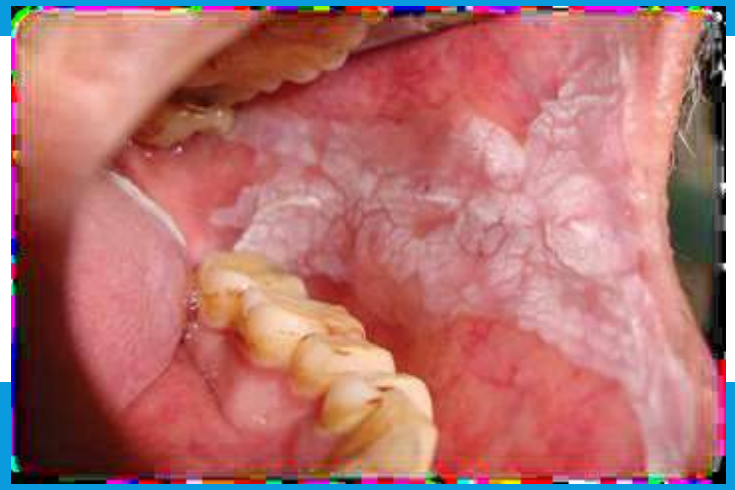
PREMALIGNANT LESIONS AND PREMALIGNANT CONDITIONS

DEFINITION

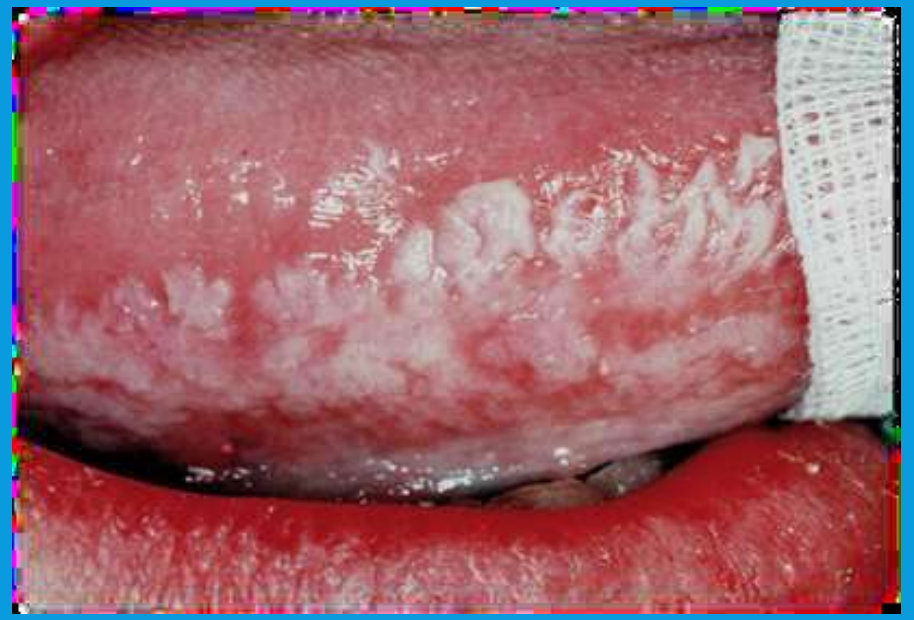
- premalignant lesion is “A morphologically altered tissue in which oral cancer is more likely to occur than in its apparently normal counterpart
- Premalignant condition is ‘a generalized state associated with a significantly increased risk of cancer

Premalignant lesions	Premalignant conditions
Leukoplakia	Oral submucous fibrosis
Erythroplakia	Oral lichen planus
Leukokeratosis nicotina palatinae	Actinic keratosis
Candidiasis	Syphilis
Carcinoma in situ	Discoid lupus erythematosus
	Sideropenic dysphagia

LEUKOPLAKIA

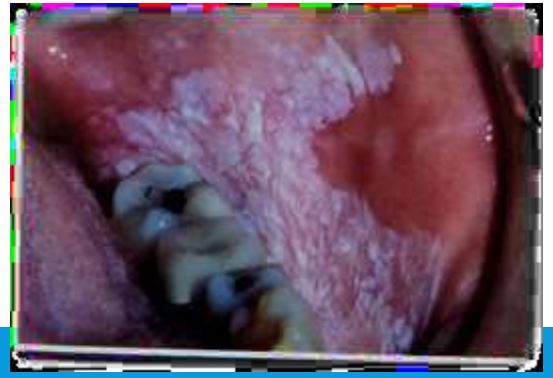


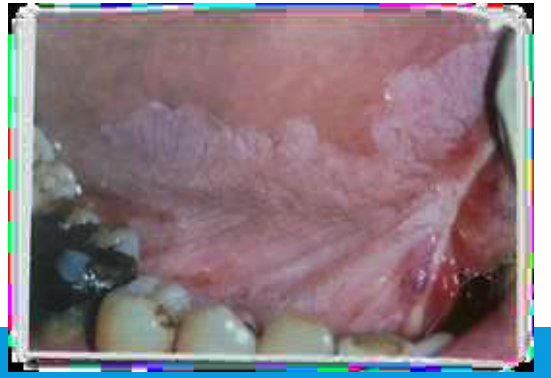
- *(leuko* = white; *plakia* = patch) is defined by the World Health Organization (WHO) as
- "a white patch or plaque that cannot be characterized clinically or pathologically as any other disease.
- The most common precancer lesion
- Causes: tobacco, alcohol, sanguinaria , trauma, infection, UV rays
- Male predilection
- Mostly occurs above 40 y/o,
- Sites: lip, vermilion of lip, buccal mucosa, gingiva



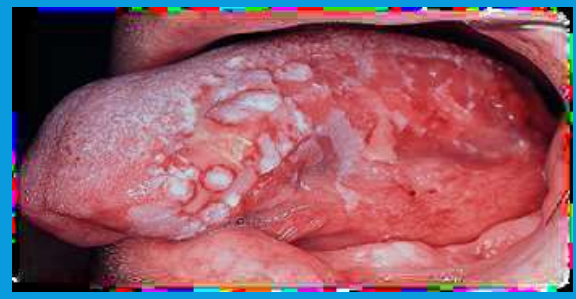
- **TWO types:**
- **1. Homogenous**
- **2. Non-homogenous**

HOMOGENOUS



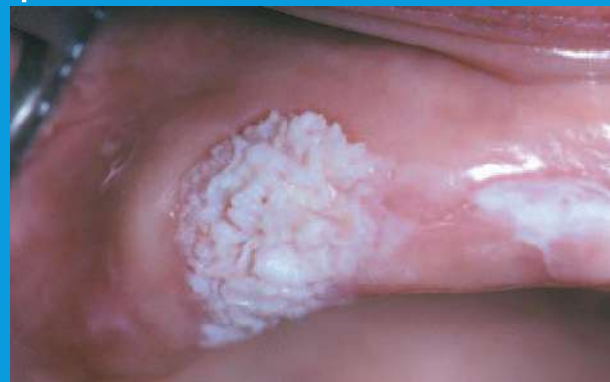


- Uniform white patch lesion with smooth or corrugated surface sometimes, slightly raised mucosa. Usually plaque like, some are smooth, may be wrinkled or fissured
- Malignant transformation – 1 to 7%.



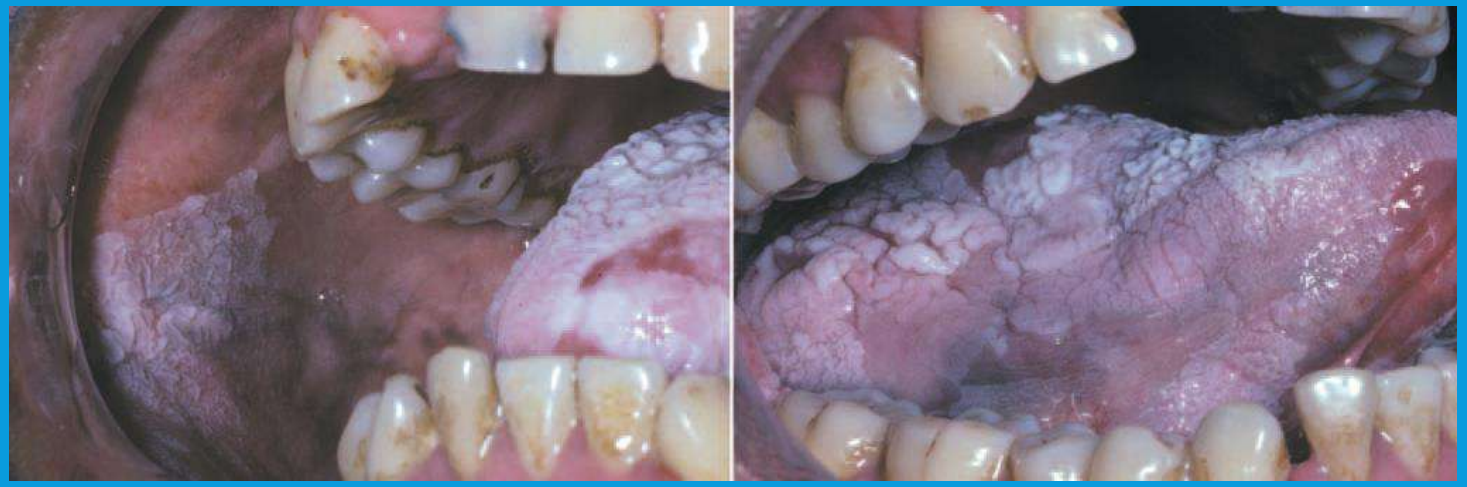
Non-homogenous : 3 types

- **Ulcerative**- Red ulcerative lesion (Atrophic epithelium) with small white specks or nodules over it.
- **Verrucous** -Warty surface (white lesion with hyperplastic surface)
- **Speckled**- Mixed red and white patches on an irregular surface



PROLIFERATIVE VERRUCOUS LEUKOPLAKIA (PVL)

- Specialized form of leukoplakia is characterized by the development of multiple keratotic plaques with roughened surface projections .
- the lesions progress, they may go through a stage indistinguishable from **verrucous carcinoma**, but they later usually develop dysplastic changes and transform into **squamous cell carcinoma** (usually within 8 years of initial PVL diagnosis).



HISTOPATHOLOGY

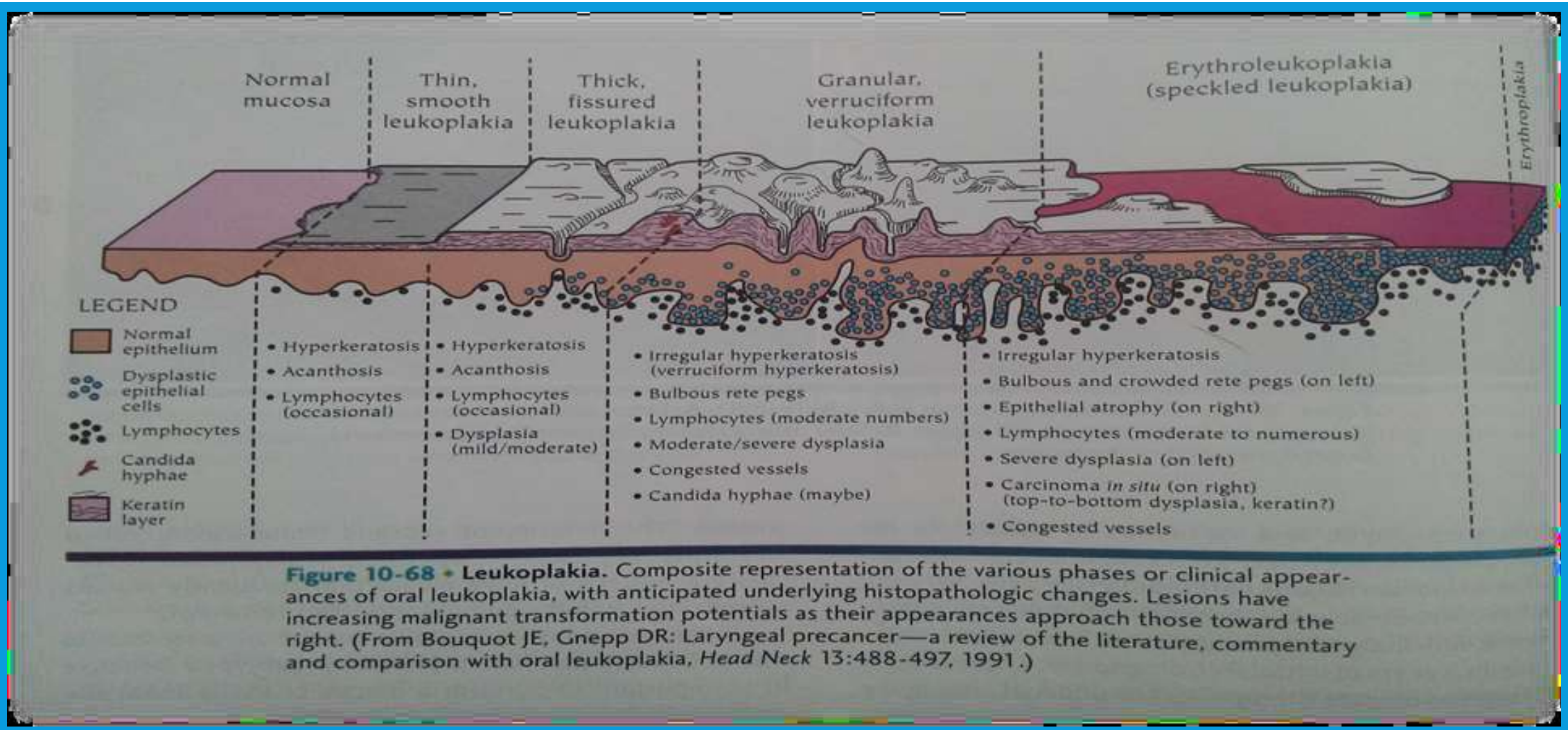
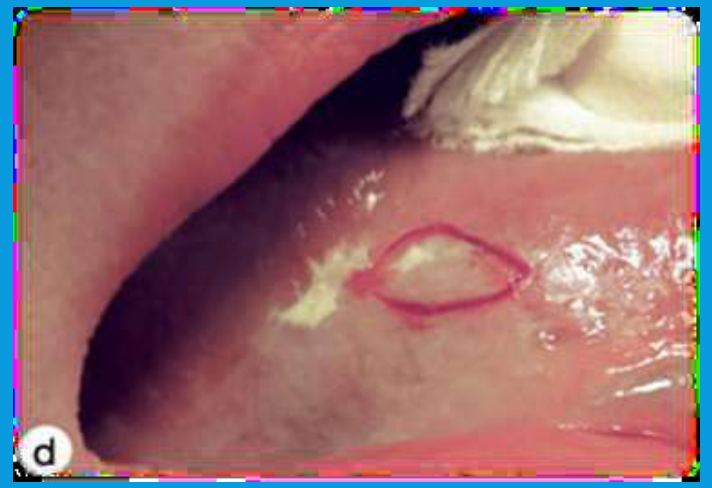


Figure 10-68 • Leukoplakia. Composite representation of the various phases or clinical appearances of oral leukoplakia, with anticipated underlying histopathologic changes. Lesions have increasing malignant transformation potentials as their appearances approach those toward the right. (From Bouquot JE, Gnepp DR: Laryngeal precancer—a review of the literature, commentary and comparison with oral leukoplakia, *Head Neck* 13:488-497, 1991.)

TREATMENT AND PROGNOSIS

- biopsy is mandatory and will guide the course of treatment.
- Mild dysplasia: eliminate risk factors + follow up
- Moderate to severe dysplasia: surgical excision by one of 4 methods:
- **1. Scalpel excision / Stripping**
- **2. Electrocautery**
- **3. Cryotherapy**
- **4. CO_2 Laser therapy**

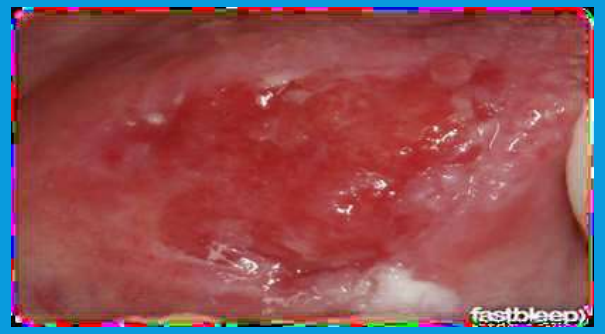


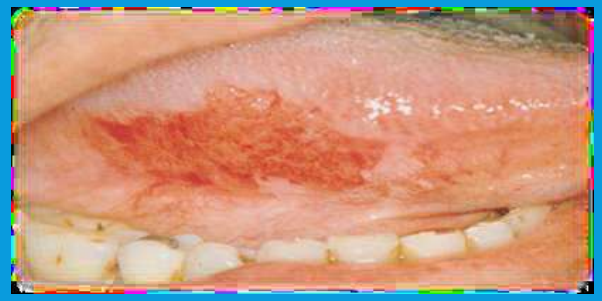
ERYTHROPLAKIA

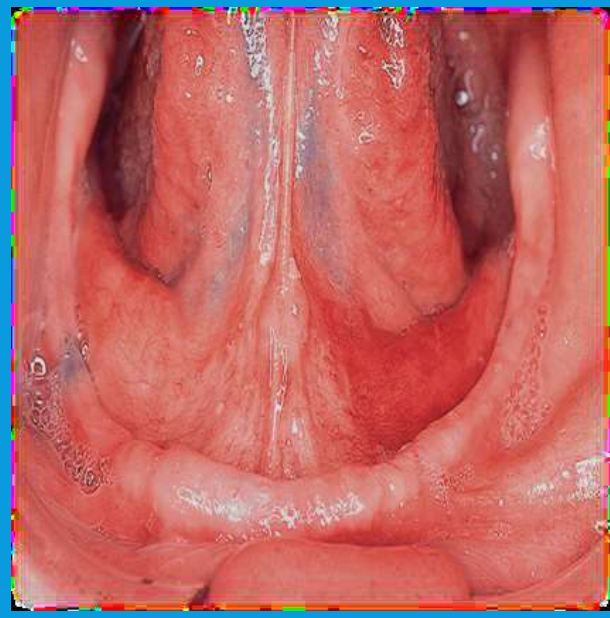
- fiery red patch that cannot be characterized clinically or pathologically as any other definable disease.
- is more common in males and occurs more frequently in the 6th and 7th decade of life.
- **CLINICAL FEATURE**
- Red, often velvety, well-defined patches. Most commonly present on

floor of mouth, retromolar trigone area, lateral tongue.

- Usually asymptomatic







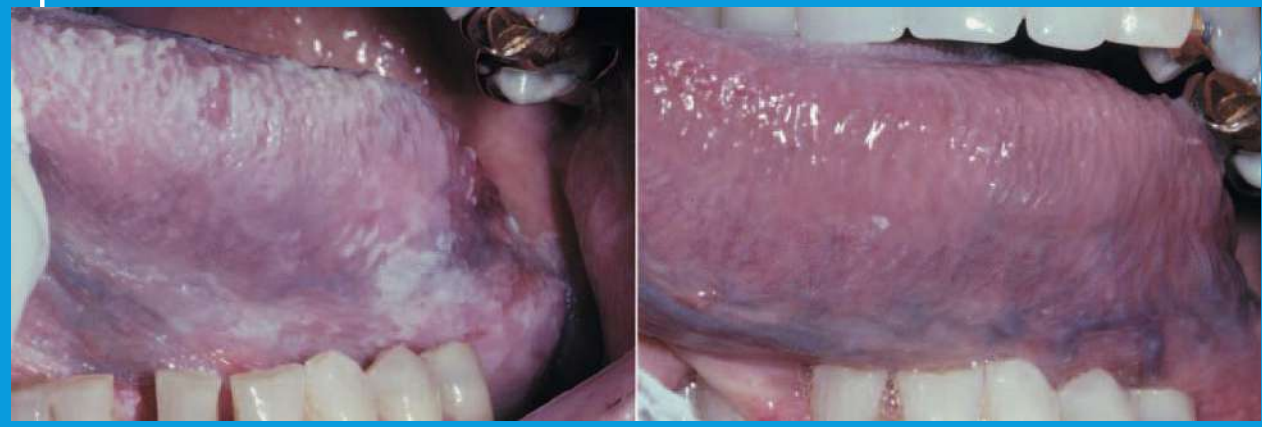
DIFFERENTIAL DIAGNOSIS

- Erythematous (atrophic) candidiasis
- Kaposi's sarcoma
- Ecchymosis
- Contact stomatitis
- Vascular malformation
- Squamous cell carcinoma
- Geographic tongue/ erythema migrans

Treatment as in leukoplakia :
Biopsy first then removed completely

CHRONIC HYPERPLASTIC CANDIDIASIS

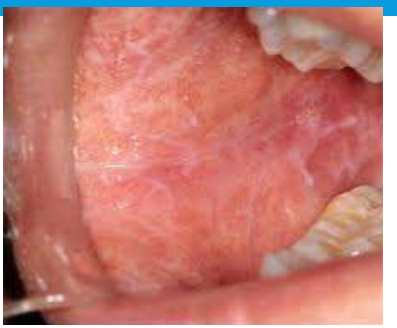
- Leukoplakia associate with candidosis
- Middle and elderly with equal predilection
- Cause: C. Albicans with co-factors
- Asymptomatic , tough adherent white plaque
- Sites: dorsum of tongue+buccal mucosa
- Biopsy show candida hyphae
- Treatment: control risk factors
- antifungal drugs
- excision for persistent lesions

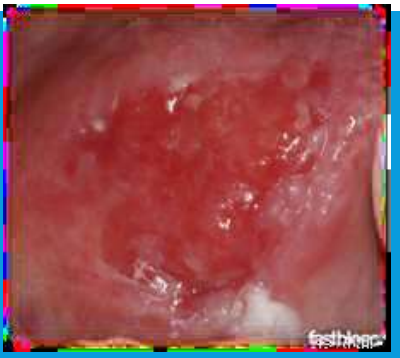


ACTINIC (SOLAR) KERATOSIS (CHEILITIS)

- is potentially malignant disorder associated with long term exposure to radiation and may be found on the vermilion border of the lips as well as other exposed skin surfaces.
- atrophy of the lower lip vermilion border, characterized by a smooth surface and blotchy pale areas.
- The areas thicken and may appear as leukoplakic lesions.
- The patient may report that the scaly material can be peeled off with some diffi culty, only to reform again within a few days.
- With further progression, chronic focal ulceration may develop.
- Treatment: In clinically severe cases without obvious malignant transformation, a lip shave procedure **(vermilionectomy)** may be performed.
- if a SCC is identified, then resection of tumor with safe margin

LICHEN PLANUS





- Autoimmune. T cell–mediated disease targeting basal keratinocytes
- F bias, above 40 y/o,
- Variants: 1) reticular (most common oral form)

2)erosive (painful)

3) Atrophic

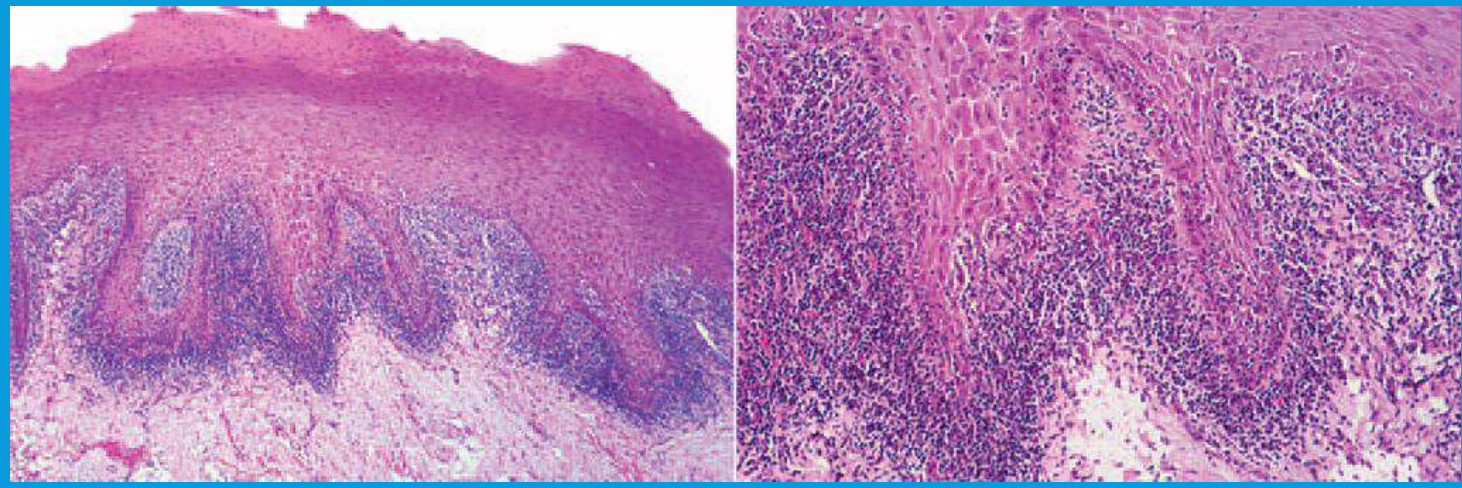
4) Plaque

5) bullous (rare)

- Bilateral and often symmetric distribution
- Oral site frequency: buccal mucosa (most frequent), then tongue, then gingiva,

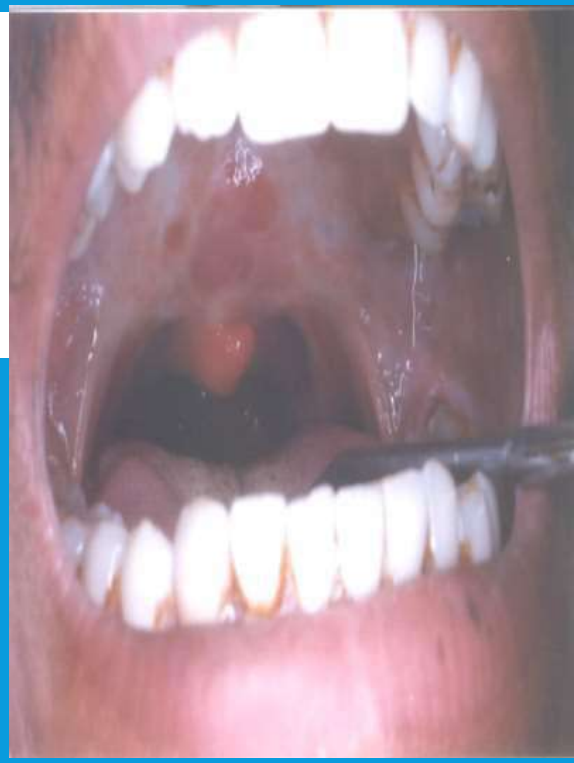
HISTOPATHOLOGY

- Subepithelial cellular infiltrate (T-lymphocytes)
- Hyperkeratosis
- Civatte bodies
- Saw-tooth appearance rete pegs



Treatment: topical steroids+/-immunosuppression

ORAL SUBMUCOUS FIBROSIS



- Premalignant disorder seen commonly in India and Indian subcontinent
- Chronic mucosal progressive disorder associated with chewing betel quids.
- S.& S.: Difficulty in opening mouth
- Inability to whistle, blow
- Difficulty in swallowing
- Referred pain to the ear
- Changes in tone of the voice due to vocal cord involvement
- Sometimes deafness due to occlusion of eustachian tubes
- Treatment:
- 1.Restriction of habits/ Behavioral therapy.
- 2.Non-surgical therapy.
- 3.Surgical therapy.
- 4.Oral Physiotherapy:

PREPROSTHETIC SURGERY BY

DR. SABAH ALHEETI

WHAT IS PREPROSTHETIC SURGERY?

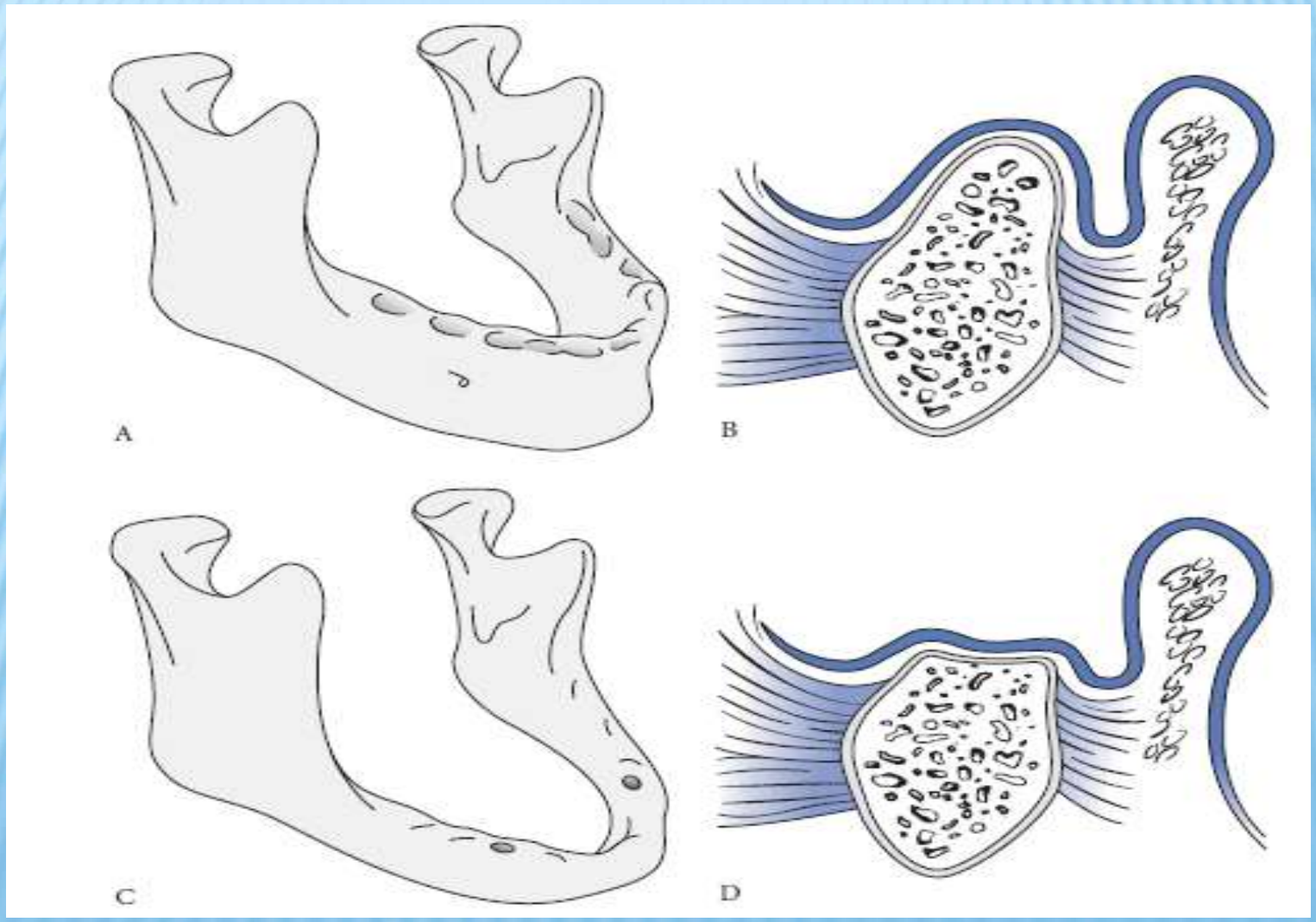
- “surgical Procedures intended to improve the denture bearing surfaces of the mandible and maxilla”
- The objective:

To provide a better anatomic environment and proper supporting structures for denture construction (improvement function and esthetic).

FUNCTIONAL EFFECTS OF EDENTULISM

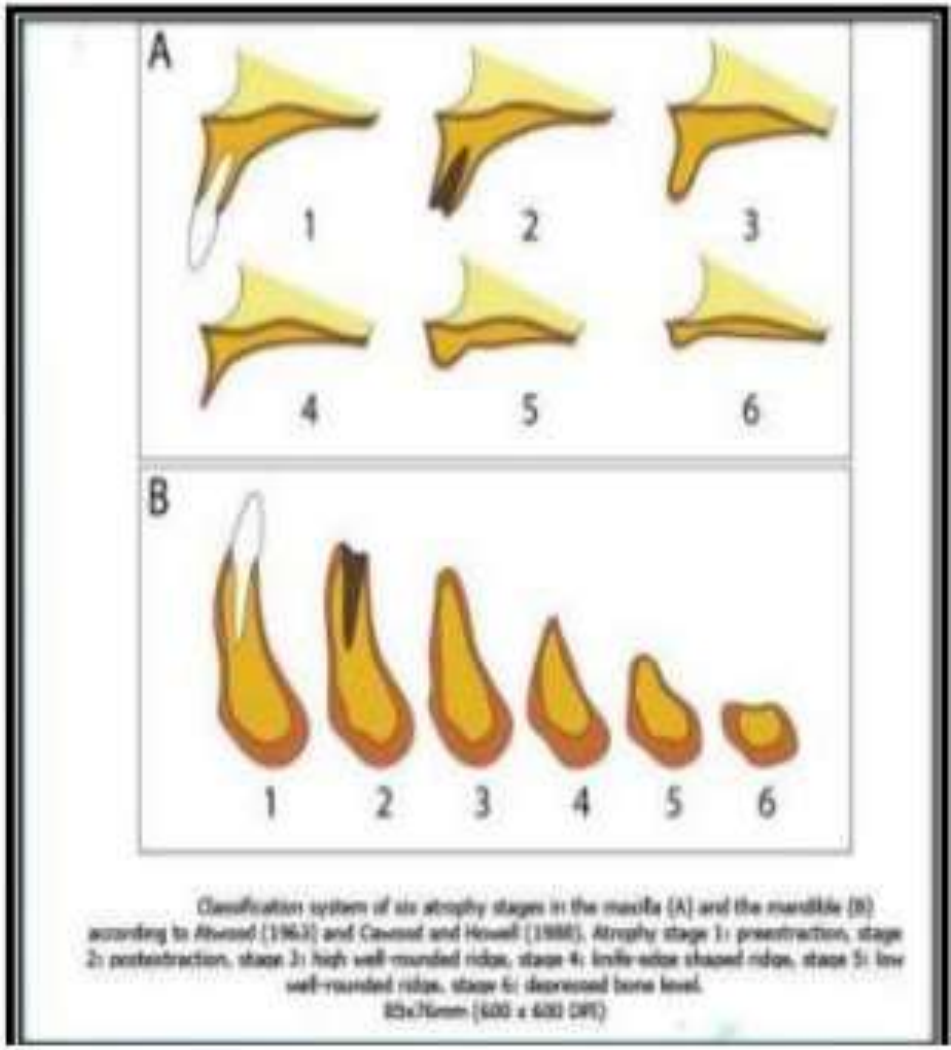
A. Decrease size and form of ridge in all dimensions
B. Decreased alveolar support for traditional prostheses
C. Encroachment of muscle and tissue attachments to the alveolar crest resulting in progressive instability of conventional soft tissue–borne prosthetic devices.
D. Neurosensory changes secondary to atrophy.
E. There is a progression toward decreased overall lower facial height, leading to the typical overclosed appearance

PATTERN OF MANDIBULAR ATROPHY



As given by ATWOOD (1963) CAWOOD & HOWELL(1988)

- Class I –Pre extraction
- Class II - post extraction.
- Class III - rounded ridge, adequate height and width
- Class IV - knife edge ridge, adequate height, inadequate width.
- Class V - flat ridge, inadequate height and width.
- Class VI - depressed ridge with varying degrees of basal bone loss, that may be extensive but follows no predictable pattern.



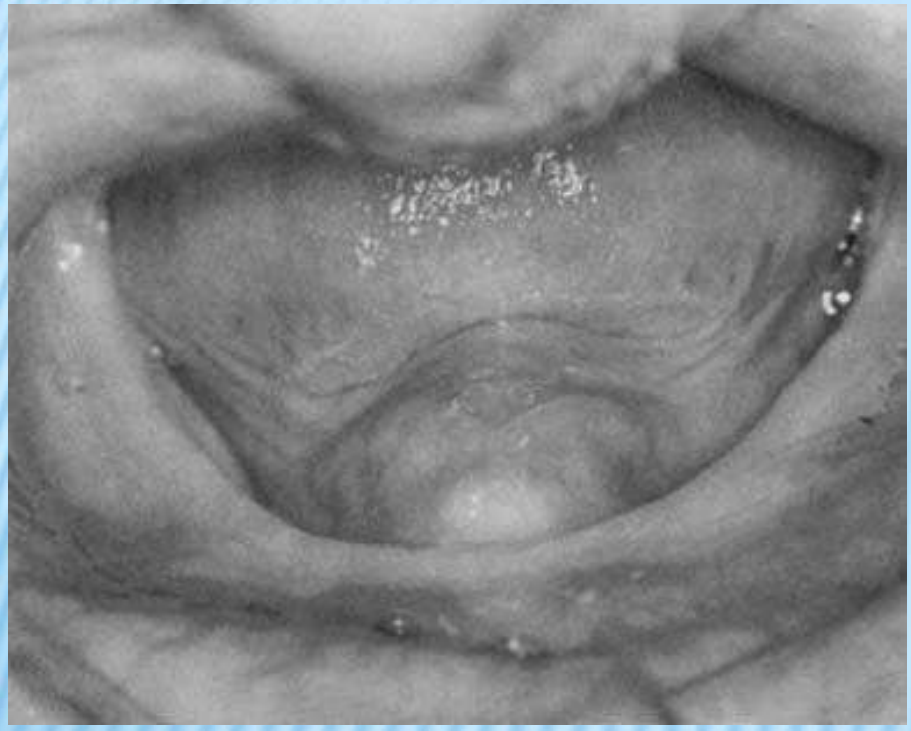
PROSTHETIC SURGERY WORK -UP

- Chief complain and patient expectation
- History
  - HPI
  - Medical Hx/ surgical Hx
  - Dental Hx
  - Social Hx

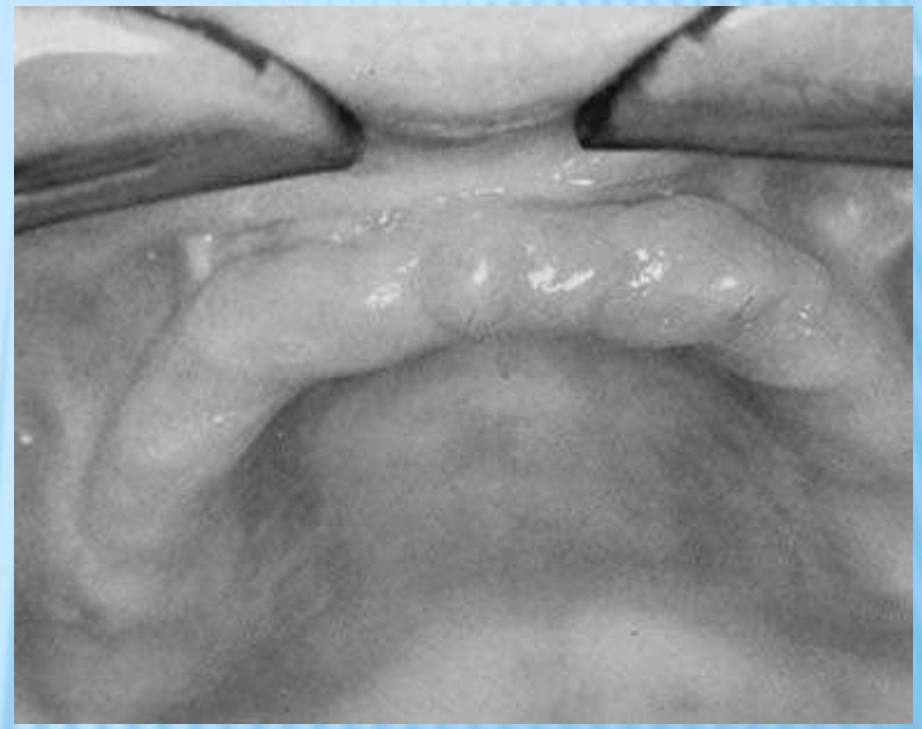
PHYSICAL EXAMINATION

- Identification of both soft tissue and underlying bone characteristics and/or deficiencies is essential to formulate a successful reconstructive plan.

MANDIBLE



MAXILLA

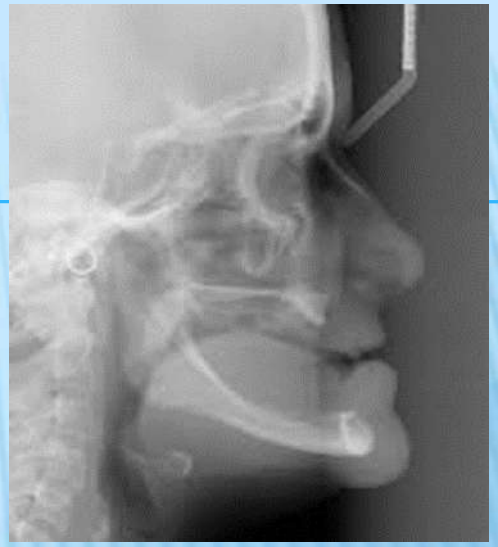


PHYSICAL EXAM

- –Visual inspection and palpation

•Existing occlusal relationships
•Existing jaw relationships / interarch relationships
•Amount of remaining bone (height/width)
•Contours of remaining bone
•Quality of existing soft tissue
•Character of existing soft tissue
•Vestibular depths
•Muscle attachments
•Anatomic relationships
•Presence of hard or soft tissue pathologic conditions

RADIOGRAPH

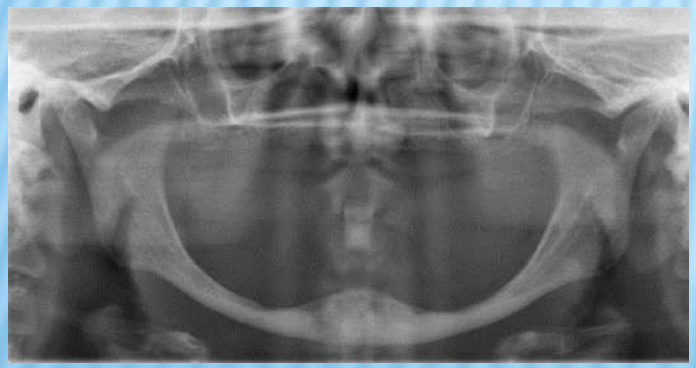


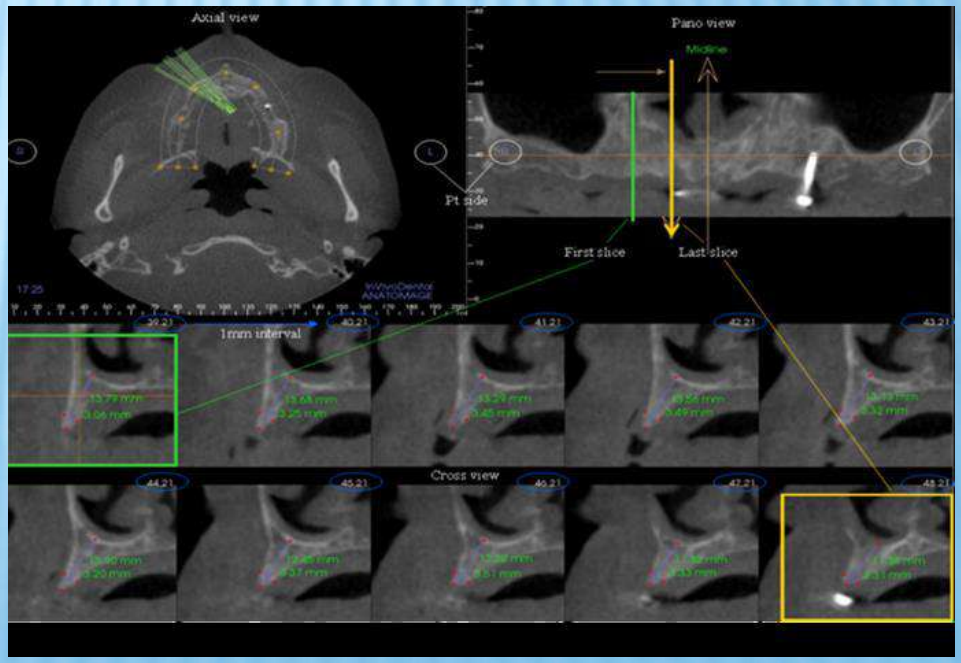
1- Plain films

2- Panoramic radiograph

3- Cephalometric radiograph

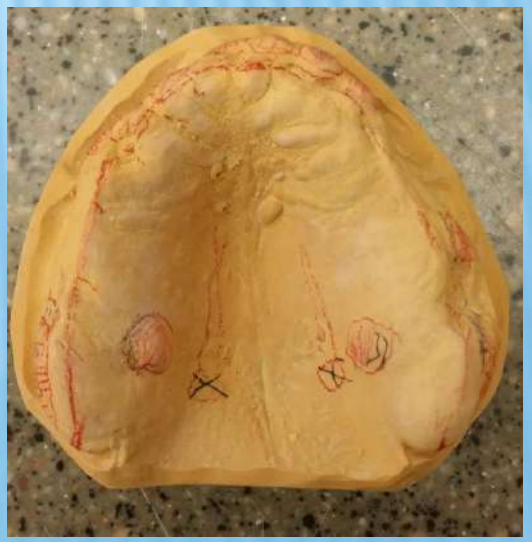
4- CBCT

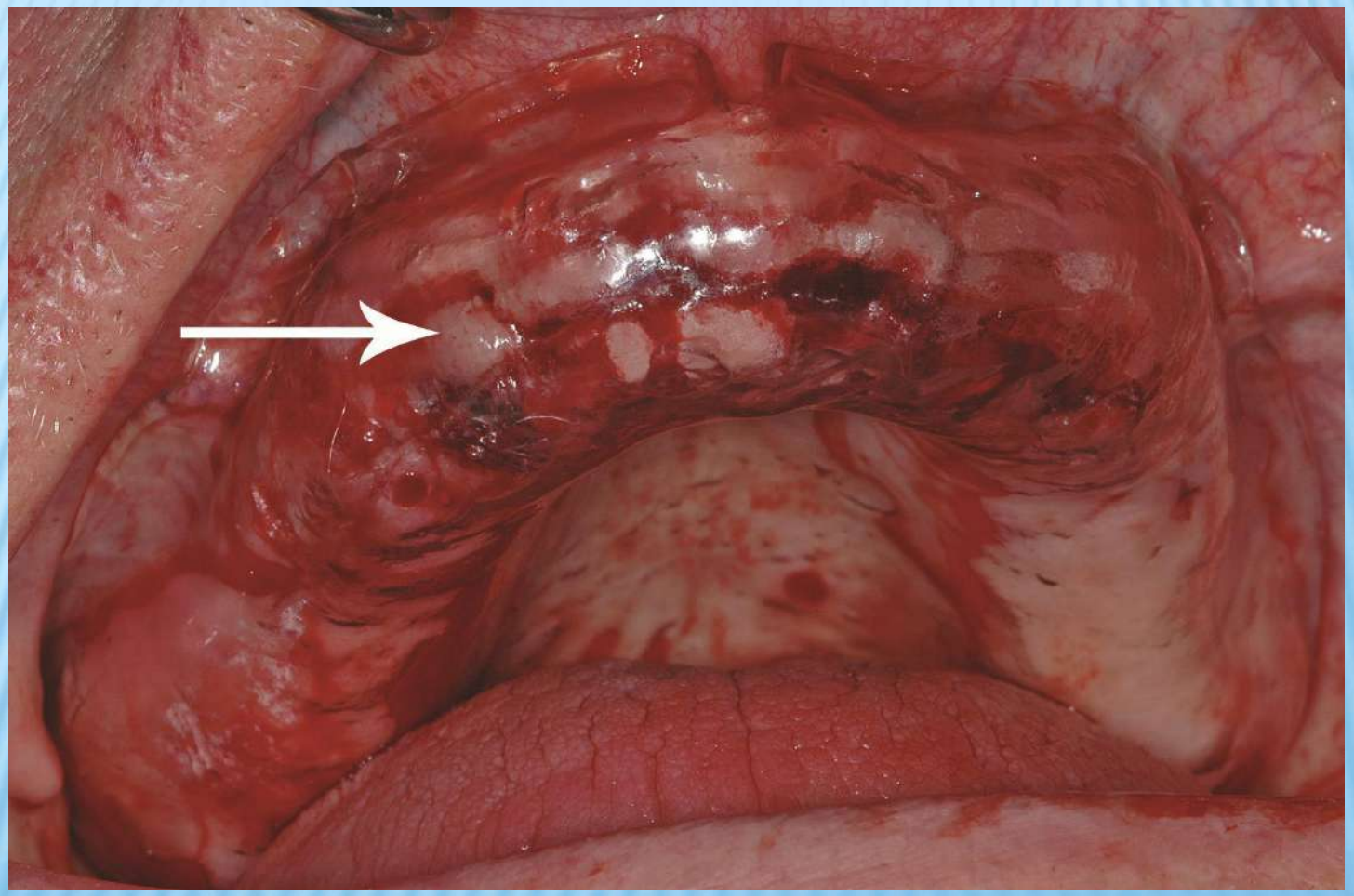




DIAGNOSTIC MODELS (CASTS)

- Provide best opportunity to coordinate cast surgery with actual surgery, and improvement of patient comfort
- Provides for verification of ridge reduction before insertion of the immediate complete denture
- Enables the surgeon to visually check for pressure points before inserting the immediate denture.
- Can be used to maintain/support repositioned tissues
- Can be used to contain grafting/augmentation materials





TYPES OF PREPROSTHETIC SURGERY

- Procedures involve hard tissue:

1.Alveoloplasty
2.Maxillary Tuberosity Reduction
3. Buccal Exostosis and Excessive Undercut Removal
4. Genial Tubercle Reduction
5. Tori Reduction
6. Mylohyoid Ridge Reduction
7. Bone augmentation

- Procedures involve soft tissue:

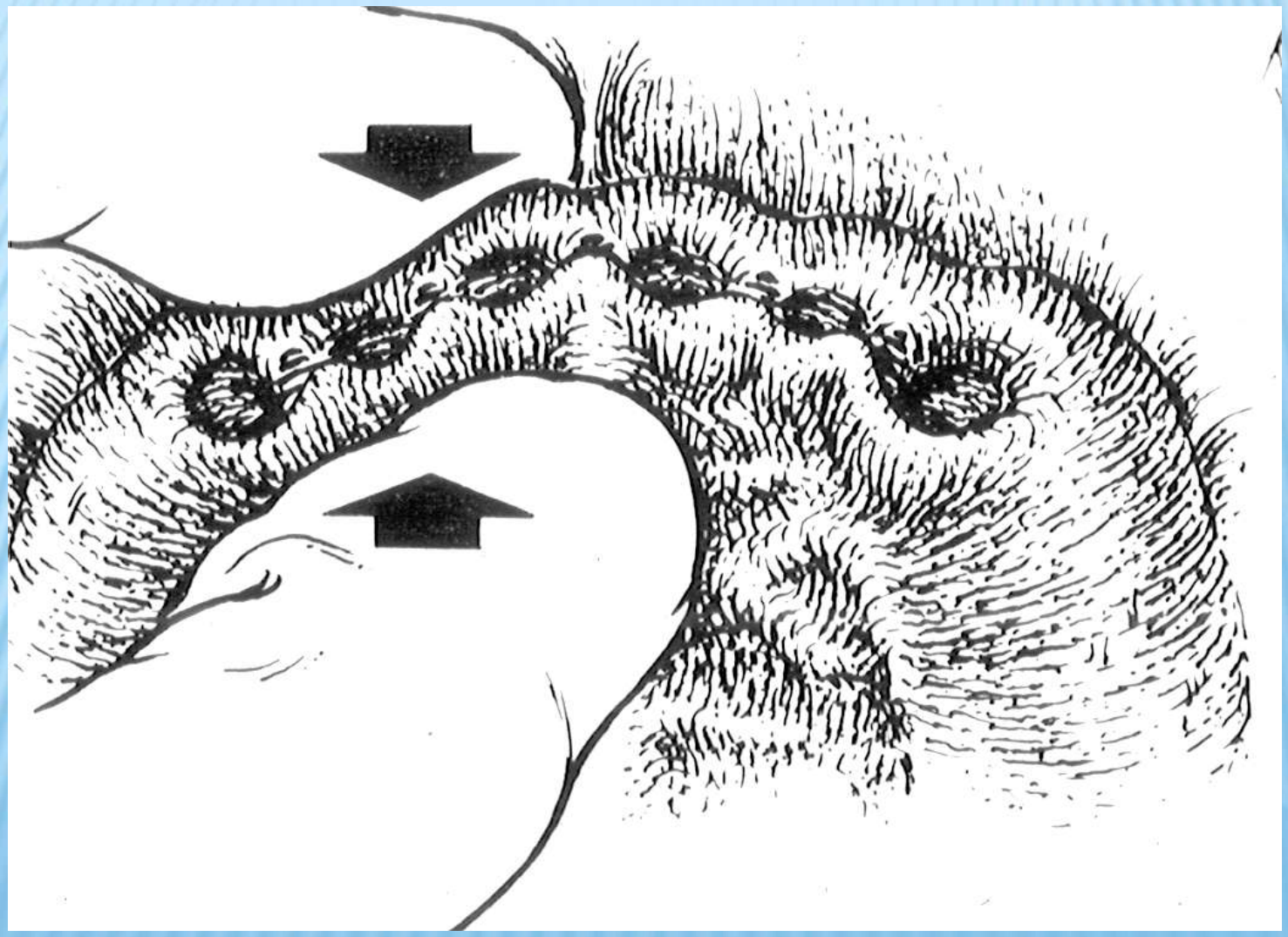
1) Maxillary Tuberosity Reduction (soft tissue)
2) Inflammatory Fibrous Hyperplasia
3) Inflammatory Papillary Hyperplasia
4) Labial Frenectomy
5) Lingual Frenectomy
6) Ridge Extension Procedures

Alveoloplasty

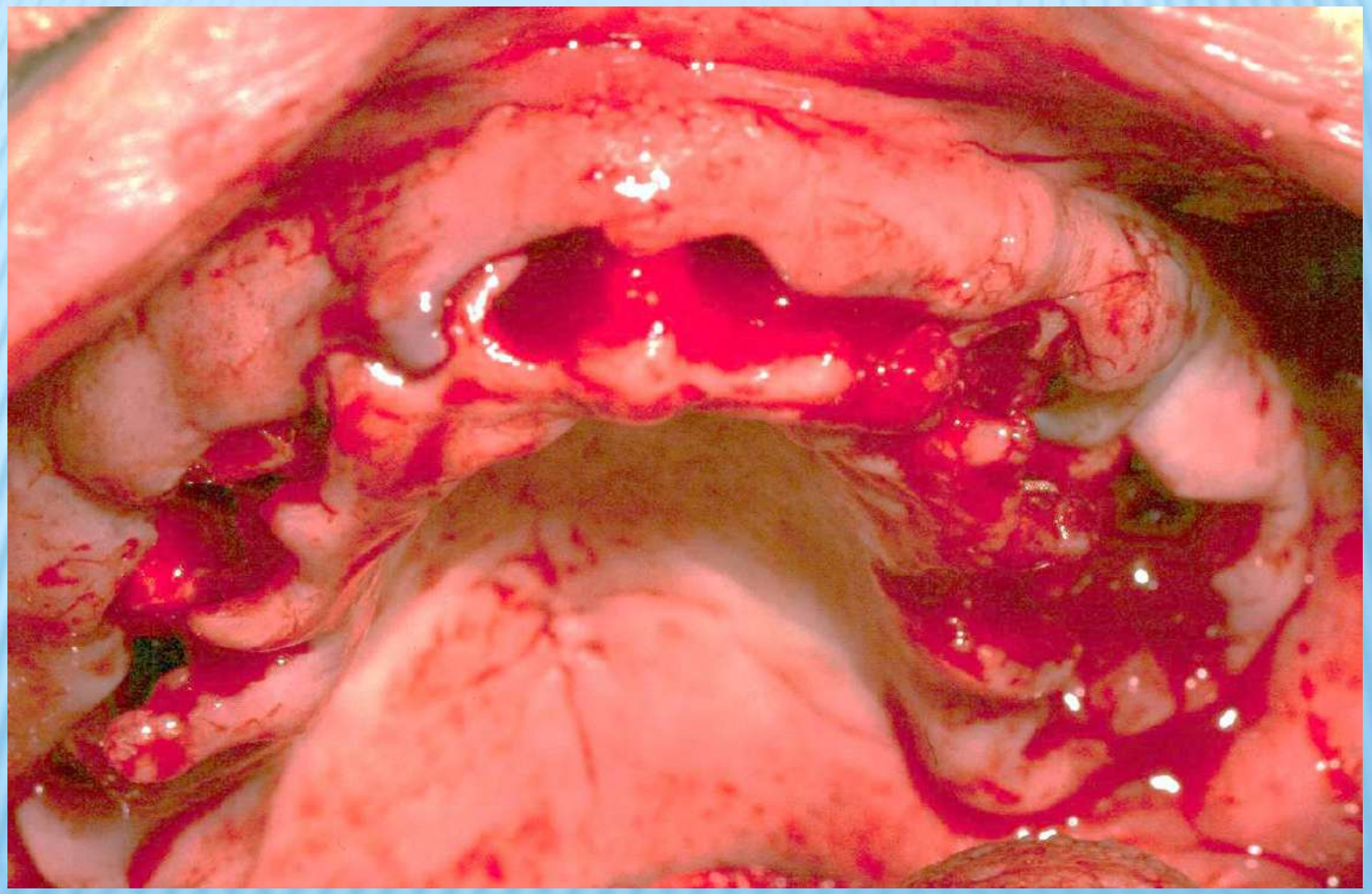
ALVEOLOPLASTY

- It involves recontouring and smoothening of alveolar arches and then covering the arches with healthy soft tissue which helps in providing a stable and retentive oral prosthesis.
- Alveoloplasty can be as simple as the compression of socket walls after closed extraction or Intraseptal alveoloplasty is another type of alveoloplasty which

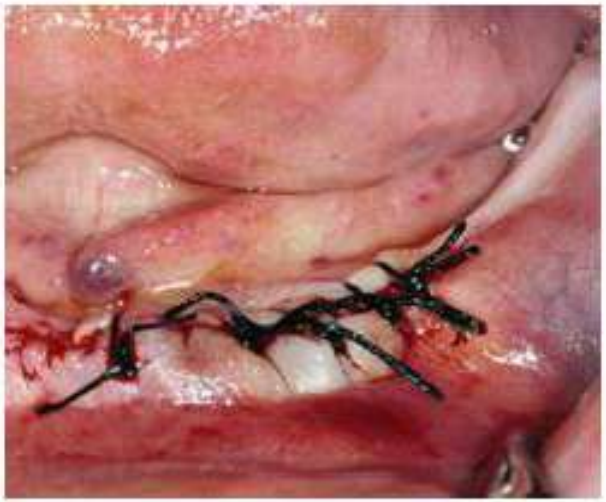
DIGITAL COMPRESSION

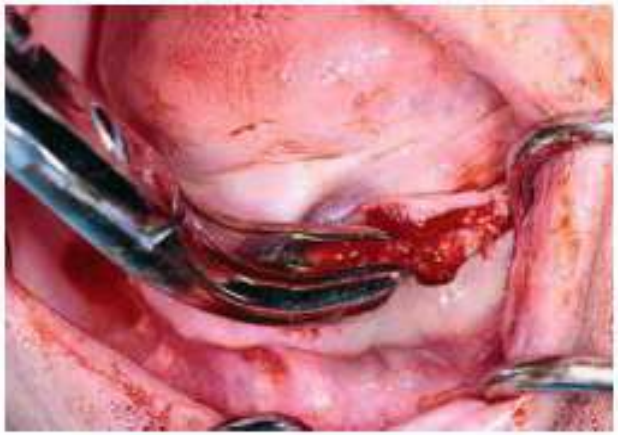


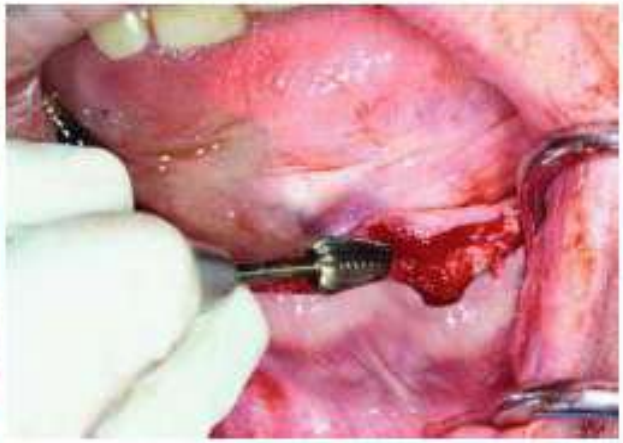
DO OR NOT?



Single Tooth Alveoloplasty with Excision of Wedges







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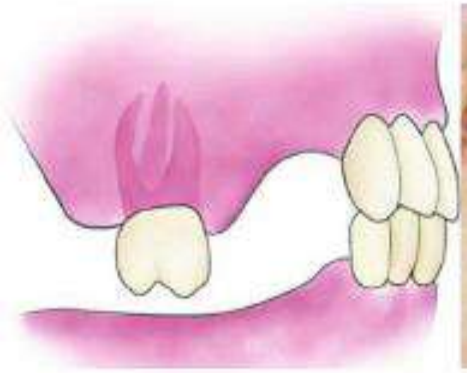
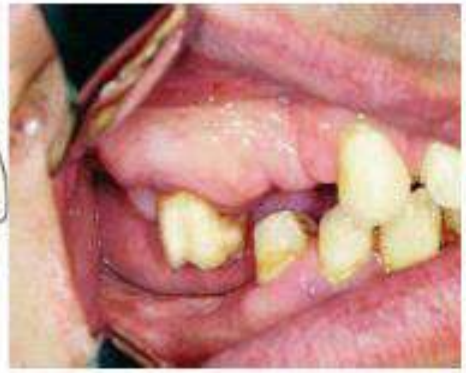
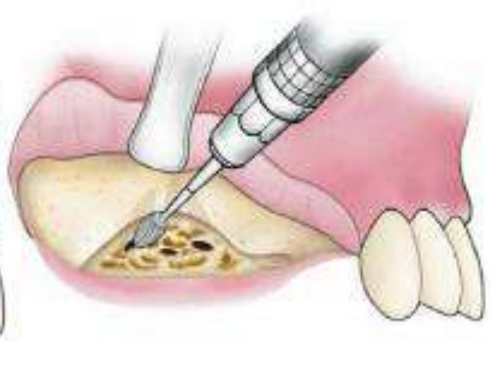
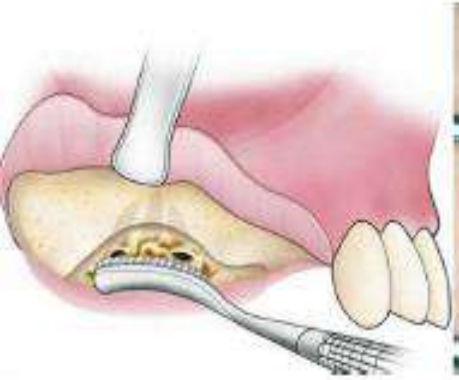
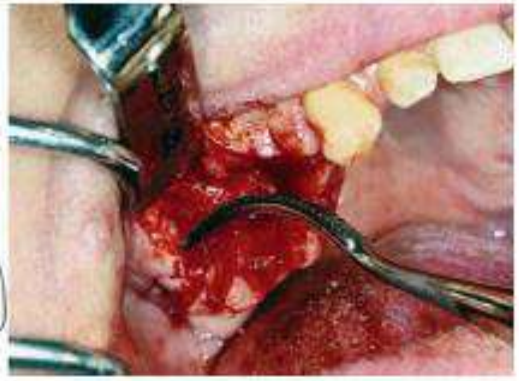
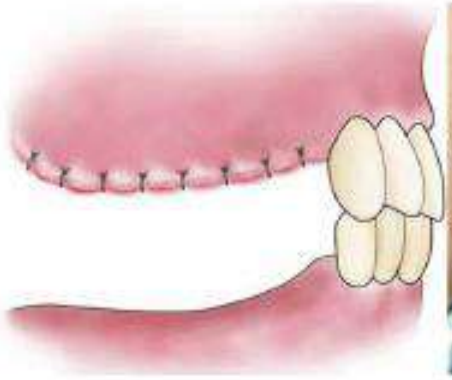
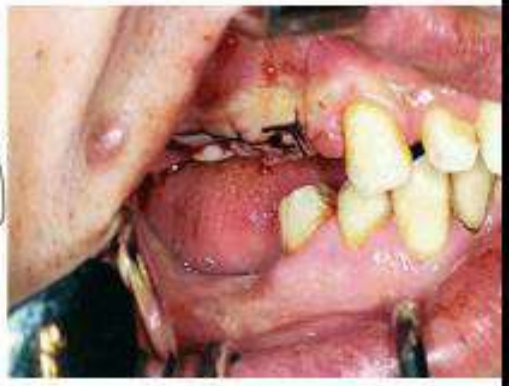
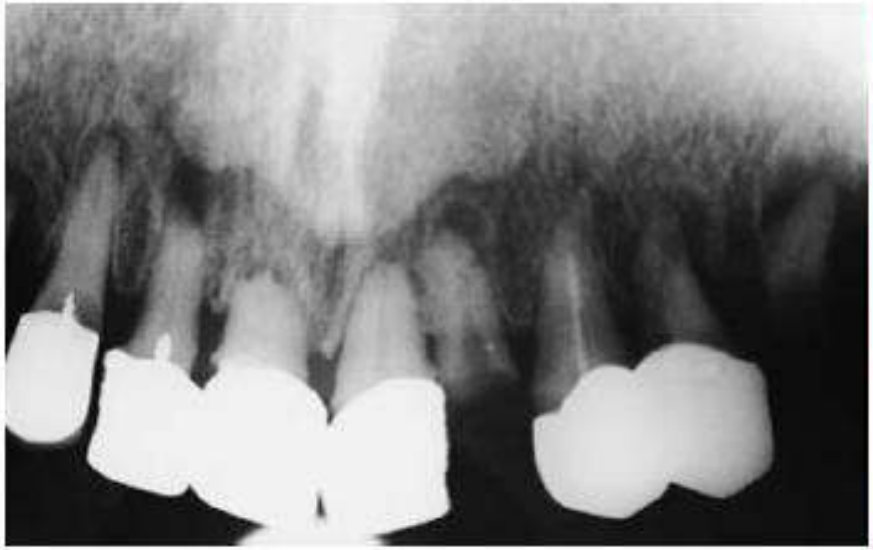
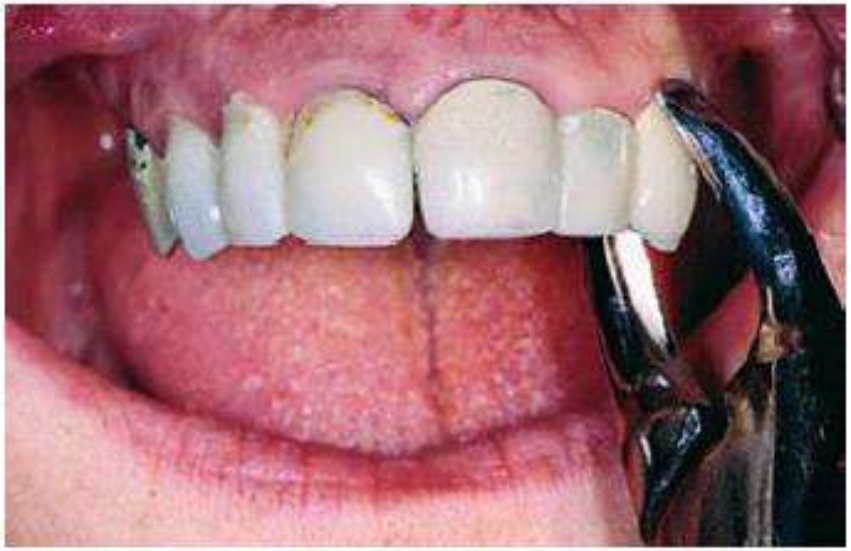
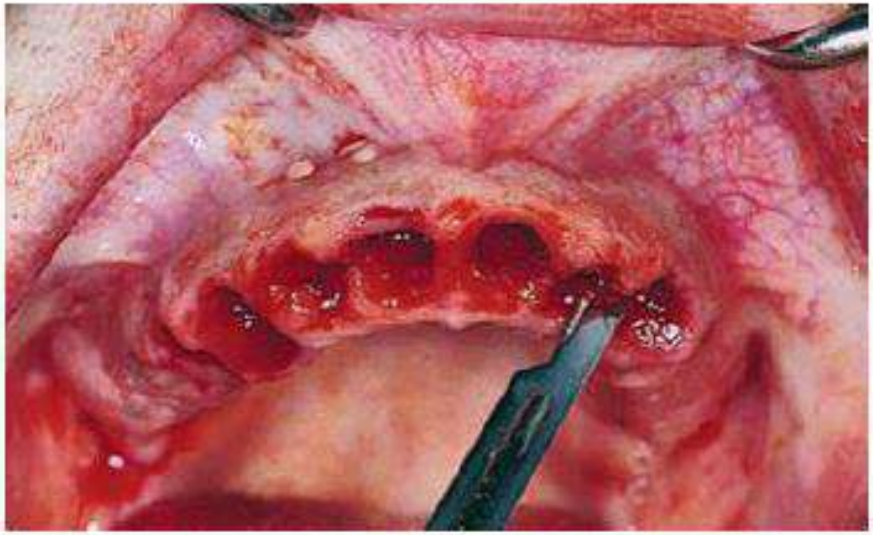



