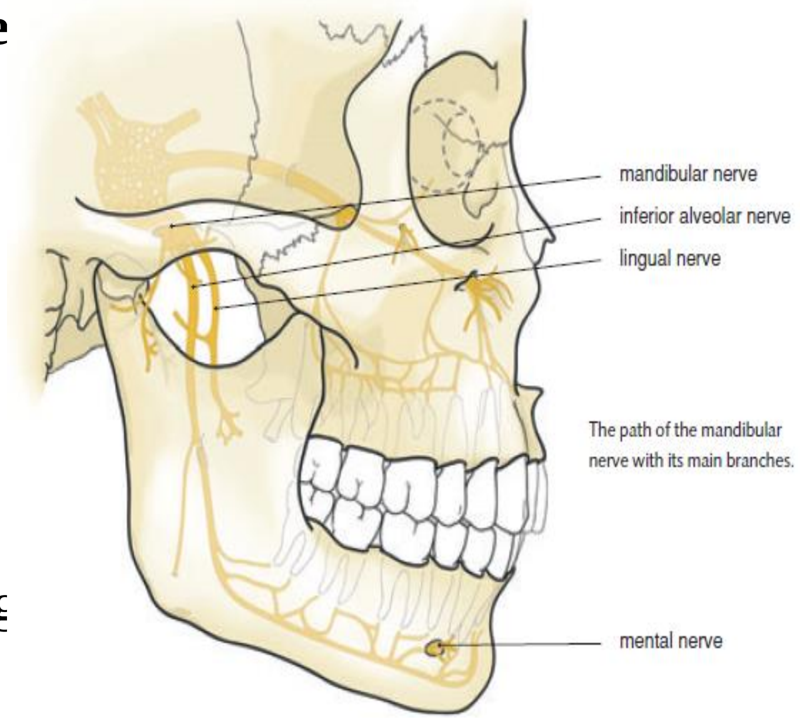
Local anaesthesia in the lower jaw

Introduction

The buccal cortical bone at the premolars and molars of the lower jaw impedes the diffusion of anaesthetic fluid to the apices of these teeth, located centrally in the jaw bone. Adults require mandibular block anaesthesia for an effective anaesthesia. In the area of the lower canines and incisors the cortical bone is thinner and the roots lie on the buccal side of the jaw. Here, infiltration anaesthesia is effective.

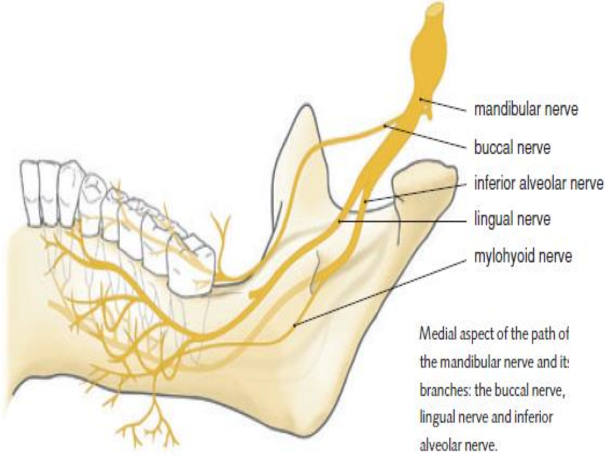
The mental nerve leaves the jaw through the mental foramen and innervates the buccal mucosa and gingiva, the lower lip and the skin of the chin. Therefore, anaesthetising the mental nerve will not anaesthetize the teeth in adults. However, in **children the molars are anaesthetised,because the apex of these through the thinner cortical bone.**

The lingual side of the mandible is innervated by the lingual nerve.
This nerve can be anaesthetised both by **block anaesthesia** as well as by **infiltration anaesthesia**.
The dentist must avoid pricking the floor of the mouth too often as this **increases the risk of a haematoma** in combination with **transport of bacteria via the injection needle.**
This may cause a **phlegmonous infection of the mouth floor**, a life-threatening complication.



The path of the mandibular nerve with its main branches.

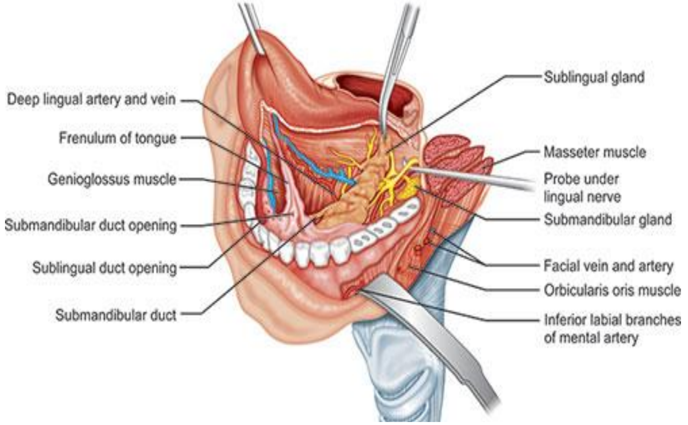
Block anaesthesia of the buccal nerve is possible. This nerve runs from high lingual and crosses the front side of the mandibular ramus above the occlusion plane. Then the buccal nerve continues caudo-ventrally to innervate the buccal mucosa and gingiva in the area of the (erupted) M3inf to P2inf.
Because the height at which the buccal nerve crosses the mandibula varies, **infiltration anaesthesia applied buccal to the respective teeth** is also an **excellent technique to anaesthetise the gingiva and mucosa**



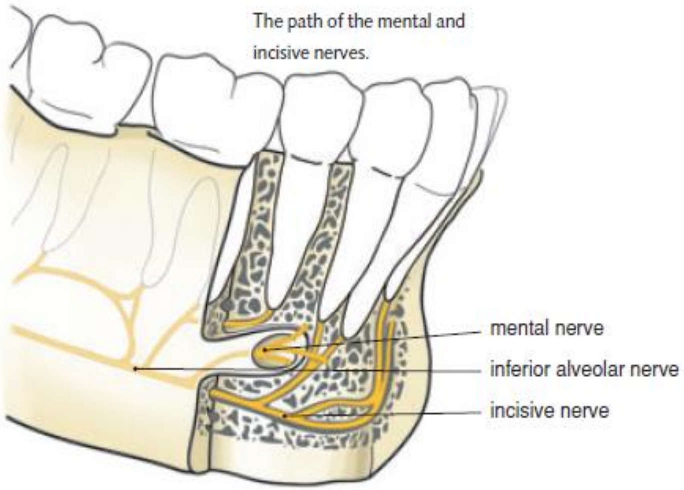
Incisors and canines:

Anatomical aspects

Once the alveolar nerve has separated from the trigeminal nerve, the nerve runs laterally and enters the mandibular foramen. The inferior alveolar nerve divides into a branch, the mental nerve, at the mental foramen and then continues as the incisive nerve. The incisive nerve no longer runs in a bony canal and divides into little branches leading to the roots of the lower canines and incisors (Figure 6.3). In the mandibular symphysis area, sensory anastomoses from the contralateral side are present, both lingually and buccally.
This must be taken into account, particularly with extensive surgical treatment in the lower frontal area.



The roots of the lower incisors and canines are found against the buccal cortical bone. The mental muscle is attached to the jaw at the height of the I2inf so that infiltration anaesthesia in this area can be painful and less effective

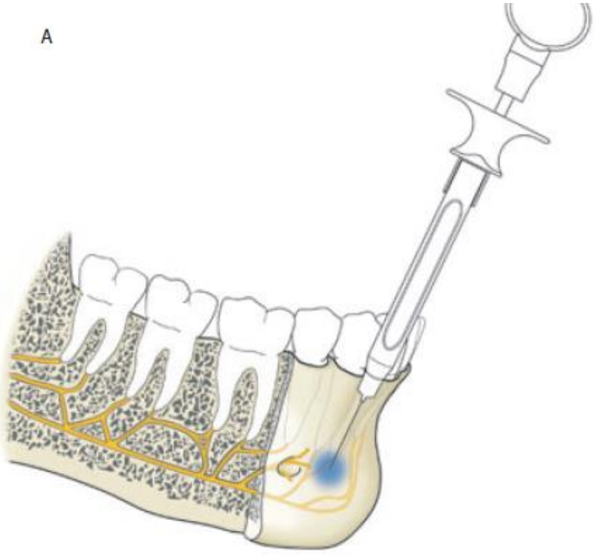


Indication

Buccal infiltration anaesthesia will be **sufficient for cavity preparations** and **endodontic treatment** in the lower frontal area, ***unless*** a **cofferdam** and/or **matrix band** and **wedges** are used.
In that case, it is necessary to **apply additional anaesthesia lingually**, or **perhaps in the interdental papilla on the lingual side**.

Infiltration anaesthesia is also used for **surgical treatment** in the lower frontal area, in which case the needle is inserted more caudally to the apex.
The dentist must take the sensory anastamoses into account by also anaesthetising the contralateral nerve branches.

The point of attachment of the mimic muscles, such as the orbicularis oris muscle and the mental muscle, also requires special attention during anaesthesia. Injecting into these muscles causes bleeding, is painful and does not lead to a good anaesthesia



A and B Drawing (**A**) and photo (**B**) of infiltration anaesthesia of the **I2 inferior ri**ght.
The buccal gingiva is also anaesthetised.
In order to anaesthetise the lingual gingiva, it is necessary to inject into the floor of the mouth.

Technique:

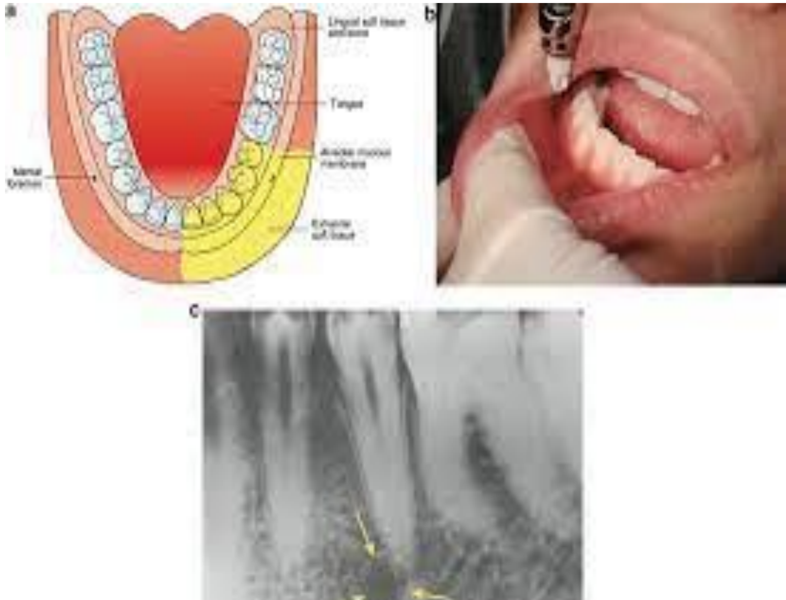
For infiltration anaesthesia in the lower frontal area, the non-injecting hand pulls the lip forwards and pinches the lip softly at the moment the needle penetrates the mucosa. The needle is inserted right under the apex of the tooth that is to be anaesthetised, up to the bone. Preferably, the needle isinserted vertically and not pushed into the periosteum. The dentist sits or stands behind the patient in an **11–1 o'clock position**.

The same position is assumed for infiltration anaesthesia of the lingual mucosa and gingiva. When anaesthetising from an **8–9 o'clock position**, it is better to bend the needle **45–90** degrees for a lingual injection but a disadvantage of this is that the needle cannot be returned to its protective cap. With lingual anaesthesia it is important to prevent a haematoma occurring.

Bilateral mandibular block anaesthesia, with additional **local infiltration anaesthesia**, is recommended for surgical treatment of the **lower front**, such as **extensive pre-implantological treatments** (e.g. a chin bone transplant) and **implantological treatments** (two or four dental implants). This reduces the required number of injections to a minimum, as well as the chance of haematomas and infections. Furthermore, a maximum of 4–6 ml of anaesthetic fluid will be sufficient.

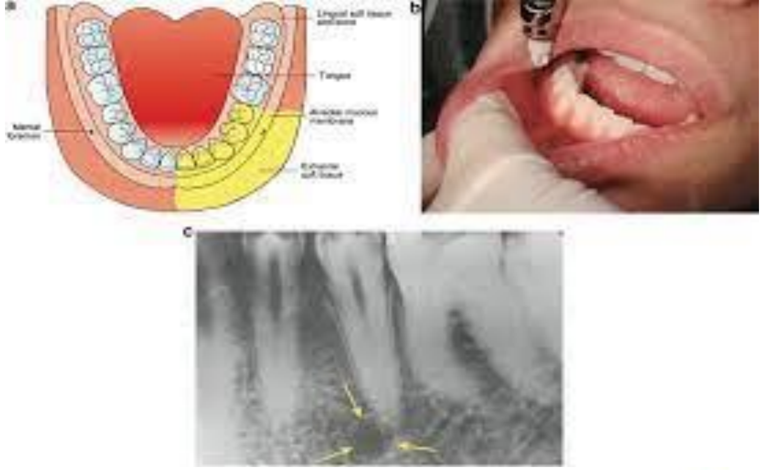
Lingual anaesthesia with a bent needle with the dentist in an 8 to 9 o'clock position.

Lingual anaesthesia with a non-bent needle with the dentist in an 11 to 1 o'clock position.



In this situation it is recommended that the dentist administers the double-sided mandibular block first and then waits until the patient spontaneously indicates that the lower lip and border of the tongue have started to tingle.

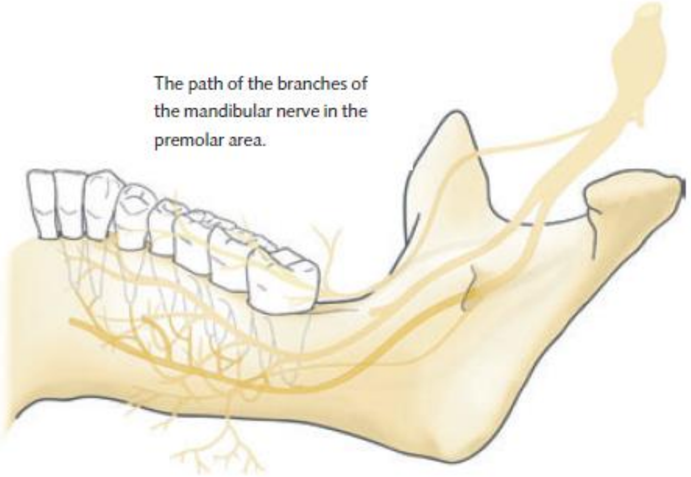
The dentist can then give a buccal infiltration anaesthesia.



Premolars:

Anatomical aspects

Innervation of the premolars in the lower jaw takes place in the bone through the inferior alveolar nerve, buccal through the buccal nerve (at the P1inf also through the mental nerve) and lingually through the lingual nerve.



The path of the branches of the mandibular nerve in the premolar area.

Because of the **thickness** and **impermeability of the buccal cortical bone**, infiltration anaesthesia is not really possible for the treatment of the (pulpa of) **premolars in adult patients.**

Use of block anaesthesia of the mandibular nerve, i.e. of the inferior alveolar and lingual nerves, is therefore a more obvious choice, if necessary supplemented with local infiltration anaesthesia of the branches of the buccal nerve

The mental nerve is found lower than the apices, exactly between the two premolars. Block anaesthesia of the mental nerve branches must be given superficially to avoid damage to the mental nerve, with subsequent long-term anaesthesia of the lower lip half.

Buccal infiltration anaesthesia at the level of the M_1 inferior right, for anaesthetising the buccal nerve branches.

If a combination of block anaesthesia of the **mandibular nerve** and **local infiltration is chosen**, then the mandibular block must, of course, be given first.
A supplementary infiltration anaesthetic is applied locally once the **patient spontaneously reports that the lower lip** half and edge of the **tongue have begun to tingle.**
When the inferior alveolar nerve appears to be anaesthetised after block anaesthesia of the mandibular nerve, but the lingual nerve does not, then **lingual infiltration anaesthesia at the level of the respective premolar will be sufficient**.
Generally, a repeated mandibular block injection should be avoided, because the partial anaesthesia that has already set in will limit the patient's ability to give a **warning if the lingual** or **inferior alveolar nerve is touched by the needle.**

Indication:

Mandibular block anaesthesia is used for cavity preparation and endodontic treatment, if necessary supplemented by buccal infiltration anaesthesia (Figure 6.6). It should also be used for extensive surgical treatments such as periodontal surgery, implantology, extraction and
apicoectomy, where the buccal infiltration anaesthesia is more extensive and more caudal. Extra attention to the path of the mental nerve is required here. **When giving additional anaesthesia, it is most undesirable** to damage this nerve by **pricking it accidentally.**

Technical aspects:

Only a single injection is required to anaesthetise the mandibular nerve, i.e. the inferior alveolar and lingual nerve. An aspirating cartridge syringe is used with a 25-gauge needle that is 35 mm long.

When anaesthetising the mandibular nerve, the point of the needle is placed in **the buccal infiltration anaesthesia at the level of the M1 inferior right**, for anaesthetising the buccal nerve branches.

Pterygomandibular space. :

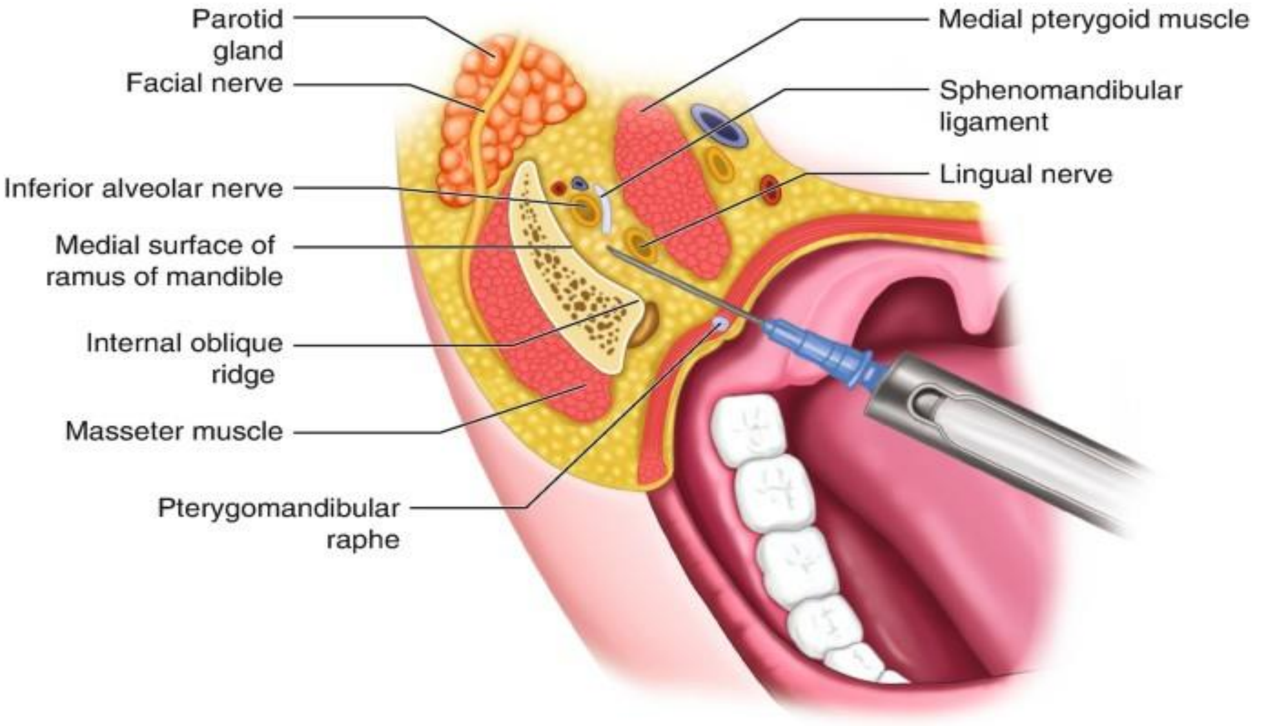
This space is bordered ventrally by the mucosa of the pharyngeal arches.

This is where the **injection needle** is inserted in the **pterygomandibular space**.

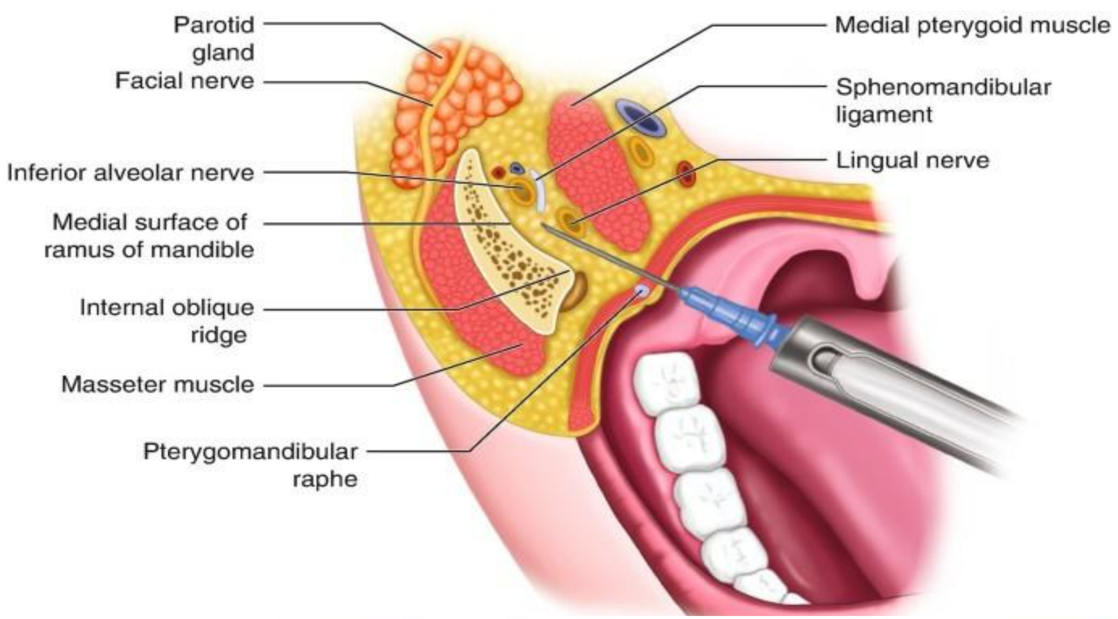
This space is **bordered laterally by the ascending branch of the lower jaw** and **dorsally by the median part of the parotid gland** and the **skin**.

The attachment of the medial pterygoid muscle is found caudally and also borders the space median. The lateral pterygoid muscle borders the space cranially.

Lingual aspect of an adult mandibula with the lingula exactly in the middle between the front and back sides.



The mandibular nerve, **inferior alveolar nerve**, **lingual nerve**, **buccal nerve** and **branches of the arteries** and **maxillary veins run within the space**.
The space runs ventro-caudally into the submandibular space and caudo-cranially into the parapharyngeal and retropharyngeal spaces, which eventually lead to the mediastinum and pericardium.



The direct and indirect technique

The inside of the mandibular ramus is more or less divergent dorsally. Therefore, various techniques can be used to anaesthetise the mandibular nerve (‘**mandibular block**’).
A distinction must be made between the **so-called direct** and **indirect technique.**

The direct technique is performed from the **homolateral side**. The risk of this is that the **anaesthetic fluid may be applied too far medially**. This has led to the indirect technique, which is performed from the **contralateral commissure**.
The danger of this is that the **medial pterygoid muscle** may be **damaged** or **anaesthetic fluid** may be injected into this muscle. **This would lead to postoperative trismus**, which may persist for days or weeks, or to a **haematoma**.

The indirect technique

The direct technique

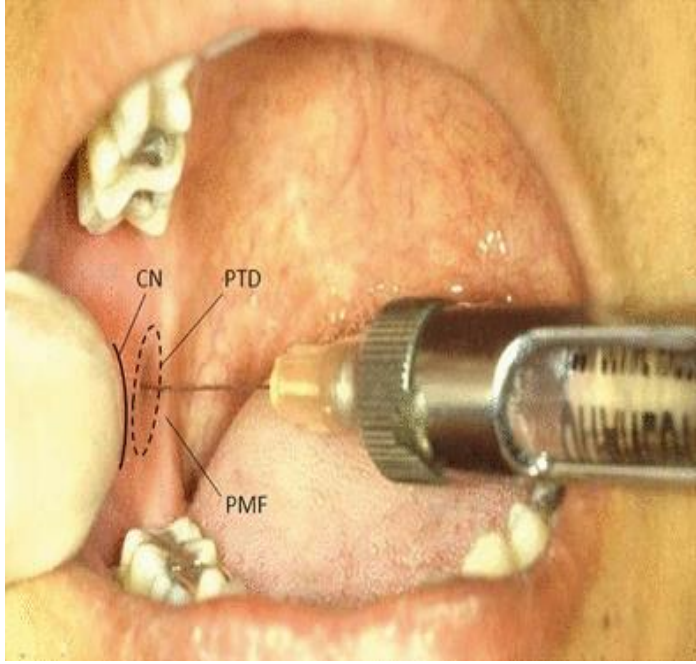
The direct technique

does not have these risks, but lacks the relative certainty of a truly effective anaesthesia. For administration of anaesthesia with either the direct or the indirect technique, a number of characteristic anatomical structures are of great importance. The following anatomical structures determine the place,

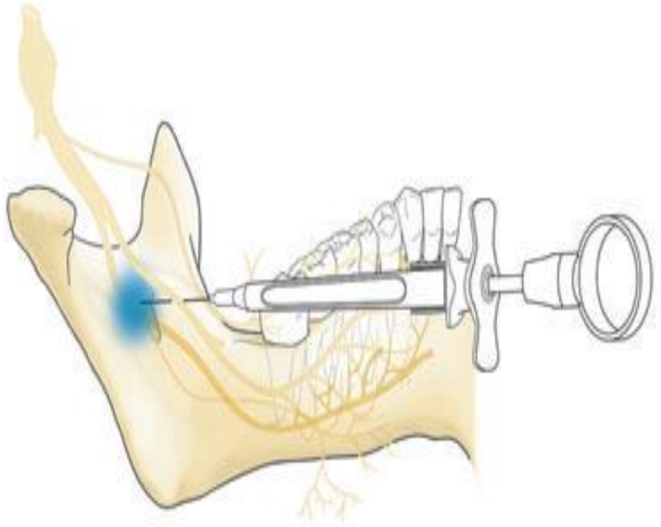
direction and penetration depth of the needle:

- ***The plane of occlusion.*** Anaesthesia is given parallel to the plane of occlusion, approx. one finger width above (1–1.5 cm).
- ***The deepest point of the front of the mandibular ramus*.** If the noninjecting hand feels along the front of the ramus, the entrance of the mandibular canal appears to be on the same level as the deepest point.
- ***The triangle at the front of the pterygomandibular space*** that is formed by the cheek mucosa that runs into the throat and pterygomandibular plica running from the palate to the retromolar pad. The needle must be inserted in the middle of this triangle.
- ***The thumb of the non-injecting hand feels along the front of the ramus,*** while **three fingers of the same hand feel along the back of the ramus**.

Exactly halfway between the **thumb** and **fingers** is the ***mandibular foramen***, where the alveolar nerve enters the jaw. In adults the foramen is found exactly in between the front and back. **In children the foramen** lies about a **third in from the front**. This determines the depth of the needle's insertion.



Mandibular block anaesthesia. The drawing shows the position of the needle point in relation to the mandibular foramen.



With the *indirect* technique the anaesthesia is given from the contralateral commissure and the syringe is moved to the middle of the mouth opening while the needle is pushed in. With the *direct technique* the anaesthesia is given from the homolateral corner of the mouth and the syringe is moved to the middle of the mouth opening if necessary while the needle is inserted.

Whilst inserting the needle, the dentist must attempt to move the needle as close to the bone as possible without touching the periosteum unnecessarily.

It is also important to hold the tip of the needle in such a way that it does not get stuck in the periosteum.

The thumb of the non-injecting hand seeks for the deepest point of the right ramus and the fingers feel along the back of it. **The needle is held parallel to the plane of occlusion, about one finger width above it.**

The needle point is then **inserted in the middle of the mucosa triangle until it makes contact with the bone**. Following this, the needle is carefully pushed up by moving the syringe to the middle of the oral cavity and holding it **parallel to the plane of occlusion.** The level of divergence inside the mandibular ramus **differs from person to person**.

When 1 cm of the needle still remains visible and the point is exactly in the middle between the front and back of the ramus, the dentist carefully aspirates. Approximately 1.5 ml of anaesthetic fluid is injected .

The needle is then pulled back about 1 cm and the dentist aspirates once again.

The technique of mandibular block anaesthesia from the left corner of the mouth (indirect technique). The needle is inserted to the bone, pulled back a millimetre and then the syringe is moved to the middle of the mouth and carefully pushed in so that about 1 cm of the needle remains visible. The injection is given after aspiration

Failing of the lingula is the most common reason for an ineffective mandibular block. The point of the injection needle is then found to be too far medially, too low or too far dorsally. Other complications may also occur. **In approximately 15% of cases, blood is aspirated**. It is also possible for the **needle to touch the lingual nerve** or the **inferior alveolar nerve.**

In the case of **positive aspiration** or when a nerve has been touched, it is enough to pull the needle back a **few millimetres**. If the needle is in **too deep**, this can lead to local anaesthesia within the **capsule of the parotid** gland.

The technique of mandibular block anaesthesia from the left corner of the mouth (indirect technique). The needle is inserted to the bone,pulled back a millimetre and then the syringe is moved to the middle of the mouth and carefully pushed in so that about 1 cm of the needle remains visible. The

This may result in one-sided paralysis of the facial nerve, which fortunately lasts only a few hours.