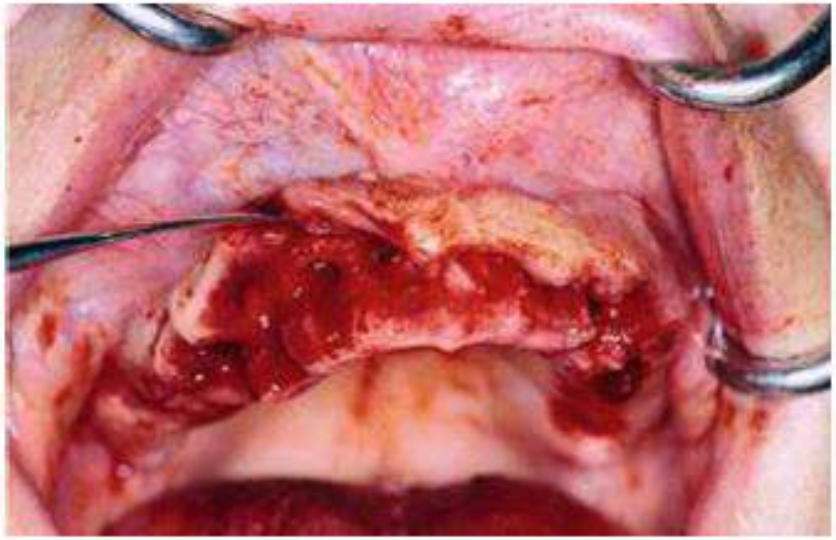






Simple Alveoloplasty

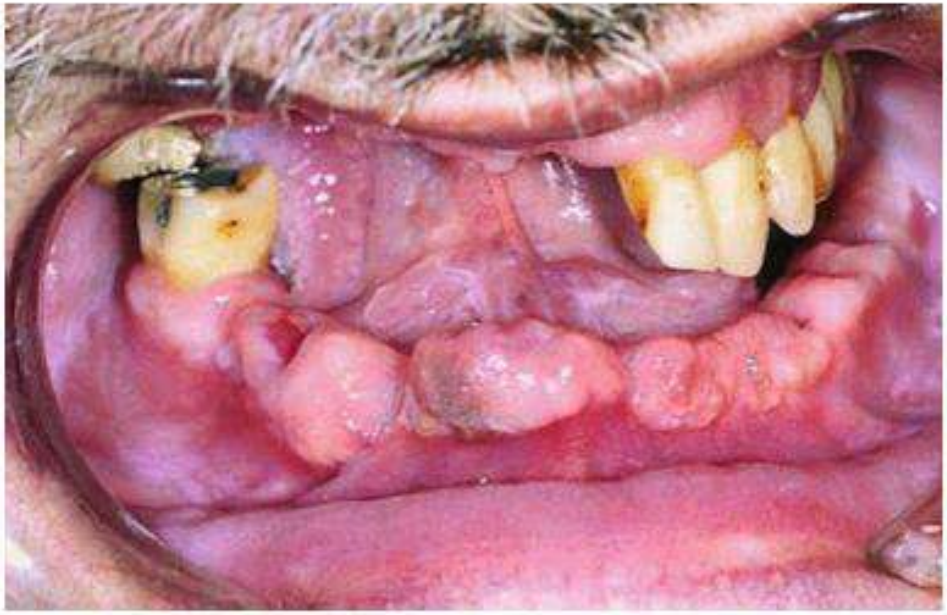


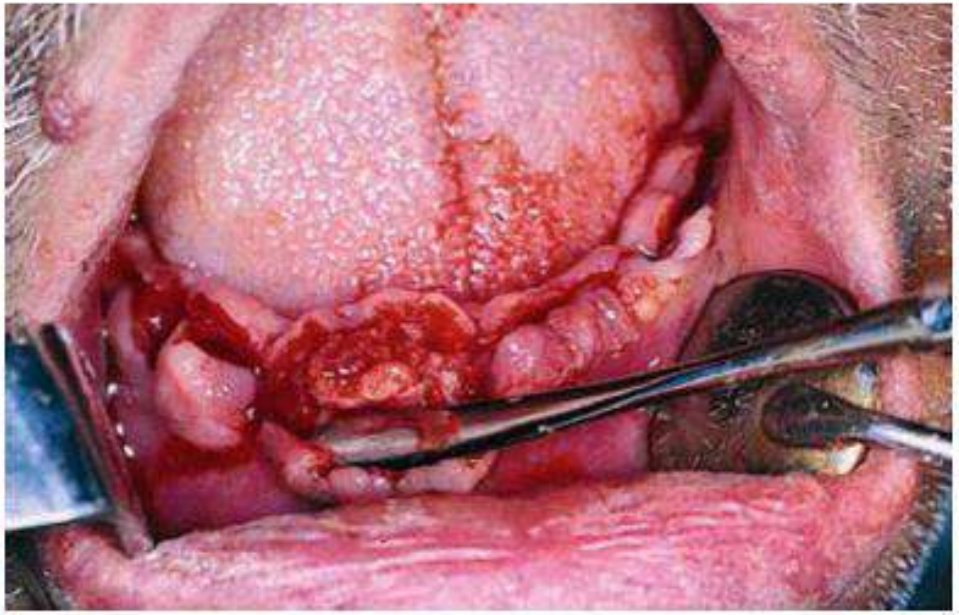


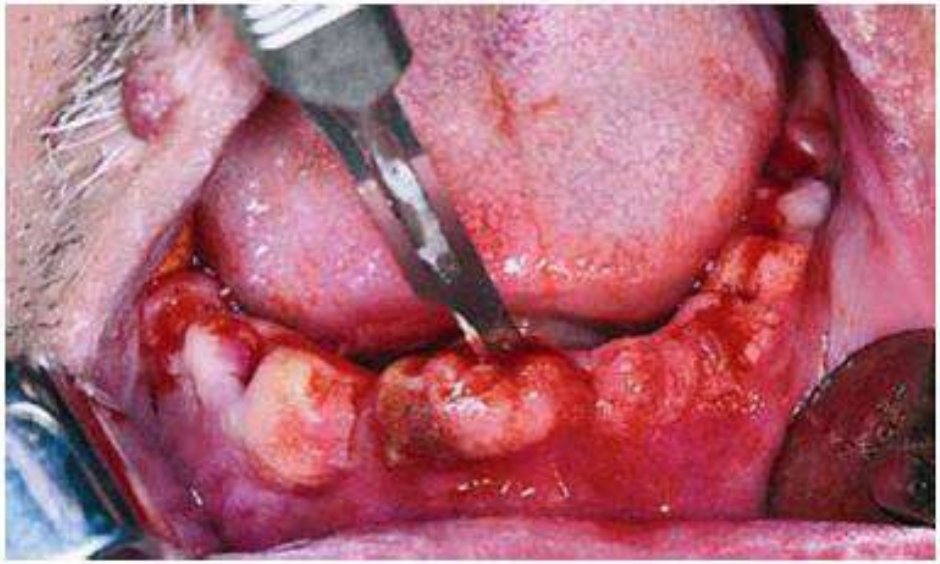


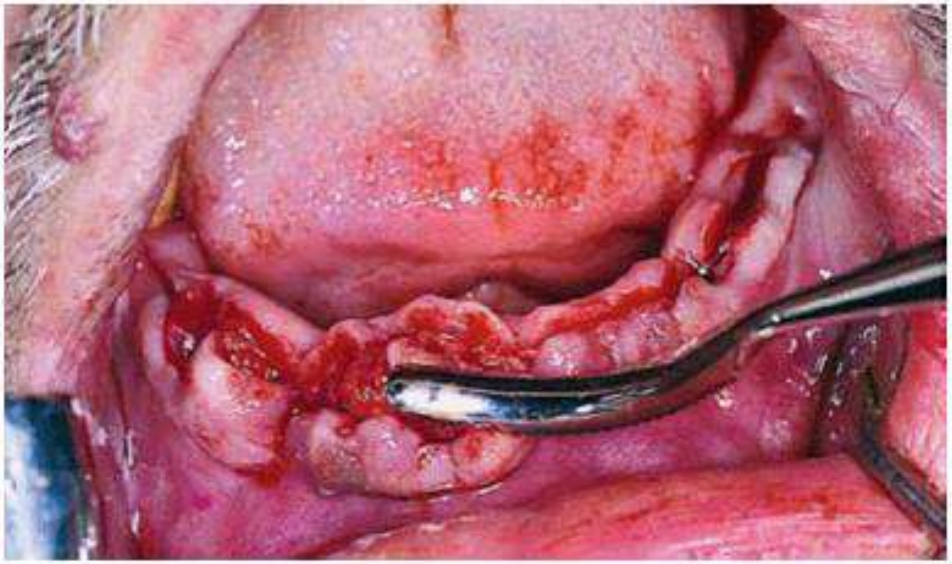


Simple Alveoloplasty

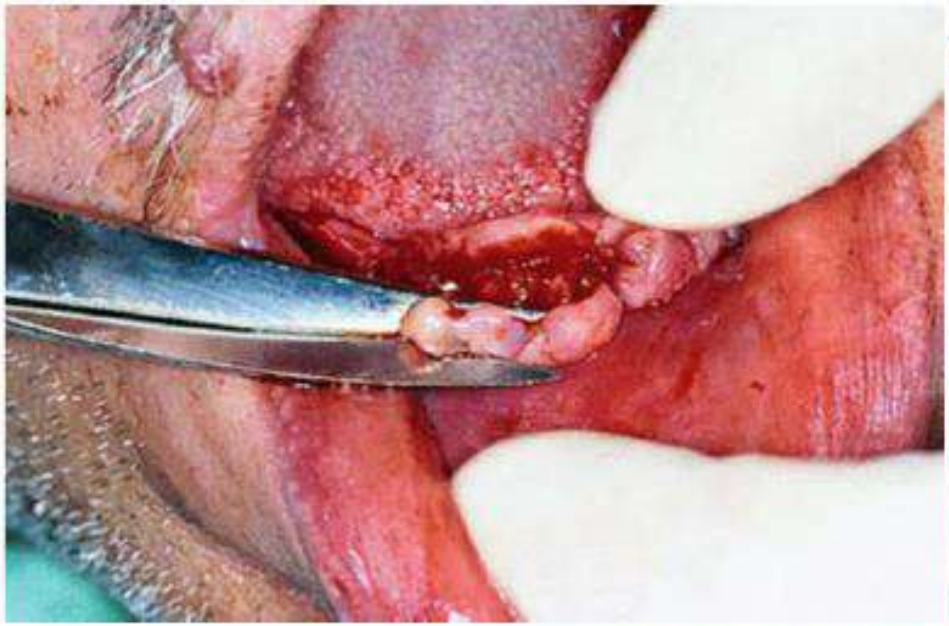


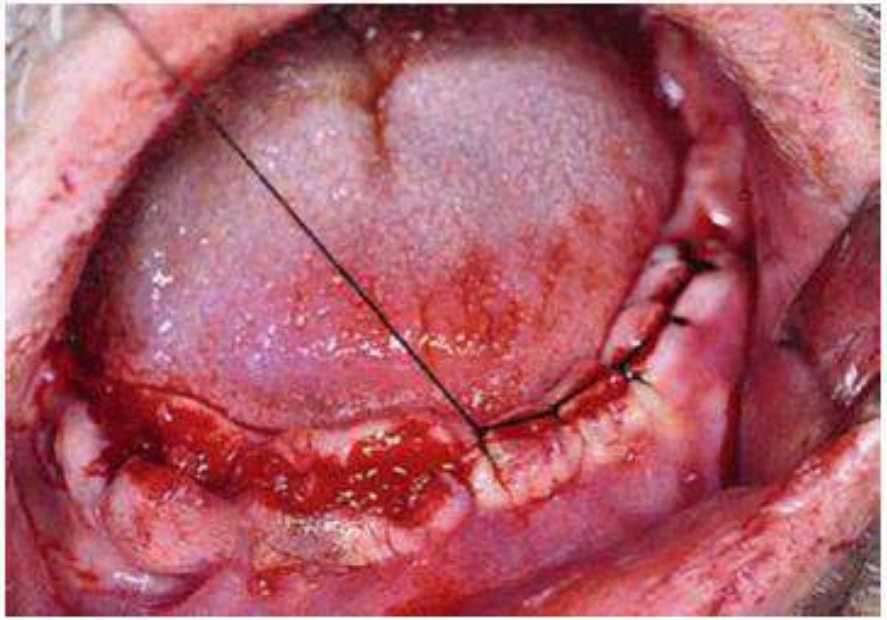


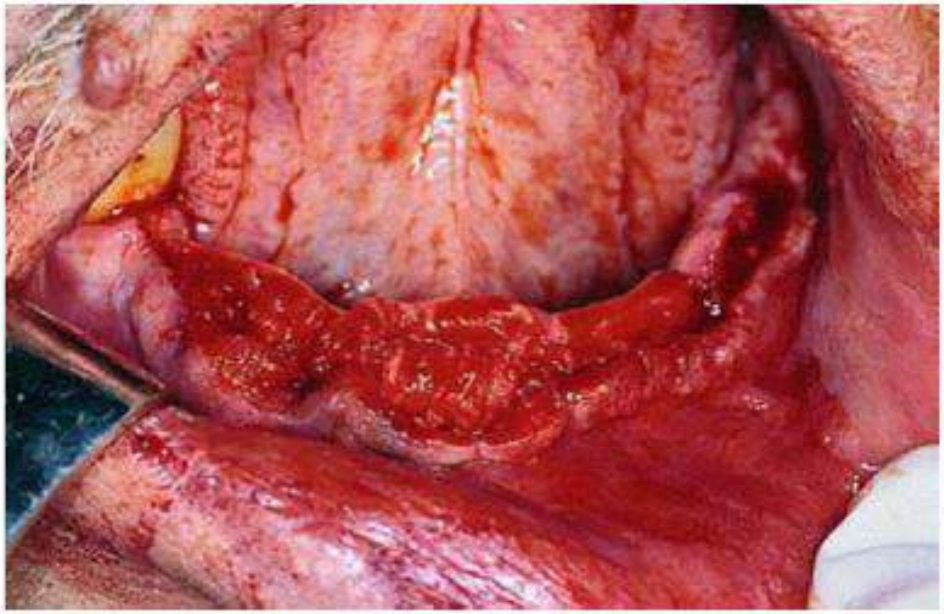


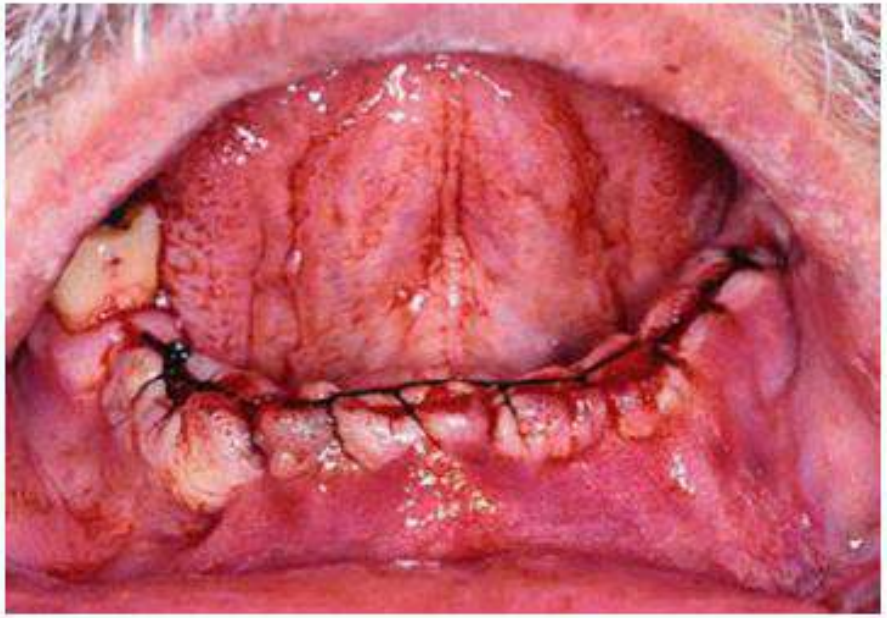


Simple Alveoloplasty



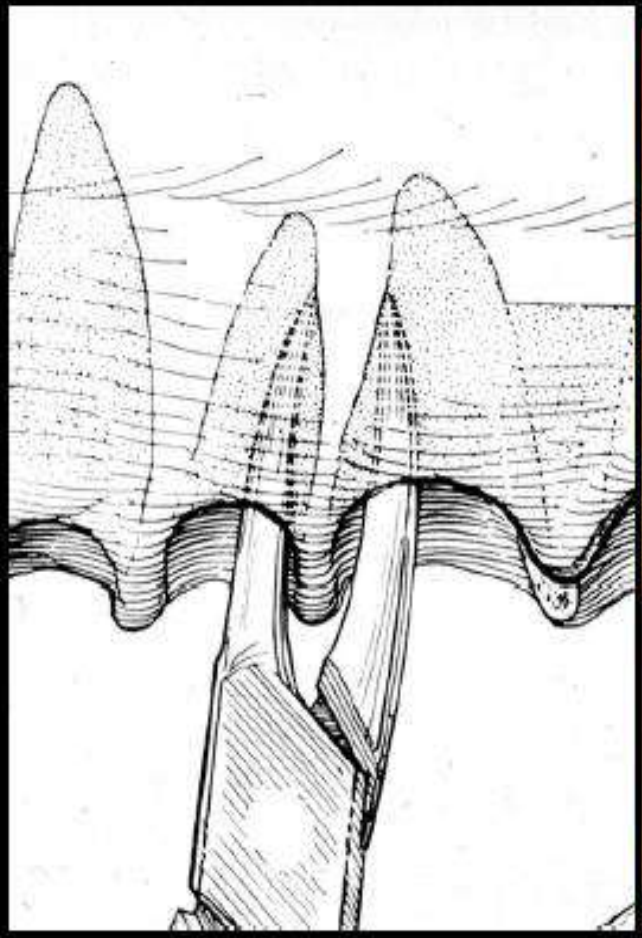




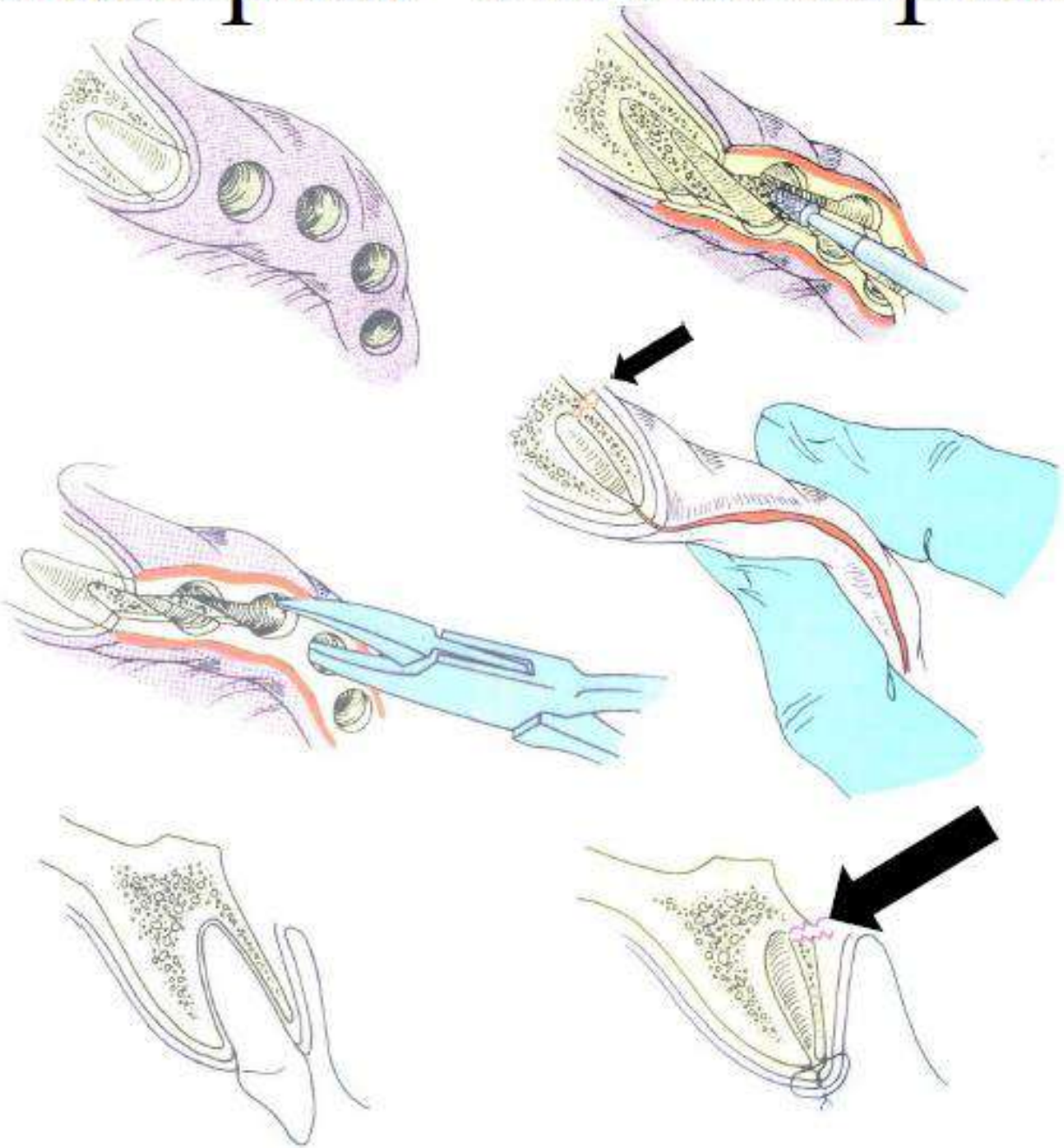


Intraseptal Alveoplasty

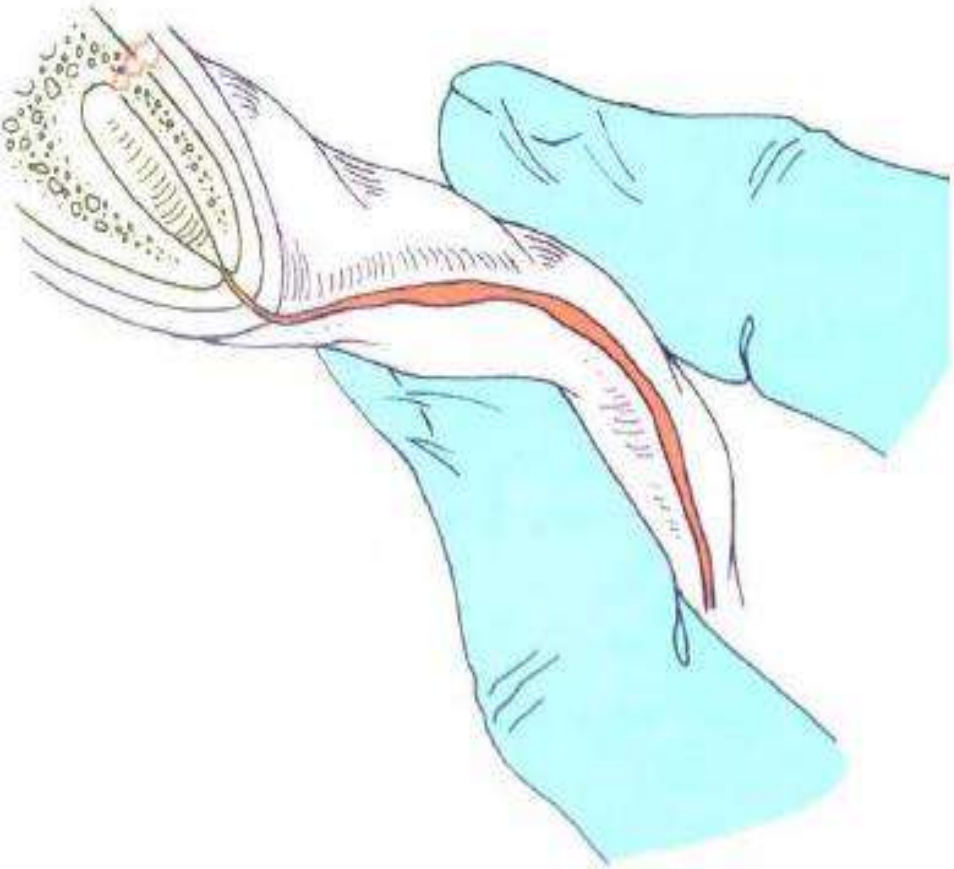
Removal of Intraseptal Bone

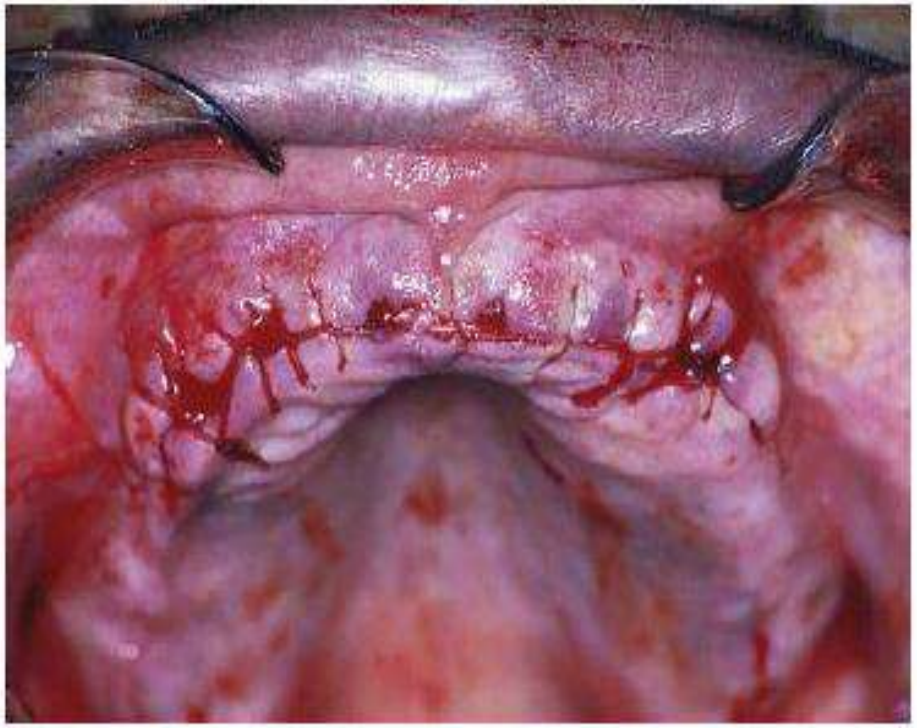


Intraseptal Alveoloplasty

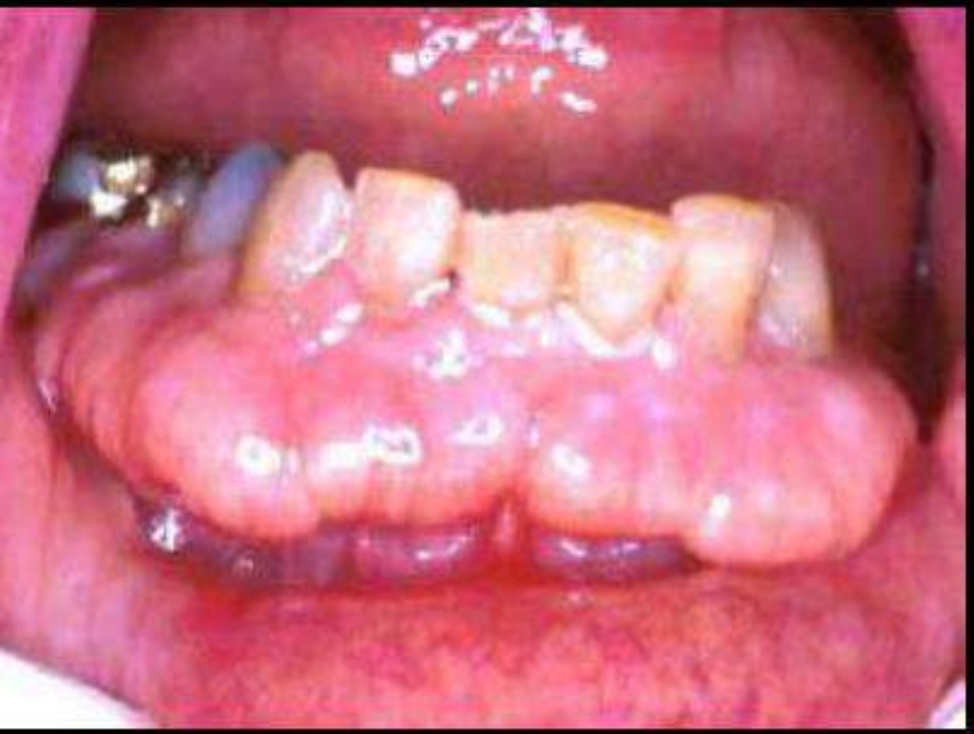


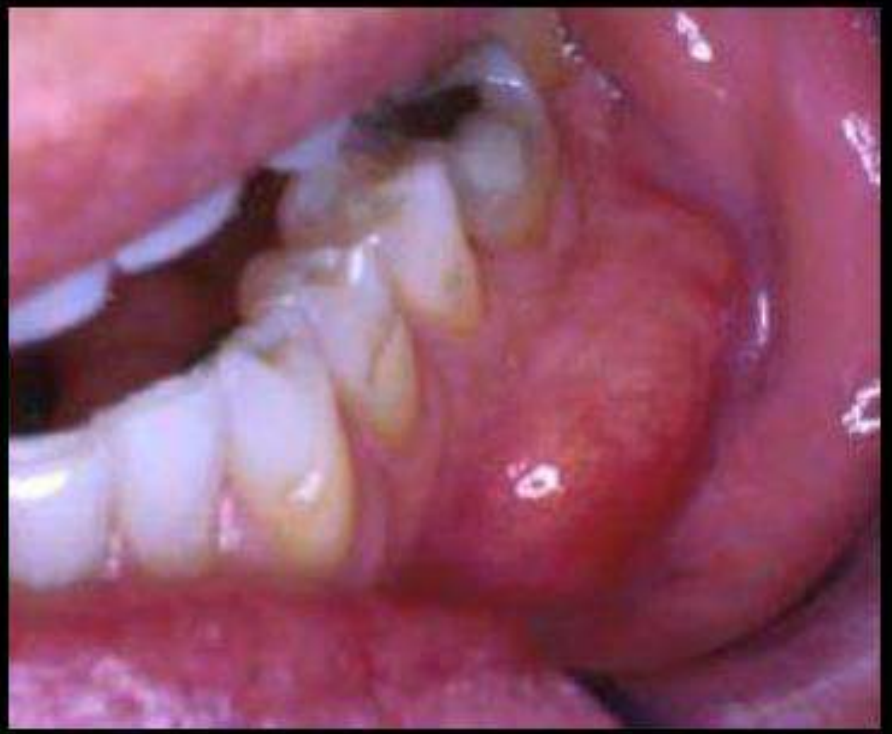
Intraseptal Alveoloplasty



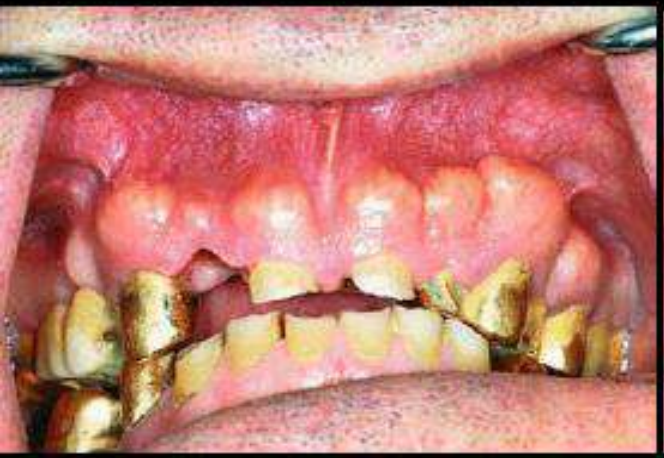
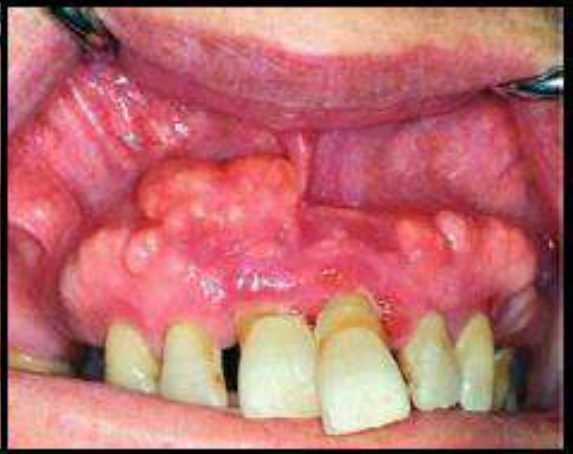
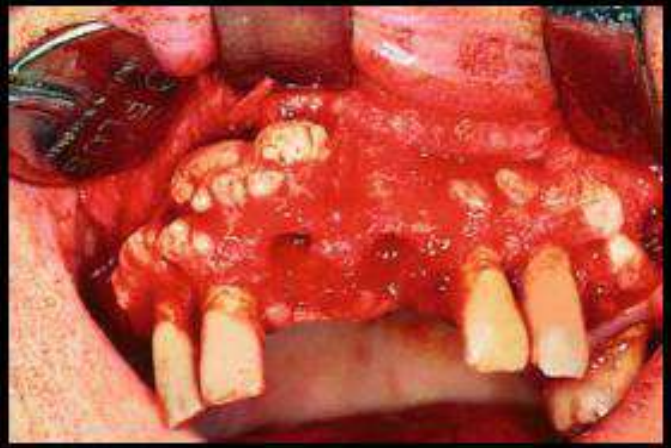
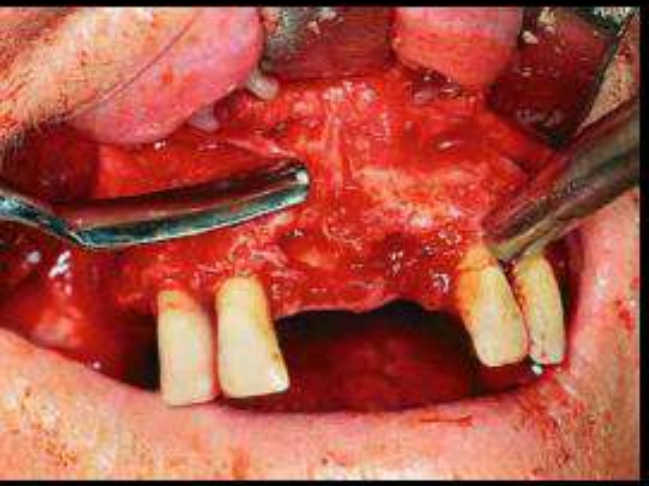
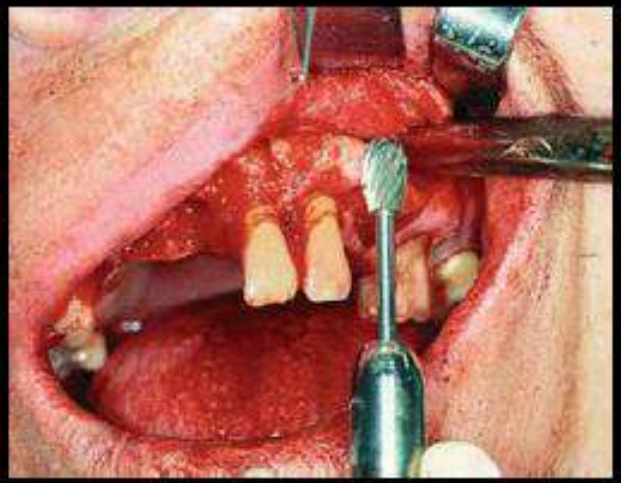
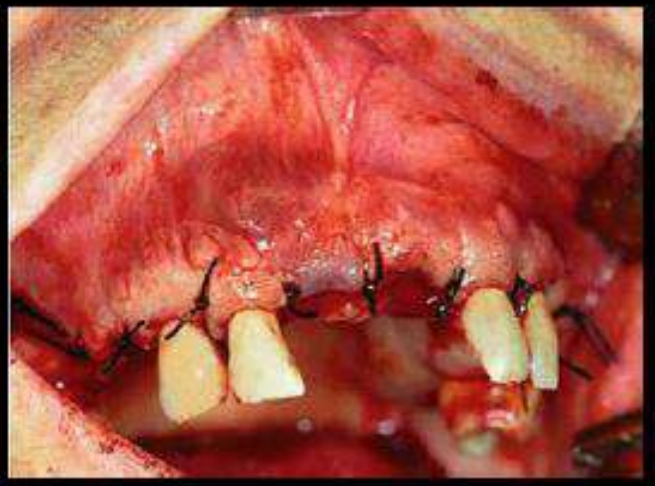


Buccal /Facial Exostosis
Excessive Undercuts

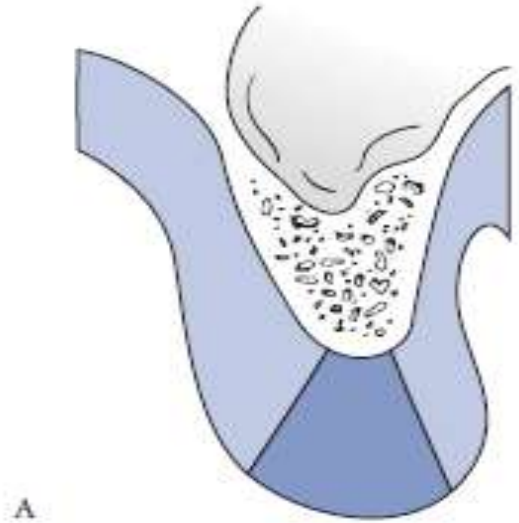


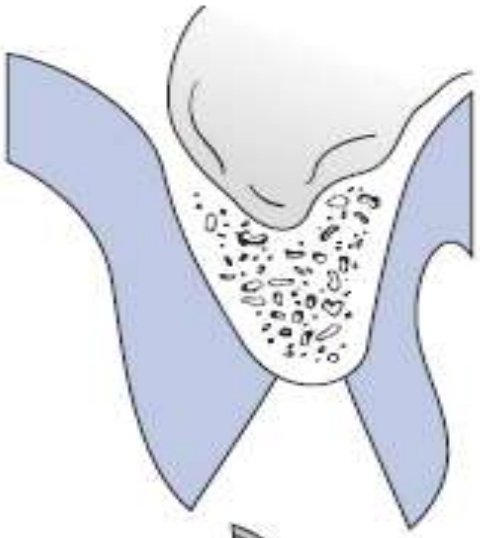


Buccal /Facial Exostosis
Excessive Undercuts

TUBEROSITY REDUCTION





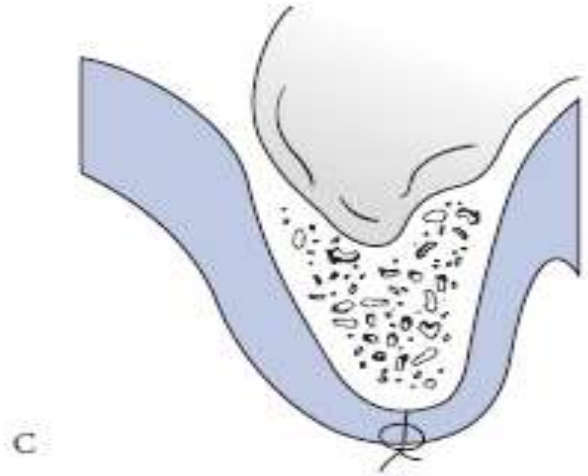
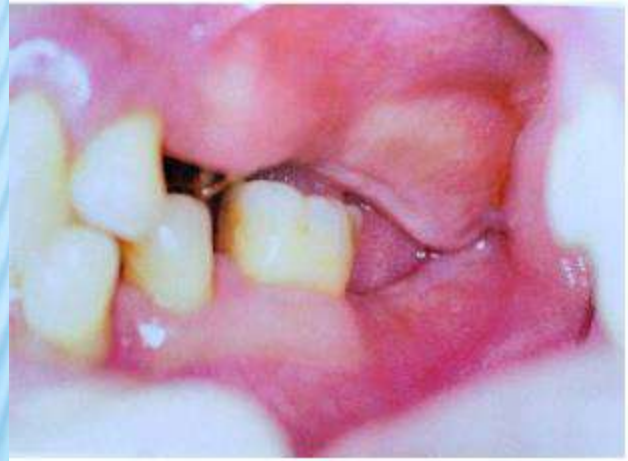
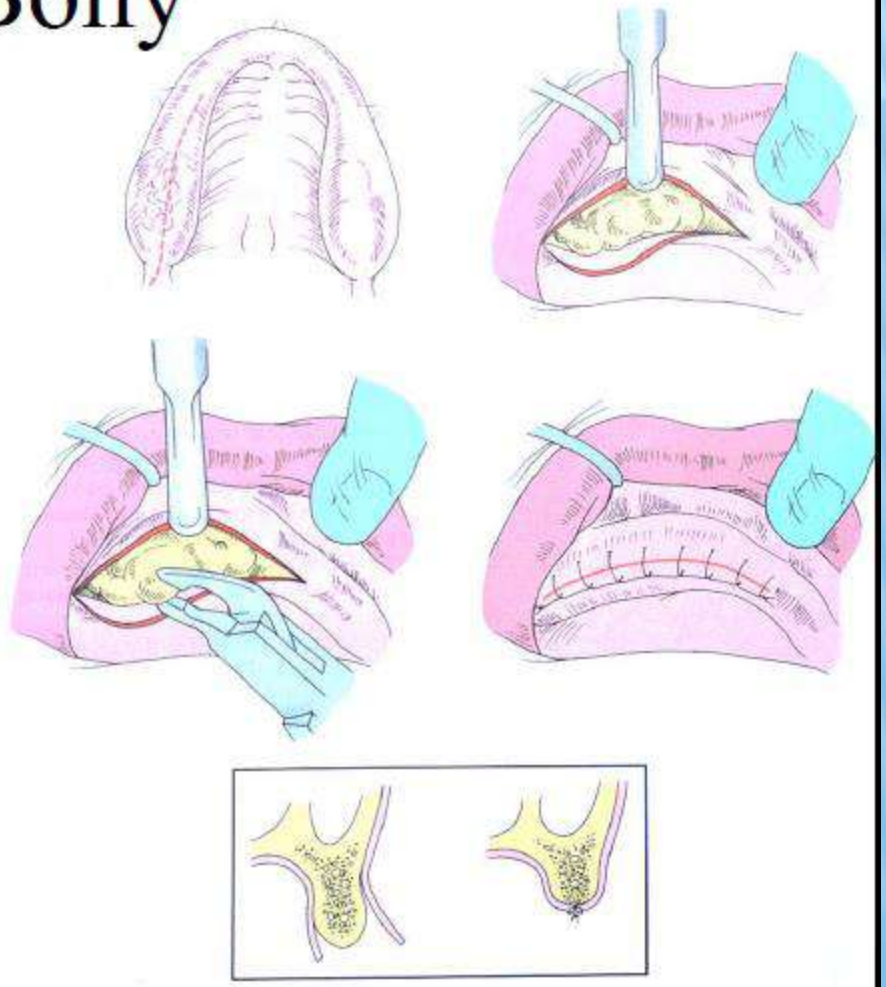
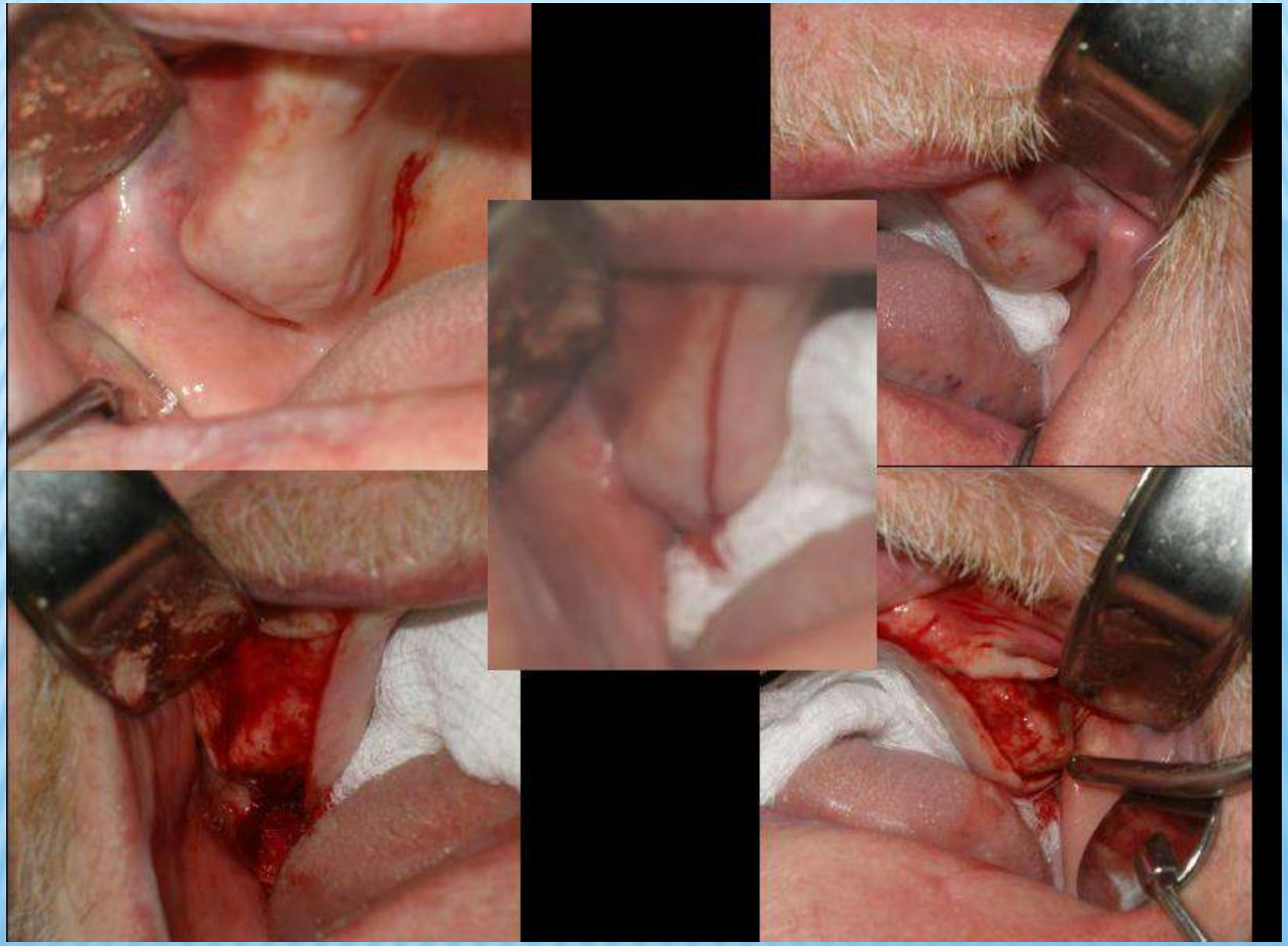


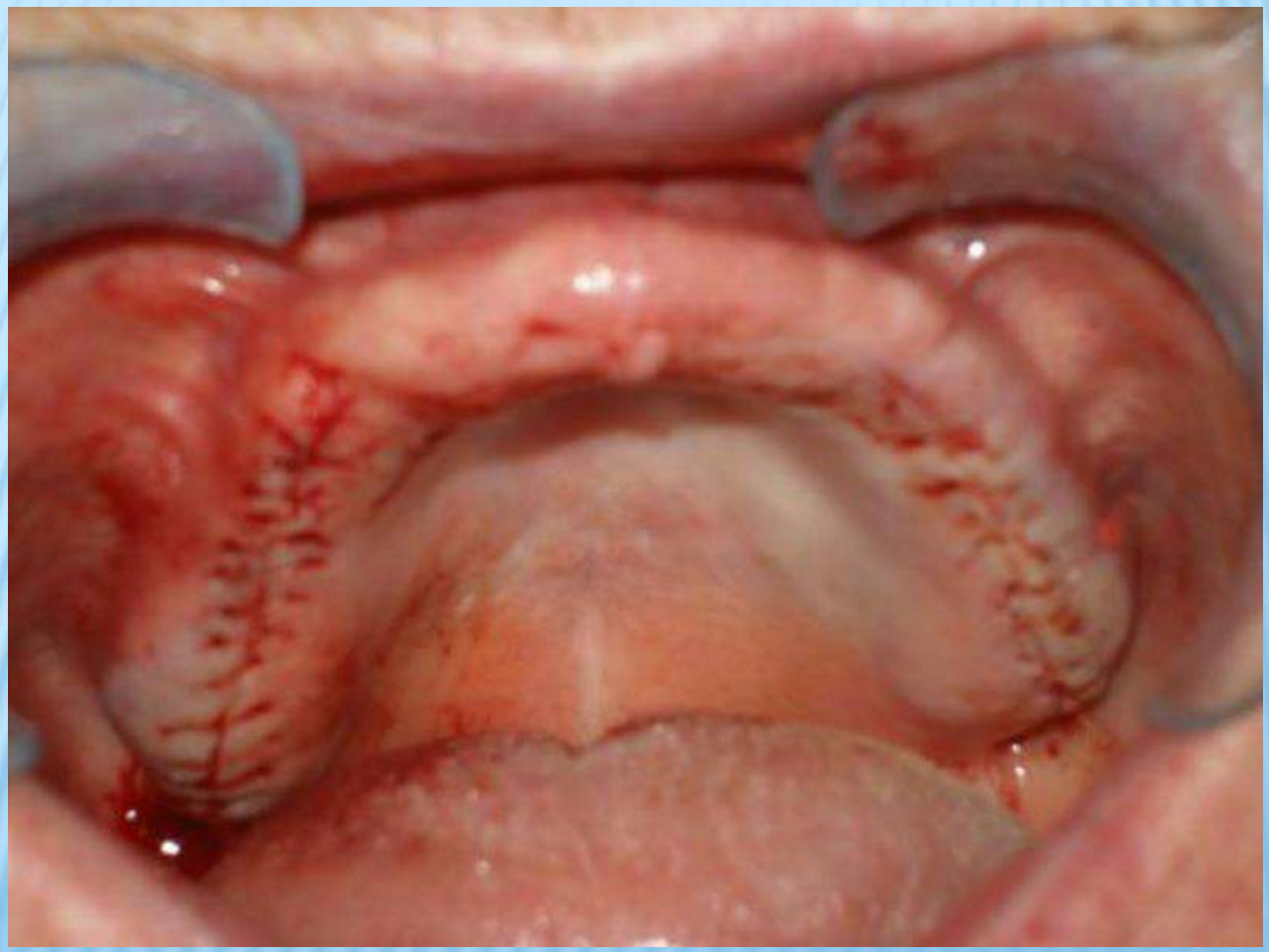
FIGURE 9-12 A, *Area of soft tissue to be excised in an elliptic fashion over the tuberosity.* B, *Removal of tissue and undermining of buccal and palatal flaps completed.* C, *Final tissue closure.*

Maxillary Tuberosity Reduction Bony

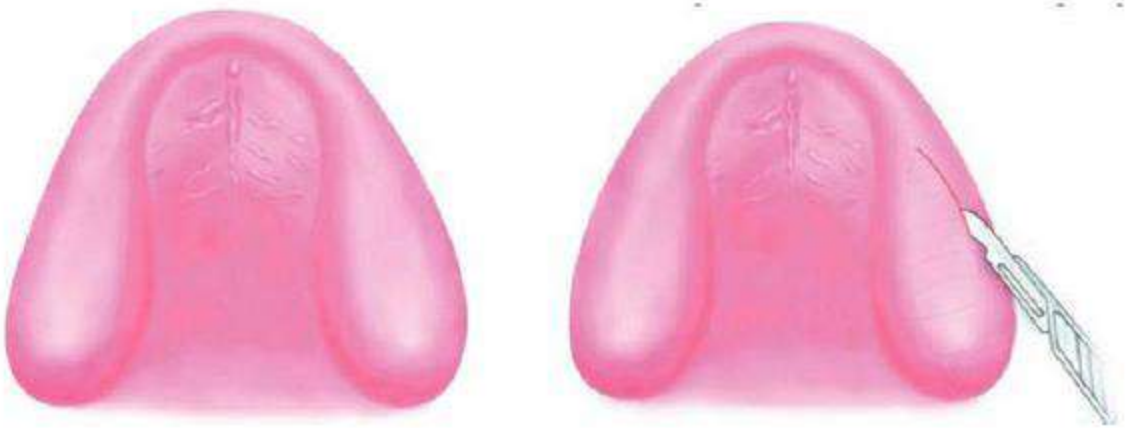


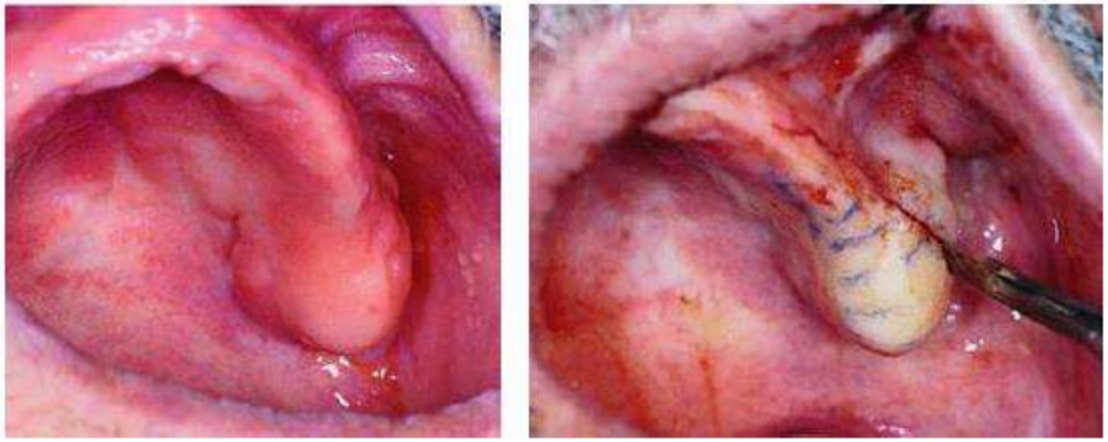




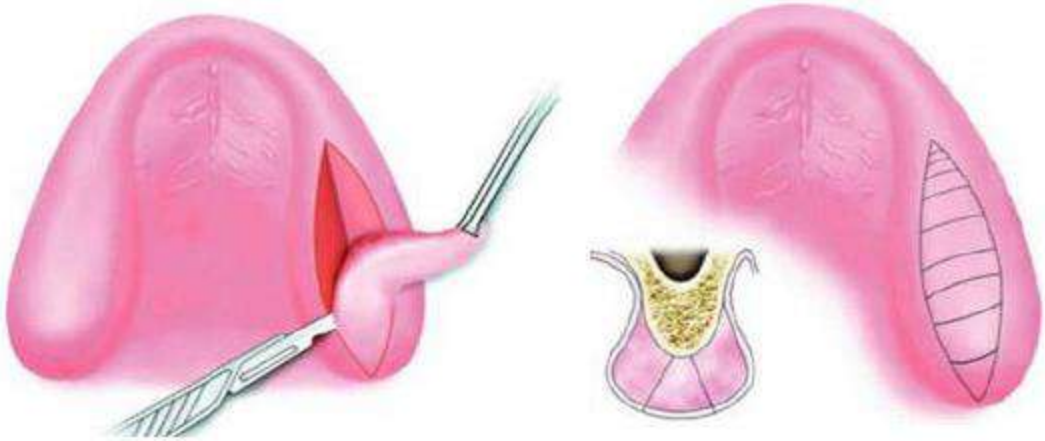


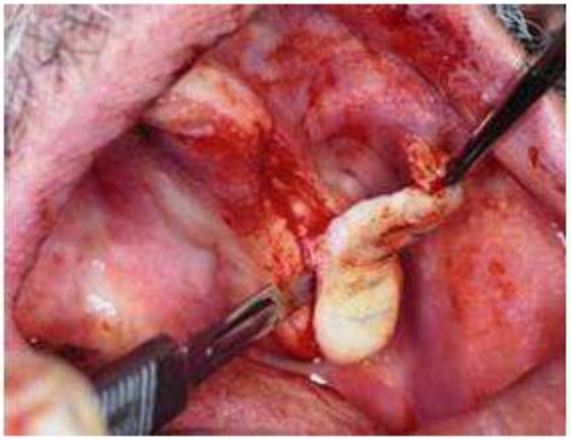
Maxillary Tuberosity Reduction Soft Tissue

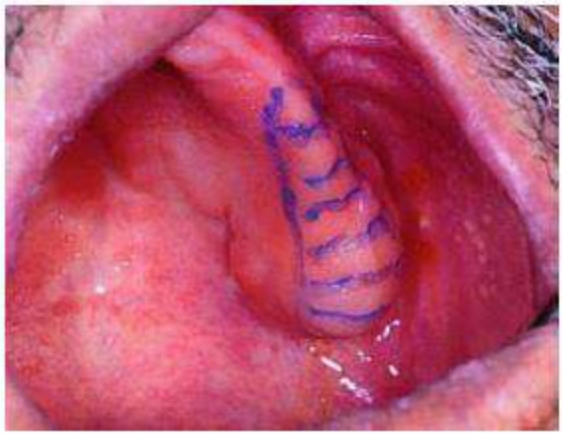




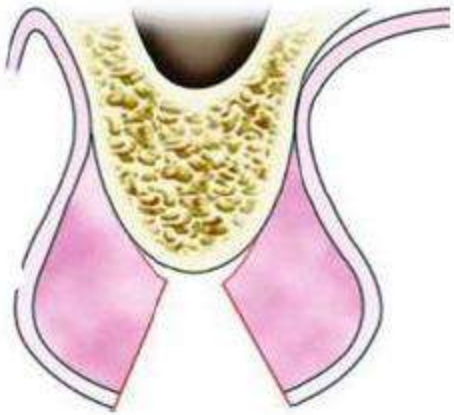
Maxillary Tuberosity Reduction Soft Tissue

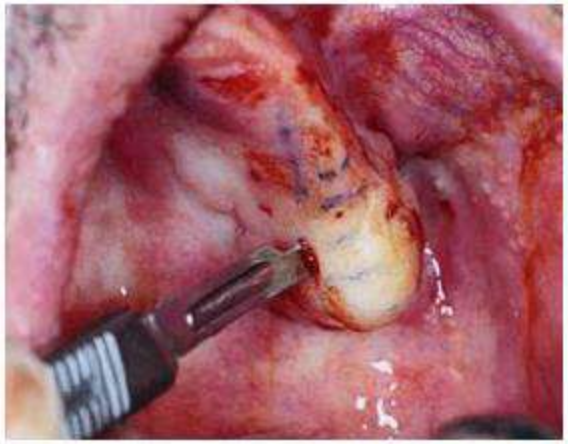


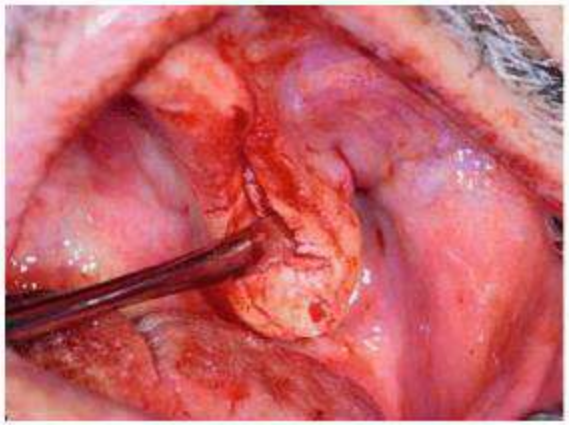




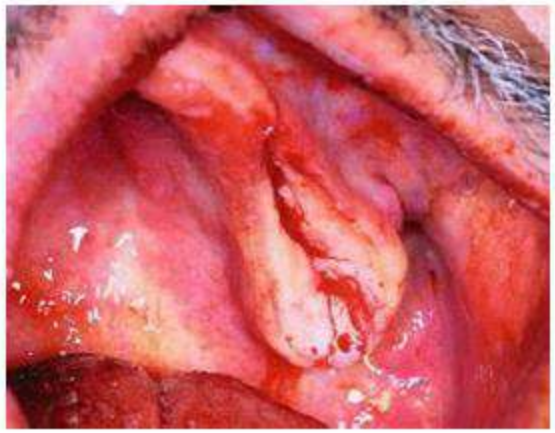
Maxillary Tuberosity Reduction Soft Tissue

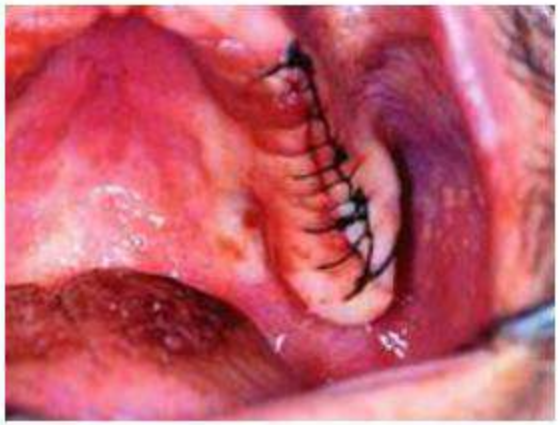




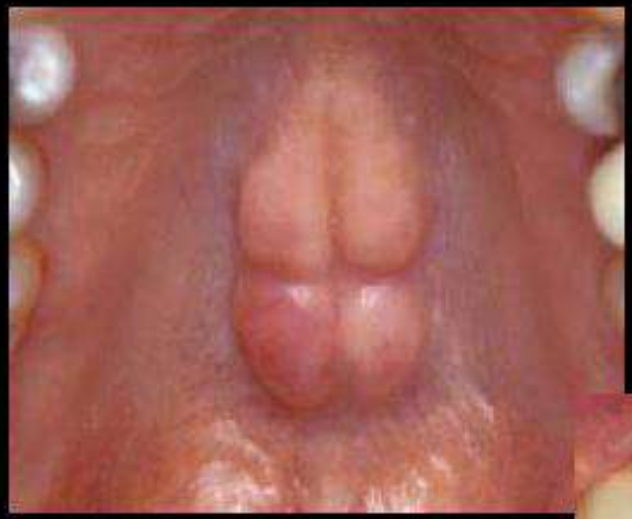


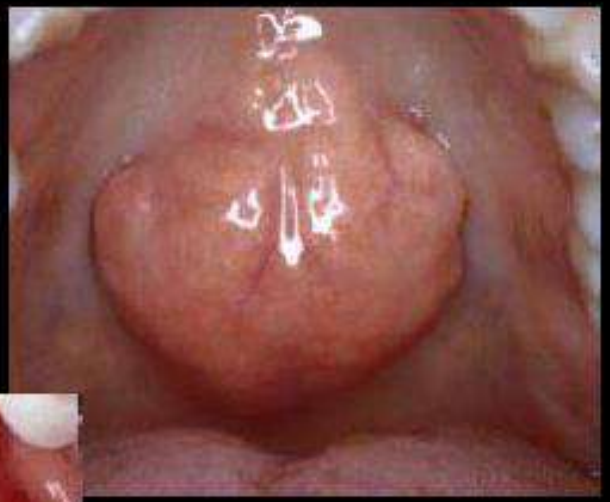
Maxillary Tuberosity Reduction Soft Tissue

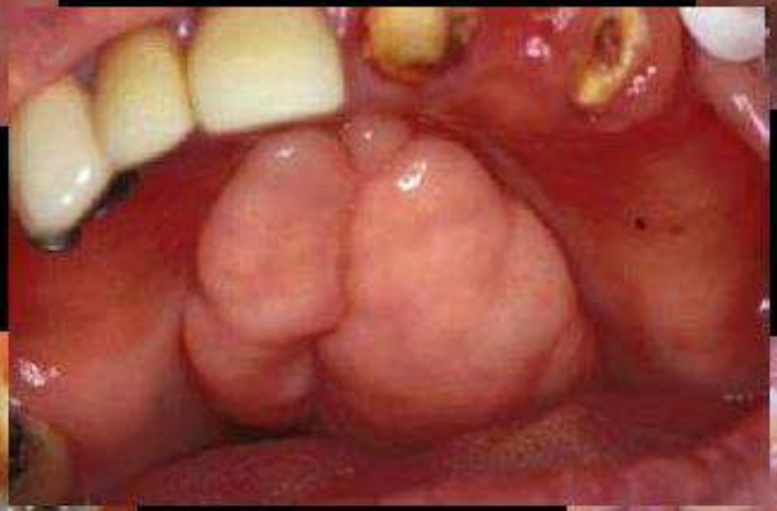


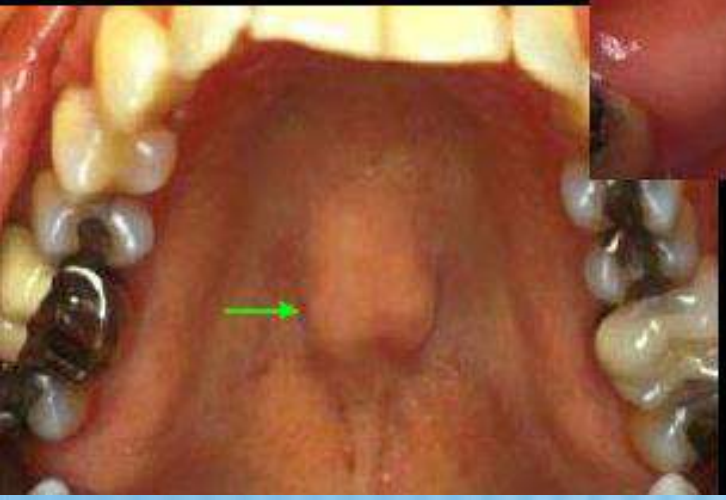


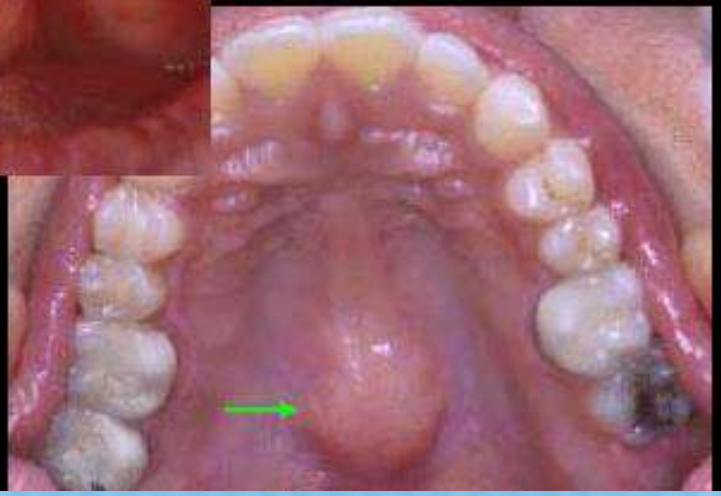
Maxillary Palatal Tori Reduction



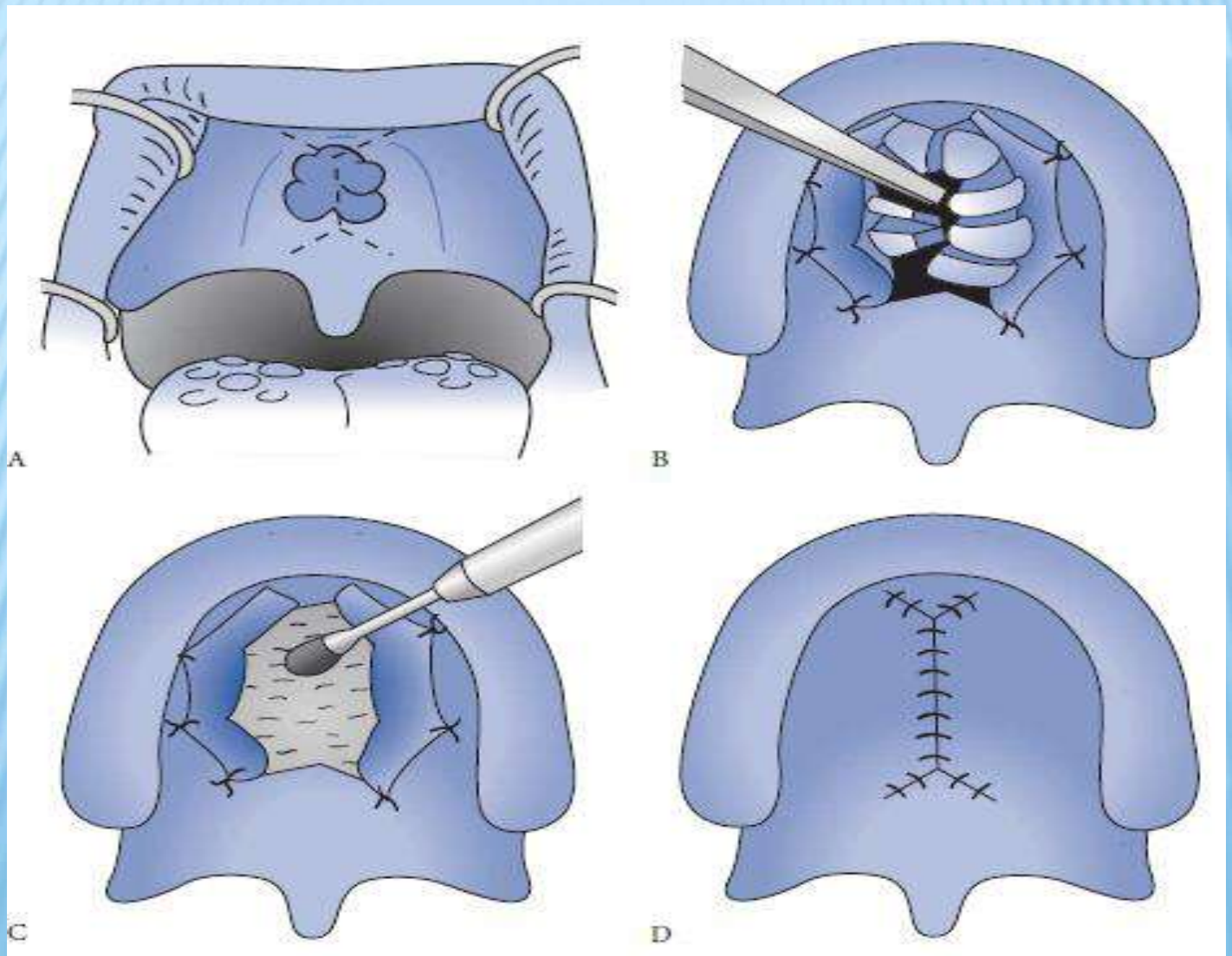




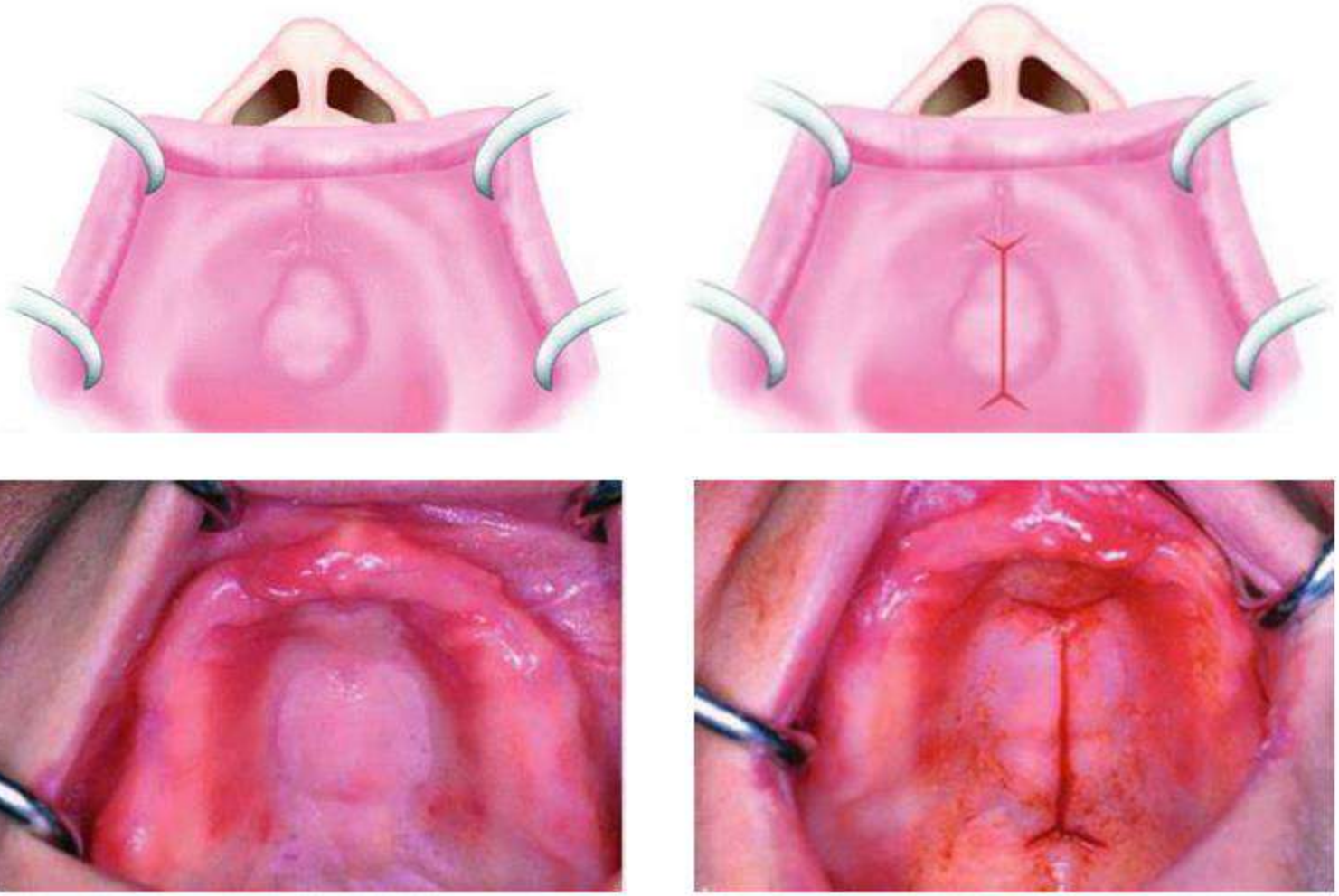




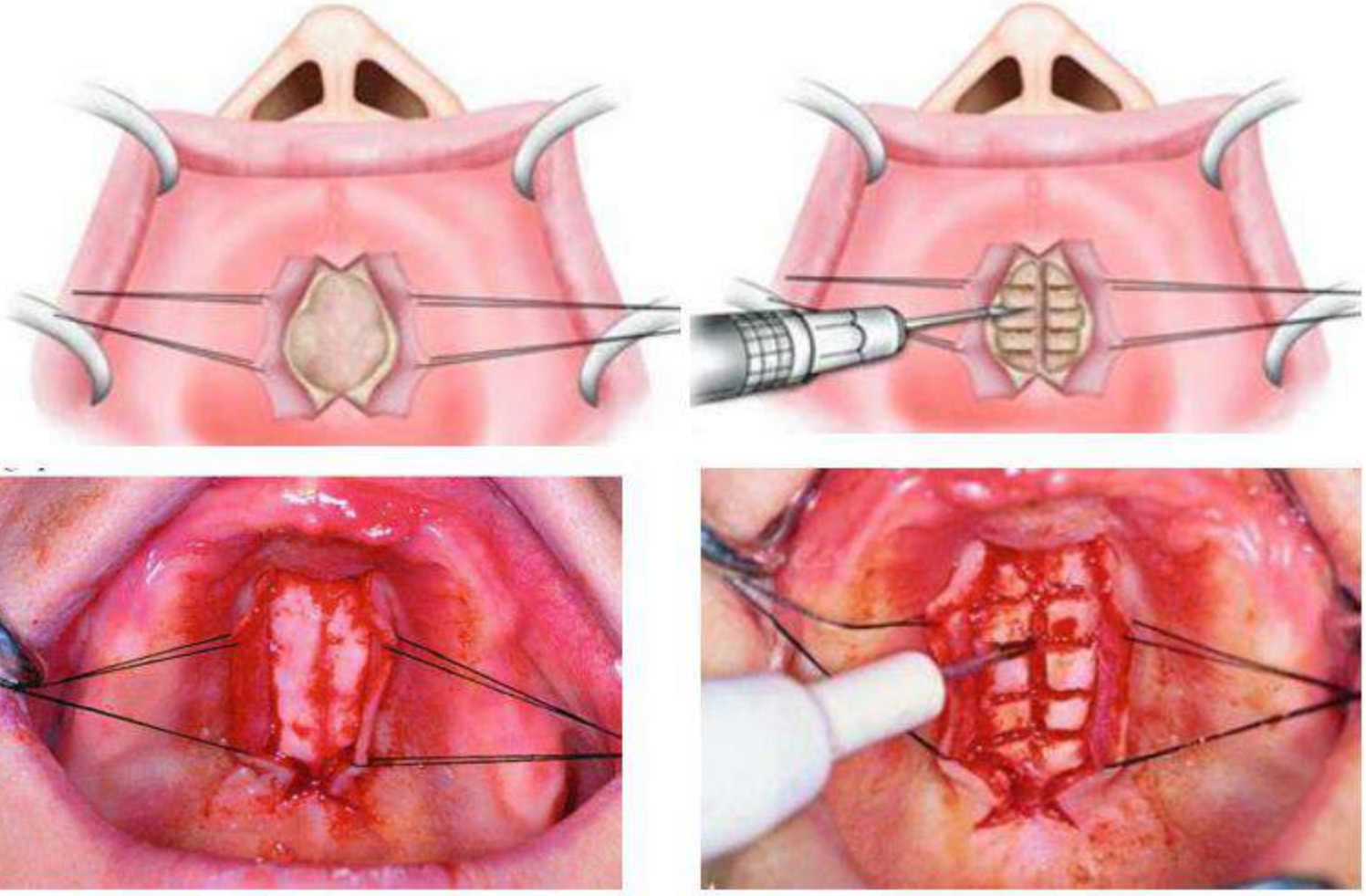
MAXILLARY TORUS



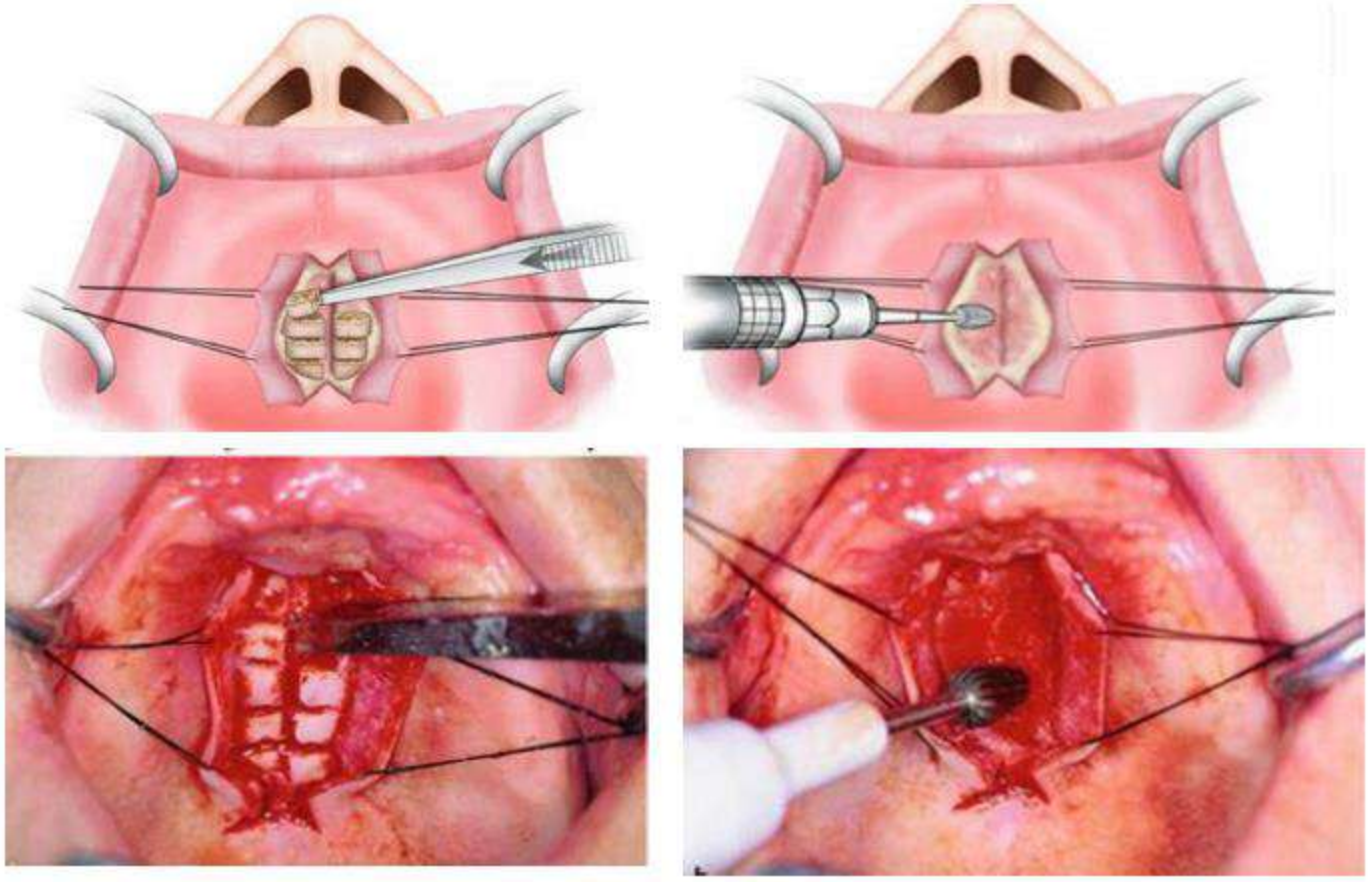
Maxillary Tori Reduction



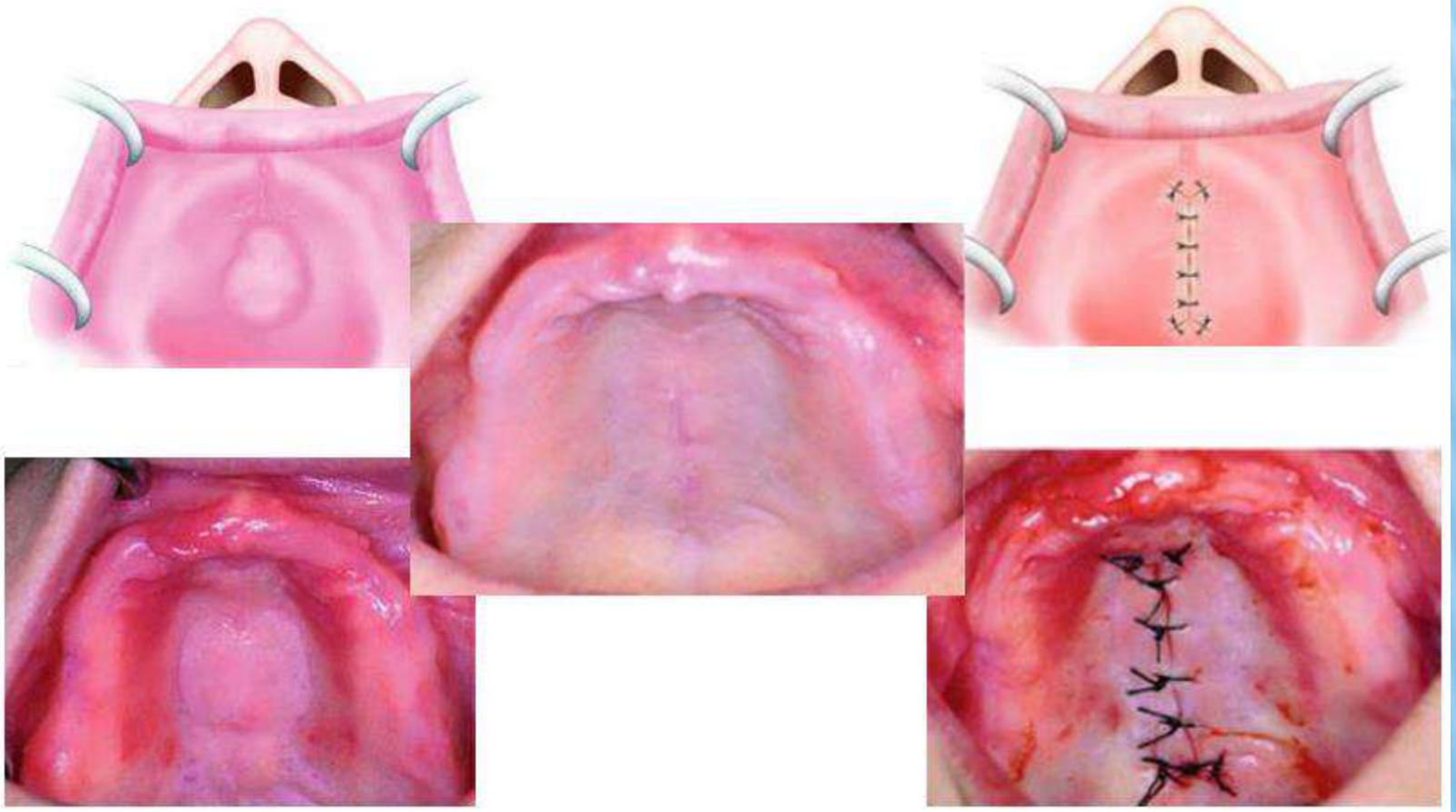
Maxillary Tori Reduction



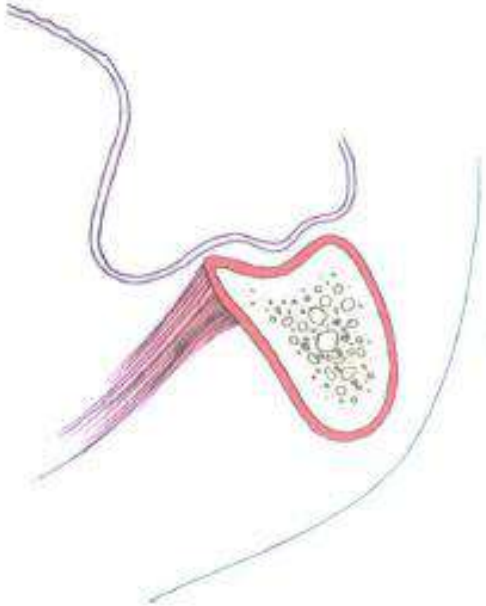
Maxillary Tori Reduction

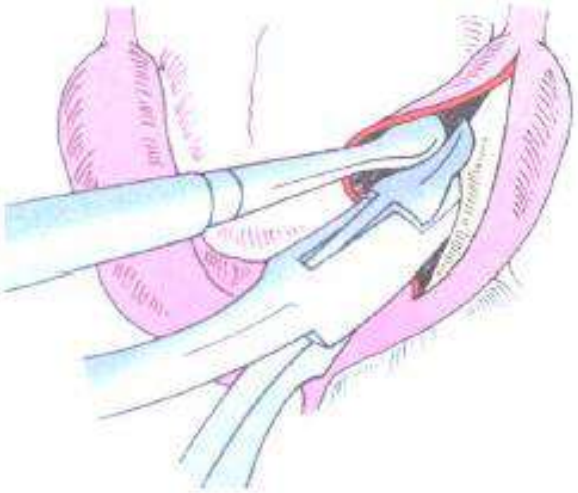


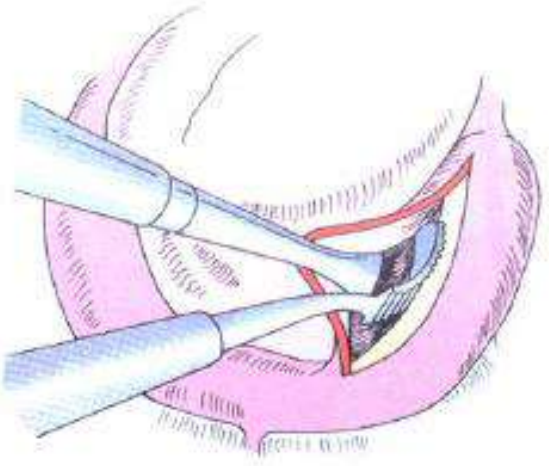
Maxillary Tori Reduction



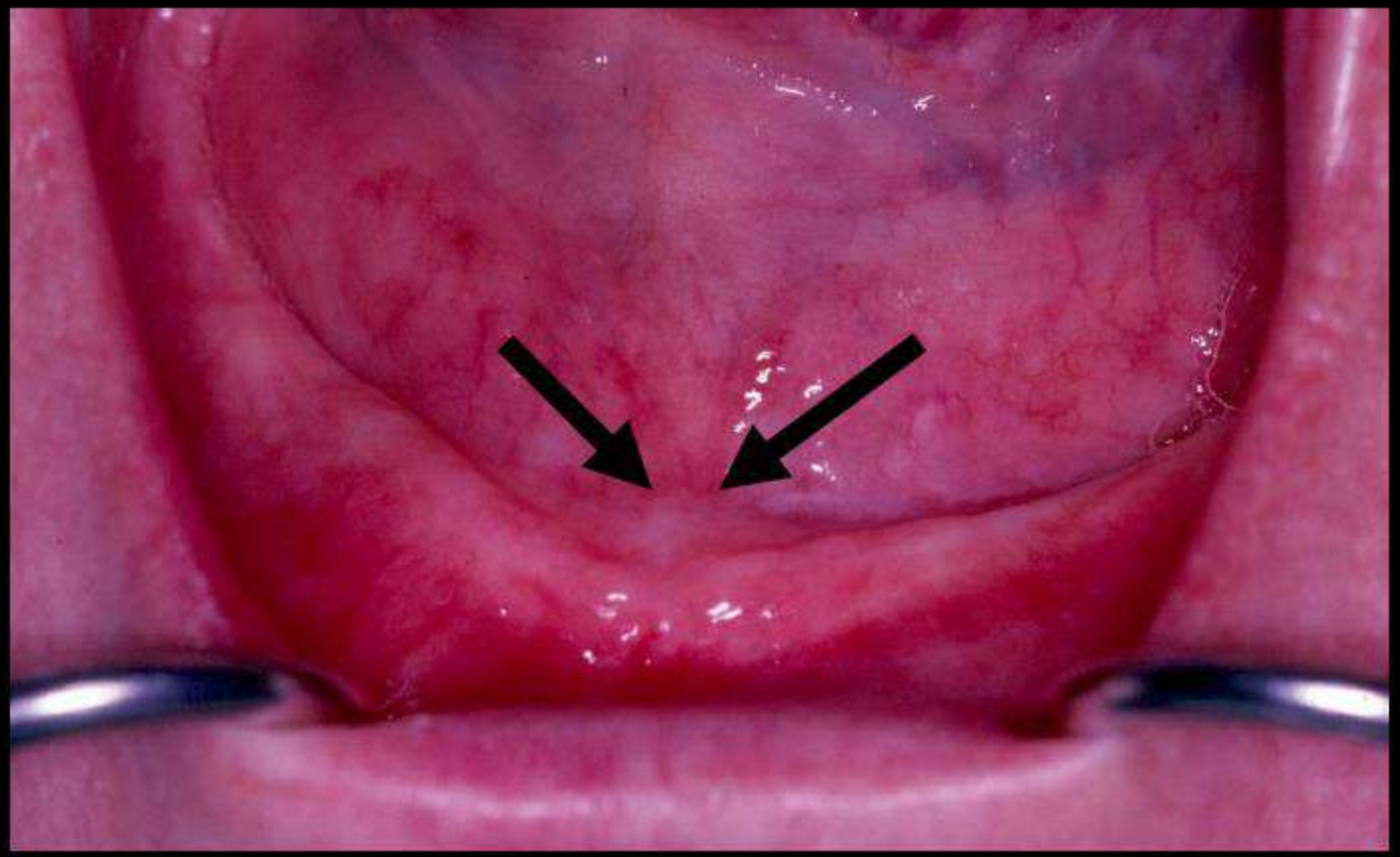
Mylohyoid Ridge Reduction







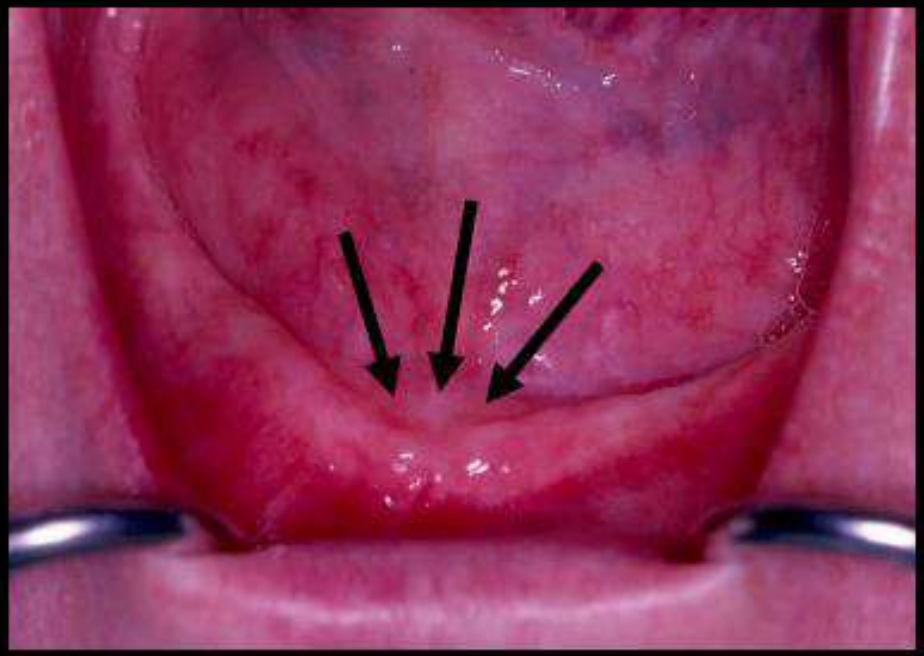
Genial Tubercle Reduction



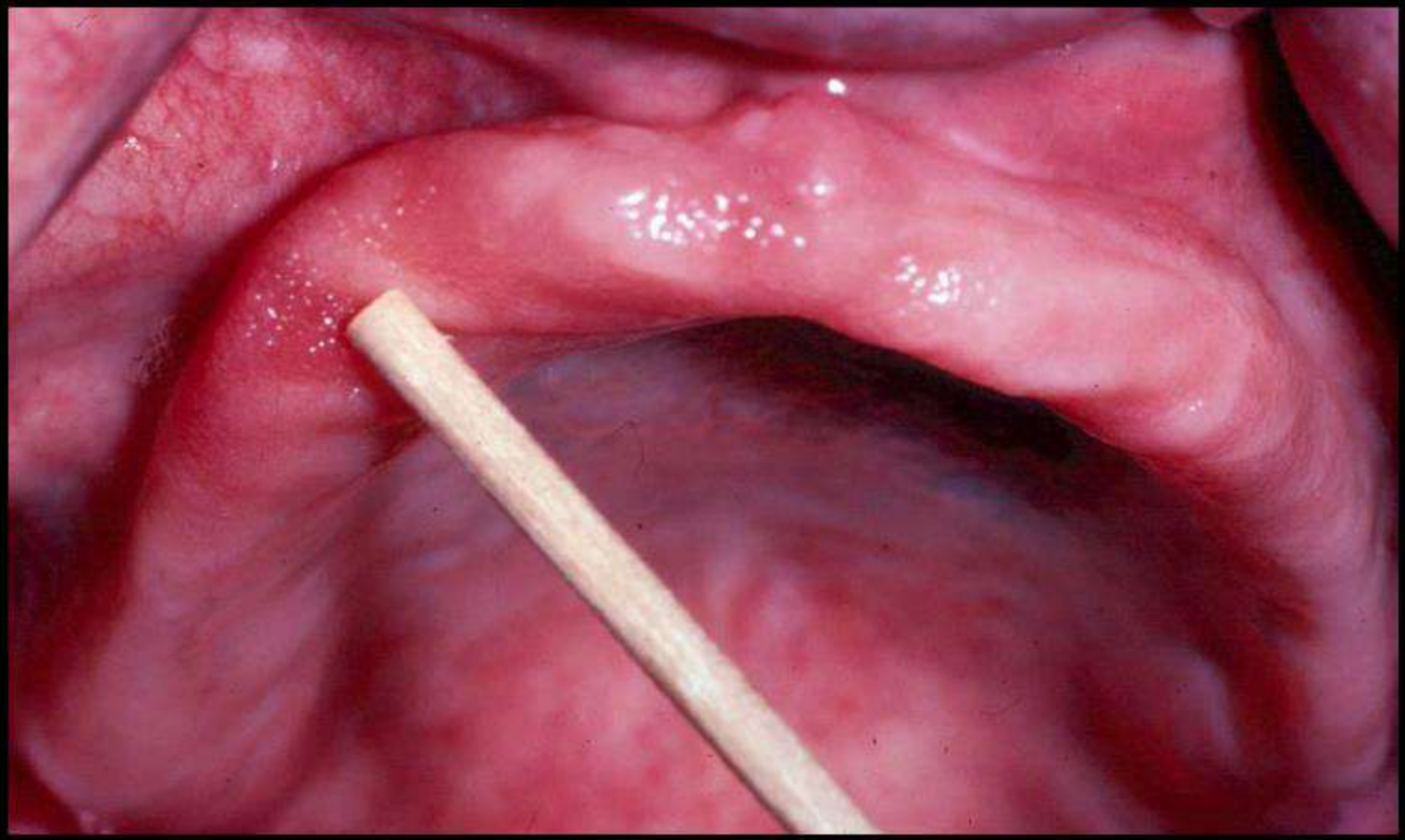
Genial Tubercle Reduction

- Resorption of the mandibular alveolus
- Continued loading of the tubercle by the genioglossus muscle

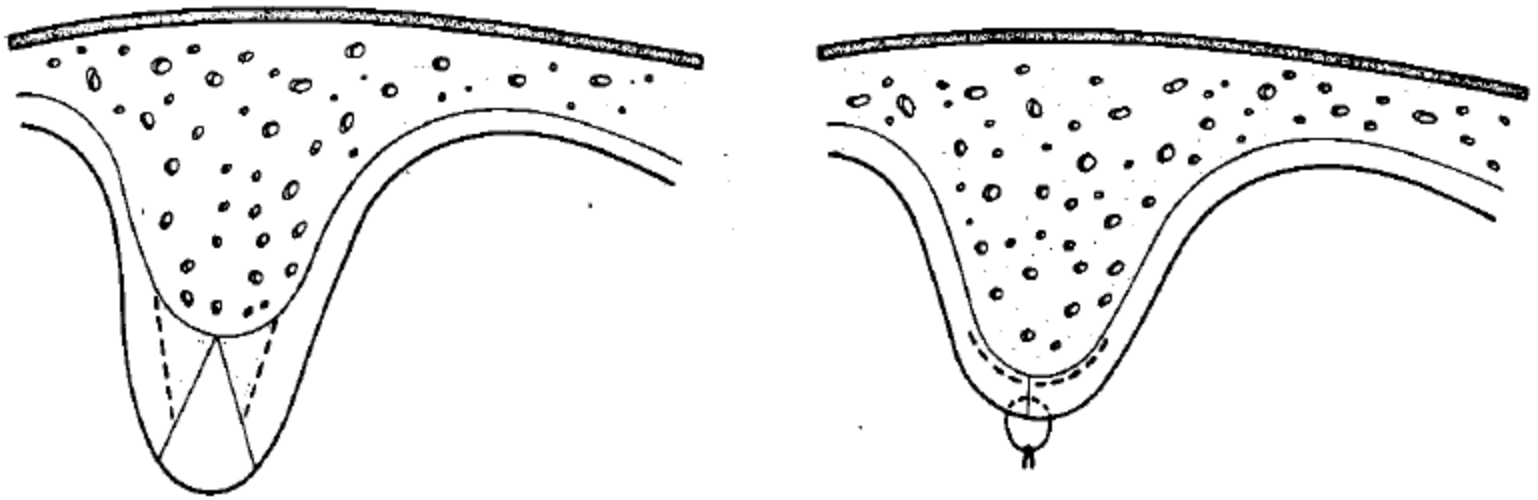
  Genial tubercle becomes increasingly prominent
- Interferes with denture fabrication / seating
- Remove tubercle vs. augmentation



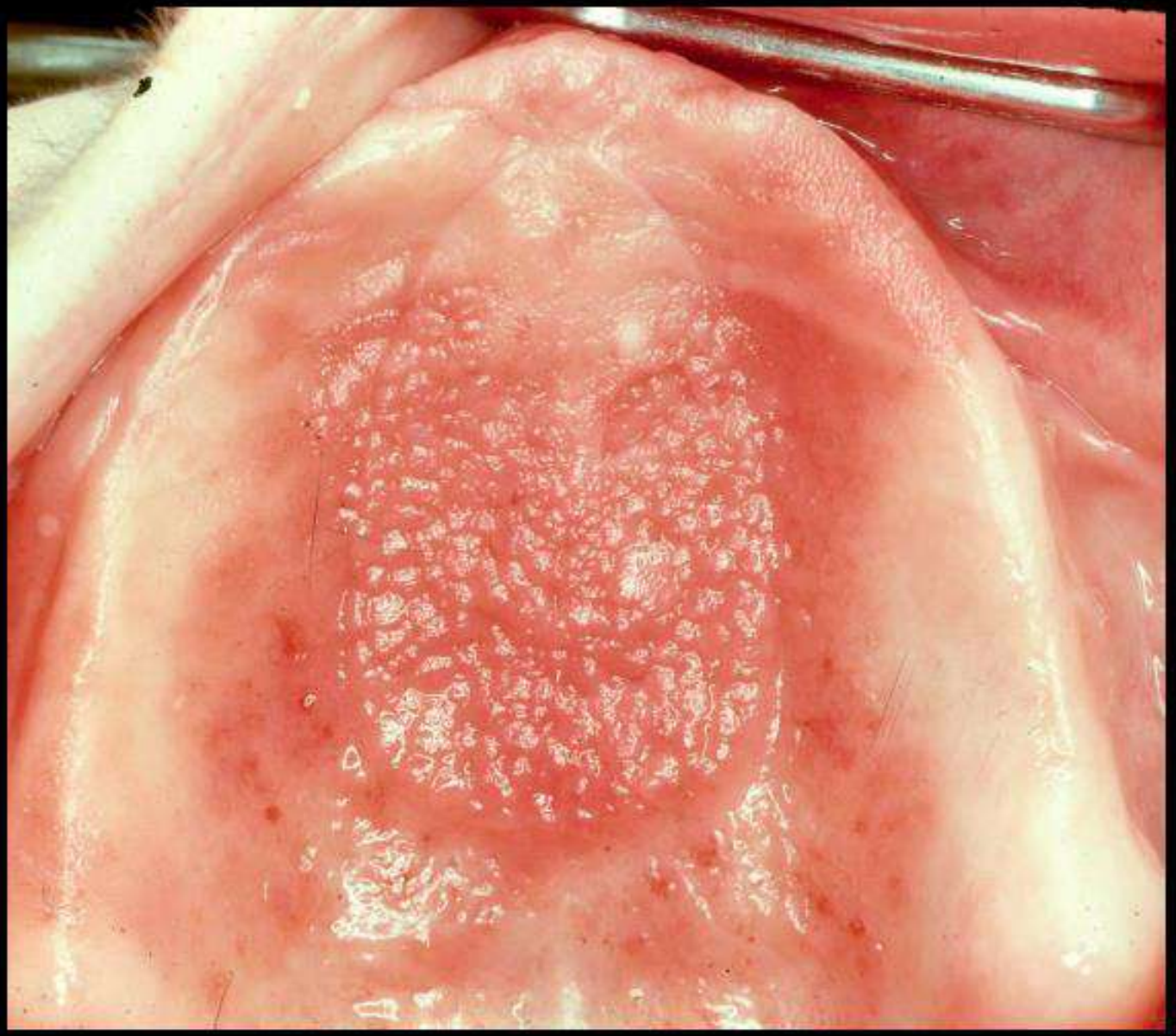
Reduction of Hypermobile Maxillary Tissue



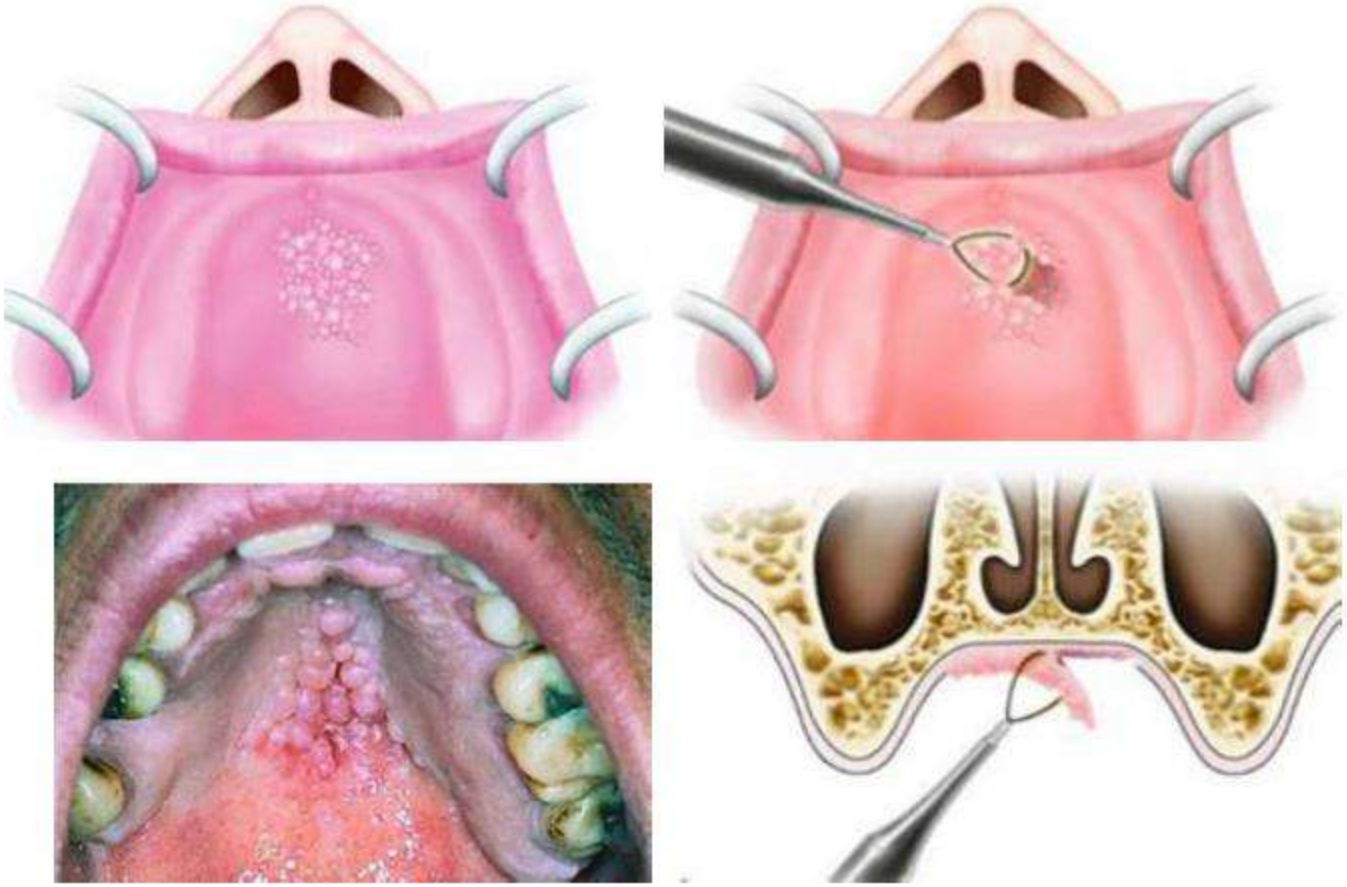
Reduction of Hypermobile Maxillary Tissue Wedge Excision



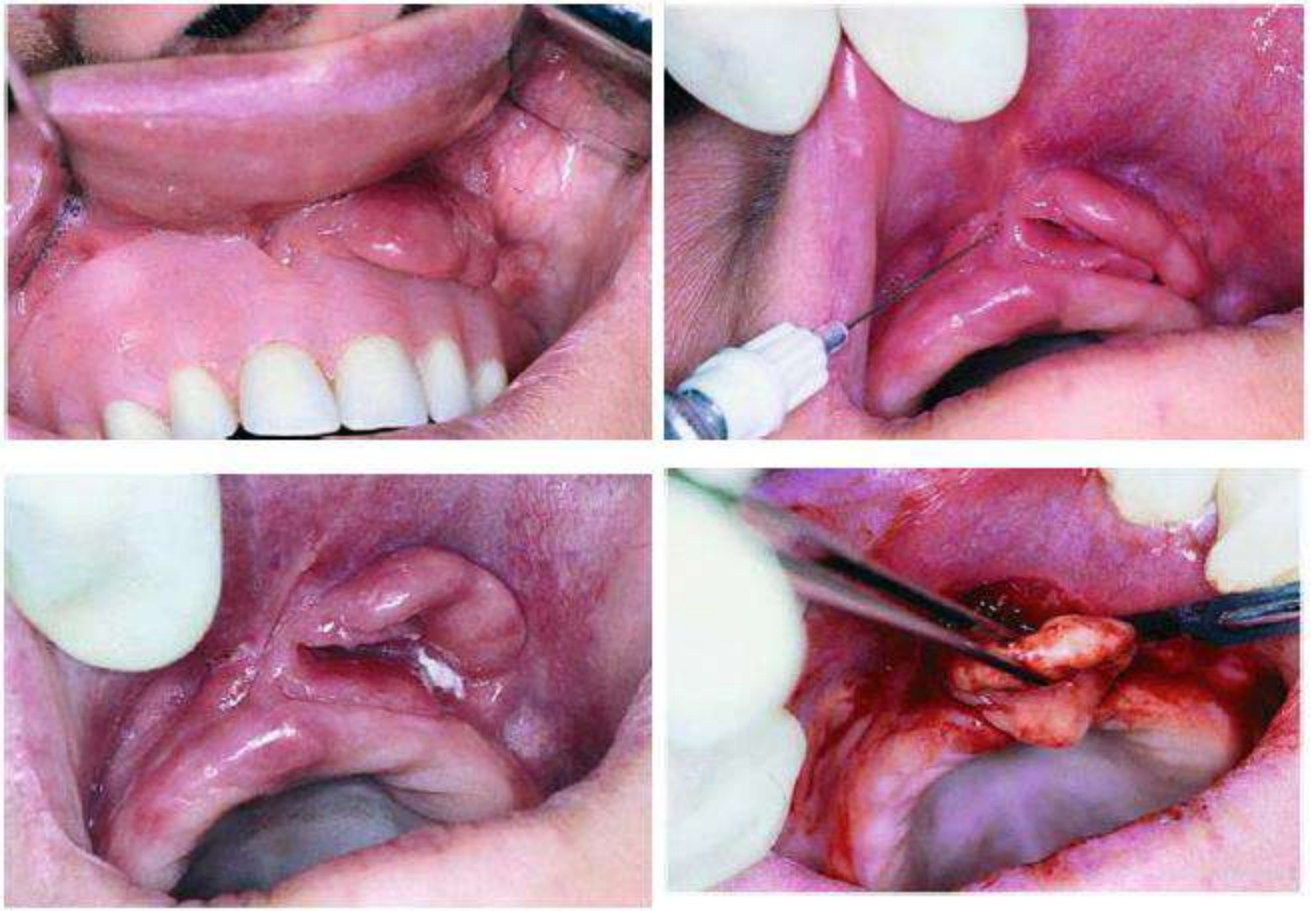
Palatal Papillary Hyperplasia



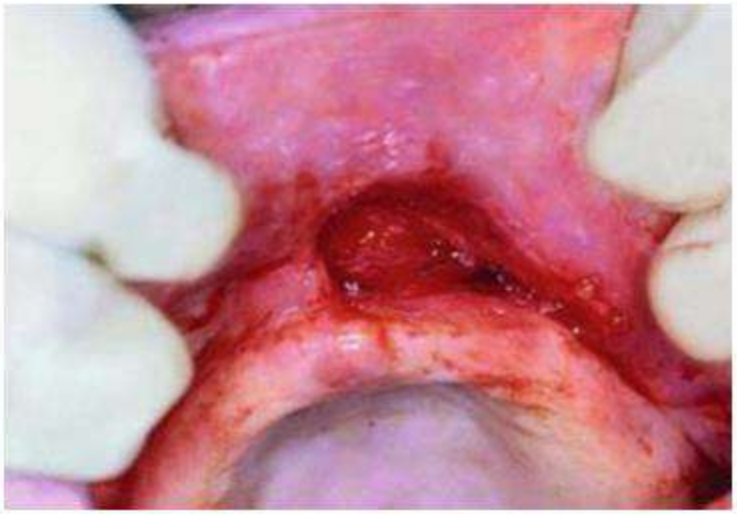
Palatal Papillary Hyperplasia

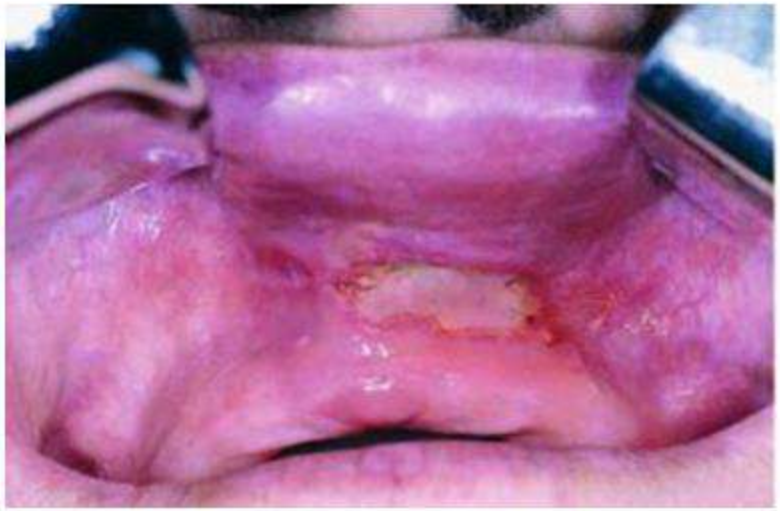


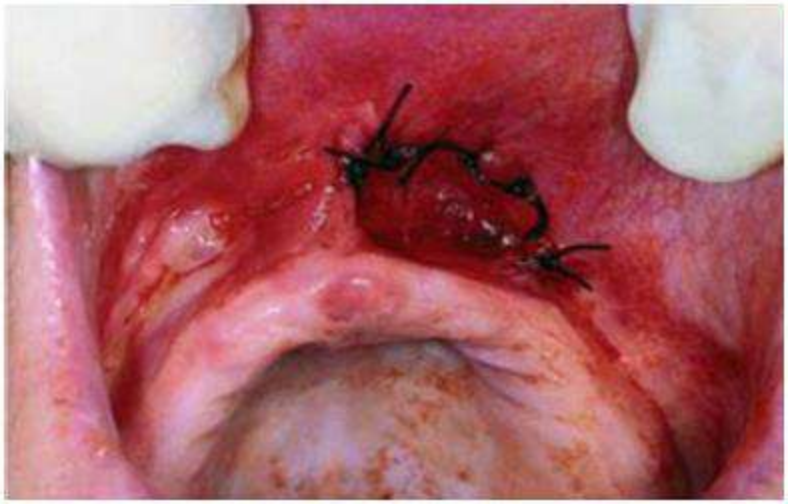
Epulis Fissuratum

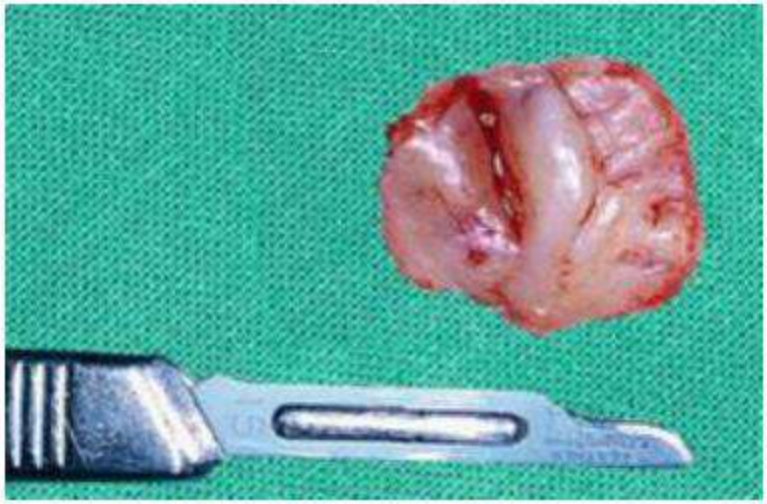


Epulis Fissuratum









Frenectomy

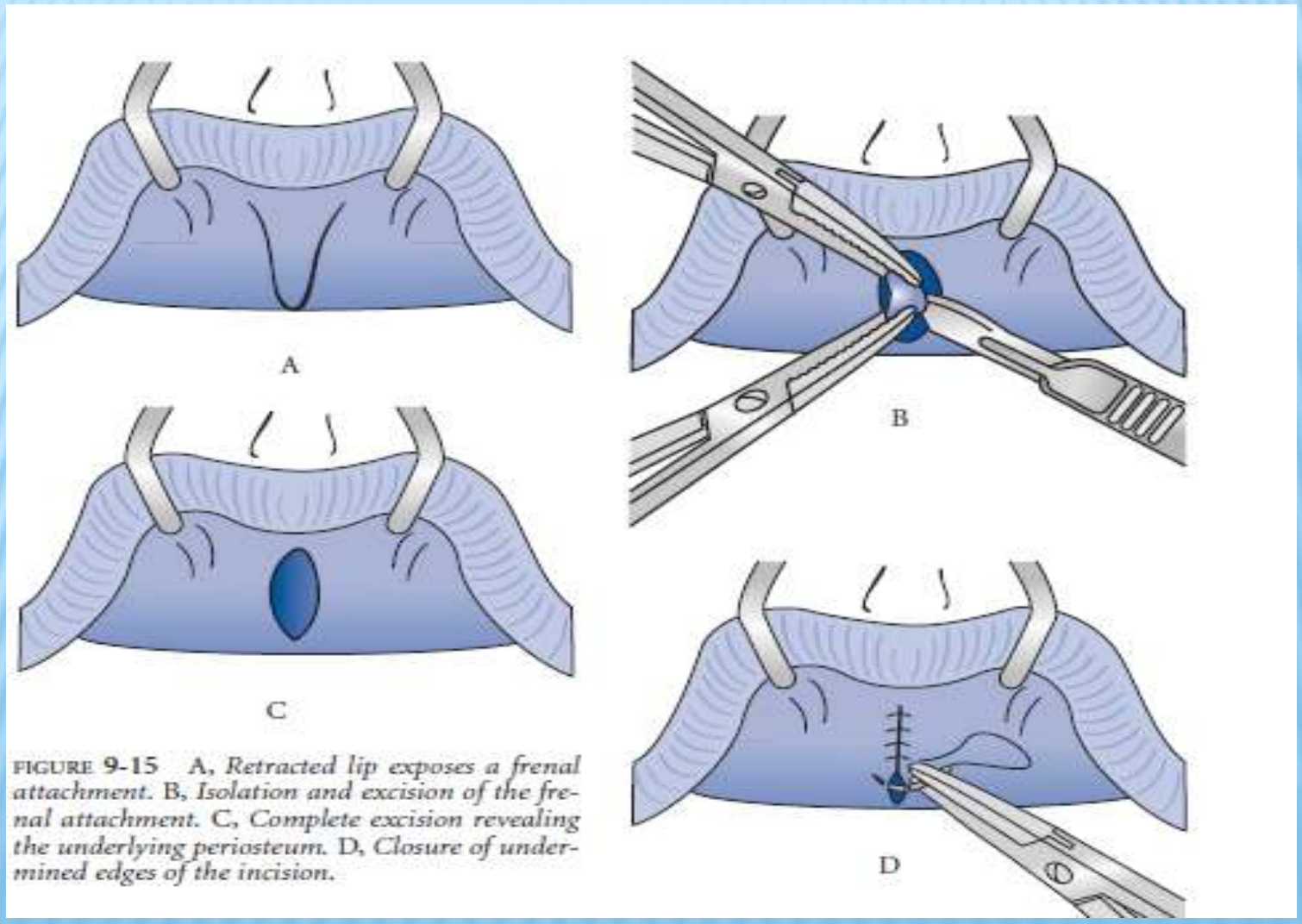


FIGURE 9-15 A, *Retracted lip exposes a frenal attachment.* B, *Isolation and excision of the frenal attachment.* C, *Complete excision revealing the underlying periosteum.* D, *Closure of undermined edges of the incision.*

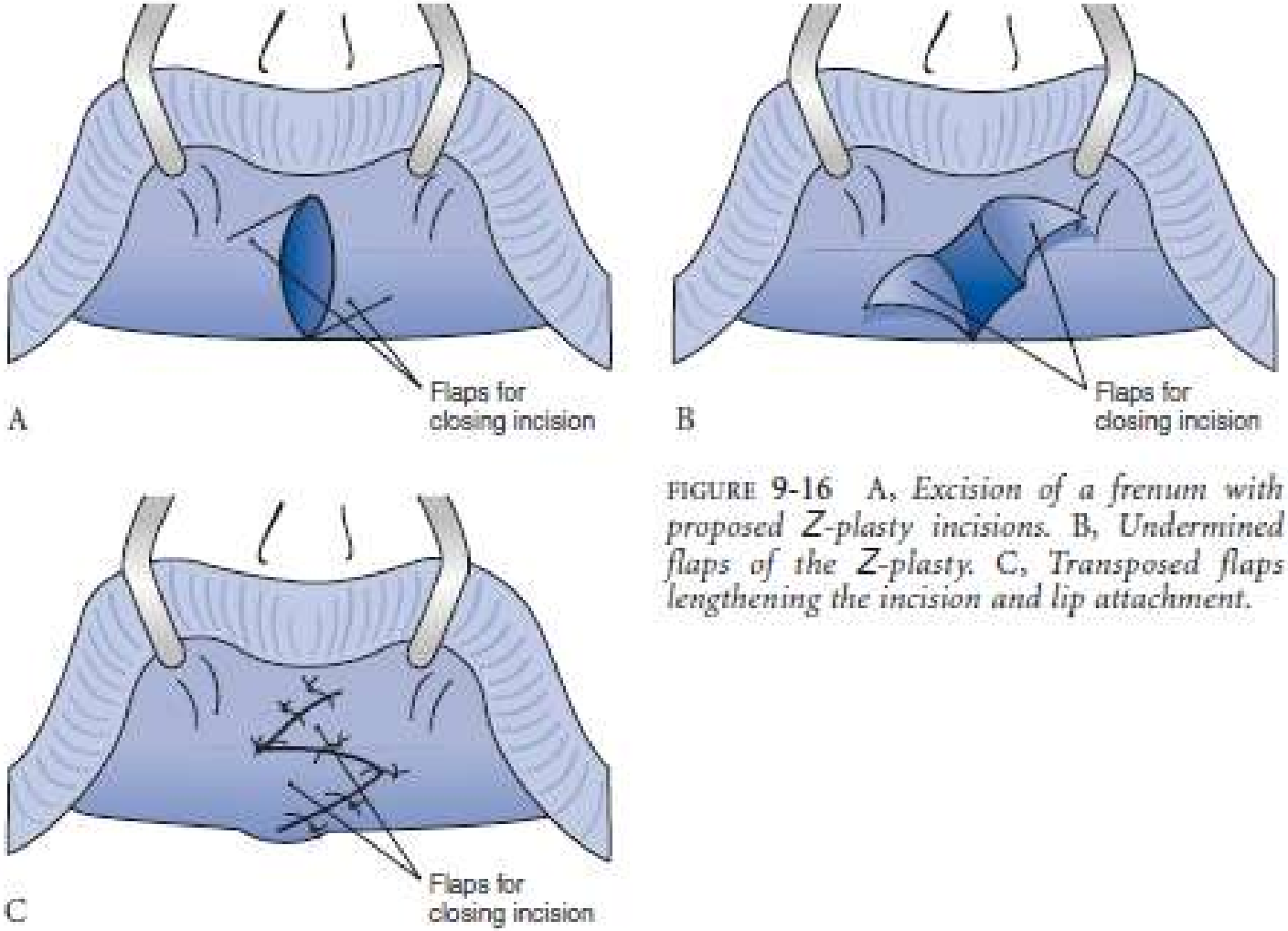
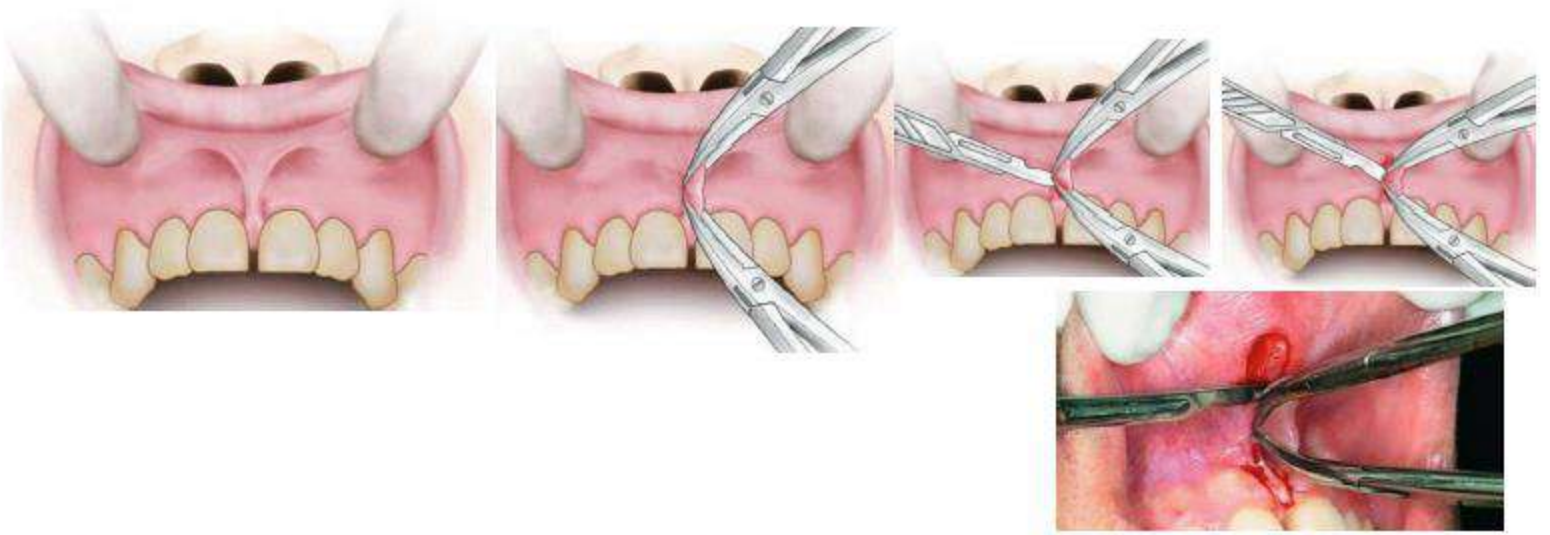
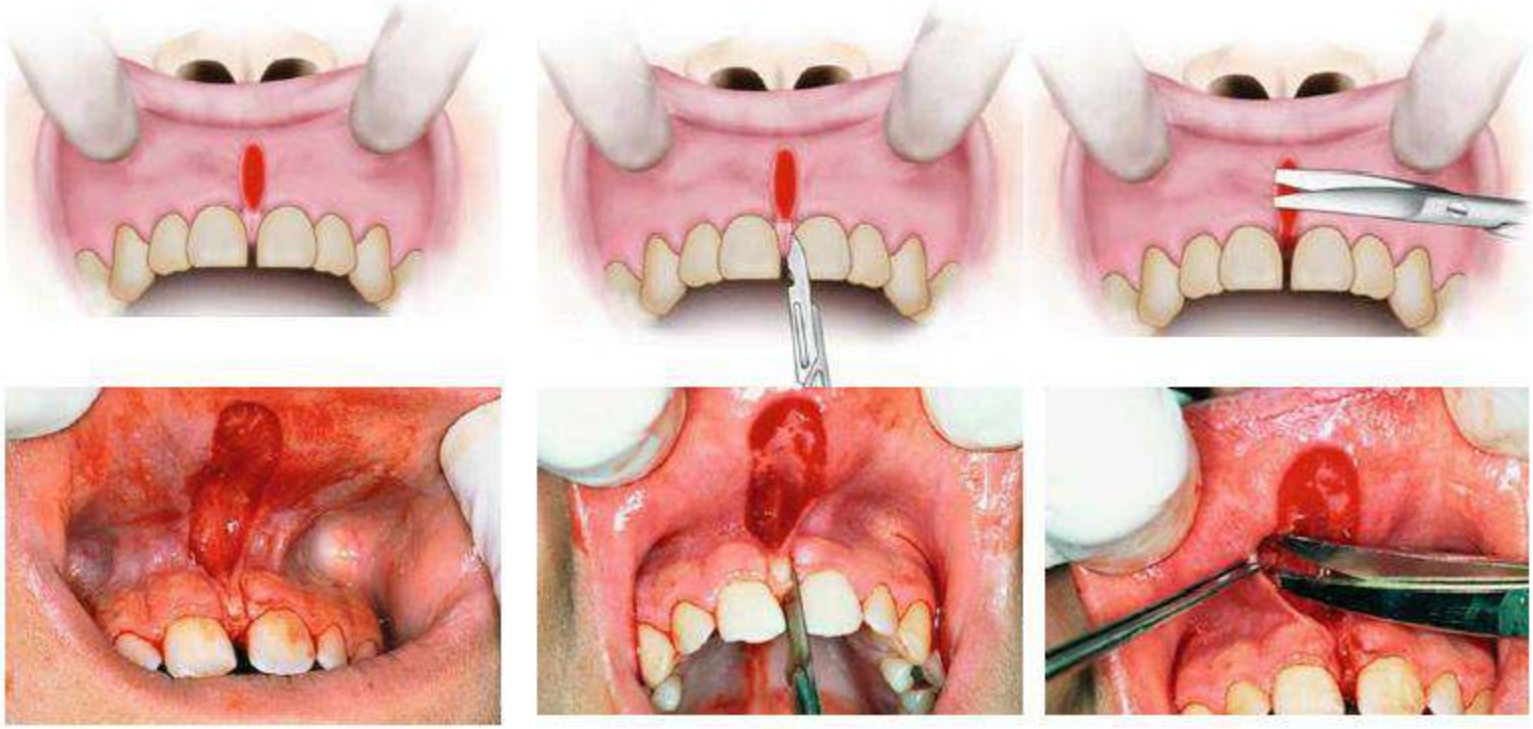


FIGURE 9-16 A, *Excision of a frenum with proposed Z-plasty incisions.* B, *Undermined flaps of the Z-plasty.* C, *Transposed flaps lengthening the incision and lip attachment.*

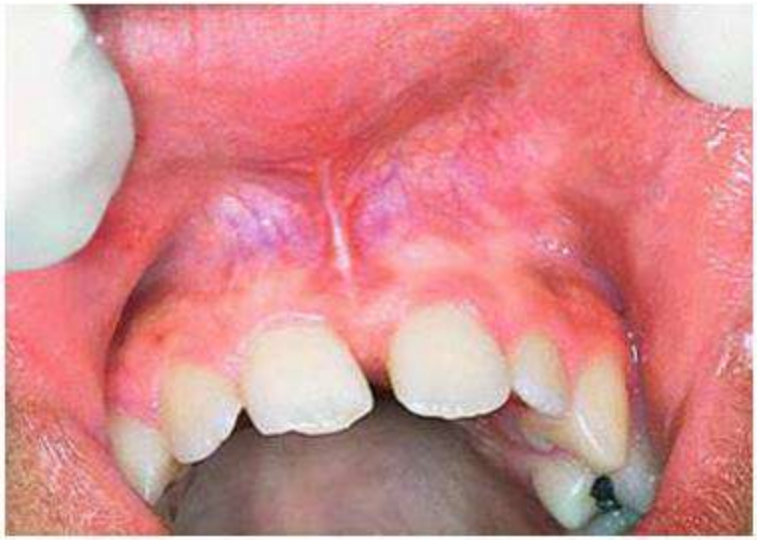
Labial Frenum

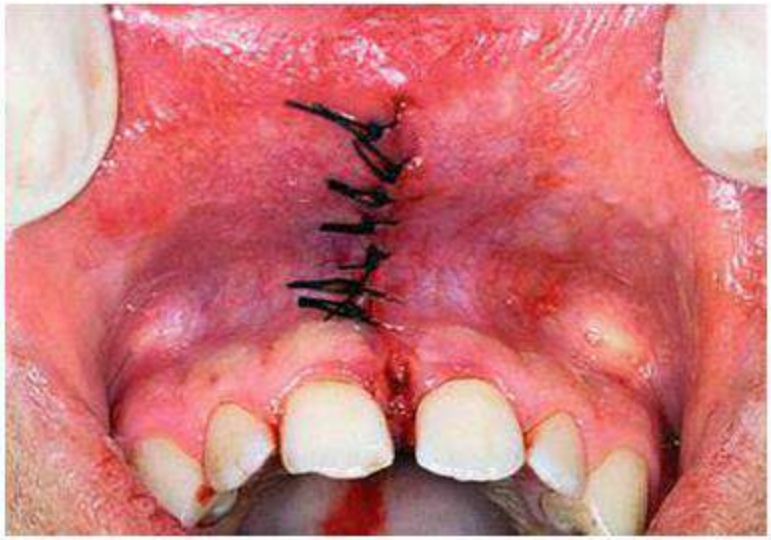


Labial Frenum



Labial Frenum





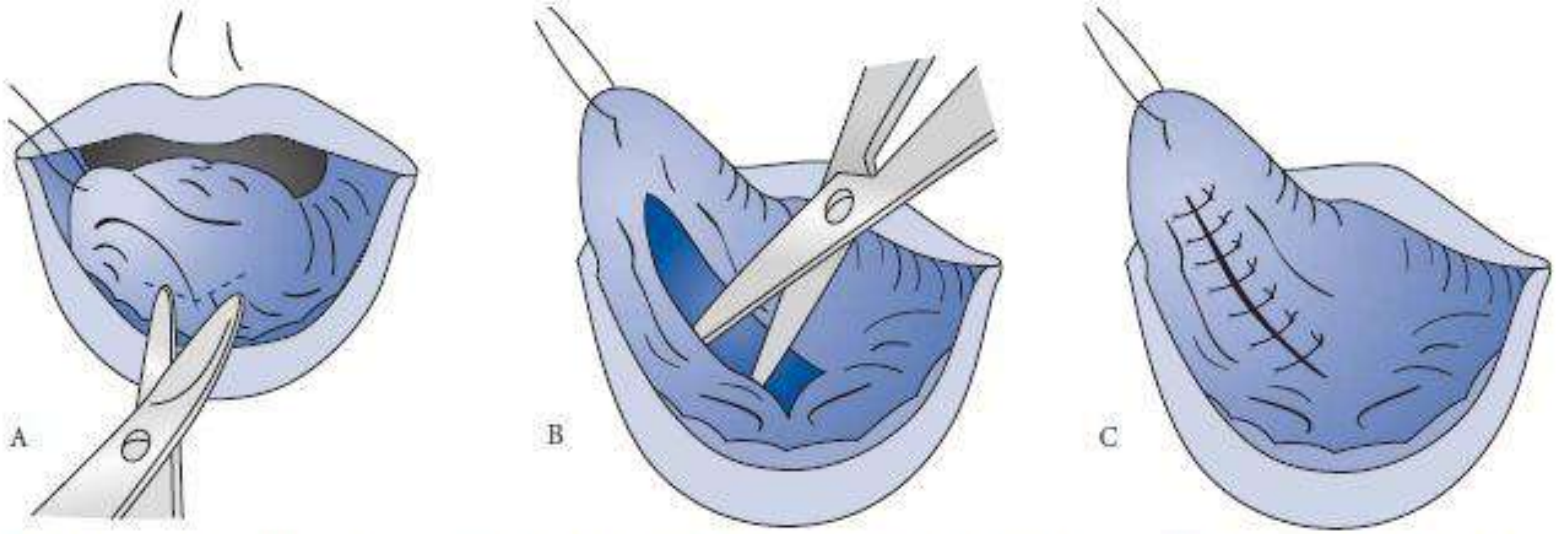
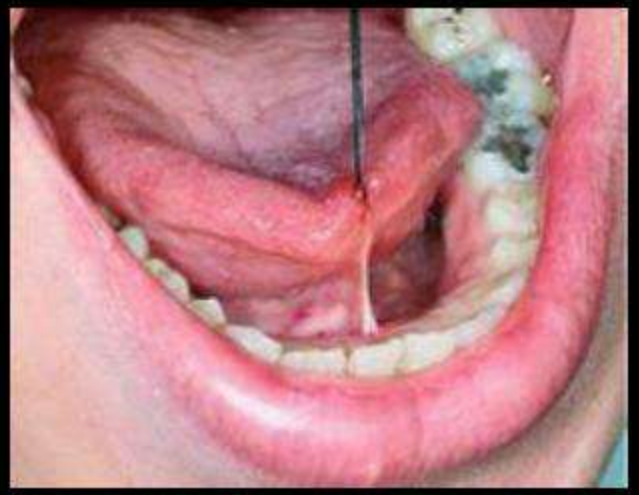
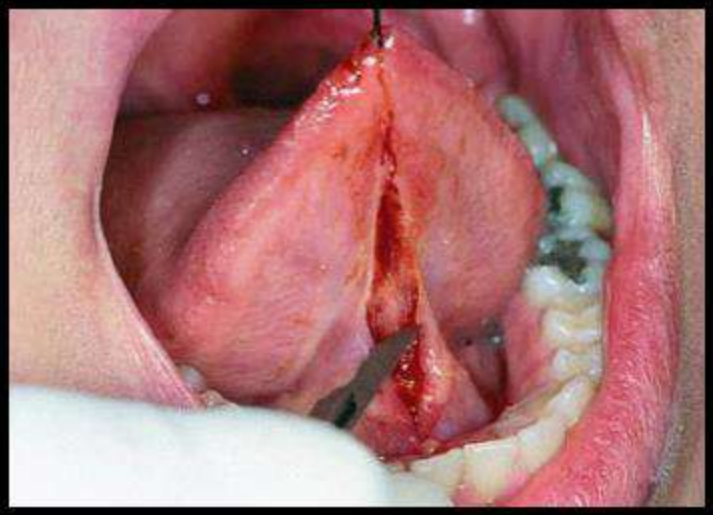
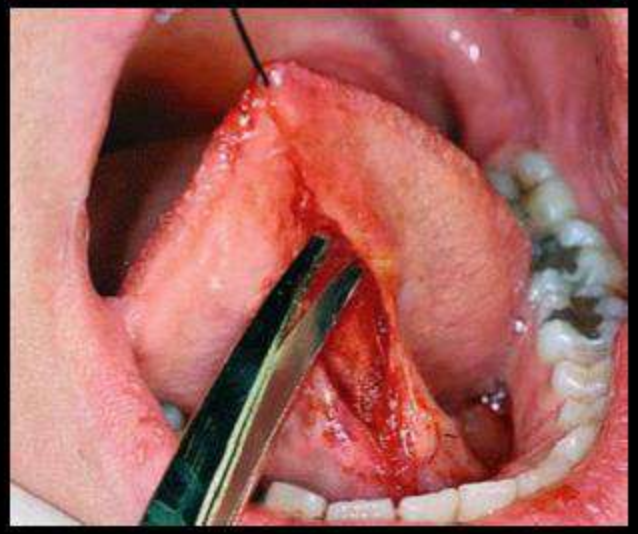


FIGURE 9-17 A, *Lingual frenum attachment encroaching on an atrophic mandibular alveolus.* B, *Excision of the frenum with undermining of mucosal edges. Note: Care must be taken to avoid causing damage to Wharton's ducts.* C, *Final closure of mucosal edges.*

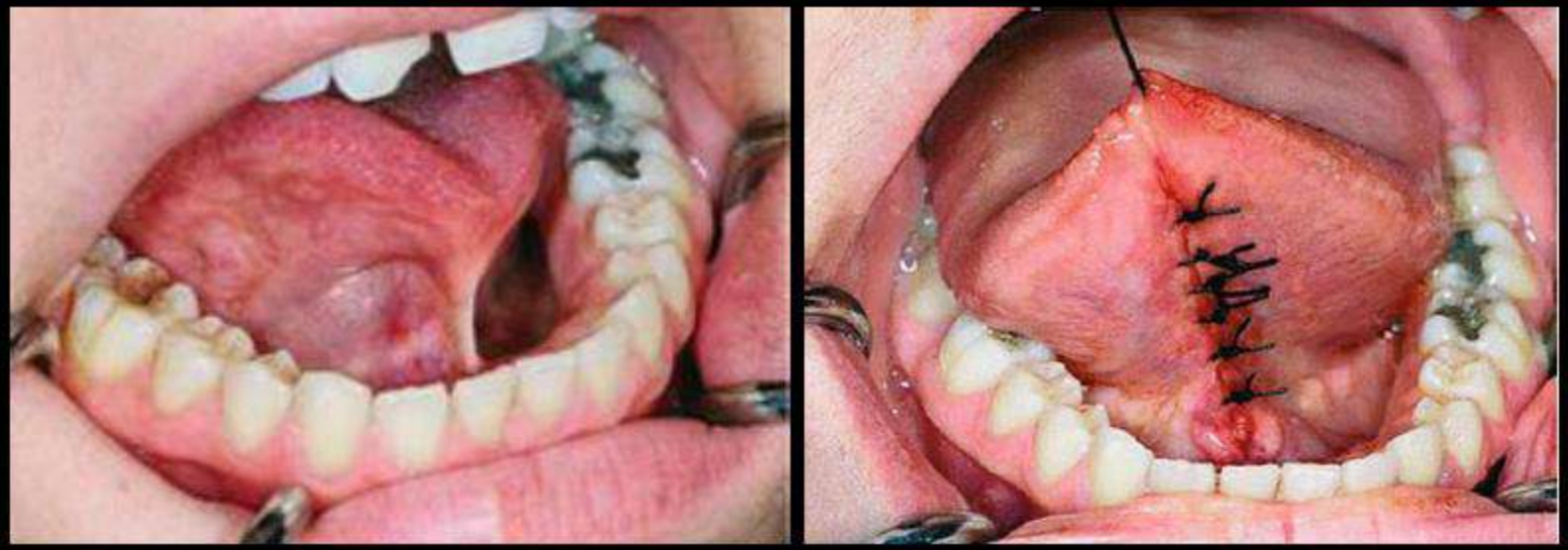
Lingual Frenectomy





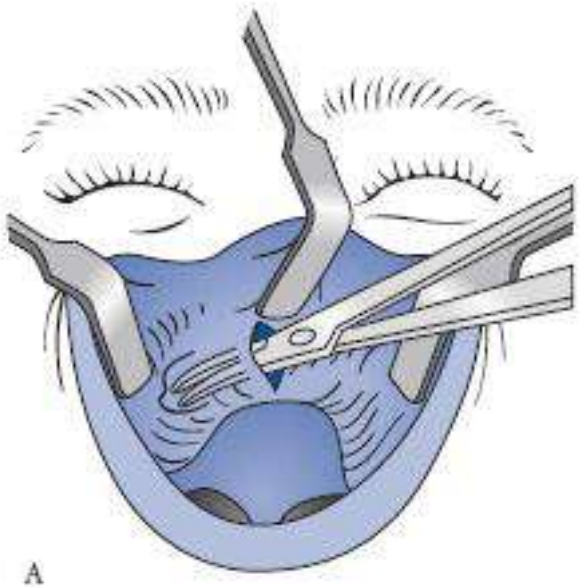


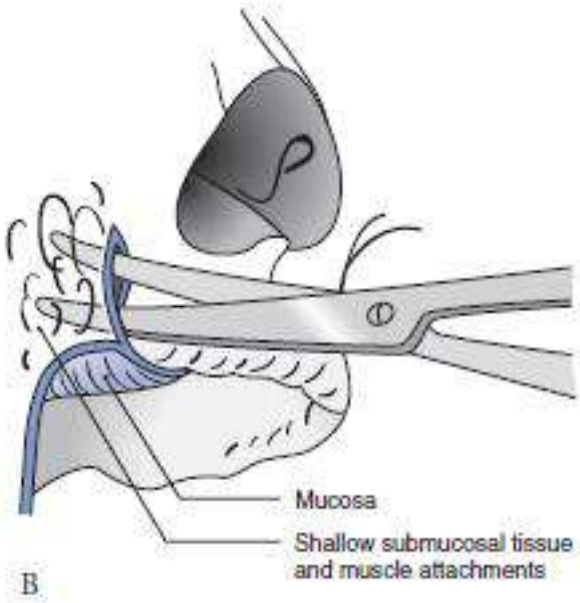
Lingual Frenectomy

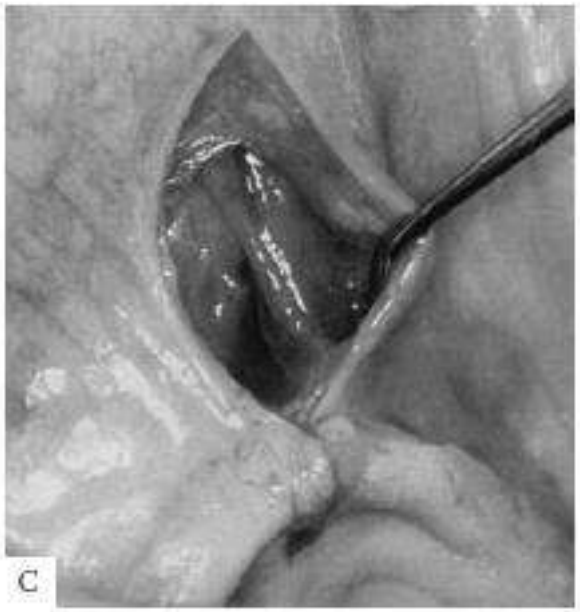


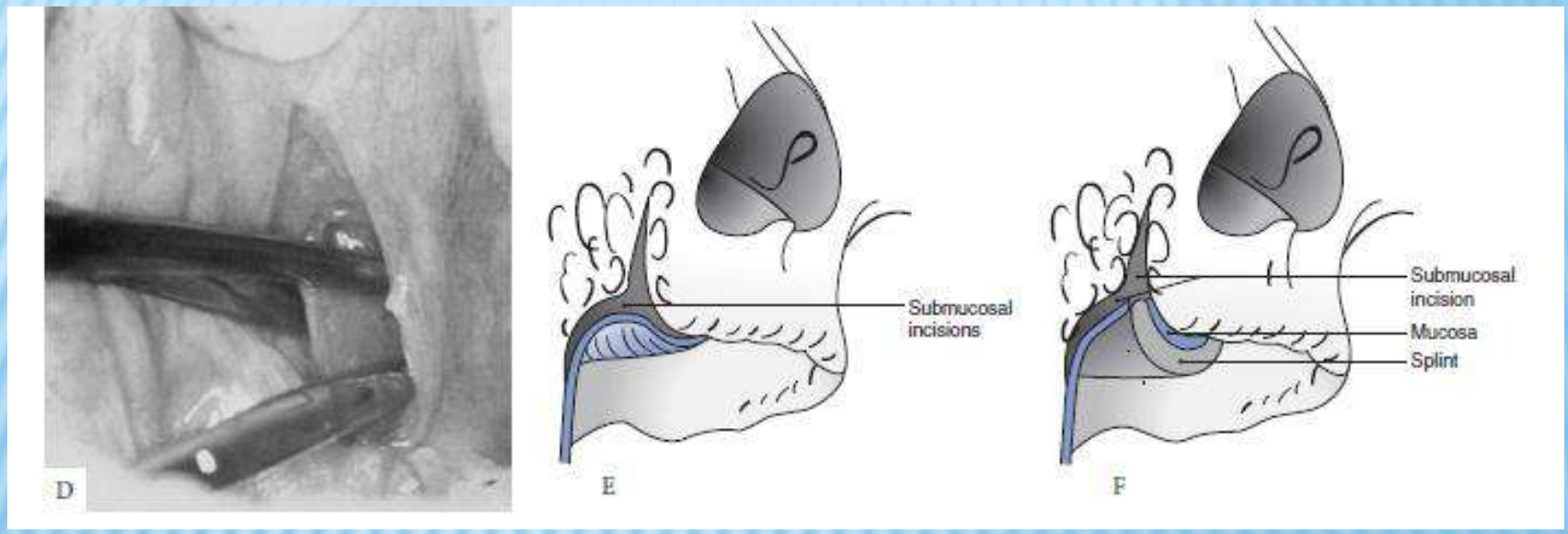
Ridge Extension Procedures in the Maxilla and Mandible

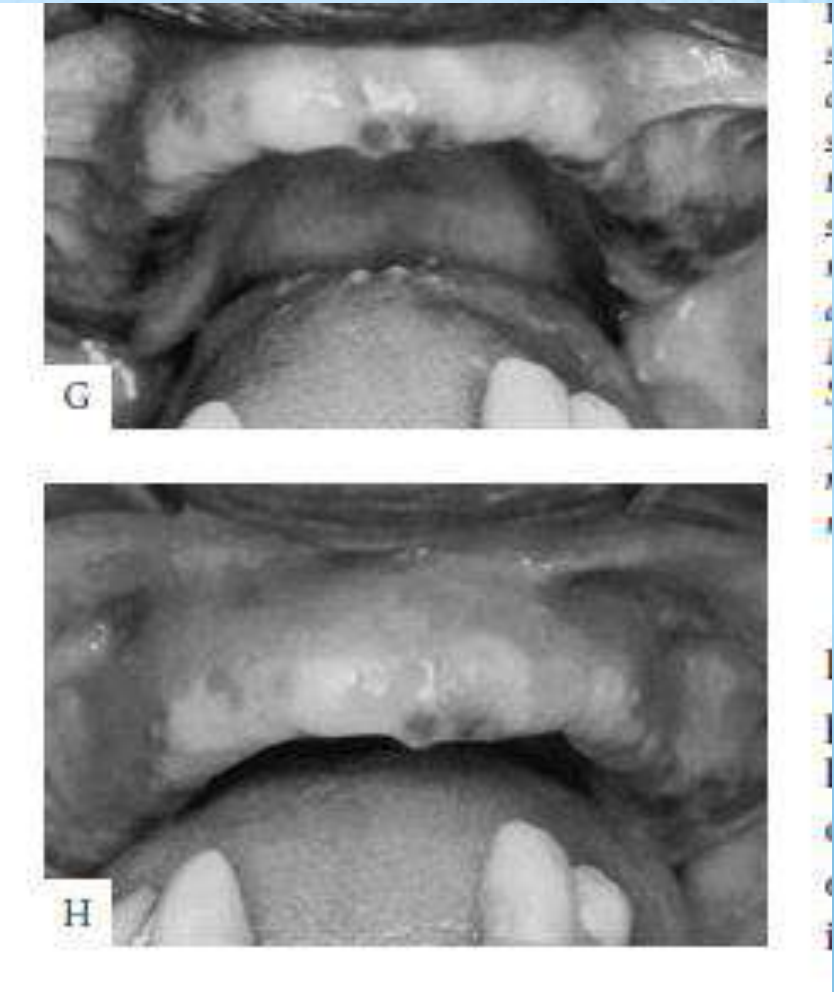
MAXILLARY SUBMUCOSAL VESTIBULOPLASTY



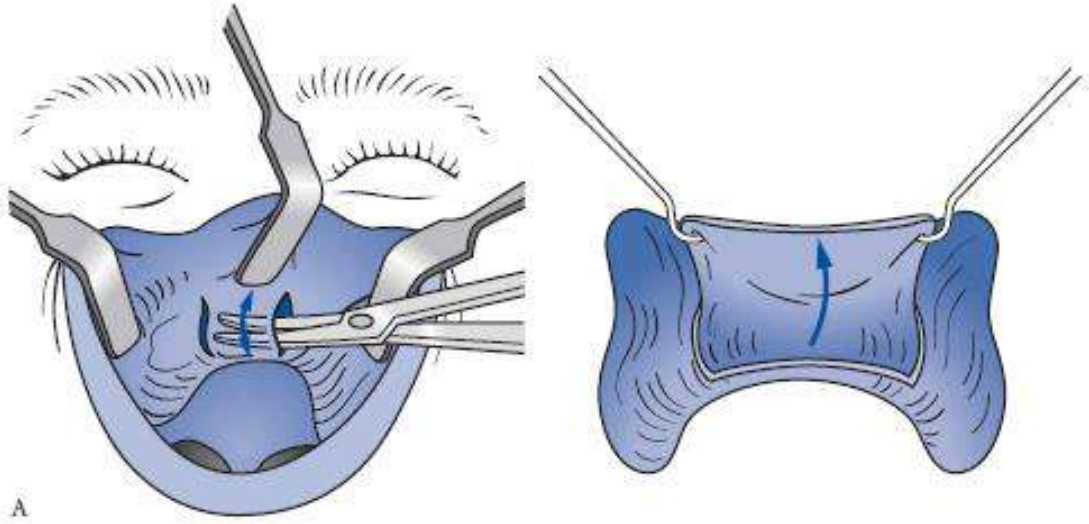


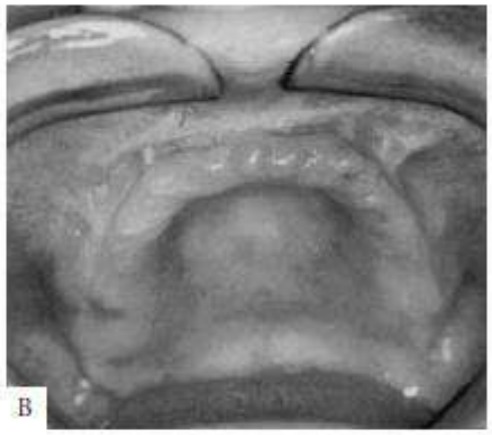


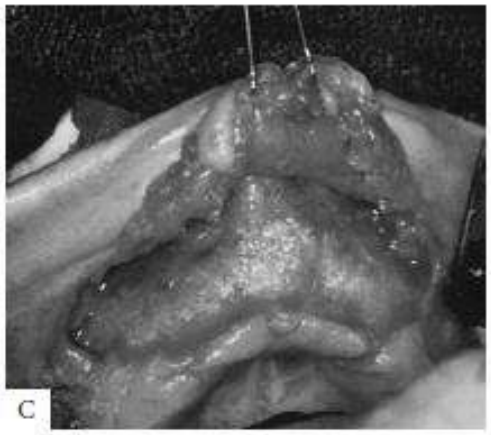

D
Submucosal incisions
E
Submucosal incision
Mucosa
Splint
F

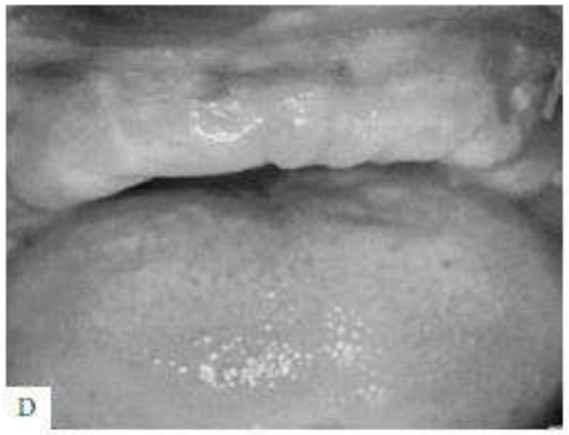

G
H

OPEN SUBMUCOSAL VESTIBULOPLASTY









KAZANJIAN FLAP VESTIBULOPLASTY

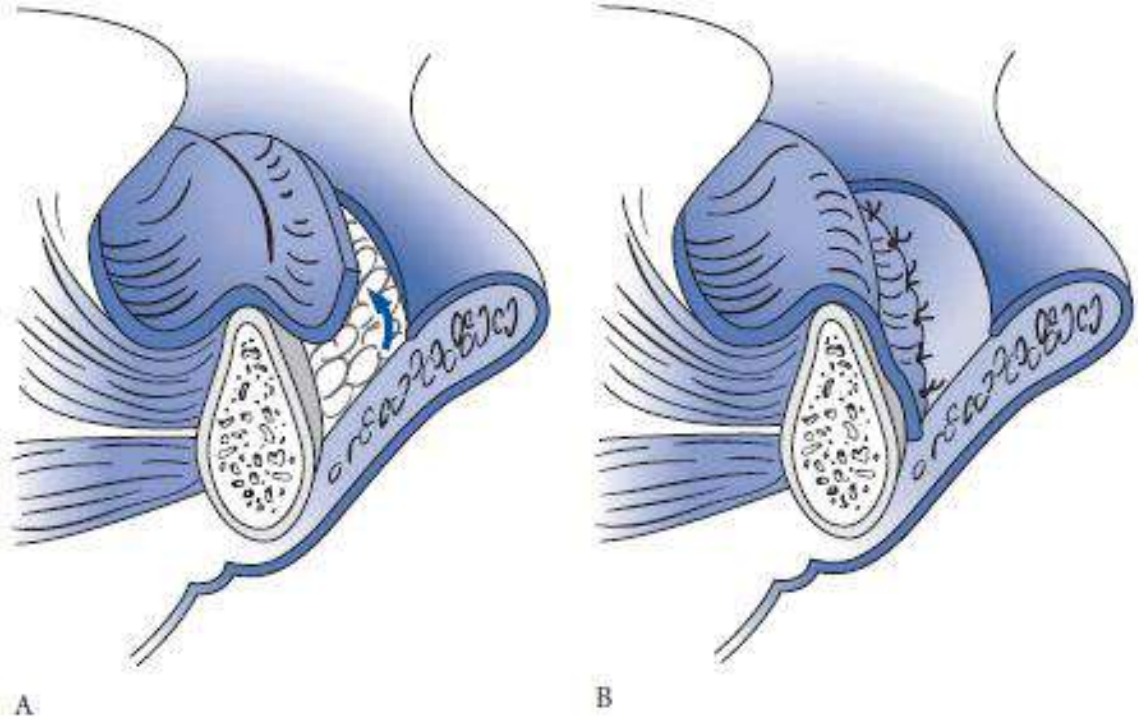
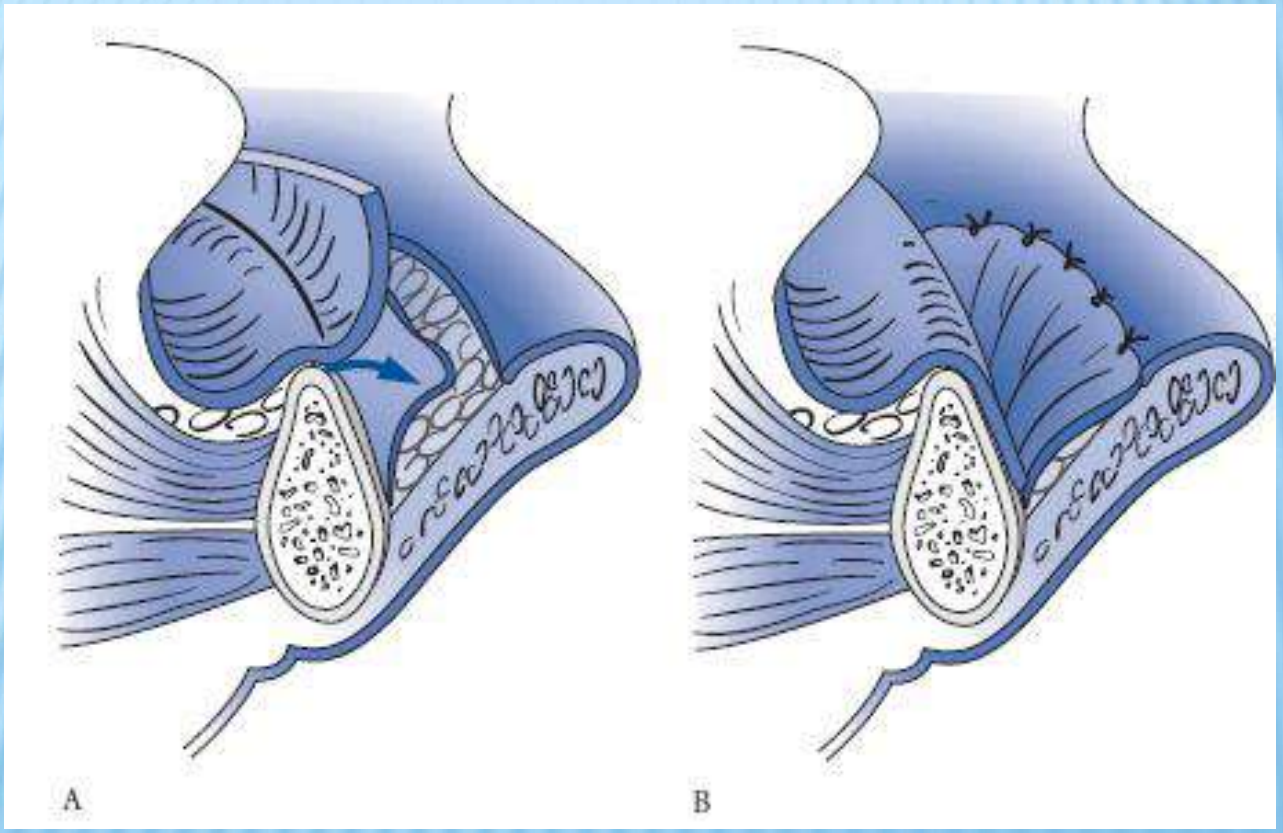
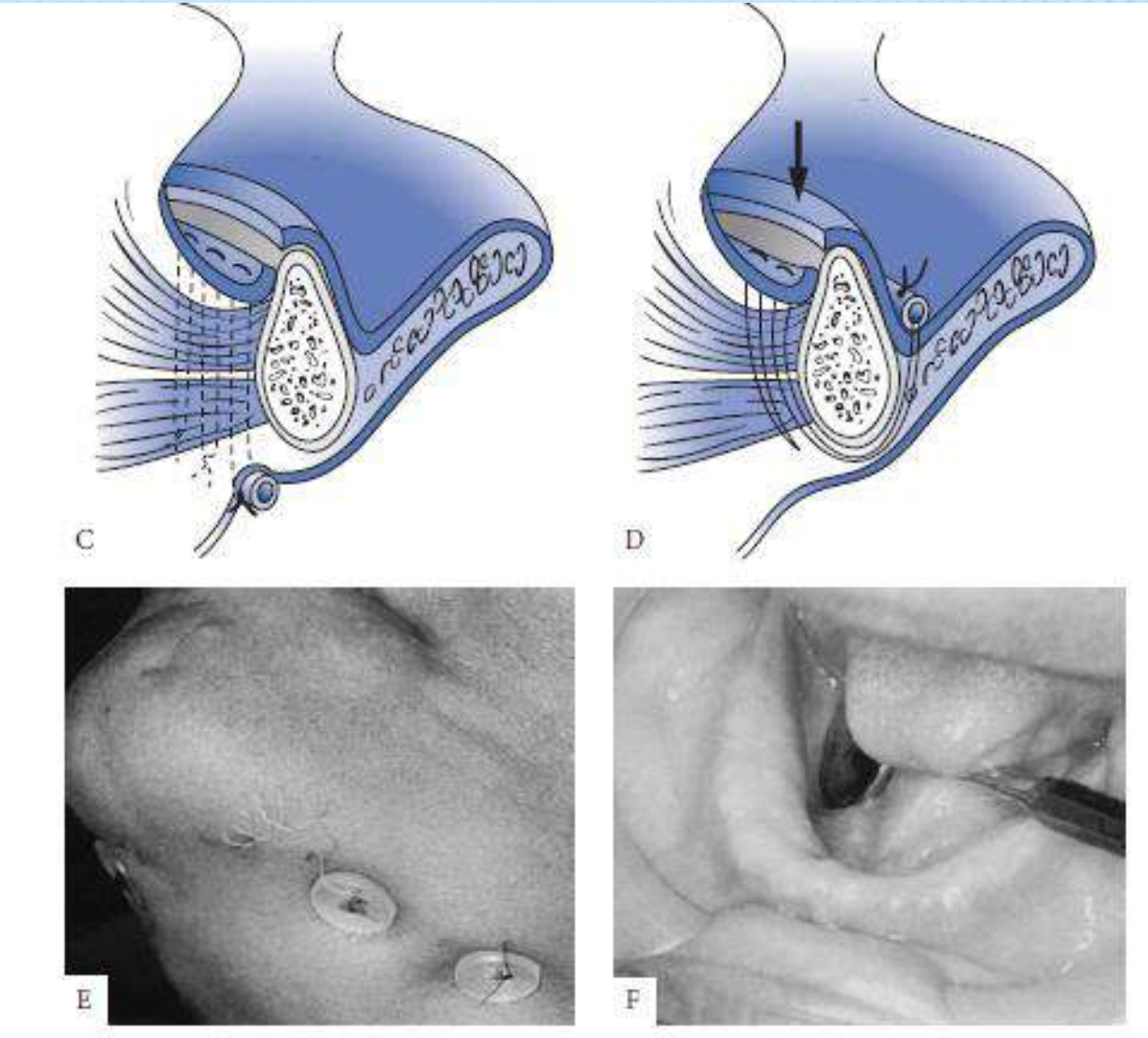


FIGURE 9-21 *Kazanjian flap vestibuloplasty. A, An incision is made in the labial mucosa, and a thin mucosal flap is dissected from the underlying tissue. A supraperiosteal dissection is performed on the anterior aspect of the mandible. B, The labial mucosal flap is sutured to the depth of the vestibule. The anterior aspect of the labial vestibule heals by secondary intention. Adapted from Tucker MR. Ambulatory preprosthetic reconstructive surgery. In: Peterson LJ, Indresano AT, Marciani RD, Roser SM. Principles of oral and maxillofacial surgery. Vol 2. Philadelphia (PA): JB Lippincott Company; 1992. p. 1120.*

TRANSPOSITIONAL FLAP (LIP-SWITCH) VESTIBULOPLASTY



FLOOR-OF-MOUTH LOWERING

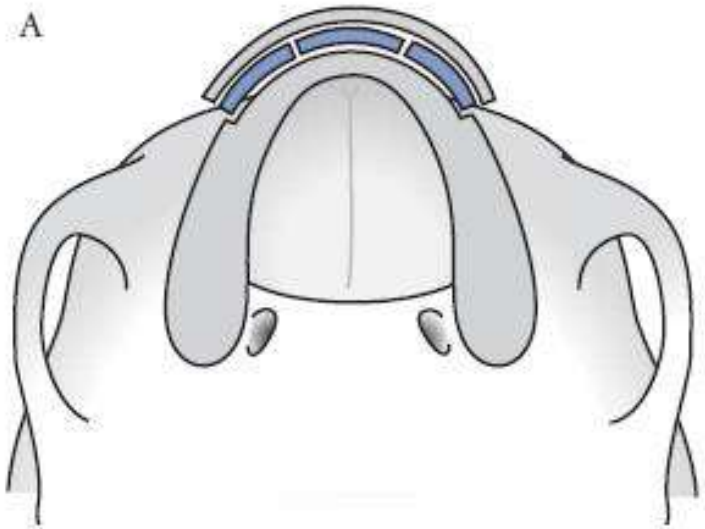


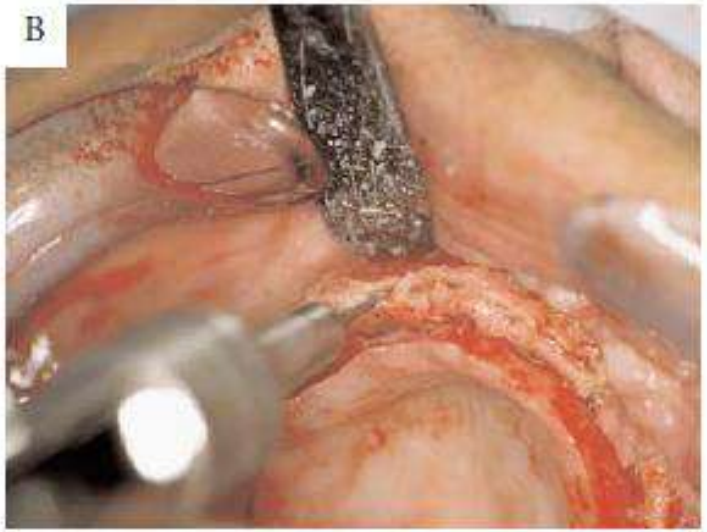
HARD TISSUE AUGMENTATION

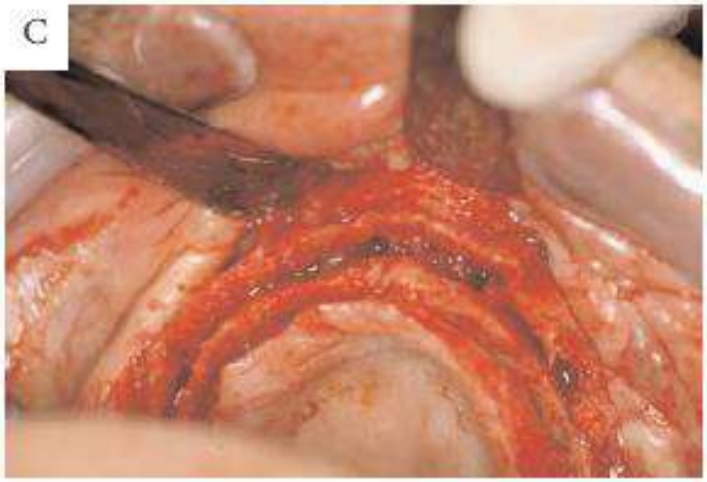
MAXILLARY AUGMENTATION (FOR IV- VI RIDGE FORMS)