When the **mandibular block** is not effective, **another injection** may be given. There is a chance that, during the **second injection**, the **patient will not notice a touching of the lingual nerve**, the **inferior alveolar nerve** or the **mandibular nerve** by the needle.

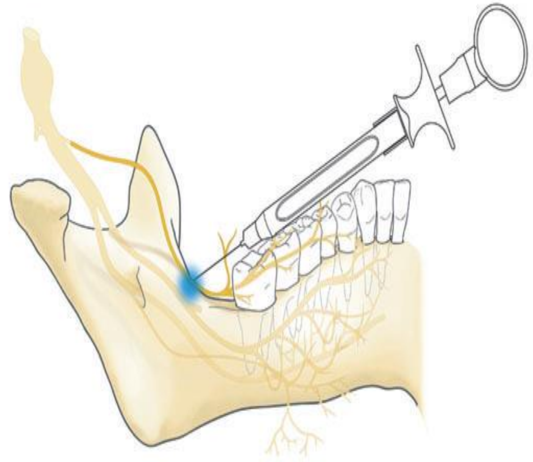
The nerve may then be damaged without the patient or dentist noticing.

It is therefore advisable to **employ intraligamentous anaesthesia**, particularly if the **lingual nerve and the inferior alveolar nerve are not anaesthetised**.

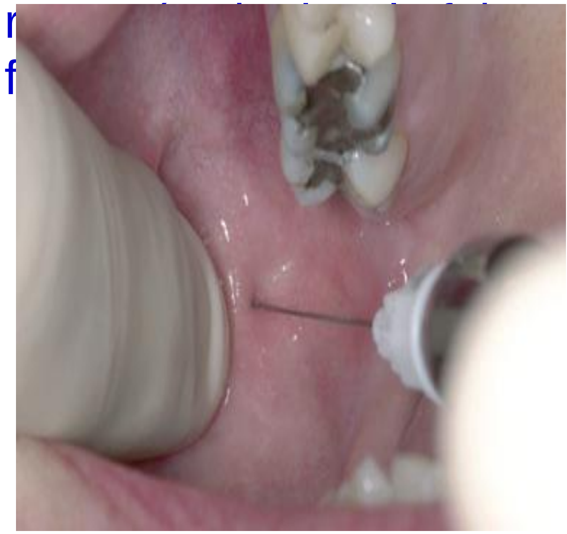
An **additional injection also** has the **disadvantage that an ‘acidic environment**’ slowly develops inside the **pterygomandibular space** due to the addition of another 1.7–1.8 ml of anaesthetic fluid with a **low pH.**

The rest of the **carpule** is then injected in order to anaesthetize the **lingual nerve .**
Now the syringe is taken **out of the mouth**.
The patient closes the mouth and is given a compliment for his/her cooperation during the anaesthesia.
The dentist also asks about the patient's experience:
'Was it painful?
Do you feel the effects of the anaesthesia?'

If necessary, the **cartridge is replacedand anaesthesia** is given to the **buccal nerve**, also one finger-width above the point of **occlusion exactly on the front side of the ramus** down to the gingiva and mucosa in the region M3–P1inf .
The path of the buccal nerve varies, however, so that the dentist may elect to give an infiltration anaesthesia buccal to the element **to be anaesthetised**.
About **2–3 minutes** after giving a **mandibular block** the patient should indicate that the corner of the mouth, lower lip, edge and tip of the tongue have begun to tingle and feel odd. One minute later the lower lip and tongue will be anaesthetised on one side and the treatment can start.
An experienced dentist, who takes the characteristic **anatomical structures** that were previously mentioned into account, will achieve a **good mandibular anaesthesia** in about **85% of cases**. **Block anaesthesia may fail due to individual anatomical differences**, such as a progenic mandibula, a **divergent angle** between the horizontal and vertical part of the mandibula, or **the absence of teeth**



Drawing (**A**) and photo (**B**)
of block anaesthesia of the
buccal nerve that runs from high lingual, crosses the front of the



In this environment, the **ionised** form of anaesthetic will increase.
This form is unable to pass through the myelin sheath, thus reducing the effectiveness of the local anaesthetic.

Mandibular anaesthesia on the **left side** of the patient is conducted in the same way as on **the right side**.
The only difference is that the **left hand injects instead** of the **right hand**.
If a dentist **still prefers to use the righthand**, he/she must move to an **11–12 o'clock position**.
The left hand holds the **patient's cheek to the side** and **feels along the front of the mandibular** ramus, while the **right hand gives the anaesthetic.**
In some situations, such as **two-sided extractions in the lower jaw,** **extensive periodontal treatment** and **(pre-)implantological treatments** in the **interforaminal area**, a **two-sided mandibular block** may be given to healthy patients.
The entire mandibula is then anaesthetised, including the **lower lip** and the **front two-thirds of the tongue.**
The tongue's motorics remain **undisturbed**, however, as well as **reflexive swallowing**. **Reflexive swallowing** begins at the back third of the tongue and pharynx. Because the foremost part of the tongue is anaesthetised, the patient will **not notice** if anything is **lying on it**, such as a broken part of a molar or a piece of filling.
The dentist and assistant must, therefore, keep a good eye on the oral cavity and throat

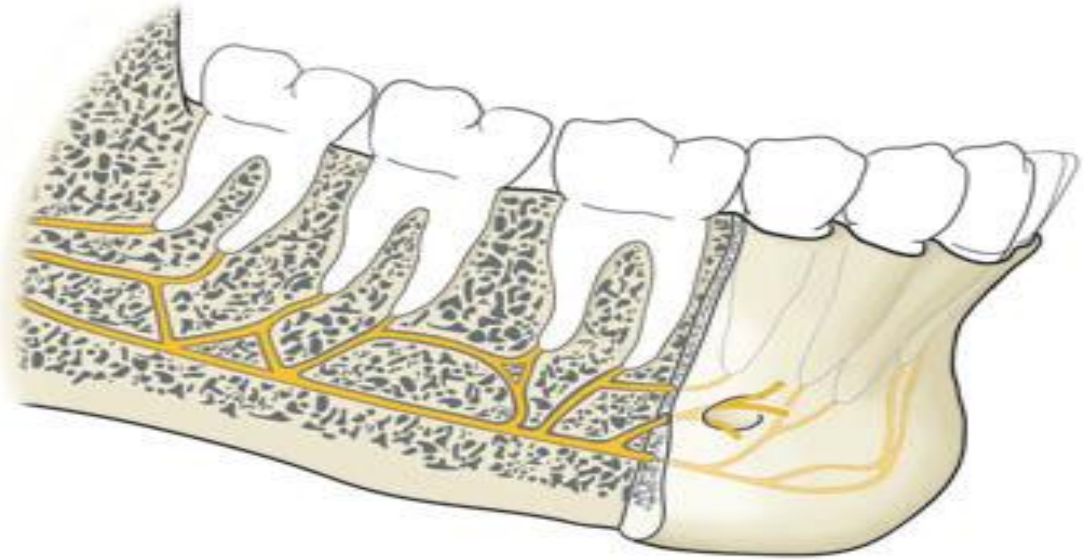
Mandibular block anaesthesia from the right corner of the mouth (direct technique). The mouth is opened as much as possible and the needle is inserted carefully on the lingual side of the mandibular ramus so that 1 cm of the needle remains

Molars

Anatomical aspects

Both on the buccal and on the lingual side, the roots of the molars are covered by **a thick layer of cortical bone**. The external oblique rim and the mylohyoid rim form an extra barrier for the **diffusion of anaesthetic fluid to the apices of the molars** .

The roots of the **molars lie on the lingual side,** usually below the **level of the mylohyoid muscle** and **buccal to the M2inf under the point of attachment of the buccinator muscle. Infiltration of these muscles must be avoided** as a **haematoma increases**the **risk of infection**.

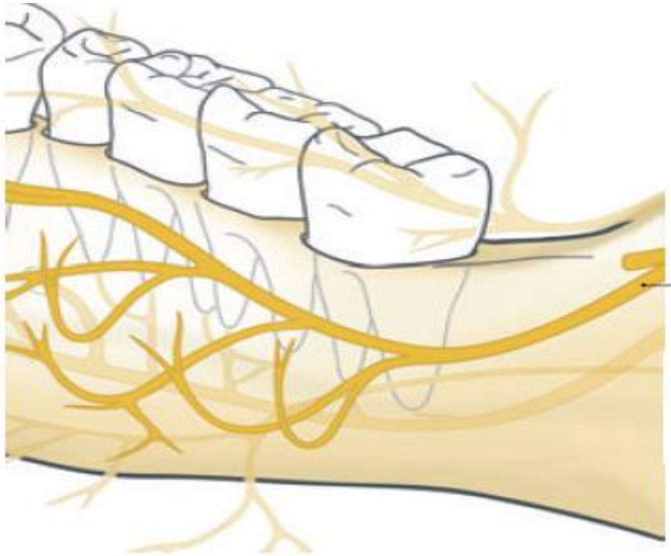


Anatomical drawing of the inferior alveolar nerve and its branches to the apices of the molars.

Indication:

Cavity preparations, endodontic treatment and surgical treatments require
a mandibular block, supplemented by **block** or **infiltration anaesthesia** of
the buccal nerve.

Intraligamentous anaesthesia should, theoretically, also suffice for cavity preparations and endodontic treatments.



Lingual aspect of the mandibula with the M_{3Inf} and lingual nerve relation right.

Technique:

see the previous text above for the technical aspects of a mandibular block and buccal nerve anaesthesia.

Third molars in the lower jaw

Anatomical aspects

The impacted M3inf lies in the **mandibular angle region**, dorsal to the M2inf. This area is innervated not only by the mandibular nerve but also by sensory branches that leave the **spinal column at C2 and C3**, and run over the platysma to the angle.

This must be taken into account when surgically removing the M3inf.

Lingual aspect of the mandibula with the M_{3inf} and lingual nerve relation right.

The lingual nerve runs **caudo-laterall**y from its source in the direction of the jaw and is found at the height of the M3inf approx. **5 mm lingually** and **caudally to the bone edge**, and dorsally to the M2inf. The path of the lingual nerve, however, shows great individual variation: the lingual nerve can also be found on the lingual side of the alveolar process above the impacted M3inf at the height of the bone

Indication:

Surgical treatments such as **trigone bone transplant**, an **operculectomy and removal of the (partly) impacted** M3inf require mandibular block anaesthesia and anaesthesia of the buccal nerve. If the M3inf is deeply impacted, anaesthesia is also needed for the sensory **branches from C2 and C3 by applying** infiltration anaesthesia deep in the fold behind the M2inf.

Technique

See the text above for the mandibular block technique and block anaesthesia of the buccal nerve.

Mental nerve block

The mental nerve leaves the mandibular canal via the mental foramen approximately 5–8 mm under the P1–P2inf **apices** and provides sensitivity to the lower lip, skin of the chin and oral mucosa, ventral to the foramen. **The lower frontal teeth**, including P1inf, are not innervated by the mental nerve.

Blocking the mental nerve is advised for **surgery of the lower lip** and the **anterior edentous alveolar process front**, and **for biopsy of the relevant are**a.

A cartridge syringe with a short **25-gauge** needle is used, and a local anaesthetic with **vasoconstrictor**.

The mouth is almost closed and the thumb holds the lip to the side. The fingers of the same hand feel the inferior border of the mandible. **The short needle penetrates the mucosa by the P1inf**, approximately **0.5 cm from the alveolar process.**

The needle point is introduced slightly medially and dorsally, so that contact with the bone occurs after approximately 1.5 cm .Half a cartridge isinjected after aspiration. The half lower lip, skin of the chin and buccal mucosa are anaesthetised within 2–3 minutes.

If the needle is inserted **too far dorsally**, the foramen will be **missed**.

When the needle is inserted too far laterally, the fluid will accumulate subcutaneously.

Additional infiltration anaesthesia is often needed for surgery of the soft parts of the lip area, because of the vasconstrictive effect.

A small injection is sufficient at the level of the corner of the mouth, where the labial artery reaches the lip area

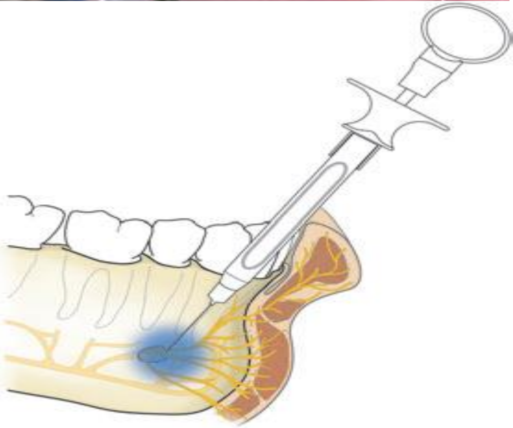


Photo of skull (**A**) and photo of patient (**B**) show block anaesthesia of the nasopalatine nerve. The needle is inserted upright into the incisive papilla and introduced carefully for approx. 1 cm into the nasopalatine canal. This runs parallel to the axis direction of the central incisor. The injection is given after aspiration.

Gow-Gates technique

Usually the direct or indirect technique is selected for a mandibular block where two consecutive injections anaesthetise first the inferior alveolar nerve and lingual nerve and second the buccal nerve. In 1973, **the Australian George Gow-Gates** described a block anaesthesia that is a mandibular block at a much higher level. **This method anaesthetizes the entire mandibular nerve with a single depot**, so that an additional block of the lingual or buccal nerve is no longer necessary.

The chance of a successful anaesthesia of the entire mandibular nerve following the Gow-Gates technique is **about 95%.**

The success rate of a classical inferior alveolar nerve **block is 85%.** The advantages of the Gow-Gates technique, **however, are limited.**

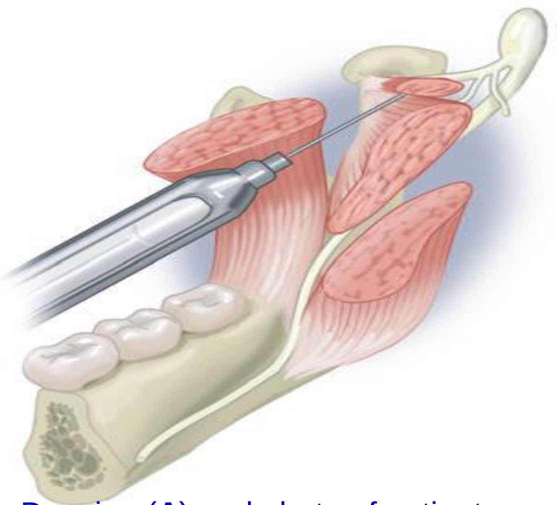
The thumb feels along the attachment of the temporal muscles to the coronoid process.

Medial to this, the needle is inserted into the mucosa at the height of the occlusal plane of the M_{2sup}. The index finger of the same hand is placed in the external auditory canal and the needle is then inserted about 25–27 mm in the direction of the index finger.

Bone contact is made with the **medioventral side of the condyle It is necessary to aspirate** because the needle point may enter the maxillary artery. After aspiration, an entire cartridge of anaesthetic fluid is injected.

After 2–3 minutes the **following branches of the mandibular** nerve will be anaesthetised: the inferior alveolar nerve, the lingual nerve and almost always the buccal nerve.

If the needle is introduced too far, the mandibular caput may be missed and the needle will shift over the mandibular incisura into the masseteric muscle.



Drawing (**A**) and photo of patient (**B**) show the Gow-Gates technique for anaesthesia of the mandibular nerve. On the lingual side of the coronoid process, at the height of the M2sup the needleis inserted in the mucosa inthe direction of the external auditory canal. The needle isintroduced almost completely until bone contact is made with the medioventral side of the condyle.

Supplemental injection techniques

The development of **safe** and **effective local anesthetic agents has been an important advancement in dental therapeutics**. Their anesthetic effectiveness, however, sometimes is inadequate, particularly after the administration of a **mandibular nerve block**. **The nerves supplying mandibular teeth and periodontal tissue are encased in the bone.** The thick cortical plate of the mandible impairs diffusion of anesthetic solutions into the mandible, often limiting the effectiveness of infiltration anesthesi
Alternative anesthetic techniques that can overcome this barrier are available. The periodontal ligament (PDL) anesthetic technique involves using high injection pressure to force the local anesthetic solution through the PDL into the cancellous medullary bone surrounding a tooth. The intraosseous (IO) anesthetic technique requires mechanical perforation of the thick cortical plate between the roots of the teeth to permit deposition of the local anesthetic into the medullary bone surrounding the tooth. These techniques permit diffusion of anesthetic around the tooth socket to anesthetize all of the nerves supplying the dental pulp. The anesthesia often is limited to the specific tooth undergoing treatment

The intraosseous (IO) anesthetic technique

The intraosseous (IO) anesthetic technique **requires mechanical perforation of the thick cortical plate between the roots of the teeth to permit deposition of the local anesthetic into the medullary bone surrounding the tooth.** Although maxillary infiltration anesthetic injection techniques may have success rates of 95 percent or higher, the **success rates for IANBs generally are 80 to 85 percent.**

Lower success rates may be due to the **greater density of the buccal alveolar plate** (which restricts supraperiosteal infiltration), **limited access to the inferior alveolar nerve** and a **wide variation in neuroanatomy**.
With pulpitis, **hyperalgesia may be another reason for anesthetic failure**. **Inflamed tissues may alter the nerves' resting membrane potentials** and **decrease excitability thresholds**, **changes that are not restricted to the inflamed pulp but affect the entire neuronal pathway**, **extending to the central nervous system**. Therefore, routine local anesthetic techniques may not prevent **nerve transmission adequately** because of the **lowered excitability thresholds**.
A description of the IO anesthetic injection technique was first published in 1910. The author described a technique for delivering local anesthetic to the root tip **via a small drilled hole**. The technique lacked popularity because dentists were reluctant to drill **into cortical bone** and **had difficulties inserting a needle precisely into the tight fit of the drilled hole**.
Early techniques included **instrumentation** with a half-round bur or a motorized endodontic reamer and a standard 27-gauge short needle.
The volume of anesthetic administered ranged from **0.5 to 1.5 mL**. Owing to the **lack of intimate** fit between the needle and the hole, the effective volume often was less than the total volume administered because of leakage at the injection site.

Instruments

As the **IO technique evolved**, instruments were designed to **control deposition** of the solution, including the **Stabident system** (Fairfax Dental, Miami) and the X-Tip dental anesthesia system (Dentsply Maillefer, Tulsa, Okla.).
The technique requires perforating the **cortical bone** by **creating a small hole between the roots of the teeth with a specialized rotary instrument**.
The dentist makes the **perforation approximately 5 millimeters apical to the buccal papilla.** Applying constant pressure when the perforator is against the cortical plate can lead to a **buildup of heat**. The X-Tip system has a unique design that leaves a guide in place after perforating the cortical bone to make it easier to insert the needle through the perforation. The administration of an injection of one-quarter to one-half of a cartridge of local anesthetic by means of a small needle guided into the trabecular bone can induce anesthesia .

Initially, dentists used the IO anesthetic technique as a supplementary technique when the IANB failed, especially in cases of **irreversible pulpitis**.

With the advent of products such as Stabident and the X-Tip, the technique has gained in popularity as a primary technique for anesthetizing a single mandibular tooth. Although dentists use the IO technique most often to provide anesthesia in a single tooth, they may use it to anesthetize multiple teeth in the same quadrant, depending on the injection site and volume of anesthetic injected.

When an IANB was supplemented with an IO injection, investigators reported a substantial increase in the overall anesthesia success rate for first molars and second premolars.

For teeth with **irreversible pulpitis**, the administration of a supplemental mandibular IO injection increased total **pulpal anesthesia success.**

The onset of anesthesia after the IO injection was administered was **almost immediate**.

Intraoral photograph showing the position of the needle for the intraosseous anesthetic injection technique

Contraindications

Contraindications to the use of the IO anesthetic injection technique include gross periodontal disease or acute peri-apical infection. Formation of fistula has been reported at perforation sites.[7]

This technique should be used cautiously in cases in which the roots of the teeth are so close together that they preclude clear access to the interdental trabecular bone. A relative contraindication is when there is difficulty perforating the cortical plate where it is thick, such as areas distal to the second molar, increasing the chance of perforator fracture.[7]

Some areas of the mandible also may have **constricted cancellous bone**, which may impede anesthetic distribution

Adverse effects and complications

There are some possible **adverse effects** and **complications of using the IO technique**. **Heart palpitations** frequently occur when a **vasopressor-containing anesthetic** is used.

To minimize the risk, a **slow injection using a local anesthetic without a vasopressor**, such as **3 percent mepivacaine**, is recommended.

Only **one-eighth to one-qu**arter of a dental cartridge should be administered **at one time until adequate anesthesia is achieved**.

Because the cancellous bone in the mandible is vascular, keep the volume of local anesthetic to the **recommended minimum** to **avoid possible rapid systemic uptake** and **overdose**.

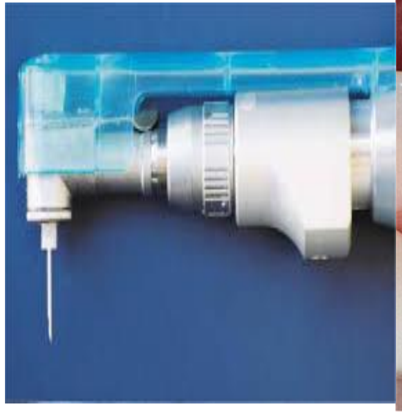
The use of vasoconstrictors is dictated by treatment needs and patients' health histories.

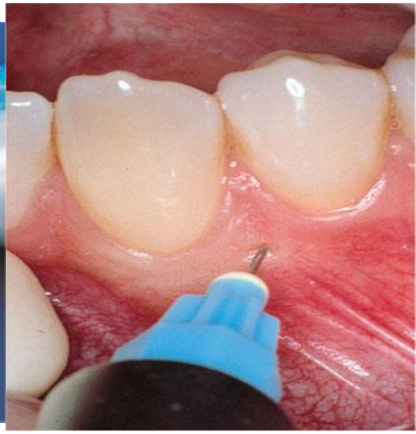
Patients with **moderate** to **severe** cardiovascular disease or who are taking **tricyclic antidepressants** or **nonselective β-adrenergic blocking agents** **are poor candidates for use of the IO anesthetic injection technique** when solutions containing **epinephrine** or **levonordefrin** are used

Investigators have reported a transient increase in heart rate after administration of vasopressor-containing anesthetic solutions by means of IO injections.

Increase in heart rate as determined by subjective questioning after administration of an IO injection of **1.8 mL of 2 percent lidocaine with epinephrine 1:100,000**.

Perforation of the lingual plate of the bone or injury to the roots of the teeth can occur. The IO anesthetic injection technique is not recommended for use in areas of mixed dentition because of insufficient cancellous bone and the possibility of damaging developing tooth buds.





Advantages of intraosseous anaesthesia

The advantages of intra-osseous anaesthesia over conventional methods include:

a smaller dose is required

a smaller area of soft tissue anaesthesia is produced

the method aids in overcoming failure of conventional techniques.

1.Smaller doses are used than in conventional regional block anaesthesia – around 1 mL is normally sufficient.

2.The amount of soft tissue anaesthesia produced is less than that caused by infiltration and regional block methods and this may reduce the possibility of self-inflicted trauma.

3.When used in combination with inferior alveolar nerve blocks, the method increases the success rate for pulpal anaesthesia compared to the use of the regional block in isolation. Similarly, supplemental anaesthesia via the intra-osseous route may be effective in teeth with irreversible pulpitis where conventional methods have failed

Disadvantages of intraosseous anesthesia

The disadvantages of intraosseous anaesthesia include:**technically more difficult** than infiltration anaesthesia

specialised equipment may be required systemic effects may be increased post-injection discomfort may be produced

teeth may be damaged.

1.The method is technically more difficult than infiltration anaesthesia as the entry point made by the perforator must be accurately located. This is simplified with some specialised intraosseous delivery systems that include a locator. This locator remains in position after removal of the perforator and directs the needle into the channel created.

2.Although it is not absolutely essential, specialised equipment makes the method easier.

3.Entry of local anaesthetic and vasoconstrictor into the circulation occurs rapidly following intraosseous anaesthesia and systemic ineffects attributable to catecholamine-entry to the circulation occur early after injection. Patients may report an increase in heart rate during intraosseous anaesthesia with epinephrine-containing solutions.

4.Post-injection discomfort may occur. Post-operative swelling and an exudate may be produced after intraosseous injections and some patients have perforation sites that are slow to heal.

5.The **method may damage teeth**. The perforators can penetrate teeth. Fortunately there is a tactile change detectable when dental tissue is encountered and strong pressure has to be used for this to occur.

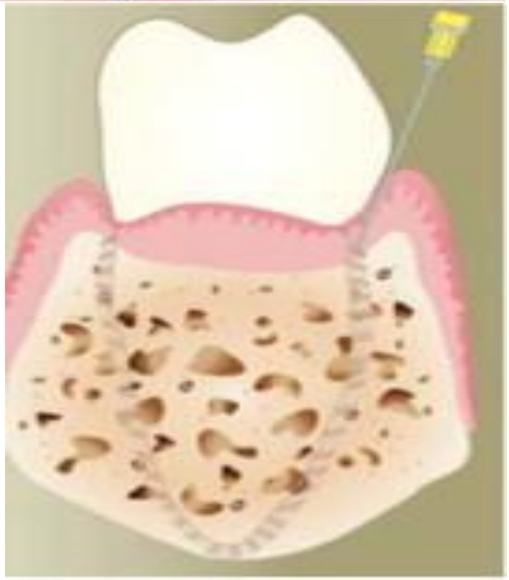
The periodontal ligament anesthetic injection

The PDL anesthetic injection technique, also referred to as the "intraligamentary injection technique," can induce local anesthesia in either maxillary or mandibular teeth.

Although occasionally it is **used as the primary anesthetic technique** (when a single tooth requires anesthesia for a short duration), dentists most often use the PDL technique when mandibular nerve **blocks are unsuccessful.**

Teeth with irreversible pulpitis generally are considered the most difficult to anesthetize and often require supplemental anesthesia.

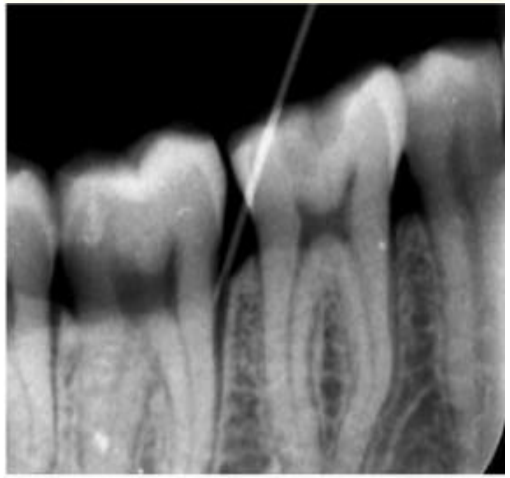
The PDL anesthetic injection technique was introduced in the **early 20th century** and gained popularity in the 1970s when dedicated high-pressure dental syringes such as the Peripress Pen (Panadent, Kent, England) and Ligmaject (Henke-Sass, Wolf, Tuttlingen, Germany) were introduced.



These syringes could be operated **with one hand** and were **capable of delivering small volumes of anesthetic from standard dental cartridges** at the **high hydrostatic pressures** required for the PDL anesthetic injection.

It is recommended using short **27- or 30-gauge** dental needles for this technique. With the tip of the needle approaching the **periodontal sulcus** on the mesial or distal aspect of the tooth, advance it to the base of the periodontal crevice. With the bevel oriented toward the root surface, advance the tip of the needle into the PDL between the root surface and the adjacent alveolar bone.

Administer a small amount (**0.2 milliliters**) of anesthetic solution slowly. To ensure that the solution is being forced into the tissue, **you must feel resistance**. Although syringes differ among manufacturers, the technique usually requires deposition of at **least 0.2 mL** for each root of the tooth.



In situations in which anesthesia of a **short duration is required**, the PDL anesthetic **injection technique** might be the **preferred treatment**.
This technique avoids the **deep needle insertion associated with mandibular regional blocks** and **may be considered a safer alternative technique for patients with bleeding disorders**.

The anesthetic efficacy of the PDL anesthetic injection technique can be unreliable if the needle is not positioned precisely. Recommended not **administering injections into inflamed** or **infected periodontal sites.**
The current American Heart Association recommendations do not provide specific guidance regarding antibiotic prophylaxis when administering the PDL injection

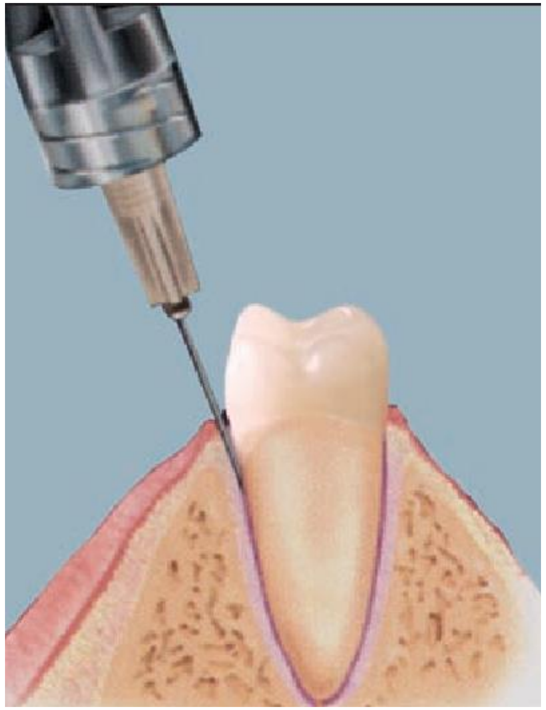
The recommendations state that antibiotic prophylaxis is not needed with routine anesthetic injections through noninfected tissue. However, even when it has been administered through healthy periodontal tissue, the PDL injection has induced **bacteremia.** Because of the potential for bacteremia to induce **bacterial endocarditis**, dentists should consider antibiotic prophylaxis when administering PDL injections, particularly when administering an injection **through inflamed periodontal tissue**.
For the few patients who have a known risk of developing **bacteremia-induced endocarditis**, **avoiding the use of the PDL anesthetic injection technique is a practical alternative when possible.**

A disadvantage of routinely using the PDL anesthetic injection technique is that some patients report **tenderness** at the injection site **for a day** or **two after treatment**.