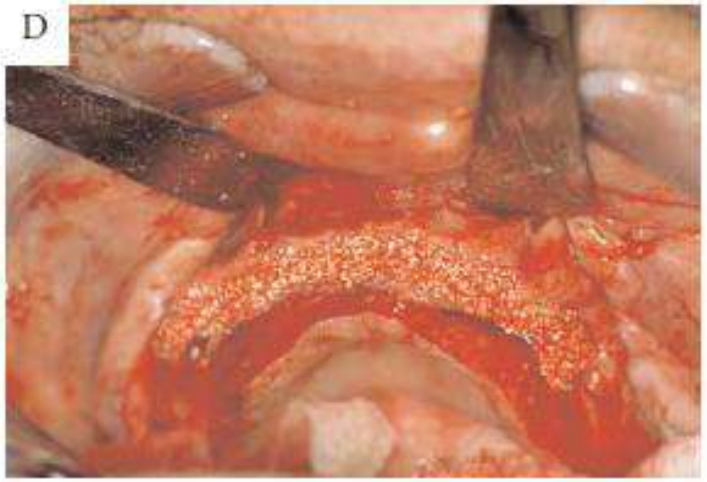
- Ridge Split Osteoplasty
- Onlay graft
- Interpositional graft
- inlay grafting

RIDGE SPLIT OSTEOPLASTY

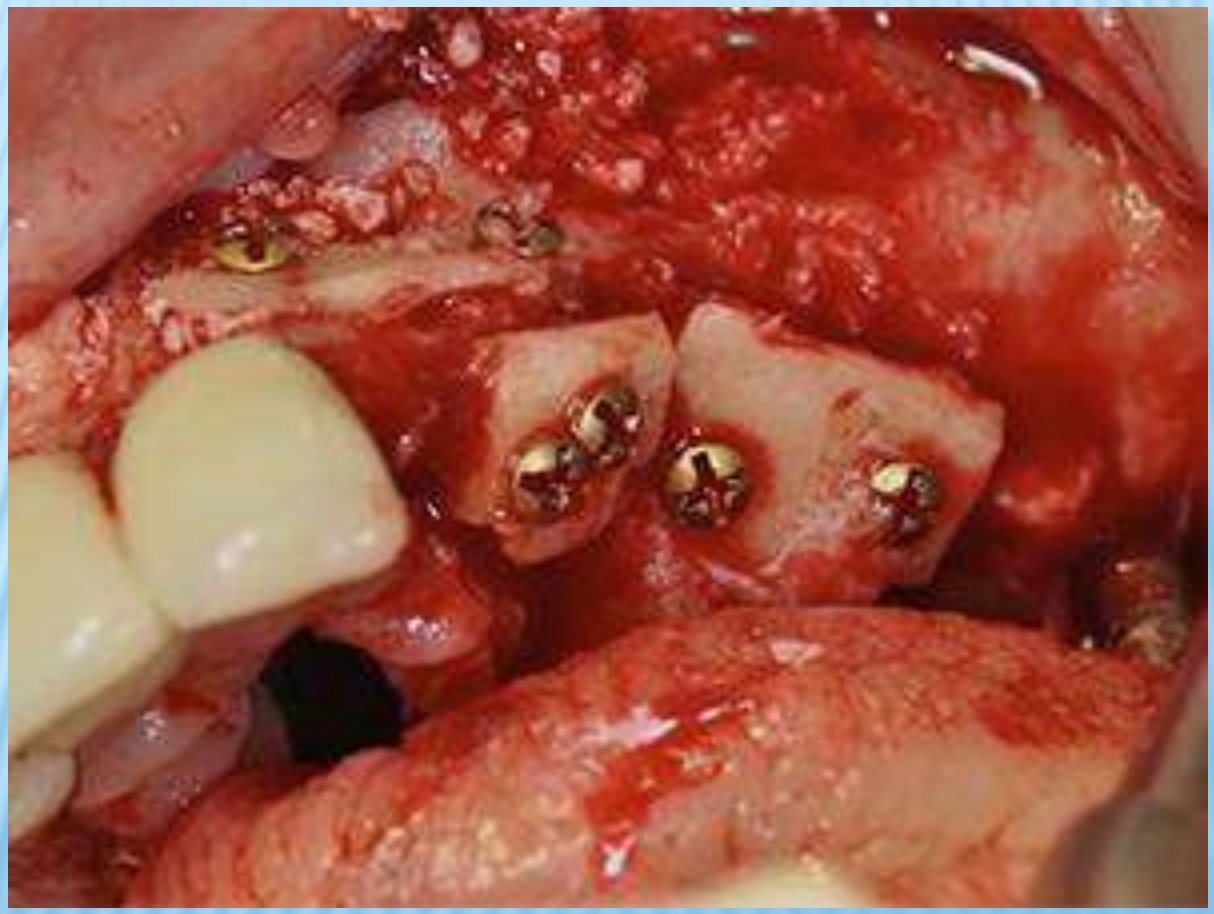








ONLAY GRAFT



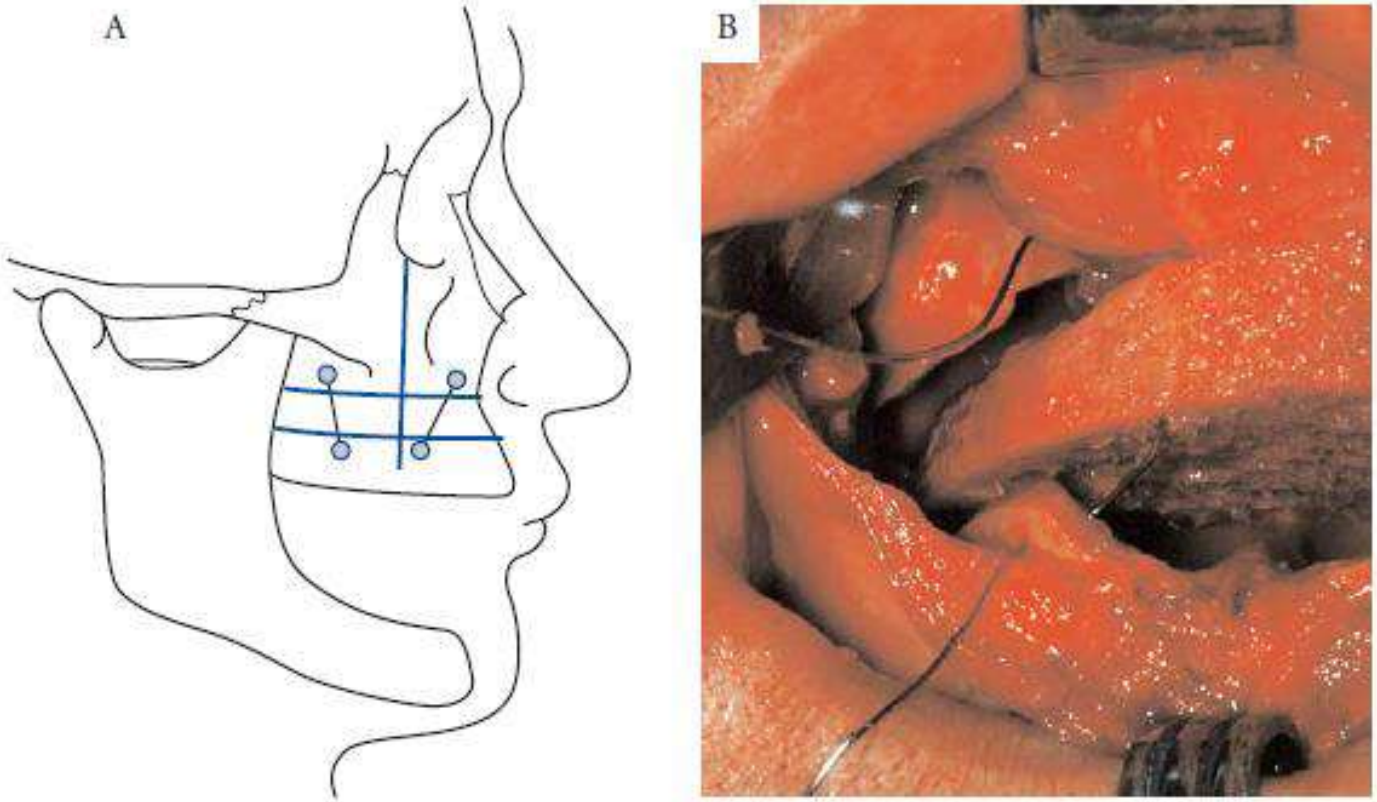
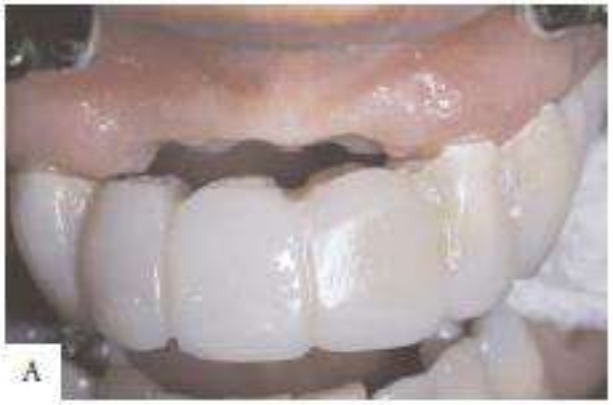
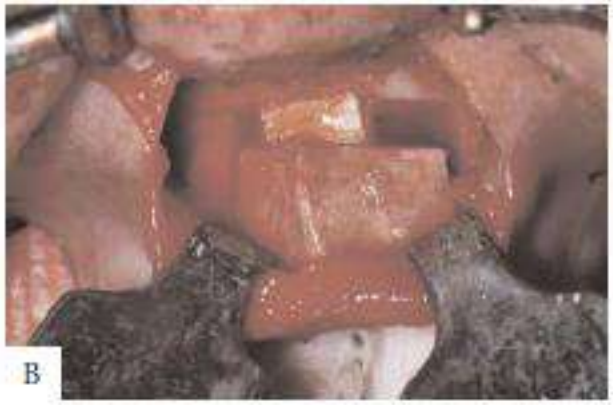


FIGURE 9-26 *Graphic (A) and clinical presentation (B) of interpositional iliac crest grafts to the maxilla.*


A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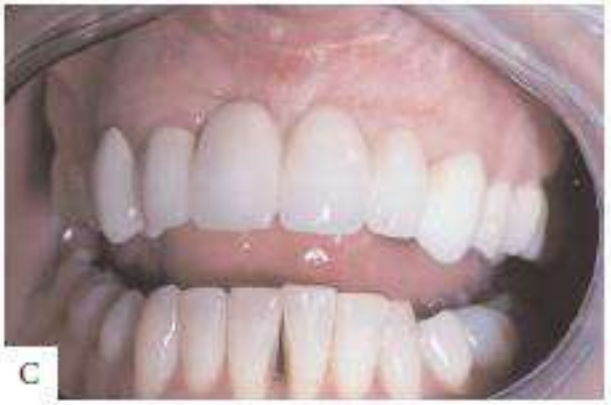
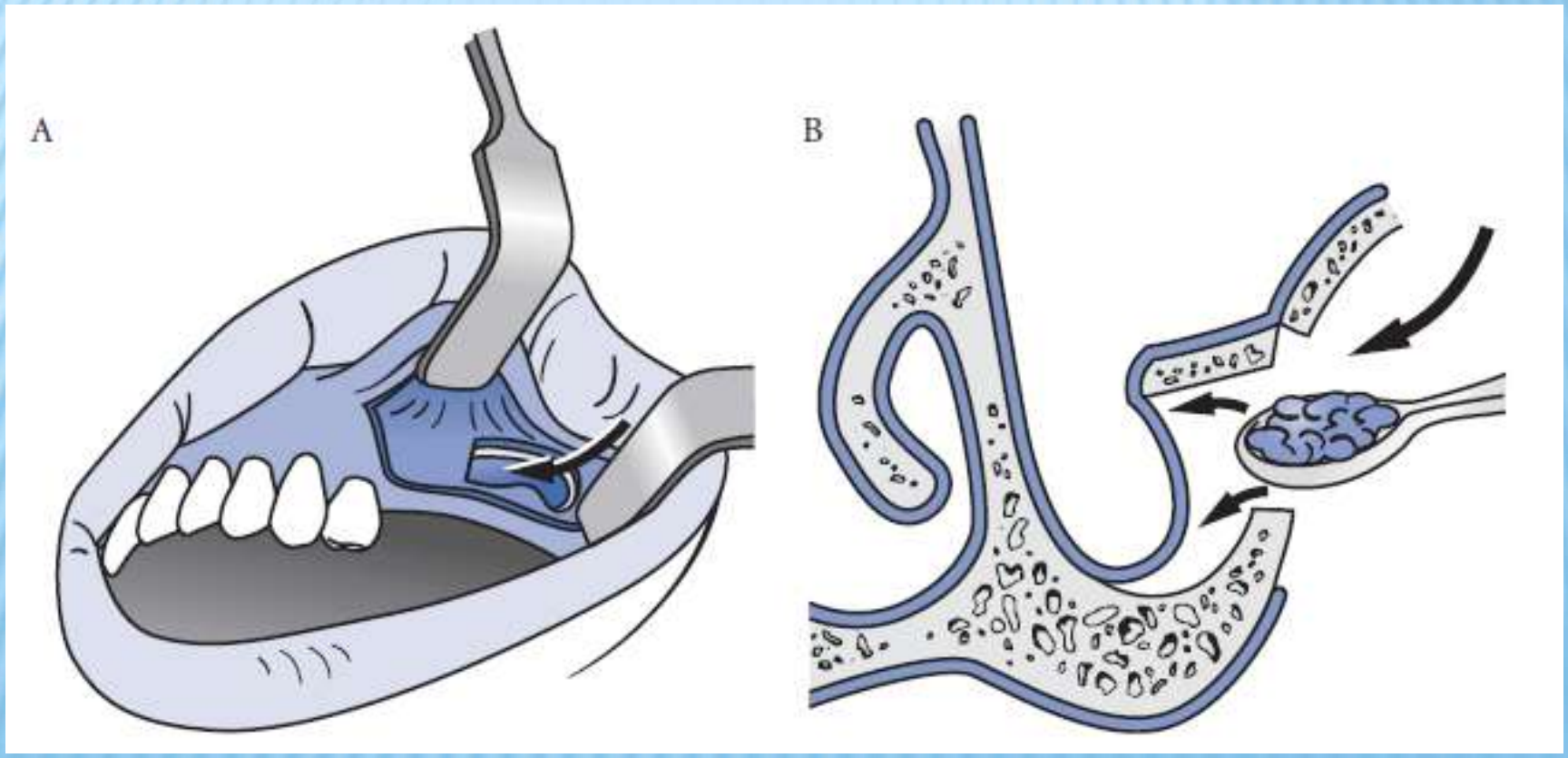

C

INLAY GRAFT AND SINUS LIFT PROCEDURE



MANDIBULAR AUGMENTATION

- Inferior and superior border augmentation
- Pedicled and interpositional grafts
- Distraction osteogenesis

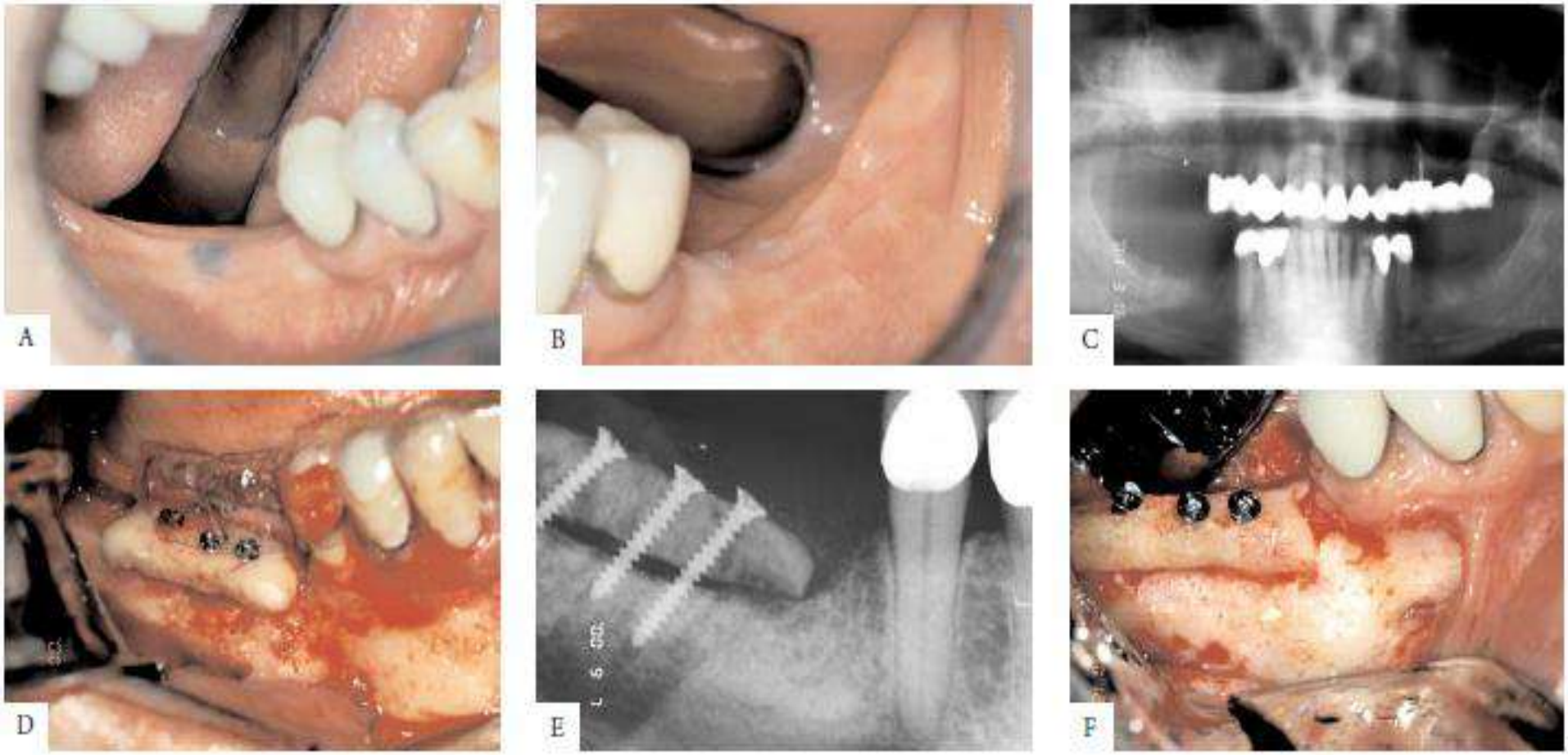
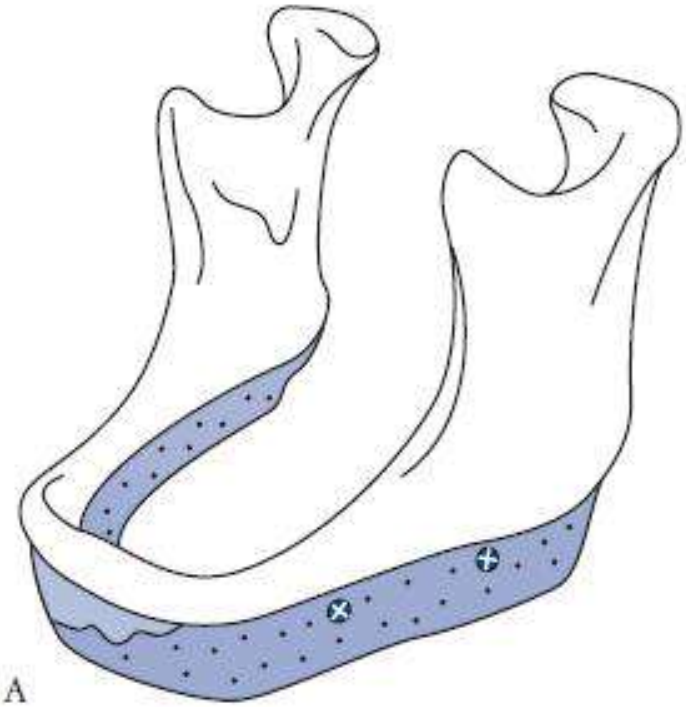
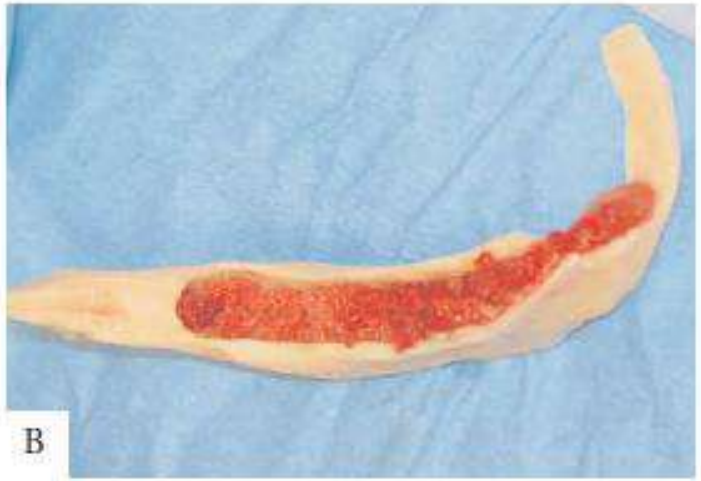


FIGURE 12-3 A, *Right posterior edentulous mandible.* B, *Left posterior edentulous mandible.* C, *Radiograph indicating bilateral posterior mandibular vertical deficiency.* D, *Block graft fixation with platelet-rich plasma application.* E, *Block graft fixation. Note butt joint at anterior recipient donor interface.* F, *Excellent block graft incorporation at 5 months.* (CONTINUED ON NEXT PAGE)



A



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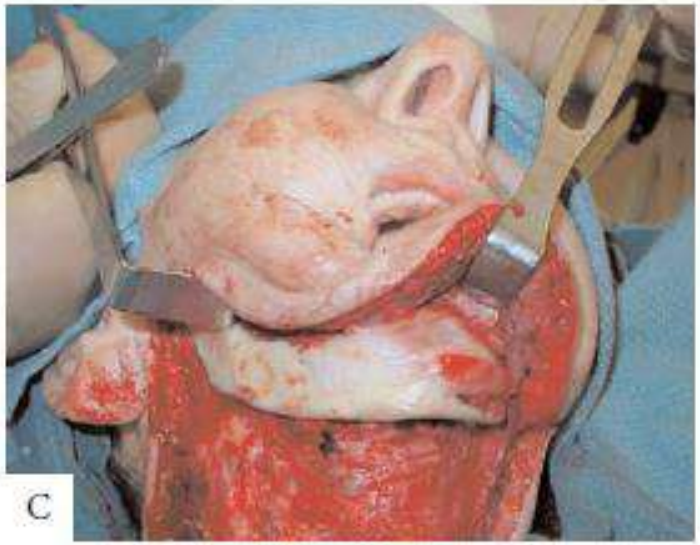


C

FIGURE 9-29 A, *Cadaveric mandible tray rigidly fixed to an atrophic mandible with autogenous cancellous bone sandwiched between native and cadaveric bone. Note that bur holes have been created to facilitate the revascularization of the graft.* B, *Cadaveric tray filled with autogenous bone before insetting.* C, *Graft and cadaveric tray inset for inferior border augmentation.*

PEDICLED FLAP

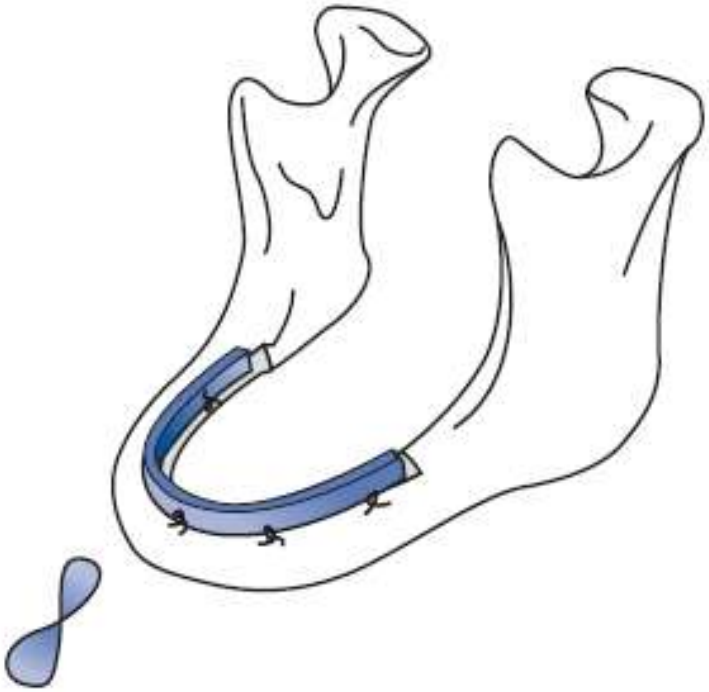


FIGURE 9-30 *Visor osteotomy devised by Harle F. Adapted from Stoelinga PJW[61] and Harle F.[63]*

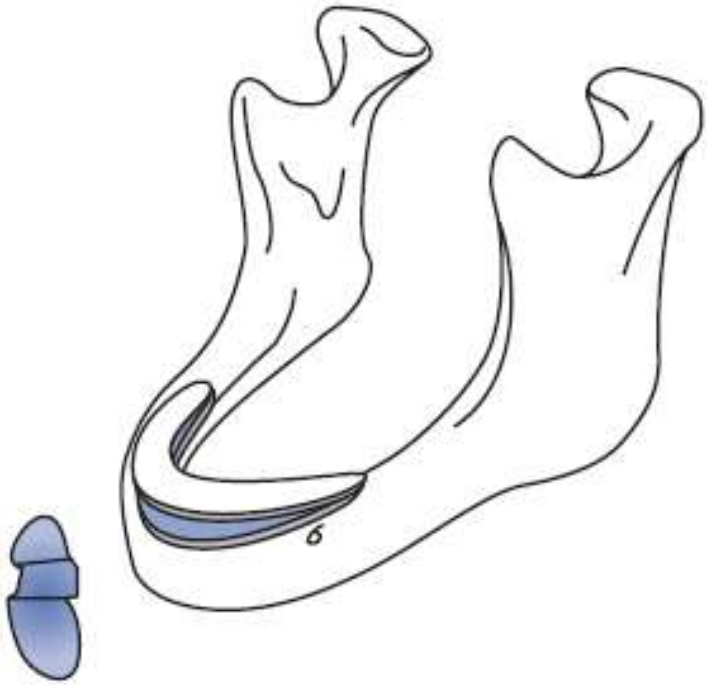


FIGURE 9-31 *Sandwich osteotomy designed by Schettler and Holtermann. Adapted from Stoelinga PJW[61] and Schettler D and Holtermann W.[64]*

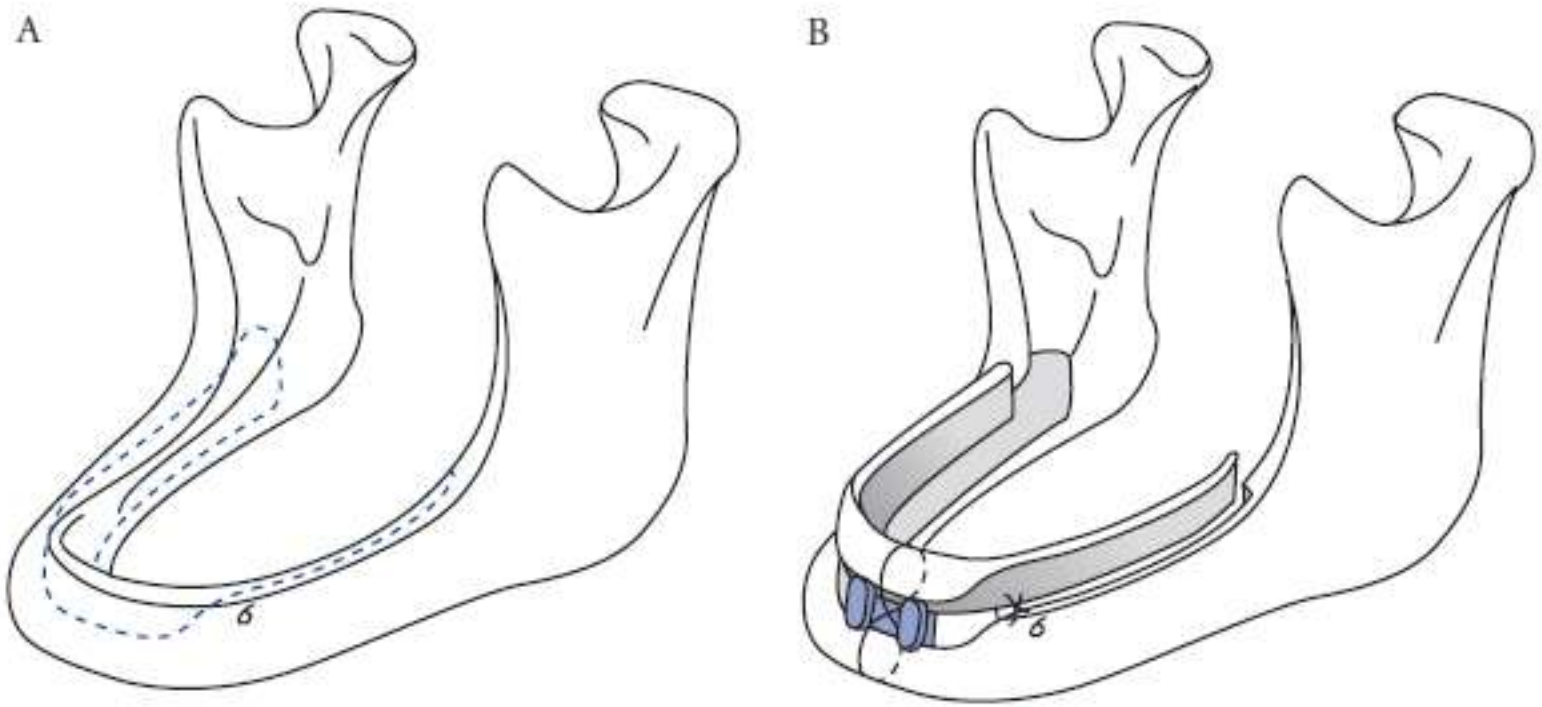
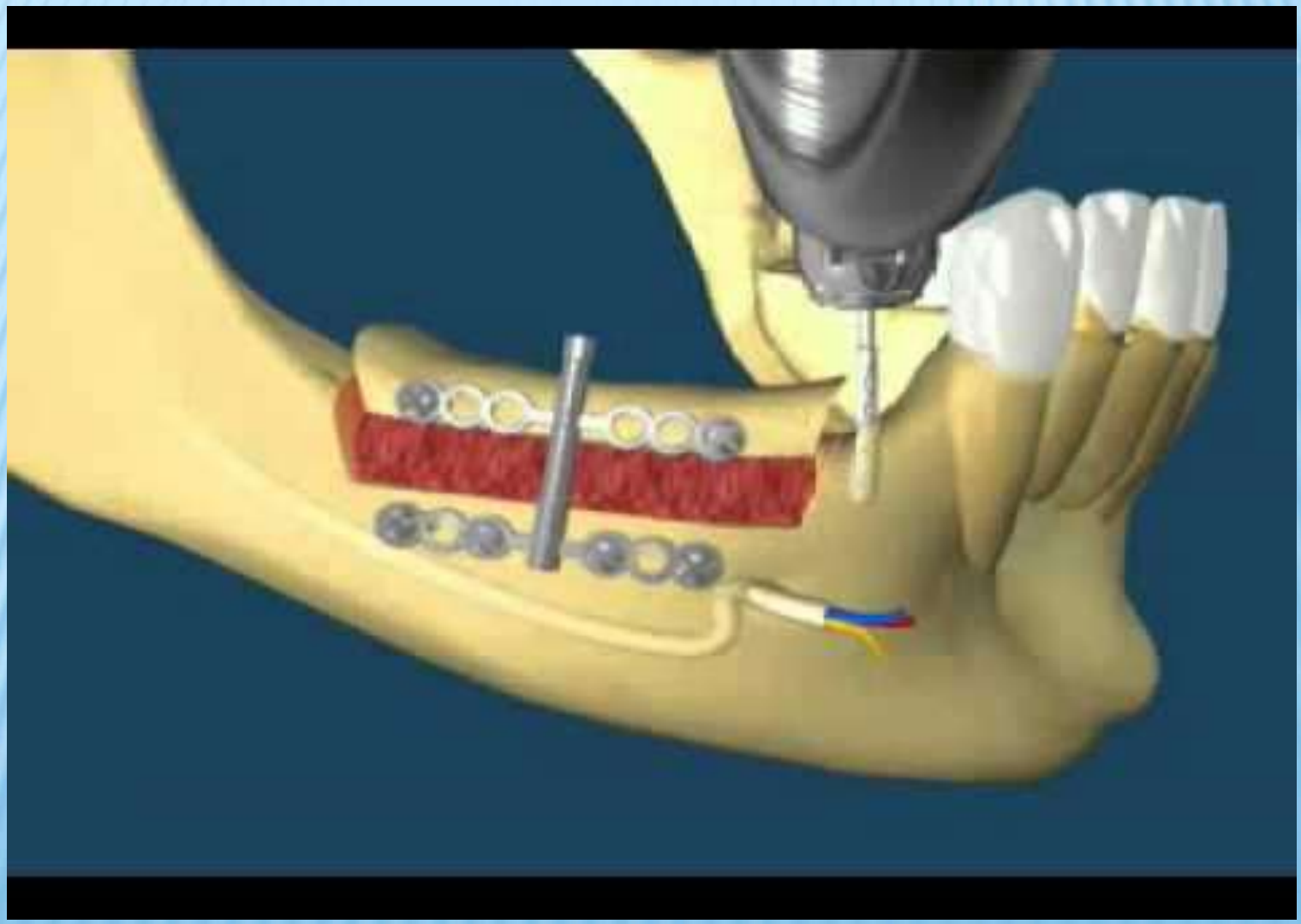
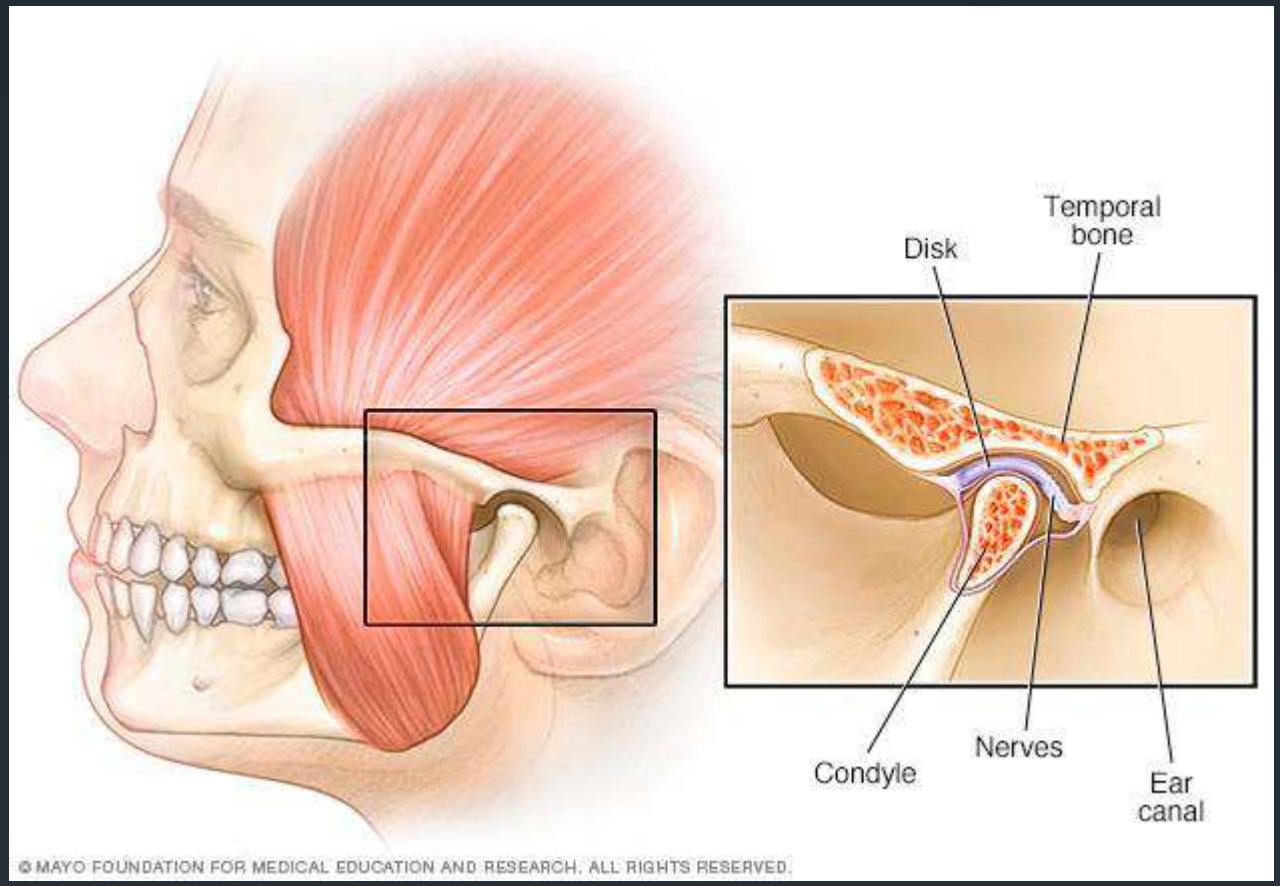


FIGURE 9-32 *Sandwich-visor osteotomy according to Stoelinga and Tideman.* A, *Bone cut is outlined* (dotted line). B, *Cranial fragment is lifted, supported by bone struts, and secured by a wire tied in a figure of eight. Adapted from Stoelinga PJW[61] and Schettler D and Holtermann W.[64]*

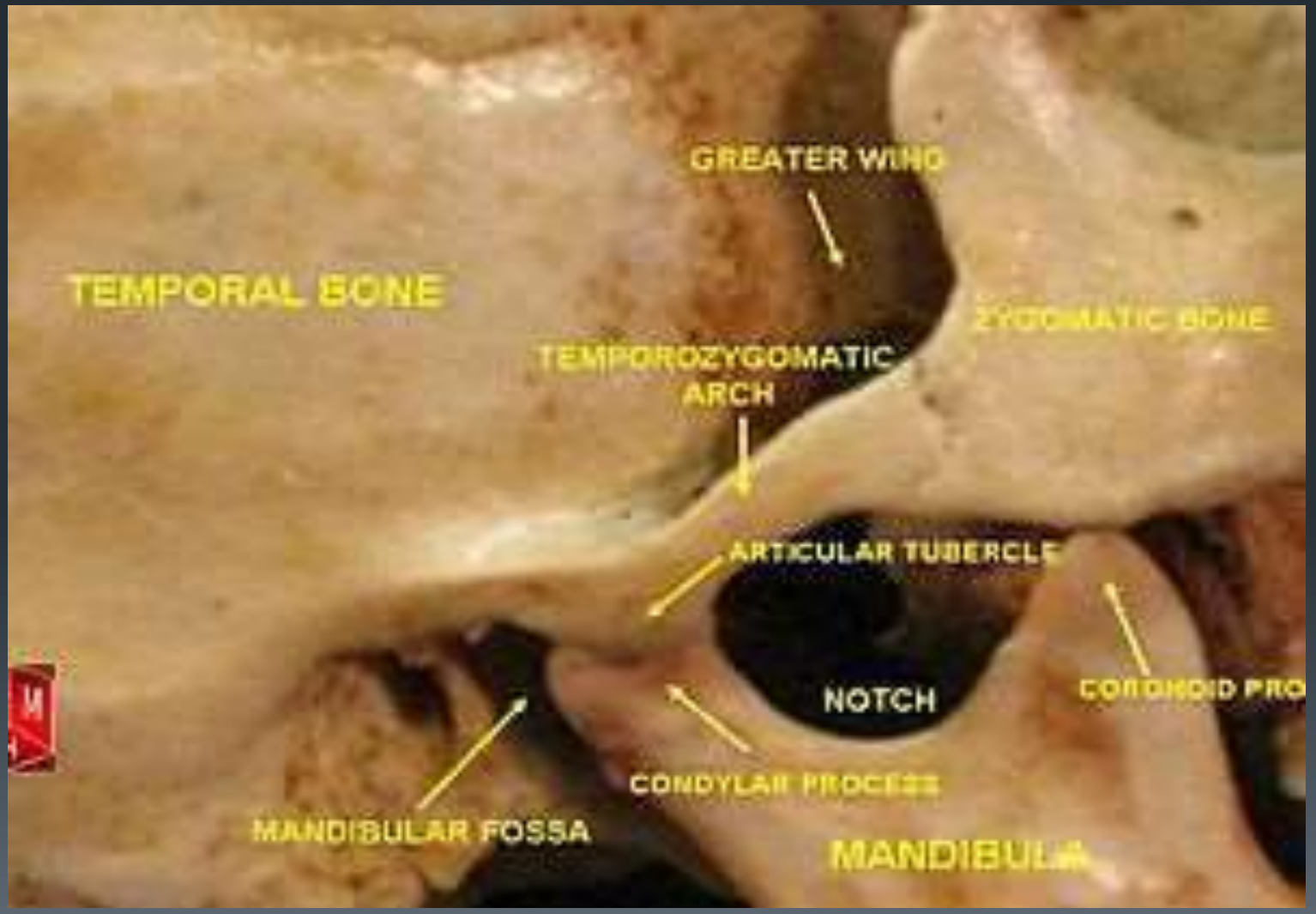
ALVEOLAR DISTRACTION OSTEOGENESIS

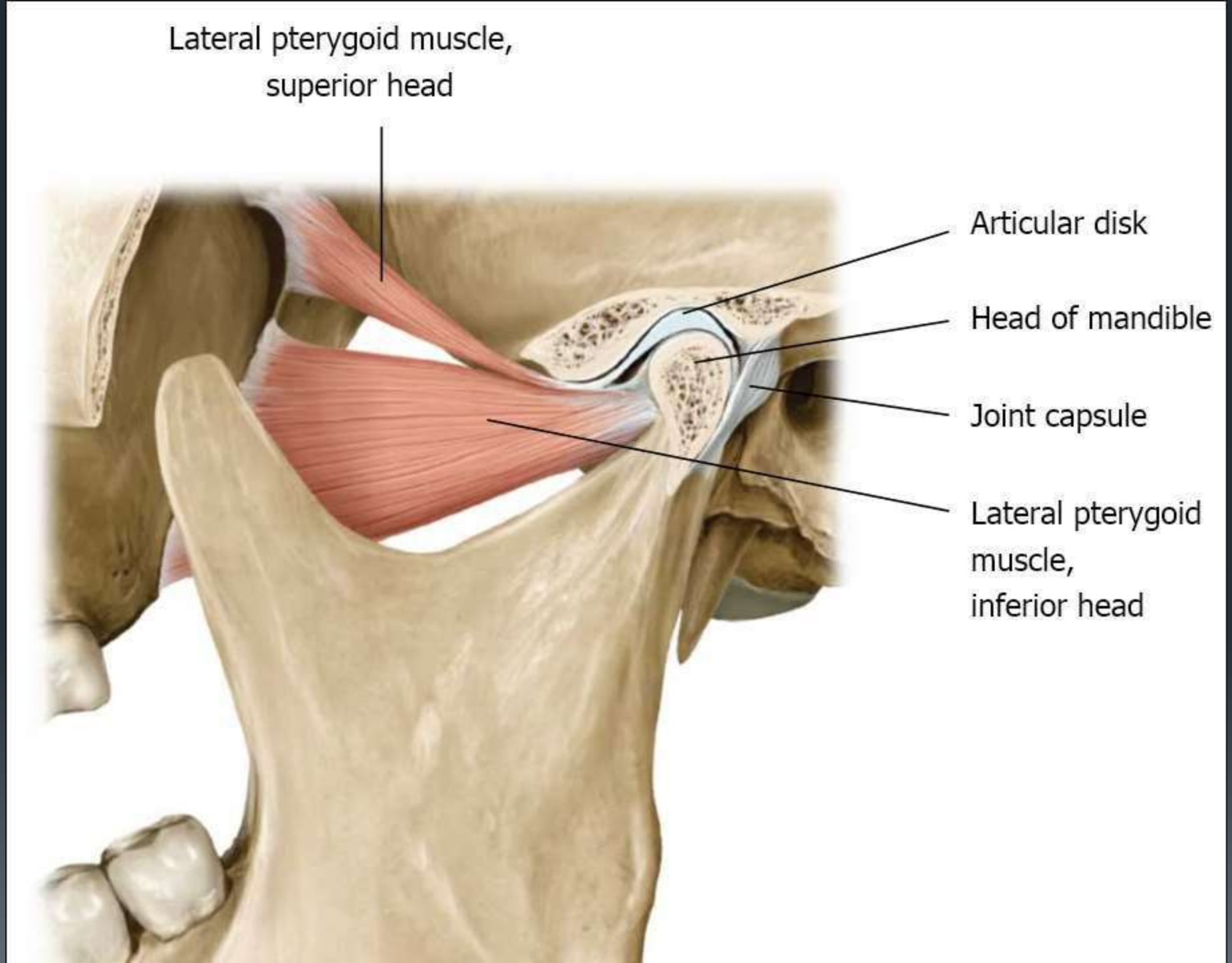


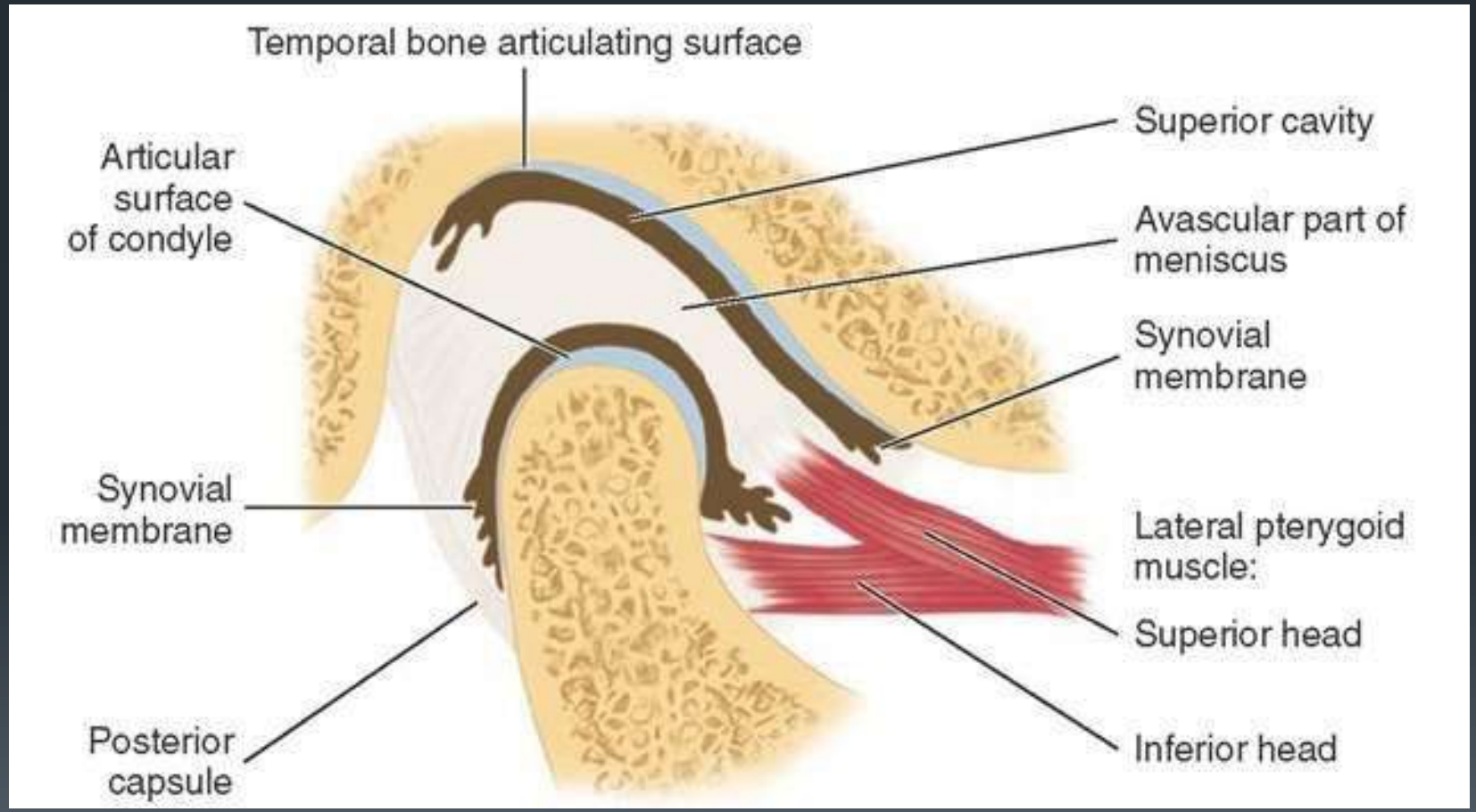


Temporomandibular Disorders

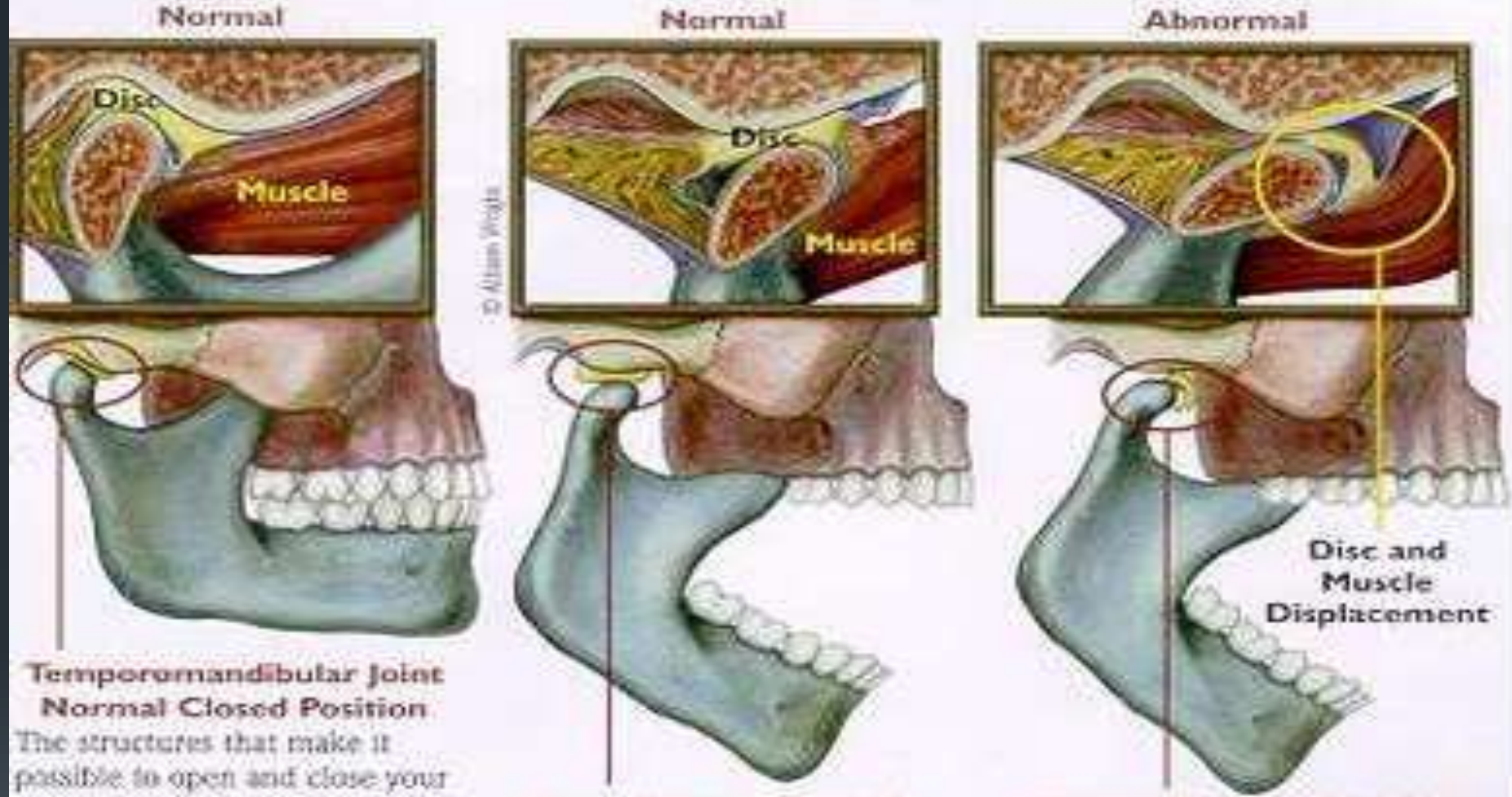
Anatomy of TMJ




Lateral pterygoid muscle, superior head
Articular disk
Head of mandible
Joint capsule
Lateral pterygoid muscle, inferior head


Temporal bone articulating surface
Articular surface of condyle
Synovial membrane
Posterior capsule
Superior cavity
Avascular part of meniscus
Synovial membrane
Lateral pterygoid muscle:
Superior head
Inferior head

Function of the Temporomandibular Joint



Temporomandibular Joint Normal Closed Position

The structures that make it possible to open and close your mouth include the bones, joints, and muscles. When functioning correctly, your jaw-bone is separated from your skull by a soft disc that acts as a cushion when you chew, speak or swallow.

Temporomandibular Joint Normal Open Position

When the joint is functioning properly, the disc stays in place when the jaw is in use, preventing the bony structures from coming in contact.

Temporomandibular Joint Dysfunctioning Open Position

When the joint is not functioning properly, the disc is commonly pulled forward when the jaw is in use, causing the bones of the skull and jaw to grind together.

The evaluation of the patient with temporomandibular pain, dysfunction

- History
- physical examination of the masticatory system
- TMJ radiography.
- Special diagnostic studies should be performed only as indicated and not as routine studies.

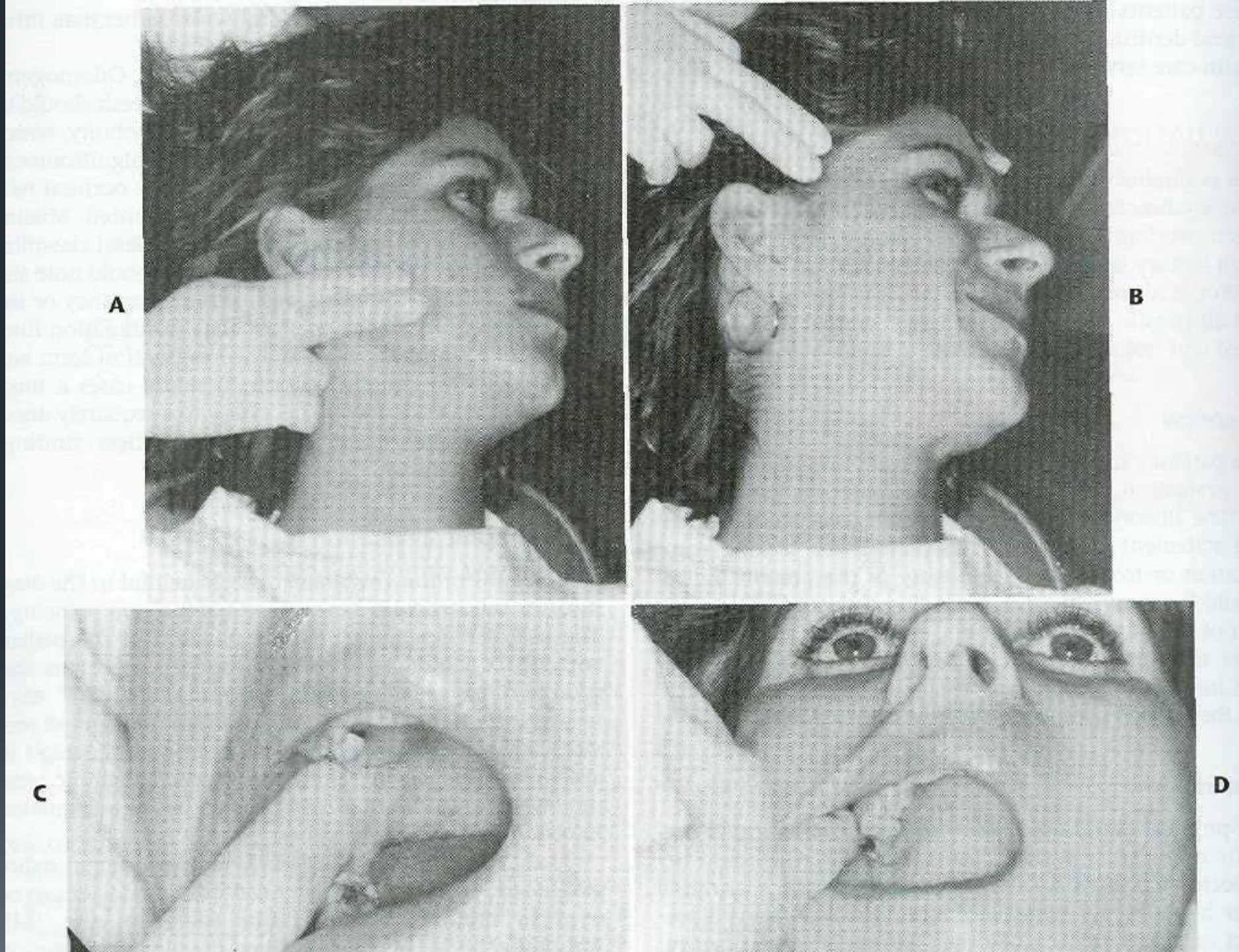
physical examination

1- Evaluation of the entire masticatory system

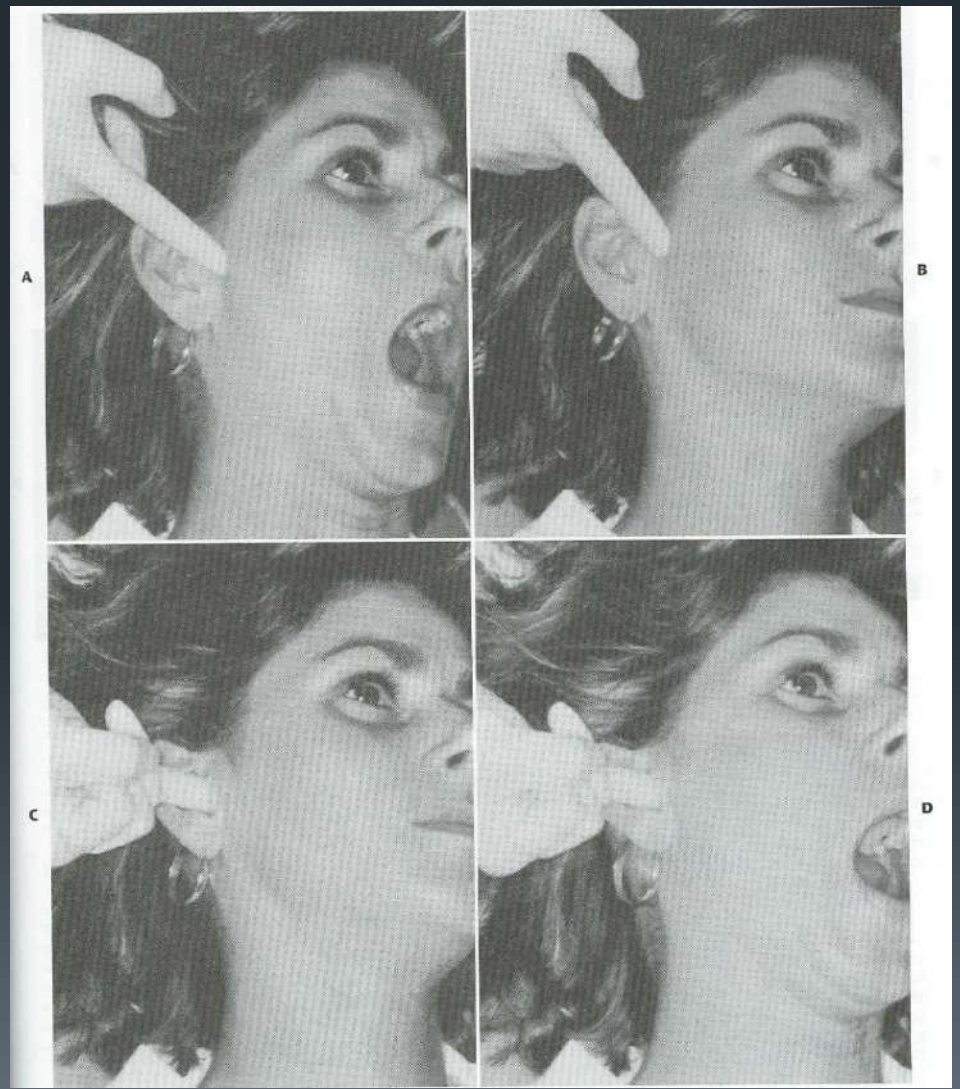
- On inspection: Asymmetry , muscular hypertrophy. The patient should be observed for signs of jaw clenching or other habits.

- On palpation: The muscles should be palpated for the presence of

I. Tenderness,
II. Fasciculations,
III. Spasm
IV. Trigger points

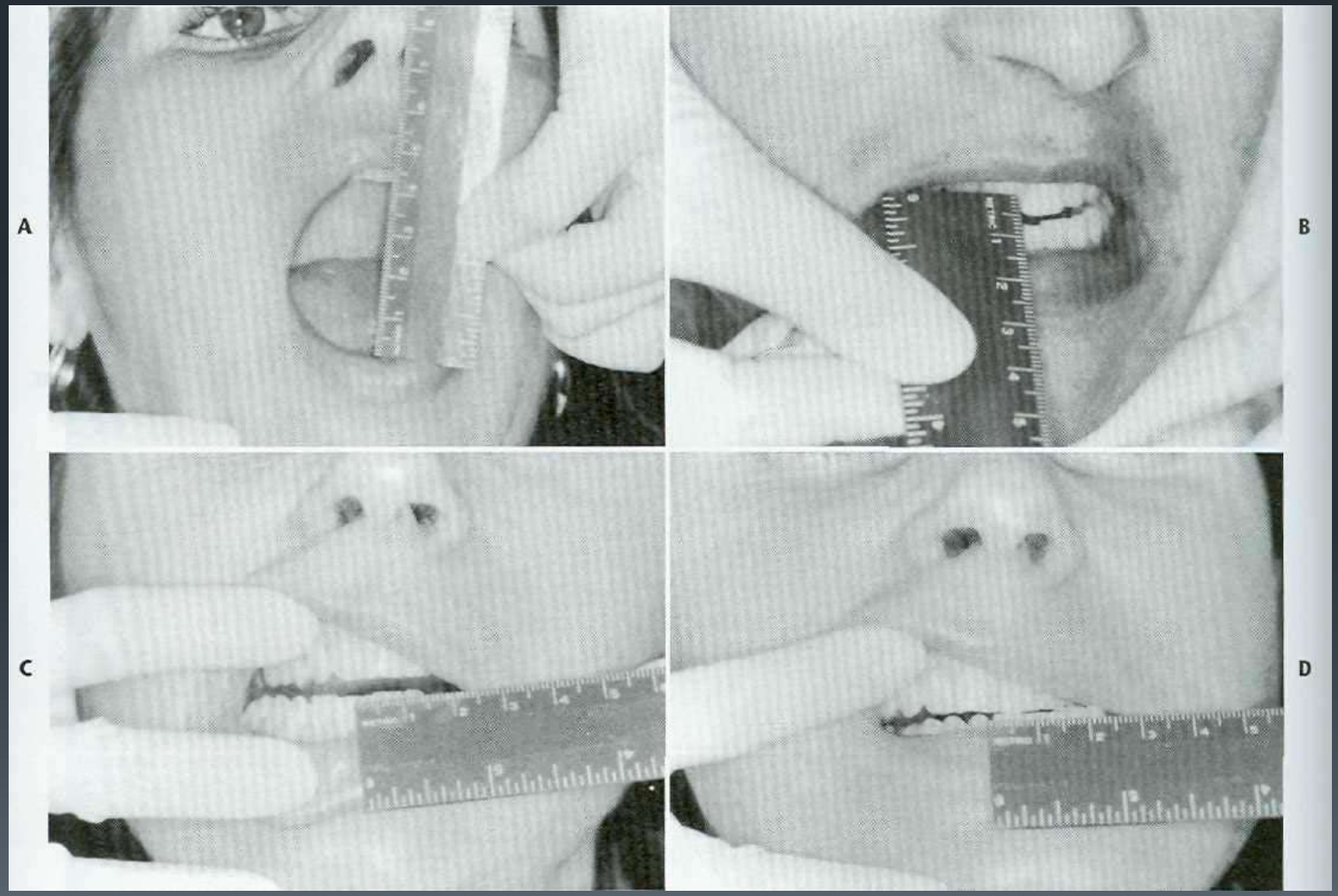

A
B
C
D

2- Examination of joint for noise and tenderness



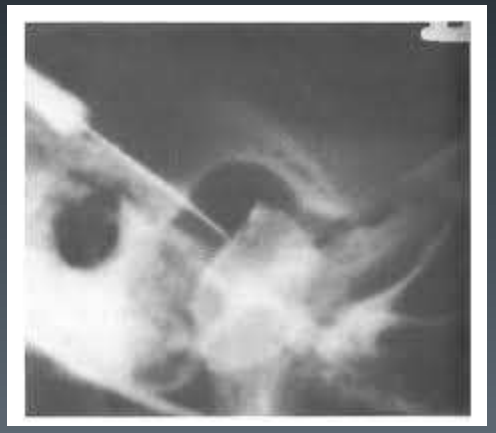
3- Examination: The mandibular range of motion should be determined.

- Normal range of movement of an adult's mandible is about 45 mm vertically (i.e., interincisally) and 10 mm protrusively and laterally

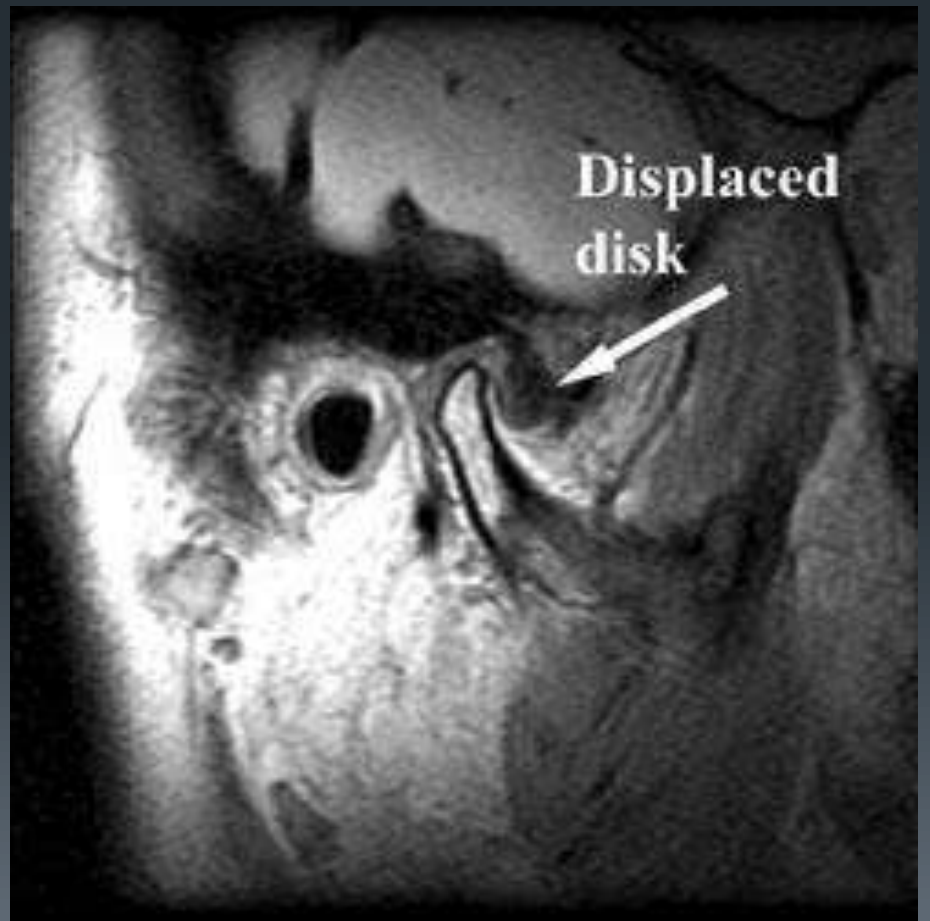


Radiographic Evaluation

1. ***Panoramic radiography:.***
2. ***Temporomandibular joint arthrography.*** Arthrography involves the injection of contrast material into the inferior or superior spaces of a joint, after which the joint is radiographed. This technique also demonstrates the presence of perforations and adhesions of the disk or its attachments.
3. ***Computed tomography.*** provide the most accurate radiographic assessment of the bony components of the joint .
4. ***Magnetic resonance imaging.*** The most effective diagnostic imaging technique to evaluate TMJ soft tissues is magnetic resonance imaging **(MRI)** (disc morphology and position)
5. ***Nuclear imaging***



MRI



CLASSIFICATION OF TEMPOROMANDIBULAR DISORDERS

- Myofascial Pain
- Disk Displacement Disorders
- Degenerative Joint Disease
- Systemic Arthritic Conditions
- Chronic Recurrent Dislocation
- Ankylosis
- Neoplasia
- infections

Myofascial pain and dysfunction (MPD)

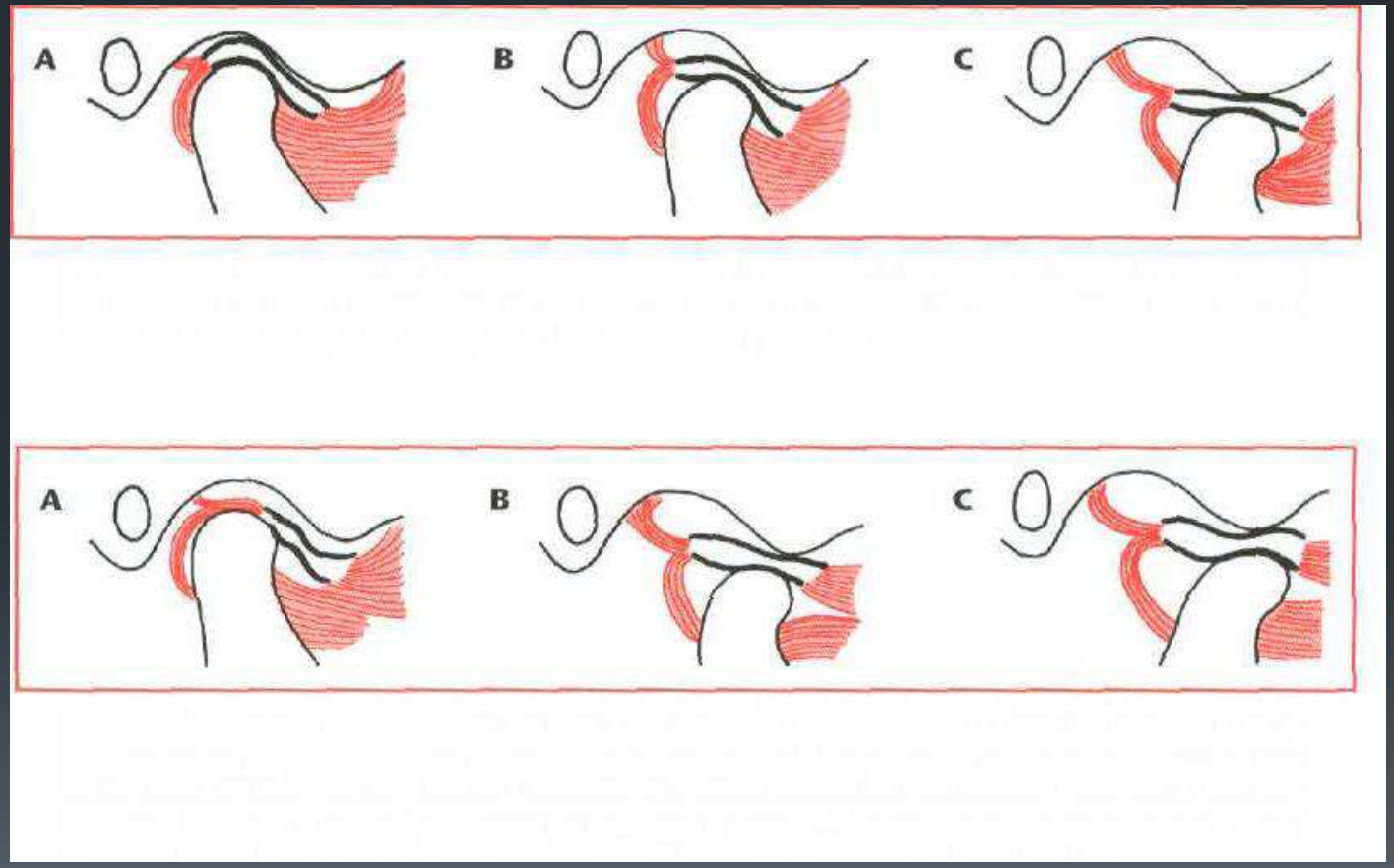
- Is the most common cause of masticatory pain and limited function
- The source of the pain and dysfunction is muscular
- Frequently but not always associated clenching or bruxism.
- Causes: stress, malocclusion, disc displacement
- C/P:

I. diffuse, poorly localized, preauricular pain
II. Tenderness of muscles of mastication
III. Decreased jaw opening with pain during functions such as chewing
IV. Headaches
V. joint noises are usually not present
VI. Radiographs of the TMJs are usually normal

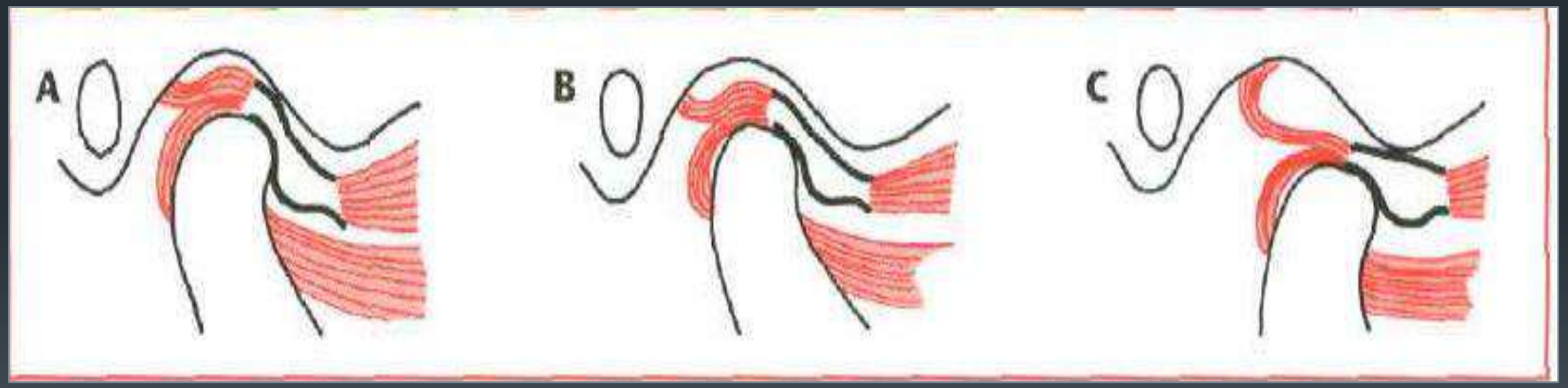
Disk Displacement Disorders

Normal TMJ

Disc displacement with reduction



Non reducing disc displacement



Disc displacement with reduction

1. Joint noise: Clicking during opening the mouth. If it occurs during opening and closing, it is called reciprocal clicking
2. Painless but Joint tenderness and muscle tenderness may be seen
3. Maximal opening can be normal or slightly limited
4. Plain TMJ radiography: normal

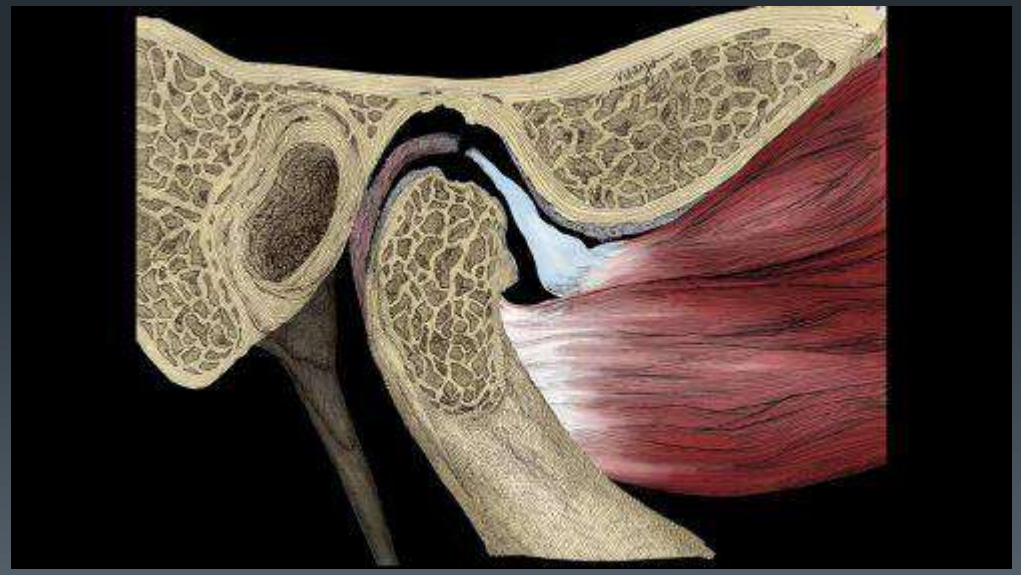
Disc displacement without reduction

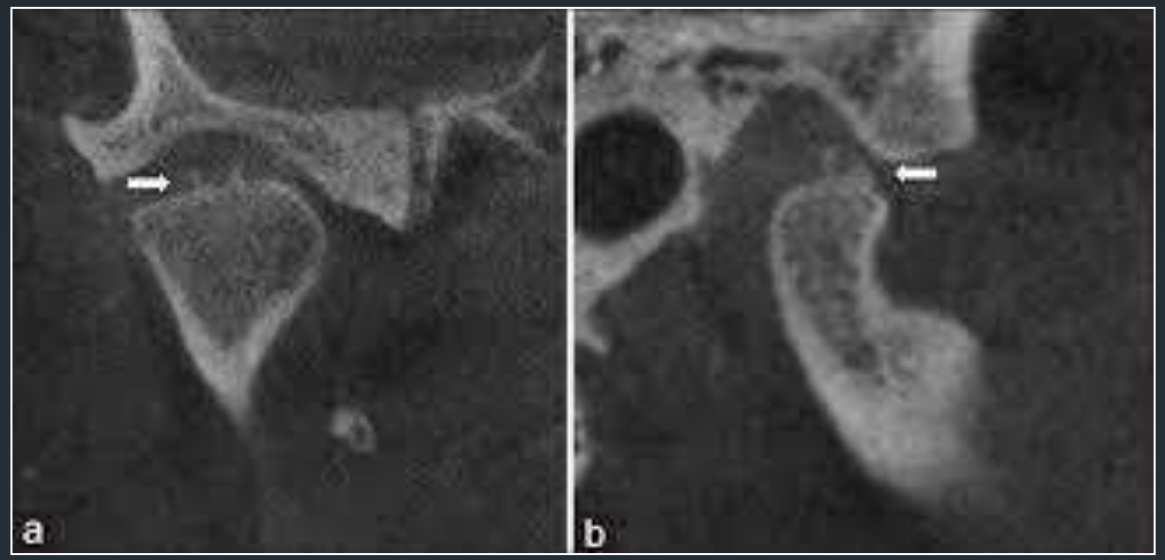
1. No clicking occurs, because they are unable to translate the condyle over the posterior aspect of the disk.
2. Joint tenderness and muscle tenderness
3. Restricted opening, deviation to the affected side, and decreased lateral excursions to the contralateral side
4. Plain Radiographic evaluation: normal

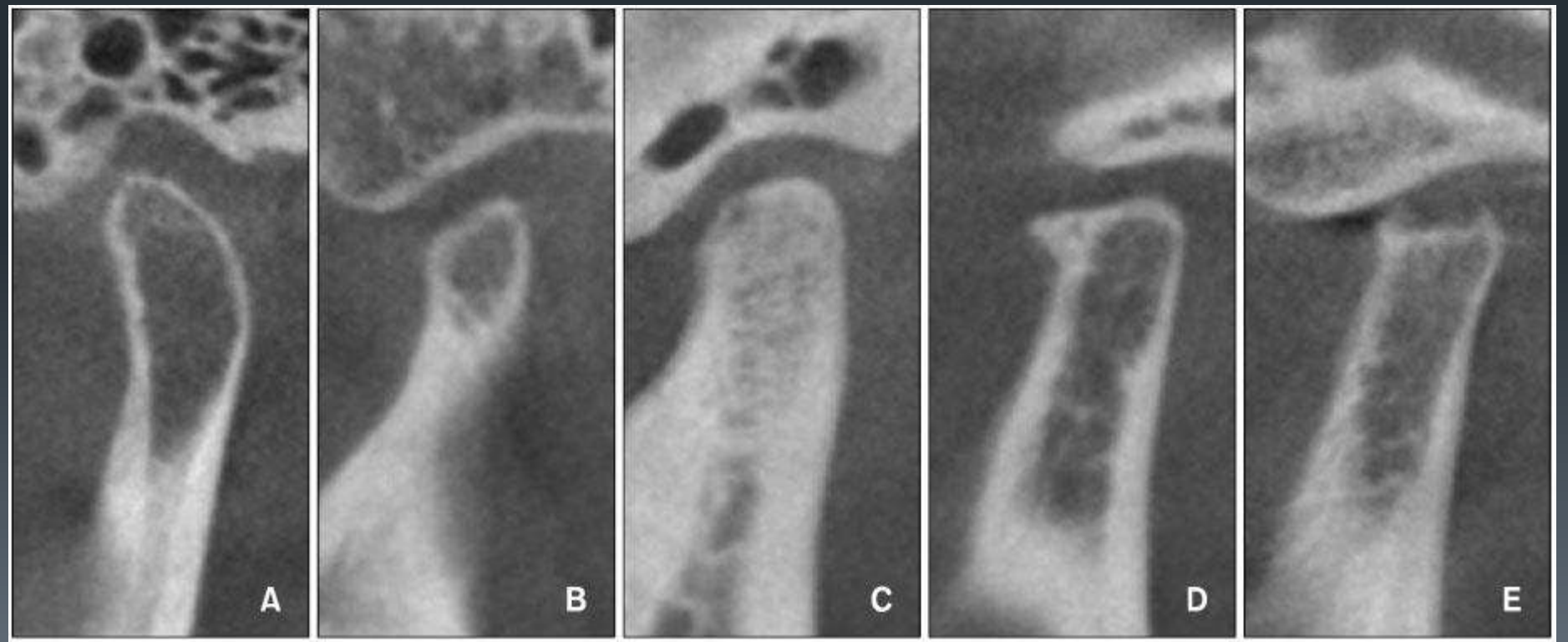
Degenerative Joint Disease (Arthrosis, Osteoarthritis

- includes a variety of anatomic findings, including

1. irregular, perforated, or severely damaged disks
2. articular surface abnormalities, such as articular surface flattening, erosions, or osteophyte formation



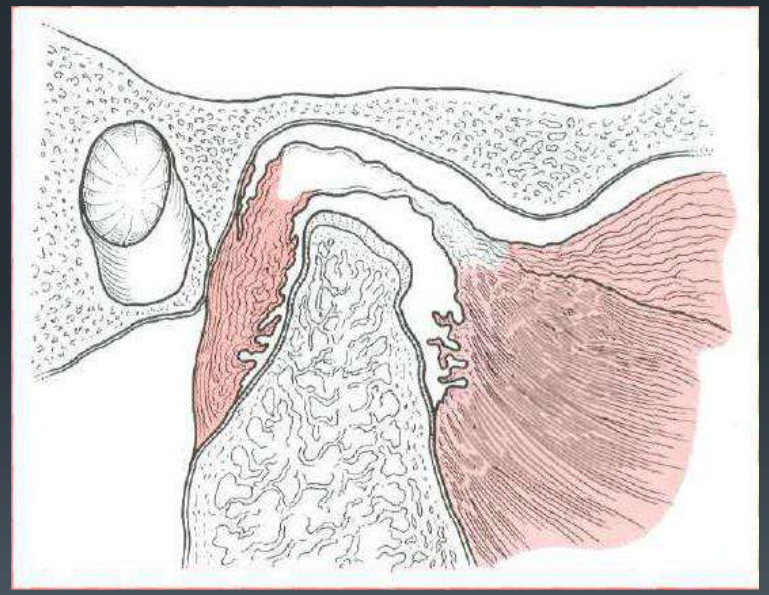

a
b


A
B
C
D
E

DJD

- pain associated with
- clicking or crepitus, located directly over the TMJ.
- Usually, an obvious limitation of opening is present
- Radiographic findings : exhibit decreased joint space, surface erosions, osteophytes, and flattening of the condylar head

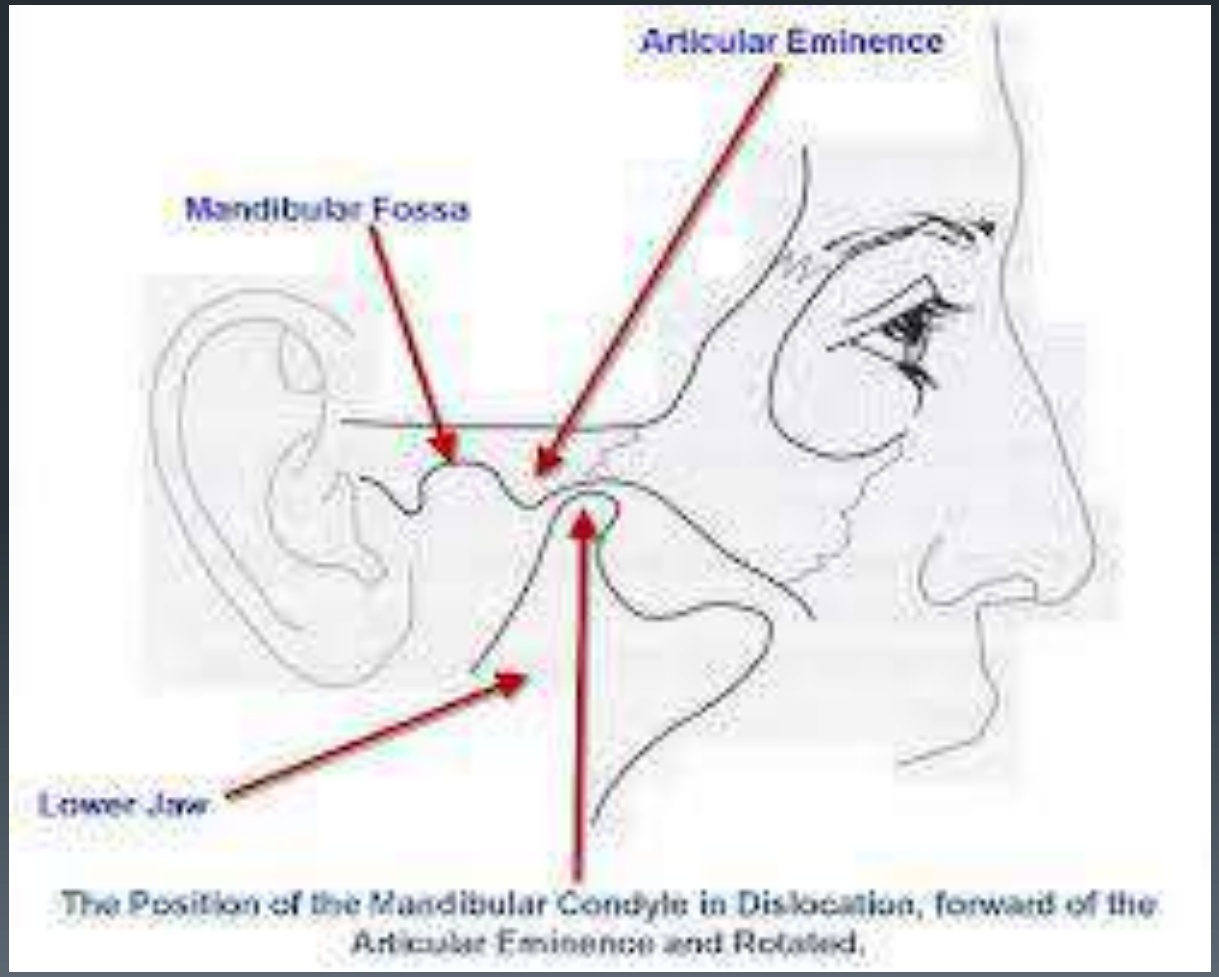
Rheumatoid arthritis.



- inflammatory process results in abnormal proliferation of synovial tissue in a so-called pannus formation.
- Same DJD Features but it is bilateral and associated with systemic manifestations
- ESR, RF level are elevated

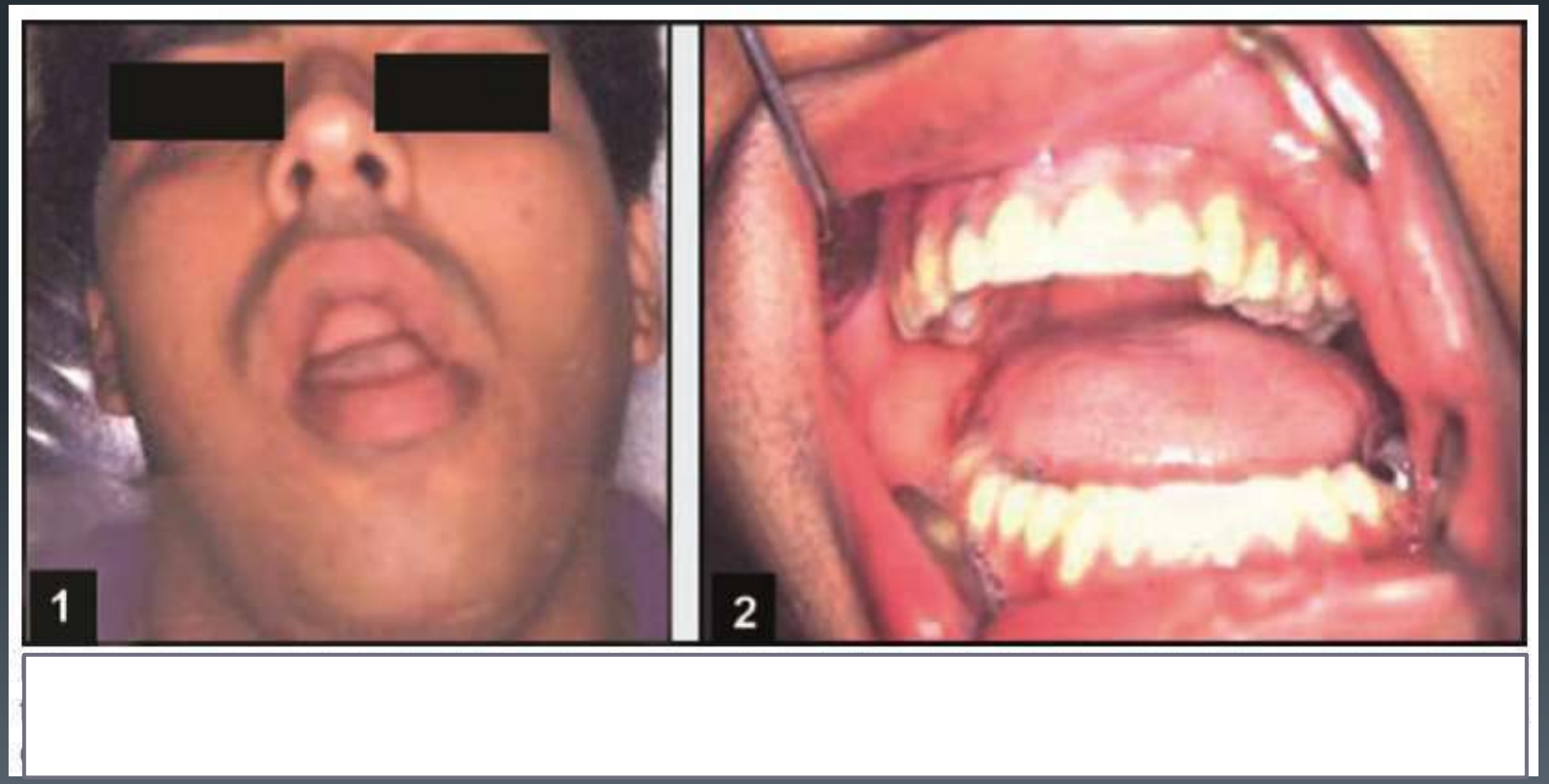
Chronic Recurrent Dislocation

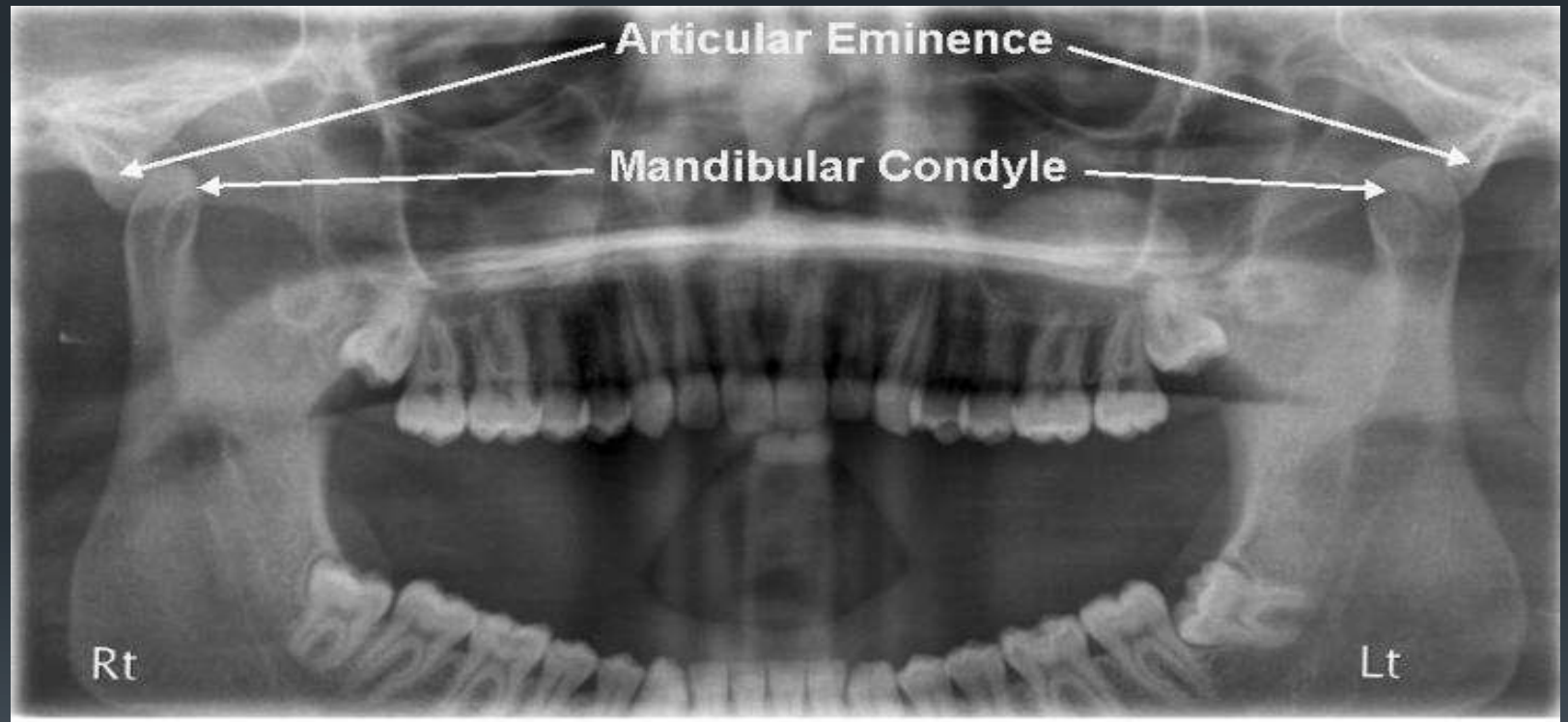
- Characterized by the condyle sliding over the articular eminence and becomes locked in that position .



The Position of the Mandibular Condyle in Dislocation, forward of the Articular Eminence and Rotated.

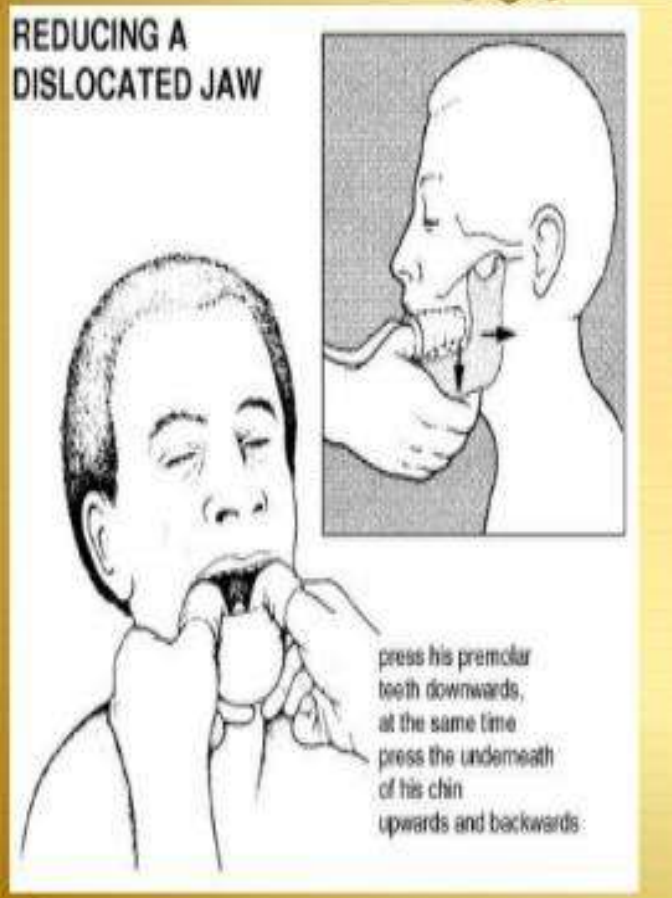
- Dislocation may be unilateral or bilateral and may occur spontaneously after opening the mouth widely, such as when yawning, eating, or during a dental procedure
- painful and is often associated with severe muscular spasms
- Dislocations should be reduced as soon as possible.

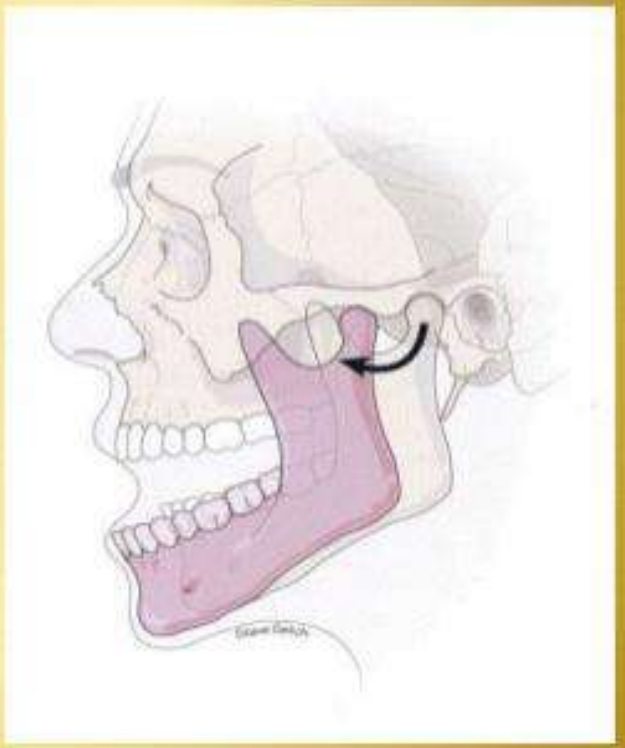


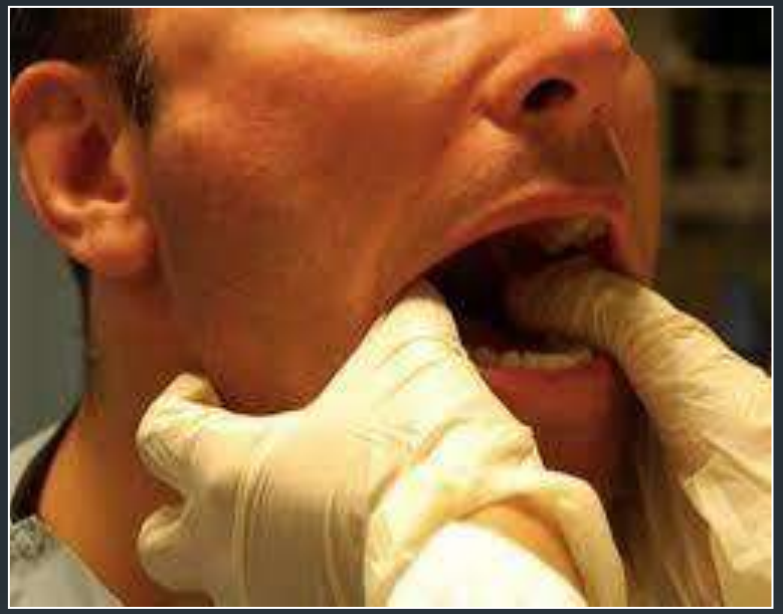


X-Ray showing a Bilateral Dislocation of the Mandible.
Notice how open the patient's mouth is.

TMJ DISLOCATION….



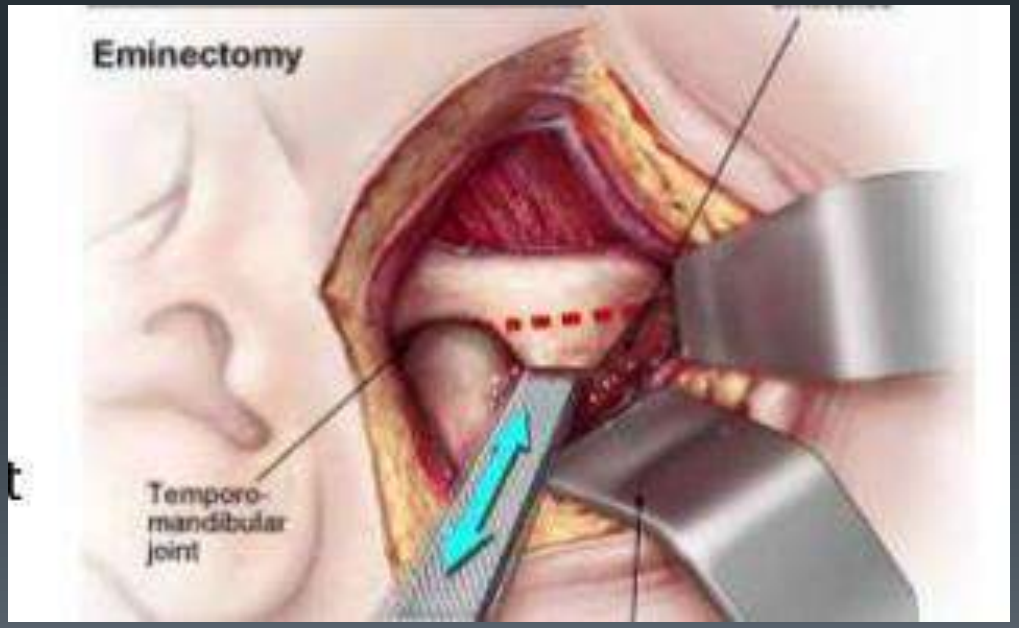


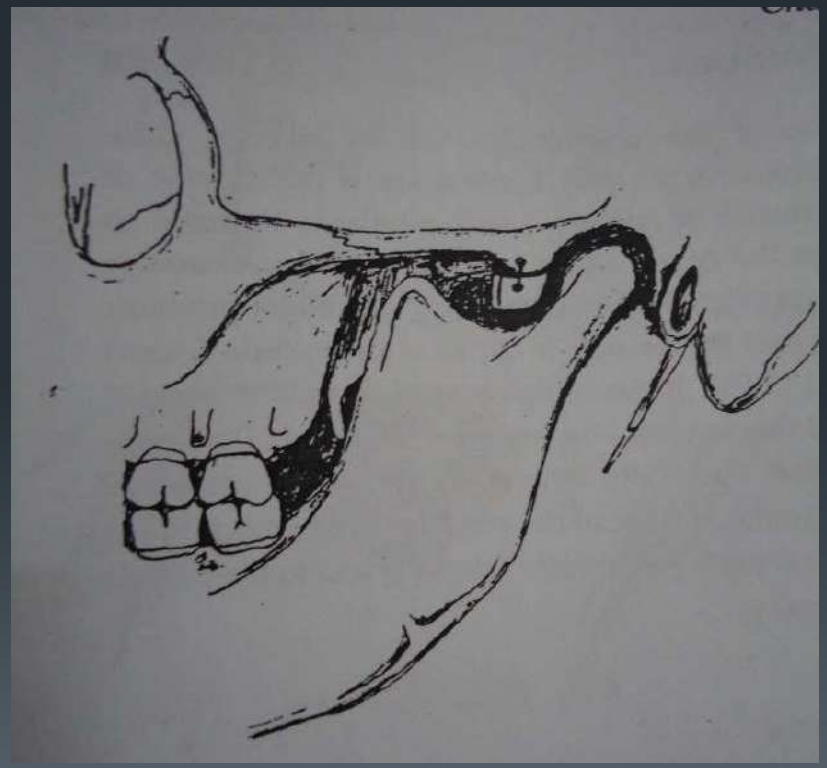


Treatment of chronic recurrent dislocation

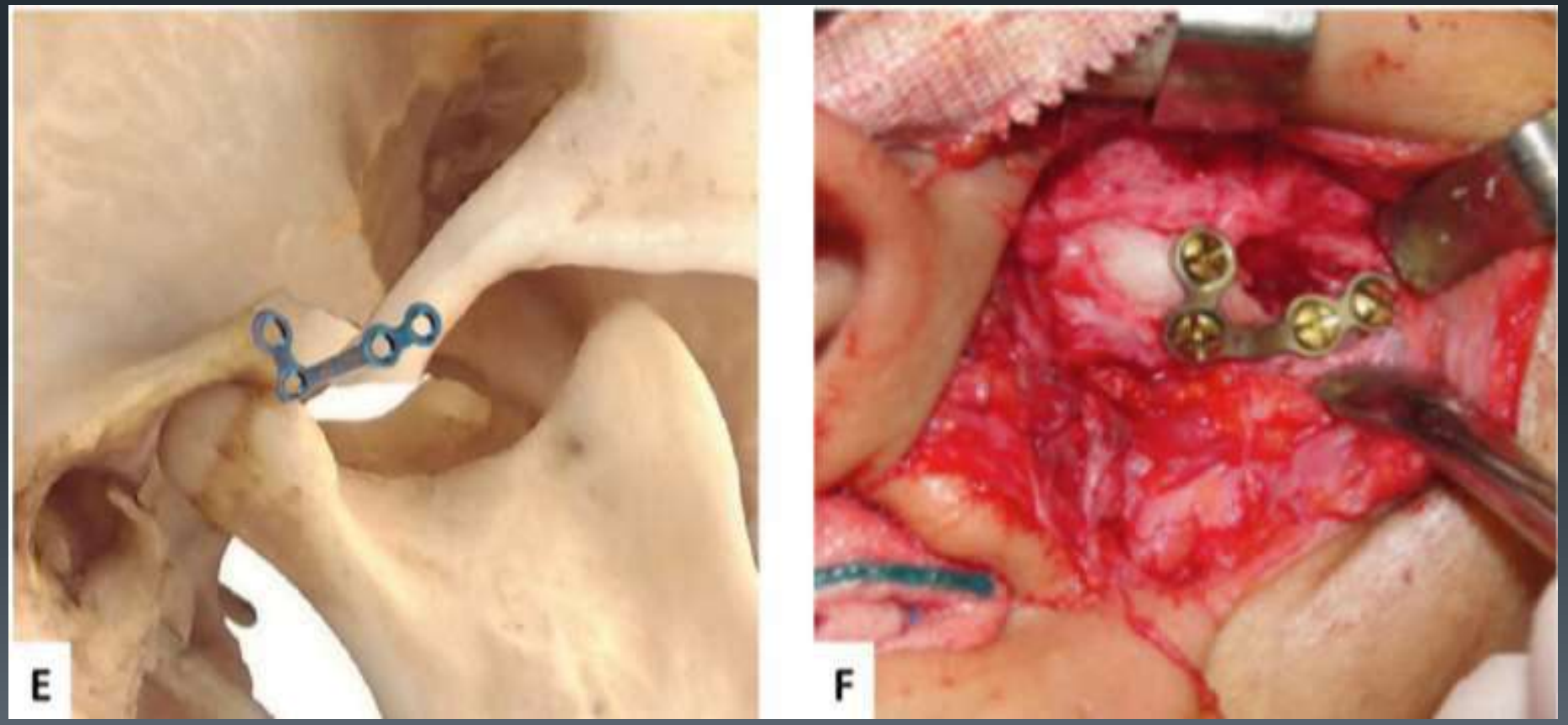
A) Conservatives methods: Injection of sclerosing agents like alcohol

B) Surgical methods:



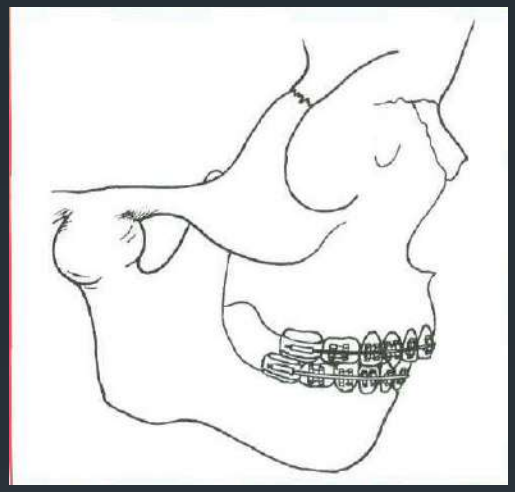


Dautrey procedure



Ankylosis

- Bony fusion between the mandibular condyle and skull base involves the TMJ.
- MAY BE BONY, fibrous or fibro-osseous
- May be intracapsular or extracapsular ankylosis



- Trauma
- Infection
- Previous surgery

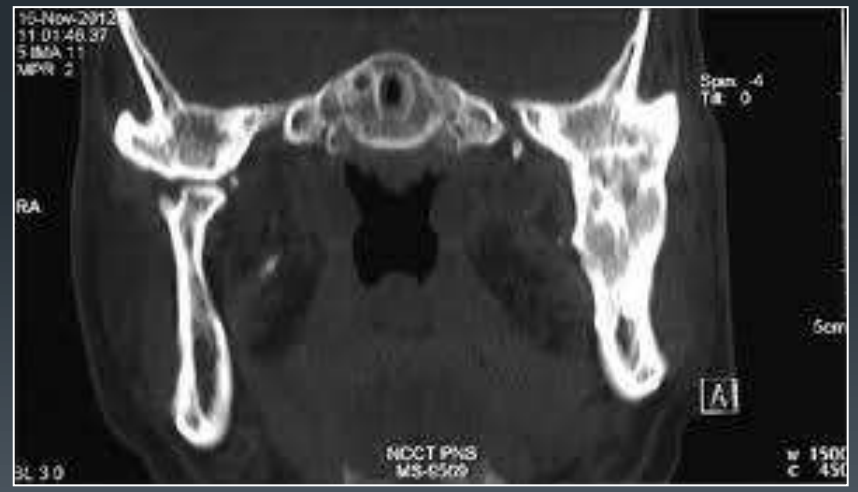
coronoid HP

infection around temporalis

zygomatic arch fracture

Features

- severe restriction of maximal opening,
- deviation to the affected side, and
- decreased lateral excursions to the contralateral side



Treatment of TMJ disorders

- **Non -surgical Treatment: must be applied to all patient**
- **Surgery**

Non-surgical treatment: Education and behavior modification
medications
splints
physical therapy

Diet

- Decreases muscle activity and loading forces on temporomandibular joints
- Controls range of motion—hinge and sliding
- Ranges from liquid diet to elimination of hard chewy food; involves cutting food into small pieces
- Eliminates gum chewing

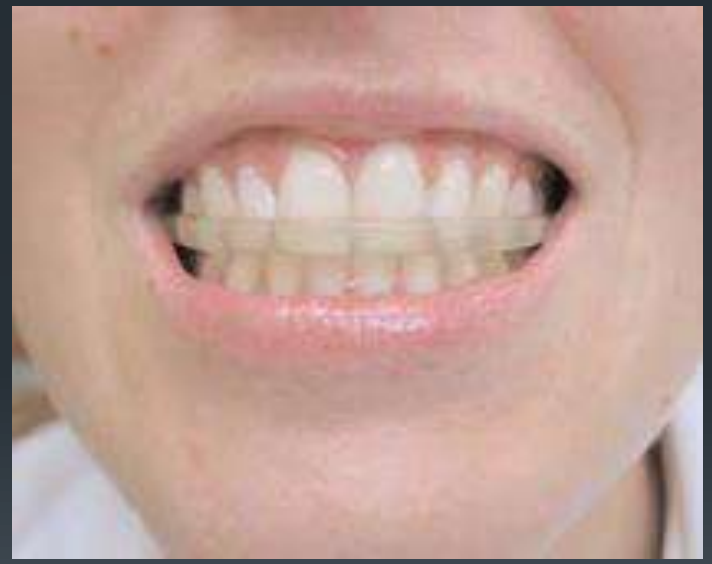
Medication

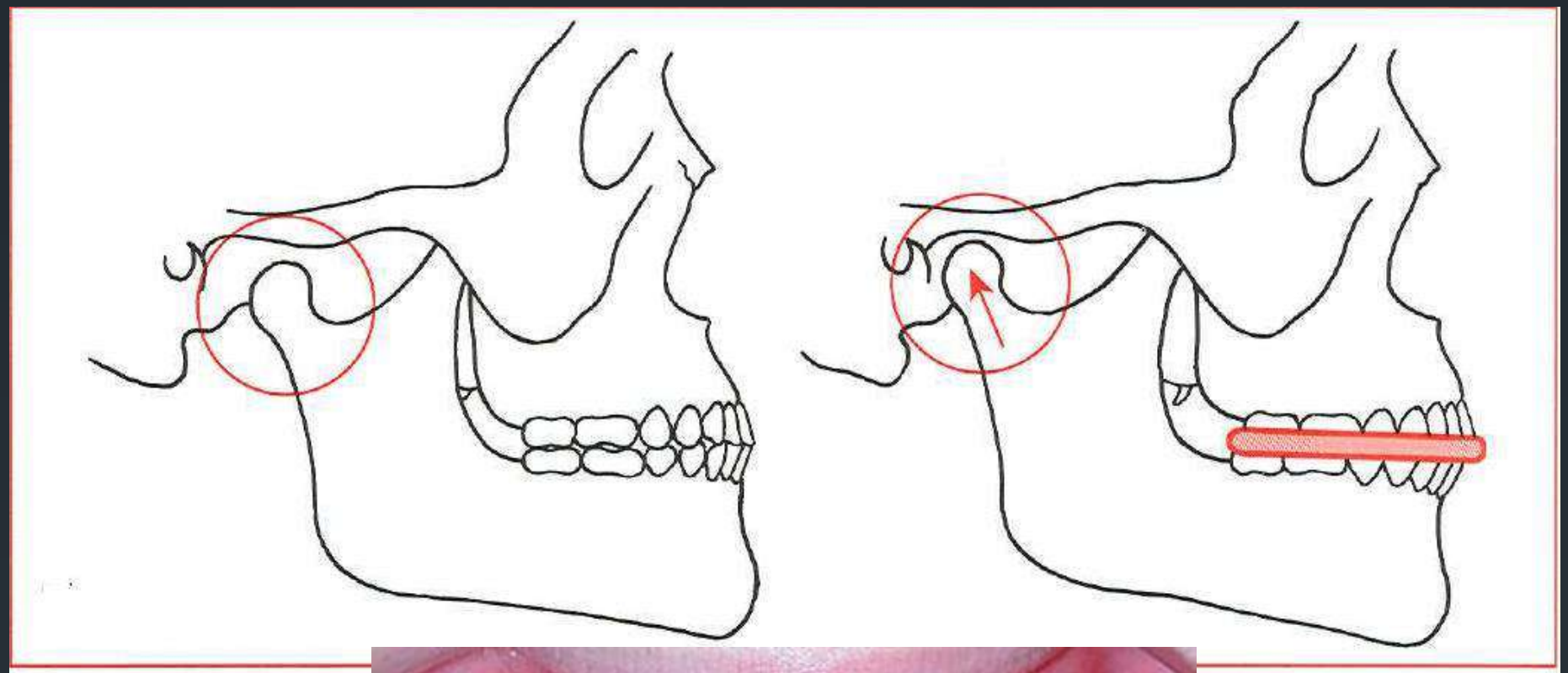
- (1) NSAIDs: meloxicam , piroxicam
- (2) analgesics: panadol, codeine
- (3) muscular relaxants: diazepam, cyclobenzaprine, myogesic
- (4) tricyclic antidepressants: amitryptaline
- .(5) anxiolytics.
- (6) local anesthetics

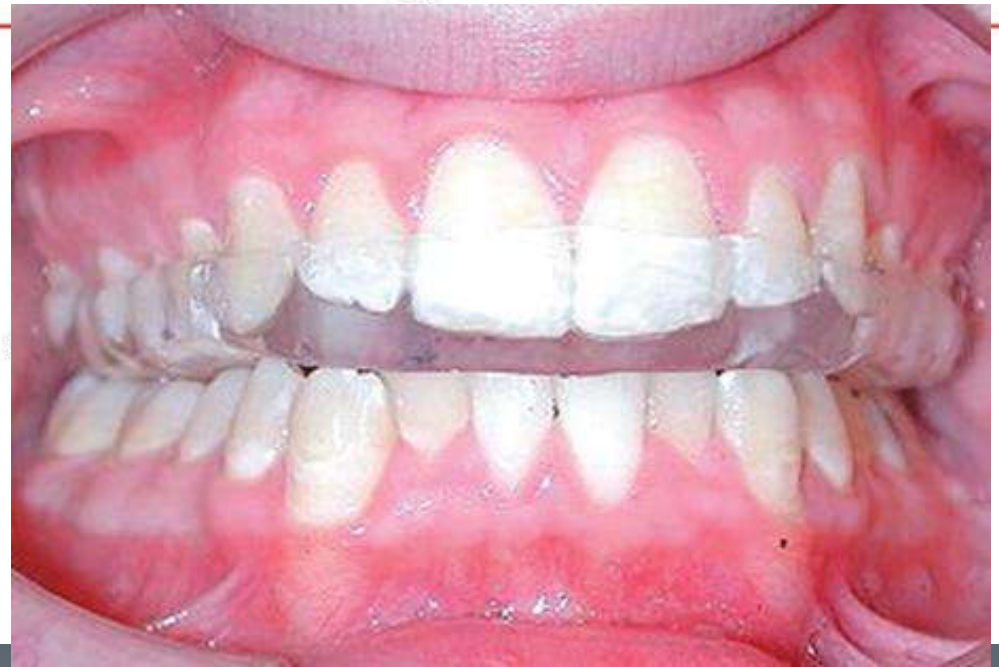
Table 48-13 Physical Therapy
Home Treatment Program (good for mild acute symptoms)
Soft diet
Decrease function
Heat/ice packs
Jaw/tongue posture opening exercise
Lateral jaw movement
Control passive motion (ie, Therabite)
Office Treatment (reduction of pain and inflammation)
Ultrasonography
Transcutaneous electrical nerve stimulation
Range of motion
Soft tissue manipulation
Trigger point injections
Acupuncture (reestablishing proper energy flow by adding electric current or heat to the placed acupuncture needle)

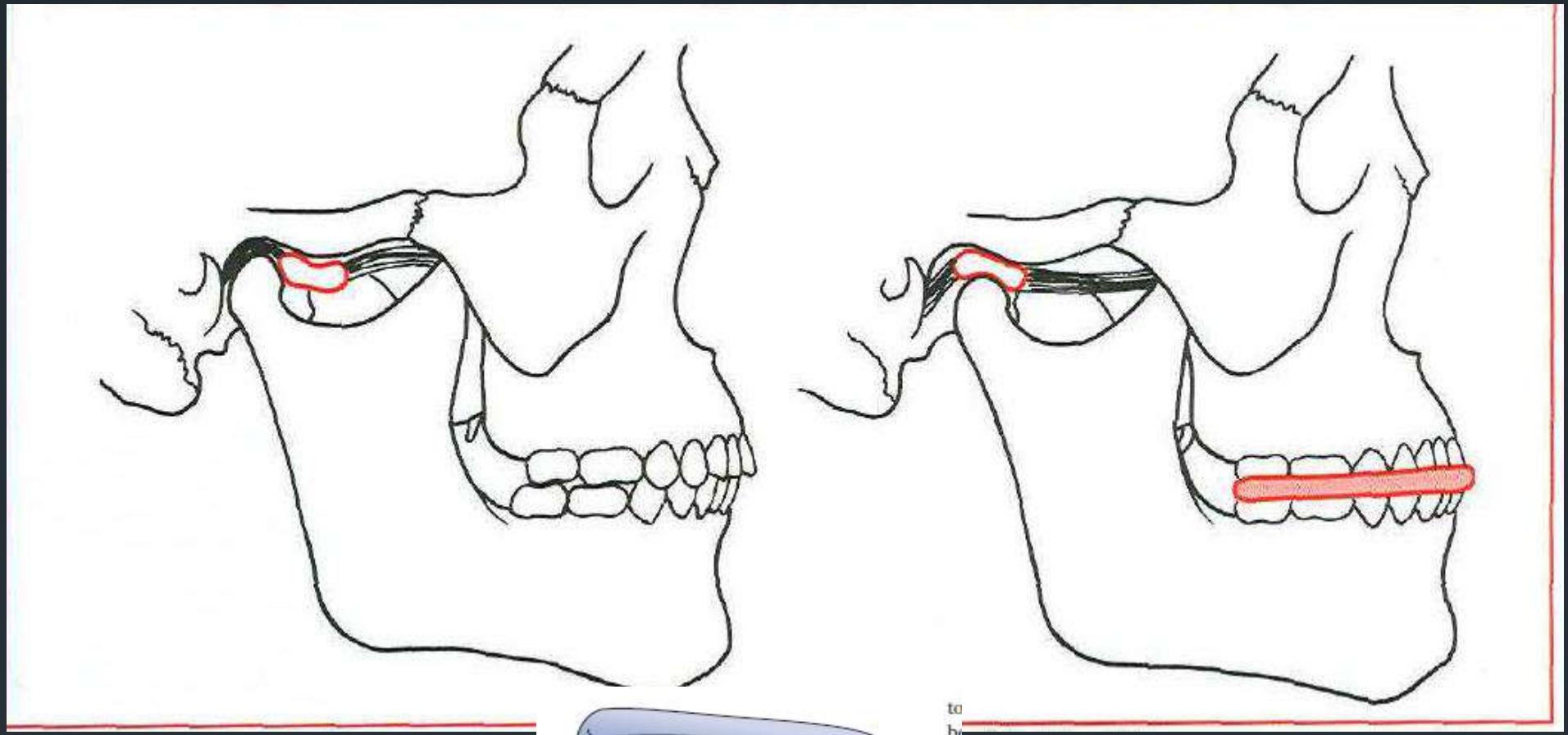
FIGURE 48-2 *Patient using passive jaw exercise device to improve interincisal opening and to break any fibrous bands. (This Ther-*

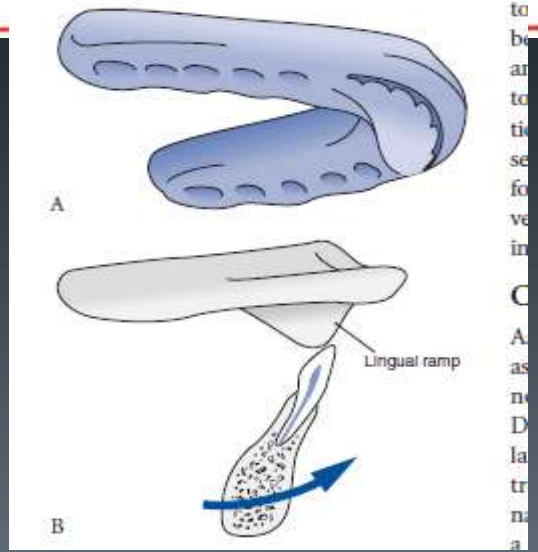
Splints










A
Lingual ramp
B

TEMPOROMADIBULAR JOINT SURGERY

1- Arthrocentesis:.

2-Arthroscopy:

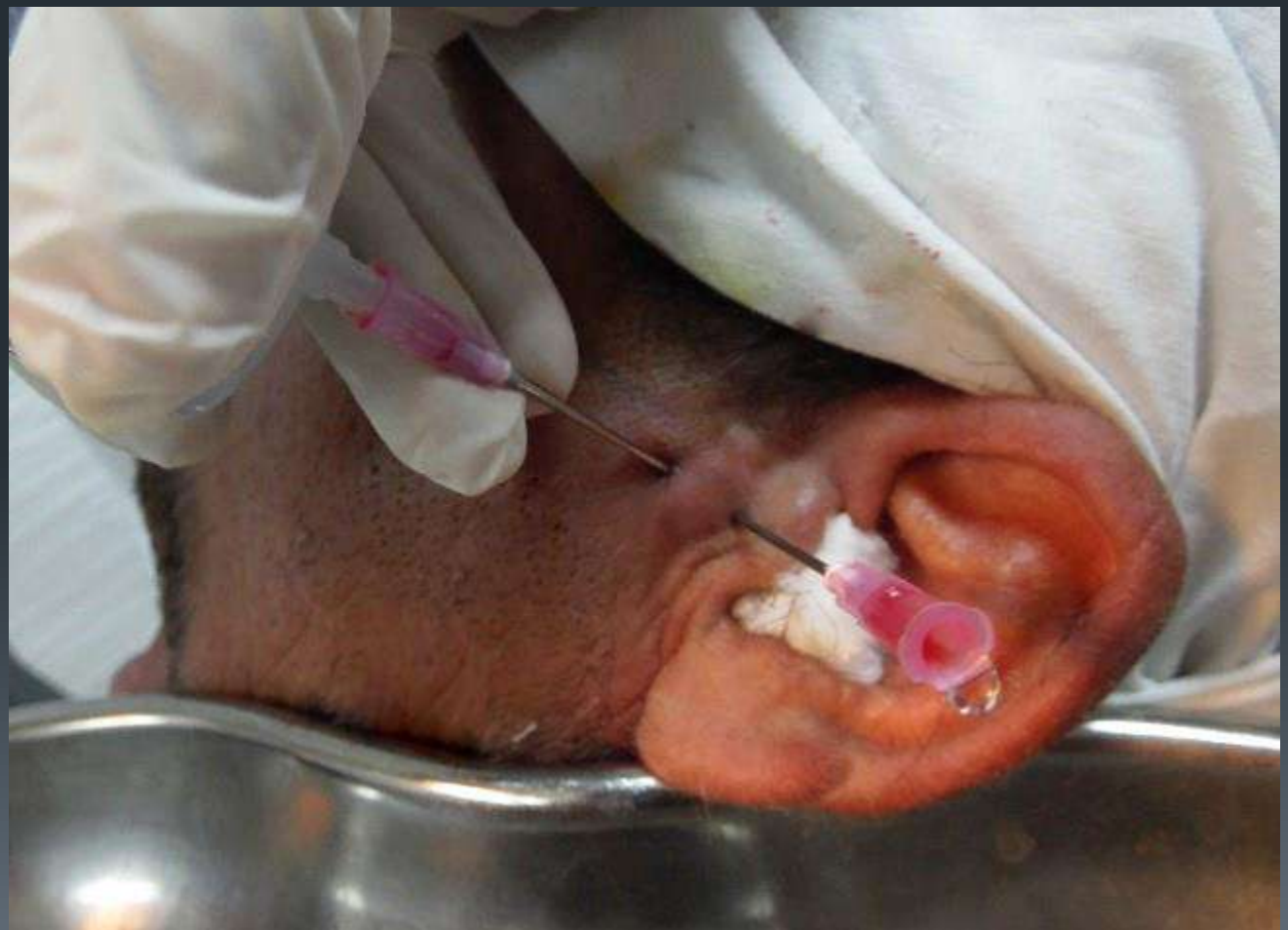
3-Disk-Repositioning Surgery

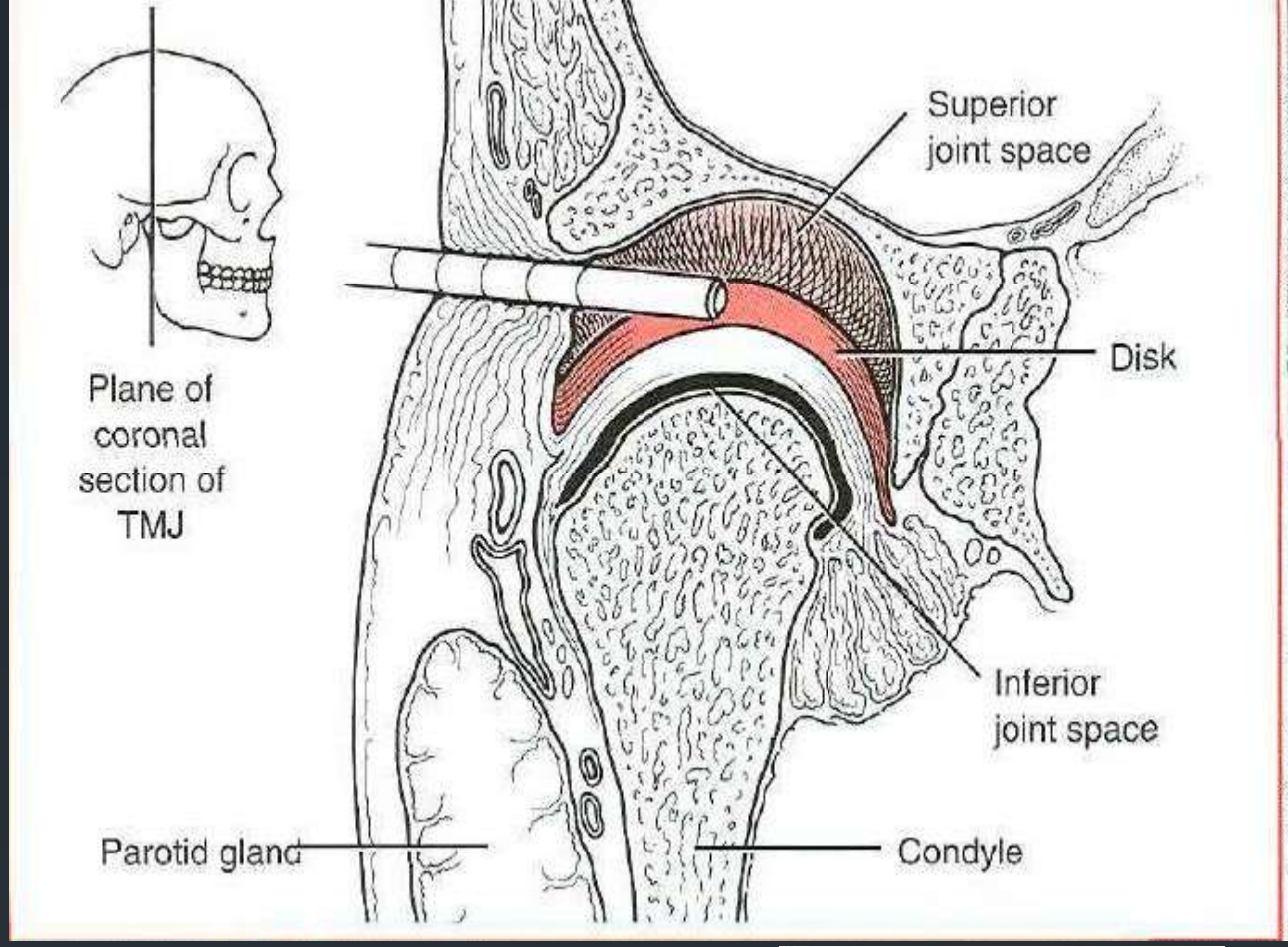
4-Disk Repair or Removal

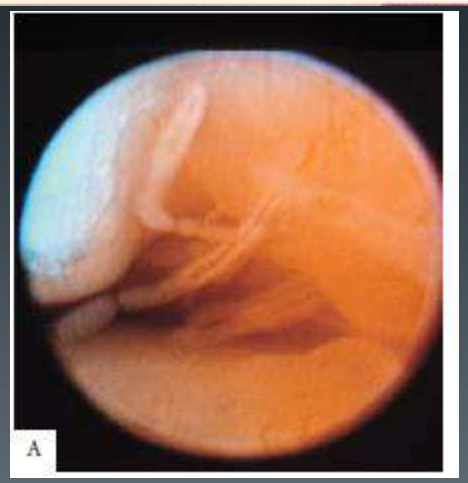
5-Condylotomy

6-Total Joint Replacement

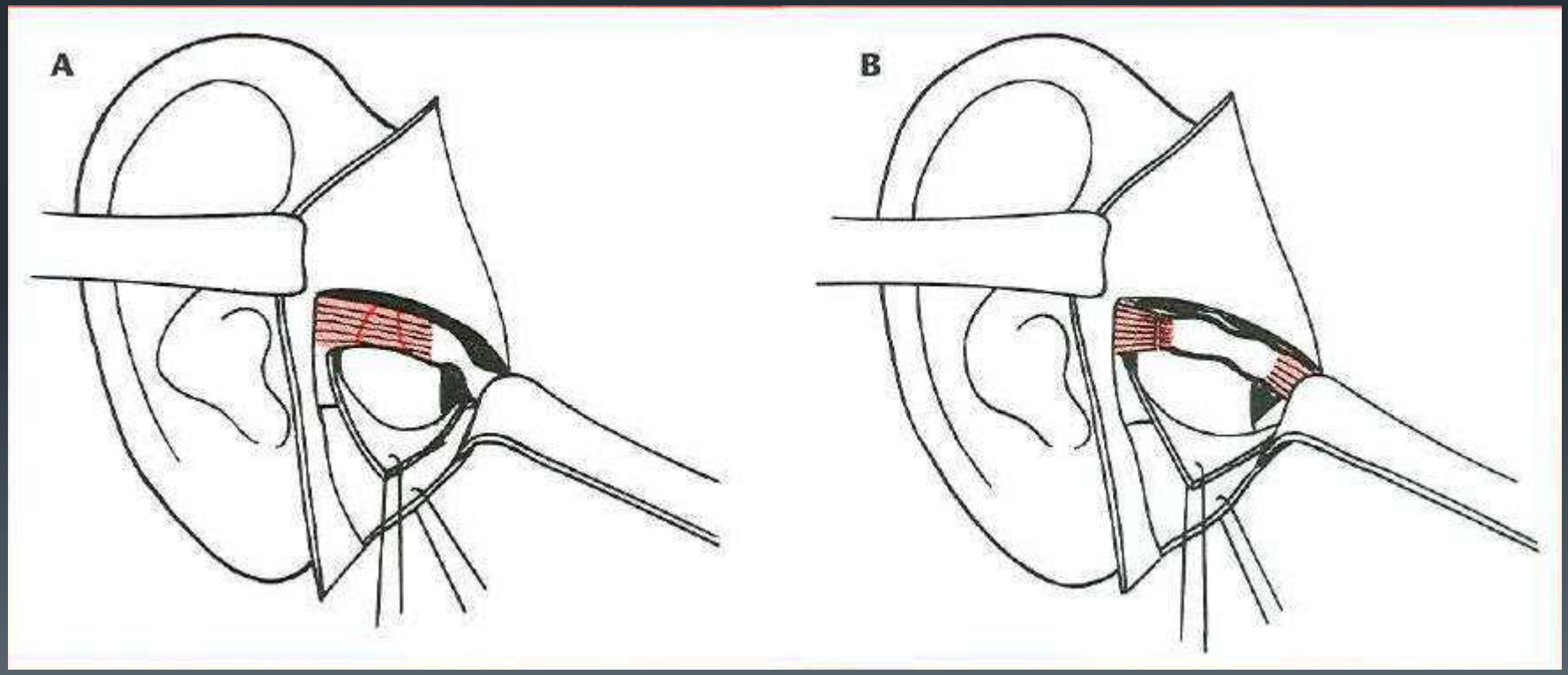
Arthrocentesis



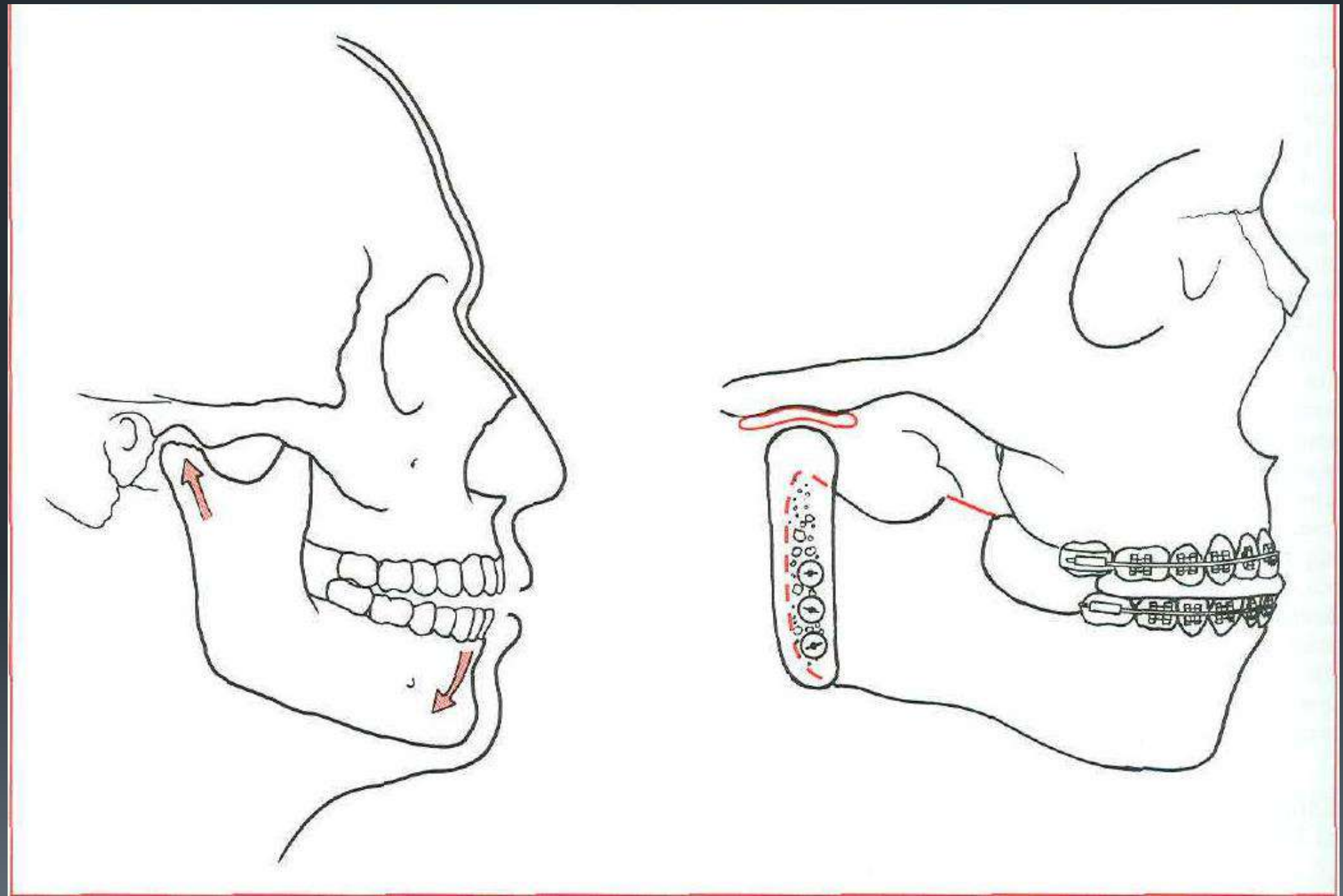

Plane of coronal section of TMJ
Superior joint space
Disk
Inferior joint space
Parotid gland
Condyle


A

Disk reposition surgery



Total joint replacement



Diagnostic Imaging

Maxillofacial imaging has evolved in parallel with the development of newer imaging technologies. Traditional plain radiography and dental imaging is now frequently supplemented by cross-sectional modalities, such as computed tomography (CT), magnetic resonance imaging (MRI) and ultrasound, together with functional imaging modalities, such as positron emission tomography (PET). It is important to be aware of the benefits and limitations of such imaging examinations such that they are applied to the appropriate clinical scenario.

Radiation Protection

Some imaging investigations use ionizing radiation which has the potential to result in biological damage. The aim of radiation protection is to provide a safe environment for the worker and patient. The Ionising Radiation (Medical Exposure) Regulations 2000 (IRMER) lay down basic measures required for protection against the harmful effects of medical radiation exposure. There are duties of the employer who provides a framework within exposures which take place, the operator who carries out the exposure, the practitioner who justifies the exposure and the referrer who requests the exposure. Key principles are that the examination should be of sufficient benefit to justify radiation exposure, that dose is optimized by the ALARA (as low as is reasonably achievable) principle and that dose limits should be recorded

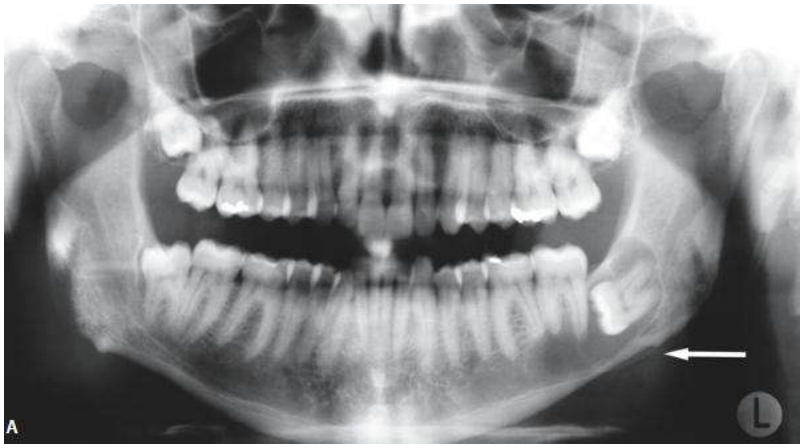
Plain Radiographs

X-rays are produced by a point source and, after passing through the body part of interest, are detected by nonscreen (dental radiography) or intensifying screen/film combinations (extraoral radiography). Selected

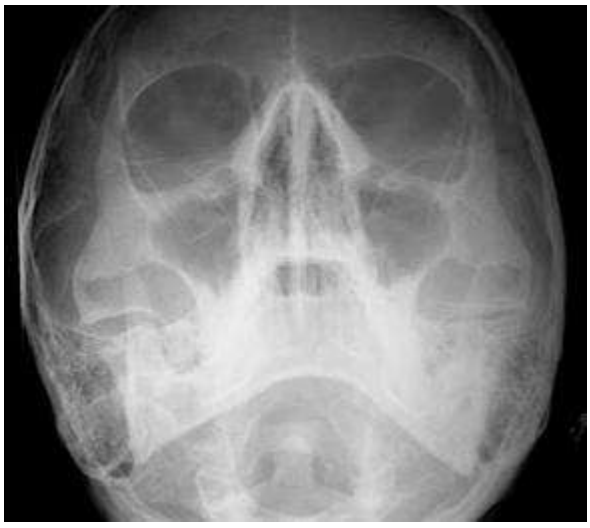
facial radiographic views are listed below. Tomography refers to a technique whereby the x-ray source and film move during the exposure. The aim is to demonstrate only a section which is in focus, whereas structures outside this section are blurred. Applications include conventional dental panoramic tomography, tomograms of the temporomandibular joints and mandibular tomograms for implant planning.

	Radiographic view	Comment
Mid and upper third	Occipito-frontal (OF) 15-20 (Caldwell view)	Used to visualize upper third of face
	OF 25 (modified Caldwell view)	Superior visualization of orbital floor relative to Caldwell view
	Occipito-mental (OM)	Used to visualize mid-third of the face
	OM 10	Less obscuration of maxillary antrum than an OM view
	OM 30	Superior view of malar arches and inferior orbital margins. Preferable to submentovertical (SMV) view
	Lateral	Supplementary for central midface injury
Lower third	Postero-anterior (PA) mandible	
	Lateral oblique	Replaces OPG if not available or impractical
	Reverse Townes	Better visualizes mandibular condyles
	Orthopantomogram (OPG)	

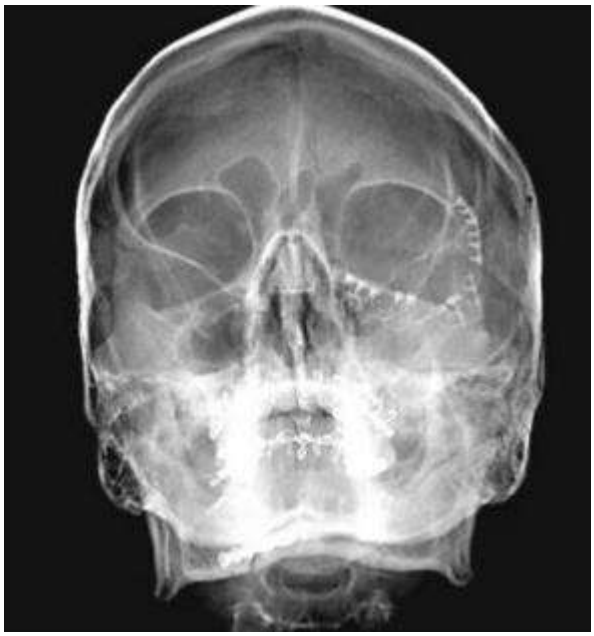
Digital radiography units (using digital receptors to intercept the x-ray beam rather than intensifying screens) are now replacing conventional units. This allows transmission of data to image processing and storage devices, as well as communications networks**.**



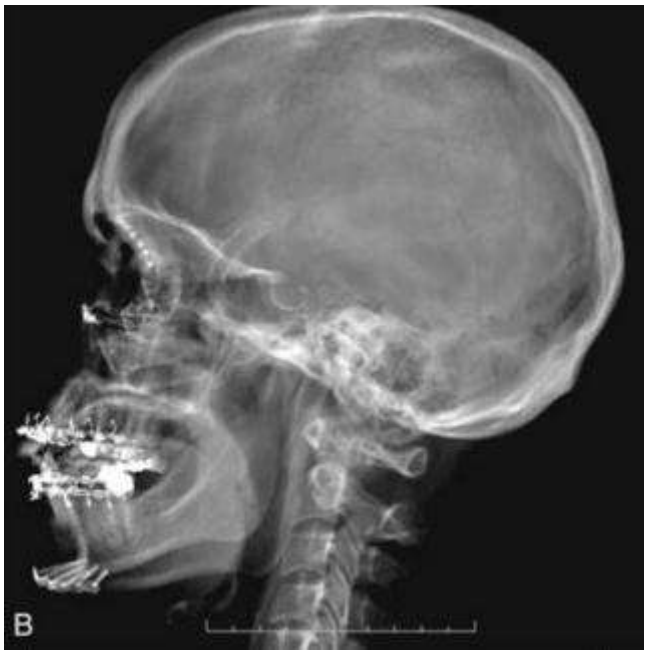
OPG Radiograph



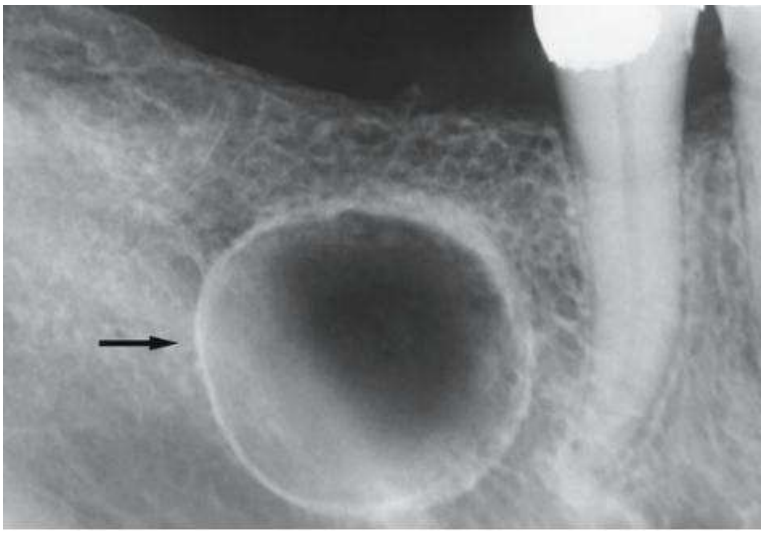
Occiptomental radiograph



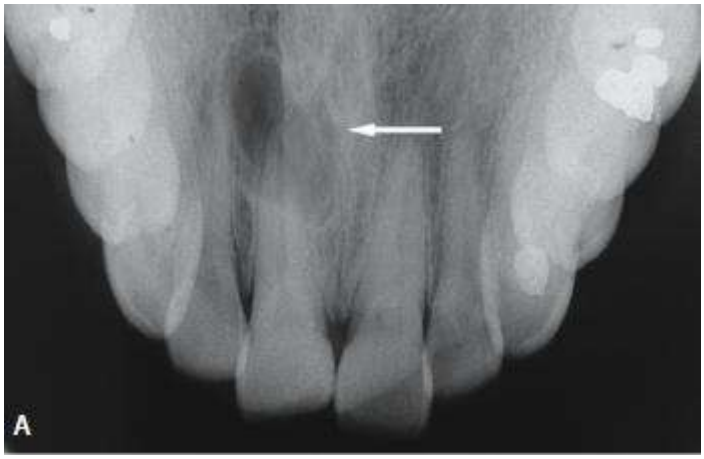
PA Skull (Mandible) Radiograph



True Lateral (Cephalometeric) Radiograph



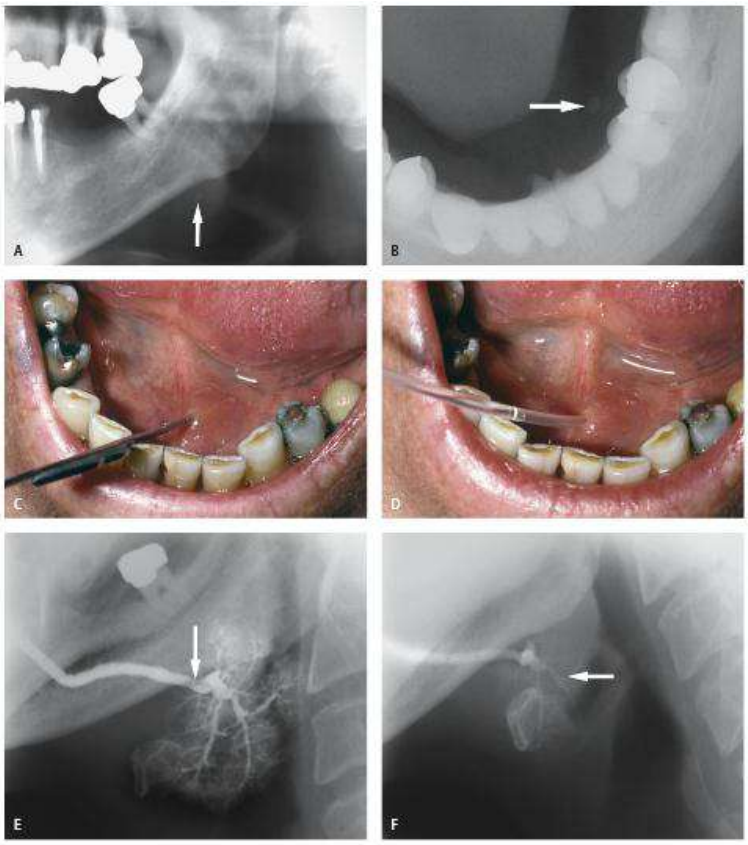
Periapical Radiograph



Occlusal Radiograph

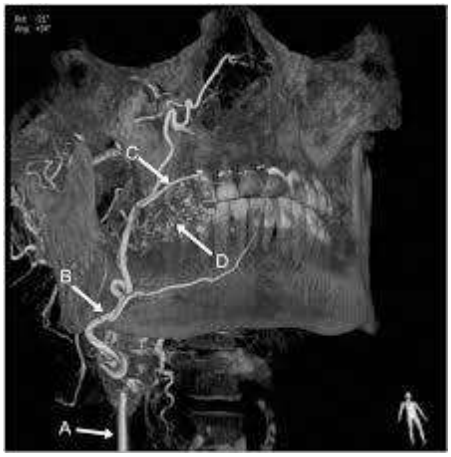
Contrast Studies

Contrast media may be introduced into a vessel, lumen or cavity in order to render it radio-opaque and hence radiographically visible. This may then be viewed in real time with fluoroscopic imaging or with serial radiographs. Contrast media used for this purpose include barium sulphate suspensions and non-ionic iodinated contrast agents. There is a small risk associated with the intravascular iodinated contrast agents which must be weighed against the potential benefits. Information which should be sought from the patient before contrast injection includes previous contrast reactions, asthma, renal problems, diabetes and metformin therapy.



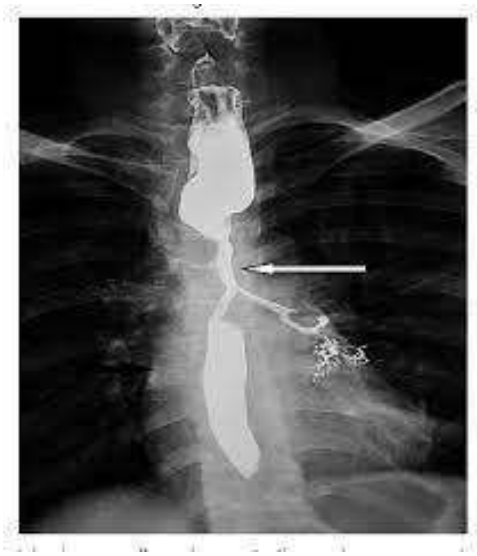
Contrast studies with maxillofacial applications are:

1 Angiography: Conventional angiography is generally performed as a precursor to interventional radiological techniques. CT and MR angiography have largely replaced diagnostic applications in maxillofacial pathology. It remains appropriate for the planning of embolization of high flow vascular malformations and tumours and for the evaluation of arterial injury (traumatic or tumour erosion). Angiographic catheters are generally introduced over a guidewire via a common femoral artery puncture. Small caliber microcatheters may be introduced into distal external carotid artery branches.

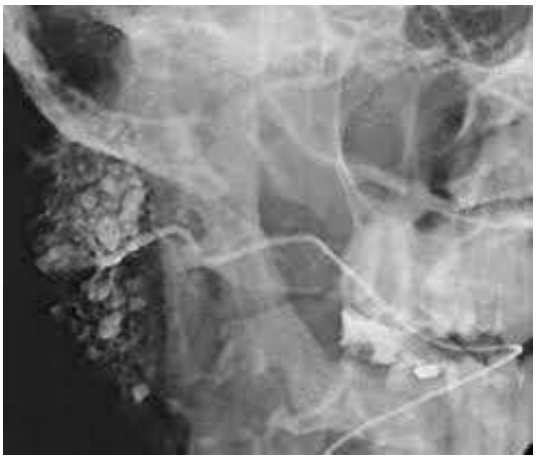


2 Barium/contrast studies: Barium swallows may be required to evaluate high dysphagia and pain. Rapid serial radiography or video recording may be used **to** assess the hypopharynx and upper oesophagus during deglutition. Barium may be combined with a gas-producing agent and an intravenous smooth muscle relaxant to produce double-contrast images of the lower

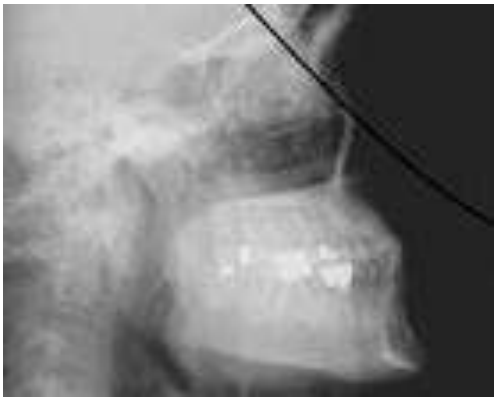
oesophagus. If aspiration or tracheooesophageal fistulation is suspected, then a low osmolar iodinated contrast medium will be used.