Among the commonly used local anesthetic injection techniques, patients described **needle placement during the administration of an IANB as most painful**, followed **by the PDL anesthetic** injection technique and the **mental nerve block injection and infiltration anesthetic injection techniques.**
They reported that the PDL anesthetic injection technique was the **most uncomfortable during solution deposition**.

The position of the needle and the **pressure of the injection can cause trauma to tissue** and **subsequent postoperative discomfort.**

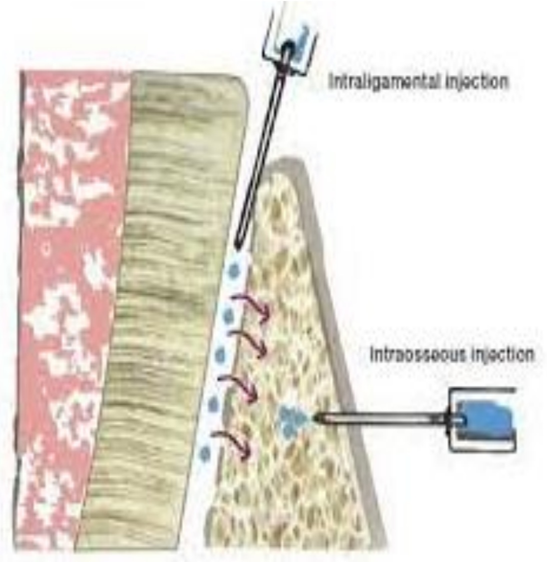
The PDL anesthetic injection technique is not recommended for primary teeth, because there have been cases of enamel hypoplasia and **hypomineralization in permanent teeth adjacent to the injection site.**



Intra Septal injections

Local anesthetic failure is an unavoidable aspect of dental practice. A number of factors contribute to this, which may be related to either the patient or the operator. Patient-dependent factors may be anatomical, pathological or psychological1-3. Work is still going on by dental clinicians and researchers in order to find an optimal local anesthetic agent which it has a high potency and rapid onset of action.4-6. However, pain free injection also play a role in improving the patient

Although the solution is **deposited into the coronal segment of the PDL**, the anesthetic is **not forced down the PDL** to the **tooth apex** but **instead is redirected into the surrounding cancellous bone through the fenestrations in the dental socket**.



Unlike the cortical plate of the mandible, the dental socket has **multiple passageways** to accommodate the blood **vessels that supply the periodontium**. Investigators used a dog model to simulate this clinical technique and assessed the distribution of the local anesthetic after the PDL injection was administered..

Studies showed The results of the PDL anesthetic injection techniques showed that success rates ranged from **60 percent** for endodontic therapies to 100 percent for periodontal therapies and tooth extractions.

Anesthesia onset was rapid, and **anesthesia duration was 30 to 45 minutes**.

Adverse reactions included **pain during administration** of the injection**, tenderness at the injection site after treatment** and **a subjective sensation that the tooth was elevated in the occlusion** or **"high" after treatment.**

Intraseptal anesthesia for pain-free dental treatment

In order to be a successful dentist, you have to implement a pain-free dental treatment. This can be merely achieved by effective local anesthetic techniques.Considering the complex nature of oral and dental tissues, attaining effective dental local anesthesia may be challenging in certain circumstances. For example, the **failure of local anesthetic injections in irreversible pulpitis** can be **8 times** **higher than health teeth**. Such issues cannot be ignored as a good number of patients with endodontic complaints attend the dental clinics on a daily basis.

Falure of the local anesthetic injections using inferior alveolar nerve block (IANB) for lower dentition and buccal infiltration for upper teeth in asymptomatic and symptomatic patients requires additional buck-up strategies to achieve pain-free dental treatment. Otherwise, the patient complains of **severe pain** and **hindering the clinician to proceed to the dental treatmen**t .Intraseptal anesthesia (ISA), and specialized equipment may be required. Intraosseous technique can lead to rapid **absorption of local anesthetic** and **vasoconstrictor in the circulation**. Hence, cardiovascular changes are attributable to **adrenaline entry into the circulation that may occur immediately after intraosseous injection**

Intraseptal injection has been reported to be more effective for controlling postoperative pain compared to intraosseous and intraligamentary injections. **The efficacy of ISA is similar** to **intraosseous injection**, and they are both more successful than the **intraligamentary injection** because a **greater amount of anesthetic solution can be delivered during the injection.**

Supplementary techniques might have a **negative effect** on the cardiovascular system, for example, **increased heart rate for a couple of minutes after intraosseous injection .**

Both intraseptal and periodontal ligament techniques is beneficial and appropriate for the **routine tooth/teeth removal.** Intraseptal technique provides local anesthesia of **one tooth including the soft-tissues**.

Intraseptal technique **anesthetizes surrounding nerve endings in the tissues of a particular tooth**.

There are a few **contraindications** such as **acute inflammation** or **infection at injection site**. However, **ISA remains a convenient local anesthesia practice for a general dental surgeon.**

The protocol used for the administration of **ISA has been described briefly**. Patient ought to be placed in the supine position. Considering the **thick of soft-tissues**, a **short injection needle** is usually **proffered**. The target region is **located 2-3 mm apical to the apex of the papillary triangle.**

Intraseptal injection technique; (a) marking for the administration of anesthesia (b) positioning of the needle 3 mm apical to the apex of the papillary triangle (c) ischemia of the soft-tissue surrounding the injection site

The needle is introduced into **the soft-tissue** and advanced until contact with bone is made. Pressure must be applied to the syringe and drive the barb slightly deeper (1-2 mm) into the interdental septum. Afterward, anesthetic solution (0.2-0.4 ml) is **deposited in a minimum of 20 s time.** Prevailing resistance to the flow/movement of the anesthetic solution and ischemic discoloration of the neighboring soft-tissues are main signs of success of this technique.This main aim of this review was to **discuss various aspects of intraseptal dental anesthesia and its role significance in pain-free treatment in the dental office**. In addition, reasons of failure and limitations of this technique have been highlighted.

Mechanism of Action for Intraseptal Injection

The route of diffusion and distribution of the anesthetic solution in the intraseptal technique is most likely through the medullary bone It offers anesthesia to the bone, delicate/soft-tissues, and root structure in the region of infusion.

It is best when **both pain control** and **hemostasis are wanted for delicate/soft-tissue and bony periodontal treatment.**

Advantages of intraseptal injection:

In contrast to IANB and local infiltration, the intraseptal technique prevents the anesthesia of tissues such as lips and tongue hence, decreases the chances of cheek or lip biting (self-trauma). It necessitates minimum or least dosage of local anesthetic and minimizes bleeding during the surgical procedure.

This technique being less traumatic has immediate or instantaneous (<30 s) onset of action and comparatively less number of postsurgical complications. Intravascular injection is extremely unlikely to occur compared to IANB or infiltration. **Assertions that ISA is immediate are properly consistent with previous clinical results**. Their findings reported that the onset of action for anesthesia was within 1 min after injection. Hence, the onset time can be considered rapid if not immediate

Disadvantages of intraseptal injection

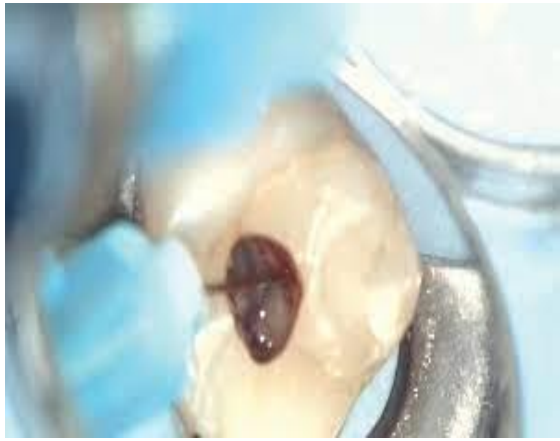
Clinical experience and multiple tissue punctures may be necessary to perform this technique. During the anesthetic procedure, the anesthetic solution may leak into the oral cavity resulting discomfort and an unpleasant or bitter taste. The effective period anesthesia for pulpal and soft-tissues is very limited .Hence multiple repeats may be required for longer surgical procedures.

Dental techniques demanding significant pulpal, bone, and soft-tissue anesthesia can be adequately and securely acquired using ISA. **It can be a first choice anesthesia for teeth extractions and restorative dental procedures**. Intraseptal injection is likewise helpful for giving **hemostasis for surgical flap procedures and periodontal curettage**

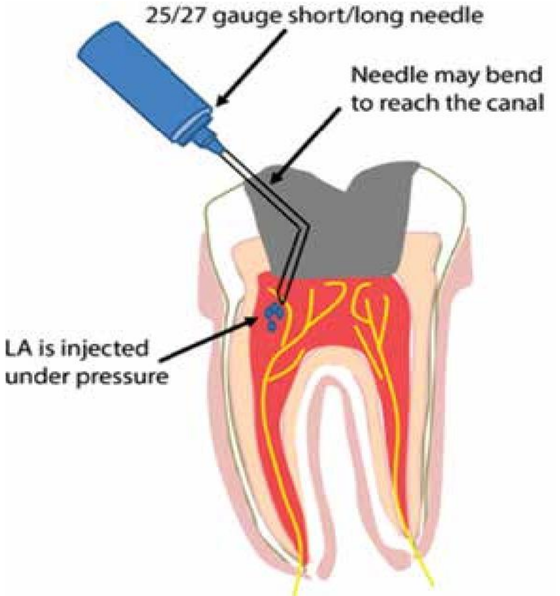
Intrapulpal injection(IPI):

In about **5 to 10%** of mandibular posterior teeth with irreversible pulpitis, supplemental injections, even when repeated, do not produce profound anesthesia; **pain persists** when the pulp is entered. **This is an indication for an intrapulpal injection**

The intrapulpal injection technique (IPI) is **one of the commonly employed supplemental anesthetic technique adjuvant to conventional maxillary infiltration anesthesia or mandibular inferior alveolar block in situations**



The **intrapulpal injection** should only be given after all other **supplemental techniques have failed**. Another disadvantage of the technique is the duration of pulpal anesthesia may be short (15 to 20 minutes). Therefore, the bulk of the pulpal tissue must be removed quickly, at the correct working length, to prevent reoccurrence of pain during instrumentation. **Another disadvan**tage is that, obviously, **the pulp must be exposed to permit direct** injection; frequently, anesthetic problems occur prior to exposure while still in dentin



The most significant factor **contributing to the success of IPI** is that its administration must be done under pressure. Monheim has suggested that prolonged pressure may lead to degeneration of nerve fibres in many instances leading to **profound anaesthesia.**

Various suggested methods that aid in pressure build up in such cases include obliteration of a large pulpal opening with either **gutta-percha** or a **cotton pelle**

The advantage of the intrapulpal injection is that it works well for profound anesthesia if given under back-pressure. **Depositing anesthetic passively into the chamber is not adequate; the solution will not diffuse throughout the pulp.**

Complications:

Discomfort during the injection of anesthetic.

The patient may experience a brief period of pain as the injection of the anesthetic drug is started.

Almost immediately, the tissue is anesthetized and the pain ceases

Failures of Anesthesia Intrapulpal Injection

Infected or inflamed tissues.

Changes in the tissue pH minimize the effectiveness of the anesthetic.

Intrapulpal anesthesia invariably works to provide effective pain control

Solution not retained in the tissue.

To correct: try to advance the needle further into the pulp chamber or root canal and re-administer 0.2 to 0.3 ml of anesthetic.

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Complications of local anesthesia

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Complications of local anesthesia

 Local Complications

- ✓ Needle breakage
- ✓ Prolonged anesthesia (paresthesia)
- ✓ Facial nerve paralysis
- ✓ Ocular complications
- ✓ Trismus
- ✓ Soft tissue injury
- ✓ Hematoma

Complications of local anesthesia

- ✓ Pain on injection
- ✓ Burning on injection
- ✓ Infection
- ✓ Edema
- ✓ Sloughing of tissues
- ✓ Postanesthetic intraoral lesions

Complications of local anesthesia

 Systemic complications

- ✓ Overdose
- ✓ Allergey

Local anaesthesia: is frequently used in dentistry and seldom leads to **serious local complications**. Nevertheless, it is of great importance to be aware of the causes of each local complication and – if necessary – to implement correct treatment.

The patient must be **informed extensively** and, if **necessary, be reassured**. The **incident must also be recorded** in detail in the patient's file.

This is especially important if there is a chance of **prolonged or even permanent symptoms**

Local Complications

Lack of effect

Needle breakage

Prolonged anesthesia (paresthesia)

Facial nerve paralysis

Ocular complications

Pain on injection

Burning on injection

Oro-Facial blanching

Infection

Trismus

Edema

Sloughing of tissues

Postanesthetic intraoral lesions

Systemic complications

Overdose

Allergy

Lack of effect (failure of LA):

Reasons for **unsuccessful in obtaining local anesthesia** can be dependent on:

1- anatomical variants,
2-pathological and
3-psychological factors,
4-choice of technique and solution, and
5-poor technique .

Anatomical factors comprise accessory nerve supply, alteration in foramen location, atypical development of the nerves (bifid mandibular canals), and bone density .

Pathological reasons for the failure of anesthesia are trismus, infection, inflammation, and previous surgery or trauma.

Inflammatory diseases altering the **pharmacokinetic**s and **pharmacodynamics** of local anesthetics cause a response to **decrease** and **unfavorable effects to increase .**Local anesthetic failure **or difficulty to obtain satisfactory** analgesia commonly occurs in the situations with **inflammations** such as **pulpitis** and apical periodontitis acute periodontal abscess or pericoronitis .

Psychological determinants such as **angs**t and **anxiety** can also cause local **anesthesia failure**.

Poor technique failure mostly occurs to obtain mandibular anesthesia. If the needle is inserted and advanced too deeply and too far dorsally, the **terminal branches of the facial nerve within the deep lobe of the parotid gland are affected**.

Direct anesthesia to the facial nerve can force a rapid onset that occurs while the anesthetic agent is being injected; **reflex vasospasms of the external carotid artery can lead to ischemia of the facial nerve**, so facial nerve palsy occurs.

The patient is unable to **wrinkle the forehead**, **raise the eyebrow**, **close the upper eyelid**, **retract the commissure of the lips to smile**, and **turn down the lower lip on the affected side**.

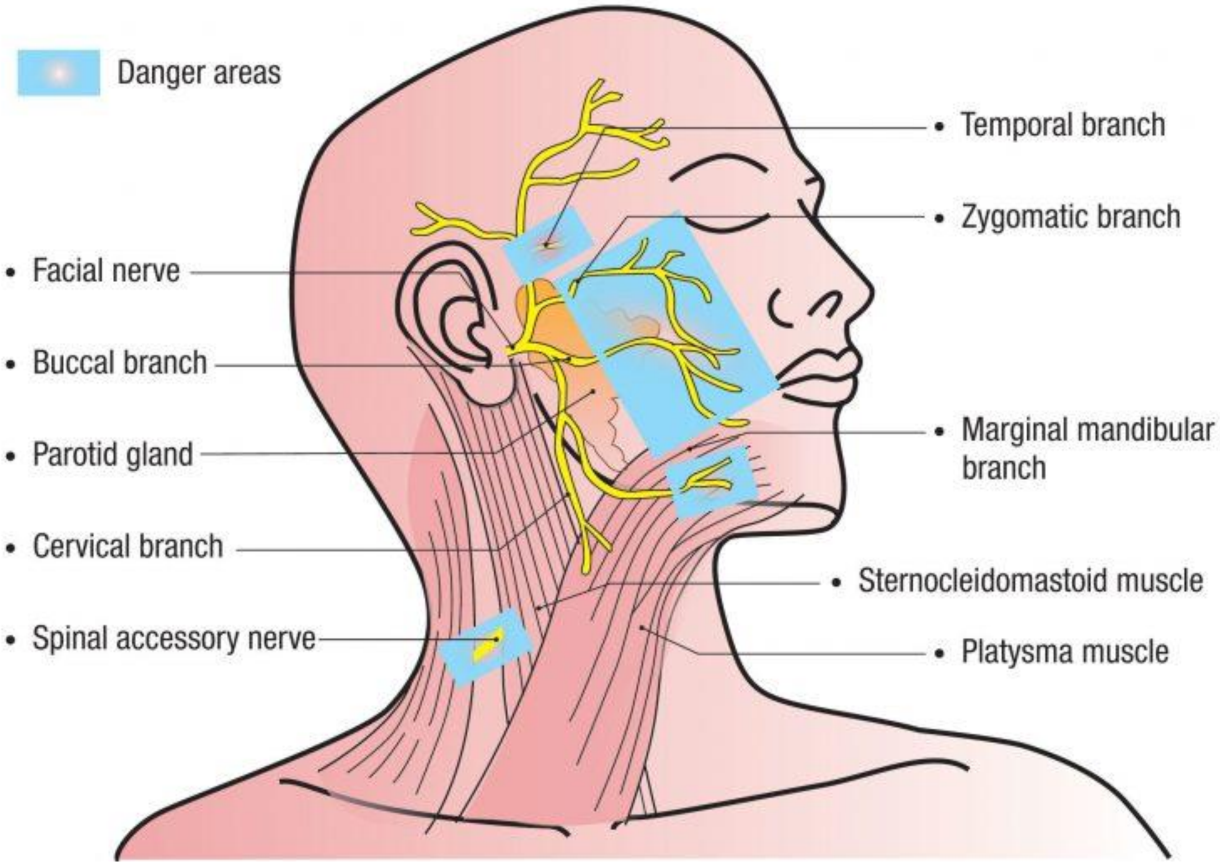
The removal of contact lenses and closing of the eye on the affected side in Bell's palsy prevent corneal abrasion or drying

If the needle is **inserted too high and deep**, N**. auriculotemporalis** will be affected, and the feeling of "**numbness" will occur**. There has been a report of **sudden unilateral deafness** following **inferior dental nerve anesthesia**

A technique suggested for patients who have **experienced local anaesthetic failure in the mandible is this:**

1-Conventional inferior alveolar and lingual block with lignocaine and adrenaline (1.5 ml), followed by long buccal nerve block with remainder of cartridge.

2-After subjective soft tissue signs of first block have taken effect a repeat inferior alveolar and lingual block injection using 3% prilocaine with 0.03 IU/ml felypressin. There is no scientific evidence that changing the active agent increases duration or depth of anaesthesia. However, there are a number of reasons why changing the solution **might offer an advantage**. **Firstly**, with the combination suggested there is an increase in the amount of local anaesthetic without increasing the amount of adrenaline administered. This can be of particular importance in some medically-compromised individuals. **Secondly,** there is some evidence that the combination of **lignocaine and prilocaine provides a greater spread of anaesthesia**, and this may be of some clinical benefit.



Facial nerve (CN7) branches and danger zones

Temporal Branch
• Frontalis muscle m. • Corrugator supercilii m. • Orbicularis oculi m. (superior portion) • Auricular m. (anterior and superior; also known as the temporoparietalis m.)
Posterior Auricular Branch
• Occipitalis m. • Auricular m. (posterior)
Zygomatic Branch
• Orbicularis oculi m. (inferior portion) • Nasalis m. (alar portion) • Procerus m. • Upper lip muscles ‣ Levator anguli oris m. ‣ Zygomaticus major m.
Buccal Branch
• Buccinator m. (muscle of mastication) • Depressor septi nasi m. • Nasalis m. (transverse portion) • Upper lip muscles ‣ Zygomaticus major and minor m. ‣ Levator labii superioris m. ‣ Orbicularis oris m. ‣ Levator anguli oris m. • Lower lip muscles (orbicularis oris m.)
Marginal Mandibular Branch
• Lower lip muscles ‣ Orbicularis oris m. ‣ Depressor anguli oris m. ‣ Depressor labii inferioris m. ‣ Mentalis m. • Risorius m. • Platysma m. (upper portion)
Cervical Branch
• Platysma m.

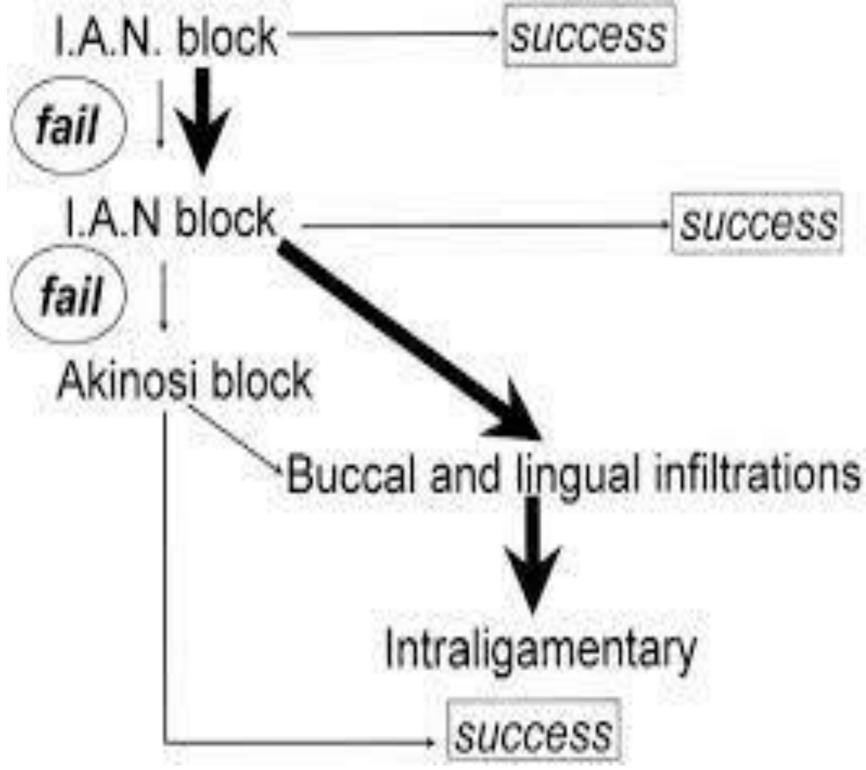
If subjective signs of inferior alveolar nerve block anaesthesia are not apparent after a second block then an Akinosi block is recommended with lignocaine and adrenaline.

3-Buccal and lingual infiltrations adjacent to the tooth of interest using around 1.0 ml of lignocaine and adrenaline in total (this to eliminate any accessory supply).

4-Intraligamentary injection of 0.2 ml lignocaine with adrenaline per root. This may seem extreme but the total volume injected is less than 6.0 ml which is acceptable in healthy adults. In the severely mentioned above is not recommendedmedically-compromised however, such as those with unstable angina this volume of an adrenaline-containing solution may be excessive and the technique in such patients.

In these individuals an adrenaline-free solution such as **3% prilocaine with felypressin should be used and a 'high' block should be considered after an initial failure**.

The supplementary infiltrations should be given with the same solution



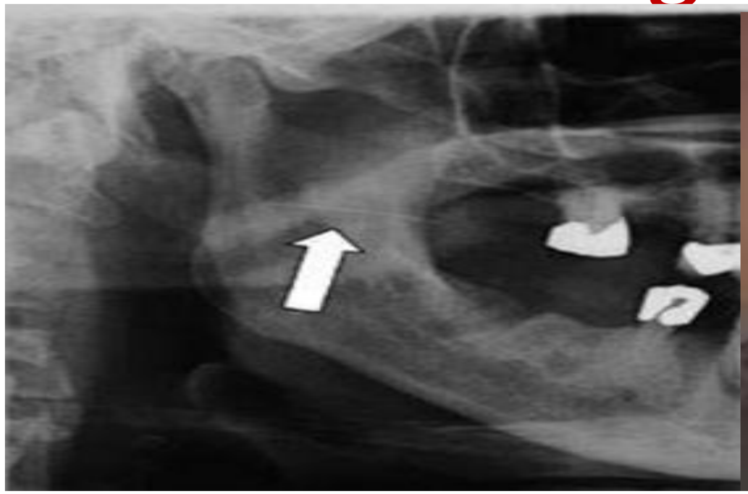
Needle breakage :

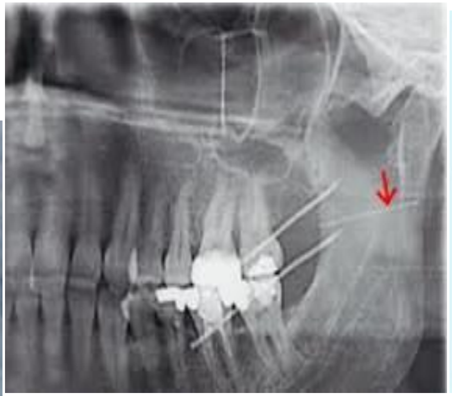
Since the introduction of **modern disposable needles**, needle breakage **is a rare complication**. Nevertheless, even these needles have a small chance of breaking, **especially with mandibular block anaesthesia** and **tuberosity anaesthesia**.
This **risk increases** if the needle is **repeatedly bent** to facilitate the **entrance into the area to be injected**.
The **unexpected movements of a scared patient**, such as a **sudden jerk of the head**, **grabbing or moving away the dentist's hand** or **suddenly closing the mouth**, can also **lead to this serious complication**.
Changing the direction of an incorrectly inserted needle also **increases the risk of needle breakage**. In such a situation, it is sensible to pull back the needle almost completely and then insert it again in the correct direction.

If the needle does break and the proximal part of the needle is **still sticking out of the mucosa**, the end may be taken **with tweezers** or **mosquito artery forceps** and **cautiously removed**.

However, if the broken **part of the needle** is **no longer visible** the patient **must be referred quickly to an oral and maxillofacial surgeon in a well-equipped hospital** .
In the meantime, the patient must be **instructed not to talk** and **to swallow as little as possible**, since such movements may allow the needle to move deeper into the tissues.

Needle Breakage



Causes:

1. Bending of the needle.
2. Sudden unexpected movement of the patient.
3. Entire length of the needle inserted into the soft tissue.
4. Use of the smaller needles (e.g. 40 gauge)

Prevention:

1. Use large-gauge needles, specially with Inferior Alveolar Nerve and Posterior Superior Alveolar Nerve.
2. Use long needles.
3. Do not insert a needle into tissues to **its hub** .
4. Do not redirect a needle once it is inserted into tissue.

Needle Breakage

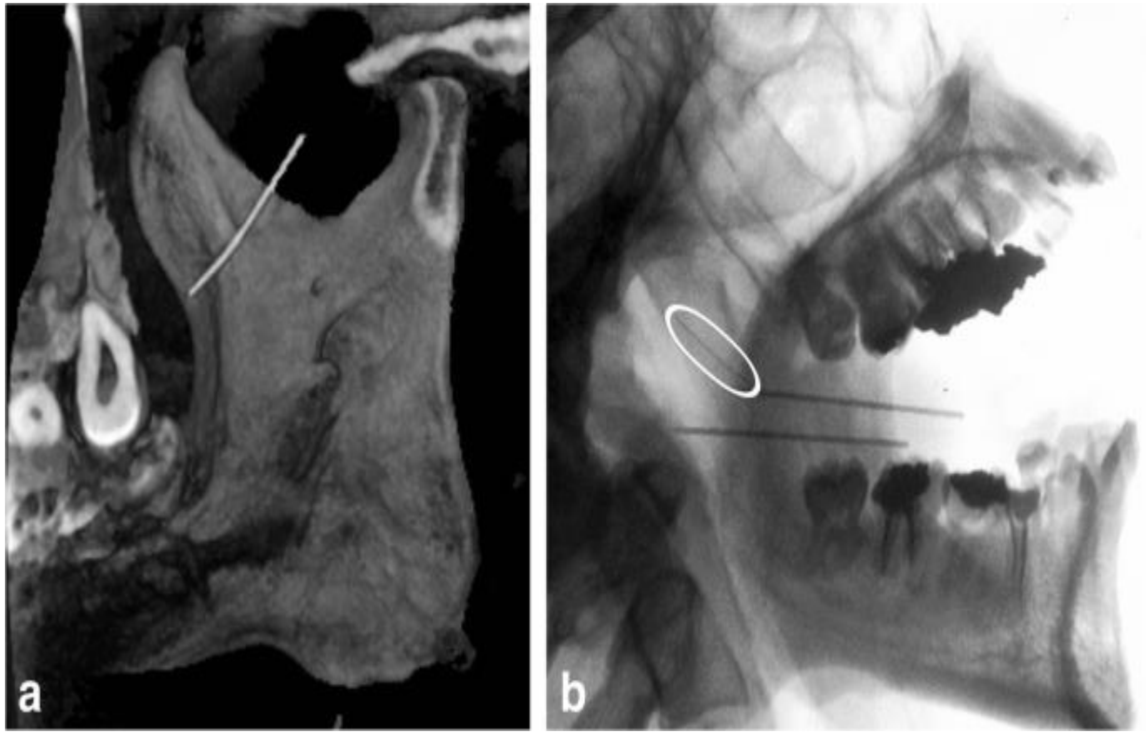
Management :

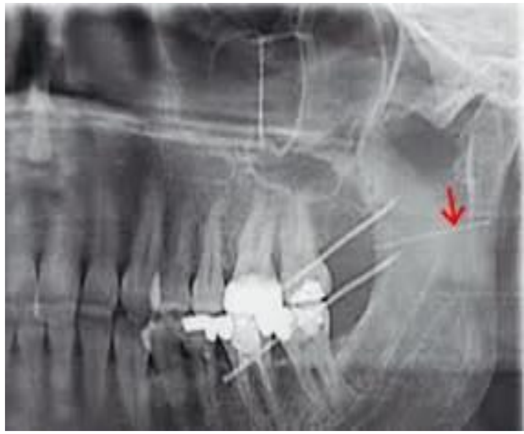
- When a needle breaks (visible):
- Stay calm.
- Instruct the patient not to move and let his mouth open.

If the fragment visible, remove it with hemostat or a Magill intubation forceps .

When a needle breaks (not-visible):

1. No incision or probing.
2. Calmly inform the patient.
3. Referral Oral Surgeon, take radiograph and determine if it is superficial, remove or leave it and follow up?!!





Prolonged anesthesia (paresthesia)

Local anaesthetics are the most used drugs in dentistry. **Paresthesia** may occur after various **dental procedures** such as **simple anesthetic injections**, surgical procedures, and **endodontic treatment**, and is **reported to range from 0.35% to 8.4%**.

The altered sensation usually **follows immediately** after the procedure, and reports of **late onset of nerve** involvement are rare

Concerning the oral paraesthesia, a possible additional symptom is the **impairment of the gustatory function** due to the **lingual nerve injury.**

In dentistry, most oral paraesthesia is caused by **direct trauma associated with a surgical procedure**, such as a **dental extraction** or orthognathic surgery aLSO can occur after **local anaesthesia** in non-surgical dentistry .

The mechanism underlying **non-surgical paraesthesia** is not known. Different hypotheses have been suggested to explain the relationship between **local anaesthetics** and **subsequent paraesthesia**:

(1) direct trauma caused by the penetration of the needle,

(2) haemorrhagic damage,

(3) nerve compression caused by the perineural oedema after the injection and

(4) neurotoxicity of the local anaesthetics .**Neurotoxicity of local anaesthetics** is a controversial topic .**Different studies** have suggested that local anaesthetic formulations may have the **potential for neurotoxicity,** in particular **articaine 4%** and **prilocaine 3–4%** .

As a matter of fact, articaine is the most used local anaesthetic in many countries , while prilocaine is scarcely used in clinical practice **.**

Evidence supporting the neurotoxicity of local anaesthetics emerged from the analysis of paraesthesia persistency after injection. 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. In dental practice, paraesthesia was described for any local anaesthetic.

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.

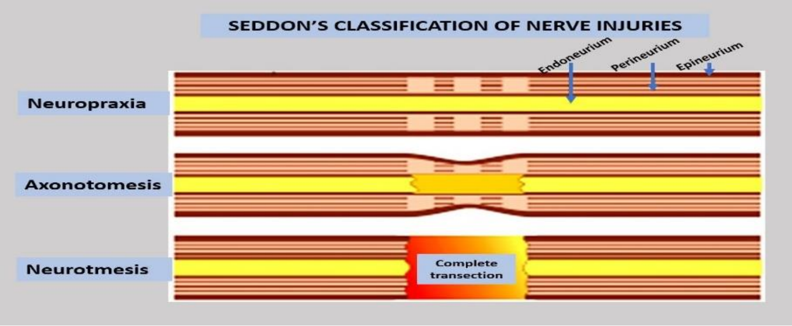
Therefore, the needle can penetrate the **nerve sheath** and **consequently could cause** (1) direct damage of nerve fibres; or (2) **damage of small blood** vessels located within the nerve, leading to intraneural haemorrhage; or (3) **damage of connective** tissues within the nerve, **producing oedema** within the nerve sheath. All 3 events can lead to paraesthesia, in a transient or permanent way

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 anesthesi**



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 **neurotmes**is. Discerning the **differences between the two entities includes:**

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 conc**entration **seems to play an important role for nerve injury**:,
Researchesrs 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 administ**ration, and they should report this **adverse event to their pharmacovigilance service**

Prevention/risk mitigation strategies:

1-Commit to refamiliarization with the **intricate anatomy of the area to include the following**:
The location and trajectory of the inferior alveolar nerve, the lingual nerve, the mental foramen, and recognized anatomical variations in the positioning of these nerves

2-Recall the rich vascularity of the pterygomandibular fossa thereby avoiding hematoma formation and the risk of a life threatening arterial bleed

3-Visualize the positions of the pterygoid, masseter and temporalis muscles to **avoid intramuscular injection** which increases the **risks of trismus** and **the possibility of needle breakage :**

• Select the appropriate needle gauge and **length for a block injection (25-27 gauge**, long) and use a new syringe/needle for additional anesthetic efforts and attempts

4-Do not bend the needle—the needle already deflects when it engages the soft tissues

5-Do not insert to **the hub of the needle**

• Calculate the anesthetic agent dosage in relation to the patient's known anesthetic history, the limits of the informed consent and the planned treatment

6- Adjust for anesthetic agent concentration **4% versus 2%**

7-Consider the effects of other medications on toxicity calculations • Adhere to a disciplined follow- up protocol for every patient and reconsider the implications of informed consent as it relates to anesthesia • In the event of persistent paresthesia, you should:

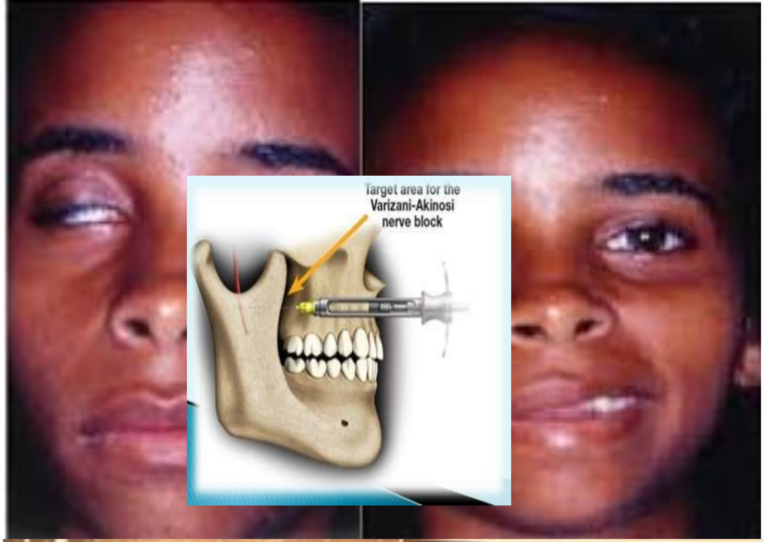
8-Reevaluate the patient at regular intervals o Map, measure, and document (photographs) the numb area to determine relevant changes o Consider referral to a specialist

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, **mandibula**r and **cervica**l **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) .
Peripheral facial nerve palsy can occur during dental treatment, either **immediately** or **delayed**, depending on the time **elapsed from the moment the anesthetic is injected until the onset of symptoms** .

In most cases, **nerve palsy begins instantly after local anesthetic injection into the retromolar region and usually resolves within 12 hours.**



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.

Prevention: Proper care and handling to injection control and cartridge

Management :

1. Reassuring the patient.
2. Contact lenses should be removed.
3. An eye patched should be applied to affected eye or manually close the lower eyelid periodically to keep the cornea lubricated.

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

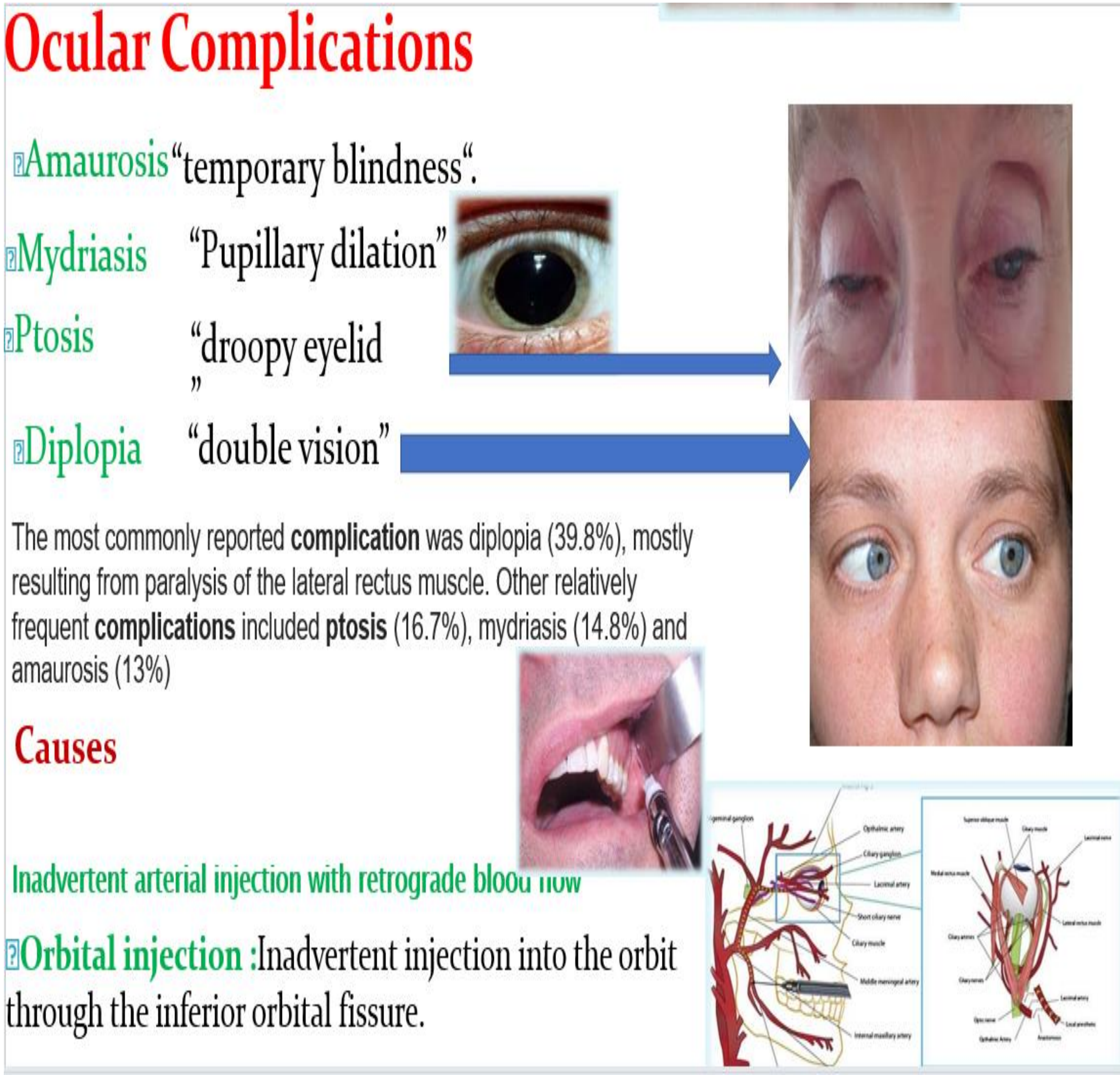
An estimated **1 in 1,000** local anaesthetic injections in the **maxilla or the mandible** lead to unwanted effects on the **ipsilateral eye**

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

Pain and burning sensation on injection:

Pain on injection

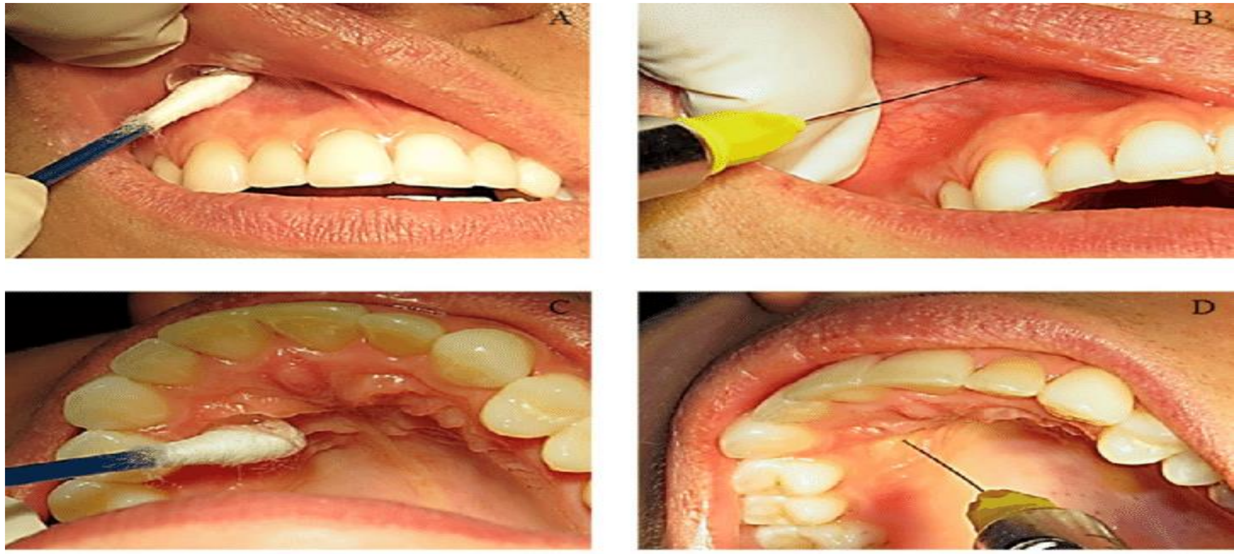
Pain on injection , Pain on injection can be due to **specific circumstances** such as:

1-temperate of the solution, 2-**velocity of i**njection, 3-**dull needles**, 4-**needles with barbs**, 5-**aggressive insertion of the needle**, 6-**damaging soft tissues**, 7-**blood vessels, 8-nerves**, or 9-t**he periosteum** and **causing more pain and other complicati**ons.

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 da**mage

Prevention:

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 injection 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.



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**

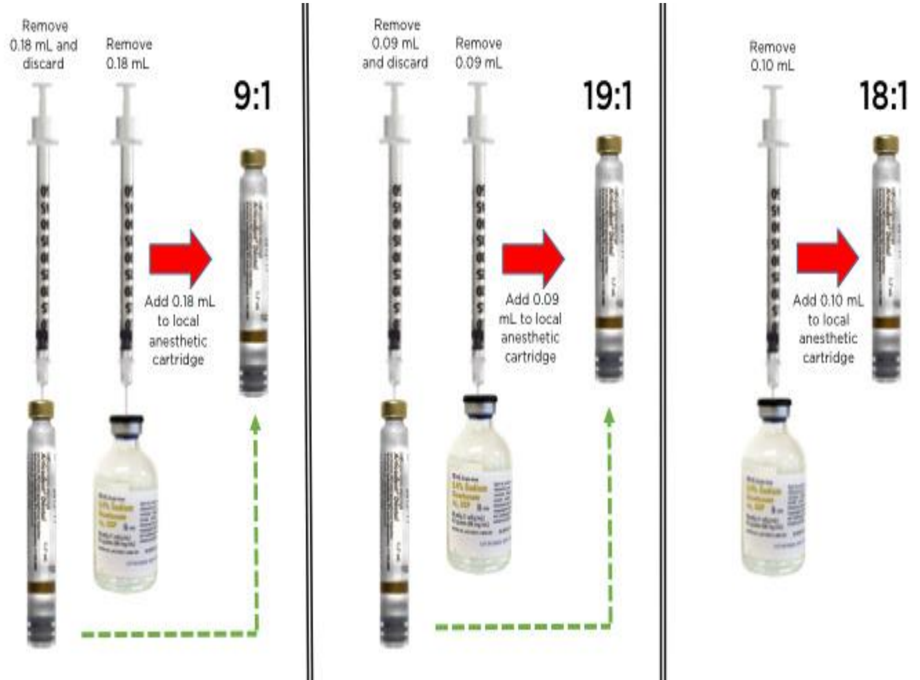
Prevention:

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.

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% $NaHCO_3$]) can considerably minimize pain. 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 milliliters).



The initial poke should be perpendicular to the skin, not angled :

Sensory nerve endings in the skin branch out like a tree. By penetrating at an angle of 90°, the needle intersects fewer nerves

A pause allows the anesthetic to alleviate the initial pain of the injection :

The first 0.2–0.5 millilitres should be injected subdermally, rather than intradermally, followed by a pause. This pause allows the local anesthetic to alleviate the pain caused by the needle being in the skin. More anesthetic can then be injected slowly, before changing the needle’s angle and moving laterally. When properly done, a large wheal will be seen beneath the skin, and the patient will not have felt any pain since the first poke.It is important to ask your patients to tell you if they feel any further pain during the injection.

Keep some visible anesthetic ahead of the needle’s tip.

Almost always, pain after the first poke is caused by the needle tip hitting nerves that have not yet been anesthetized. When moving laterally beneath the skin, it is important to keep five millimetres of palpable or visible local anesthetic ahead of the tip. This technique anesthetizes the subdermal nerve endings and should make a pain-free experience more likely for the patiet

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

Infection :

Infection complication **is rare** since the **usage of disposable needles** and **glass cartridges**.
Infection may extend to tissues by penetration of the needle through a contaminated tissue, because of the needle being contaminated before an **operation or improper preparation of local anesthetic diluted solutions**. On the other hand, **a latent viral infection may be reactivated due to the trauma of the procedure which may be responsible for neural sheath inflammation.**

Prevention :

The area to be **penetrated should be cleaned** with a **topical antiseptic prior to insertion of the needl**e.
Antiseptic mouthwash solutions such as **chlorhexidine gluconate should be considered for all regional techniques**.
The local anesthesia should not be injected through the infected area. Injecting local anesthesia during the presence of **infection is important to increase the pH of anesthetic agent in order to increase efficiency** because the infected **tissue is more acidic.**
This process is called **anesthetic buffering** and leads **to patient comfort during injection**,

Recommendation for treatment of fast onset of anesthesia, and lower postinjection tissue injury.
Recommendation for treatment of infection is antibiotics (**penicillin V 500 mg every 6 hour for 7–10 days**), **analgesics, heat, drainage, and physiotherapy**

Trismus

Trismus is defined as a **painful circumstance** with **inability to open the mouth normally**.
Several factors cause trismus such as:
1- multiple injections in a short period of time in the same area,
2-intramuscular injections inside the muscle or trauma to muscles (either the lateral pterygoid muscle or the temporal muscle) which cause hematoma formation and fibrosis,
3-needle fracture in the muscles inserting to styloid process, I
4-inaccurate positioning of the needle when giving the inferior nerve block or maxillary posterior injections or inflammation of the masseter and other masticatory muscles,
5-a low-grade infection, and excessive volumes of local anesthetic solution deposited into a bounded region which cause expansion of tissues.

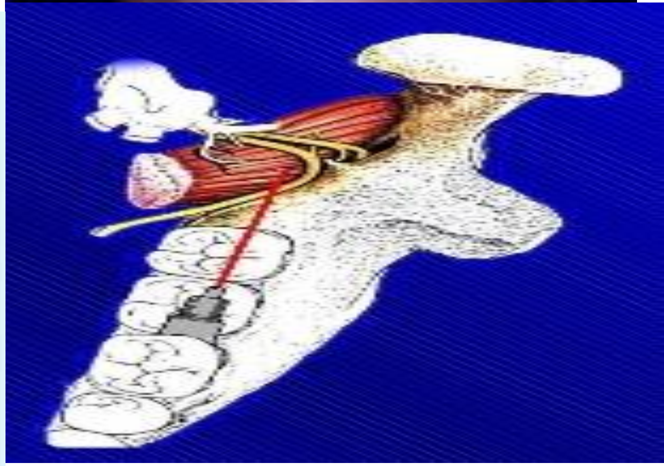
In the acute phase, pain from **hemorrhage leads to muscle contraction** and **limitation of motion.**
Once trismus develops, some cases will resolve spontaneously.
Progression of trismus to **chronic hypomobility** and **fibrous ankylosis** may be ***prevented b***y the early institution of treatment consisting of **heat therapy**; **soft diet**; **prescription of analgesics**, **anti-inflammatory drugs**, **antibiotics, muscle relaxants;** or **physiotherapy**.

Trismus caused by an infection needs to be treated by antibiotics. Usually, trismus will resolve in **6 weeks**, with a range of **4 to 20 weeks.**

Prevention:

Awareness of the **anatomical landmarks** and **muscles: palpation of bony anterior ramus for temporalis muscle**, **pterygomandibular fold for pterygoid muscle**, and **appropriate angulation of the needle and bone contact before injecting are good methods for avoiding trismus via local anesthesia.**

Intraorally the Vazirani-Akinosi technique, the closed-mouth mandibular nerve block technique, or extraoral techniques can **provide anesthesia to trismus patients**



Soft-Tissue Injury

Lip or tongue biting or chewing can occur on **children** with **special ne**eds or **disabled patients**, following dental local anesthesia with the **unfamiliar sensation of being numb** .

Shorter-acting local anesthetics such as **plain mepivacaine** should be **chosen**, and the **patient** or the **guardian** should be **warned about eating**, **drinking hot fluids**, and **biting on the lips** or **tongue to test for anesthesia**; **cotton rolls can be placed between the teeth and soft tissues to prevent chewing**.

In order to accelerate **recovery time for sensation**, an **alpha-adrenergic receptor**, **phentolamine mesylate** (Oraverse), may be injected. ***Oraverse*,** an anesthetic **reversal agent**, can return the patient to normal sensation and function in about half the time.

This first and only proven safe and effective product of its kind can quickly reverse the effects of local dental anesthetics, so patient can carry on **without impairment of talk, smile** and **drink after dental procedures**.

Using dosage:

For adults, the proposed dosage is **1 to 2 cartridges of phentolamine mesylate** (a dose of 0.4 to 0.8 mg), while for children the proposed dosage is **0.5 to 1 cartridge** (0.2 to 0.4 mg).

Prevention :

- A cotton roll placed between the lips and the teeth.
- Warn the patient.
- Self-adherent warning sticker

Soft-Tissue Injury

Management :

Analgesic for pain.
Antibiotic if there is infection.
Warm saline rinse to aid in decreasing the swelling. Petroleum jelly to cover the lesion and minimize the irritation.

Hematoma

Hematoma formation as a complication of **local anesthesia** is the result of a **venous or arterial laceration**; intra-arterial blood pressure increase causes **effusion of blood** into the surrounding soft tissues. While injecting, if there is a high pressure, it may be a warning injecting against the bloodstream.The effusion of the blood into extravascular spaces can result from inadvertently a blood vessel. **Casued** by nicking to the artery or vein. Most occur with IANB and PSA nerve block. 7 to 14 days the hematoma will be presented

Heamatoma

Hematoma formation as a **complication of local anesthesia** is the result of a **venous** or **arterial laceration**; **intra-arterial blood** pressure increase causes effusion of blood into the surrounding soft tissues. **While injecting**, if there is a high pressure, it may be a warning **injecting against the bloodstream**.

The size of a **hematoma depends** on the **density** and **compactness of the affected tissue**; when a vein **rupture is concerned**, **hematoma does not necessarily occur**. Discoloration on the area, a bruise may accompany hematoma

From the anatomical point of view, **different nerve effects cause hematoma on specific regions** such as:

1- anterior superior alveolar (infraorbital) nerve block below the lower eyelid,

2-incisive (mental) nerve block at the chin area,

3-buccal nerve block or any palatal injection within the mouth, and

4-posterior superior alveolar nerve block extraoral in the lower buccal region of the mandible,

5-intraoral distal to maxillary tuberosity.

Prevention :

Hematoma formation can be prevented by :

1. Knowledge of normal anatomy.
2. Use shorter needle for PSA nerve block.
3. Minimize the number of the needle penetration.
4. Never use a needle as a **probe in the tissue**.

5-Aspirating before injecting the anesthetic solution,

Management :

1-When swelling forms immediately after injection, localized pressure should be pplied with a **minimum of 2 minutes. This will stop the hemorrhage.**

2. Apply cold moist towels to affected area each 20 min. every hour.
3. Advice the patient about soreness and limitation of the mouth opening possibility.

Both swelling and **discoloration usually subside in 10 to 15 days**. Ice packs should be held for the first 24 hours after surgery following which intermittent hot moist packs can be used to resolve the condition and massage therapy using a heparin cream. Antibiotics should be prescribed if the hematoma is large in order to prevent the development of a wound infection.

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 desquation

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

• Sterile abscess

1-Secondary to prolong ischemia , lways 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

Toxicity:

Local anesthetics are commonly used in most **medical** and **dental practices**. While **adverse effects are rare**, the rising prevalence of local anesthetics in practice has **resulted in a greater incidence of local anesthetic toxicity**. From **minor symptoms** to **major cardiac or central nervous system (CNS) effects.**
local anesthetic systemic toxicity (LAST) is an important consequence of which to be aware.

Systemic toxicity was originally associated with **seizures** and **respiratory failure**. **Bupivacaine-associated fatal cardiac toxicity** was discovered in healthy adults. This activity reviews the mechanisms, frequency, clinical characteristics, treatment, and prevention of local anesthetic toxicity

Classification of toxicity

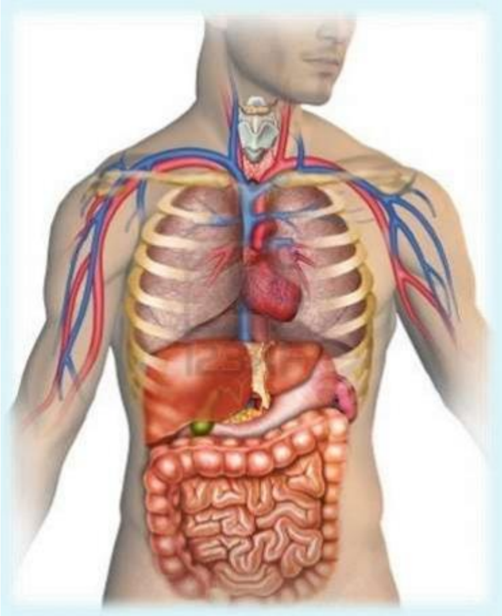
1. Toxicity caused by direct extension of pharmacological effects

A-Side effects B-Over dose C-Local toxic effects

2. Toxicity caused by alteration in the recipient

A-Presence of disease ,B- Emotional disturbances ,C-Genetic aberrations ,D-Idiosyncrasy ,

3. Toxicity caused by allergy to the drug



Local anesthetic systemic toxicity develops when a sufficient (toxic) concentration of anesthetic drug in the blood level reaches to the **central nervous system** and **cardiovascular systems**. **Local anesthetics inhibit** many components of the **oxidative phosphorylation pathway**. Thus LAST affects the 2 organs that are inherently less tolerant of anaerobic metabolism, the **heart**, and **brain.**

Cardiac toxicity is an important component of **local anesthetic systemic toxicity** (LAST), and most instances are related to accidental intravascular injection.
Local anesthetics **bind to and inhibit voltage-gated sodium channels**. Leading to **conduction disturbances, contractile dysfunction**, and **ventricular arrhythmias** that are seen in **local anesthetic-induced cardiac toxicity**.
The incidence of **cardiac toxicity increases** with **bupivacaine, a longer-acting anesthetic**.
Bupivacaine avidly blocks inactive sodium channels during the cardiac action potential at a concentration of 0.2 micrograms/ml.

This is done in a “fast-in/slow-out fashion,” **meaning bupivacaine binds very quickly to a large proportion of sodium channels during the cardiac action potential**, but releases from the **channels slowly during diastole, resulting in a large proportion of the medication accumulating at 60 to 150 beats per minute**.

Lidocaine at 5 to 10 micrograms/ml will also result in substantial sodium channel blockade during a cardiac action potential.

However, in contrast to **bupivacaine, lidocaine** follows the **“fast-in/fast-out”** principle, meaning it releases from sodium channels rapidly during diastole. This allows for a quicker recovery, and a reduced incidence of cardiac toxicity when compared to **bupivacaine.**
Initial symptoms are characterized by **central nervous system signs** such as excitation, convulsions, followed by loss of consciousness and respiratory arrest.

These symptoms are often accompanied by **cardiovascular** signs such as hypertension, tachycardia, and premature ventricular contractions. The clinical signs and symptoms usually show objective symptoms such as **quick talking**, **flicker,** and **tremor in the extremities** .

Predisposing factors are associated with :

Patient Factors:

Age.

Weight.

Medications.

Gender.

Presence of disease.

Genetics

Mental attitude.

Drugs factors:

Vasoactivity.

Concentration.

Dose.

Route of administration.

Rate of injection.

Vascularity of the injection site.

Presence of vasoconsrictor

CNS toxicity is another important consequence of LAST. While it is comprised of many **initial prodromal features**, it most often manifests as **seizures.**

One mechanistic theory is centered around **Twik-related acid-sensitive K+ channels (TASK**). These pH-sensitive channels generate **neuronal potassium "leak" currents**.

Local anesthetic inhibition causes **membrane depolarization** and **increased neuronal excitability**. As these channels are expressed **throughout the brain**, this is the suggested mechanism for **seizures in this setting**.

Overdose reaction is occurring when the drug access to the circulatory system .Normally there is constant absorption of the drug from **its site of admission into the circulatory system** and a steady removal from the blood by the liver

Predisposing Factors

Patient Factors:

Age : The function of absorption, metabolism, and excretion are diminished in old people – increasing the half-life of the drug in circulation blood.

Weight : Greater body weight – larger dose.

Medications : Meperidine”narcotic analgesic ”, phenytoin”anticonvalsun”, quinidine ”antidysrhythmatic ”, and desipramine ”antidepressant” – increase local anesthesia blood level, because protein bending competition.

Gender : Renal function during pregnancy may impaired leading to increase local anesthesia blood level.

In adult women the seizure threshold is 5.8 mg\kg, in newborn 18.4, in the fetus 41.9mg\kg. Placenta clearance of lidocaine.