3 Sialogram: Iodinated contrast medium may be introduced into the salivary duct ostium via a polythene catheter. Fluoroscopy or radiography is used including delayed images after administration of a sialagogue. This is compared with preprocedure control films.

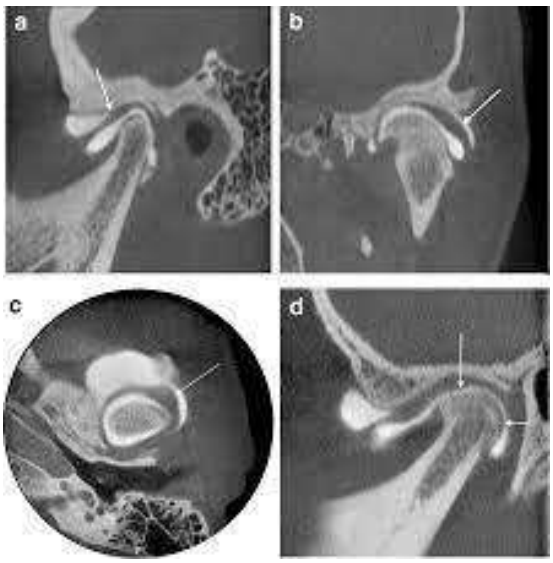


4 Sinogram/fistulogram: A sinogram involves the insertion of a fine catheter into the orifice of a sinus and injection of contrast medium in order to delineate a sinus or fistula. If there is a complex tract then it may be combined with CT.

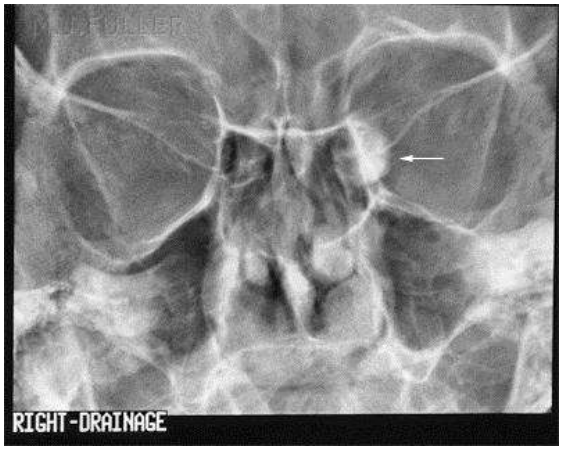


5 Temporomandibular joint (TMJ) arthrogram:

Iodinated contrast medium is injected into the joint under fluoroscopic guidance and double-contrast studies may be achieved by contrast withdrawal and replacement with air.



6 Dacrocystogram: The nasolacrimal sac and duct may be cannulated and injected with contrast medium in patients with epiphora. The lacrimal drainage system may also be evaluated with CT and MRI following conjunctival application of contrast medium.



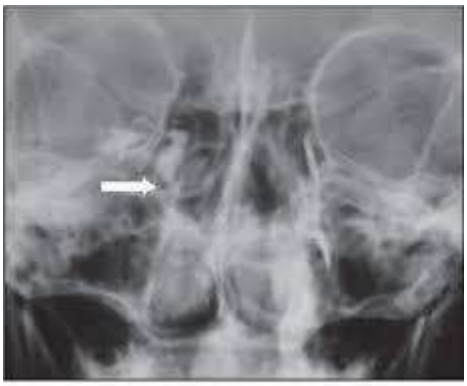
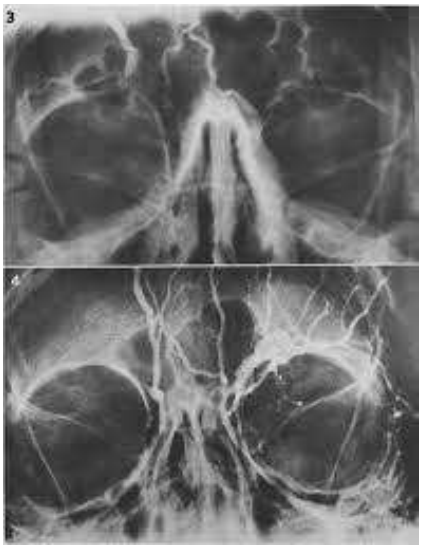


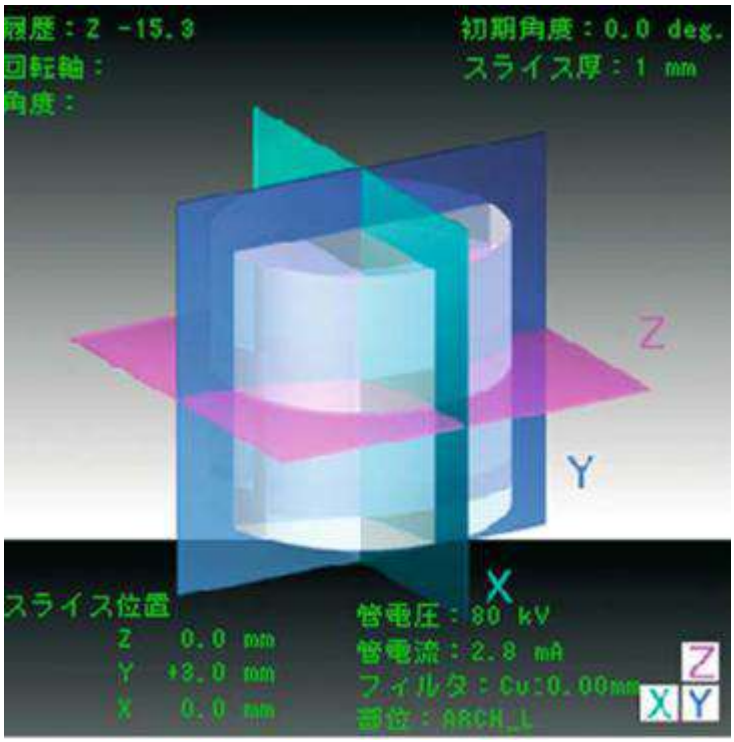
Figure 3. Conventional dacryocystography. Complete obstruction of the right lacrimal pathway at the level of the Krause's valve (arrow). Pervious and morphologically normal left lacrimal pathway.

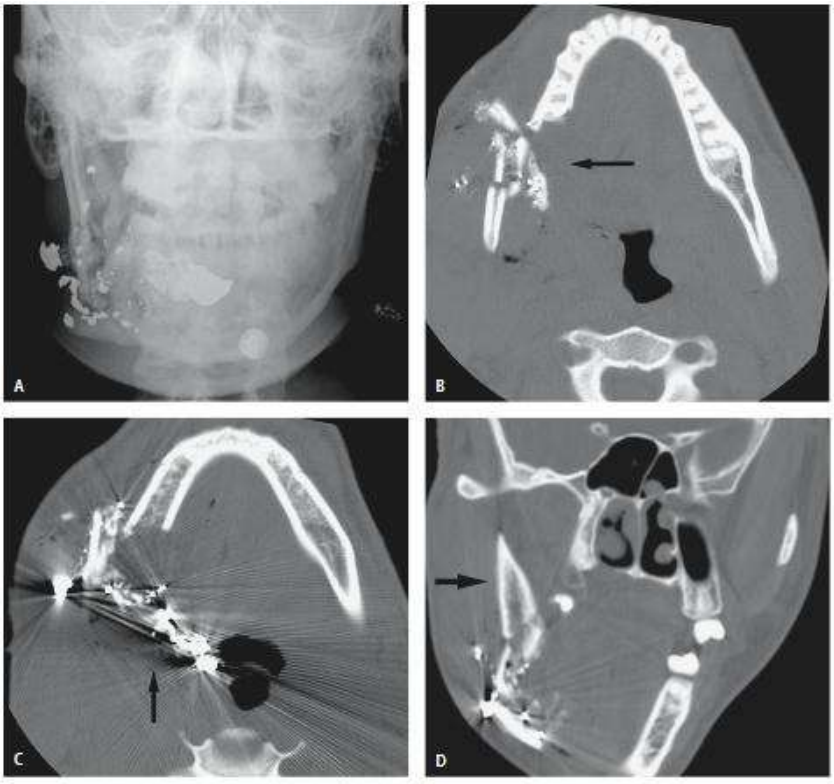
7 **Percutaneous venogram**: Percutaneous venography may be used as a precursor to sclerotherapy for the assessment of volume and venous run off in the setting of low flow venous malformations. A similar technique (lymphogram) is used for a lymphatic malformation. Ultrasound is used to guide the needle placement if the lesion is not clinically palpable.



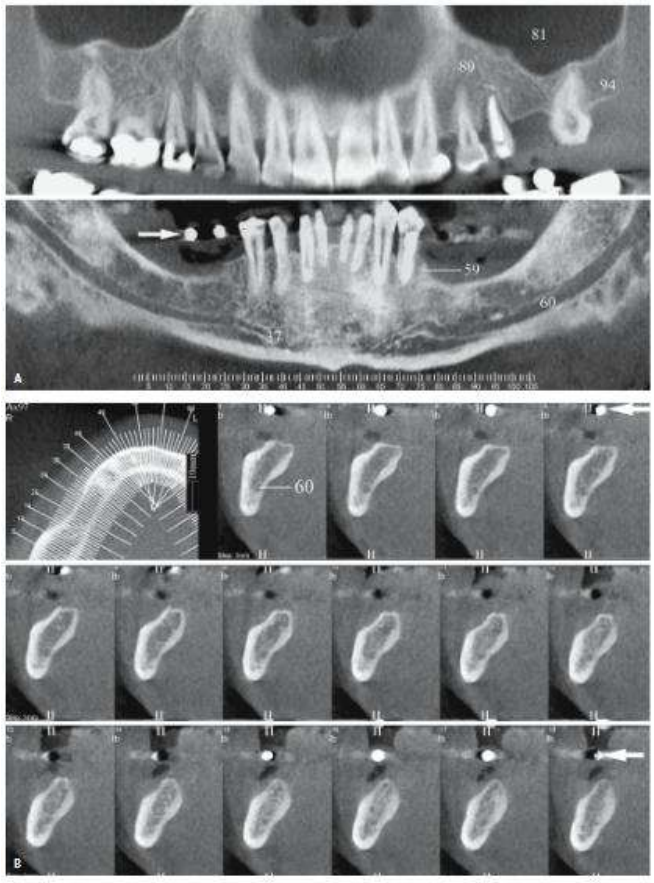
Computed Tomography

A CT scanner consists of an x-ray tube which sends a fan of x-rays through the patient and the attenuation of the beam by the patient is detected. The process is repeated as the tube and detectors rotate and the patient is advanced through the scanner. The degree of x-ray absorption by each volume of tissue (voxel) is displayed as a pixel which is allocated a number (Hounsfield unit). This information may be digitally manipulated so as to best demonstrate the tissues of interest (e.g. by changing the range of numbers in the grey scale or window width or by using algorithms to alter the sharpness of the image). The same information may be used to provide multiplanar reformats or rendering of three-dimensional objects to facilitate visual assessment. Imaging of soft tissues generally requires the administration of iodinated contrast medium to enhance pathological tissues and help delineate vascular structures from other soft tissue, such as lymph nodes. The availability of CT fluoroscopy and in-room CT controls/monitors has improved the safety and efficacy of CT-guided biopsies of deep facial and skull base lesions. Contemporary multislice computed tomography (MSCT) differs in that a number of slices (current scanners are typically 64 slice) are obtained per tube rotation. Multislice CT has the potential to scan standard volumes with shorter acquisition times so reducing movement artefact (e.g. due to swallowing) or the requirement for sedation and optimizing vascular opacification (e.g. for CT angiographic studies). It also allows the scanning of larger volumes or the use of narrower section thickness so optimizing the three-dimensional dataset for post-processing and interactive 3D image-guided surgery.


履歴：Z -15.3
回転軸：
角度：
初期角度：0.0 deg.
スライス厚：1 mm
Z
Y
X
スライス位置
Z 0.0 mm
Y +3.0 mm
X 0.0 mm
管電圧：80 kV
管電流：2.8 mA
フィルタ：Cu:0.00mm
部位：ARCH_L
Z
X Y


A
B
C
D

Cone beam CT has developed as a technique which povides high resolution 3D data at low radiation doses (e.g. equivalent to 2–8 OPGs). The equipment may resemble that of a conventional dental panoramic tomography unit (patient erect) or may mimic a conventional CT scanner (patient supine). A cylinder- or sphere-shaped volume of data is rapidly acquired with a single tube rotation. Some cone beam CT equipment is designed to simulate intraoral radiographs by imaging small volumes (e.g. two or three teeth) at high resolution, whilst other equipment is designed to image the whole maxillofacial region (e.g. 15-cm3 spheres). The low tube currents utilized to reduce the radiation dose unfortunately preclude adequate imaging of soft tissue structures.

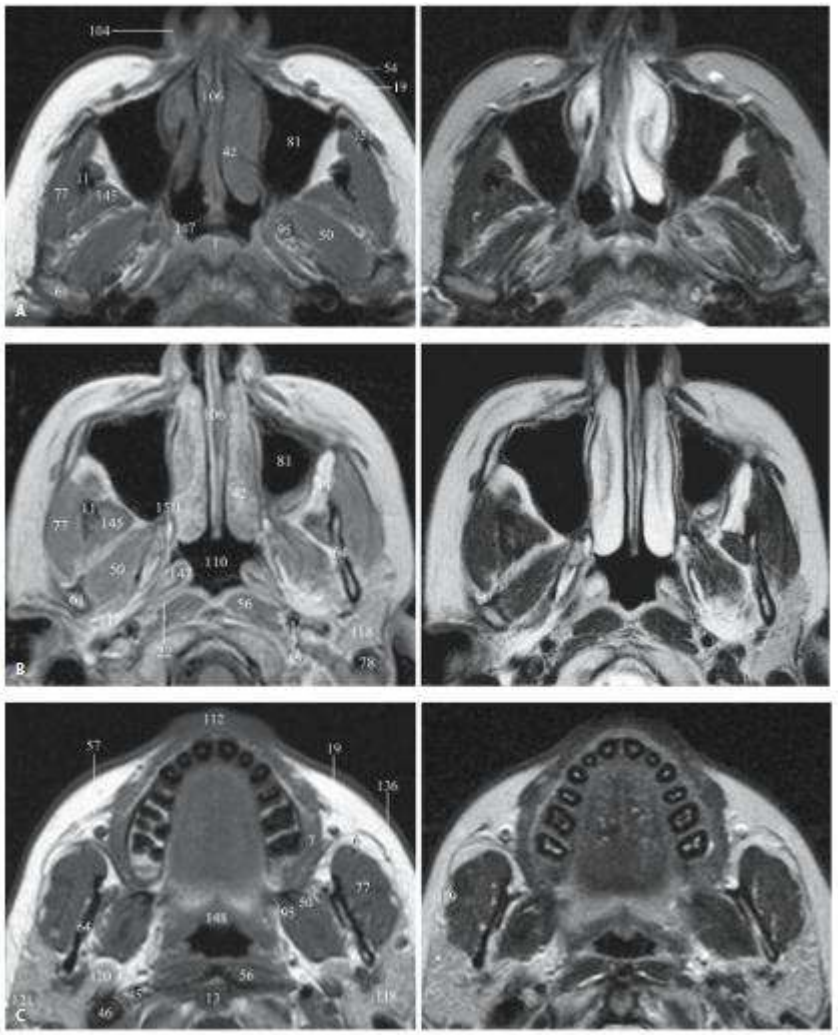


Magnetic Resonance Imaging (MRI)

Magnetic resonance imaging does not require ionizing radiation so should be preferred in cases where it would provide similar information to CT and both are available. There are some definite contraindications to the use of MRI, including metallic foreign bodies in the orbit, intracranial aneurysm clips, cardiac pacemakers and cochlear implants.

MRI signal is tissue dependent and is based on the behaviour of protons within that tissue when they are exposed to radiofrequency pulses within a magnetic field. Signal can be resolved into two components (T1 and T2). Selecting appropriate pulse sequences allows images to reflect the T1-weighted or T2-weighted characteristics of tissues. Most pathology results in increased water content relative to normal tissues and thus is shown as decreased signal on T1-w images and increased signal on T2-w images. There are various other tissues and substances which may be distinguished by differing MRI signal. Pre- and post-gadolinium (contrast medium) sequences should be performed with T1-w. T1-w sequences may also be combined with fat saturation postgadolinium, such that increased signal due to enhancement is not masked by that due to fat. Pathological lesions undergo variable enhancement and gadolinium is used to help characterize lesions. Normal structures that markedly enhance include mucosal linings and lymphoid tissue. The STIR (short time inversion recovery) sequence has been shown to be very sensitive to pathology which generally demonstrates increased signal. Multiplanar imaging (coronal and axial imaging as a minimum) is routinely performed with 4–5-mm section thickness. Typical imaging sequences for a study of the face and neck would include: T1-w axial, T2-w axial, T1-w postgadolinium axial, STIR coronal, T1 fat saturated post-gadolinium coronal images. MR angiography may demonstrate flow in

relation to a vessel lumen with or without the use of gadolinium. Other MRI techniques (such as spectroscopy, diffusion and perfusion imaging), higher field magnets (3 Tesla as opposed to standard 1.5 Tesla) and novel contrast agents, have been applied to the face and neck although clinical utility has not yet been established.



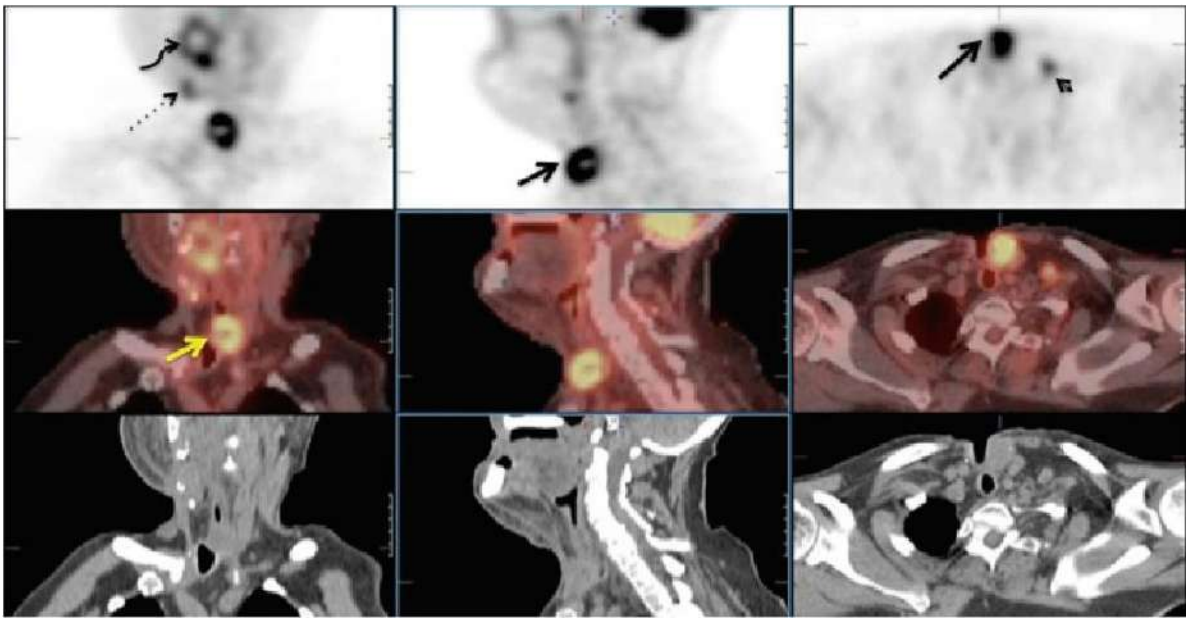
	CT	MRI
Advantages	Widely available	Does not require ionizing radiation
	Rapid so less prone to movement artefact	Usually less image distortion than CT from metallic foreign bodies
	Demonstrates cortical bone and calcification well	Delineates bone marrow pathology well (e.g. mandible/central skull base)
	May be combined with imaging of the lungs	Superior for skull base and intracranial imaging
	Excellent spatial resolution and 3D post-processing	Excellent contrast resolution with direct multiplanar imaging
Disadvantages	Ionizing radiation	Absolute contraindications preclude some patients
	May require iodinated contrast media (incidence of severe reactions is 0.04%)	Claustrophobia precludes some patients
		Time consuming and prone to motion artefact if patient breathless/unwell
		Expensive

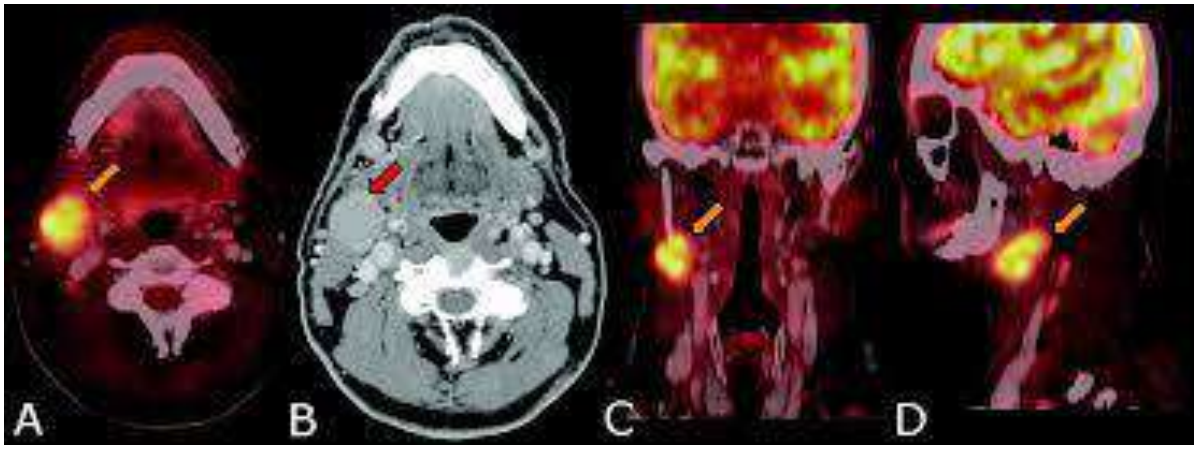
Positron Emission Tomography (PET) And Other Radioisotope Imaging

Positron emission tomography differs from the previously mentioned anatomical techniques in that it provides functional imaging of metabolic activity. This has proved very useful in the setting of maxillofacial malignancy with improved diagnostic accuracy relative to CT and MRI. Most PET imaging studies of the head and neck use the short-lived radiotracer 18-fluorodeoxyglucose (18FDG) which allows an examination of altered glucose metabolism as a marker of tumour activity. This unstable

radioisotope releases a positron over a short distance after which it annihilates with an electron and emits the photons that are detected. This process of photon production implies a lower limit of spatial resolution , so PET does not provide the same anatomical detail as CT or MRI. To improve the localization of pathology, PET images were initially co-registered with CT or MR images; however, techniques have now progressed such that functional and anatomical CT images (PET-CT) may be obtained on the same scanner. It should be noted that the CT component of such PETCT scanners may be performed without contrast medium and does not generally use the same parameters as diagnostic CT so is not a direct substitute. Multiple slices are obtained and multiplanar reformats are routine. A dedicated head and neck field of view may be followed by a separate half body study.

PET must be interpreted with an awareness of the limitations in detecting small volume (particularly <3–4 mm) disease, including superficial mucosal lesions, lymph node micrometastases and necrotic lymph nodes. Some tumours, such as salivary gland tumours, are not 18FDG avid. Some centres use an objective measure of FDG uptake standardized uptake value (SUV)) to help distinguish a malignant lesion. There are also pitfalls due to false-positive findings resulting from normal tracer distribution (e.g.salivary and thyroid gland, muscle activity and Waldeyer's ring) and inflammatory tissue (e.g. lymph nodes, early stages post-tumour treatment and healing bone).




A
B
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D

Other radioisotopes used in the investigation of maxillofacial disease include:

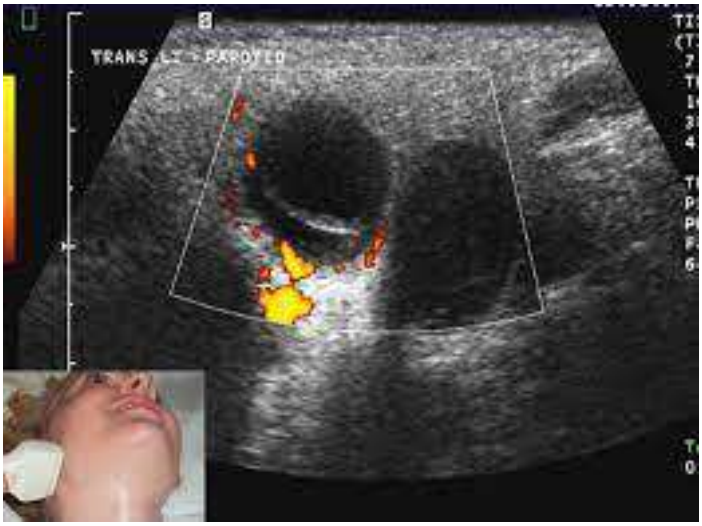
- 1 99mTc-MDP for the evaluation of bone disease (e.g. condylar hyperplasia, degree of activity in fibro-osseous lesions, bone metastases, bone invasion by tumour, osteomyelitis, integrity of blood supply in radionecrosis or vascularized grafts).
- 111Indium-labelled and 99mTc-HMPAO-labelled leukocytes together with 67Ga-gallium citrate for the diagnosis and localization of infection or inflammation in soft tissues.
- 99mTc-pertechnetate for dynamic salivary gland imaging or to detect ectopic thyroid tissue

Ultrasound imaging

Ultrasound imaging does not require ionizing radiation and is a relatively inexpensive, non-invasive and readily available technique which is well tolerated by patients. It is particularly useful in examining superficial structures (less than 5 cm deep to the skin surface), where the use of a high frequency linear probe (7.5–12 MHz) produces high definition images in multiple imaging planes. The spatial resolution achieved by ultrasound surpasses that of either computed tomography (CT) or magnetic resonance imaging (MRI), and when combined with tissue sampling techniques (percutaneous fine needle aspiration (FNA) for cytology or core biopsy for histopathology), ultrasound is a highly specific diagnostic tool.

Clinicians who have detailed knowledge of the anatomy of the head and neck region may choose to learn how to use ultrasound as an adjunct to clinical examination and as an aid to biopsy techniques. This chapter aims to

give an overview of the use of ultrasound in the neck with relevance to clinicians who either want to gain a greater understanding of the technique or who wish to begin to use ultrasound in their practice of ultrasound waves caused by variation in acoustic impedance by the various tissues being scanned. A detailed discussion of the physics involved is beyond the scope of this text, but essentially the ultrasound probe acts as both transmitter and receiver for sound waves. Images are generated by computerized analysis of the sound waves reflected back to the probe. The higher the frequency of the sound wave generated, the greater the resolution obtained, but there is a resultant fall off in penetration with higher frequencies. Typically, 8–12 MHz probes are used in assessment of the neck, giving improved resolution for superficial structures but with reduced penetration, i.e. a failure to generate images of deeper structures. This trade off is not usually a problem in the neck. Air causes marked scattering of the ultrasound wave, hence gel is used as the interface between skin and probe to optimize the though put of the sound wave signal. Gas and bone represent a problem as far as ultrasound is concerned; gas will cause scatter which results in a ‘white out’, while bone and other calcified structures transmit little sound causing acoustic shadowing (black hole).

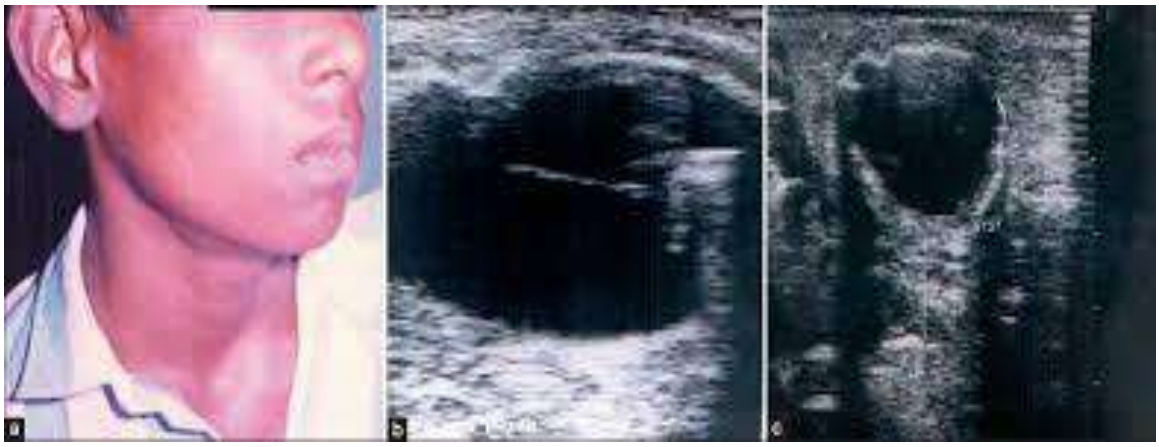


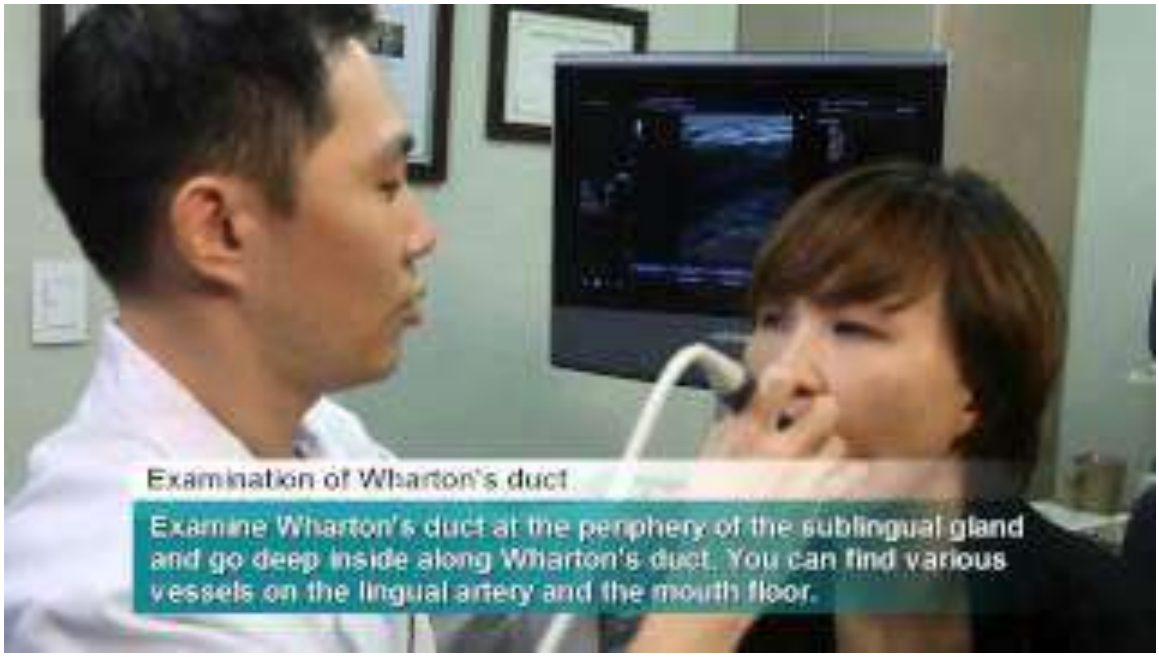
In general, highly reflective tissues appear echogenic (white) on an ultrasound image, whereas structures with poor reflectivity (e.g. blood within the internal jugular vein (IJV)) will be hypo-echoic (black) on an ultrasound image. The high reflectivity of some tissues may be desirable (e.g. identification of a core biopsy needle with ultrasound) or undesirable (e.g. a calcified thyroid lamina which prevents assessment of the larynx).

Sadly for the uninitiated, not all hypo-echoic structures are cystic or fluid in composition. Solid structures in the neck that may appear typically hypo-echoic or 'pseudocystic' (i.e. black) include lymphoma, salivary pleomorphic adenomata, nerve sheath tumours and parathyroid adenomata. Conversely, some cysts do not abide by the rules of physics – a true cyst should be hypo-echoic or black on ultrasound, but the congenital cysts of the neck (e.g. branchial and thyroglossal duct cysts) often appear echogenic, i.e. pseudosolid in appearance.

Colour and power Doppler may be used to assess flow in normal vascular structures (e.g. assessment of carotid arteriopathy and venous thrombosis) and abnormal flow in pathological processes (e.g. hilar vessels

in metastatic nodal disease). Colour flow Doppler is standard on most modern machines and can help the beginner to find vascular structures. A power Doppler function is useful for assessing flow patterns, such as the vascularity in lymph nodes.





Indications for head and neck ultrasound

The following indications will be considered:

- lymph node assessment
- salivary glands
- imaging lumps and bumps
- ultrasound-guided FNA and percutaneous core biopsy

1. Lymph nodes

Sonographic criteria for lymph node assessment have been extensively described in the literature. Normal nodes have a well-defined ellipsoid or fusiform shape, with an intermediate to low reflectivity homogeneous cortex and highly reflective central hilus. Abnormal nodes display reduced reflectivity (i.e. tend to be hypo-echoic or 'black') with a tendency to lose the central echogenic hilus. Short axis measurements increase, giving a rounder rather than elongated shape. Vascularity may increase and have a disordered pattern. Peripheral or subcapsular vessels, in particular, are a strong sign of malignancy

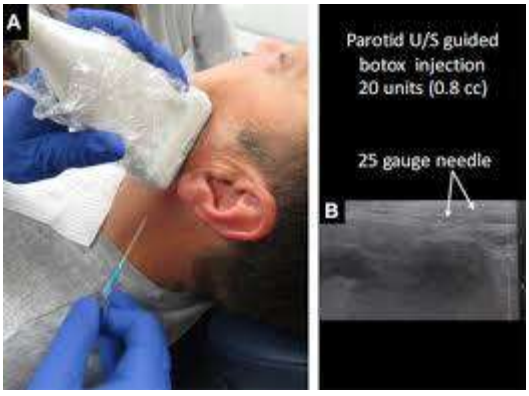
2. Salivary glands

The most common problems encountered include sialolithiasis, inflammatory conditions and tumours.

- **Submandibular Gland**

The normal submandibular glands are homogeneous echogenic (bright) structures lying inferolateral to the mylohyoid muscle in the submandibular space. Intraglandular ducts are visible as short defined hyperechoic lines, but Wharton's duct is only usually visualized when it is dilated. Lymph nodes in the submandibular space are exclusively extraglandular.

- **Parotid Gland**



Sialolithiasis

Intraglandular calculi are easier to identify than ductal stones. Frank duct dilatation or sialectasis may be seen and ultrasound will also demonstrate the complications of calculi, abscess formation and sialocele. Ultrasound cannot definitively exclude calculi, if there is a strong clinical suggestion of salivary duct obstruction and ultrasound examination is negative, sialography will be required in order to exclude a stone/stricture.

Inflammation

Acute salivary gland inflammation occurs in response to suppurative sialadenitis and viral infection. Inflammation causes gland hypertrophy and hypo-echogenicity, i.e. the salivary glands lose their normal bright echotexture. Ultrasound can be used to exclude abscess formation and may demonstrate hyper-reflective microbubbles of gas in suppurative sialadenitis, which usually affects a single gland, along with reactive nodes. In the case of abscess formation in acute suppurative sialadenitis, ultrasound-guided percutaneous drainage combined with antibiotic therapy may avoid surgical intervention.

Tumours

Approximately 80 per cent of salivary tumours are benign, 80 per cent occurring within the parotid with 80 percent of these being pleomorphic adenomata. The vast majority of parotid tumours lie within the superficial portion of the gland, allowing easy assessment with ultrasound. However, in the case of large or deep masses, the deep extent of a lesion can be difficult to assess (necessitating CT or MRI). Ultrasound cannot always predict whether salivary gland lesions are benign or malignant (although irregularity, abnormal vascularity and the presence of enlarged or suspicious

nodes aids accuracy), and is usually used in conjunction with fine needle sampling.

3. Miscellaneous lumps and bumps

Lipoma

Lipomas are benign encapsulated subcutaneous lesions which are frequently encountered in the neck. Typical sonographic features include hyperechogenicity, linear internal echoes perpendicular to the ultrasound beam , compressibility and a lack of internal vascularity on colour flow or colour Doppler imaging. Intramuscular lipomas can mimic muscle and may be difficult to define with ultrasound.

Haemangioma

The head and neck is a relatively common site for haemangiomas. They are frequently seen in the masseter, trapezius and sternomastoid muscles. Haemangiomata may have large cavernous spaces and possess capillary and/or lymphatic elements. Phleboliths may be demonstrated within the lesion in 70 per cent of cases. In large or intramuscular haemangiomas, MRI is better at depicting the extent of the lesion.

Branchial Cleft Cyst

Most branchial cysts arise from the second branchial arch remnants and present as a mass at the angle of the mandible, often following an infection. The typical location is abutting the posterior aspect of the submandibular gland, lying lateral to the carotid vessels and immediately anterior to the anterior border of the sternomastoid. On ultrasound, these lesions may be cystic, but more commonly the presence of debris, haemorrhage or infection gives rise to a pseudosolid appearance and the cyst wall thickens in the presence of infection. It may be impossible to distinguish between a second branchial cleft cyst and a necrotic lymph node metastasis due to squamous

cell carcinoma. Branchial cysts may extend between the carotid artery and lateral pharyngeal wall or have associated sinuses and these features are better demonstrated on MRI or CT than ultrasound.

Thyroglossal Duct Cyst

Thyroglossal duct cysts can arise at any position along the course of the thyroglossal duct remnant, but the majority are related to the hyoid bone, with most occurring at the level of or inferior to the hyoid. On ultrasound, thyroglossal duct cysts may appear cystic, heterogeneous or pseudosolid due to varying content of debris, haemorrhage or infection. Classically, they are embedded in the strap muscles, often 'splitting' the strap muscles. Malignant degeneration of the epithelial lining occurs rarely and any solid component which appears to contain microcalcification (i.e. suggestive of papillary carcinoma) should undergo sampling.

Dermoid Cyst

Dermoids can be identified by their site, i.e. either midline or peri-orbital. In the peri-orbital region, they are typically (60 per cent) found in the upper outer quadrant of the orbit. These lesions arise from sequestration of the ectoderm from adjacent sutures, most commonly the frontozygomatic suture. Dermoid cysts arise from more than one germ cell layer and therefore will contain one or more dermal adnexal structures. Sebaceous glands, hair and fat are commonly found in dermoids, but they may also be purely cystic. They may therefore have a heterogenous appearance with the presence of fat manifesting as a fluid/fluid level or often as rounded echogenic masses within the cyst (representing sebaceous rests within the dermoid). The typical location for midline cysts is in the submental region either superficial or deep to mylohyoid.

Abscess

Infection in the submandibular region frequently arises from dental disease. Ultrasound can differentiate between infection with a fluid component (abscess) and cellulitis, and identify associated lymphadenopathy and venous thrombosis.

4. Ultrasound-guided fine needle aspiration and core biopsy

Ultrasound is a very useful adjunct in percutaneous sampling procedures, allowing direct visualization of the needle and structures to be avoided (such as vessels). A metallic needle is a reflective surface and if placed parallel or slightly oblique to the transducer surface the needle will be imaged as a very reflective or echogenic structure. Thus the needle must be in the plane of the ultrasound beam and as parallel to the probe surface as possible in order to optimally visualize it.

Suggestive Readings

- L. Baert, LeuvenK. Sartor, Heidelberg. Medical Radiology Diagnostic Imaging, Springer-Verlag Berlin Heidelberg 2006.
- T. A. LarheimP.-L. Westesson. MaxillofacialImaging. Springer-Verlag Berlin Heidelberg 2006

Principles of Reconstructive Surgery

(Flap Surgery)

Flap surgery is a technique in plastic and reconstructive surgery where any type of tissue is lifted from a donor site and moved to a recipient site with an intact blood supply. This is similar to but different from a graft, which does not have an intact blood supply and therefore relies on growth of new blood vessels. This is done to fill a defect such as a wound resulting from injury or surgery when the remaining tissue is unable to support a graft, or to rebuild more complex anatomic structures such as the jaw.

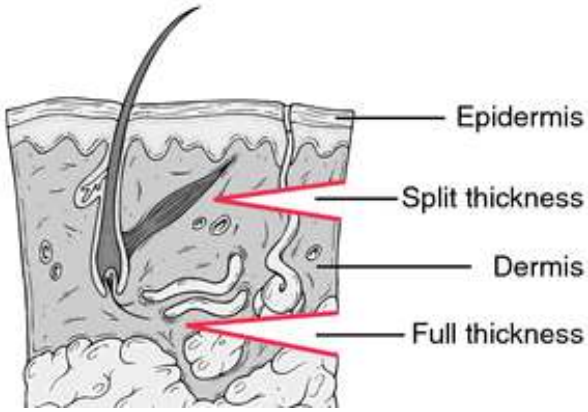
There are many causes of tissue loss, including

- Trauma
- Pathologic processes
- Congenital anomalies

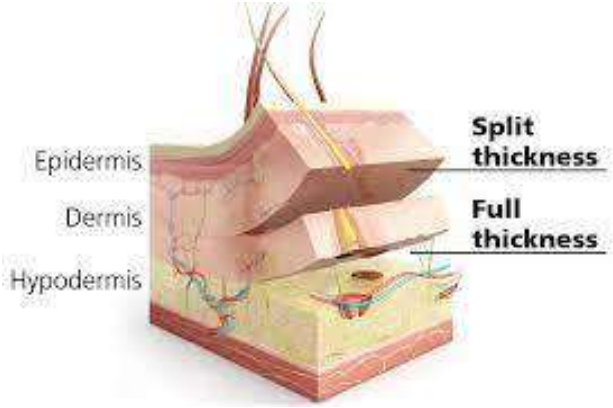
The resulting characteristics such as the size, geometry, and vascularity of the defects decide the surgical options available for treatment.

Choice for soft tissue reconstruction include

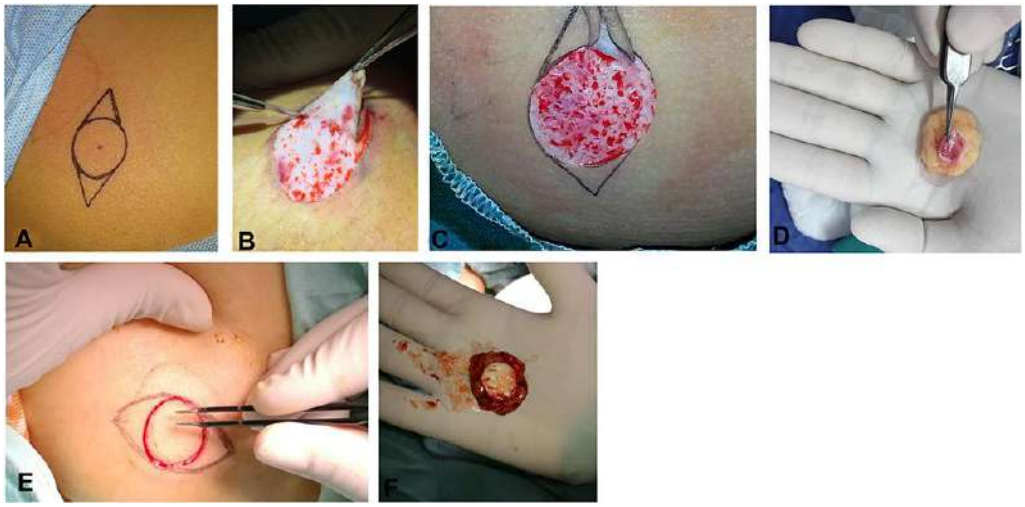
A-Full thickness skin or mucosal graft: Full thickness skin graft consist of entire thickness of epidermis and dermis. This graft taken from the donor area by using scalpel. Autogenous skin grafts have been used in oral and maxillofacial surgery, full thickness graft used in plastic surgical reconstruction of large facial defects., it is simple method of reconstruction, low complication rate, good colour match and minimal contracture also in the presence of failure, subsequent healing by secondary intention may give good result.



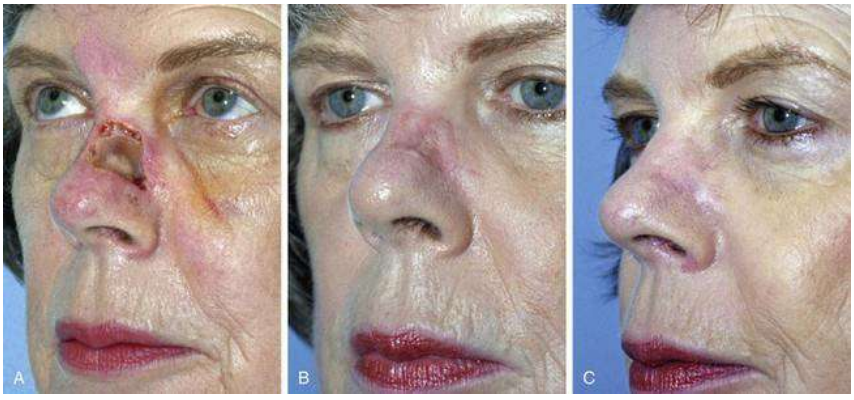
***B-Split skin grafts or mucosal graft*:** Contain all of epidermis and only part of dermis .Further subdivided in to **thin,medium or thick** according to amount of dermis containt . The graft is taken by either dermatome or a humby knife. It is most commonly used graft in head and neck cancer surgery .Resurfacing occur due to the remnant tissue in the donar area, so little care needed for donar area. It may be used to cover donar sites or secondary defects, to line flaps, to cover muscle when flap pedicles are exposed or rotated to replace small area of skin loss.



***C-Dermal and fat graft*:** Rarely used in head and neck surgery, dermal graft either pure dermis or dermis and fat, obtained by using dermatom , the epidermis is shaved off but left attached at one end then a strip of dermis is then taken as the graft, finally the epidermis is replaced and sutured.



***D-Full thickness skin and cartilage (composite) graft*:** consist of a full thickness skin graft with underlying cartilage may be taken from the auricular region used in reconstruction of small and moderate defects where tissue loss indicates that skin and underlying cartilage are required.



Transferring the grafts

Its life span depend on temperature, but when wrapped in gauze , moistened in saline and stored in fridge at 4c, it may live for up to 3 weeks. To survive permanently it

must be planted. Skin graft adheres to its new bed by fibrin .Fibrous tissue form by 5 days reasonable anchorage has occurred

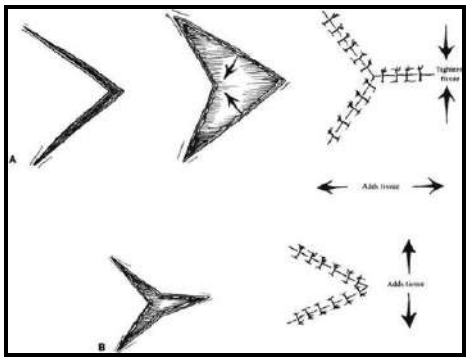
Site for grafting

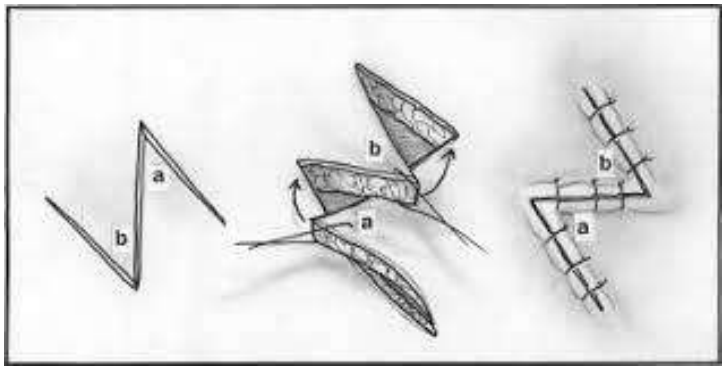
Split skin can easily be taken from the thigh, the upper arm and the flat surface of abdomen. Common head and neck full thickness skin graft donar sites are post auricular , preauricular and lower neck.

Classification of flaps

Flaps can also be classified according to tissue configuration

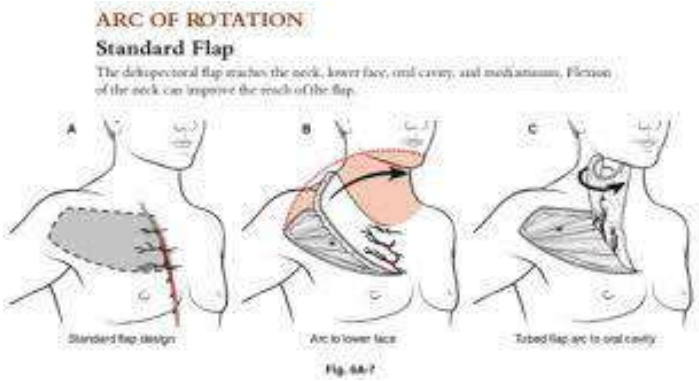
This describes the geometric shape of the flap. These flaps include rhomboid, bilobed, z-plasty, v-y, rotation, and others.

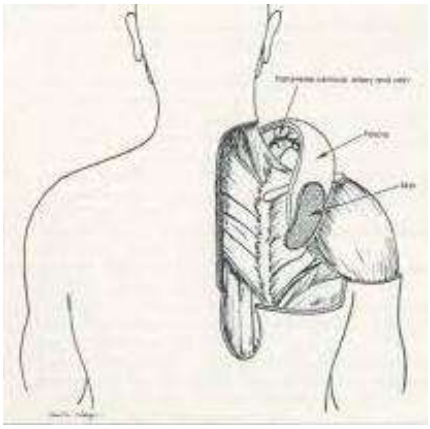




Flaps can also be classified by their tissue content.

These flaps include: *cutaneous* (skin and subcutaneous tissue), *myocutaneous* (composite of skin, muscle, and blood supply), and *fasciocutanous* (deep muscle fascia, skin, regional artery perforators).



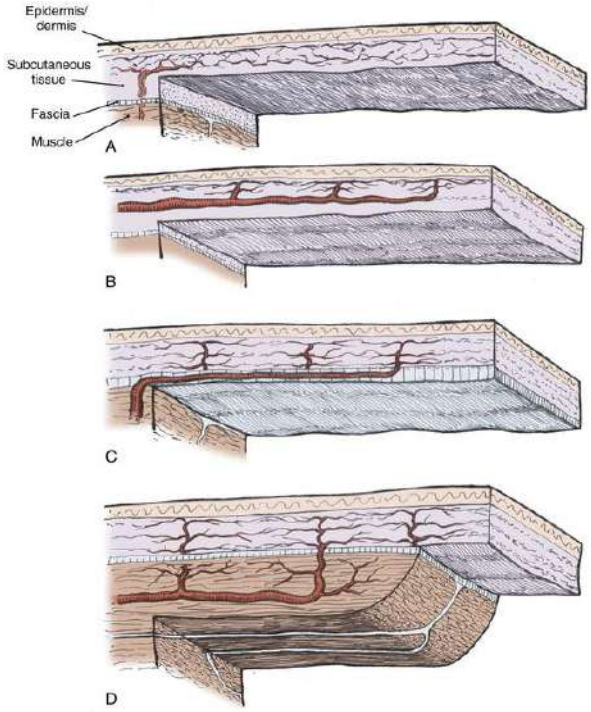


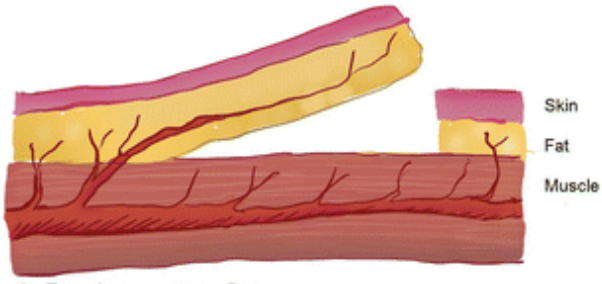
Flaps can also be classified according to arterial supply.

Axial Pattern Flap – A single flap which has an anatomically recognized arterio-venous system running along its long axis. Such a flap, because of the presence of its axial arterio-venous system, is not subject to many of the restrictions which apply to flaps in general.

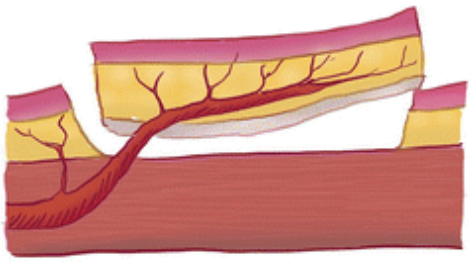
Random Pattern Flap - has no named blood supply, rather, it uses the dermal (mucosal) and subdermal (submucosal) plexus as its blood supply.

Pedicled flaps- remain attached to the donor site via a pedicle that contains the blood supply (in contrast to a free flap).

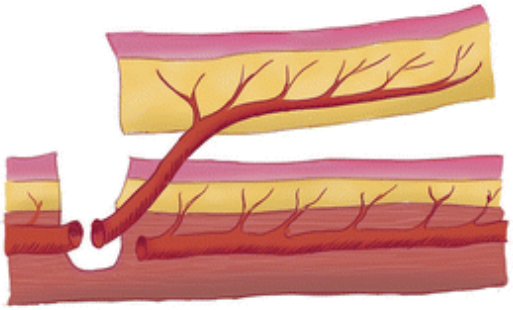




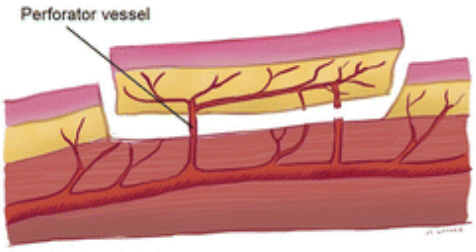
A: Random pattern flap



B: Axial pattern flap



C: Free flap



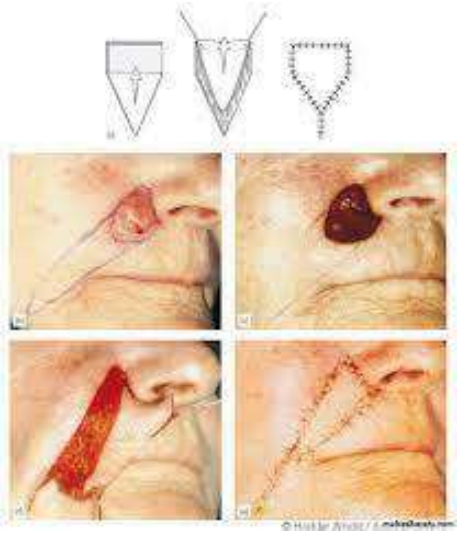
D: Perforator flap

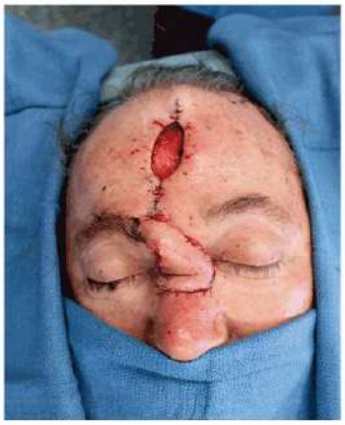
Classification can also be based on the relative location of the donor site.

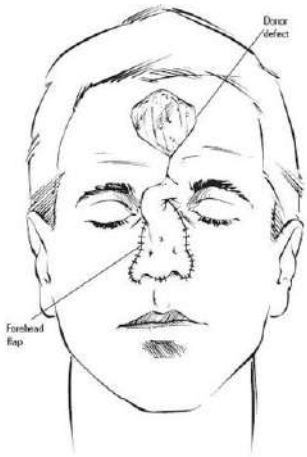
Local flaps are considered adjacent to the primary defect.

Regional flap donor sites are located on different areas of the same body part.

If different body parts are used as the donor site, the flap is termed a *Distant flap*.

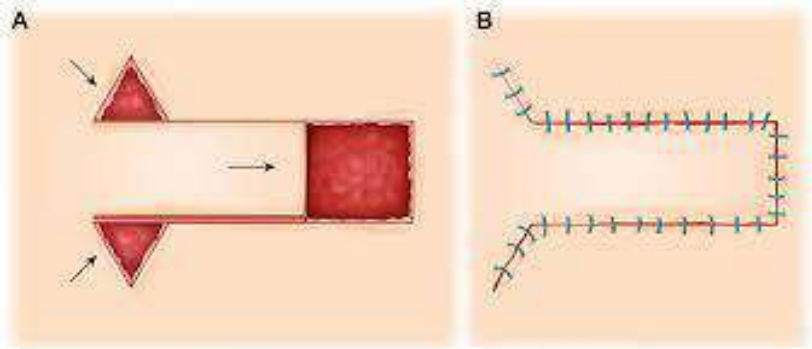




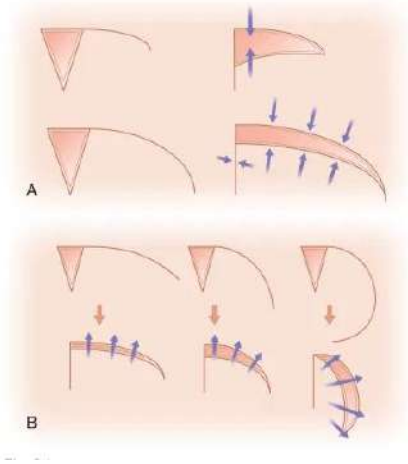


Classification can also be based on the flap movement.

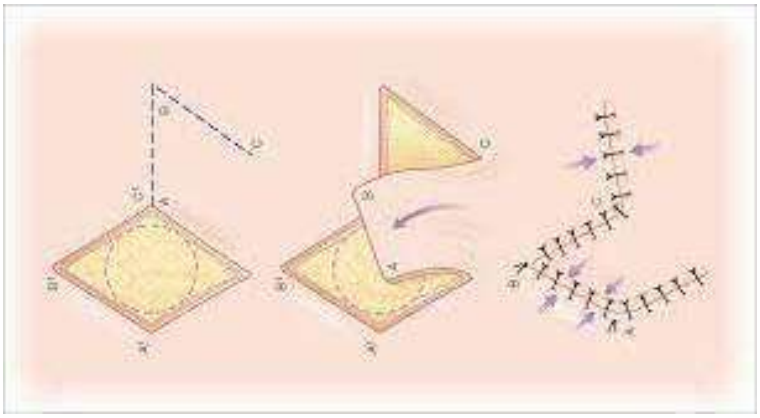
Advancement flaps use mobilized tissue in a direction toward the primary defect.



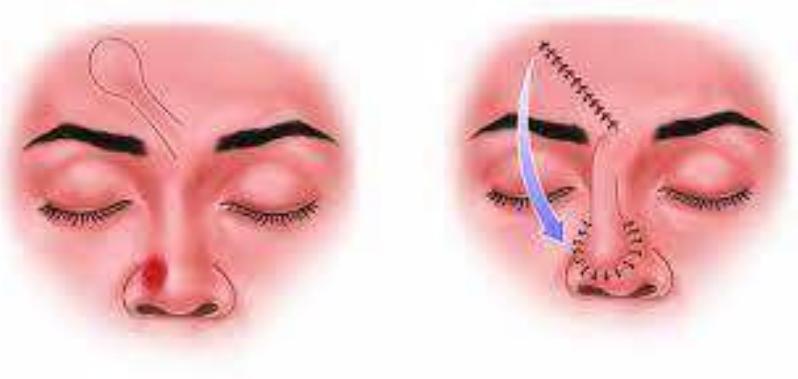
Rotation flaps pivot mobilized tissue around a point toward the primary defect.



Transposition flaps are mobilized tissues that traverse adjacent tissue by rotation and/or advancement in an effort to close the primary defect.



Interpolated flaps are mobilized tissues that traverse over or beneath an otherwise non-compromised skin bridge in the form of a pedicle to close the primary defect. The pedicle consists of skin (possibly subcutaneous fat and muscle) and/or an individual artery and vein used, with adjacent tissue, to maintain vascularity of the flap. At least one additional procedure is required to divide a pedicle.



Finally, *microvascular free tissue transfer* utilizes tissue transferred from a different part of the body and, unlike local or regional flaps, distant or microvascular free flaps require the detachment of the feeding vessels and transfer of the flap to the recipient site and anastomosing the vessels to a recipient artery and vein or veins. The advantage of this method of reconstruction is that the surgeon is no longer limited to the amount of tissue in the vicinity of the defect nor the arc of rotation of the flap. It enables the use of small to large or simple to complex tissue transfer. The obvious disadvantage is that when the skin in the head and neck needs to be reconstructed, the color match and texture will be significantly different.

Movement of the local tissue occur in one of two ways

1- The tissue may be advanced in a forward direction using **advancement technique**

2-Lateral movement using the pivot principle . When there is movement around a pivot point. The technique of **transpostion** and **rotation** based on this principle. When a flap is designed, it remains attached the body. Usually by its distal end which referred to as the base , and it is through this area that the blood supply enters.

Important consideration in flap planning

Appearance and amount

1- The anatomy and physiology of the skin including colour, texture ,

2-Local muscle anatomy, vascular supply, nerve supply and lymphatic drain

3-The esthetic of the area

4-Possible sites for incision placement.

5-Area of local tissue avaliable in relation to the area to be reconstructed.

Any one flap has advantages and disadvantages, Indications and contraindications

Local flaps

Because skin is elastic and stretches and it is possible to take tissue and move it from areas where it is redundant in to area where it is needed. The stretching of the skin is a mechanical property which related to the viscoelastic properties of the collagen bundles. It is time dependent ex- the longer one pulls the more it stretches.

Areas on the face which possible donar site

Particular areas on the face not only facilitate direct closure but also provide lax skin for transfer include, **glabellar area, temporal, nasolabial, mandibular and masseteric region.** The surgeon use pinch test to identify lax tissue, then the flap drawn out according to the size of primary defect.

1- Rotation flap-The flap defined as a large arc of semicircle where the triangular primary defect represent a small arc approximately one eighth the size of the flap, the flap is elevated then the difference in lengths of the two sides of the defect is made up by suturing with different tension. If closure is a little tight rotation may be facilitated by back cut. It is used to close oro-antral fistula (palatal flap).

2-Tranposition flap: It is rectangle which designed raised and elevated in to a triangulated defect. This leaves a donar site defect to be closed. This technique has little application in head and neck.

3-Panthographic expantion. This is variation of advancement

4-Transposition Z-plasty

The problem of skin advancement , when the tissue has been advanced, unless it is stopped where, it is, it tend to return whence it came one way to prevent this is to break up the scar with a Zplasty. This is ideal for reconstruction of lower eyelid which are excised as a triangle and tissue advanced laterally.

Flaps in Oral Surgery

The main concern of the dentist performing surgical procedures involves fundamental principles of surgery, asepsis and antisepsis, to prevent pathogenic microbes from entering the body as well as spread of certain infectious diseases from one patient to another. Sterilization of instruments, as well as preparation of the patient and dentist are therefore considered necessary.

The following fundamental rules apply to every surgical procedure, concerning the incision and flap:

- The incision must be carried out with a firm, continuous stroke, not interrupted strokes. During the incision, the scalpel should be in constant contact with bone. Repeated strokes at the same place, many times, impair wound healing.
- Flap design and incision should be carried out in such a way that injury of anatomic structures is avoided, such as: the mental neurovascular bundle, palatal vessels emerging from the greater palatine foramen and incisive foramen, infraorbital nerve, lingual nerve, submandibular duct, parotid duct, hypoglossal venous plexus, buccal artery (of concern when incision of an abscess of the pterygomandibular space is to be performed), facial nerve and facial artery and vein, which are of concern basically for the drainage of abscesses performed with extraoral incisions.

 Vertical releasing incisions should begin approximately at the buccal vestibule and end at the interdental papillae of the gingiva.

 Envelope incisions and semilunar incisions, which are used in apicoectomies and removal of root tips, must be at least 0.5 cm from the gingival sulcus.

 The elliptic incision, which is used for the excision of various soft tissue lesions, comprises two convex incisions joined at an acute angle at each end, while the depth of the incision is such that there is no tension when the wound margins are coapted and sutured.

 The width of the flap must be adequate, so that the operative field is easily accessible, without creating tension and trauma during manipulation.

 The base of the flap must be broader than the free gingival margin, to ensure adequate blood supply and to promote healing.

 The flap itself must be larger than the bone deficit so that the flap margins, when sutured, are resting on intact, healthy bone and not over missing or unhealthy bone, thus preventing flap dehiscence and tearing.

 The mucosa and periosteum must be reflected together. This is achieved (after a deep incision)when the elevator is continuously kept and pressed firmly against the bone.

 When the incision is not made along the gingival sulcus, for esthetic reasons, and especially in people with broad smiles, the scar that will result must be taken into consideration, particularly on the labial surface of the front teeth.

 Excessive pulling and crushing or folding of the flap must be avoided, because the blood supply is compromised and healing is delayed.

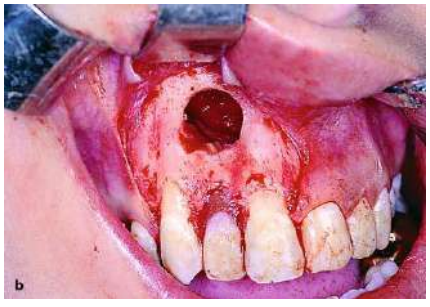
Local Flaps

The basic flap types are: trapezoidal, triangular, envelope, semilunar, flaps created by and incisions, and pedicle flaps.

- **Trapezoidal Flap:** The trapezoidal flap is created after a □-shaped incision, which is formed by a horizontal incision along the gingivae, and two oblique vertical releasing incisions extending to the buccal vestibule. The vertical releasing incisions always extend to the interdental papilla and never to the center of the labial or buccal surface of the tooth. This ensures the integrity of the gingival proper, because if the incisionwere to begin at the center of the tooth, contraction after healing would leave the cervical area of the tooth exposed. A satisfactory surgical field is ensured when the incision extends at least one or two teeth on either side of the area of bone removal. The fact that the base of the resulting flap is broader than its free gingival margin ensures the necessary adequate blood supply for the healing process. The trapezoidal flap is suitable for extensive surgical procedures, especially when the triangular flap would not provide adequate access.

  Advantages. Provides excellent access, allows surgery to be performed on more than one or two teeth, produces no tension in the tissues, allows easy reapproximation of the flap to its original position and hastens the healing process.

  Disadvantages. Produces a defect in the attached gingiva (recession of gingiva).



- **Triangular Flap:** This flap is the result of an L-shaped incision, with a horizontal incision made along the gingival sulcus and a vertical or oblique

incision. The vertical incision begins approximately at the vestibular fold and extends to the interdental papilla of the gingiva. The triangular flap is performed labially or buccally on both jaws and is indicated in the surgical removal of root tips, small cysts, and apicoectomies.

Advantages. Ensures an adequate blood supply, satisfactory visualization, very good stability and reapproximation; it is easily modified with a small releasing incision, or an additional vertical incision, or even lengthening of the horizontal incision.

Disadvantages. Limited access to long roots, tension is created when the flap is held with a retractor, and it causes a defect in the attached gingiva.

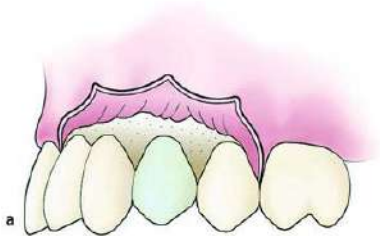
- **Envelope Flap:** This type of flap is the result of an extended horizontal incision along the cervical lines of the teeth. The incision is made in the gingival sulcus and extends along four or five teeth. The tissue connected to the cervical lines of these teeth and the interdental papillae is thus freed. The envelope flap is used

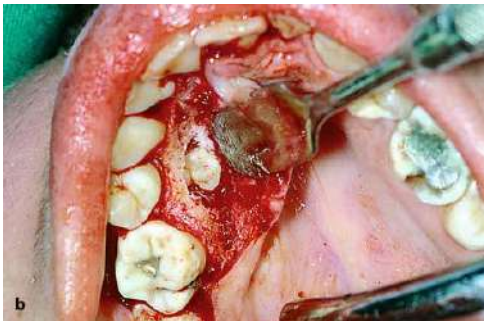
  for surgery of incisors, premolars and molars, on the labial or buccal and palatal or lingual surface , and is usually indicated when the surgical procedure involves

the cervical lines of the teeth labially (or buccally) and palatally (or lingually), apicoectomy (palatal root), removal of impacted teeth, cysts, etc.

Advantages. Avoidance of vertical incision and easy reapproximation to original position.

Disadvantages. Difficult reflection (mainly palatally), great tension with a risk of the ends tearing, limited visualization in apicoectomies, limited access, possibility of injury of palatal vessels and nerves, defect of attached gingiva.



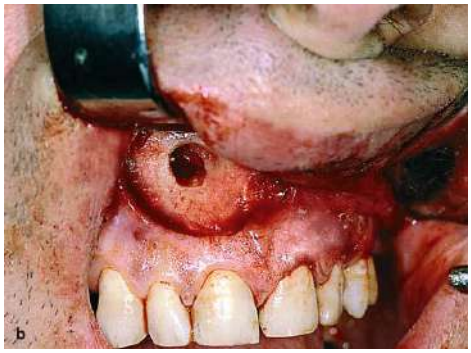


- **Semilunar Flap:** This flap is the result of a curved incision, which begins just beneath the vestibular fold and has a bowshaped course with the convex part towards the attached gingiva . The lowest point of the incisionmust be at least 0.5 cm fromthe gingivalmargin, so that the blood supply is not compromised. Each end of the incision must extend at least one tooth over on each side of the area of bone removal. The semilunar flap is used in apicoectomies and removal of small cysts and root tips.

Advantages. Small incision and easy reflection, no recession of gingivae around the prosthetic restoration, no intervention at the periodontium, easier oral hygiene compared to other types of flaps.

Disadvantages. Possibility of the incision being performed right over the bone lesion due to miscalculation, scarringmainly in the anterior area, difficulty of

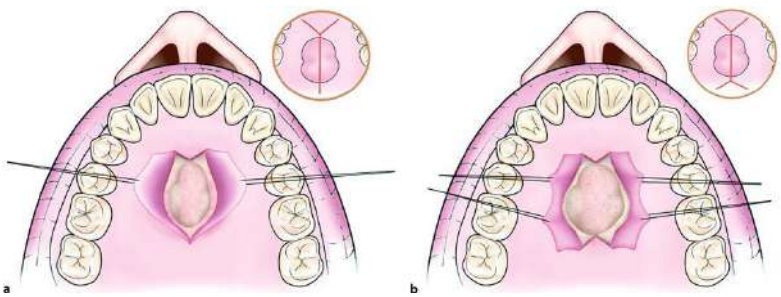
reapproximation and suturing due to absence of specific reference points, limited access and visualization, tendency to tear.



- **Other Types of Flaps:** Other types of flaps are the result of Y -shaped and X -shaped incision. These flaps are used in surgical procedures of the palate, mainly for the removal of exostoses (torus palatinus).

Flap Resulting from Y-shaped Incision. An incision ismade along themidline of the palate, as well as two anterolateral incisions, which are anterior to the canines. This type of flap is indicated in surgical procedures involving the removal of small exostoses.

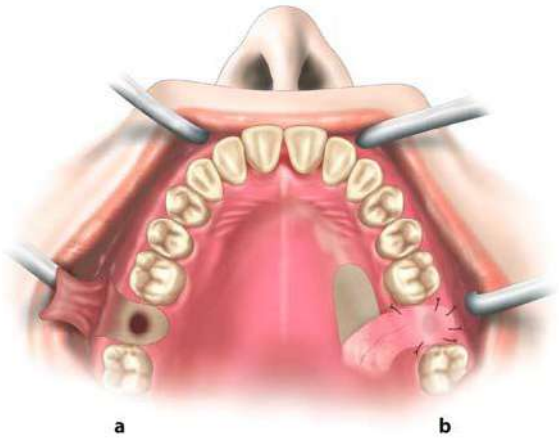
Flap Resulting from X-shaped Incision. This type of flap is used in larger exostoses, and is basically an extension of the -shaped incision. The difference is that two more posterolateral incisions are made, which are necessary for adequate access to the surgical field. This flap is designed such that major branches of the greater palatine artery are not severed.



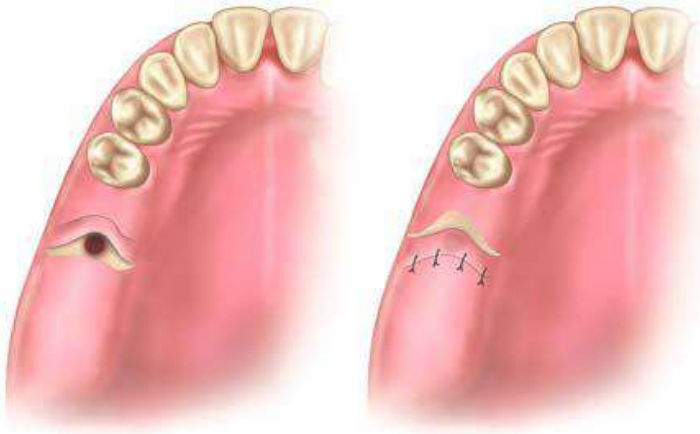
- **Pedicle Flaps:** The three main types of pedicle flaps used for closure of an oroantral communication are: buccal, palatal, and bridge flaps.

Buccal Flap. This is a typical trapezoidal flap created buccally, corresponding to the area which is to be covered, and is usually used on dentulous patients. It is the result of two oblique incisions that diverge upwards, and extend as far as the tooth socket . After creating the flap, the periosteum is incised transversally, making it more elastic so that it may cover the orifice that results from the tooth extraction. The oblique buccal flap is a variation of the buccal flap. It is the result of an anteroposterior incision, so that its base is perpendicular to the buccal area, posterior to the wound. The flap is rotated about 70°–80° and is placed over the socket. Both cases require that, before placing the flap, the woundmargins must be debrided.

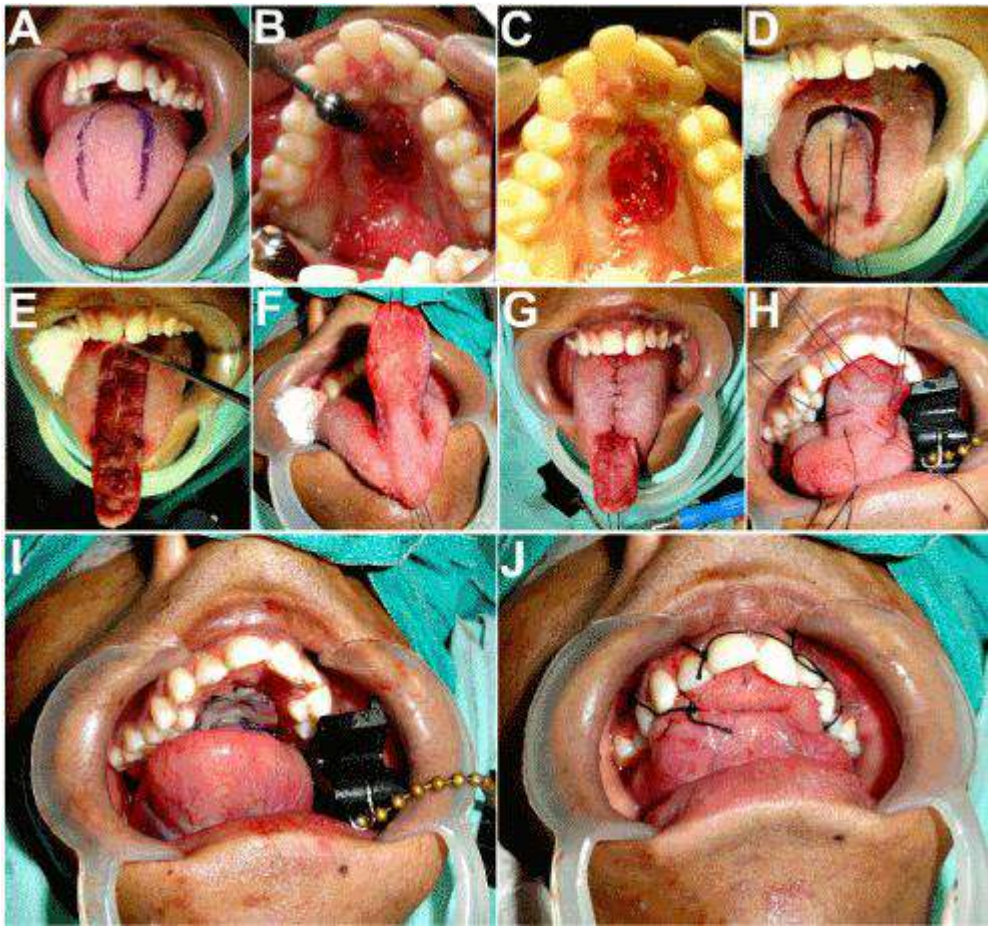
Palatal Flap. This type of flap is used in edentulous patients so that the vestibular depth is maintained. The resulting palatal mucoperiosteal flap is rotated posteriorly and buccally, always including the vessels that emerge from the corresponding greater palatine foramen. After rotation, the flap is placed over the orifice of the socket, the wound margins are debrided, and the flap is sutured with the buccal tissues. A gingival dressing is applied for a few days at the void created and healing is achieved by secondary intention.



Pedicle Bridge Flap. This flap is palatobuccal and is perpendicular to the alveolar ridge. After creation, the flap is rotated posteriorly or anteriorly, to cover the orifice of the oroantral communication, without compromising the vestibular fold. This type of flap is used only on edentulous parts of the alveolar ridge.



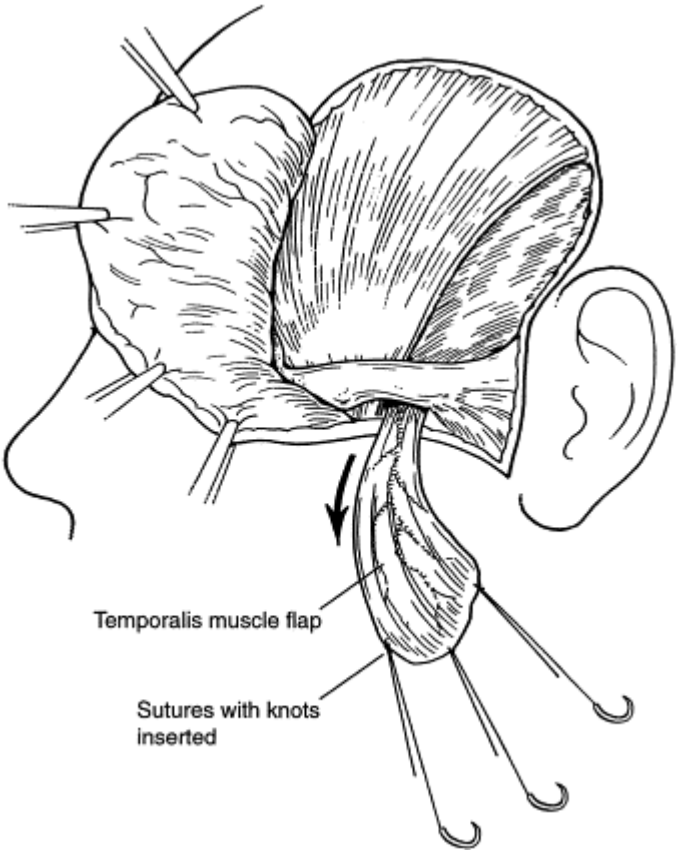
Tongue flap: The tongue flap is a robust, versatile flap that can be used for reconstruction of oral, pharyngeal, and perioral defects of congenital, traumatic, and ablative origin. The rich blood supply and ease of use make the tongue flap a reliable and predictable reconstructive technique for indicated defects.