Presence of disease :

*Hepatic , renal dysfunction and congestive heart failure decrease liver perfusion – **increase amide local anesthesia blood level.***

Prevention:

In order to prevent **systemic toxicity**,
1-the patient should be **evaluated**.
2-The volume of local anesthesia should be **decreased**,
3-young or lightweight patients should not be treated all four quadrants at one visit using local anesthetic alone;
4-accurate and slow injection technique, adjustment of dosage **divided administration** and **aspirating technique**, using agents with **low toxicity** such as **ropivacaine** and **levobupivacain**e, and performing an **aspiration test are recommended .**

Preventing from a toxic dose complication,

1-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-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

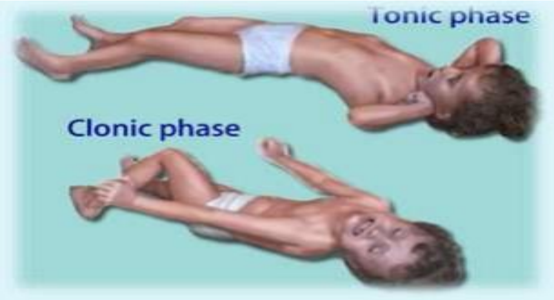
3-Treatment at the office includes **airway support**, **administration of 100% oxygen**, **supine positioning**, and **protection from injury in the event of seizure activity**, treating **convulsions (benzodiazepines** or **thiopental)**

propofol cannot be used in patients with unstable **blood pressure, heartbeat) .**
If severe **hypotension arrhythmia** occurs, administration of the infusion of a 1.5 mL/kg 20% lipid emulsion over approximately 1 minute and then starting with continuous application at 0.25 mL/kg/min = 1000 mL/h.
Studies have reported a resuscitation effect at a total dose of ≤10 mL/kg; therefore, 12 mL/kg can be used as an approximate **estimate for the maximum dose.**

4- use aspiration syringe., use a needle no smaller than 25 gauge.aspirate in at least two planes before injection, slow inject the anesthetic.

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, Myocardiac Depression and cardiac arrest



Management

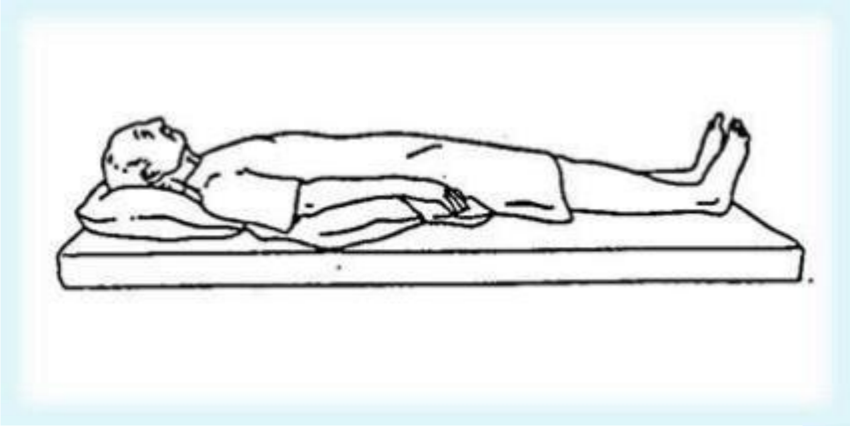
Mild Overdose:

1. **Slow onset (>5 minutes)**
2. **Slow onset (>15 minutes)**

Severe Overdose:

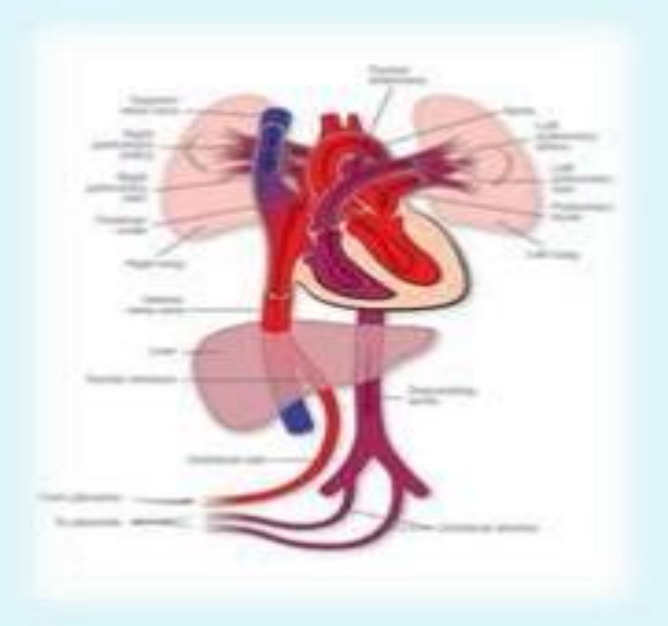
1.Rapid onset (within 1 minute)

2. **Slow onset (5 to 15 minutes)**



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

- 1. **P**osition.
- 2. **C**irculation.
- 3. **A**irway.
- 4. **B**reathing.
- 5. **D**efinitive Care



Mild Overdose: "Patient conscious"

Slow onset (>5 minutes): P-C-A-B-D

Reassure the patient.

Administer oxygen via nasal canal.

Monitor and record vital signs.

IV anticonvulsants (diazepam 5 mg\min. or midazolam 1 m\min.)

Emergency medical assistance before patient discharge.

Severe Overdose: "Patient unconscious"

Rapid onset (within 1 minute) P-C-A-B-D

Protect the patient.

Immediately summon emergency for medical assistance.

Continue Basic life support (BLS)

IV anticonvulsants (diazepam 5 mg\min. or midazolam 1 m\min.) "if seizures protract more than 4 min."


Basic Life Support (BLS)
Collapsed Patient Assessment
D Check for DANGERS
R Check for RESPONSE
S SHOUT for HELP
A Assess & Open AIRWAY
B Assess for NORMAL BREATHING &
C NORMAL signs of life (CIRCULATION) for 10 Seconds
NHS
QE Gateshead

Allergic Reactions

Hypersensitive state, acquired through exposure to a particular allergen.

Allergic reactions cover a broad spectrum od 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 the cartridge**
- **Topical Anesthesia:**

  Mostly ester.

  Preservatives containing such as methylparaben, ethylparaben, or propylparaben

Signs and Symptoms:

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

Allergy reaction to LA

Allergy is also known as hypersensitive reactions, initiated by immunological mechanisms acquired through exposure to a specific allergen; re-exposure to which produces a heightened capacity to react. The prevalence of allergic reactions to amide group local anesthetics is rare. It is predicted that less than 1% of all complications are caused by an allergy.

Many of the **complications doubt to be allergic are actually anxiety-induced reactions** **Ester-type local anesthetics** are more **allergenic than amide-type local anesthetics**. Therefore, amide-type anesthetics are broadly used, among which lidocaine is **the most commonly used for dental anesthesia epinephrine involving form**.

Adverse reactions to local anesthesia are caused by preservatives (e.g., **methyl-p-hydroxybenzoate**), antioxidants (e.g., bisulfate), antiseptics (e.g., chlorhexidine), vasoconstrictor (e.g., sulfites), and other antigens such as latex, as well as local anesthetic **drugs themselves Allergic reactions may include mild symptoms**, such as **urticaria**, erythema, and intense itching, as well as severe reactions in the **form of angioedema and/or respiratory distress.**

Even more severe life-threatening anaphylactic responses include symptoms of apnea, hypotension, and loss of consciousness .

In order to diagnose allergies, the skin prick test is the most endorsed. When skin prick test results are determined to be negative, intradermal testing should be performed for patients who have a history of allergy to local anesthetics intradermal tests become obligatory

The following treatments a local anesthetic patient had tested negative in the allergy tests, should be useThe initial treatment for an allergic reaction in office at the first step should be the removal of the causative agent. For the management of mild symptoms, oral or intramuscular antihistamine-diphenhydramine (Benadryl), 25 or 50 mg, should be given. Additionally, hydrocortisone cream may be prescribed to relieve skin itching or erythema. In life-threatening cases basic life support, intramuscular or subcutaneous epinephrine 0.3–0.5 mg, and hospitalization services should be given.

Anaphylaxis is an **acute potentially life-threatening hypersensitivity reaction**. The clinical symptoms of anaphylaxis are depending on the **organ systems involved**.

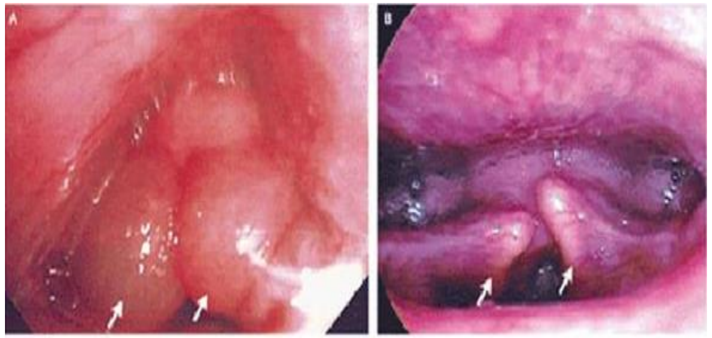
Uncontrolled **co-existing asthma**, **mast cell disorders**, and patients with specific allergens such as **peanut** and **tree nut allergy** are the **risk factors for anaphylaxis**. **Respiratory reactions:**

Signs and Symptoms:

• Fever • Edema • Urticaria • Dermatitis • Bronchospasm • Systemic Anaphylaxis Respiratory distress Dyspnea Wheezing,Flushing, Cyanosis , Perspiration ,Tachycardia, Anxiety,

Laryngeal edema :

- Extension of edema to the larynx
- Life threatening emergency.



Emergency management of anaphylaxis in the office:

Steps of management :

1-The patient should lie flat, but also in the case of breathing difficulty, the patient is allowed to sit.

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.

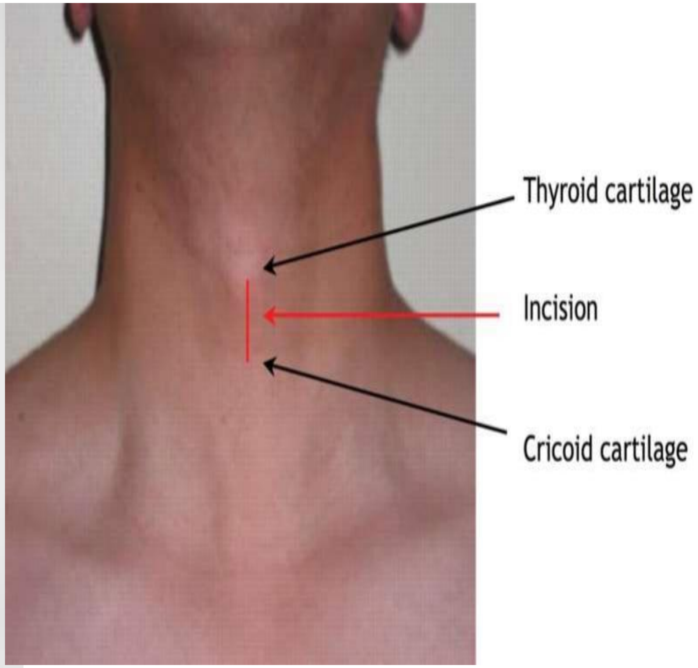
Adrenaline should be administered for anaphylaxis by intravenous (IV) route only in the case of profoundly hypotensive patients or **patients who develop a cardiopulmonary arrest** or **those who fail to respond to multiple doses of IM adrenaline because of the potential cardiovascular adverse effects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

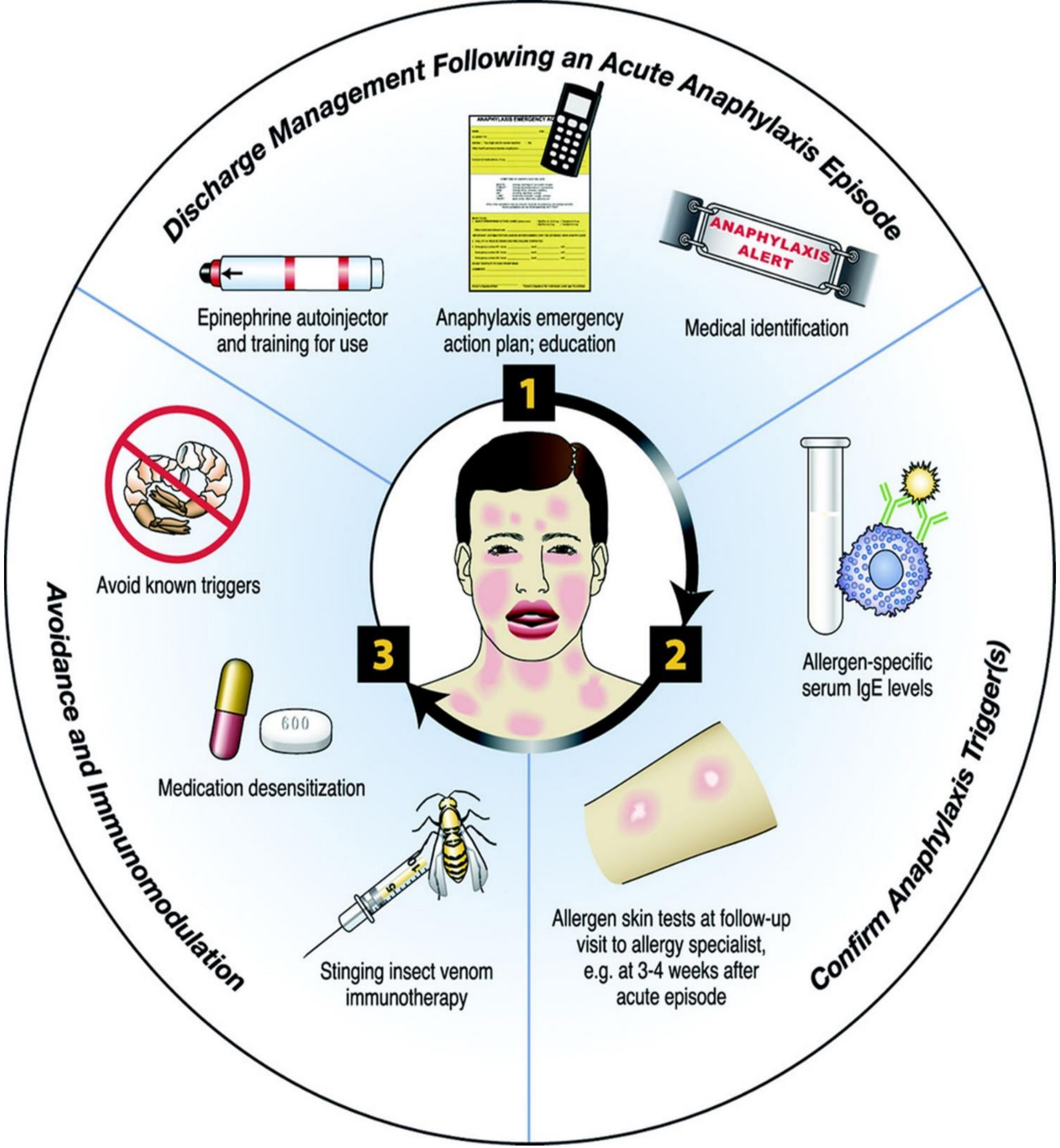
Intramuscular injection is the **first line of treatment** in the treatment of anaphylaxis. But the use of **antihistamines** and **glucocorticoids is controversial**.

Antihistamines is not effective as adrenaline on upper or lower airway obstruction, hypotension, or shock, but it decrease the side effects **urticaria**, **flushing**, **headache**, **hypotensio**n, and **rhinorrhea.**

Glucocorticoids were reported to be the second most widely available medications (after epinephrine) for anaphylaxis treatment globally, even though some **claim glucocorticoids have no proven benefit in anaphylaxis**

As a result first step of treatment must be **epinephrine** additionally **glucocorticoids** and **antihistamines** may use to treat severe systemic reactions.





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Academic Year 3

Medical Emergencies During Dental Treatment

Assistant Prof Dr. Hamid Hammad Enezei

Ph.D in Oral & Maxillofacial Surgery

Medical emergencies during dental treatment

- Overview of medical emergencies
- Basic measures, equipment and drugs
- Common emergencies

✓ Collapse
✓ Anaphylaxis
✓ Cardiac arrest
✓ Diabetic collapse due to hypoglycemia
✓ Fits and convulsions
✓ Adrenal crisis
✓ Acute severe asthma
✓ Chest pain

Medical emergencies during dental treatment

Medical emergencies that have occurred in dental practices include **vasovagal syncope, angina, hypoglycaemia, epileptic seizures, choking, asthma, anaphylaxis and cardiac arrest**.

It is estimated that, on average, a general dental practitioner (GDP) will experience a medical emergency at least **once every two years**

Common emergencies

Sudden collapse

Adrenal crisis

Anaphylaxis

Cardiac arrest

Diabetic collapse due to hypoglycemia

Fits and convulsions

Acute severe asthma

Chest pain

Syncope

Risk management can play an important part in reducing the risk of medical emergencies in the dental practice. It is therefore recommended that all primary care dental facilities have a process for medical risk assessment of their patients.

Anticipation of potential medical emergencies that may arise should be highlighted by taking a thorough medical history which is revised, updated and checked each time the patient presents for treatment. It has been suggested that the presence of an updated medical history may help to minimize the risk of a medical emergency occurring.

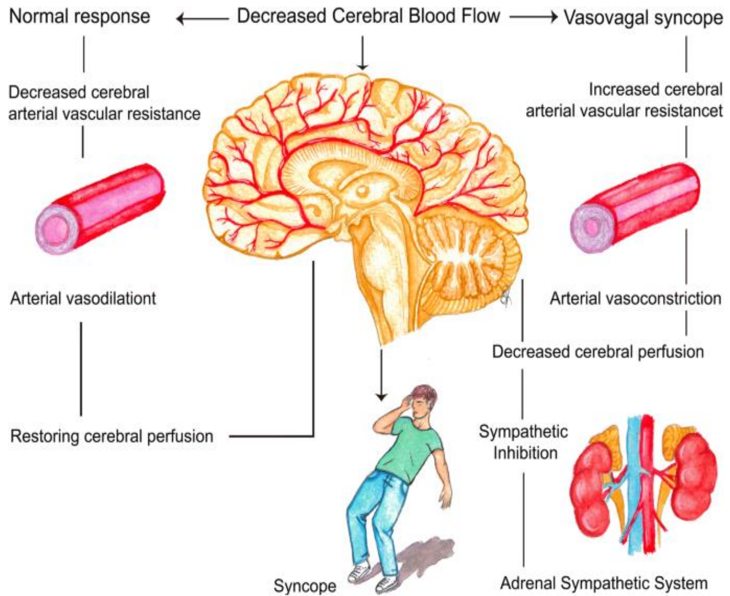
Importance of training:

It is essential that all **dental professionals** must be trained in dealing with **medical emergencies**, including **resuscitation**, and **possess up to date evidence of capability .**

1-Planning ahead, **there should be at least two people available within the working environment to deal with medical emergencies when treatment is scheduled to take place** (in exceptional circumstances, the second person could be **a receptionist** or **a person accompanying the patient**).

2 -Members of the dental team **must maintain their knowledge** and **competence to effectively** and **safely deal with a medical emergency**, an **important aspect of all dental professionals' continuing professional development (CPD) once a year .**

Vasovagal syncope is the most common emergency encountered. **Interestingly**, one study showed that 4.8% of all GDPs observed 22% of all syncopes and that the prevalence of vasovagal syncope tends to decrease with **professional experience**



Basic measures, equipment and drugs

Recommended practice

Practices should ensure they have emergency medicines and equipment to keep patients safe.

Professional guidelines: **NICE: Prescribing in dental practice (Medical emergencies in dental practice)**

1.These should be available to manage common medical emergencies:

1. adrenaline/epinephrine injection, adrenaline 1 in 1000, (adrenaline 1 mg/mL as acid tartrate), 1 mL amps
2. aspirin dispersible tablets 300 mg
3. glucagon injection, glucagon (as hydrochloride), 1 - unit vial (with solvent)
4. glucose (for administration by mouth)
5. glyceryl trinitrate spray
6. midazolam oromucosal solution treated epilepsy
7. medical oxygen
8. salbutamol aerosol inhalation, salbutamol 100 micrograms/metered inhalation.

Professional guidelines: **Quality standards: Primary dental care**

2.This is the minimum equipment recommended:

2. adhesive defibrillator pads
3. automated external defibrillator (AED)
4. clear face masks for self-inflating bag (sizes 0,1,2,3,4)
5. oropharyngeal airways sizes 0,1,2,3,4
6. medical oxygen cylinder
7. oxygen masks with reservoir
8. oxygen tubing

2. pocket mask with oxygen port
3. portable suction, for example Yankauer
4. protective equipment – gloves, aprons, eye protection
5. Razor or blade
6. scissors
7. self-inflating bag with reservoir (adult)
8. self-inflating bag with reservoir (child)
9. if there are ampules in the medical emergency drugs kit, there must be adequate numbers of suitable needles and syringes.

3.Oxygen cylinders should be easily portable but must allow adequate flow rate (for example 15 L min) for thirty minutes or until the arrival of emergency services. Local policy should dictate the precise size of cylinder and whether a second cylinder is needed in case the first one is at risk of running out.

4.Quality Assurance Process: **At least every week, check:**

4. expiry dates for emergency medicines
5. equipment and availability of oxygen. The manufacturer’s instructions must be followed about the use, storage, servicing and expiry of equipment. A planned replacement programme should be in place for disposable equipment items that have been used or that reach their expiry date.

Adrenal crisis

People with **Addison's disease** must be constantly aware of the risk of a **sudden worsening of symptoms**, called an **adrenal crisis**. This **can happen when the levels of cortisol in body fall significantly**. An adrenal crisis is a medical emergency. **If left untreated, it can be fatal. Patient all the time under steroid therapy**

Steroid supplementation is required **before the dental treatment to prevent adrenal crisis** in a patient with primary adrenal insufficiency (for example, Addison's disease) undergoing a dental procedure:

•Minor dental procedures (for example, scale and polish, replacement filling) - advise the patient to take an additional oral dose of glucocorticoid one hour before their appointment

•More complex dental procedures (for example, root canal work under local anaesthetic) - prescribe steroid supplementation before the procedure and for a full 24 hours afterwards

•Dental extractions and minor oral surgery - refer to secondary care.

Patients with primary adrenal insufficiency should be **advised to bring their emergency hydrocortisone injection kit to** all dental appointments, as well as their **personalised adrenal crisis letter which provides medical treatment guidance.**

If the patient develops adrenal crisis, call **health emergencey line** immediately, stating 'Addisonian crisis', and if the patient's emergency hydrocortisone injection kit is available, administer intramuscular (IM) hydrocortisone. The recommended dose, which should be stated on the patient's adrenal crisis letter, depends on the **patient's age:**

•Adults: 100 mg

•Children six years of age or older: 50-100 mg (use clinical judgement depending on the age and size of the child)

•Children one to five years of age: 50 mg

•Infants up to one year of age: 25 mg.

Anaphylaxis

Anaphylaxis is a body reaction to some thing **causes the immune system to release a flood of chemicals that can cause you to go into shock** — blood pressure drops suddenly and the airways narrow, blocking breathing. Signs and symptoms include a **rapid**, **weak pulse**; a **skin rash**; and **nausea and vomiting.**

Anaphylaxis is likely when all of the following 3 criteria are met:

•**Sudden onset and rapid progression of symptoms.**

•The person will feel and look unwell.

•Most reactions occur over several minutes. Rarely, reactions may be slower in onset.

•The time of onset of an anaphylactic reaction will depend on the type of trigger. For example, an intravenous trigger will cause a more rapid onset of reaction than stings, which tend to cause a more rapid onset than orally ingested triggers.

•The person will appear anxious and can experience a '**sense** of impending doom'.

Anaphylaxis is a clinical diagnosis; a precise definition is not important for treatment.

Recognise anaphylaxis based on:

•Sudden onset and rapid progression of symptoms

•Airway and/or Breathing and/or Circulation problems

•Skin and/or mucosal changes (flushing, urticaria, angioedema) – but these can be absent in up to 20 per cent of cases.

Signs and symptoms

•**Airway problems:** lip and tongue swelling/angioedema, nasal congestion, sneezing, tightness of throat/hoarse voice/stridor
•**Breathing problems:** increased respiratory rate, bronchospasm causing wheeze, increased mucous secretions, exhaustion, cyanosis, respiratory arrest
•**Circulation problems:** low blood pressure, fast heart rate (tachycardia), arrhythmia, chest pain, cardiac arrest
•**Disability/Neurological problems:** confusion, agitation, loss of consciousness
•**Exposure – skin and mucosal:** urticaria, erythema, pruritus
•**Gastrointestinal:** stomach cramps or spasm, nausea, vomiting, diarrhoea
•**Other:** feeling of impending doom

Differential diagnosis:

Commonly anaphylaxis **can be mistaken** or **confused for:**
•Asthma (can present with similar symptoms and signs to anaphylaxis, particularly in children)
•Septic shock (hypotension with petechial/purpuric rash)
•Vasovagal episode
•Panic attack or hyperventilation syndrome
•Idiopathic (non-allergic) urticaria or angioedema

Management

Scenario: Angio-oedema without anaphylaxis

How should I manage a person with angio-oedema without any airway symptoms or signs of circulatory collapse?

For people with rapidly developing angio-oedema *without* anaphylaxis:

•Give **slow intravenous (IV)** or intramuscular (IM) chlorphenamine and hydrocortisone.

•Arrange emergency hospital admission.

•Arrange a review after the person has been discharged from hospital.

For people with stable angio-oedema *without* anaphylaxis:

•**Try to identify the underlying cause so that further episodes can be avoided.**

- For people taking an angiotensin-converting enzyme (ACE) inhibitor:
  - Stop treatment immediately, and consider starting an alternative drug treatment.
  - If possible, avoid angiotensin-II receptor antagonists as these can also trigger episodes of angio-oedema.

•**For people with mild symptoms of angio-oedema,** treatment may not be needed.

•**For people with symptoms requiring treatment:**

- Offer a **non-sedating antihistamine** (such as cetirizine, fexofenadine, or loratadine) for **up to 6 weeks** (use clinical judgement to determine the duration of treatment).
- If symptoms are severe, give a short course of an **oral corticosteroid** (for example prednisolone 40 mg daily **for up to 7 days**) in addition to the non-sedating oral antihistamine.
- Advise the person to seek immediate medical help (by dialling EMERGENCEY LINE or attending A&E) if symptoms progress rapidly or i**f symptoms of anaphylaxis develop.** e.

- **Refer the person to a dermatologist or immunologist if:**
    - Hereditary or acquired angio-oedema is suspected.
    - Symptoms persist or reoccur 3 months after stopping treatment with an ACE inhibitor.
    - The cause of angio-oedema is not identifiable or avoidable.
- **For people awaiting specialist review who are at risk of anaphylaxis,** seek specialist advice about prescribing an adrenaline auto-injector device to be used in the event of anaphylaxis before their hospital appointment.
    - People at risk of anaphylaxis include people with co-existing asthma, chronic obstructive pulmonary disease, or heart disease, people who have experienced angio-oedema with trace amounts of an allergen/trigger, and people who cannot easily avoid an allergen.
    - If an adrenaline auto-injector device is indicated in a person taking a beta-blocker, consider discontinuing the beta-blocker if possible, as it can interfere with the action of adrenalin

Scenario: Anaphylaxis with or without angio-oedema

If a person presents with features of anaphylaxis with or without angio-oedema:

- **Treat as a medical emergency.** Call for an ambulance and ask for help from colleagues.
- **Assess the person.**
    - Look for and relieve airway obstruction.
    - Check for normal breathing.
    - **If the person is unresponsive and not breathing normally:**
        - Start cardiopulmonary resuscitation (CPR) immediately.

•Repeat the **IM adrenaline dose after 5 minutes** if there is no improvement in the person's condition.

•If there is no improvement after **two IM doses**, give **IM adrenaline every 5 minutes until there has been an adequate response.**

•**Do not give intravenous (IV) adrenaline in primary care.**

•**Remove the trigger if possible.**

•For example, remove the stinger after a bee sting. Early removal is more important than the method of removal.

•After food-induced anaphylaxis, attempts to make the person vomit are not recommended.

•Do not delay definitive treatment if removing the trigger is not feasible.

•**Where skills and equipment are available:**

•**Give oxygen** at the highest concentration possible as soon as available using a mask with an oxygen reservoir.

•Adjust the inspired oxygen concentration to achieve an **oxygen saturation of 94-98%** (in people at risk of hypercapnic respiratory failure, consider a target range of 88-92%).

•**Attach monitoring (pulse oximetry**, **blood pressure**, **ECG)** as soon as possible — this will help assess the person's response to adrenaline.

•In the presence of hypotension/shock, or poor response to an initial dose of adrenaline obtain IV access and give a rapid IV fluid bolus (for example, with Hartmann's or normal saline) using 500–1000 mL in an adult, or 10 mL/kg in a child. Give further fluids as necessary.

•**Consider inhaled salbutamol or ipratropium therapy if the person is wheezy** (especially in people with known asthma).

•**Arrange a review after the person has been discharged from hospital.** See the section on Follow up for more information.

•Ensure that help is on its way as early **advanced life support is essential.**
•**For people who do not require CPR:**
•Examine the chest for signs of lower and upper airway obstruction.
•Check the pulse and blood pressure for signs of circulatory collapse.
•Check the skin and inside the mouth for urticaria and angio-oedema.

•**Place the person in a comfortable position — take into account the following factors:**
•Fatality can occur within minutes if the person stands, walks or sits up suddenly. They must not walk or stand during acute reactions. Use caution when transferring people who have been stabilized.
•People with airway and breathing problems may prefer to be in a semi-recumbent position, as this will make breathing easier.
•Lying flat with or without leg elevation is helpful for people with low blood pressure.
•Place people who are breathing normally and unconscious on their side (recovery position). Monitor breathing continuously and prepare to intervene if this changes.
•Pregnant women should lie on their left side to prevent aortocaval compression.

•**Give intramuscular (IM) adrenaline 1:1000 as per age-related guidelines.**
•The injection can be given in the anterolateral aspect of the middle third of the thigh (ideally) or arm, depending on access.
•Assess the response to treatment after 5 minutes — measure vital signs (respiratory rate, oxygen saturations, heart rate, BP, level of consciousness) if possible and auscultate for wheeze.

Cardiac arrest:

As oral health professionals, being prepared can spell the difference between life and death for patients experiencing a cardiac emergency in the dental office. The general populations’ increasing overall age (and subsequent medical involvement) raise the probability of encountering sudden cardiac arrest in **oral health care settings**.
Once the heartbeat becomes **arrhythmic**, **defibrillation** is the only **resolution**. An automated external defibrillator (AED) is an essential tool in every dental office to manage these situations.

Early defibrillation with these easy-to-operate devices will convert **two of the most common lethal cardiac dysrhythmias**, **ventricular fibrillation** and **ventricular tachycardia**, into a **normal sinus rhythm** and **restore oxygen perfusion to vital organs**.

Survival rates decline by 7% to 10% with each passing minute an affected individual goes without defibrillation.

Sudden cardiac arrest and sudden cardiac death (SCA/SCD) involve the rapid arrest or death of an individual due to cardiovascular-related issues.

The heart's electrical rhythm becomes chaotic — either through **ischemic provocation** or **another arrhythmic trigger** — and **ultimately stops beating**.
Congestive heart disease is the most common underlying pathology for **sudden cardiac arrest** in adults over the age of 35.
Risk factors for congestive heart disease, **including diabetes mellitus**, **obesity**, **hypertension** and **high cholesterol**, have a **direct relationship with these events**.

Diabetes plays a role in the development of **cardiovascular disease** by increasing the amount of blood and workload placed on the heart. Its chambers are damaged through **excessive dilation** and **hypertrophic enlargement**, making **each pump strenuous**.
Obesity increases the **incidence of lethal arrhythmia** and **atrial fibrillation** by structurally **damaging the heart**.
When **obesity** and **hypertension coexist**, the heart grows even larger as its **chamber volume is filled beyond c**apacity.
Sudden cardiac death in young people, who may be affected by congenital cardiac structural abnormalities, is typically **associated with high cholesterol.**

Dental teams should be able to **recognize the physical manifestation of cardiac arrest** or **clinical death**, including loss of **consciousness**, **sudden collapse**, or a **lack of pulse and breathing**. Making certain that staff members hold current **Basic Life Support (BLS) for Healthcare**

Providers certification, and equipping all dental facilities with an AED, emergency kit and monitoring equipment — as well as developing and rehearsing an emergency action plan — will provide these patients with the

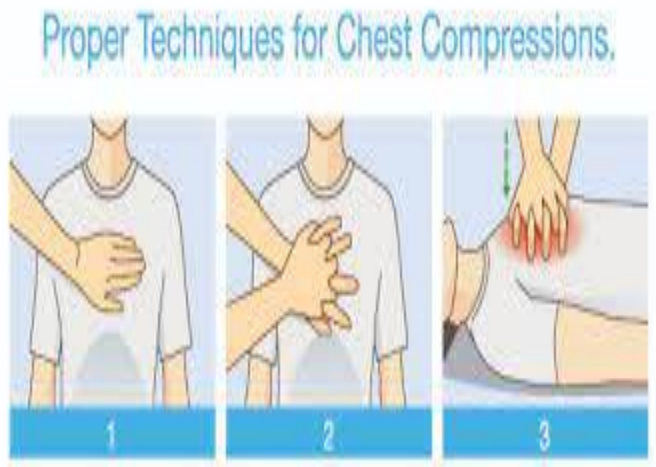
Dental staff should be suspicious of cardiac arrest in any patient presenting with seizures. On confirming cardiac arrest, stay with the patient and start chest compressions immediately while asking colleagues to call health emergency line for an **ambulance** and to **fetch automated external defibrillator (AED)/resuscitation equipment**

High-quality chest compressions

High-quality CPR performance metrics include: **Chest compression fraction >80%** Compression rate of 100-120/min.

Compression depth of at least 50 mm (2 inches) in adults and at least 1/3 the AP dimension of the chest in infants and children

•Start chest compressions as soon as possible.
•Deliver compressions on the lower half of the sternum ('in the center of the chest').
•Compress to a depth of at least 5 cm but not more than 6 cm.
•Compress the chest at a rate of 100–120 min with as few interruptions as possible.
•Allow the chest to recoil completely after each compression; do not lean on the chest.
•Perform chest compressions on a firm surface whenever feasible.



Rescue breaths
•If you are trained to do so, after **30 compressions**, provide **2 rescue breaths**.
•Alternate between providing **30 compressions** and **2 rescue breaths.**
•If you are **unable or unwilling to provide ventilations**, give **continuous chest compressions.**

Compressions before defibrillation

•Continue CPR until an AED (or other type of defibrillator) arrives on site and is switched on and attached to the person.

•Do not delay defibrillation to provide additional CPR once the defibrillator is ready.

Fully automatic AEDs

•If a shock is indicated, fully automatic AEDs are designed to deliver a shock without any further action by the rescuer. The safety of fully automatic AEDs has not been well studied.

Safety of AEDs

•Many studies of public access defibrillation have shown that AEDs can be used safely by bystanders and first responders. Although injury to the CPR provider from a shock by a defibrillator is extremely rare, do not continue chest compression during shock delivery.

Safety

•Make sure you, the person and any bystanders are safe.

•Members of the public should start CPR for presumed cardiac arrest without concerns of causing harm to those not in cardiac arrest.

•Members of the public may safely perform chest compressions and use an AED as the risk of infection during compressions and harm from accidental shock during AED use is very low.

automated external defibrillator

Diabetic collapse due to hypoglycemia

Dental practices have a mandatory requirement to keep **medication and equipment to monitor and treat hypoglycemia**.

This includes **blood glucose monitors**, **oxygen and masks**, **glucose tablets**, **glucogel** and **glucagon**.


If a patient is **showing signs of low blood sugar** or the results of a **blood glucose test are less than 3mmol per liter** then the **following regime should be adopted**.

If a patient is conscious and cooperative give them a glucose drink or tablets to chew.

If the patient is conscious but uncooperative then administer **a tube of glucose gel in the buccal sulcus**.

If however, the patient is **unconscious then call the ambulance and administer glucagon in**tramuscularly.
Glucagon is a hormone which helps to **raise blood glucose levels**. It is the **opposite of insulin which lowers blood glucose levels**.
Glucagon kits consist of a vial of 1 mg of freeze-dried glucagon and a **syringe containing a 1 ml of diluting solution**.

The kit should be **stored at a temperature of 2–8°C in a refrigerator**, but it must **not be frozen**. If stored in the refrigerator the shelf life from the manufacturer is 36 months.

Glucagon kits can be stored outside the refrigerator at a temperature not exceeding 25°C for 18 months provided that the expiry date is not exceeded.

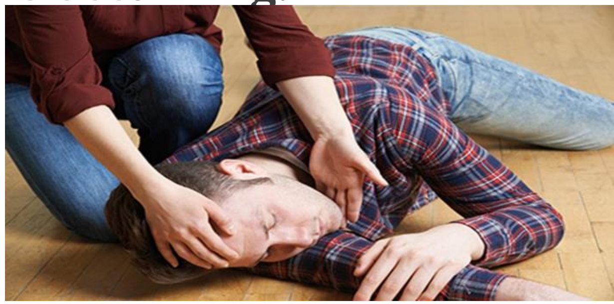
It should be stored in the original package in order to **protect from light**. This is common practice as it needs to be easily accessible for emergency use.

However DENTAL PROFESSIONAL must be able to demonstrate either when the product was out of refrigerated storage, for example, label the product with the date it was taken out of the fridge or a **revised expiry date**, or how the product is safe for use, for example by referring to the purchase invoice showing that 18 months has not elapsed from delivery.

In addition, if you are storing the glucagon kit in the fridge you must ensure that the fridge is monitored and that weekly checks are carried out to limit the temperature range between 2-8 degrees centigrade. No food items or laboratory items should be stored in the same fridge.

Once glucagon has been administered, oxygen can be given and the patient monitored with special attention to the airway. On recovery, **a glucose drink and a complex carbohydrate can be given whilst awaiting the ambulance services.**

As always it is better to carry **out risk assessments for diabetic patients and a detailed medical history and checking whether patients have eaten before appointments will often prevent problems occurring.**



Fits and convulsions

A seizure is defined as **a fit** or **convulsion** that occurs when a sudden burst of electrical activity in the brain temporarily interferes with the normal messaging processes. The brain controls the whole body, so depending on where the **seizure occurs in the brain**, different parts of the body may be affected.

There are many different types of **seizures** and a **multitude of causes**. Any head injury or stress to the brain can cause fitting, as can **brain tumours**, **meningitis**, **malaria**, **eclampsia in pregnancy**, **poisoning**, **lack of oxygen, raised body temperature**, **epilepsy as well as drug and alcohol use and withdrawal.**

It is possible that **someone experiencing a cardiac arrest may appear to have a seizure as their brain struggles with depleted oxygen – always ensure they are breathing.**

It is particularly common for **babies** and **young children** to experience febrile convulsions. These are seizures triggered by a rise in temperature when the child is unwell. **These seizures don't usually cause any long-term problems** and **the child usually grows out of their pre-disposition.**

Observing how someone behaves during a seizure can often be extremely helpful to aid the neurologist's diagnosis and treatment.

Different types of seizures

Fits, **seizures** or **convulsions** can take many forms. They may cause rigid out of control movements; the casualty may experience absence seizures where they become rigid and unresponsive; there may be full thrashing around **tonic/clonic fits**; or anything in between.

How to help someone experiencing a ***tonic-clonic fit*** *or* **generalized seizure**

What might happen

Tonic phase – the casualty will collapse as they lose consciousness. The body goes stiff and rigid and they may cry out as if in pain. This is due to an involuntary action as the muscles force air out of the lungs – the casualty is not in pain and is usually unaware of the noise they are making. They can begin to appear blue around their mouth and finger tips.

Clonic phase – They may rigidly jerk around as the muscles alternately relax and tighten. They may make a snoring noise as the tongue flops to the back of the airway; they could be incontinent and might bite their tongue.

Post-ictal phase – Once the jerking stops, they may be confused, sleepy, agitated or relatively unresponsive (if you are worried about their airway put them into the recovery position).

Help for a generalized seizure in a dental practice

1-Clear all dental instruments away from the patient.

2-If the patient is on the dental chair: place the dental chair in a supported, supine position as near to the floor as possible. If the patient is not on the chair, ease them onto the floor and protect their head from injury by gently cushioning with a pillow or coat. Move things away from them to protect from injury. If dental treatment has begun, try and ease the patient onto their side to reduce the possibility of them aspirating secretions and recent dental work.

3-Do not restrain the patient.

4-Do not put your fingers in his or her mouth (you might be bitten), or try and put anything in their mouth for them to bite on.

5-Time the seizure. Call the emergency services if the seizure lasts longer than five minutes or the patient experiences repeated seizures.
6-Loosen any tight clothes around their neck and loosen belts. **Protect the patient's dignity.**
7-Call the emergency services if the patient **looks cyanotic** [blue] from the onset.
8-Administering **oxygen** may be helpful.
9-If the seizure **lasts longer than five minutes** or **for repeated seizures**, administer **buccal midazolam** or appropriate emergency **anti-epileptic drugs** from your emergency drugs. **Contact the emergency services if unsure**.
10-Be aware of the possibility of compromised airway or uncontrollable seizure.

Once the seizure has finished, place the patient in the **recovery position**, on his or her side. These guidelines should also be followed:

1-Do not undertake further dental treatment that day.
2-Try to talk to the patient to evaluate the level of consciousness during the post-ictal phase.
3-Do not attempt to restrain the patient, as he or she might be confused.
4-Do not allow the patient to leave the practice until you are sure they have made a full recovery.
5-Contact the patient's family, if he or she is alone.
6-Do a brief oral examination for sustained injuries.
7-Depending on post-ictal state, discharge the patient home with a responsible person, to his or her family physician or call the emergency services.

Acute severe asthma

An acute episode of asthma in the dental office **may be precipitated by extrinsic factors such as inhaled allergens, as well as intrinsic factors such as fear or anxiety**.
An asthma episode should be **considered a medical emergency** and must be **treated promptly by inhalation of a bronchodilating agent.**
A history of asthma in the dental patient should alert the dentist to implement strategies that may prevent an acute attack and to be prepared to manage this potentially life-threatening medical emergency appropriately. The chronic use of **bronchodilating inhalers** and/or **glucocorticoids for the management of asthma** can increase the likelihood of **oral candidiasis**, particularly in **patients who have additional risk factors** such as **smoking**, **denture use**, or the use of **xerostomic medication**

GUIDELINES TO BE FOLLOWED

A) BEFORE TREATMENT

1.Patients appointment should be late morning or afternoon.
2.Assess severity of ASTHMATIC condition.
3.Consider antibiotic prophylaxis for immuno-suppressed patients.
4.Consider corticosteriod replacement for adrenally suppressed patients.
5.Avoid using dental materials that may elicit an ,**Asthmatic attack** i.e. dentifrices ,Fissure sealants ,Methyl meth acrylate, Fluoride trays & cotton rolls can trigger Asthmatic events.

6.If asthmatic patients does not use a broncodilator ,make sure the emergency kits has both a **bronchodilator & oxygen.**

B) DURING TREATMENT

1.Rubber dams should be used cautiously.

2.**Use technique to reduce patient stress:**

•Avoid **prolonged supine positioning**

•Avoid **nitrous oxide** in people with severe ASTHMA.

•Avoid using BARBITURATES.

3.Avoid using LA containing **SODIUM METABISULFIDE**.

4.Use vasoconstrictor judiciously.

C) AFTER TREATMENT

1. Be cautious while prescribing **NSAID** to ASTHMATIC patient.

2.TETRACYCLINE should be used cautiously.

3.Avoid use of ERTHROMYCIN in patients taking THEOPHYLLINE.

4.Avoid use of PHENOBARBITALS in patients taking THEOPHYLLINE.

5.**Analgesic** of choice for these patients is ACETAMINOPHEN.

WHAT TO DO DURING ACUTE ASTHMATIC ATTACK IN DENTAL CLINIC OR HOSPITAL?

1. Allow patient to **assume a comfortable position** & discontinue the dental procedure.

2.Establish & maintain patient **airway & administer beta2 agonists** VIA inhaler or nebulizer.

3.Administer **oxygen 6-10 liters** VIA face-mask, nasal hood or cannula.

if no signs of improvement is absorbed & symptoms are worsening ,administer EPINEPHRINE subcutaneously (1:1,000 solution ,0.01 milligram/kg of body weight to a maximum dose of 0.3mg)

4.Alert medical emergency service.

5.Maintain a **good oxygen level until the patient stops**,**wheezing** /or medical assistance arrives.

6.Begin basic life support C,A,B & D 's activity as needed.

Acute chest pain :

The most common causes of acute chest pain encountered in dental situations include **hyperventilation, pulmonary embolism, angina pectoris and myocardial infarction**. Stress and fear often **cause rapid breathing** or **hyperventilation** .

Cause:

Diminished blood supply to myocardium due to an imbalance between myocardial oxygen supply and demand.

Signs/Symptoms:

Chest pain – substernal pressure or crushing sensation. May radiate to neck, left shoulder and down arm, and left side of jaw. About 10% of heart attacks report **jaw pain** as a symptom. Though, that is usually **throughout the jaw** and not localized to a single tooth.

The most common description is pain throughout the lower jaw. Some people describe this pain but don't have any accompanying chest pains, yet they were still having a heart attack. It is always better to have pain looked at. Each person's anatomy and responses vary. There isn't a single common symptom to look out for.

Some Other Symptoms of a Heart Attach

•Pressure, tightness, pain, or a squeezing or aching sensation in your chest or arms that may spread to your neck, jaw or back

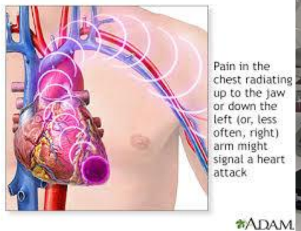
•Nausea, indigestion, heartburn or abdominal pain

•Shortness of breath

•Cold sweats

•Fatigue

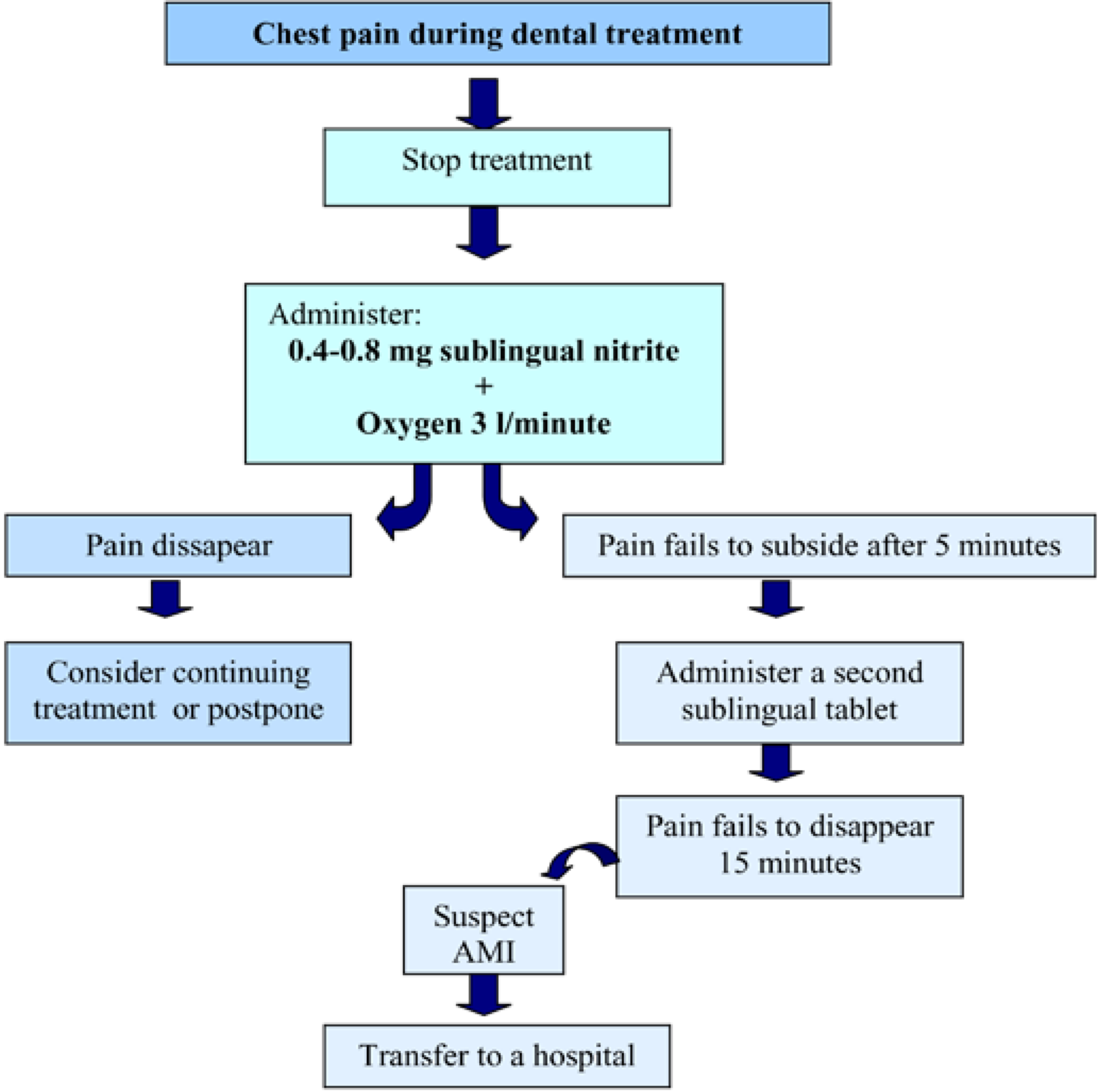
•Lightheadedness or sudden dizziness



The two cardiovascular conditions that cause most deaths are ischemic heart disease and cerebrovascular disease, with heart failure in third place. In addition to their associated morbidity, such disorders are important due to the number of affected individuals and the many patients subjected to treatment because of them. Patients with cardiovascular disease constitute risk cases in dental practice, particularly in the absence of adequate medical control.

Treatment of Angina:

1) Terminate procedure - The stress of the procedure itself might be what increases the heart rate, and precipitates angina. Sometimes, just halting the procedure, or removing the stressful stimulus (syringe, hand piece, etc) will allow the angina to resolve itself.
2) Position the patient - The patient in angina is usual **conscious** and **apprehensive**.



cardial infarction

Position the patient in the most comfortable position, which is **usually sitting up**. Lying down for this patient, usually makes **the pain feel more intense.**
3) **Administer Oxygen** - Since the cause of the problem is a **relative lack of oxygen** to the heart muscle, any thing that increases the amount of oxygen in the blood stream will increase the amount of oxygen the heart muscle receives. Administering oxygen via a nasal hood or canula is an easy way to do this.
4) **Administer Nitroglycerin(NTG) -** Nitroglycerin is a potent vasodilator, expecially for the cardiac vessels. This opening of the cardiac blood vessels allows more oxygen rich blood to reach the heart. **The dose is 0.4mg either by pill or metered**
spray sublingual.

There are two precautions.

First is a real contraindication. **Patients that have taken a phosphodiester inhibitor medication such as Viagra or Cialis, in the last 48 hours should not be given nitroglycerin.** There are synergistic effects from these drugs that can **result in a severe**, life **threatening hypotension**.

The second is a precaution with **hypotensive patients**. NTG has effects on the whole body, and a more **generalized vasodilation** can **lead to hypotension**. If the patient is **already severely hypotensive**, administering NTG may exacerbate the hypotension.

Patients with systolic blood pressures **below 110 or more than 30% below the patients baseline may need to be placed flat or given a IV fluid bolus prior to administering NTG.** This may be **impractical in your office and best to wait for EMS to arrive**.
4) Repeat NTG **q3-5** minutes **after three doses**, you have to **suspect that this is not angina**, and is a **true heart attack/myocardial infarction(MI) occurring**. **Procede to treatment of an MI you should follow.**
5) Modify dental therapy to **prevent recurrence**. Administer **oxygen** while treating the patient, and **using stress reduction protocols.**

Stress Reduction Protocol

Seventy Five percent of emergencies in the office are stress related. Most patents have the capacity to deal with this increase in stress. However, our medically compromised patients have less capacity to deal with it.

The body's response to dental stress increases the cardiovascular workload and metabolism, which puts stress on the **respiratory** and **endocrine systems**. Together, these can overwhelm the compromised patient's ability to deal with this stress. **To minimize the stress to the patient, the stress reduction protocol was established**.

The Stress Reduction Protocol

Short Appointments - Some people only have so much tolerance for dentistry. Don't go into "overtime" with these patients. Short appointments, may be best for them.

Profound Local Anesthesia - It's obvious how this reduces stress. Work on having multiple techniques for local, different anesthetics and differnent technique. Not all patients respond to one type of local or one technique of administration.

Supplemental Oxygen - Increasing the blood level of oxygen makes more available to the heart, lungs and brain. Even if cardiac, respiratory or metabolic workload goes up. There is more oxygen available for these organ systmes to work.

Pre-op Sedatives - Sedated patients are calm patients. Used appropriately, sedated patients have a fewer spikes in pulse and blood pressure, which decreases cardiac, respiratory or metabolic workload.

Sedation during appointment - See above.

Prophylactic Meds - These optimize the patient's cardiac, respiratory or metabolic system before treating them.

•Inhalers , Glucose ,Nitroglycerin

Pre Op Vitals - Comparison of these values to base line vitals taken at an earlier visit serve as a physical status on any givent day. In addition to pre-op pulse and blood pressure, I like to get a blood sugar in my diabetic patients.

Syncope:

Vasovagal syncope (VVS), known as common **faint**, is a neurally mediated syndrome associated with hypotension and relative bradycardia due to cerebral hypoperfusion.

Syncope is the most **common emergency in dental practices**. Nonetheless, the vast majority of dentists do not seem **competent nor prepared to manage this emergency**.

Psychogenic factors seem to play an important role in **provoking syncope**. Placing the patient **in a supine reclined position with raised legs in combination with the administration of oxygen seems effective for regaining consciousn**ess.

Although valuable in many aspects, risk assessment by medical history taking is not proven to result in fewer episodes. The strength of these conclusions is low based on GRADE guidelines

Causes:

- External fluid loss;
- Internal sequestration of fluid;
- Decrease in cardiac output;
- Arrhythmias;
- Hypocapnia (hyperventilation);
- Hypoglycemia.

Signs/Symptoms:

1. Blood pressure low normal or elevated; Tachycardia.
2. Orthostatic hypotension and increased pulse rate.
3. Altered mental states; anorexia; apathy, weakness.
4. Cold clammy skin.

Treatment:

1-proposes early intervention by placing the patient in a supine position with **feet elevated 10°** whilst maintaining an open airway in order to reinstate cerebral perfusion

2-Maintain patient airway – turn head to one side to prevent aspiration.

3-Administer 100% oxygen.

1. DO NOT use aromatic spirits of ammonia because it stimulates the sympathetic system and augments arrythmogenicity.

Prevention:

Dentists and other oral health care workers have an important role to play since they are responsible for: (1) preventing episodes from (re)occurring, (2) diagnosing and differentiating between banal or severe incidents and (3) acting adequately to regain one's consciousness. In most clinical situations however, dental practitioners don't feel confident handling such medical emergencies.

Low confidence in managing emergencies is associated with insufficient training or education.The common absence of a blood pressure monitor, electrocardiogram (ECG) monitor or an on-site specialist in dental practices poses an additional challenge to the practitioner.

Dr. Hamid Hammad Enezei

PhD

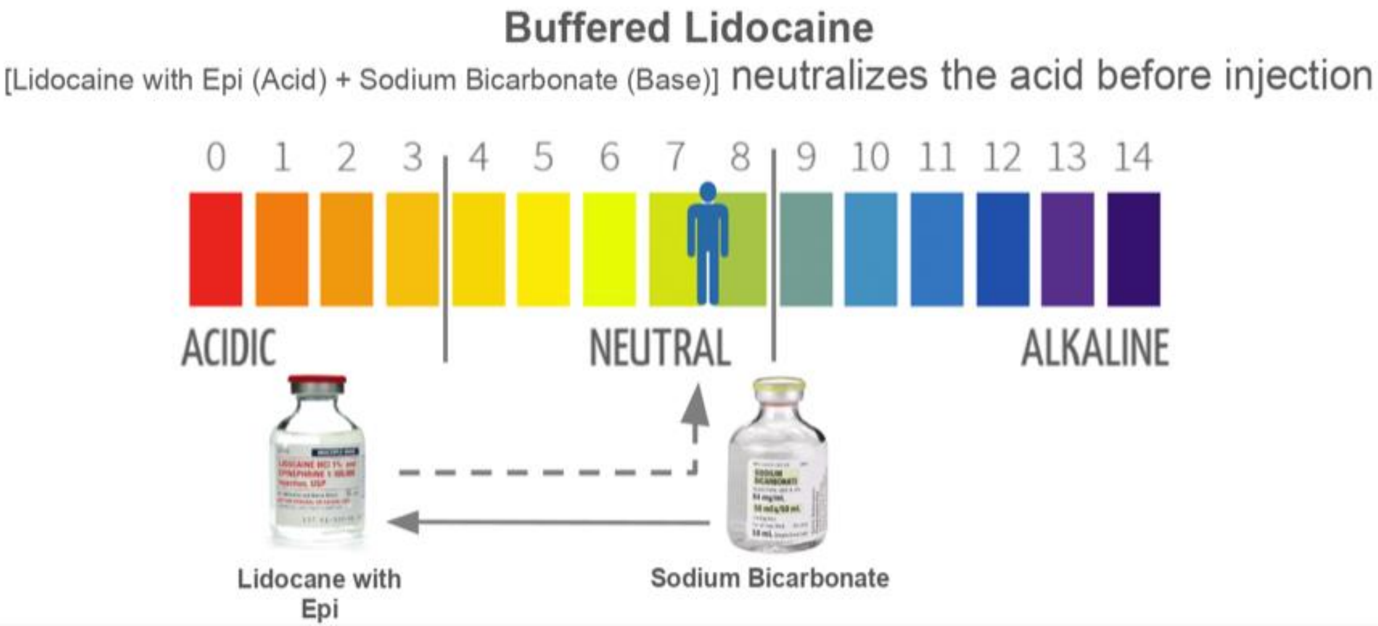
Oral & Maxillofacial Surgery

Academic Year 3

Advances in local anesthesia

Assistant Prof Dr. Hamid Hammad Enezei

Ph.D in Oral & Maxillofacial Surgery



Advances in local anesthesia

- Computer controlled local anesthetic delivery
- Articaine hydrochloride
- Local anesthesia reversal
- Buffering of local anesthetic solution
- Nasal local anesthetic mist for maxillary nonmolar teeth

Although **local anesthesia remains the backbone of pain control** in dentistry, researches are going to seek new and better means of managing the pain.

Most of the researches are focused on improvement in the area of anesthetic agents, delivery devices and technique involved.

Newer technologies have been developed that can assist the dentist in providing enhanced pain relief with reduced injection pain and fewer adverse effects.