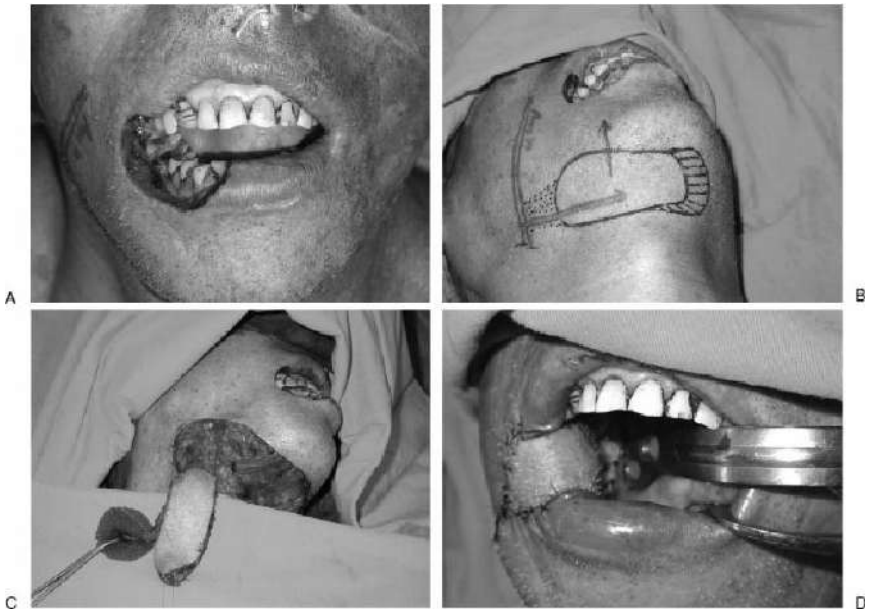
Regional & Distant flaps

Temporalis muscle flap: The external cheek, orbital exenteration, as well as maxillary and oral defects can be reconstructed using this flap. The temporal muscle elevates the mandible from its origin in the temporalis line and the infratemporal crest for insertion into the coronoid process. The temporal fascia consists of the superficial

temporoparietal and deep temporal fascia, further divided into superficial and deep layers. The muscle lies beneath the deep temporal fascia. These layers feature their own vasculature, with the superficial temporal fascia stemming from the superficial temporal vessels and the temporal muscle stemming from the deep temporal arteries originating at the internal maxillary artery. When harvesting the muscle flap, temporary removal of the zygomatic arch provides additional length to the flap. The flap measures from 12- to 16-cm-long and 0.5- to 1-cm-thick. Major drawbacks include a risk of injury to the facial nerve, postoperative trismus and temporal hollowing.



Submental Flap: In 1993, Martin presented the submental flap, a perforator or pedicled cutaneous flap from the submental region based on the submental branch of the facial artery. This flap features good colour match, good reach to the anterior mouth and the donor site is directly closed; typically, it offers an abundance of tissue, particularly in elderly patients. The skin paddle can reach up to 10 cm by 16 cm; the pedicle reaches up to 5 cm and the platysma muscle, a part of the mylohyoid, as well as the anterior digastricus muscle are included. The submental flap is also applicable in facial vessels proximally divided through a reverse flow, and can also be used as a free flap. The submental flap is ideal for reconstructing bearded areas in men.

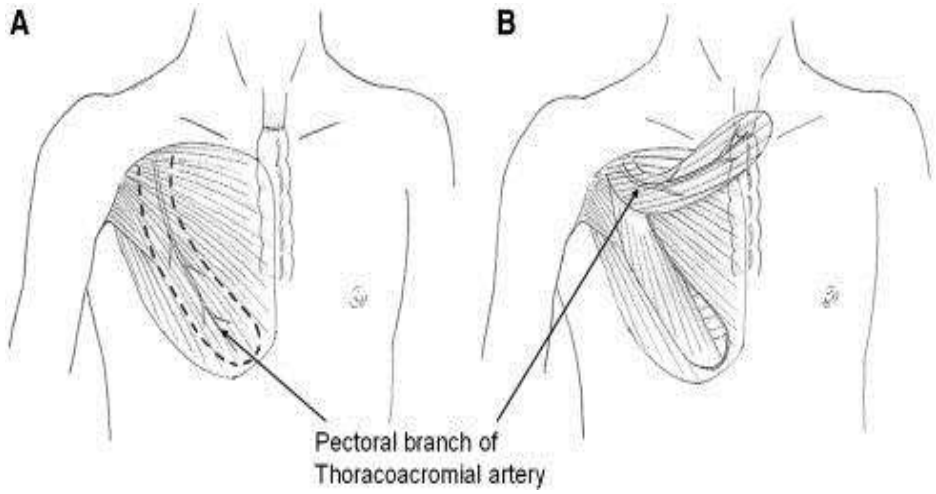


Deltopectoral flap : It is axial pattern flap designed on the anterior chest wall between the line of the clavicle and the level of the anterior axillary fold . It is based on branches of internal mammary artery. The flap will extend to any site in the neck and occasionally up to level of the zygoma. Tissues of the pectoral area such as skin and pectoralis major muscle are used in safe and extended flaps for cervical and neck reconstructions. As blood supply is derived from medial vessels (internal mammary

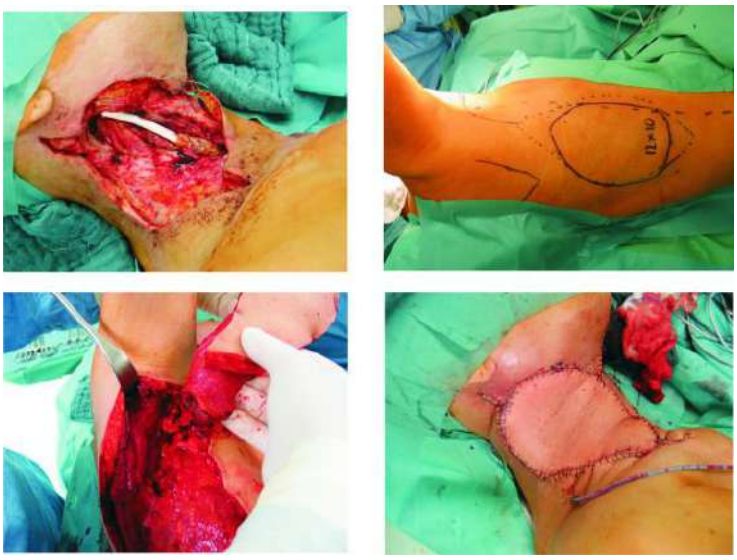
artery) or lateral (thoracodorsal and lateral thoracic arteries), 2 different flaps can be constructed: medial and lateral deltopectoral flaps. Medial deltopectoral flap was developed by Bakamjian as an axial-pattern skin flap, and its blood supply depends on perforating branches from the internal mammary artery. The successful use of this lateral deltopectoral flap in an extended cervical and thoracic reconstruction after resection of a giant basal cell carcinoma demonstrates that it must be considered as an alternative technique.

Pectoralis major flap: The pectoralis major myocutaneous (PMMC) flap has been used as a versatile and reliable flap since its first description by Ariyan in 1979**.** The flap receives its blood supply from the thoraco acromial artery and the secondary segmental perforators arising from the internal mammory artery. The lateral thoracic artery does not usually contribute significantly to the vasculature of the pectoralis muscle. The pectoralis major myocutaneous flap and myofascial flap variation are utilized in a large variety of head and neck reconstructive procedures that can include coverage of mucosal and/or cutaneous defects. The extent of coverage and the reach of the flap are dependent on the anatomy of the patient, modifications of the standard techniques of elevation, and inset. The upper limits are generally considered the zygomatic arch area externally and the superior tonsillar pole internally - patient body habitus may either limit extension short of these landmarks, or permit extension beyond The myofascial flap variation carries no skin paddle and is utilized primarily to close small mucosal defects, to protect major vascular structures, and to support primary

mucosal closure in a patient at increased risk of wound breakdown (prior radiation, diabetic, weight loss).

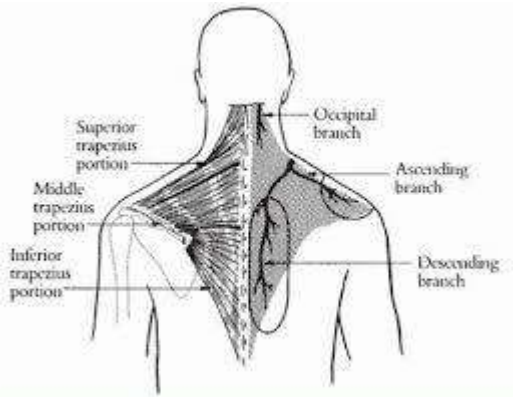


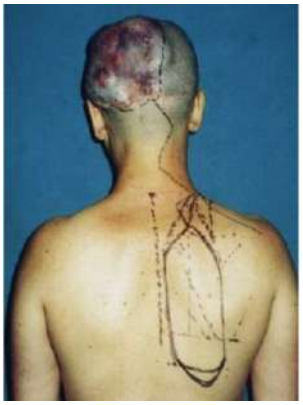
Latissmus dorsi flap: The latissimus dorsi flap was introduced by Tansini in 1906 for the coverage of extensive mastectomy defects. Subsequently forgotten, it was re-described by Olivari in 1976 for the coverage of large radiation ulcers of the chest wall. Boswick (1978) adopted Olivari's idea and developed a latissimus island flap for breast reconstruction. A further development, together with the technical progress of microsurgery, was its use as a free musculocutaneous flap. The latissimus dorsi flap, either as pedicled or as microvascular free tissue transfer, is one of the most commonly used flaps in reconstructive surgery, with large vessel diameters and a long reliable pedicle. Its size and versatility make it an extraordinary graft that has been a workhorse in reconstructive surgery for more than two decades. It can be elevated as a muscle, a musculocutaneous or an osteomyocutaneous flap. As a composite graft including variable amounts of muscle, skin, and subcutaneous tissue, it can be used in any variety for reconstruction in every area of the body. The harvested flap can be as large as 20 × 35, cm but numerous combinations with other flaps nourished by the subscapular system are possible to simultaneously reconstruct more complex defects with several flaps, based on a single pedicle.

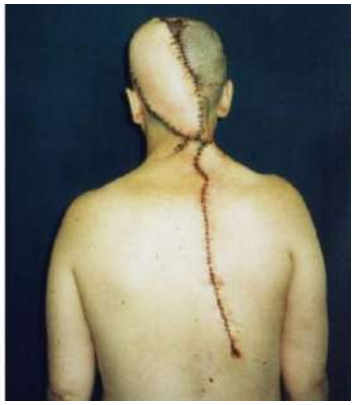


Sternomastoid flap: The sternocleidomastoid muscle has long been used for local reconstruction in the head and neck. It remains an incredibly versatile flap by providing bulk, an area of hairless skin with excellent color match for the face, and opportunity to transpose periosteum or bone for correcting head and neck defects. The middle third of the SCM muscle mostly receives its blood supply from 1 branch of the superior thyroid artery and the external carotid artery. In nearly one third of the specimens (8 [27%] on the right side and 8 [26%] on the left), these 2 arteries almost equally shared the blood supply

Trapezius flap: The trapezius flap is a large, thin, myocutaneous, pedicled flap. The wide arc of rotation and pliable tissue makes this flap ideal for reconstruction of H&N defects. The trapezius flap has been utilized for a range of H&N defects, The upper trapezius myocutaneous flap is based on the occipital artery , while the middle transverse trapezius myocutaneous flap is based on the transverse cervical artery (TCA) branches. The vertical paravertebral or lower trapezius myocutaneous flap is supplied by the deep branch of the TCA The trapezius muscle is the source of three myocutaneous flaps used in head and neck reconstruction: the superior trapezius flap, the lateral island trapezius flap, and the inferior or lower island trapezius flap. These flaps are used for lateral neck and lateral skull defects when a free vascularized flap is not considered. The lateral island trapezius flap, or trapezius osseomyocutaneous flap, is the only one of the three flaps that enables the transfer of bone pedicled with the muscle. It is the only reliable pedicled flap that enables the transfer of well-vascularized bone for mandibular reconstruction as well as skin for intraoral and extraoral defects. Before the use of microvascular techniques, this flap was used for mandibular reconstruction, and various authors report that its use has good functional and esthetic results. It is now accepted that osseointegrated implants are standard components of a complete mandibular reconstruction and the trapezius osseomyocutaneous flap fulfills the criteria of implantability.







Suggested Reading

- Robert Dolan,Facial plastic, Renstructive Truma Surgery, Marcel DeKker, NewYork,2003.
- Peter Ward Booth, Maxillofacial Surgery: Churchill Livingstone, 2006

Principles of Reconstructive Surgery

(Bone Graft)

Defects of the facial bones, especially the jaws, have a variety of causes, such as eradication of pathologic conditions, trauma, infections, and congenital deformities. The size of the defects that are commonly reconstructed in the oral and maxillofacial region varies considerably from small alveolar clefts to mandibulectomy defects. Each defect poses a unique set of problems that reconstructive surgical intervention must address.

Goals of Reconstruction

- Restoration of function
- Restoration of cervicofacial symmetry and form
- Creation of barriers between cavities and spaces in the head and neck
- Facial reanimation
- Dental rehabilitation
- Return of sensation

Types of Grafts

Several types of bone grafts are available for use in reconstructive surgery. A useful classification categorizes the bone grafts according to their origin and thus their potential to induce an immunologic response. Because of their origins and the preparations used to help avoid an intense immune response, the grafts have different qualities and indications for use.

i. Autogenous Grafts

Also known as autografts or self-grafts, autogenous grafts are composed of tissues from the same individual. Fresh autogenous bone is the most ideal bone graft material. The autogenous graft is unique among bone grafts in that it is the only type of bone graft to supply living, immunocompatible bone cells essential to phase I osteogenesis. The larger number of living cells that are transplanted, the more osseous tissue that will be produced.

Autogenous bone is the type used most frequently in oral and maxillofacial surgery. The bone can be obtained from a host of sites in the body and can be taken in several forms. Block grafts are solid pieces of cortical bone and underlying cancellous bone. The iliac crest is often used as a source for this type of graft. The entire thickness of the ilium can be obtained, or the ilium can be split to obtain a thinner piece of block graft. Ribs also constitute a form of block graft. Particulate marrow and cancellous bone grafts are obtained by harvesting the medullary bone and the associated endosteum and hematopoietic marrow. Particulate marrow and cancellous bone grafts produce the greatest concentration of osteogenic cells, and because of the particulate nature, more cells survive transplantation because of the access they have to nutrients in the surrounding graft bed. The most common site for the procurement of this type of graft is the ilium.

Autogenous bone may also be transplanted while maintaining the blood supply to the graft. Two methods can accomplish this:

The first involves the transfer of a bone graft pedicled to a muscular (or muscular and skin) pedicle. The bone is not stripped of its soft tissue pedicle, preserving some blood supply to the bone graft. Thus the number of surviving osteogenic cells is potentially great. An example of this type of autogenous

graft is a segment of the clavicle transferred to the mandible, pedicled to the sternocleidomastoid muscle.

The second method by which autogenous bone can be transplanted without losing blood supply is by the use of microsurgical techniques. A block of ilium, tibia, rib, or other suitable bone is removed along with the overlying soft tissues after dissecting free an artery and a vein that supply the tissue. An artery and a vein are also prepared in the recipient bed. Once the bone graft is secured in place, the artery and veins are reconnected using microvascular anastomoses. In this way the blood supply to the bone graft is restored. Both of these types of autogenous grafts are known as composite grafts because they contain soft tissue and osseous elements. The first type described, in which the bone maintains a muscular origin, is a pedicled composite graft. The pedicle is the soft tissue remaining on it, which supplies the vasculature. The second type of composite graft is a free composite graft, meaning that it is totally removed from the body and immediately replaced, and its blood supply is restored by reconnection of blood vessels.

Although these types of grafts may seem ideal, they have some shortcomings when used to restore defects of the jaws. Because the soft tissues attached to the bone graft maintain the blood supply, there can be minimal stripping of the soft tissue from the graft during procurement and placement. Thus the size and shape of the graft cannot be altered to any significant degree. Frequently, inadequate bulk of bone is provided when these grafts are used to restore mandibular continuity defects. Another problem is the morbidity to the donor site. Instead of just removing osseous tissue, soft tissues are also removed with composite grafts, which cause greater functional and cosmetic defects.

The **advantages** of autogenous bone are that it provides osteogenic cells for phase I bone formation, and no immunologic response occurs.

A **disadvantage** is that this procedure necessitates another site of operation for procurement of the graft.

ii. Allogeneic Grafts

Also known as allografts or homografts, allogeneic grafts are grafts taken from another individual of the same species. Because the individuals are usually genetically dissimilar, treating the graft to reduce the antigenicity is routinely accomplished. Today, the most commonly used allogeneic bone is freeze-dried. All of these treatments destroy any remaining osteogenic cells in the graft, and therefore allogeneic bone grafts cannot participate in phase 1 osteogenesis. The assistance of these grafts to osteogenesis is purely passive; they offer a hard tissue matrix for phase II induction. Thus the host must produce all of the essential elements in the graft bed for the allogeneic bone graft to become resorbed and replaced. Obviously, the health of the graft bed is much more important in this set of circumstances than it is if autogenous bone were to be used.

Advantages are that allogeneic grafts do not require another site of operation in the host and that a similar bone or a bone of similar shape to that being replaced can be obtained (e.g., an allogeneic mandible can be used for reconstruction of a mandibulectomy defect).

The **disadvantage** is that an allogeneic graft does not provide viable cells for phase I osteogenesis.

iii. Xenogeneic Grafts

Also known as xenografts or heterografts, xenogeneic grafts are taken from one species and grafted to another. The antigenic dissimilarity of these grafts is greater than with allogeneic bone. The organic matrix of xenogeneic bone is antigenically dissimilar to that of human bone, and therefore the graft must be treated more vigorously to prevent rapid rejection of the graft.. Bone grafts of this variety are rarely used in major oral and maxillofacial surgical procedures.

Advantages are that xenografts do not require another site of operation in the host, and a large quantity of bone can be obtained.

Disadvantages are that xenografts do not provide viable cells for phase I osteogenesis and must be rigorously treated to reduce antigenicity.

Osteoinduction, Osteoconduction and Osteogenesis

Osteoinduction: refers to new bone formation from the differentiation of osteoprogenitor cells, derived from primitive mesenchymal cells, into secretory osteoblasts. This differentiation is under the influence of bone inductive proteins or bone morphogenic proteins (agents from bone matrix). Osteoinduction implies that the pluripotential precursor cells of the host will be stimulated or induced to differentiate into osteoblasts by transplanted growth factors and cytokines.

Osteoconduction: is the formation of new bone from host-derived or transplanted osteoprogenitor cells along a biologic or alloplastic framework, such as along the fibrin clot in tooth extraction or along a hydroxyapatite block. Osteoconductive grafts provide only a passive framework or scaffolding. These grafts are biochemically inert in their effect upon the host. The grafted material therefore does not have the ability to actually produce

bone. This type of graft simply conducts bone-forming cells from the host bed into and around the scaffolding.

Osteogenesis: is the formation of bone from osteoprogenitor cells. Spontaneous osteogenesis is the formation of new bone from osteoprogenitor cells in the wound. Transplanted osteogenesis is the formation of new bone from osteoprogenitor cells placed into the wound from a distant site.

Osteogeneic grafts include the advantages of osteoinductive and osteoconductive grafts in addition to the advantage of transplanting fully differentiated osteocompetent cells that will immediately produce new bone. Autogenous bone is the only graft that possesses all these criteria.

Goals of Maxillofacial Reconstruction

- *Restoration of continuity*
- *Restoration of alveolar bone height*
- *Restoration of osseous bulk*
- *Preservation of normal speech, swallowing, and velopharyngeal function*
- *Close oral-antral and/or oral-nasal fistulae*
- *Maintain nasal patency*
- *Obliterate postoperative dead space*
- *Expedite wound healing and transition to adjuvant therapy*
- *Maximize mouth opening and masticatory function*
- *Maintain functional lip competence*
- *Provide vertical support to the globe and associated facial soft tissues*
- *Create a stable preprosthetic framework for implant reconstruction and/or obturator fabrication*

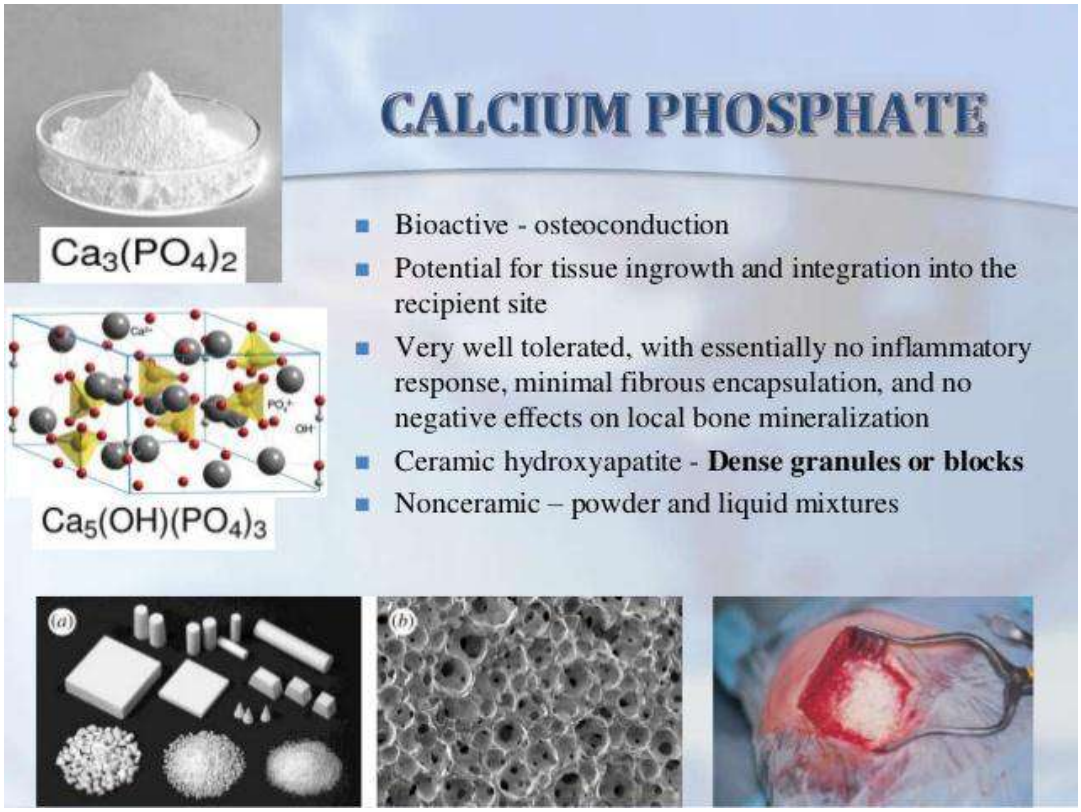
Alloplastic Materials:

An **alloplastic graft** is composed of material that is not taken from an animal or human source. **Alloplastic grafts** can be derived from natural sources (such as an elements or minerals), synthetic (man-made) substances, or a combination of the two**.**

Alloplasty is a surgical procedure performed to substitute and repair defects within the body with the use of synthetic material. It can also be performed in order to bridge wounds. The process of undergoing alloplasty involves the construction of an alloplastic graft through the use of computed tomography (CT), rapid prototyping and "the use of computer-assisted virtual model surgery." Each alloplastic graft is individually constructed and customised according to the patient's defect to address their personal health issue. Alloplasty can be applied in the form of reconstructive surgery. An example where alloplasty is applied in reconstructive surgery is in aiding cranial defects. The insertion and fixation of alloplastic implants can also be applied in cosmetic enhancement and augmentation. Since the inception of alloplasty, it has been proposed that it could be a viable alternative to other forms of transplants. The biocompatibility and customisation of alloplastic implants and grafts provides a method that may be suitable for both minor and major medical cases that may have more limitations in surgical approach.

Although there has been evidence that alloplasty is a viable method for repairing and substituting defects, there are disadvantages including suitability of patient bone quality and quantity for long term implant stability, possibility of rejection of the alloplastic implant, injuring surrounding nerves, cost of procedure and long recovery times. Complications can also occur from inadequate engineering of alloplastic implants and grafts, and poor implant fixation to bone. These include infection, inflammatory reactions, the fracture

of alloplastic implants and prostheses, loosening of implants or reduced or complete loss of Osseointegration.



Surgical Principles of Maxillofacial Bone Grafting Procedures

Several important principles should be followed during any grafting procedure. They must be strictly adhered to if a successful outcome is desired. The following are a few that pertain to reconstructing maxillofacial defects:

1- *Control of residual bone segments:*

2- *A good soft tissue bed for the bone graft:*

3- *Immobilization of the graft:*

4- *Aseptic environment:*

5- *Systemic antibiosis:*

Suggested Reading

- *Peter Ward Booth, Maxillofacial Surgery: Churchill Livingstone, 2006*

SURGICAL AIDS TO ORTHODONTICS

Surgery in the orthodontic patient will often be an integral part of a treatment plan. Surgical interventions that may be required in orthodontic treatment include: extraction of erupted teeth, management of impacted, supernumerary and dilacerated teeth, corticotomy and excision of labial frenum.

Management of Impacted teeth

Impacted tooth is one that has failed to erupt into normal functional position beyond the time usually expected for such appearance.

It occurs where there is prevention of complete eruption due to:

- Lack of space in the dental arch (main cause).
- Obstruction by another tooth.
- Development in abnormal position.
- Dense overlying bone.
- Thick fibrous tissue.
- Odontogenic cysts or tumors.

The mandibular third molar is the most commonly impacted tooth in the mouth followed by maxillary third molar, maxillary canine, mandibular canine, mandibular second premolar, maxillary second premolar and second molars.

Medical History

A detailed medical history is necessary because useful information may be found concerning the general health of the patient to be operated on. This information determines the preoperative preparation of the patient, as well as the postoperative care instructions.

Clinical examination

The position of the tooth may be obvious by the presence of a bulge either palatally or buccally. The palatal impaction is more common than the buccal one. Palpation of the maxilla through the labiobuccal sulcus may reveal the presence of the bulge buccally. The lateral incisor may be proclined due to the presence of the canine labial to the root or may be retroclined if the canine is palatal.

Radiographic examination and assessment

The radiographic views that can be used include; periapical, occlusal, OPG, lateral skull view cone beam CT (CBCT) can also be used.

The periapical radiograph provides a detailed view of the tooth, surrounding bone, root formation, the presence of root resorption of the adjacent lateral incisor or the presence of any pathology.

Localization of the canine is important especially when it cannot be determined clinically. Methods of localization include:

- Buccal object rule (parallax method, tube shift technique); in which two periapical films are taken, shifting the tube horizontally distally between exposures, if the unerupted tooth moves in the same direction in which the tube is shifted it is localized palatally, if it moves in opposite direction it is buccally located, in a rule called SLOB (Same Lingual Opposite Buccal).
- Vertex occlusal projection; which produces an axial view of the incisors, will demonstrate the buccopalatal localization of the canine.
- Periapical-occlusal method; uses a standard periapical view and an occlusal view to give two different views of the impacted tooth.

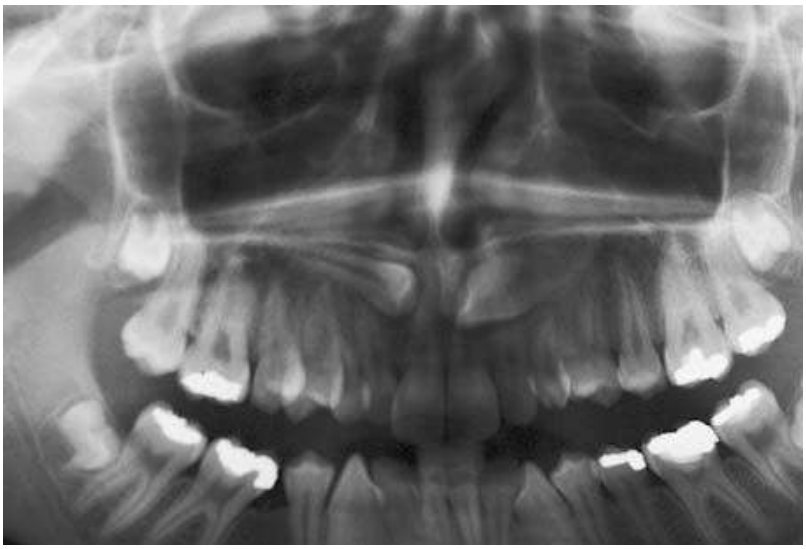
- OPG; can be used to localize impacted canine on the basis that palatally impacted canine appear magnified. It can also demonstrate the vertical angulation and its height.
- Lateral skull view or cephalometric.
- CBCT.

Impacted Canines

Impacted maxillary canines are quite common, and approximately 12%–15% of the population present with impacted canines. They are localized palatally more often than labially. Even though positions vary, the impacted canine presents five basic localizations (contralateral or ipsilateral and deep in the bone) as follows:

1. Palatal localization
2. Palatal localization of crown and labial localization of root
3. Labial localization of crown and palatal localization of root
4. Labial localization
5. Ectopic positions

In young people aged 20 years or slightly older, impacted maxillary canines may be correctly aligned in the dental arch after surgical exposure and orthodontic treatment. In older patients, especially after the age of 30 years, the above procedure is not a method of choice, because the risk of failure is greater. In such cases, surgical removal is preferred, if deemed necessary of course. The technique for removing impacted canines depends on the position of impaction (palatal or labial), the relationship of the impacted tooth to adjacent teeth, as well as the inclination of its crown. These factors should be assessed before planning the surgical procedure.



The localization of impacted canines is achieved using various radiographic techniques together with careful clinical examination. The most commonly used intraoral projections are occlusal projections, periapical radiographs and panoramic radiographs, while the technique employed for exact localization of the labial or palatal position of the impacted tooth is based on the tube shift principle. As far as the clinical examination is concerned, a palpable protuberance of the area designates the position of the tooth quite accurately. Based on the data from the clinical and radiographic examination, the surgical removal of impacted canines may be performed in three ways: with the labial approach, the palatal approach or a combination of the two.

Options of treatment

Retention or leave in situ; indicated when:

✓ The canine is asymptomatic and its extraction may lead to damage to the adjacent teeth.

✓ There is absence of any pathology like infection, abnormal widening of the follicle, resorption of the adjacent roots or any other associated pathology.

✓ Aesthetically acceptable.

The patient should be kept under annual review to verify that these complications have not arisen, the opinion of an orthodontist is important.

Surgical exposure and orthodontic traction; is the procedure that allows natural or orthodontically guided eruption of the impacted teeth, an active collaboration with an orthodontist is essential for planning this procedure. Certain criteria must be fulfilled:

✓ There should be adequate space in the arch to accommodate the tooth.

✓ There should be an unobstructed path of eruption.

✓ After eruption the tooth should be in near to normal position in all planes.

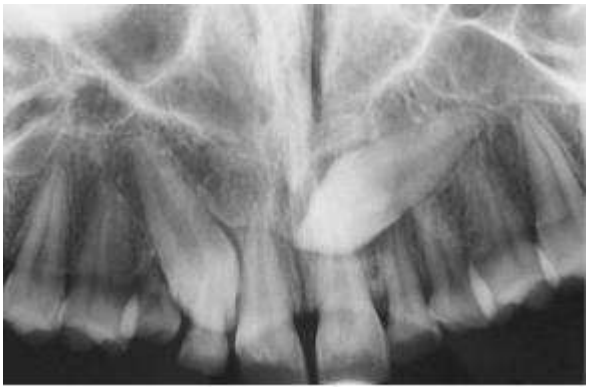
✓ The timing of the procedure should be as close as possible to the normal eruption time.

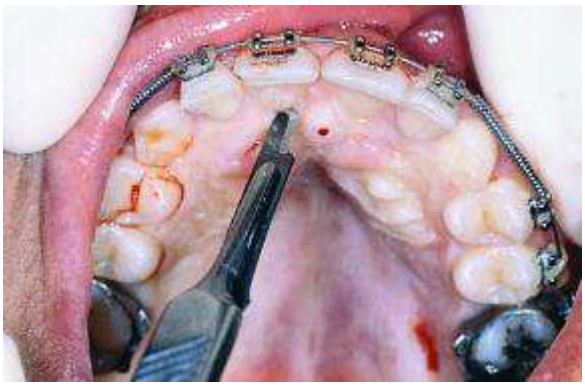
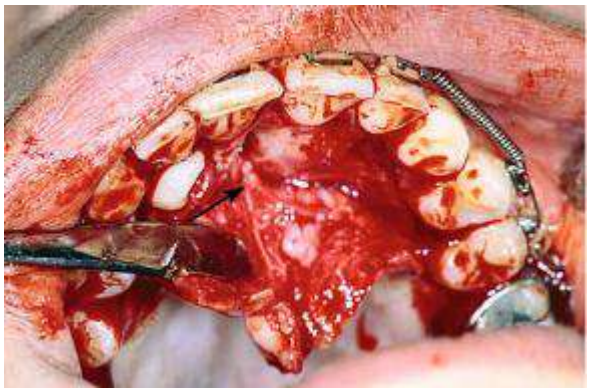
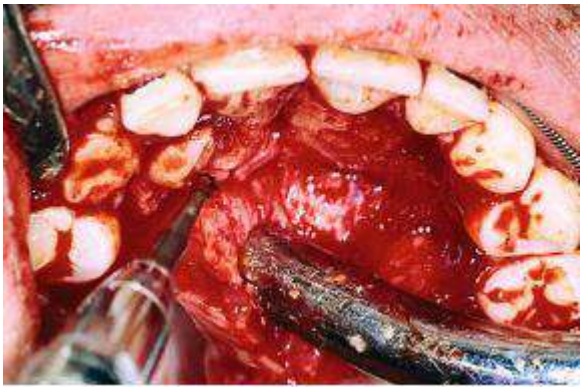
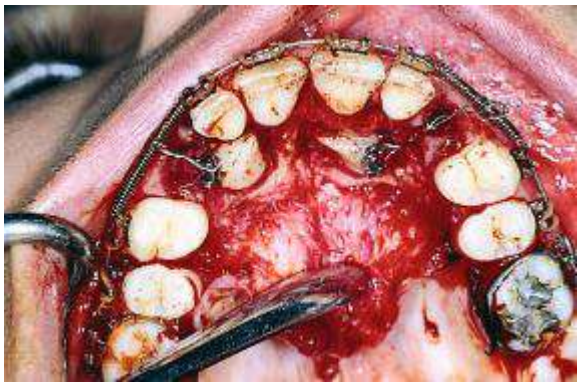
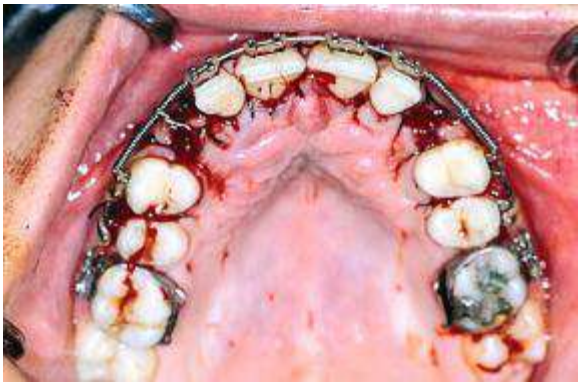
The approach is through a palatal envelope flap, extending from the first molar to the first molar on the other side in bilateral impaction cases, or from the first molar to the first premolar on the other side in unilateral impaction cases.

Buccally impacted teeth are approached through a 3-sided buccal flap, depending on its location.

After reflection of a full mucoperiosteal flap, the crown is exposed conservatively taking care not to expose the cementoenamel junction (CEJ) as this may result in increased incidence of external root resorption.

In palatally positioned canine, a window is excised in the soft tissue before replacing the flap, if the bracket is not attached at the same operation the window is packed with a suitable pack until it epithelializes for 2-3 weeks. In buccal approaches it is more appropriate to suture the flap above the crown (apically repositioned flap) and the area below covered with a pack to ensure that the tooth will erupt into an area of keratinized mucosa.



Transplantation; in this procedure the canine is carefully extracted and transferred to a surgically prepared socket in the dental arch with minimum delay. The transplanted tooth should be splinted in its new position for about a month with an orthodontic appliance.

It is essential to have sufficient space to accommodate the crown of the canine. Success rate is increased when the unerupted teeth still have open apex and

when the handling of the root is kept to minimum to ensure the viability of the cementum and periodontal membrane. Endodontic treatment should be performed as soon as possible after surgery (about 6-8 weeks), periodic follow up is required to allow early detection of root resorption which is common.

Surgical Removal (Extraction); surgical extraction maybe performed when the other options are unavailable. The main indications include:

- ✓ Before construction of a dental prosthesis.
- ✓ To permit orthodontic alignment of other anterior teeth.
- ✓ When there is resorption of the roots of adjacent teeth.
- ✓ When a follicular cyst has developed.
- ✓ Infection although uncommon.

Extraction can be performed with retention of the primary canine with restorative procedures to improve esthetic contour, extraction can also be accompanied with extraction of the primary canine and orthodontic closure of the space by the first premolar. Implant supported crown can also be used to close the space created by extraction of the impacted canine and the primary canine.

Palatally positioned teeth are approached through palatal envelope flap, while buccal teeth are approached through buccal flap.

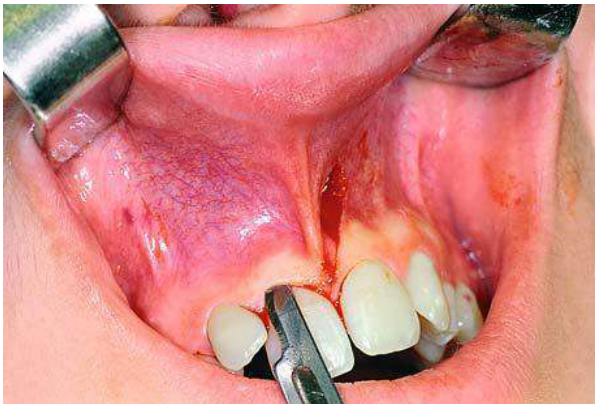
Occasionally tooth sectioning is required after bone removal and the tooth is extracted in segments.

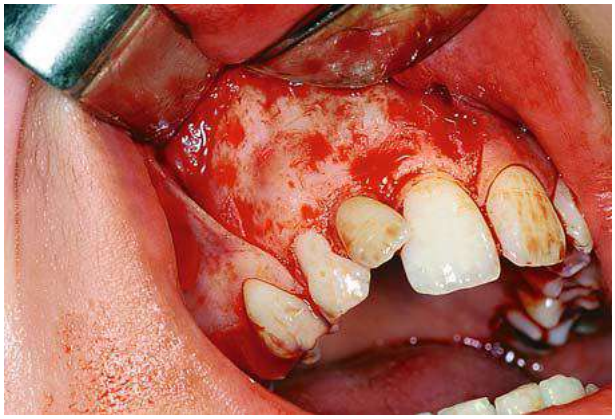
Possible complications include:

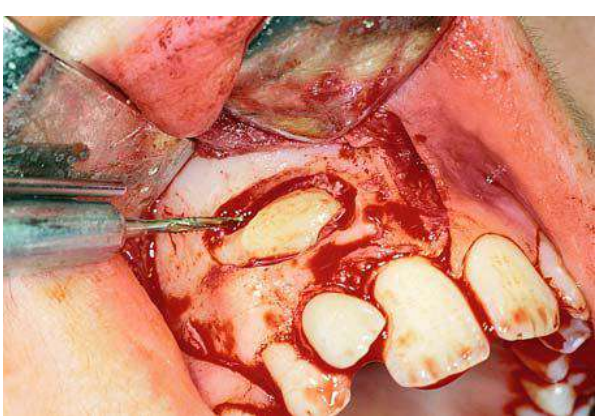
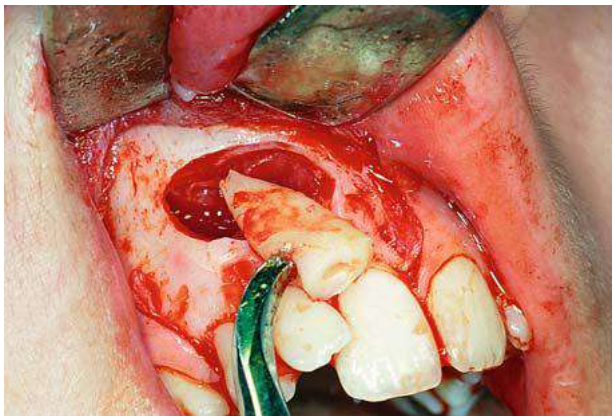
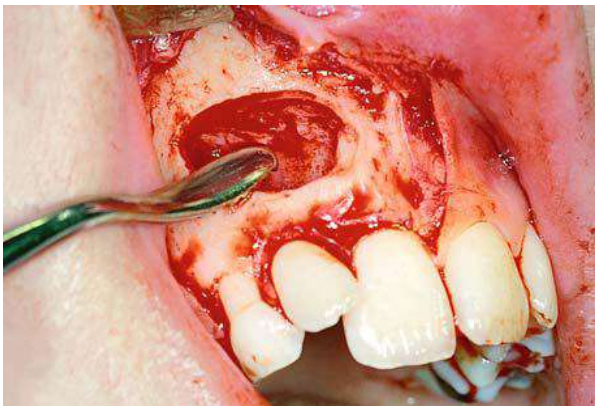
- Palatal hematoma formation, this can be prevented by an acrylic splint to support the soft tissue.
- Perforation into the floor of the nose, but it rarely causes a problem.

Extraction Using Labial Approach

If the impacted tooth is localized labially and is entirely covered by bone, the procedure for its removal is as follows. First a trapezoidal incision is created and the mucoperiosteum is then reflected. The bone covering the tooth is removed using a round bur, with a steady stream of saline solution, until the entire crown of the tooth and part of the root are exposed. A groove is then created at the cervical line using a fissure bur, in order to separate the crown from the root. Separation is achieved using a straight elevator, which is placed in the groove. Upon rotation, the instrument separates the tooth into two segments. The crown is removed first and the root is then luxated, after creating a purchase point on the surface of the root for placement of the tip of the elevator blade. After smoothing the bone, the area is thoroughly irrigated with saline solution, and the wound is sutured. When the impacted tooth is not entirely covered by bone, but the crown of the tooth is covered by overlying soft tissues, removal of the tooth is easier, since it does not have to be sectioned into two pieces.



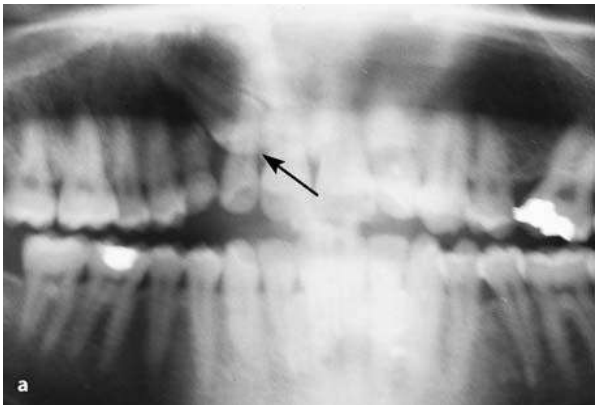


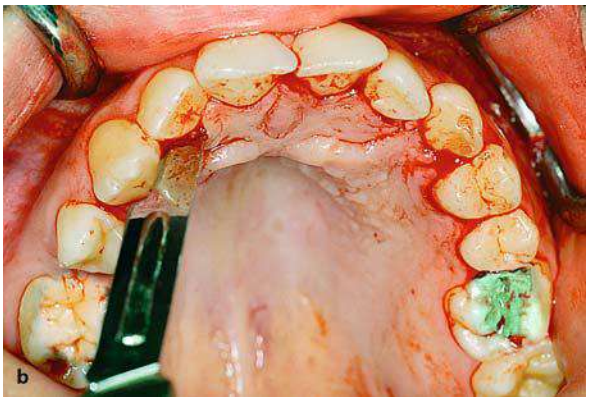




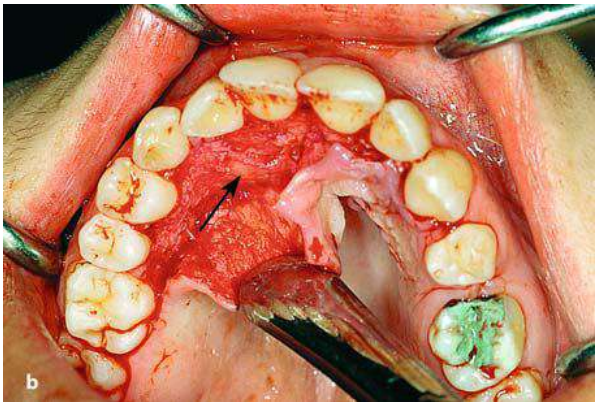
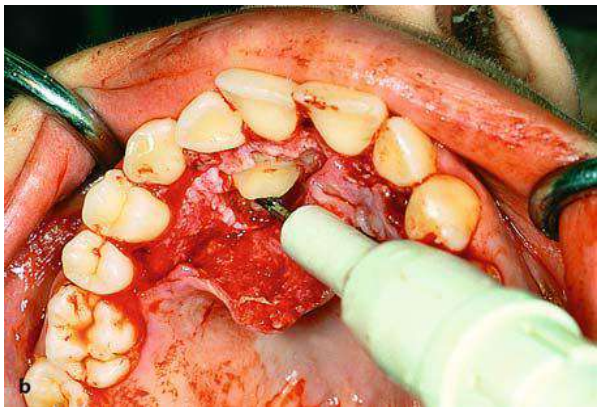
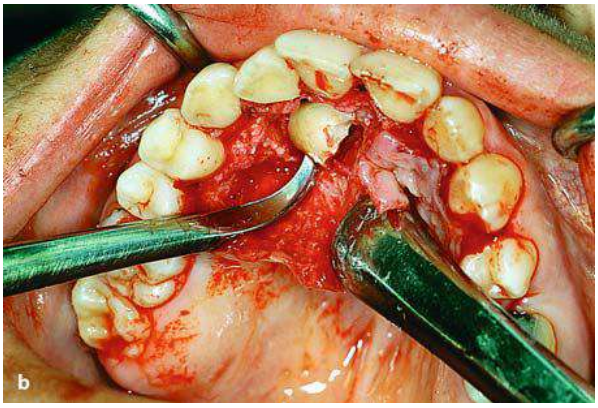
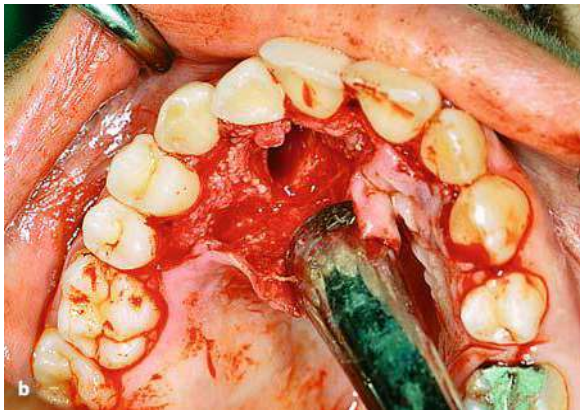
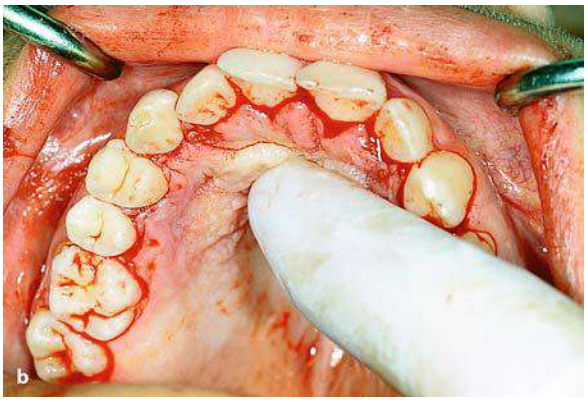
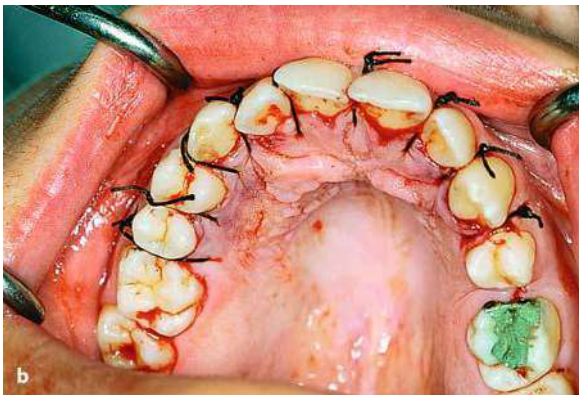

Extraction Using Palatal Approach

When the impacted tooth is positioned palatally , the approach is achieved using a bilateral palatal flap. The incision for creation of the flap begins at the first or second ipsilateral premolar and, after continuing along the cervical lines of the teeth, ends at the first premolar on the contralateral side. After careful reflection of the mucoperiosteum, part of the crown of the tooth may be exposed, or the entire crown may be covered by bone, resulting in protuberance at that site. Either way, enough bone must be removed to expose the entire crown, so that the tooth may be extracted using forceps or an elevator. If the tip of the crown is positioned between the roots of the lateral and central incisors, there is a risk of injuring their roots during the exposure attempt. That is why extraction of the canine must be achieved using the technique of separating the crown from the root. More specifically, a

groove is created on the cervical line of the tooth using a fissure bur and, after placing the elevator blade in the groove created, the instrument is rotated until the crown is separated from the root. The crown is then removed, and, after using the round bur to create a purchase point on the root for placement of the angled elevator's tip, the root is elevated from its bed. After this procedure, the bone edges are smoothed, and the area is thoroughly irrigated with saline solution, while the flap is repositioned and sutured with interrupted sutures bone must be removed to expose the entire crown, so that the tooth may be extracted using forceps or an elevator. If the tip of the crown is positioned between the roots of the lateral and central incisors, there is a risk of injuring their roots during the exposure attempt. That is why extraction of the canine must be achieved using the technique of separating the crown from the root. More specifically, a groove is created on the cervical line of the tooth using a fissure bur and, after placing the elevator blade in the groove created, the instrument is rotated until the crown is separated from the root. The crown is then removed, and, after using the round bur to create a purchase point on the root for placement of the angled elevator's tip, the root is elevated from its bed. After this procedure, the bone edges are smoothed, and the area is thoroughly irrigated with saline solution, while the flap is repositioned and sutured with interrupted sutures .



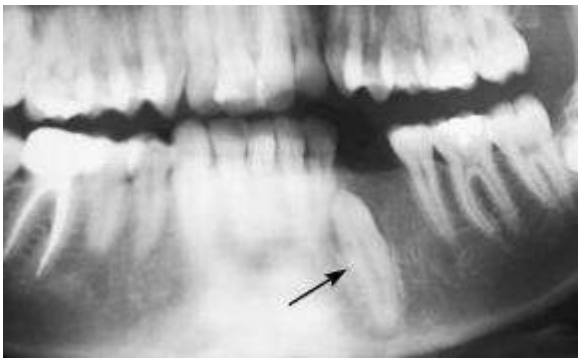


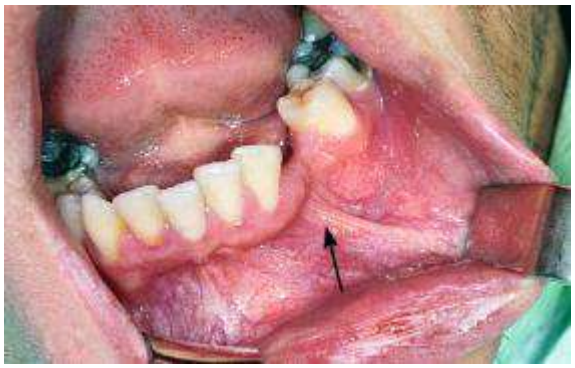







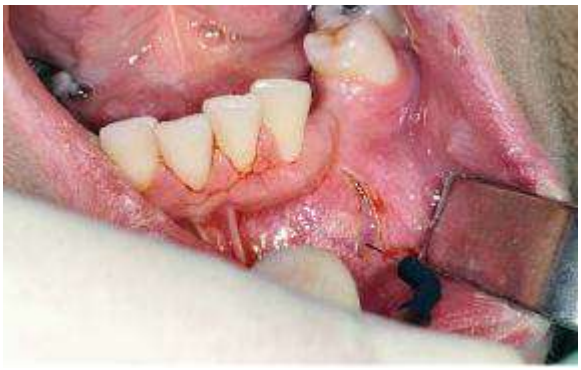
Impacted Mandibular Canine

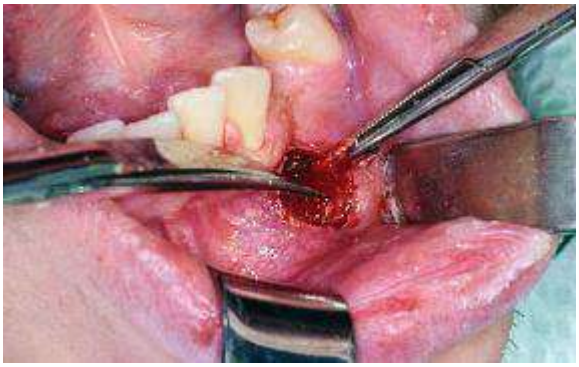
Exposure of the tooth may be achieved in the area locating the impacted canine presents as light protuberance and the crown of the tooth is covered by soft tissue o

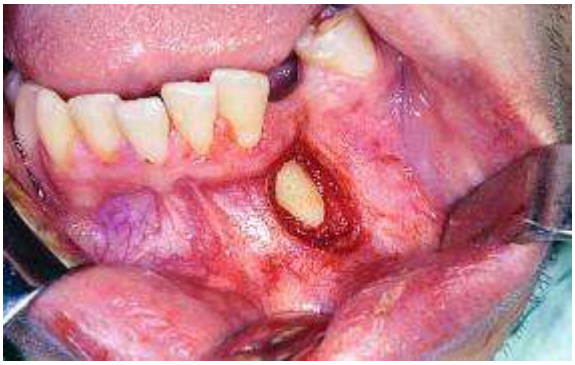
To expose the tooth, first an incision using an electrosurgical blade is made over the crown, and then the soft tissue is excised using scissors and a periosteal elevator, so that exposure is adequate. Afterwards, a surgical dressing is applied to the wound until the day the orthodontist bonds the bracket for traction of the tooth to its nor-malposition in the dental arch .The second technique involves exposure of the crown by creating a f lap. More specifically, after creating an L-shaped incision, a small flap is reflected and the crown of the impacted tooth is exposed. The tooth is then dried and after the orthodontist has placed the bracket on the crown of the tooth, the flap is repositioned and the wound is sutured.

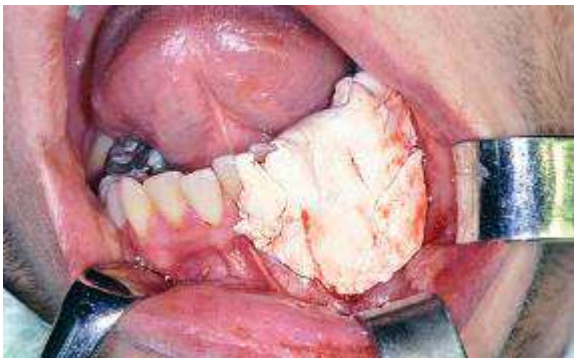






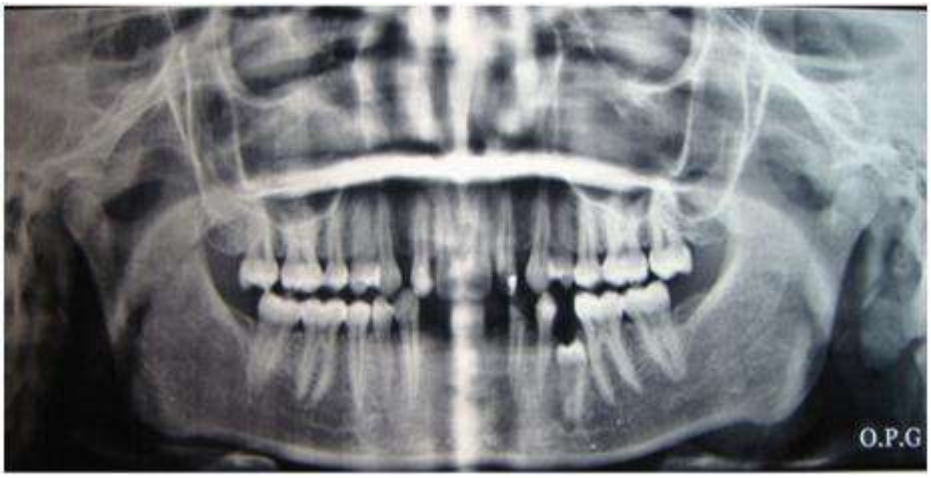


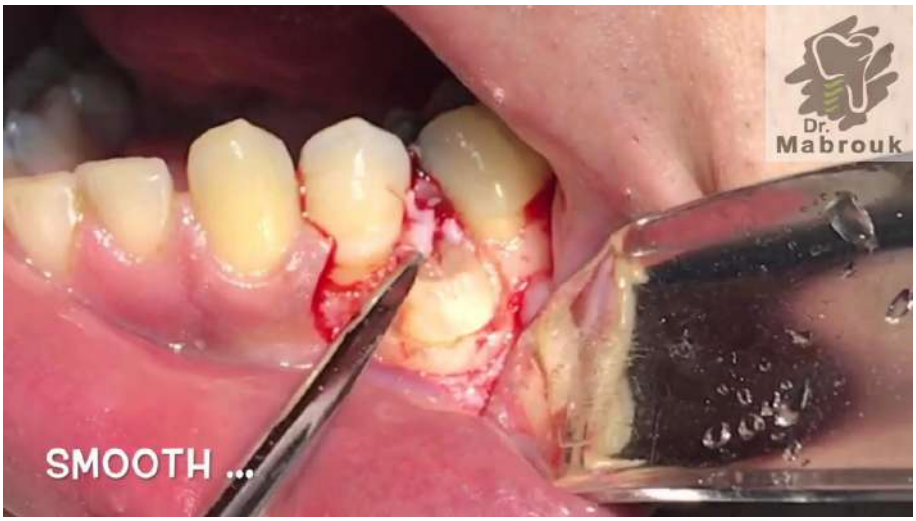




Impacted lower premolars

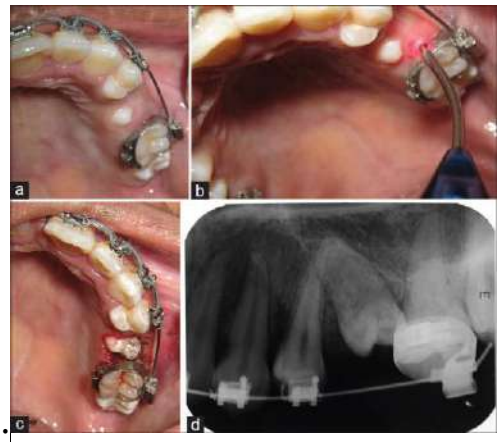
It occurs mostly due to loss of space by drifting forward of the first permanent molar after early extraction of the second deciduous molar. Localization is by periapical film, OPG with occlusal view to demonstrate the buccolingual position or CBCT. Removal is by raising a buccal flap, with preservation of the mental nerve, bone removal, sectioning of the tooth if needed and extraction of the tooth. Consultation with orthodontist before extraction is essential.



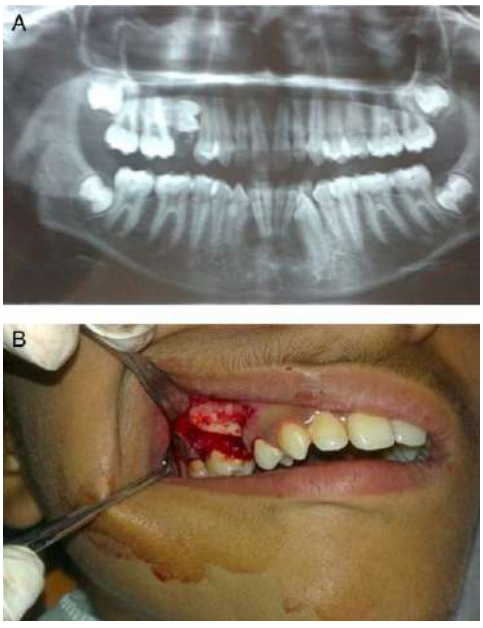


Impacted maxillary premolars

It is usually impacted with its crown palatally, or it may be within the arch between adjacent roots. It can be partially erupted, completely buried or the crown may be wholly exposed, in the latter case extraction is easy with an elevator or forceps. Completely impacted teeth require a palatal envelope flap extending from the second molar to the lateral incisor on the same side, bone removal and extraction of the tooth

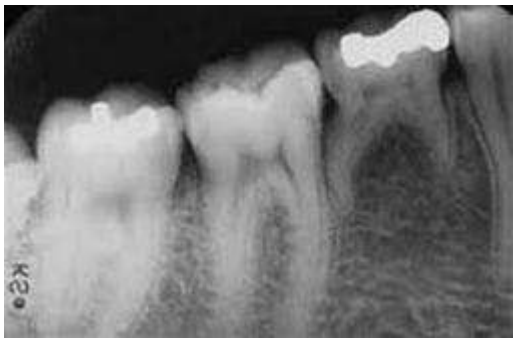


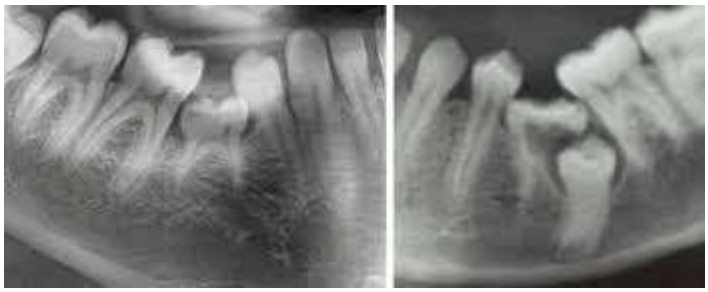
Buccal approach is needed in cases where the tooth is within the arch between the standing teeth, sectioning of the tooth is needed when the root is curved. Care is taken not to damage the adjacent teeth.



Buried deciduous molars

These are usually ankylosed and should be removed surgically through buccal approach, bone removal and tooth sectioning if necessary.



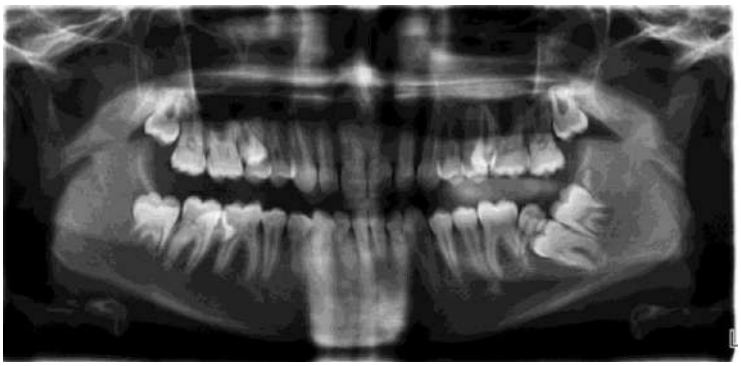


Supernumerary teeth

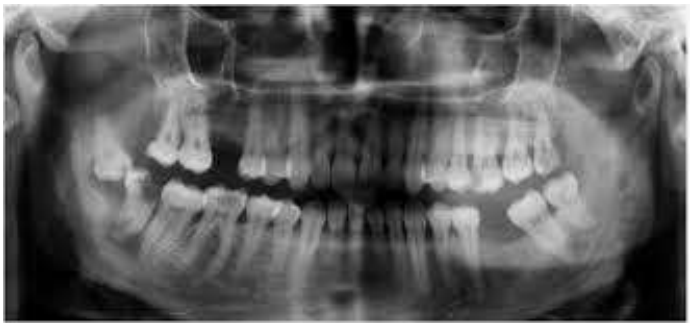
These are more in the males than in the females, they can be present in the primary dentition as well as in the permanent dentition. Supernumerary teeth can be classified according to their position into:

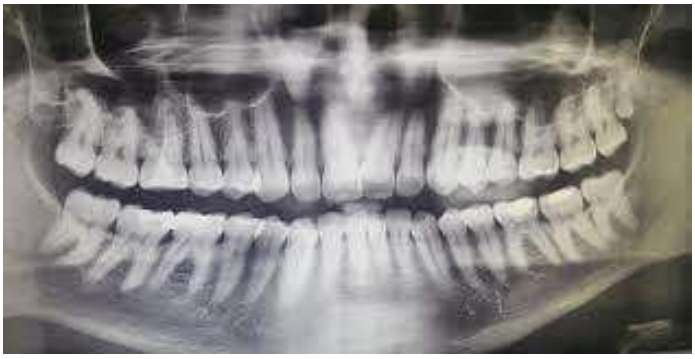
Mesiodens; is situated in the premaxilla in the midline and it is commonly conical, it can have a horizontal or inverted position. Supplemental teeth may also occur in the anterior maxillary region.

Paramolar; appear in the premolar or molar region and is situated buccally to the teeth, they can be conical or supplemental.



Distomolar; appear as a fourth molar usually distal to the standing molars and they are either normal or smaller in size.



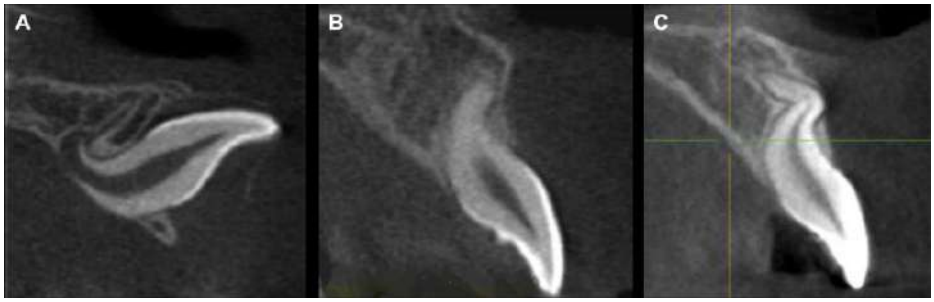


According to the shape they can be either; **conical (peg-shaped)** or **supplementary**; which have the shape and size of a normal tooth or they can have conventional shape with smaller or larger size.

Supernumerary teeth can have no effect on other standing teeth or they can cause failure of eruption of the other teeth, crowding, malposition or misalignment, resorption of the roots of the adjacent teeth or they can be associated with other pathologies (e.g. cysts). Erupted supernumerary teeth are extracted easily especially if they are conical in shape.If they need removal they should be localized accurately using periapical films (buccal object rule may be applied), vertex occlusal view or CBCT. OPG is needed to determine the vertical position of the tooth and its position in relation with the floor of the nasal cavity or maxillary sinus. They are approached palatally through palatal flap or buccally, bone removal and tooth sectioning may be needed, sometimes combined palatal and buccal approach is necessary.

Dilacerated incisors

Trauma to the deciduous incisors especially in the 2-3 years of age can cause damage to the underlying permanent incisor tooth germ causing root development to take place at an angle. Exposure and orthodontic traction can be performed if possible, but if not, these teeth should be removed and the lateral incisors allowed filling their space. It is essential to seek the opinion of an orthodontist.



Corticotomy-assisted orthodontic treatment

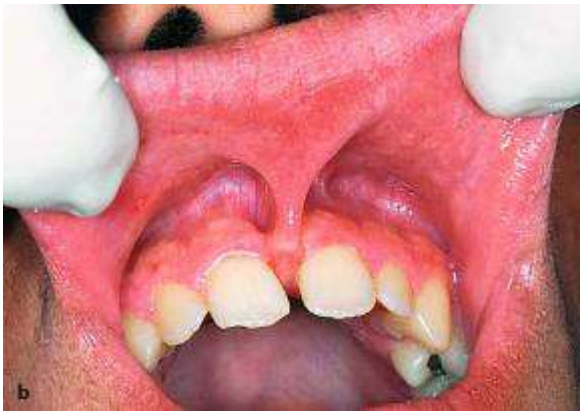
It can be defined as a linear cutting technique in the cortical plates surrounding the teeth to produce accelerated tooth movement. This process differs from the osteotomy, in which cortical and cancellous bone is cut for the purpose of repositioning blocks of bone with associated teeth. Corticotomy is contraindicated in patients with active periodontal disease or gingival recession. The traditional corticotomy procedure entails raising full-thickness buccal or buccal and palatal/lingual mucoperiosteal flaps and performing vertical linear interradicular corticotomy cuts (about 0.5 mm in depth), with or without joining horizontal subapical corticotomy cuts, or by drilling multiple holes that penetrated the cortical plate instead of linear cuts.After surgery orthodontic force can be applied either immediately or within 2 weeks after surgery.

Frenectomy

In many cases, the placement of a complete denture of the maxilla, or orthodontic procedures in younger persons requires the removal of the labial frenum, especially if it is hypertrophic . Also, in the mandible, the lingual frenum may create problems, causing partial or complete ankyloglossia. This case is due to attachment of the frenum to the floor of the mouth or to the alveolar mucosa. It may even be the result of an extremely short frenum that is connected to the tip of the tongue. Ankyloglossia greatly limits movements of the tongue, resulting in speech difficulties.

Maxillary Labial Frenectomy

Excision of the labial frenum is easy, within the reach of the general practitioner, andmay be performedwith various techniques. The method usually employed is that of excision using two hemostats. In this case, the procedure used is as follows. After local anesthesia, the lip is pulled upwards, and the frenum is grasped using two curved hemostats, which are positioned at the superior and inferior margins.The lip is then further retracted and a thin scalpel blade incises the tissue found behind the hemostat, first behind the lower hemostat and then behind the upper hemostat . If the frenum is hypertrophic and there is a large space between the central incisors, the tissues found between and behind the central incisors are also removed. Interrupted sutures are placed along the lateral margins of the wound in a linear direction, after the mucosa of the wound margins is undermined using scissors .

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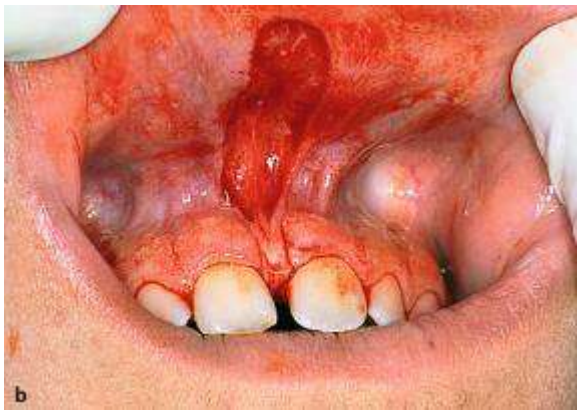
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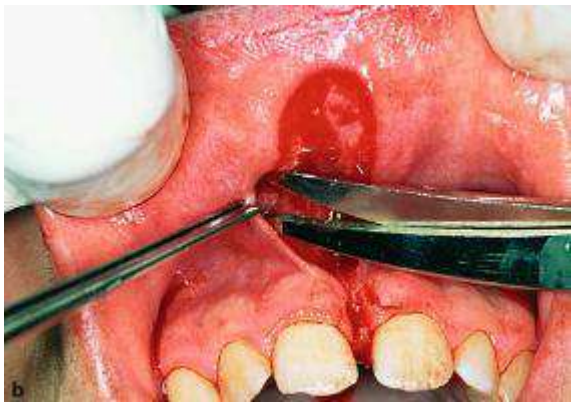
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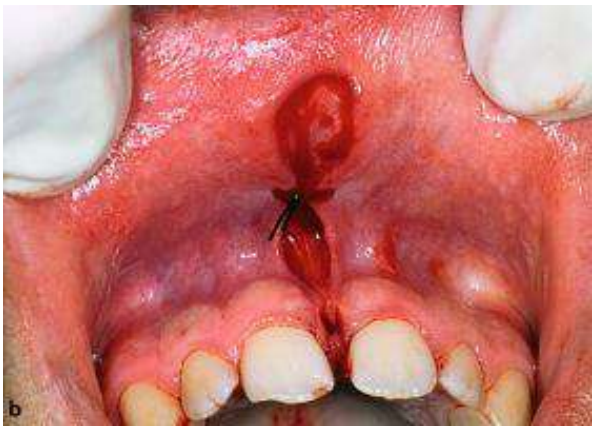
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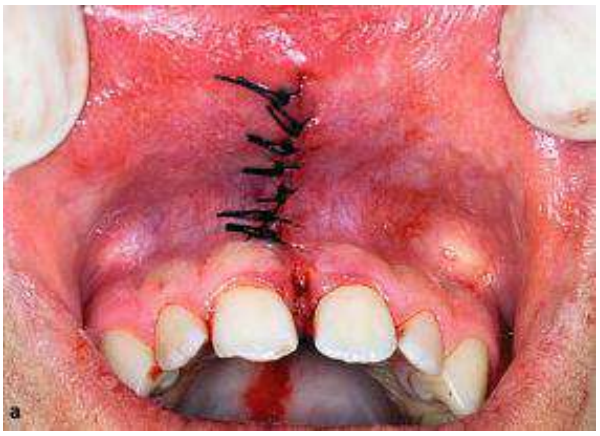
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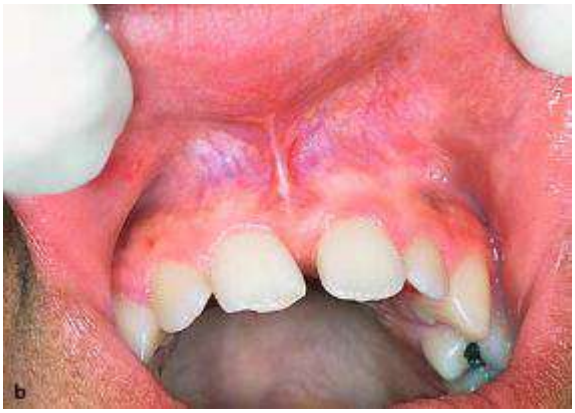
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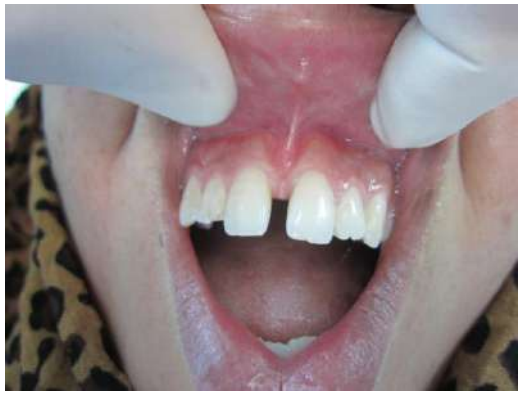
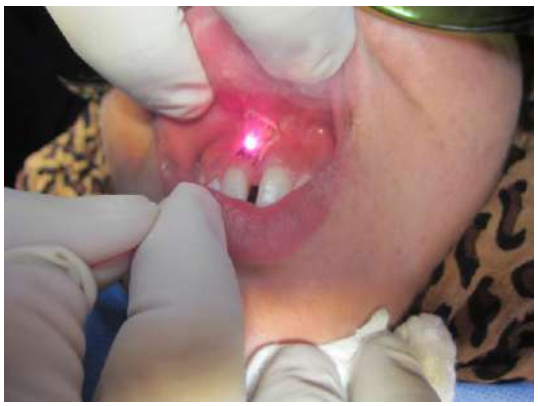
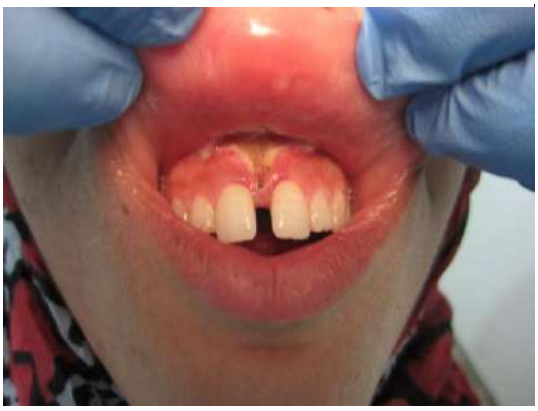
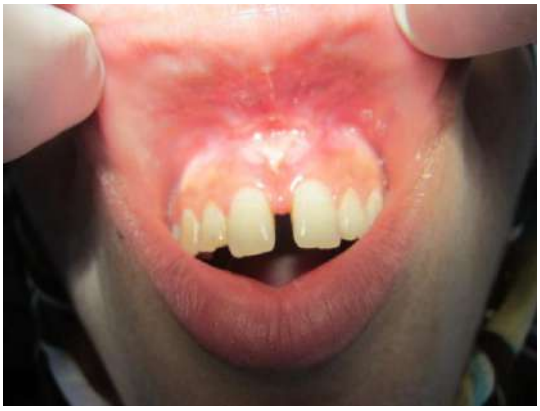
The laser-assisted frenectomy

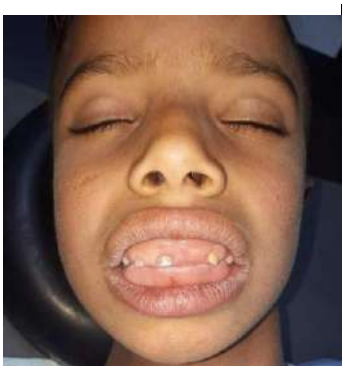
Frenectomy can be performed by laser. Diod laser, Nd:YAG laser, Er:YAG laser and CO_2 Laser have been reported. The main advantages of laser are; less bleeding during surgery, no need for suturing or periodontal dressing, with minimal postoperative swelling and discomfort.