Computer-controlled local anesthetic delivery (CCLAD):

Computer-controlled local anesthetic delivery (**CCLAD**) is one the method to **reduce patient pain during local anesthesia**; it is a device that slowly administers anesthetics by using a computerized device to control the injection speed

Dental fear is the most common reason for patients to avoid visiting the dentist.

Dental fear can occur for a variety of reasons, including **noise** and **vibration from tooth-cutting devices such as dental handpieces**, **smell of drugs or materials used in dentistry**, **pain during dental treatment**, and **irrational fear of local anesthesia**. Because dental treatments may be **painful, appropriate local anesthesia is necessary to reduce pain during such treatments**.

However, paradoxically, patients often **fear pain caused by anesthetic injections more than pain from dental treatment itself** .

Despite careful anesthetic procedures, **dental local anesthesia can cause pain for various reasons,** including **soft tissue damage during penetration of the oral mucosa**, pressure from the spread of the anesthetic solution, **temperature of anesthetic solution**, **low pH of anesthetic solution**, and **pain from the characteristics of the drug**.

In order to reduce pain during local anesthesia, **swabbing anesthesia** is often performed on the injection point; **similarly**, local anesthetic techniques that can **anatomically reduce pain**, such as **infiltration anesthesia**, should be used rather than **subperiosteal or intraosseous injections** that **can cause pain**. **In addition**, the anesthetic ampoule must be used administered at a temperature similar to body temperature; sterile local anesthesia should be used; and effort should be made to slow the injection speed .

Although reducing the **injection speed is the most effective method of reducing pain**, **controlling and maintaining the amount or speed of injection in actual clinical settings is difficult**.

Many devices have been introduced that can inject local anesthetic into the tissues at **a set speed**. Collectively, these **"painless anesthetic devices"**, are termed **"computer-controlled local anesthetic delivery" (CCLAD) devices**.

CCLAD also collectively refers to devices that not only **slow** and **maintain the injection speed**, but also **maintain a constant speed while taking into account the anatomical characteristics of the tissues being injected** .

The most widely known devices of this type include the **Wand® (Milestone Scientific, Livingstone, NJ)**, The Wand eliminates the "bee sting" effect - the painful consequence from a surge of fluid into a confined tissue area. Even more, certain injections made possible with the Wand minimize the unnecessary numbness of the tongue, cheek, and face. Imagine leaving the dentist's office without a "fat lip!"

The Wand handpiece provides painless injections for all routine dental treatment including root canals, crowns, fillings, and cleanings.

The Wand's Key Benefits:

Higher Patient Comfort

Reduced Patient Anxiety

Faster numbing onset and sensation recovery

Especially useful for pediatric patients

The Wand's computer-controlled local anesthetic delivery system doesn't look like a syringe. It doesn't feel like a syringe. And, what's more, it works better than a syringe resulting in a more pleasant experience. Ask us about Wand at your next visit. It's the comfortable alternative to the syringe

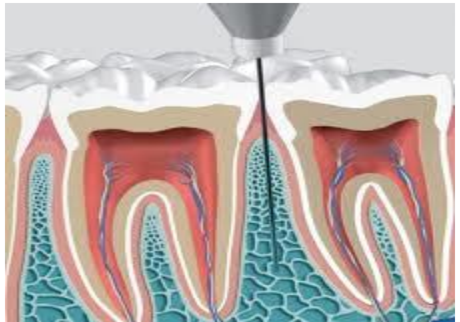
Comfort Control Syringe (CCS; Dentsply, USA),

The Comfort Control™ Syringe (CCS™) is an electronic, pre-programmed delivery system for local anesthesia that dispenses the anesthetic in a slower, more controlled and more consistent manner than a traditional manual syringe.

The Comfort Control™ Syringe has a special delivery system. The injection begins at a slow rate to minimize the discomfort associated with rapid injection.

In short, the Comfort Control™ Syringe can make injections easier for the dental professional and more comfortable for the patient.

QuickSleeper (Dental HiTec, France),



New techniques

Electronic dental anesthesia (EDA):

This technique is based **on the TENS** (transcutaneous electronic nerve stimulation) and the electronic waves are used to disrupt neural pain transmission to the brain. Research on this technique continues for use in the dental field.

Needle-free injection:

The injection system is based on a piston-pressure system and several systems are introduced such as PED-O-JET, SYRIJET and MED-E-JET. These techniques are widely used for **daily injections of insulin in diabetics**. In studies, these systems showed less pain compared to conventional injections with a needle gauge of 25

Computer controlled injection:

In this technique, computer **controls the speed** and **injection pressure**. C-CLADS (computer controlled local anesthetic delivery system) has less pain and discomfort for patients than conventional syringe injections, **but requires** greater facilities, more space and higher costs

Jet–injection:

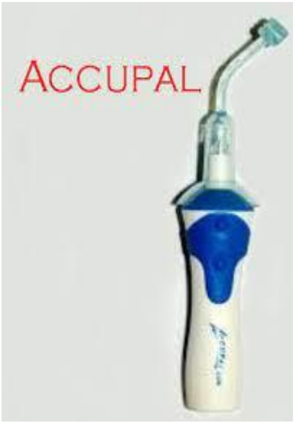
In this technique, a small amount of anesthetic drug driven into the submucosa without a needle. The air pressure is used for the infiltration of the drug into the mucosa through tiny pores. This method is particularly useful for topical anesthesia for palatal injection

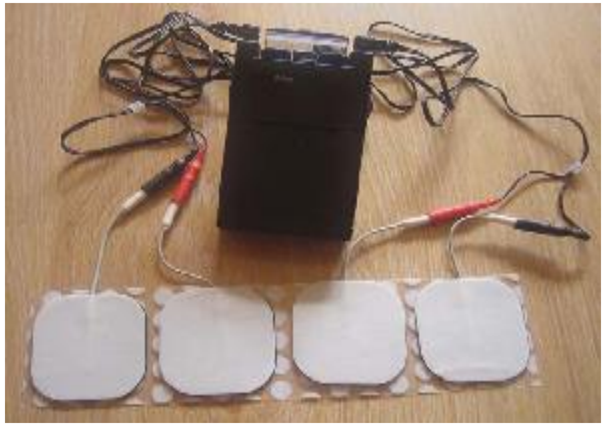
Vibrajet:

It is a device that provides high frequency vibrations in the dental injection syringe which causes a relative decrease in pain during the injection.

Accupal:

This is a tool to create pressure and vibration at the injection site. These mentioned irritate the larger nerve fibers and cause the lack of sensitivity during the penetration of the needle





TENS (transcutaneous electronic nerve stimulation):

The result of this method in patient comfort and it provides less pain during the injection. This has been demonstrated especially for IAN nerve block techniques, while topical anesthesia does not cause significant changes to reduce pain during the injection. This technique stimulates the nervous system and it starts before injecting and the pulse rate increases to make a good shake to the patient. The needle is inserted at an area between the electrodes of TENS while generated impulses are continuing at the same level. After withdrawing the injection and removing the needle, pulses are **slowly reduced** and stopped .

STA system device:

This is an **auxiliary system** for injection especially made **for PDL injections** where the dynamic pressure sensory system **improves the quality** and **reduces the side effects of injections**.

Low-pressure dynamic injection in this technique prevents tissue damage and pain during the injection. In addition, the injected anesthetic drug leakage is detected and prevents creation of an unpleasant taste in the patient’s mouth.

However, this technique requires the computer system tools .

Figure 2—The STA-Intraligamentary Injection uses the STA-System real-time dynamic pressure-sensing technology to guide the needle to the PDL. Accurate and objective feedback is provided to the operator

Intranasal local anesthesia:

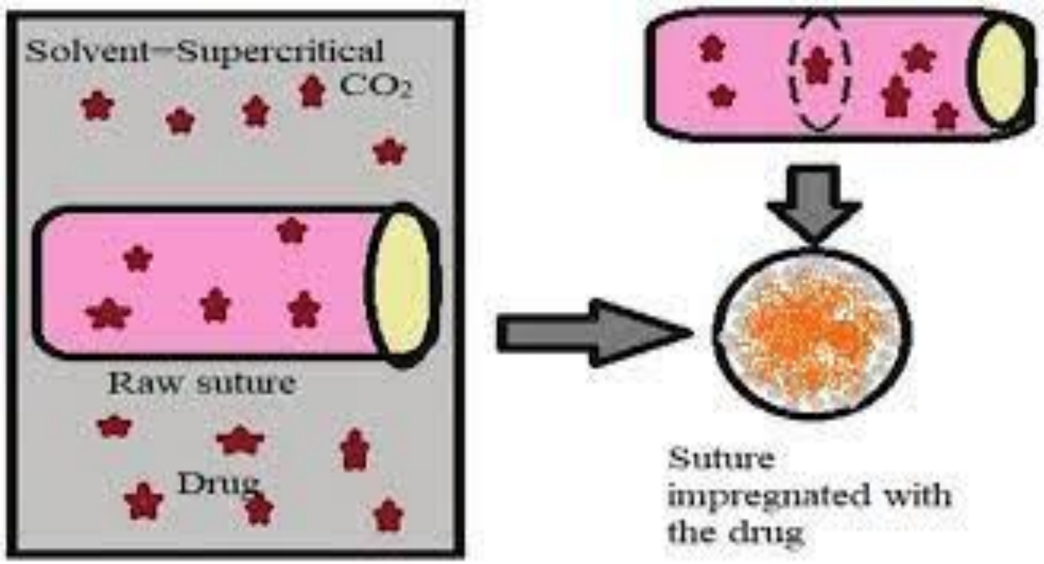
In the past, the use of nasal mucosa was conventional due to the high blood supply and ability to achieve the systemic effects of drugs.

Nowadays for the nasal mucosa and even upper teeth numbness, anesthetic drugs (especially tetracaine) are used on the **nasal mucosa**.

Studies have shown that the use of **intranasal tetracaine with a vasoconstrictor such as oxymetazoline** can provide tooth anesthesia for **the first molar on one side to the first molar on the other side** and dental procedures can be performed for the teeth, **without need to inject anesthetic drugs**

Electrospun drug-eluting suture:

Contains absorbable sutures with PLGA chemical structures that are **combined with bupivacaine**. The sutures can slowly release the drug to the surgical site within 12 days and provide appropriate analgesia. Higher concentrations of the anesthetic drug cause a decrease in the suture tensile strength. The suture tissue reaction is comparable to regular PLGA sutures without the combination of anesthetic drugs



Intraoral lidocaine patch (dentipatch):

This patch contains 10-20% lidocaine which is placed on dry mucosa for **15** minutes and provides suitable anesthesia for the mandible and maxilla

Local anesthesia reversal

Phentolamine mesylate (Oraverse):

Patients can have their dental work performed in a pain-free fashion. Of course, the problem with dental anesthetics is that **for some people**, it takes hours for their mouths to **recover from the numbing effect**. This issue often translates into a **problem for busy working people** who can't afford to **spend half a day feeling numb** in the face. Instead of a quick morning filling appointment being a simple affair, it can **negatively impact a person's work** performance by **impairing speech and impeding a person's ability to present them publicly without humiliation**.

Fortunately, mouth-related numbness doesn't have to be an all-day affair thanks to a new anesthetic reversal agent called *OraVerse* .**OraVerse is the first and only local dental anesthesia reversal agent in the market proven to accelerate the reversal of anaesthetic effect (numbness) after dental procedures**.

To administer OraVerse, dentist will inject it into the area of the patient's mouth that has been worked on and is therefore already numb

The injectable form of **phentolamine (alpha adrenergic receptor antagonist)** can be used to **terminate drug-induced** **local anesthesia** when it is **not required.**

Especially in high risk populations, where children and the elderly can inadvertently damage the tissues inside the mouth.

OraVerse®
Phentolamine Mesylate

Soft tissue numbness causes problems with normal functions such as talking, laughing, eating and drinking and can sometimes cause tissue damage.

To prevent this situation, a 1.7 mL dental cartridge containing 0.4 mg phentolamine mesylate is used. In this way, the approximate time for the return of normal sensation will be about half. For example, the normal sensation of the tongue will return within **60 minutes with phentolamine mesylate and 125 minutes without it**

Children and **adults** **have a tendency to bite their lips and even the inside of their cheeks** when they are numb from anesthesia. They can't feel themselves biting their lip and so they continue (unfortunately) until they have chewed so hard they have damaged their lips and they have **become swollen** and **sore,** sometimes to the point of **being cut and bleeding!**

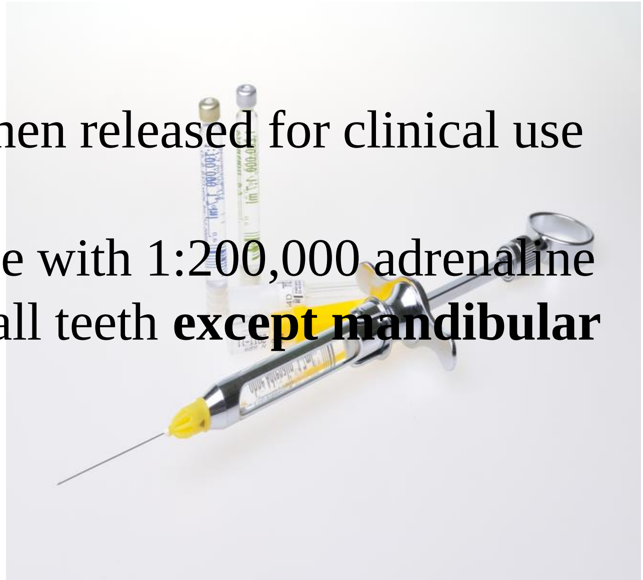
Adults and **young people** become embarrassed being numb because they can't speak clearly, smile or even drink properly they feel uncomfortable and self-conscious. Drooling becomes an issue.
Dental patients were administered a dose of 0.2, 0.4 or 0.8 mg of *OraVerse*. The majority of adverse reactions were mild and resolved within 48 h. There were no serious adverse reactions and no discontinuations due to adverse reactions

Articaine :

mandibular buccal infiltrationFor a mandibular buccal infiltration of the first molar, **4 percent articaine with 1:100,000 epinephrine will result in a higher success rate** than will **2 percent lidocaine with 1:100,000 epinephrine**, but the duration of pulpal anesthesia will **decline over 60 minutes with either formulation.**

Articaine pharmacology

Articaine, 4-methyl-3[2-(propylamino)-propionamido]-2-thiophene-carboxylic acid, methyl ester hydrochloride, belongs to the amide family of LAs, which also includes **lidocaine, mepivacaine, bupivicaine** and **prilocaine**. **Articaine** is unique amongst the **amide family**, containing an **ester group and having a thiophene instead of a benzene ring.4–6 The thiophene ring**, an integral feature of articaine's LA potency7 increases **articaine's lipid solubility** facilitating more efficient diffusion of the anaesthetic through the **nerve cell lipid membrane and into surrounding**



Articaine was first synthesized in Germany in **1969** under the label, HOE 40-045, and then released for clinical use in **1976** under the name(Carticaine hydrochloride)

In 1971 finding that 2% articaine with 1:200,000 adrenaline was superior to 2% lidocaine with 1:200,000 adrenaline in anaesthetic duration and extent, and that articaine produced profound anaesthesia for all teeth **except mandibular molars.**

In 2006, The FDA **approved 4% articaine with 1:200,00 adrenaline.**

Articaine efficacy :

Articaine LA onset takes between **1.5 and 1.8 min for a maxillary infiltration** and **1.5–3.6 min for mandibular block anaesthesia**.

Articaine pulpal anaesthesia lasts between 30 and 120 min, **a duration longer than lidocaine, mepivacaine and prilocaine**.

Articaine soft tissue anaesthesia lasts ~2.25 h for maxillary infiltrations and 4 h for mandibular blocks.

Articaine safety ,is well-tolerated and safe for use in routine clinical dentistry.

Both anaesthetics are appropriate and effective for clinical use.

Articaine's toxicity is comparable to that of **lidocaine**, ***cautioned*** **use of both lidocaine and articaine in patients with liver or cardiovascular impairment as amide biotransformation occurs in the liver and the anaesthetics can decrease myocardial function for patients with advanced cardiovascular disease**.

Lidocaine and **articaine** use in dentistry Lidocaine has proven safe and efficacious for routine clinical treatment.

Articaine seems to be the local anesthetic of **first choice in tissues with suppurative inflammation, for adults, children (over 4)**, **elderly, pregnant women, breastfeeding women, patients suffering from hepatic disorders and renal function impairment**.

In Articaine solutions (1: 200,000) epinephrine is in low concentration, thus in patients at high risk adverse responses are **maximally decreased**. In these patients articaine should be used with careful consideration of **risk/benefit ratio**.

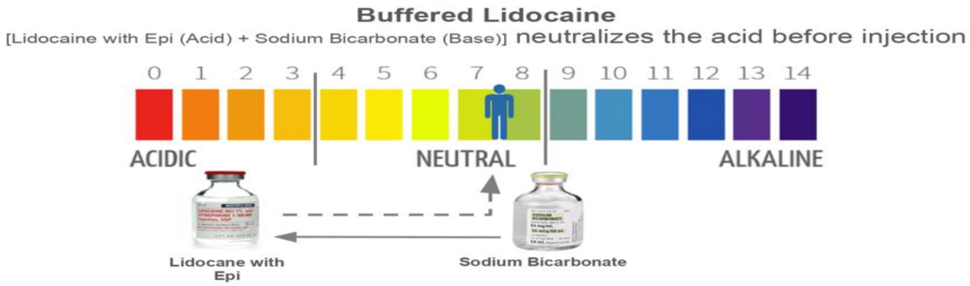
Articaine solutions must not be **used in persons who are allergic or hypersensitive to sulphite, due to content of**

Sodium metabisulfite as vasoconstrictor's antioxidant in it.

Incidence of serious adverse effects related to dental anesthesia with articaine is very low. Toxic reactions are usually due to an **inadvertent intravascular injection** or **use of excessive dose**.

To avoid overdoses maximum recommendation dose (MRD) **must not be exceeded** and **aspiration test always performed prior all LA injections**.

Buffered Local Anesthetic



What is Buffered Local Anesthetic?

One downside to a local anesthetic that contains the epinephrine vasoconstrictor is that the ph or "acid-base balance" tends to be more on the acid side of the equation as delivered by the manufacturer. This results in two minor, but manageable problems:

First, as the local anesthetic is initially injected into the patient's soft tissue, the lower "acid" **ph** results in a **slight stinging effect felt by the patient**. Due to the fast action of most local anesthetics, the surrounding tissue becomes numb quickly and subsequent injections, even at the lower ph are not felt by the patient.

Secondly, also due to the lower **ph,** less number of anesthetic chemical molecules are initially released into the area that is being numbed. **Because of this effect, multiple injections may be required and a time delay of the local anesthetic to profoundly numb an inflammed or infected tooth**.

The human body's soft tissues eventually **neutralizes or "buffers"** the lower **ph** of the local anesthetic back to a **neutral balance** that gets and keeps a **patient profoundly numb**. The potential benefits of buffering local anesthetic solutions prior to injection, such as decreased injection pain, faster onset, and greater depth of anesthesia, may be particularly advantageous in patients who have difficulty achieving profound anesthesia for clinical dentistry, and for anesthetizing infected areas

Dentists can effectively buffer local anesthetic preparations using commercially available mixing systems or by utilizing a hand-mixing technique. Rather than using a remove and replace technique, practitioners may consider a direct injection technique, adding 0.1 mL of 8.4% sodium bicarbonate directly into any local anesthetic cartridge regardless of local anesthetic concentration.

The Benefits of Buffering:

Since buffered anesthetic can cross the nerve membrane more readily than unbuffered ones, patients become numb **very quickly** so that the clinician can stay in the operatory and begin the procedure **almost immediately**. **Using buffered anesthetic can save as much as 17-20 minutes per hour**. Not only do the **dentists and patient** save significant time, but discomfort is minimized. Both impact and improve the patient experience. This combination is priceless

Predictability: Buffering can **elevate the depth of anesthesia**, increasing the likelihood of patients **getting numb the first time**. It allows dental professionals to give multiple injections without having to **reload the cartridge or risk wasting premixed anesthetic**. ***When using this buffering system, 64% of appointments were at least 15 minutes shorter than scheduled.***

Efficiency: Buffered anesthetics can cross the **nerve membrane more readily**, allowing a patient to become numb quickly. ***When using this buffering system, 16.7 minutes is the average time saved per appointment (saves 2 hours per day.)***

Reduced Pain: Mixing anesthetic with sodium bicarbonate creates a bi-product of CO2, which by itself contains the ability to create a numbing effect. A single injection is often sufficient, but when subsequent injections are required, the fast-acting buffered anesthetic is already working and patients don’t feel multiple injections. *When using this buffering system, on a scale of 1-10, 1 being no pain and 10 being extreme pain, patients rate their pain experience 1.81 out of 10.*

Reduced Pain: **Mixing anesthetic with sodium bicarbonate creates a bi-product of CO_2, which by itself contains the ability to create a numbing effect.** A single injection is often sufficient, but when subsequent injections are required, **the fast-acting buffered anesthetic is already working and patients don't feel multiple injections**. ***When using this buffering system, on a scale of 1-10, 1 being no pain and 10 being extreme pain, patients rate their pain experience 1.81 out of 10.***

How is the buffering procedure performed?

•1. A sterile dental anesthetic carpule (1.8cc) is loaded into a standard dental syringe.

•2. The dentist then extrudes and discards approximately **10% of the solution (0.18cc) out of the anesthetic cartridge**.

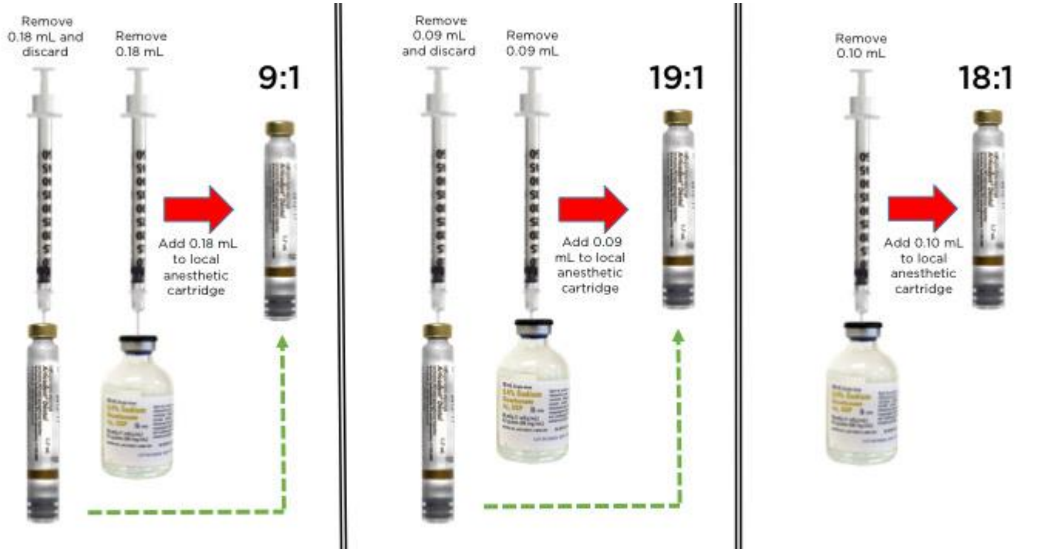
•3. Using a separate sterile plastic syringe and needle, approximately **1cc of 8.4% sterile sodium bicarbonate (an alkaline base solution) is drawn into the 3cc syringe from the bottle of sodium bicarbonate**.

•4. Remove the needle from the 3cc syringe containing the sterile 8.4% sodium bicarbonate and insert the dental cartridge's anesthetic needle into the open ended syringe of sodium bicarbonate and proceed to aspirate **0.18cc** of sodium bicarbonate back into the dental syringe anesthetic carpule.

5. This process essentially replaces **approximately 10% of the local anesthetic** solution of the anesthetic carpule with the 8.4% sodium bicarbonate. When the **two solutions are mixed together**, the local anesthetic solution (low ph- "acid") + sodium bicarbonate (high ph- "base") **they immediately balance each other out and the final solution becomes a neutral ph=7.0, which is equal to the body's ph of 7.0.**

This final, neutralized solution is used to numb the patient's tooth. **Ph paper is used** to verify the correct ph of the final solution: the color of the ph paper will change from yellow (new carpule: ph=3.8) to green (buffered carpule: ph=7.0).

BUFFERING SYSTEM EQUIPMENT



Anutra Medical Cassette set up for buffering

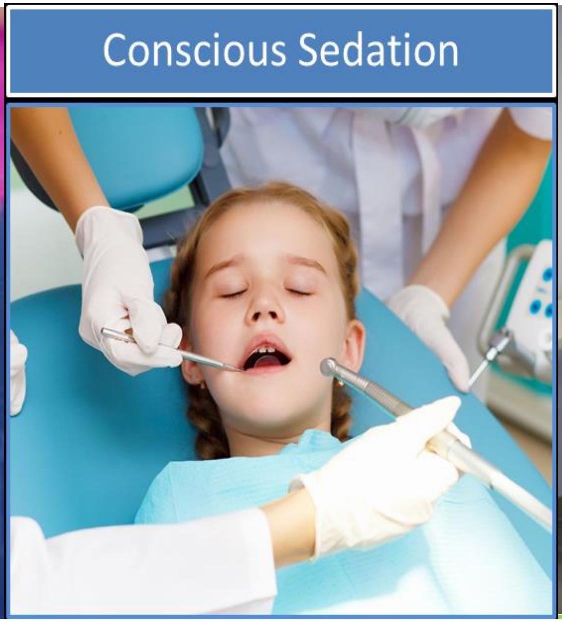
Dr. Hamid Hammad Enezei
PhD
Oral & Maxillofacial Surgery

Academic Year 3

Conscious Sedation and General Anesthesia

Assistant Prof Dr. Hamid Hammad Enezei

Ph.D in Oral & Maxillofacial Surgery



Conscious sedation

- Sedation techniques: Oral, sublingual, transdermal, intranasal, intramuscular, intravenous and inhalational
- Nitrous oxide
- Complications and medicolegal considerations

Sedation in dentistry :

Sedation in dentistry has been a **controversial topic** due to questions being raised over its safety, especially in dental chair. **Dental fear and anxiety are not only common in children** but **also significantly prevalent among adults due to high intensity of pain**.
Sharing of airway between the **anesthesiologist and the dentist remains the greatest challenge**.

Conscious sedation is a technique in which the **use of a drug** or **drugs produces a state of depression** of the central nervous system (CNS) **enabling treatment to be carried out**, but during which verbal contact with the patient is maintained **throughout the period of sedation**. The drugs and techniques used to provide conscious sedation for dental treatment **should carry a margin of safety wide enough to render loss of consciousness unlikely**.
Conscious sedation retains the **patient's ability to maintain a patent** airway **independently** and **continuously**

Conscious sedation is a **drug-induced depression of consciousness** during which the **patient responds purposefully** to **verbal commands**, **either alone** or **accompanied by light tactile stimulation**. No interventions are required to maintain a **patent airway**, and **spontaneous ventilation** is adequate. **Cardiovascular function is usually maintained**.

Careful presedation evaluation with **respect to** airway, **fasting**, **and understanding about the pharmacodynamics** and **pharmacokinetics of the drugs** must be **established**.

Availability of airway management equipment, venous access, and appropriate intraoperative monitoring and well-trained staff in the recovery area must be ensured.

Conscious sedation can be administered through various routes such as **oral**, **intramuscular**, **intravenous**, and **inhalational**

Challenges in dental conscious sedation:

The challenges in dental conscious sedation are as under:

1.Shared airway between the **dentist** and the **anesthesiologist**

2.Phobia and anxiety

3.Coexisting medical conditions such **as cardiac anomalies**, **mental instability**, and **epilepsy**

4.Chances of **arrhythmias during surgery due to trigeminal nerve stimulation**

5.Enlarged tonsils and **adenoids in children** likely to **precipitate respiratory obstruction**

6.Risk of patient **losing consciousness**, **respiratory,** and **cardiovascular depression**

7.**Vasovagal syncope** **due to the dependent position of legs in dental chair**.

The anesthesiologist should be well prepared to **face** and **tackle all the anticipated challenges as enumerated above**. A detailed and thorough **presedation checkup comprising assessment of airway**, **cardiorespiratory system**, any **congenital abnormality**, **medication history**, and **allergy must be done.**

The operating area should be well equipped with all the resuscitation drugs/equipment required to resuscitate the patient in case of emergency

INDICATIONS FOR CONSCIOUS SEDATION

1-Dental phobia and anxiety

2-Traumatic and long dental procedures

4-Medical conditions aggravated by stress such as angina, asthma and epilepsy

5.Children more than 1 year of age

6.Mentally challenged individuals

7.Ineffective local anesthesia due to any reason. PREPARATION FOR CONSCIOUS SEDATION

Preparation for conscious sedation involves the preparation of patients as well as preparation in an operating area to meet any unforeseen challenges.

Patient preparation

1.Consent for treatment: Valid informed consent is necessary for all patients receiving dental care under conscious sedation, and this should be confirmed in writing. In case of children, valid consent should be signed by the legal guardian

2. Presedation checkup: Patient's detailed history and examination are performed so as to classify according to the American Society of Anesthesiologists (ASA) classification. Only patients who satisfy the criteria of ASA Grade I and II should be considered for sedation in dental surgery outside hospital. For pediatric patients, it is recommended that only the ASA Grade I patients are sedated outside a hospital environment

ASA class	Description
I	A normal healthy patient
II	A patient with mild systemic disease
III	A patient with severe systemic disease
IV	A patient with severe systemic disease that is a constant threat to life
V	A moribund patient who is not expected to survive without the operation
VI	A declared brain-dead patient whose organs are being removed for donor purposes
E	Suffix for patients undergoing emergency procedures

C-detailed airway examination is done for pediatric patients to look for adenotonsillar hypertrophy or any other anatomical airway abnormalities. In case of any underlying medical or surgical condition, the concerned specialist should be consulted for optimization before taking up the patient for dental procedure

D-Fasting instructions: Preoperative fasting for sedation is controversial and considered unnecessary by some authorities within dentistry for conscious sedation. Airway reflexes are assumed to be maintained during moderate and minimal sedation. It is not clear where the point of loss of reflexes lies. The chances of inadvertent oversedation and loss of protective airway reflexes at some point cannot be ruled out

E-For elective procedures using conscious sedation, the 2-4-6 fasting rule applies (that is 2 h for clear fluids, 4 h for breast milk, and 6 h for solids.). For emergency procedures where the fasting cannot be assured, the benefits of treatment and risk of the lightest effective sedation can be analyzed. Delaying the procedure may benefit the patient.

Operating/procedure setup

The institution/clinic should have **monitoring** and **resuscitation equipment** and trained manpower available to handle any emergency situation.

Monitoring: Monitoring equipment such as ECG, pulse oximeter, $ETCO_2$, NIBP, and defibrillator should be handy in working condition

Crash cart should be available with all the resuscitation equipment and drugs required to resuscitate a patient

Every procedure should be carried out after ensuring the availability of appropriate size suction catheter, adequate oxygen supply, functioning flowmeters and tubings for **oxygen delivery to patient**, and appropriate-sized airway equipment.

PHARMACOLOGY OF DRUGS USED FOR CONSCIOUS SEDATION

It is mandatory to secure an **intravenous (IV) li**ne with the help of an appropriate-sized IV cannula before administering any drug or inhalational anesthesia. In many cases, mild anxiolytic along with local anesthesia is sufficient to reduce fear and anxiety in the patient.

Nitrous oxide

Mixture of nitrous oxide (N_2O) and oxygen is used as a sedative. N_2O is a colorless gas used as an inhalational anesthetic agent. It is an anxiolytic/analgesic agent that causes CNS depression and varying degree of muscle relaxation and euphoria with hardly any effect on the respiratory system.

Recent research shows analgesic effects of **N_2O is initiated by the neuronal release** of endogenous opioid peptides with **activation of opioid receptors** and **descending gamma-aminobutyric acid (GABA)** and **noradrenergic pathways that modulate nociceptive processing at the spinal level**.

Anxiolytic effect involves the activation of $GABA_A$ receptor through benzodiazepine-binding site. The anesthetic effect appears to be caused by **inhibition of N-methyl-D-aspartate (NMDA) glutamate receptors**, thus removing its excitatory influence in the nervous system.

The technique employs subanesthetic concentrations of N_2O delivered along with oxygen from dedicated machinery through a nasal mask. **The N_2O/oxygen delivery systems are manufactured with oxygen fail-safe devices that stop the flow of N_2O when the flow of oxygen is stopped**. The safety mechanism ensures delivery of **at least 30% oxygen** in all situations. N_2O has low tissue solubility and high minimum alveolar concentration which enables rapid onset of action coupled with a rapid recovery; thus ensuring a controlled sedation and quick return to normal activities. It is very safe as the patient remains awake and responsive and reflexes are retained. **The use of N_2O** is **contraindicated** in patients with **common cold**, **porphyria**, and **COPD.**

Sevoflurane

Sevoflurane is an ether **inhalational anesthetic agent** with **low pungency**, a **nonirritant odor**, and a **low blood–gas partition coefficient**. Its low solubility facilitates precise control over the depth of sedation and rapid and smooth induction and emergence from sedation.

Sevoflurane, therefore, remains an **ideal induction agent before starting infusion of a total IV anesthetic** such as **propofol** to maintain sedation.

Benzodiazepines

Benzodiazepines, including **diazepam** and **midazolam**, have proved to be **safe and effective for IV conscious sedation**. Their sedative and selective anxiolytic effects and wide margin of safety contribute to their popularity in dentistry. Apart from anxiolysis and amnesia, **benzodiazepines are known to possess skeletal muscle relaxation and anticonvulsant activity**; however, these drugs have no **analgesic properties**. Mechanism of action **is through GABA-mediated opening of chloride channels**. They have **high lipid solubility** giving **rise to rapid onset of action**.They are normally added to N_2O/oxygen for conscious sedation, as N_2O produces the **analgesic effects**. The most commonly used **benzodiazepine is midazolam**. Its high first-pass metabolism makes it a short-acting one. It is used for **conscious sedation** in the **pediatric dentistry**. It is mixed with a **sweet vehicle**, such as simple **syrup**, and used **orally either via drinking cu**p or through a **syringe without needle** and **deposited in the retromolar area**. Syrup can **be given 20 min before the procedure**.

Dose under 25 kg is **0.3–0.5 mg/kg** in adults but should be administered in a **hospital setup only**. It can also be given **intramuscularly**, **intravenously**, **rectall**y, and **nasally**. Its effects are **enhanced by various drugs** such as **opioids**, clonidine, antidepressants, antipsychotics, erythromycin, antihistaminics, alcohol, and antiepileptics and **should be avoided or used with caution.**

All practitioners using these drugs must have **flumazenil**, the specific **benzodiazepine receptor antagonist**, as one of the **emergency drugs** in the institution. **Flumazenil causes rapid reversal of all benzodiazepines**. However, it is contraindicated in patients taking **benzodiazepines for seizure disorder** or **high doses of tricyclic antidepressants**.

Ketamine

Ketamine, a phencyclidine derivative, is an **N-Methyl-D-aspartate receptor (NMDAR) antagonist**. It is a unique drug giving complete anesthesia and analgesia with preservation of vital brain stem functions. This "**dissociative**" state has been described as "a functional and neurophysiological dissociation between the neocortical and limbic systems." Dissociation results in a state of lack of response to pain with preservation of cardiovascular and respiratory functions despite profound amnesia and analgesia, described as "Catalepsy." This trance-like state of sensory isolation provides a unique combination of amnesia, sedation, and analgesia. The eyes often remain open, though nystagmus is commonly seen. Heart rate and blood pressure remain stable and are often stimulated possibly through **sympathomimetic actions**. **Functional residual capacity** and **tidal volume are preserved** with **bronchial smooth muscle relaxation** and **maintenance of airway patency and respiration**.

The most commonly seen **disadvantage of ketamine** is **emergence phenomenon** which occurs in **5%–50% of adults** in 0%–5% **in children. Ketamine increases salivary and tracheobronchial mucus gland secretions, so it is recommended to use an antisialagogue before administering ketamine**.

The emetic side effect of ketamine producing an **incidence of vomiting in 10% of children** can be lessened by administering atropine which reduces salivary flow.

Laryngospasm reported in 0.4% of cases can be managed with positive pressure ventilation with 100% oxygen.

Ketamine can be given intramuscularly at a dose of 3–4 mg/kg or intravenously at a dose of 1–2 mg/kg .However, administering **a lower dose of the drug may be safer to achieve adequate levels of sedation in children** due to the problem of potential severe respiratory depression.

Propofol

Propofol is chemically described as **2,6-diisopropylphenol**. Being **insoluble in water**, it is available in **whit**e, **oil-in-water emulsion which facilitates IV delivery of this fat-soluble agent**. Propofol is readily oxidized to quinine which turns the suspension yellow in color after approximately 6 h of exposure to air. Propofol exerts its hypnotic actions by activation of the central inhibitory neurotransmitter **Gamma-aminobutyric acid (GABA).**High lipophilicity ensures rapid onset of action at the brain, and rapid redistribution from central to the peripheral compartment **causes quick offset of anesthetic action**

Elimination half-life is 2–24 h. The most significant hemodynamic effect is **a decrease in arterial blood pressure** and **heart rate**. Sedative doses actually have little or no effect on the respiratory system. Apfel *et al.* studied six interventions for the prevention of postoperative nausea vomiting (PONV) and found that the use of propofol reduced risk for PONV by 19%.[17] Sedative doses are not analgesic, and a large proportion of patients experience pain on injection. To use an antecubital vein instead of a hand vein for the propofol, lidocaine admixture is a simple and effective way to avoid pain. Volatile anesthetic agents are used for the induction of anesthesia to avoid the struggle to get IV access before the child is asleep. With sevoflurane, propofol is given usually at a dose of 1 mg/kg body weight, followed by maintenance dose ranging from 0.3 to 4 mg/kg/h.[18]

Opioids

All of the **above-mentioned drugs** do not have **analgesic effects except ketamine**. **Opioid analgesic**, therefore, needs to be supplemented. **Fentanyl is a short-acting opioid 60–80 times** more **potent than morphine** and with a **rapid onset of analgesia** and **sedation**.

Duration of action is 30–60 min. Fentanyl can be administered by **parenteral**, **transdermal**, **nasal**, and **oral routes.** A “lollipop” delivery system is more acceptable to children than any other route. Fentanyl being a lipophilic drug is absorbed from the buccal mucosa, metabolized in the liver, and secreted in the urine. Recommended dose is 1 µg/kg/dose IV which can be repeated by **1 µg/kg increments if required**. **Constipation**, **nausea**, and **vomiting** are **common side effects**. Dose-dependent respiratory depression and occasionally bradycardia and chest wall rigidity are associated with rapid IV injection.

Sufentanil : **Sufentanil** is a **synthetic opioid analgesic drug**, which **is 5–10 times more potent than its parent drug fentanyl** and **500 times as potent as morphine**. It has **shorter distribution** and **elimination half-lives**. For outpatient surgery, **IV sufentanil produces equivalent anesthesia** to isoflurane or fentanyl. **Recovery is rapid,** and **postoperative analgesia requirement is less**.However, side effects such as **reduced chest wall compliance** and **high incidence of nausea** and **vomiting** and **prolonged discharge time** as compared to **midazolam** make it an unpopular **choice for premedication**

Complications and medicolegal considerations:

Clinicians who provide conscious sedation should be able to **recognize** and manage sedation-related complications. With regards to **inhalational sedation**, there are few potential complications which may occur. These include **equipment failure, inadequate sedation, poor patient experience and, rarely, nausea or vomiting.**
Conscious sedation is a common treatment modality for patients with dental anxiety.
In adults, **intravenous sedation using** Midazolam is the most **common method of conscious sedation**. For inhalational sedation, **Nitrous Oxide and Oxygen are used frequently**. Clinicians should be aware of the potential complications associated with **delivery of intravenous sedation** which can occur **pre-**, **peri-** and **post-sedation.**
An awareness of the **pharmacology of the drugs as well as careful patient assessment** and monitoring can help clinicians to **anticipate and reduce complications** such as
minor post treatment complications occurred in

agitation

sleepiness

drowsiness

pain

dental bleeding

Information and Consent Form

Consent

A dentist has a legal obligation to **obtain the valid** and **voluntary consent of the patient** to the treatment proposed. Consent is a **continuous communication process** and not just a single one-off event-it should be established and **reaffirmed verbally** with the **patient at all stages of treatment**.

Where sedation is provided then the patient should also provide written consent. It is important to remember that a signature on a **form can be misleading** and the mere presence of such a signature does not guarantee that the consent obtained is valid

Intravenous sedation makes lengthy or complex dental treatment more comfortable and easy for you. It can help remove anxiety, stress, discomfort, memory and awareness of the procedure. You enjoy a relaxing sleep whilst you maintain your ability to breathe

for yourself, maintain your reflexes and your ability to respond to us if required. You will still receive a local anesthetic after you are sedated. Following your procedure you will be given appropriate recovery time in which you will be closely monitored

These risks include but are not limited to:

1. You may be relatively aware of the procedure.
2. You may experience nausea and vomiting.
3. You may remain drowsy and lack time and space judgment after the procedure is completed. This could last up to 8 hours.
4. Discoloration of the skin or bruising in areas of access to veins or attempted access may persist for some time.
5. You may experience complications including but not limited to respiratory depression, persistent generalized pain, areas of numbness, swelling, bleeding, allergic reactions and pneumonia.
6. There is an **EXTREMELY** remote possibility that complications may require transportation to a hospital for treatment. Serious complications may result in brain damage, myocardial infarction, cardiac arrest, stroke, coma or death.

Things to know prior to your sedation appointment:

1. You will need an escort.. The onset of the medication is almost immediate and the peak effects last approximately an hour. After that, it starts wearing off and most peop le feel back to normal after 24 hours. For safety reasons do not drive or operate machinery for 24 hours following your sedation.
2. You should not eat or drink anything for at least 8 hours prior to your surgery to ensure a safe and effective sedation procedure.

Sedation
Consent

I ______________________ hereby consent to the

procedures described below which have described to me by

______________________ (Surgeon).

1. The administration of Conscious Sedation by intravenous injection and

2. The following dental procedure(s)

______________________ ______________________

______________________ ______________________

______________________ ______________________

I also consent to such further or alternative operative procedures as may be found necessary during the procedure and to the medications necessary for the purpose of this procedure.

Alternatives have been fully discussed.

Sign (patient/ representative): ______________________

Date: ______________________

I confirm that I have explained the nature and explained the nature and effect of the above procedure to the person who has signed above.

Sign: ______________________

Date:

3.Please let us know if:
a.you have a known allergy to benzodiazepines (Valium, Ativan, Versed, etc.) or Fentanyl
b.you are pregnant or breast feeding
c.you have liver or kidney disease
d.you have severe asthma , respiratory,or significant seizure disorder
e.you have narrow angle glaucoma f.
you have a diagnosed supraventricular tachycardia
g.you are taking nefazodone (Serzone) or levodopa (Dopar or Larodopa).
4.If you are taking an MAO inhibitor (Marplan, Nardil, Emsam, or Parnate) inform the Doctor as this is a contraindication to sedation.
5.If you are taking any illegal drugs, inform the doctor as this could adversely affect the sedation drugs.
6.Read the "post operative" instructions and follow them. Call our office .
If you have any questions or concerns. After hours, the answering service will be able to reach the doctor on call.
I understand these considerations and am willing to abide by the conditions stated above. I have had an opportunity to ask questions and have had them answered to my satisfaction.
Signed (patient)______________________
Date__

____
Signed (guardian if <18
_______________ Date_________________
Doctor ____ Date_________________

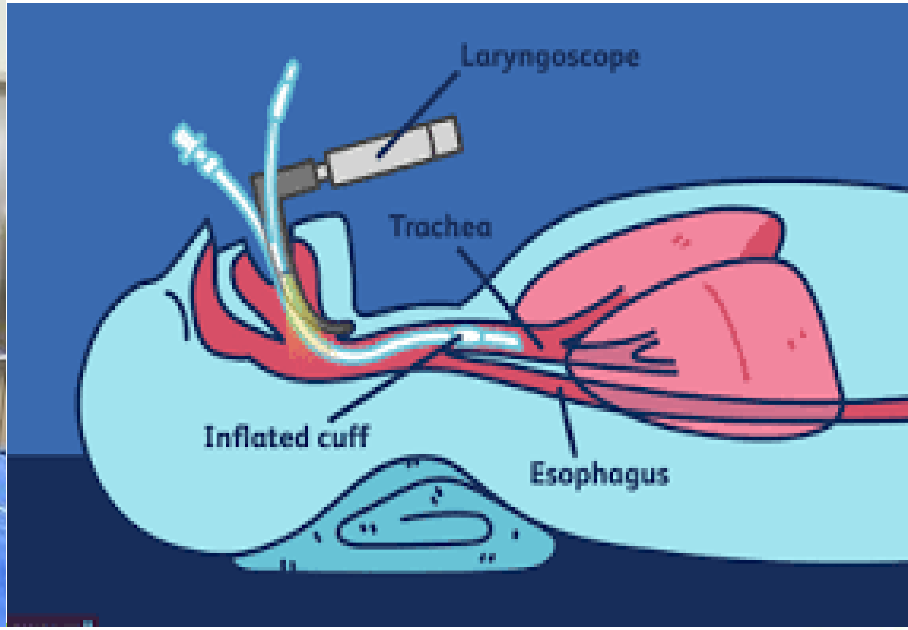
Dr. Hamid Hammad Enezei
PhD
Oral & Maxillofacial Surgery

Academic Year 3
Fundamentals of General Anesthesia

Assistant Prof Dr. Hamid Hammad Enezei

Ph.D in Oral & Maxillofacial Surgery



Fundamentals of general anesthesia

- Types of general anesthesia used in dentistry
- Advantages
- Disadvantages
- Indications
- Contraindication

❖ Fundamentals of general anesthesia

General anesthesia is commonly used to facilitate dental treatment in patients with anxiety or challenging behavior, many of whom are children or patients with special needs.
When performing procedures under general anesthesia, dental surgeons must perform a thorough pre-operative assessment, as well as ensure that the patients are aware of the **potential risks and that informed consent has been obtained**.
Such precautions ensure optimal patient management and reduce the frequency of morbidities associated with this form of sedation. Most guidelines address the management of pediatric patients under general anesthesia. However, little has been published regarding this **method in patients with special needs**.

The field encompasses a **wide variety of patients**, including those with **intellectual disabilities**, physical impairments, **psychiatric problems**, and **complex medical backgrounds** .

In many cases, these conditions can have significant effects on oral health. Additionally, they often influence the manner in which patients receive oral healthcare.
Part of the role of the special needs dentist is to adapt dental treatment to the individual needs of the patient. In many situations, this may necessitate **pharmacological sedation**, ranging from **minimal sedation**—achieved through oral, transmucosal, or **inhalation anxiolysis—**to **deeper forms of sedation and general anesthesia**

General anesthesia is **a state of unconsciousness produced by an anesthesiologist where the patient does not remember or feel anything during the procedure**. In addition, the patient remains still and does not move. The anesthesiologist must monitor the patient continually and ensure that the patient remains safe until the patient emerges from anesthesia.

Due to the complexity of this type of care, the office should have special monitoring and emergency equipment to ensure patient safety. In fact, there are national standards that require additional gas equipment and alarms when dental offices provide general anesthesia.

First, the patient and their family should meet with both the dentist and the anesthesiologist for a consultation prior to the day of surgery. During this consultation, **the dentist will decide** if the treatment requires general anesthesia. If it does, then the anesthesiologist will discuss the option of general anesthesia for dental care and ensure that you are healthy enough to have this performed at a Category 1 compliant office.

The dental assessment of any patient undergoing general anesthesia would ideally involve a thorough clinical examination and pre-operative radiographs to allow a treatment plan to be established. This would facilitate appropriate **informed consent** and **adequate planning for the procedure**.

As mentioned above, one of the main indications for treatment of patients under general anesthesia **is their lack of compliance during basic procedures**—often including examination. As a result, a large part of treatment planning comprises estimations of the patients' treatment needs, as well as ensuring that adequate time, facilities, and equipment are available to accommodate these needs.
There has been minimal discussion in the literature about whether other forms of sedation are sufficient to increase cooperation in patients with special needs and thus enable a more thorough pre-operative assessment

Not all patients are good candidates for general anesthesia in an office.

During the consultation, the anesthesiologist will also review the risks and benefits of anesthesia, answer all the patient’s questions , and review pre and post operative instructions.

One of these instructions is the last time atient can **eat** or drink before surgery appointment.

General anesthesia is a pharmacologically produced state of unconsciousness.

This is what patients routinely **receive during medical procedures at the hospital or outpatient surgical facilities**.
During general anesthesia, a patient **does not react or remember any of the surgical procedure**.
A trained anesthesia provider should administer **such a state of anesthesia**.

Ideally, that anesthesia provider **should not be the same person doing the surgical procedure**, especially in **children or patients with special healthcare needs**.

Patients should realize that **general anesthesia is quite different than "just going to sleep."** While under **general anesthesia**, the patient must be **monitored continuously** and **may need physiologic support from the anesthesiologist**, such as **assistance with breathing**. Although most medical procedures could be **accomplished with local and regional anesthesia**
it is generally **expected that general anesthesia will be provided**.
The opposite is true in dentistry, where most dental procedures are provided under local anesthesia (nerve blocks) without sedation or anesthesia. **This need not be the case though.**
The treatment area should look more like an operating room than your normal dental operatory. It should also have sufficient room to accommodate the team and emergency equipment such as a stretcher. In addition, we strongly recommend that you have met your dentist and anesthesiologist prior to the procedure and that they answer any and all of your questions fully.
Remember, general anesthesia for dentistry is aregular trip for patient to just any dental office.
Drugs used for anesthesia are CNS depressants with action that can be induced and terminated more rabidly than conventional sedative and hypnotics
Most sensitive site of action for general anesthetics is the reticular activating system of the brainstem (RAS).
Anesthetic dose: does not cause **depression of cardiac**, **vasomotor** or **respiratory centers**
Has a small margin of safety.

Stages of G. anesthesia

Stage 1:analgesia

•Decreased pain awareness, sometimes with amnesia, conscious may be impaired but not lost

Stage 2:disinhibition

Delirium, excitation, amnesia, enhanced reflexes, irregular respiration and incontinence

Stage 3: surgical anesthesia

•Unconsciousness, no pain reflex, regular respiration and maintained blood pressure

Stage 4:medullary depression

•Severe CVS and respiratory depression and the patient require pharmacological and ventilatory support

Preanaesthetic Medication

Anesthesia protocols

1. **Induction and maintenance for general anesthesia:**
   - For extensive surgical procedure
   - The protocol commonly includes IV drug for induction, inhaled agent (with or without IV) for maintenance and neuromuscular junction blockers to cause muscle relaxation
2. **Conscious sedation techniques:**
   - For minor procedure, that combine IV agent with local anesthetics
   - These can provide profound analgesia, with retention of the patient ability to maintain a patent airway and response to verbal commands
3. **Neuroleptanalgesia:**
   - combine a major neuroleptic (droperidol) and a potent opioid analgesic (fentanyl) to produce a detached, pain-free state. (state of analgesia and amnesia)
4. **Dissociative anesthetic**
   - Produced by ketamine
   - The patient is patient remains conscious but has marked catatonia, analgesia, and amnesia

- **Airway Assessment**
- This picture represents a Mallampati Class One airway. The entire uvula and tonsillar pillars are seen. This individual should be easy to mask ventilate or to intubate with a laryngoscope and endotracheal tube.
- This picture represents a Mallampati Class Three airway. None of the uvula or tonsillar pillars are seen. This individual may hard to mask ventilate, and quite difficult to intubate
- This image is representative of an extremely short thyromental distance, indicating tremendous difficulty in tracheal intubation, and possible difficulty establishing a satisfactory mask seal.

Provider Responsibilities

Intraoperative Responsibilities

Informed consent signed prior to sedation

Name, dose, route and time of all medications documented

Procedure begin and end times

Prior adverse reactions

Pre-medication time and effect

Vital Signs

BP

Heart Rate

Respiratory Rate

Oxygen Saturation

Level of ConsciousnessPost-operative Responsibilities

Vital Signs at least every 5 minutes

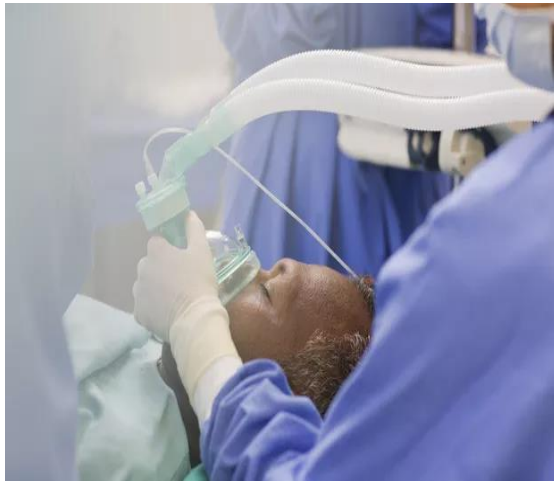
BP

Heart Rate

Respiratory Rate

Oxygen Saturation

Level of Consciousness



ASA Classification	Definition	Examples
ASA I	A normal healthy patient	Healthy, non-smoking, no or minimal alcohol use
ASA II	A patient with mild systemic disease	Mild diseases only without substantive functional limitations. Current smoker, social alcohol drinker, pregnancy, obesity (30<BMI<40), well-controlled DM/HTN, mild lung disease
ASA III	A patient with severe systemic disease	Substantive functional limitations; One or more moderate to severe diseases. Poorly controlled DM or HTN, COPD, morbid obesity (BMI ≥40), active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, ESRD undergoing regularly scheduled dialysis, history (>3 months) of MI, CVA, TIA, or CAD/stents.
ASA IV	A patient with severe systemic disease that is a constant threat to life	Recent (<3 months) MI, CVA, TIA or CAD/stents, ongoing cardiac ischemia or severe valve dysfunction, severe reduction of ejection fraction, shock, sepsis, DIC, ARD or ESRD not undergoing regularly scheduled dialysis
ASA V	A moribund patient who is not expected to survive without the operation	Ruptured abdominal/thoracic aneurysm, massive trauma, intracranial bleed with mass effect, ischemic bowel in the face of significant cardiac pathology or multiple organ/system dysfunction
ASA VI	A declared brain-dead patient whose organs are being removed for donor purposes	

Complications of General Anesthesia

Most healthy people tolerate general anesthesia without issue. Those who do suffer side-effects or complications typically have mild, transient symptoms that are easily managed. The following are possible complications of general anesthesia:

- Sore throat
- Nausea and vomiting
- Damage to teeth
- Lacerations (cuts) to the lips, tongue, gums, throat
- Nerve injury secondary to body positioning
- Awareness under anesthesia
- Anaphylaxis or allergic reaction
- Malignant hyperthermia
- Aspiration pneumonitis
- Respiratory depression
- Stroke
- Hypoxic brain injury
- Embolic event
- Cardiovascular collapse, cardiac arrest
- Death

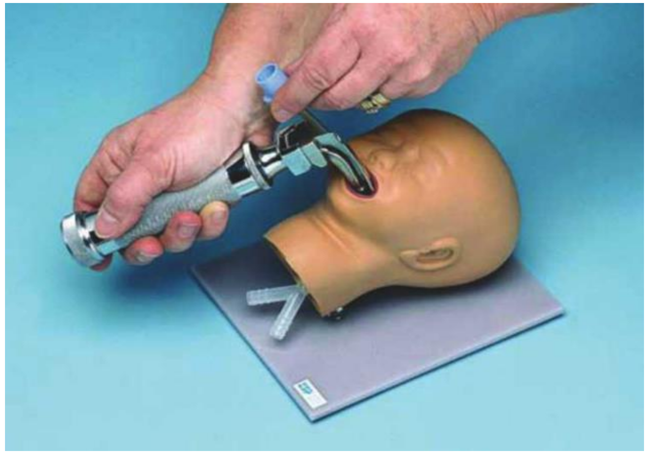
Awareness Under Anesthesia

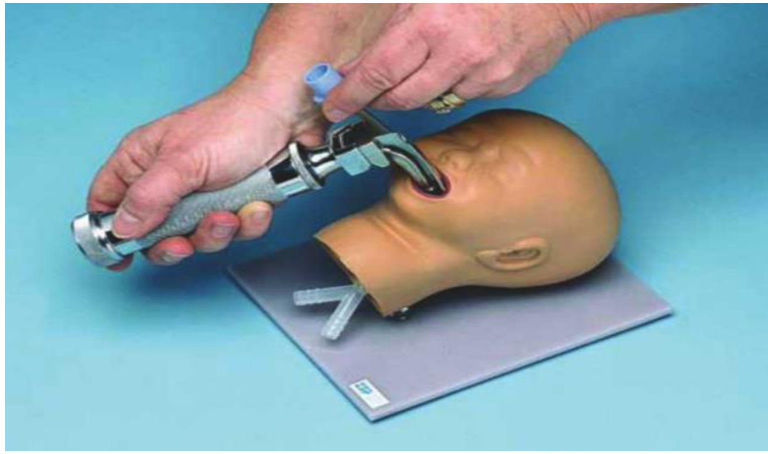
Awareness during general anesthesia seems to be **one of the biggest concerns** for patients, but is very rare. Approximately **1-2 patients per 1000 general anesthetics may briefly become aware of their surroundings**, but usually do not feel pain.

In extremely rare instances, a patient can have awareness of their situation and experience pain while under general anesthesia.

Because of paralytics given to facilitate surgery, the patient may not be able to move to make others aware of their distress. People who truly experience this level of awareness may **develop psychological issues similar to post-traumatic stress disorder (PTSD).**

Certain situations are associated with a **higher risk of some level of awareness**. Also known as unintended **intra-operative awareness, it may occur with:**





- Emergency surgery
- Trauma surgery
- Cesarean surgery under general anesthesia
- Heart surgery involving cardiopulmonary bypass
- Depression
- Daily alcohol use
- Errors by the anesthesiologist-may may include improper monitoring of the patient and/or the amount of anesthesia administered during a procedure

❖ Disadvantages

Dental treatment under GA has its disadvantages such as:

1-the absence of patient’s protective reflexes,

2-depression of vital signs.

3-higher rate of intra- and postoperative complications compared to local anesthesia (LA).

❖ Indications

The main use of G.A is to those patients who are unable to cooperate during their dental appointment and fail to help in their treatment due to:

-Fear.

- Apprehension.

-Extreme nervousness.

-Hysteria

- Mental deficiency or insanity.

-Young children cannot distinguish between pressure and pain so they will be uncooperative.

❖ **Contra- indication of G.A. in dental chair:-**

1-Diseases that impairs either respiratory efficiency or air way

2-Acute infection of the floor of the mouth.

3-Edema of the glottis and laryngeal obstruction.

***In all above diseases, if surgical intervention is indicated, so G.A. is preferable in hospital.

4-Old patient with hypertensive or ischemic heart disease.
5-Pregnant woman: fear of fetus damage, abortion or miscarriage.
6-very large and very obese patients are often **unsuitable** for G.A. in the dental chair

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