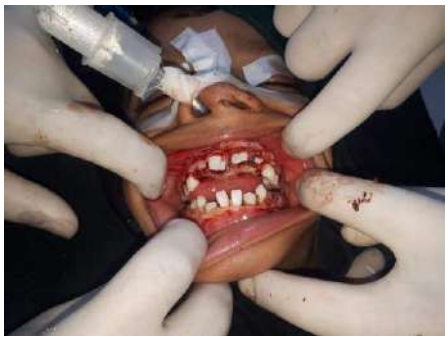
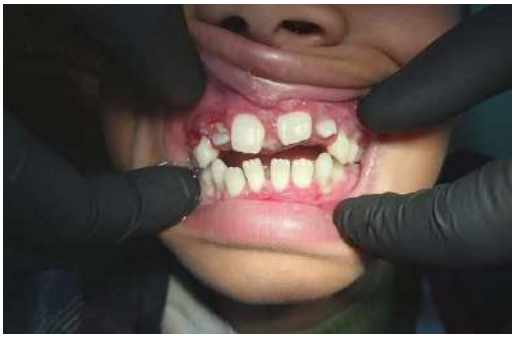
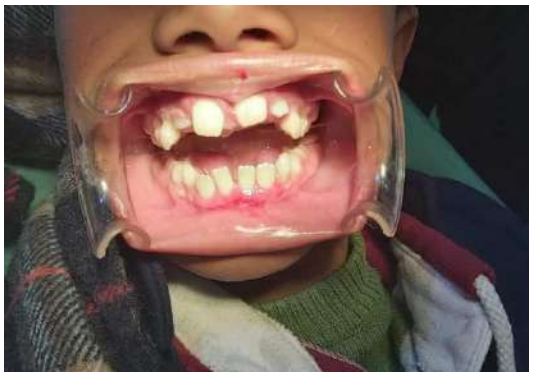





Gingival Fibromatosis

This is a benign condition, which is characterized by slow progressive swelling of the gingivae proper (attached gingivae) and alveolar mucosa (loose gingivae). The lesion may be generalized or localized and is due to hereditary or acquired causes. Clinically, gross hyperplasia of the gingivae is observed, which may partially or completely cover the crowns of the teeth, depending on the case. The surface of the gingivae is lobular, reddish, and firm to palpation, while the inflammation and bone resorption vary. Treatment is surgical and consists of

segmental excision of the gingivae using cconventional surgical technogue or by laser application**.**



Suggestive Reading

Fragiskos D. Fragiskos. Oral Surgery, Springer-Verlag Berlin Heidelberg 2007

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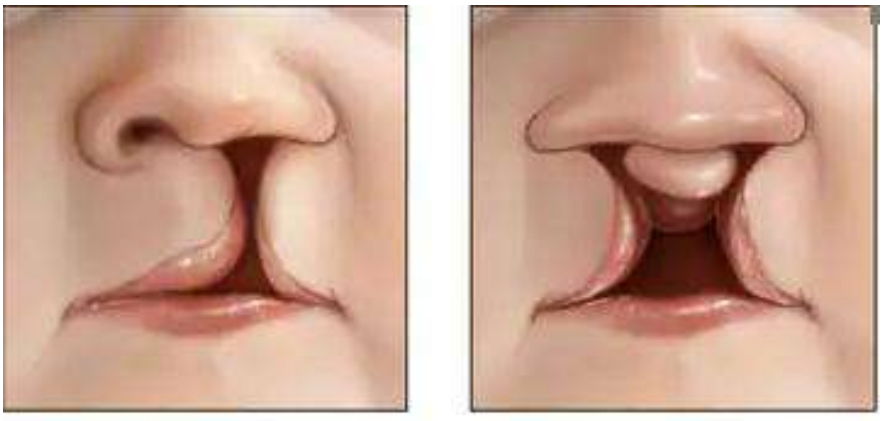
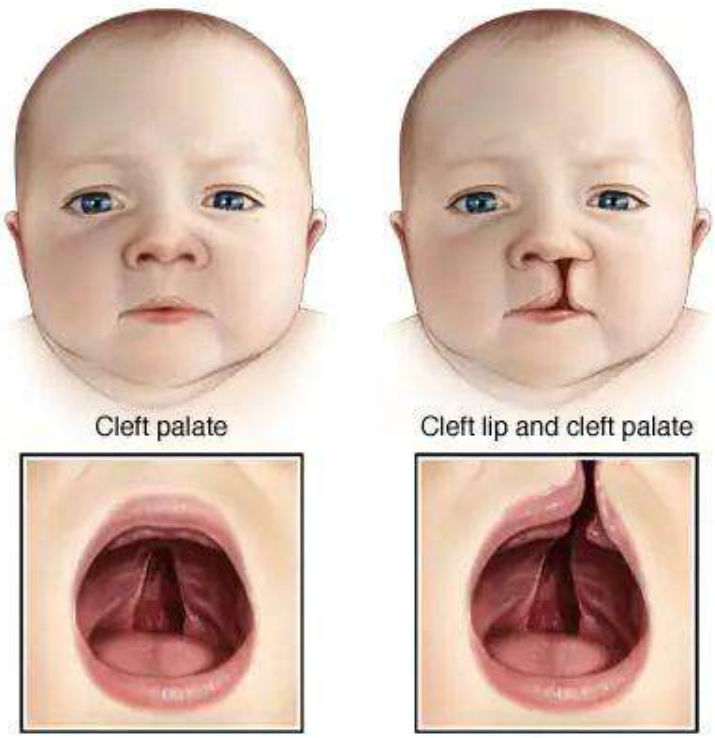
BDS, CDI, MSc, FIBMS, FIAOMS, DLMFS

Cleft Lip and Palate

Orofacial clefts include a range of congenital deformities most commonly presenting as cleft lip with or without cleft palate or isolated cleft palate. Cleft lip and cleft palate are openings or splits in the upper lip, the roof of the mouth (palate) or both. Cleft lip and cleft palate result when facial structures that are developing in an unborn baby don't close completely. Cleft lip and cleft palate are among the most common birth defects. They most commonly occur as isolated birth defects but are also associated with many inherited genetic conditions or syndromes. Having a baby born with a cleft can be upsetting, but cleft lip and cleft palate can be corrected. In most babies, a series of surgeries can restore normal function and achieve a more normal appearance with minimal scarring. Cleft lip and palate (CLP) is a three-dimensional deformity involving soft and skeletal tissue that changes with growth and function. Its comprehensive treatment requires thoughtful consideration of the anatomic complexities of the deformity and the delicate balance between intervention and growth.



Unilateral cleft lip Bilateral cleft lip



History of the Procedure

Chinese physicians were the first to describe the technique of repairing cleft lip. The early techniques involved simply excising the cleft margins and suturing the segments together. The evolution of surgical techniques during the mid-17th century resulted in the use of local flaps for cleft lip repair. These early descriptions of local flaps for the treatment of cleft lip form the foundation of surgical principles used today. Tennison introduced the triangular flap technique of unilateral cleft lip repair, which preserved the Cupid's bow in 1952. The geometry of the triangular flap was described by Randall, who popularized this method of lip repair

Epidemiology

Cleft lip and palate are hereditary diseases in which environmental and genetic factors together play a role, leading to different clinical outcomes. Generally, cleft lip and palate cases are divided into two groups: cleft lip with/without cleft palate (CL/P) and isolated cleft palate (CP). Depending on the presence of certain anomalies, the cases are also classified as syndromic and non-syndromic (nsCLP) clefts.

Approximately 70% of CL/P patients and 50% of CP patients are non-syndromic. In the remaining patients, a wide range of malformation syndromes can manifest, including chromosomal anomalies and teratogens as well as more than 500 defined Mendelian syndromes

Children born with cleft lip and palate develop complications such as nutritional difficulties, respiratory failure, and hearing and speech difficulties. In addition to health problems, affected individuals face long-term social difficulties such as acceptance. Even if surgical intervention is performed in the early period, deformities due to scarring and abnormal facial development cause persistence of functional and psychosocial problems throughout the patient's life.

It is necessary to understand the factors underlying these defects in order to foresee the long-term course of development of individuals with a cleft lip and palate that should be treated using a multidisciplinary approach including a plastic surgeon, otolaryngologist, speech therapist, audiologist, orthodontist, psychologist, social worker, and nurse. Epidemiological studies and observational reports have shown that folic acid supplements taken by the mother before pregnancy have a protective effect in reducing the incidence of cleft lip and palate, whereas smoking

and alcohol consumption before pregnancy increase the risk of cleft lip and palate formation

The estimated incidence of CLP ranges from 1:500 live births to 1: 2000 live births, the incidence is highest in Asians (1:500) followed by whites (1:1000) while the lowest incidence is in African-Americans (1:2000), but this racial variation is not observed in cases of isolated cleft palate (CP) with reported incidence of 1:2000 live births.

- Isolated cleft lip (CL) is reported in 21%-25% of the cases.
- Isolated CP is reported in 33%-40% of the cases.
- CLP is reported in 35%-46%.
- Unilateral clefts are 9 times more common than bilateral clefts and they are more frequent on the left side than on the right.
- Males are predominant in CLP population, whereas isolated CP occurs more commonly in females.
- CLP can be associated with syndromes such as Van der Woude syndrome, hemifacial microsomia, velocardiofacial syndrome or DiGeorge syndrome, ectrodactyly-ectodermal dysplasia-clefting syndrome, Stickler syndrome, trisomy 13 and trisomy 18.

Aetiology

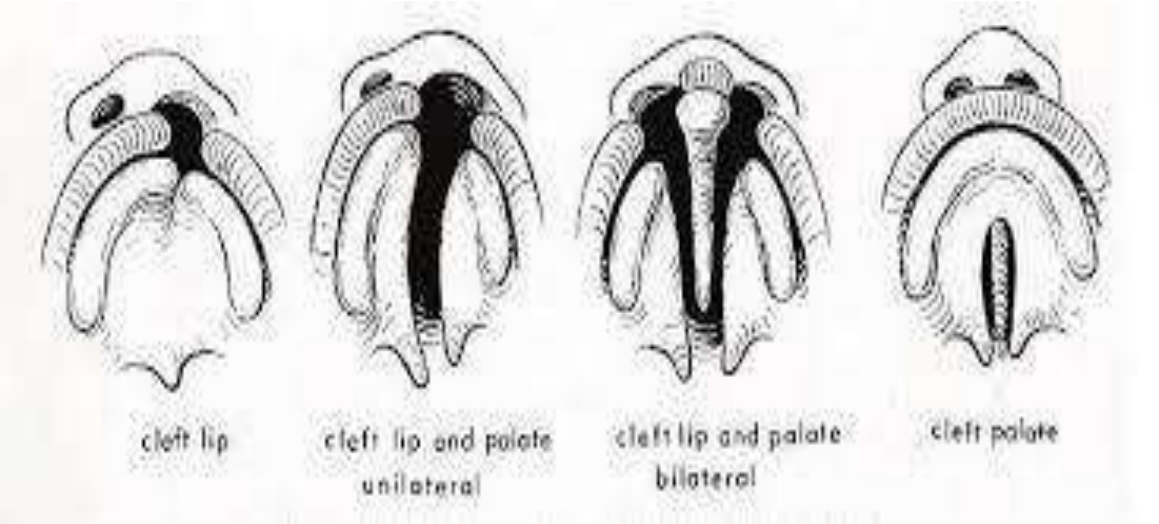
Cleft lip and or palate occurs secondary to a failure of fusion of the medial nasal swellings with the maxillary swellings. The vast majority of cases of cleft lip and/or palate are idiopathic; however, a number of drugs have been implicated, including phenytoin, carbamazepine, steroids and diazepam. In addition, chromosomal disease and malformation syndromes are also associated with facial

clefting. In trisomy 13 the facial clefting is usually median (midline cleft) or bilateral cleft lip and palate, and occurs in up to 65% of cases. Facial clefting is seen in 30% of fetuses with triploidy, up to 15% of fetuses with trisomy 18 and 0.5% of fetuses trisomy 21.

Classification

Cleft lip is classified as being complete or incomplete based on the extent of the cleft, as well as unilateral or bilateral depending on whether it affects one or both sides. An incomplete cleft of the lip does not involve the complete thickness of the lip, but has a band of tissue intact across the cleft. Conversely, a complete cleft of the lip involves the entire vertical thickness of the lip and is more often associated with a cleft of the alveolus.

Cleft palate is defined in a similar fashion, as either complete or incomplete as well as unilateral or bilateral. A complete cleft of the palate involves both the primary and secondary palates as well as the alveolus. There is also the less common possibility of an isolated cleft palate which usually involves the secondary palate, posterior to the incisive foramen.



Successful management of the child born with CLP requires coordinated care provided by a number of different specialties including oral and maxillofacial surgery, plastic surgery, otolaryngology, genetics, speech therapy, orthodontics, prosthodontics and others.

Specific goals of treatment include the following:

- ✓ Normalized esthetic appearance of the lip and nose
- ✓ Intact primary and secondary palate
- ✓ Normal speech, language, and hearing
- ✓ Nasal airway patency
- ✓ Class I occlusion with normal masticatory function
- ✓ Good dental and periodontal health
- ✓ Normal psychosocial development

The management of CLP patients can be divided into:

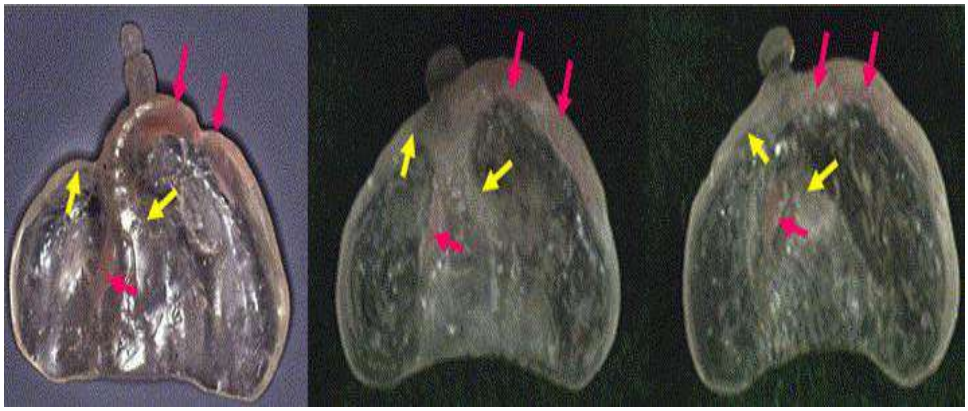
Preoperative management

Feeding : One of the major concerns during this phase of management is feeding, this concern is not very critical in children born with isolated CL, as they can feed quite well and even have the opportunity to breastfeed in most instances. Infants with cleft palate, on the other hand, can have difficulty in feeding due to the inability to form an adequate seal between the tongue and palate for creation of sufficient negative pressure to suck fluid from a bottle. Specialized nipples and bottles are necessary to improve feeding.

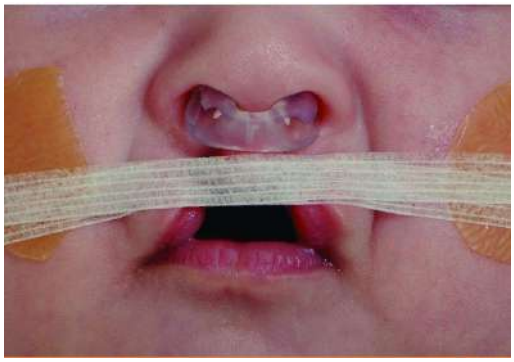
Presurgical orthopedics (PSO): PSO entails using devices to mold the perioral structures of the infant with a CLP to reposition the nasolabial and maxillary segments closer to each other. It is mainly used in the first few weeks after birth and in the months prior to palate repair.

PSO treatment can be achieved by intraoral or extraoral devices or appliances or a combination of the two, examples of PSO are:

- Alveolar molding



- Lip strap/lip taping



- Nasoalveolar molding (NAM)

Treatment planning and timing

The timing of CLP repair is controversial. The decision to surgically manipulate the tissues of the growing child should take into account the possible growth restriction that can occur with early surgery. Historically the anesthetic risk-related data suggested that the safest time period for surgery in this population of infants could be outlined simply by using the "*rule of 10's*." This referred to the idea of delaying lip repair until the child was at least 10 weeks old, 10 pounds in weight, and with a minimum hemoglobin value of 10 g/dl.

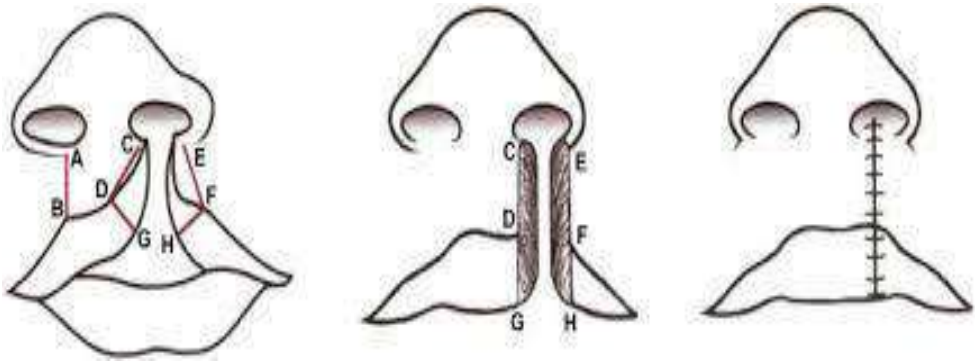
Procedure	Timing
CL repair	After 10 weeks
CP repair	9-18 months
Pharyngeal flap or pharyngoplasty	3–5 years or later based on speech development
Maxillary/alveolar reconstruction with bone grafting	6–9 years based on dental development
Cleft orthognathic surgery	14–16 years in girls, 16–18 years in boys
Cleft rhinoplasty	After age 5 years but preferably at skeletal maturity; after orthognathic surgery when possible
Cleft lip revision	Anytime once initial remodeling and scar maturation is complete but best performed after age 5 years

Primary operative management *(Surgical procedures for CL & CP)*

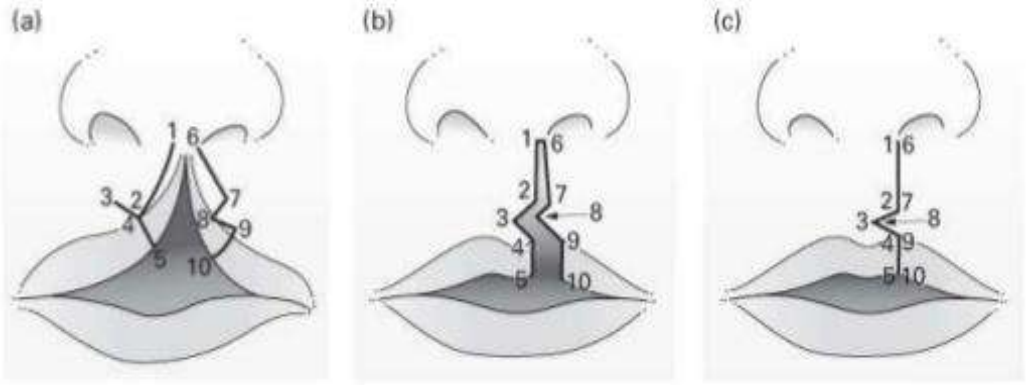
Surgical procedures for CL

The basic premise of the repair is to create a three-layered closure of skin, muscle, and mucosa that approximates normal tissue and excises hypoplastic tissue at the cleft margins. Numerous techniques, as well as modifications to popular techniques, have been extensively described in the literature

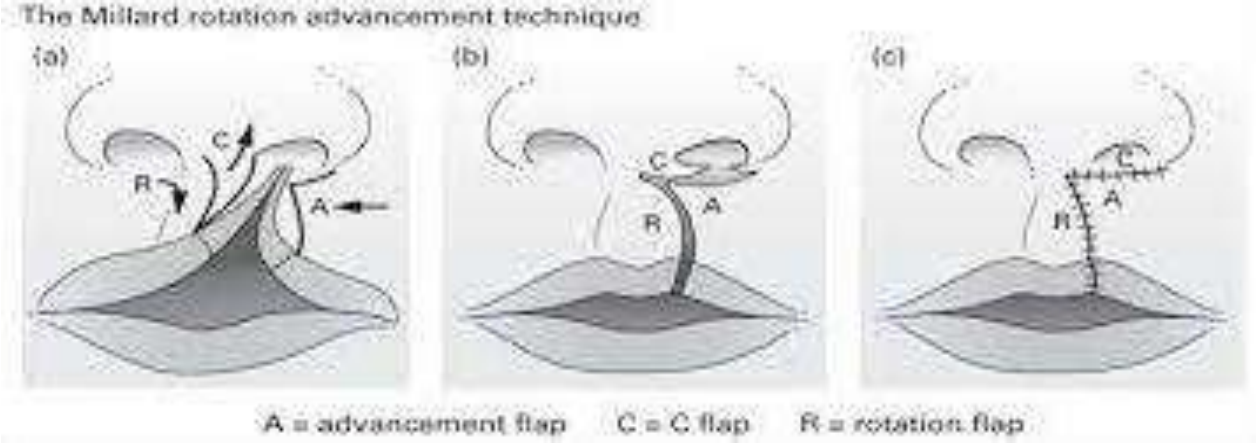
- **Straight line repair :** Straight line closure, or the Rose-Thompson closure may be indicated for microform clefts and it is rarely used as primary technique for cleft repair as it results in notching of the lip and vertical scar contracture.



- **Tennison-Randall triangular flap repair:** It utilizes the interdigitation of triangular flaps (geometric design). The concept underlying the technique can be similarly compared to a Z-plasty reconstruction of the lip. This technique is based on careful measurements of specific landmarks.



- **Millard's rotation-advancement flap repair:** It is the most prevalently used technique in cleft lip repair. The technique utilizes downward rotation of the superiorly displaced medial lip segment with advancement of the lateral lip flap to correct the defect below the nose. Many modifications for the original procedure were described.

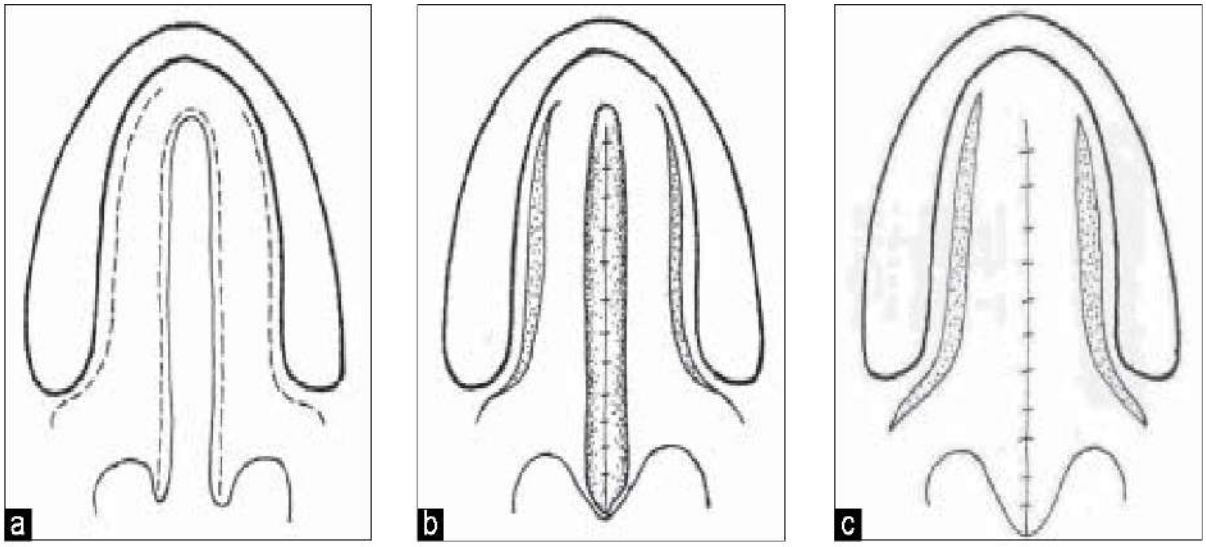


Surgical procedures for CP

CP repair requires the mobilization of multilayered flaps to reconstruct the defect in a layered fashion by first closing the nasal mucosa and then the oral mucosa, the surgeon must also reconstruct the musculature of the velopharyngeal mechanism. Therefore, the soft palate is closed in three layers by approximating the nasal mucosa, velar musculature, and the oral mucosa. The hard palate portion is closed in two layers using nasal mucosa flaps and then oral mucosa flaps. Both the hard and soft palate repairs must be done in a tension-free manner to avoid wound breakdown and fistula formation.

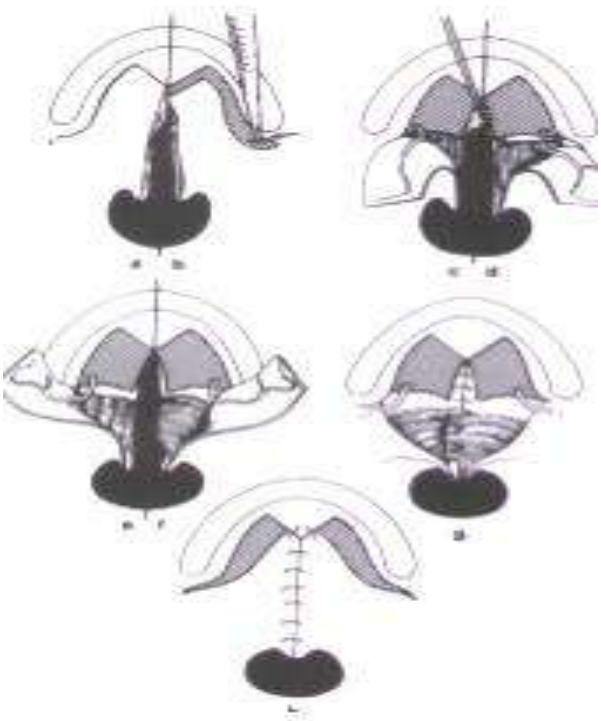
The von Langenbeck technique

It involves creating bipedicled mucoperiosteal flaps on both sides of the cleft. The nasal side of the cleft is closed first, using redundant mucoperiosteum from the incision along the cleft edge. Then the bipedicled flaps are approximated to cover the oral surface of the cleft.



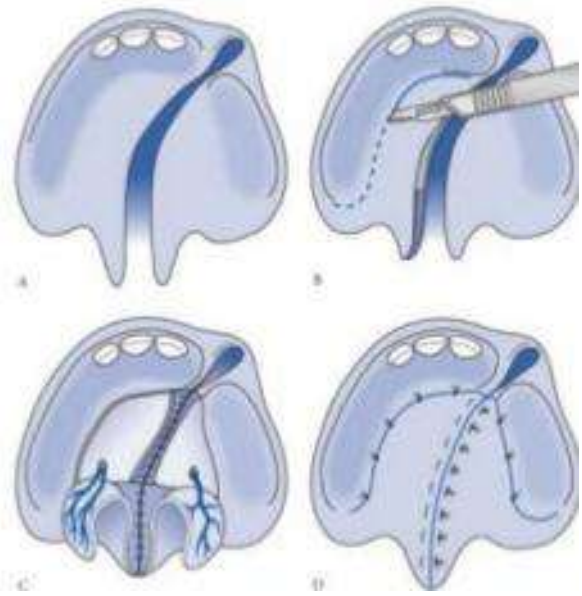
V-Y pushback technique

Many authors believed that the von Langenbeck procedure resulted in insufficient length of the soft palate and advocated the V-Y pushback technique. It has the advantage of lengthening the palate and repositioning the levator muscle in a more favorable position. However, this technique involves extensive dissection and the denuded palatal bone from which the mucoperiosteal flaps are raised adversely affects midfacial growth in cleft palate patients.



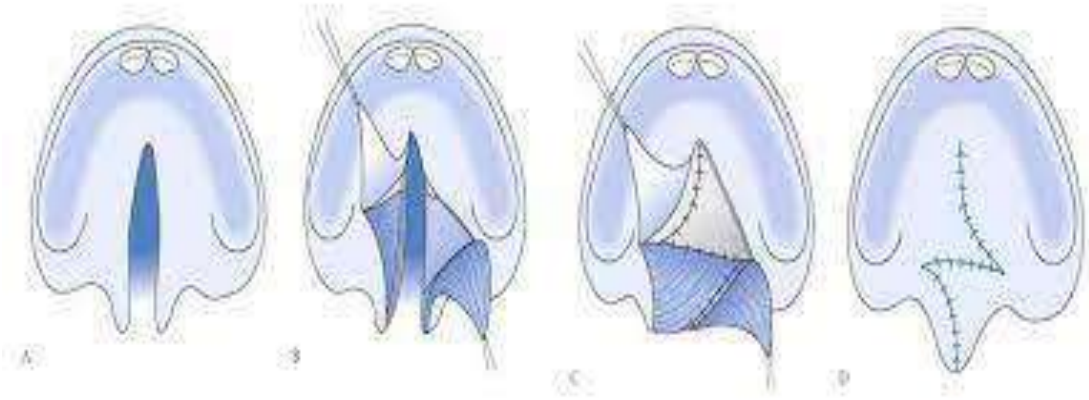
Two-flap technique

In this procedure a full-thickness mucoperiosteal flap is elevated on each side of the cleft, which preserves the palatal neurovascular bundle.



The Furlow technique

It essentially consists of repairing palatal clefts using Z-plasties of the oral and nasal mucosa. The theoretical advantage is that the soft palate may be lengthened while preventing longitudinal scar contracture and palatal shortening.



Secondary operative management *(Alveolar bone grafting)*

Goals of alveolar bone reconstruction

- To provide bone support and adequate attached gingival width for teeth adjacent to the cleft.
- To close the remaining oronasal fistula.
- To improve support of the nasal alar base and lip on the affected side(s).
- To allow normal eruption of the permanent teeth in the cleft area and providing sufficient bone for the placement of dental implants, where needed.
- To create an appropriate ridge form to allow for optimization of orthodontic care and dental alignment.
- To allow for stabilization of the premaxillary segment and to provide continuity of the maxilla as a whole.
- To improve nasal symmetry.
- To provide support for the upper lip.

Timing of the alveolar bone reconstruction was one of the most controversial issues. Generally it can be grouped according to timing into:

Primary (early) grafting; which was defined as that performed simultaneously with lip repair or as grafting performed before the palate is repaired at age younger than 2 years.

Secondary (delayed) grafting; which can also be divided into:

- Early secondary; before the eruption of the permanent incisor teeth (3-6 years).
- Secondary; before the eruption of the maxillary canine, (6-12 years).
- *Late grafting*; after 12 or 13 years of age, after the eruption of the permanent canine.

Sources of bone graft materials

- Iliac crest;
- Cranium;
- Tibia
- Mandibular symphysis.
- Bone graft substitutes

Complications

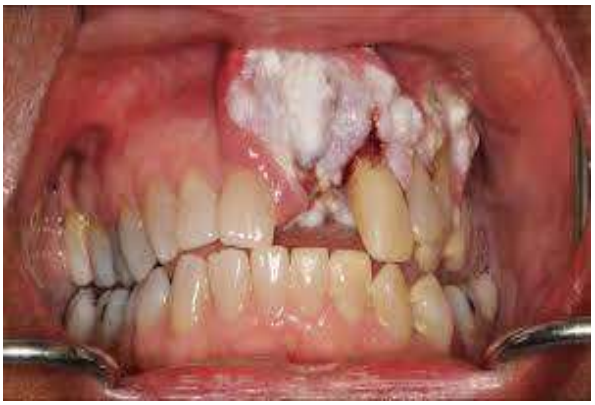
- Wound dehiscence which is managed by debridement and antimicrobial mouth washes with or without systemic antibiotics.
- Infection which is managed by conservative debridement, daily irrigation and packing with oral antibiotics.
- Persistent fistula which may require subsequent procedures.
-

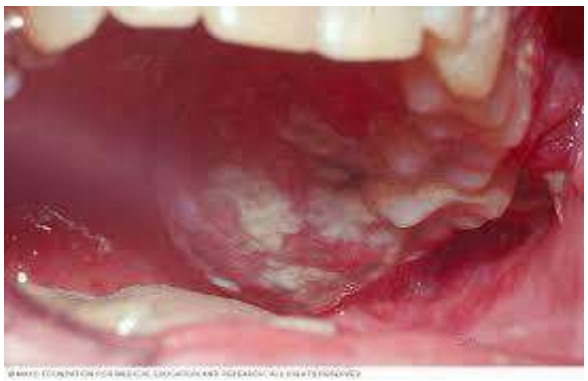
Suggested Reading

Peter Ward Booth, Maxillofacial Surgery: Churchill Livingstone, 2006

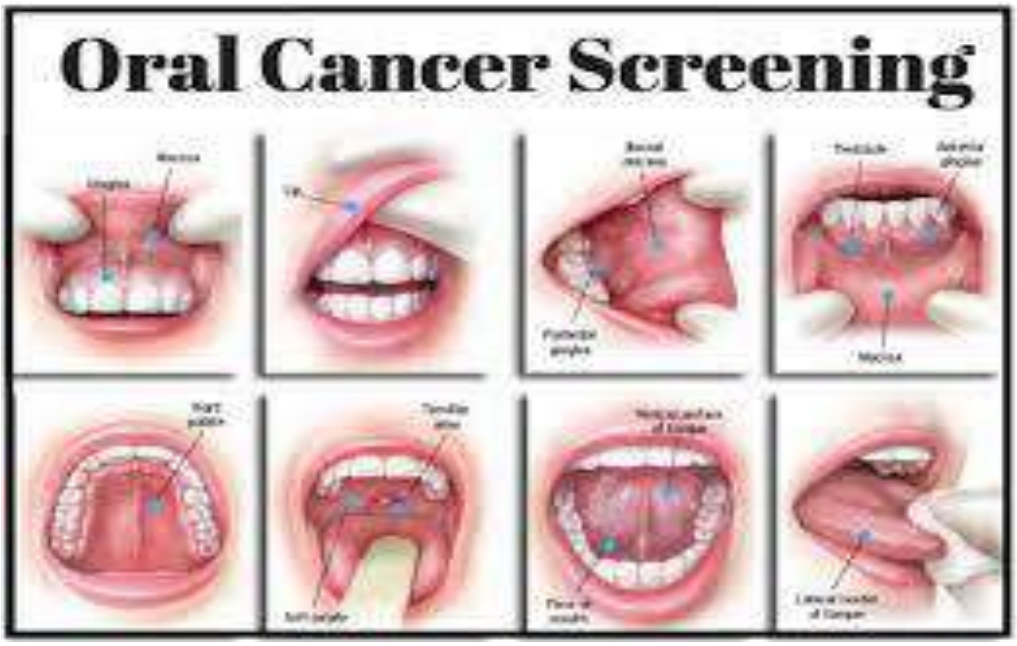
ORAL CANCER

Oral cancer is a term used for general description of malignant tumors of the oral cavity .Over 94% of oral cancers are squamous cell carcinoma (SCC). Oral squamous cell carcinoma (OSCC) is defined as a malignant neoplasm in the oral cavity which is derived from squamous epithelial cells. The term oral include all intra- oral structures such as tongue, gingiva, buccal mucosa, floor of mouth, palate, vestibules, oropharynx as well as lip. Oral cancer accounts for 4% of all cancers in the world but it is the sixth most common cancer in males and the twelfth most common in females. It is uncommon before the age of 40 years and the highest incidence of oral cancer is in the 5^{th} - 6^{th} decades of life with sex incidence being a 3:1 male predominance. Geographically, the oral cancer is found world wide, but there is significant variation in incidence in African and Asian countries.

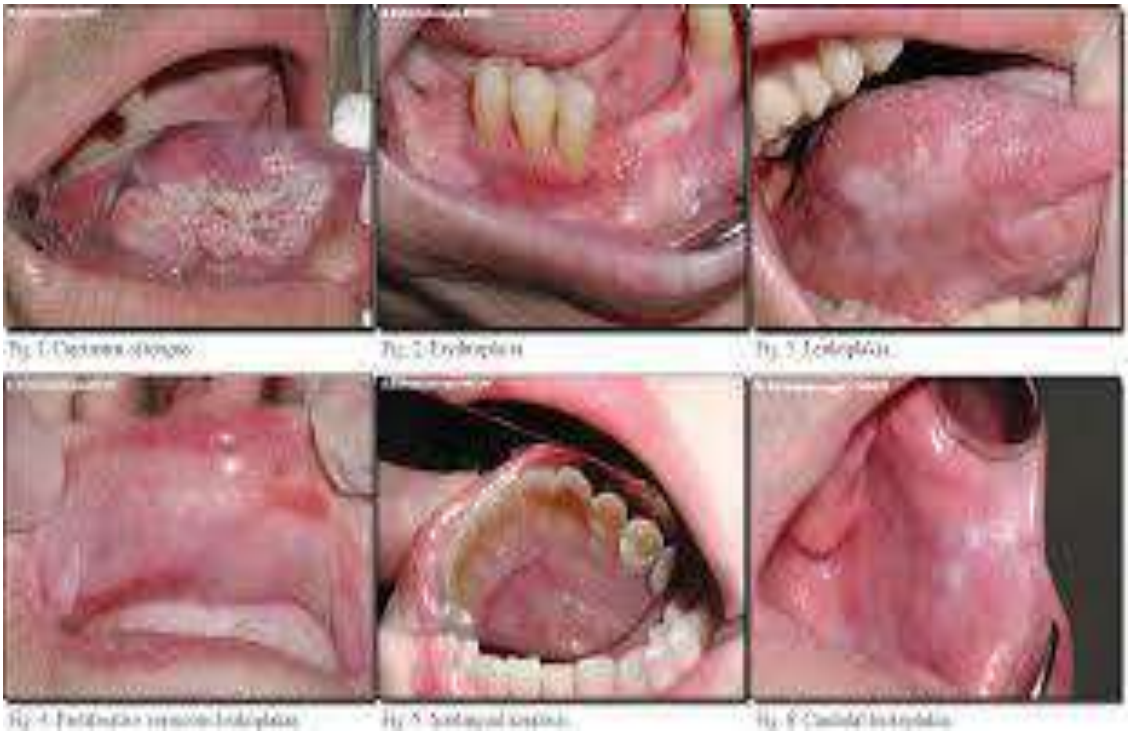




In some countries, such as India and Sri Lanka (however, exceptional) cancer of the mouth accounts for approximately 40% or more of all cancer. In Iraq cancer of the mouth accounts for approximately 4.5% and most of this percentage are SCC (91.5%) according to the Iraqi Cancer Registry. Oral cancer is an age-related disease, 98% of patients are over the age of 40 years. The risk of intra-oral cancer development increases with increasing age, especially for males. In the United States, white men have a higher risk of intra-oral cancer after 65 years, while, the highest annual incidence rate in middle age is seen in American males of African ancestry. Advanced age is probably the most important factor in the mortality associated with the development of any cancer. With all respect of molecular carcinogenesis of cancer development, the age of the patients have a great effect in the proper function of immune system which tends to become less efficient at recognizing and eliminating aberrant cellular growths which arise from time to time in people of all ages.



Females, whether white or non-white, have a much lower annual incidence rate than males at all age levels. The lower lip is the most frequent site of oral cancer, while the tongue is the most affected site within the mouth. Cancer of the tongue is accounted about 40% of all cases in the oral cavity proper, 70% of oral cancer are concentrated in the lateral borders of tongue, the adjacent floor of mouth and lingual aspect of the alveolar margin, forming U-shape area extending back to oro-pharynx. About 40% of OSCC begin on the floor of mouth or on the lateral or ventral surfaces of the tongue. About 38% of all OSCC occur within lower lip due to chronic sun exposure and presence of solar-related cancers on the external surface.Lip cancer is in some countries mainly Neozeland the most frequent malignant neoplasm of the oral cavity, reaching 25% of all cases. Lip cancer has a variable incidence around the world, with the highest rates being reported in the south of Australia and in some regions of Canada and Spain.



Squamous cell carcinoma :Represent 90 % or more of all oral malignant neoplasm.Male : female ratio of intra oral carcinoma is 2:1 or 3:1 in most country while lip cancer is 6-8 time more common in men .SCC occur mostly in old who have been aware of alteration in oral cancer site for 4-8 month before seeking professional help .

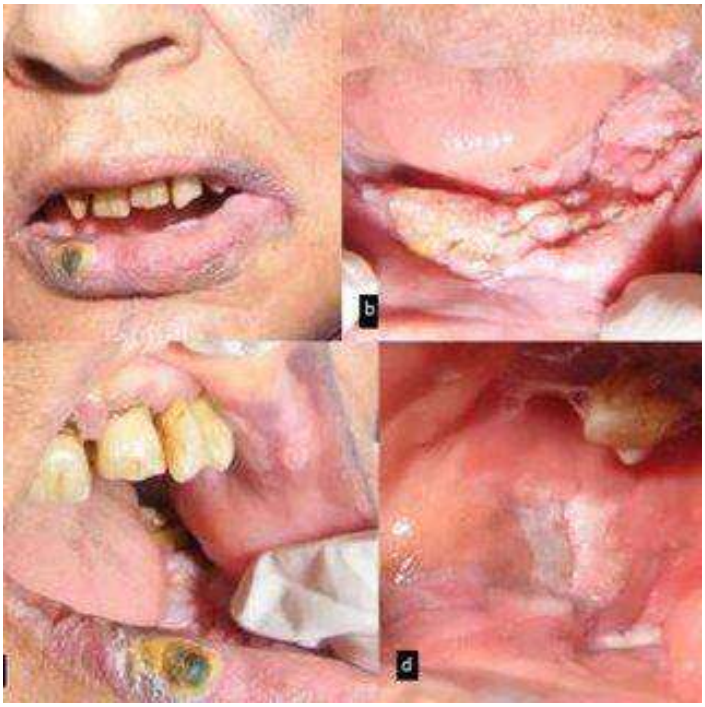
Etiology and risk factors:

The causes of OSCC are multifactorial, no single causative agent or factor (carcinogenic) has been clearly defined or accepted, it is likely that more than a single factor is needed to produce such a malignancy (carcinogenesis). In general term it can be defined into Extrinsic and Intrinsic factors.

1. Tobacco smoking:

Tobacco smoking (pipes, cigars, cigarettes, reverse smoking) is thought to be implicated in well over 80% of cases of OSCC, particularly of cigarettes. Chronic exposure of the epithelial surfaces of head and neck to this irritation is thought to result in a "field cancerization" sequence of hyperplasia, dysplasia and carcinoma. Tobacco is a known carcinogen, which means that it is known to damage cellular DNA and causes the cellular reproductive machinery to malfunction, which is the first step in the growth of malignant cellular masses (cancer). Thus tobacco causes malignant mutations in the cellular DNA.There is a dose-relationship between exposure to tobacco smoke and the development of oral cancer. Smokers are up to 25 times more likely to develop head and neck cancer than their non-smoking counterparts. Tobacco chewing ,snuff-dipping, betel chewing and tobacco sachets (smokeless tobacco) where it's particularly noticeable in India and Sweden, cause extensive hyperkeratosis plaques which may lead to development of verrucous carcinoma as well as SCC. Pipe- smoking is also associated with oral cancer development but this habit has steadily

declined in most westernized countries and has never become popular with women.

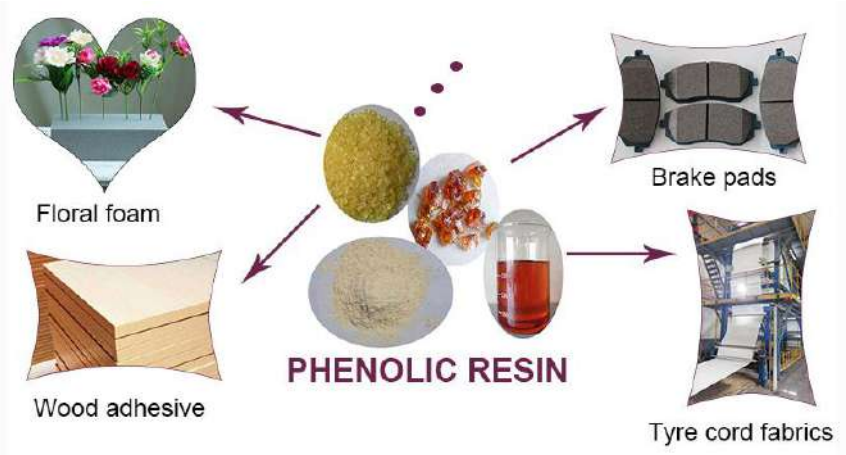


2. Alcohol:

Alcohol is known to inhibit a gene that functions in response to DNA damage. This gene is responsible for initiating cell death in cells with DNA damage (apoptosis gene), plus alcohol inhibits the body's natural defense against malignancy. Some studies show a dose-response relationship with heavy drinkers being at a greater risk. In addition, drinkers of spirits may be at a greater risk than those who drink wine.Moreover, there is a fairly convincing evidence that alcohol is carcinogen when acts synergistically with tobacco, hence it has been known for more than 30 years that tobacco and alcohol have the major role in the etiology of OSCC. This is perhaps because alcohols act as solvents and facilitate penetration of the oral mucosa by other carcinogens that may be present in the mouth e.g. tobacco.

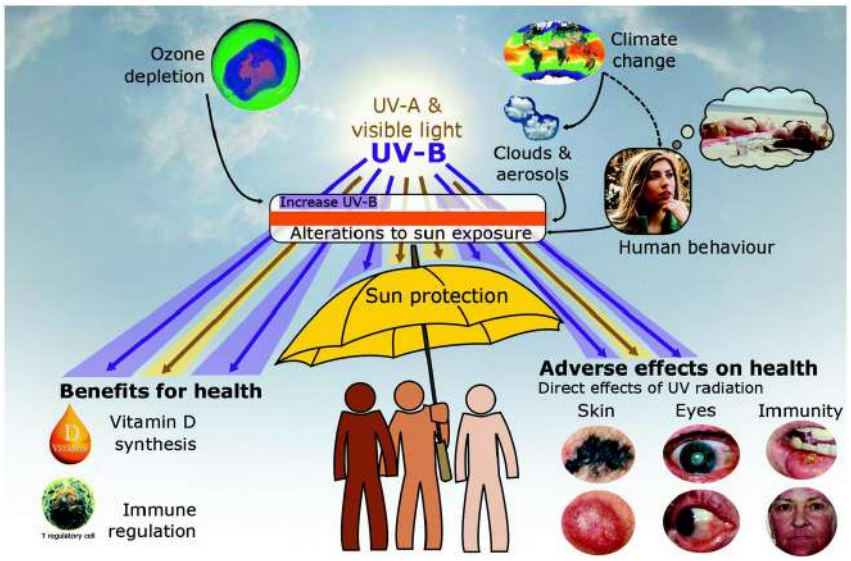
3. Phenols:

Recent evidence has pointed to an increased oral cancer risk for workers in the wood products industry chronically exposed to certain chemicals, such as phenoxyacetic acids. Moreover, it has long been known that these workers are at increased risk for nasal and nasopharyngeal carcinoma.



4. Radiation:

Whatever its source (ultra-violet rays (UVR) of sun light, x-rays, nuclear fission and radio nuclides) is a carcinogen. Ionizing radiation causes chromosome breakage, translocations and less frequently point mutations leading to genetic damage and carcinogenesis. UVR induces the formation of pyrimidine dimmers within DNA, leading to mutations. Therefore U.V.R. can give rise to S.C.C. and melanomas of the skin. The incidence is increased between farmers and fishermen and it is considered as a disease of outdoor workers.



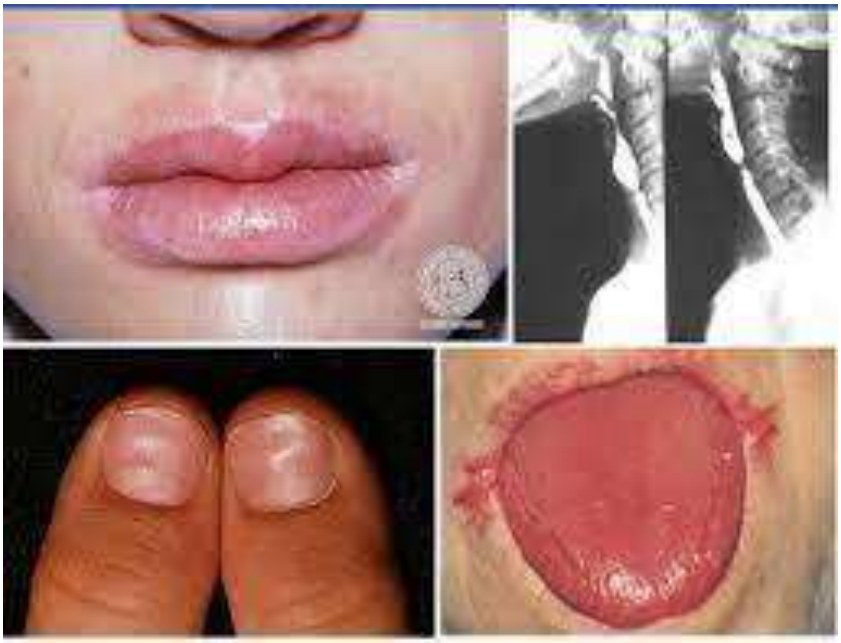
5. Malnutrition:

It can be considered as an extrinsic factor. In India, malnutrition is widespread and may contribute together with betel quid chewing, to the high incidence of SCC. This factor can be subdivided into many deficiencies:

- *Iron deficiency;*

The increased risk of oral cancer associated with primary sideropenic anemia (Plummer –Vinson or Patterson- Kellsy) has been recognized for many years. Irons is essential for maintenance of oral epithelium, thus in deficiency

states these epithelium cells turnovers more rapidly and produce an atrophic or immature mucosa, and it is possible that atrophic changes in iron deficiency anemia render the mucosa more susceptible to chemical carcinogens).



- *Vitamin deficiency:*

Vitamin A important in the maintenance of stratified squamous epithelium; vitamin A deficiency produces excessive keratinization of mucous membrane. Several epidemiological studies have shown that individual whose diet are high in the antioxidant vitamin A,C and E have a protective effect and decreased risk of oral cancer**.**

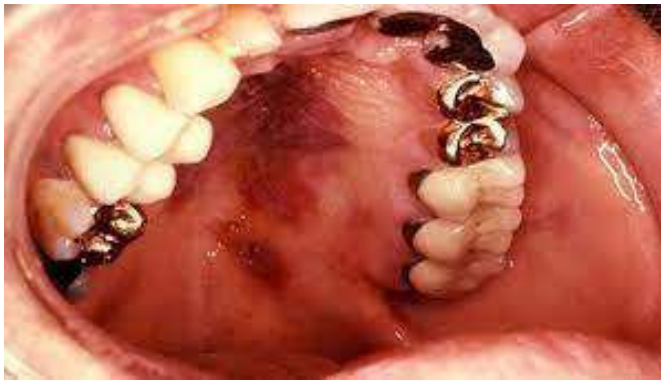
6. Oncogenes viruses

Viruses may play a major role in a wide variety of cancers. Viral agents capable of integration into the host's genetic material may be particularly dangerous and potentially could commander the host's ability to regulate normal growth and proliferation of the infected cells. The oncogenic viruses that facilitating malignancy transformation are:

- Human papilloma viruses (HPV).
- Herpes simplex viruses (HSV).
- Epstein-Bar viruses (EBV).
- Human immunodeficiency viruses (HIV).

7. Immunosupression

Reports have been published of an increased risk of carcinoma of the lip in patients with AIDS, renal and organ transplantation especially when tobacco smoking and alcohol abuse are present .

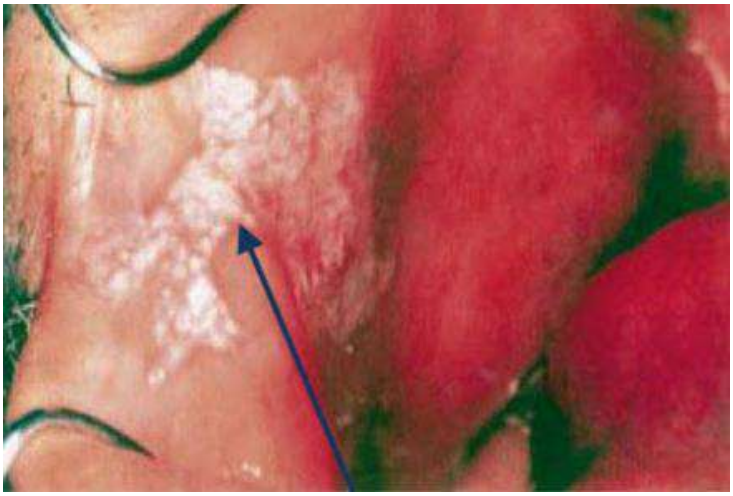


8. Chronic infection

- *Chronic candidal infection*

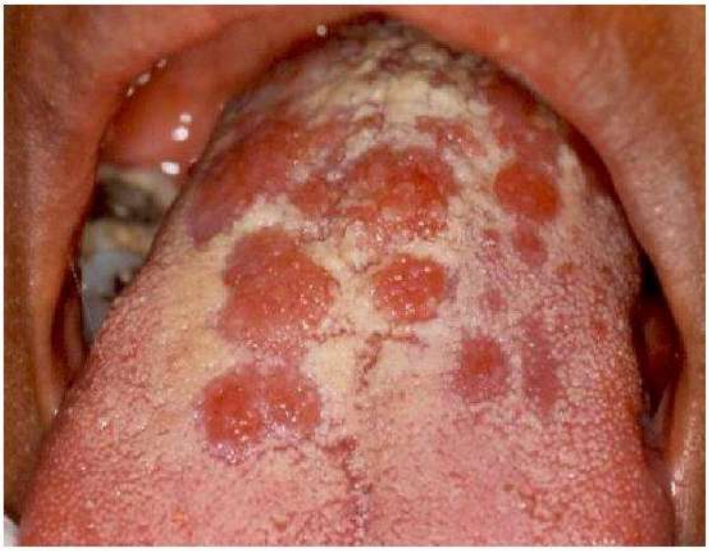
White plaque candidal leukoplakia is cited as an oral precancerous condition, because this lesion appear as a white plaque that can't be rubbed off and superimposed candidiasis. Some studies improved that; some strains of Candida albicans produce nitrosamines which are chemicals that have been

implicated in carcinogenesis.The role of candidal infection in malignant lesions must be regarded as uncertain.



- *Syphilis*

Tertiary stage of syphilis has a strong association with the development of dorsal tongue carcinoma. However, syphilis-associated oral malignancies are rare today because the infection is typically diagnosed and treated before the onset of the tertiary stage.



9. Occupation

High incidence of oral cancer has been reported in:

- Textile workers, particularly in those exposed to dust from raw cotton and wool .
- Air pollution and environmental exposure to the burning of fossil fuels, which is considered as one of contributing factors of oral cancer etiology.

10. Pre-existing oral lesion

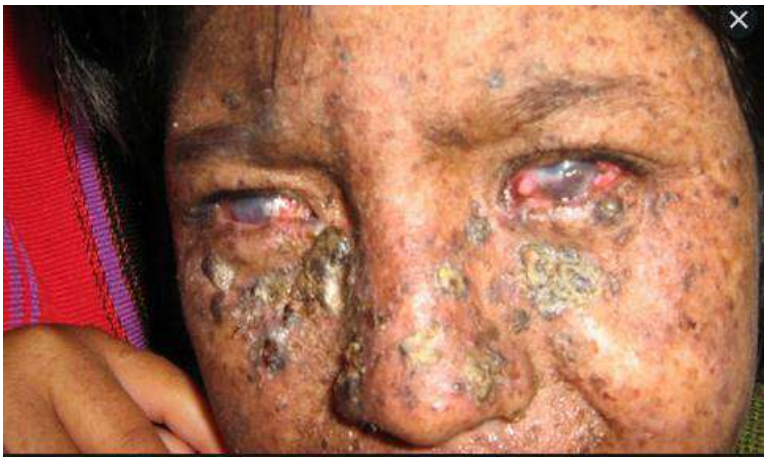
There is general agreement that pre-malignant oral lesion is defined as "a morphological altered tissue in which cancer is more likely to occur than in its apparently normal counterpart".The most important pre-malignant oral lesions are:

- Proliferative verrucous leukoplakia.
- Nicotinic palatines in reverse smokers.
- Erythroplakia.
- Oral sub mucous fibrosis.
- Erythro leukoplakia.
- Granular leukoplakia.
- Laryngeal keratosis.
- Actinic cheilosis.

- Smooth, thick leukoplakia.
- Smooth red tongue of Plummer-Vinson syndrome.
- Smokeless tobacco keratosis.
- Lichen planus.
- Smooth, thin leukoplakia.

11. Familial factors

There are a few genetic disorders that contribute with cancer development but such disorders are rare so they received less attention. Multiple primary tumors are often seen in hereditary cancer syndromes includes Xeroderma pigmentosum and Dyskeratosis congenital.



12. Socio-economical factors

The biological behavior of oral cancer

After transformation of cells and cancer development, cancer grows by progressive infiltration, invasion, destruction and penetration of the surrounding tissue. Most OSCCs are extremely locally destructive. Malignant neoplasm disseminate by one of 3 pathways:

- **Seeding:**

Spread by seeding within body cavities.

- **Lymphatic spread:**

It is the favored route for dissemination of OSCC into the surrounding lymph node. "A sentinel lymph node SLN" is defined as the first lymph node in a regional lymphatic basin that receives lymph flow from a primary tumor. A SLN is very important in determination the spread of tumor, and can be used to plan treatment by surgeons, determine most important lymph nodes in head and neck region are: parotid, buccal, sub mental, submandibular, deep cervical and superficial cervical. However; because of delayed diagnosis approximately 21% of patients have cervical metastasis at diagnosis

- **Hematological spread:**

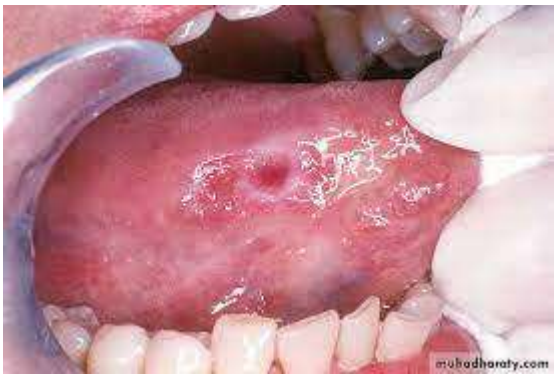
It is favored pathway for dissemination of sarcomas. In OSCC the hematological dissemination occur late in the clinical course of the disease.. The most common sites are: lung, liver and bones, but any part of the body may be affected.

Clinical presentation of OSCC

OC has a varied clinical presentation including:

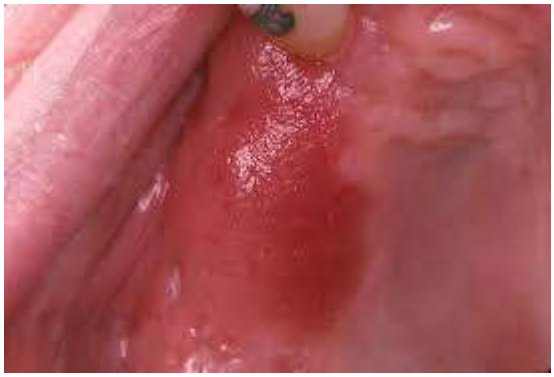
- *Exophytic (mass forming, fungating, papillary and verruciform).*

- *Endophytic (invasive, burrowing and ulcerated).*

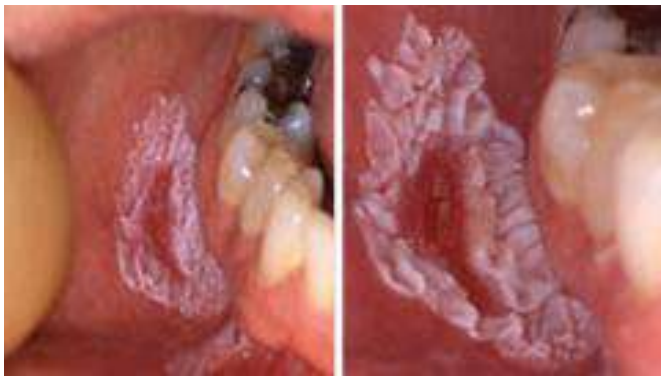


- *Leukoplakia (white patch).*

- *Erythroplakia (red patch).*



- *Erythro-leukoplakia (combined red and white patch)*



Pain is not a reliable indicator as to whether a particular lesion may be malignant. Larger; advanced carcinoma will often be painful, but many early oral cancers will be totally asymptomatic or may be associated with only minor discomfort.The symptoms which the patient may complain can be summarized as follows:

Early symptoms:

- Any white or red speckled patch.
- A non-healing ulcer or sore.
- Any lump or thickening.
- Persistent soreness or discomfort.

Later symptoms:

- Ulceration of the oral mucosa.
- Difficulty in the tongue or jaw movement.
- Difficulty in the chewing or swallowing.
- Numbness of the tongue or other parts of the mouth.
- A lump under the lower ridge or in the neck.

Clinical Staging of Cancer

The clinical characteristics of the tumor can be staged using TNM system (Tumor – Node –Metastasis), which serves as a therapeutic guide. It allows the definition of prognosis and comparison of results, given that they have a common language. The TNM system depended on three basic clinical features; where;

T= Size of primary tumor in centimeters.

N =Involvement of local lymph nodes.

M=the presence or absence of distant metastasis.

TNM system for Oral Cancer

Primary Tumor (T) size	
Tx	No available information on Primary tumor
T0	No evidence of primary tumor
Tis	Carcinoma in situ at primary site
T1	Tumor 2 cm or less in greatest diameter
T2	Tumor more than 2 cm but not more than 4cm in greatest diameter
T3	Tumor more than 4cm in greatest diameter
T4a	(Lip) Tumor invades through cortical bone, inferior alveolar nerve floor of mouth or skin of face (i.e. chin, nose). Tumor is respectable.
T4a	(Oral cavity) Tumor invades through cortical bone into deep extrinsic tongue muscles (genioglossus, hyoglossus, palatoglossus & styloglossus) ,maxillary sinus or skin of face.Tumor is resectable
T4b	Tumor involve masticator space, pteyrgoid plates or skull base and/or encases internal carotid artery. Tumor is unresectable.

Regional lymph nodes (N) involvement	
Nx	Regional lymph node can not be assessed
N0	No regional lymph node metastasis
N1	Metastasis in single ipsilateral lymph node, <3 cm
N2a	Metastasis in single ipsilateral lymph node, >3 cm but <6 cm
N2b	Metastasis in multiple ipsilateral lymph node, <6 cm
N2c	Metastasis in bilateral or contra-lateral lymph nodes, non >6 cm
N3	Metastasis in a lymph nodes >6 cm in greatest diameter.
Distant metastasis (M)	
Mx	Metastasis can not be assessed
M0	No distance metastasis
M1	Distance metastasis

TNM Clinical staging

Stage		**TNM classification**	**Oral cavity (5year survival rate)**	**Lip (5 year survival rate)**
Stage I		T1 N0 M0	68%	83%
Stage II		T2 N0 M0	53%	73%
Stage III		T3 N0 M0 or T1,T2 or T3 N1 M0	41%	62%
Stage IV	IVA	T4a N0 or N1 M0 or T1,T2,T3 or T4a N2 M0	27%	47%
	IVB	Any T N3 M0 or T4b any N M0		
	IVC	Any M1 lesion		

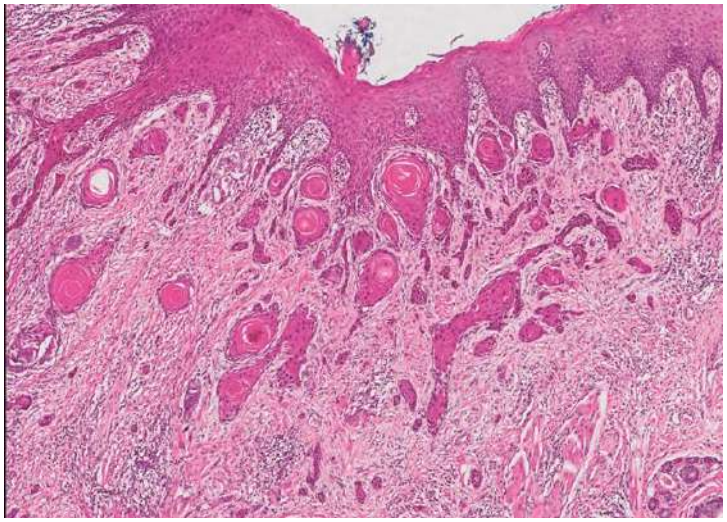
Histopathological grading

According to the method originally described and adopted by the WHO, 2003 which takes into account a subjective assessment of the degree of keratinization, cellular and nuclear pleomorphism, and mitotic activity, the histological variations showed in OSCC is related to the degree of differentiation (grade) exhibiting by the tumor cells and how closely the tissue architecture resembles normal stratified squamous epithelium.

The WHO, 2003 grading system recommends three categories:

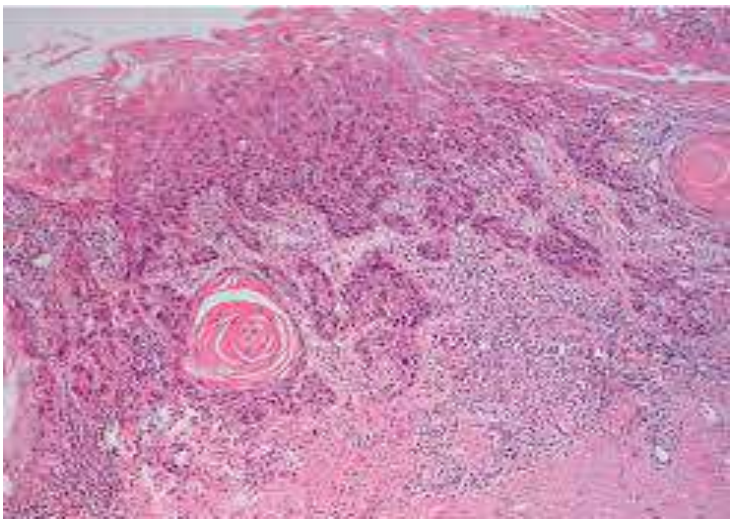
Grad 1: Well differentiated

These are tumors that produce significant amounts of keratin and exhibits some features of maturation from the basal cells to the keratinized surface layer and it seems to grow at a slightly slower pause and metastasis later in its course so called low-grade tumor.



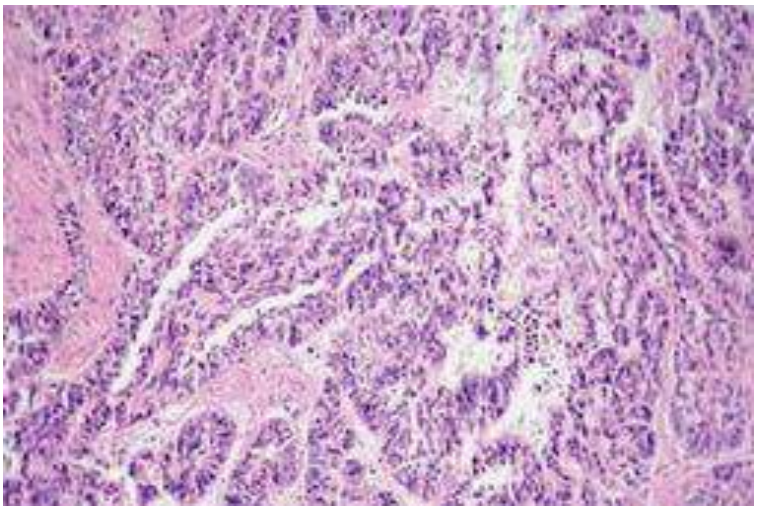
Grad 2: Moderately differentiated

The biological behavior of tumor cell somewhere between these two extremes. It produces no or little keratin but tumor cells are still recognizable as stratified squamous epithelium despite its significant deviation from normal.



Grad 3: Poorly differentiated

High –grade tumors or anaplastic carcinomas which have a biological behavior with extremely cellular and nuclear pleomorphism with little or no keratin production and rapidly metastasis in its course.



Biological Behavior, Oncogenes And Tumors-Suppressor Genes

Oral cancer has a multifactorial aetiology and is the result of genetic damage allowing uncontrolled proliferation of cells. It is a multistep process involving multiple sequential mutations which accumulate within the cell. Mutations in the genes which regulate cell growth and proliferation are particularly important. These genes are the growth-promoting proto-oncogenes found in normal cells, and the tumour-suppressor genes that encode for growth inhibitory proteins.Under normal circumstances cellular proliferation is controlled by the balance between these growth-promoting and growth-inhibiting genes. During carcinogenesis a proto-oncogene may undergo mutation and become an activated oncogene, resulting in enhanced activity, and/or tumour-suppressor genes may be mutated or their products inactivated.There sultan both cases leads to deregulation of cell proliferation and tumour formation. Oncogenes (for example, the c-myc and ras families) encode for a range of growth-promoting proteins such as growth factor receptors, signal-transmitting proteins, and stimulatory cell-cycle regulating proteins. In contrast, tumour-suppressor genes encode for growth-inhibitory proteins, such as p53 which plays a vital role in inhibiting the cell cycle and, if necessary, arresting the cycle and switching cells into apoptosis. The most important oncogenes and tumorsuppressor genes so far identified appear to influence pathways controlling the first stages of the cell cycle, i.e. the progression through the G1 phase (the phase before DNA synthesis) into S phase (the phase of DNA synthesis). Most oncogenic agents probably exert significant effects during the G1 phase of the cell cycle and the G1 to S transition is carefully regulated by inhibitory proteins, particulary p53. Thus, cells with damaged DNA are normally blocked at this G1 checkpoint. This allows time for repair of the

damaged DNA, or, if that fails, to switch the cell into apoptosis, so preserving the integrity of the genome. Mutations of the p53 gene can therefore result in loss of regulation of the checkpoint, allowing cells with damaged DNA to undergo replication. Mutation of the p53 gene is a common and significant event in many cancers throughout the body.

The cervical lymph nodes are usually divided into five groups. Tumours tend to metastasize initially to the nodes in the superior drainage groups (levels 1 and II), with progressive involvement of the more inferior groups in the chain as metastatic disease spreads. As the metastatic carcinoma destroys and replaces the nodal lymphoid tissue it may also invade through the capsule of the node into the surrounding tissues, resulting in fixation of the node on clinical examination. Extracapsular spread is an important feature which has an adverse affect on prognosis. Blood-borne metastases occur later in the clinical course of the disease. Previously, many patients died before distant metastases became apparent, but their incidence is now increasing as a result of better local and regional control of the primary tumour. The risk of distant metastases increases with increasing involvement of nodal metastases in the neck.

Main lymph node groups in the neck

Level I: nodes of the submandibular and submental triangles.

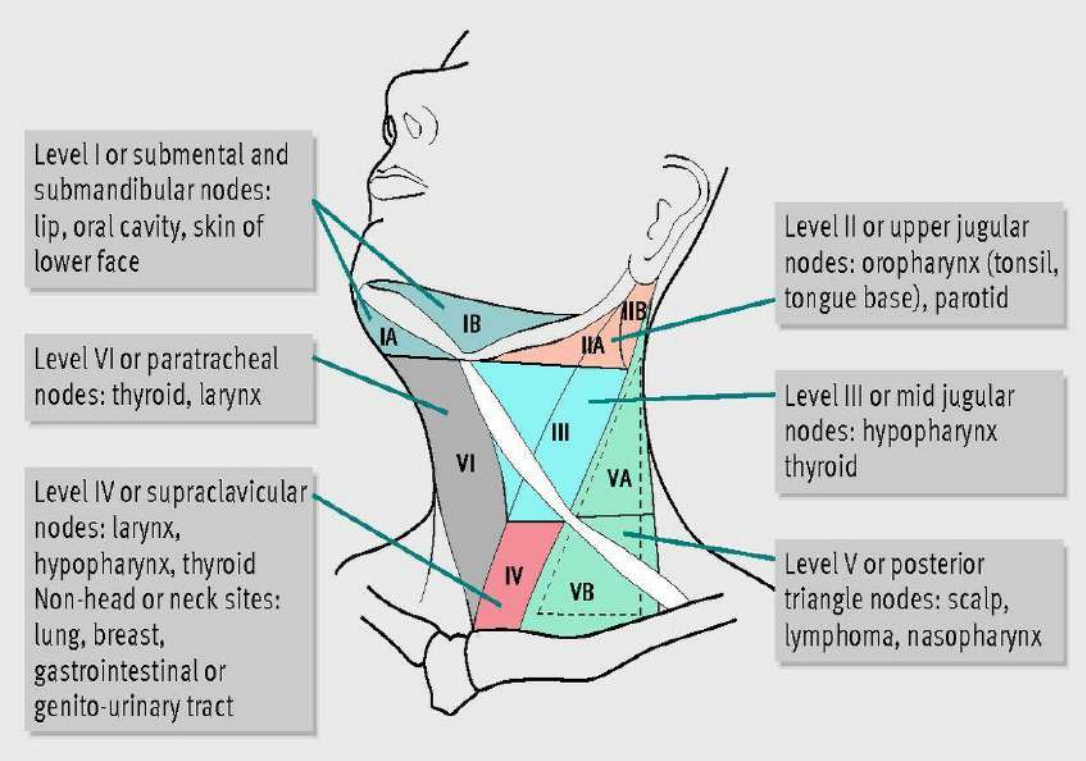
Level II: nodes of the upper cervical (jugular) chain.

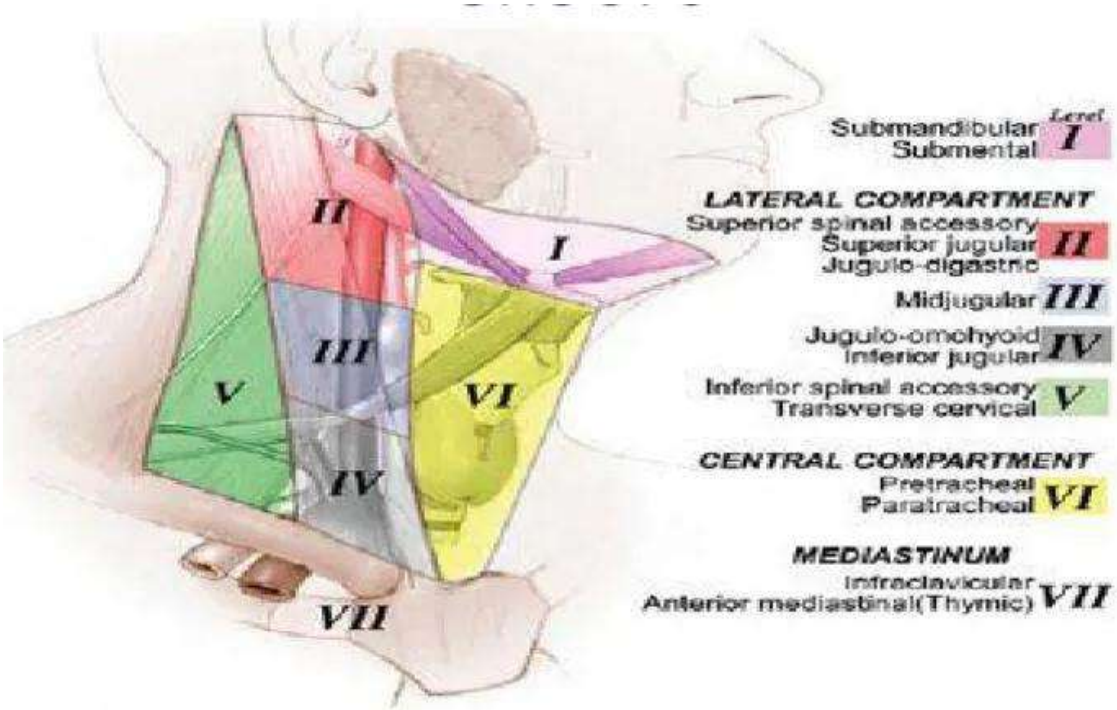
Level III: nodes of the mid-cervical (jugular) chain.

Level IV: nodes of the lower cervical (jugular) chain.

Level V: nodes of the posterior triangle of the neck.

Level I is bounded by the digastric muscle. Levels II, III, and IV nodes lie deep to the upper, mid, and lower thirds of the sternocleidomastoid muscle and are related to the internal jugular vein. The omohyoid muscle separates levels III and IV.


Level I or submental and submandibular nodes: lip, oral cavity, skin of lower face
Level VI or paratracheal nodes: thyroid, larynx
Level IV or supraclavicular nodes: larynx, hypopharynx, thyroid Non-head or neck sites: lung, breast, gastrointestinal or genito-urinary tract
Level II or upper jugular nodes: oropharynx (tonsil, tongue base), parotid
Level III or mid jugular nodes: hypopharynx thyroid
Level V or posterior triangle nodes: scalp, lymphoma, nasopharynx
IA
IB
IIA
IIB
III
VI
VA
IV
VB


Submandibular
Submental
Level I
LATERAL COMPARTMENT
Superior spinal accessory
Superior jugular
Jugulo-digastric
II
Midjugular
III
Jugulo-omohyoid
Inferior jugular
IV
Inferior spinal accessory
Transverse cervical
V
CENTRAL COMPARTMENT
Pretracheal
Paratracheal
VI
MEDIASTINUM
Infraclavicular
Anterior mediastinal(Thymic)
VII

Diagnostic Aids

- Lab. Investigations (Hb., ESR….etc)
- Biopsy (incisional, excisional, FNAC, SLN, Drill Biopsy…..etc)
- Conventional radiograph (PA, occipitomental, true & oblique lateral, OPG)
- UlS examination
- CT and MRI
- Immunohistochemistry
- In situ hybridizations

Treatments

- **Surgery:** complete excisions of the tumour with safe margin, guided by frozen sections(surgical pathologist)

- **Chemotherapy:** cytotoxic drugs in general interfere with division and proliferation of cells, the ultimate target is the DNA of the cell nuclei. Chemotherapy (chemo) is the use of anti-cancer drugs that are given into a vein or taken by mouth. These drugs enter the bloodstream and can reach cancer that has spread to organs beyond the head and neck. It may be used in several different situations: Chemotherapy combined with radiation therapy may be used instead of surgery to control larger cancers that are confined to the head and neck region. Chemotherapy (sometimes given with radiation) is sometimes given to shrink the cancer before surgery. This is called neoadjuvantorinduction chemotherapy. Chemotherapy (often together with radiation) has also been used to reduce the severity of symptoms of cancers of the head and neck that are too large to be completely removed and which radiation treatment alone has not been able to control

The uses of chemotherapy in head and neck cancer as follows

1. as adjuvant to other modalities in three patterns

i. Before surgery or radiotherapy to reduce bulk of tumor and control micrometastesis.

ii. During(concurrent)with radiotherapy (act as radiosensitizing agents)

iii. Following surgery or radiotherapy, to eradicate micrometastesis.

2. Palliative: patients with significant symptoms caused by advanced or recurrent tumor stage.

3. Alone as radical (therapeutic) chemotherapy, it is unreliable agent, some patient may respond dramatically but for short period of live, recurrence is high after 3-6 months.

4. combination chemotherapy allow a high antitumor response rate but it is more toxic

Types of chemotherapy

- *alkylating agents e.g cyclophosphmide, chlorambucil, nitrosurea.*
- *antimetabolite e.g methotrexate, 5-fluorouracil.*
- *plant derivative e.g vincrestine.*
- *antitumors antibiotics e.g Bleomycin, doxorubicin.*
- *miscellaneous e.g cis-platinum*

- **Radiotherapy**: it is the use of ionization radiation to treat disease.

1. radical radiotherapy which aims to cure the patient.

2. palliative radiotherapy which aims to relief symptoms of diseas with out attempting to cure the patient.

3. adjuvant radiotherapy

Types of radiotherapy

1. Teletherapy (external beam radiation) which use a machione to deliver a beams of radiation directed to the tumor from out side of patient.
2. Plesiotherapy(Brachytherapy, interstitial radiotherapy), the source of radiation is placed within or in close proximity to the tumour.

So that tissues either radio sensitive or radio resistant depending on type of tissues, size of tissues, oxygenation of tumour cells.

- **Immunotherapy**: method of therapy directed to augmenting the immune response against cancer, it is divided into

1. passive immunotherapy which is administration of externally stimulated immunological component that are initially obtained from patients being treated. E.g. thymic factors such as thymosin
2. active immunotherapy which is administration of agents to tumor – bearing host that are able to elicit an immune reaction designed to control or eradicate malignant diease. E.g. BCG, cytokines (IL1,IL2, interferon…etc)

- **Thermal Applications**

1. cryosurgery and Cryotherapy (liquid nitrogen and liquid nitrouoxide)
2. heat application. E.g cautry

- **Laser Application**
- **Gene Therapy**
- **Chromosomal Therapy**
- **Monoclonal Therapy**
- **Gamma Knife**
- **Apoptosis**

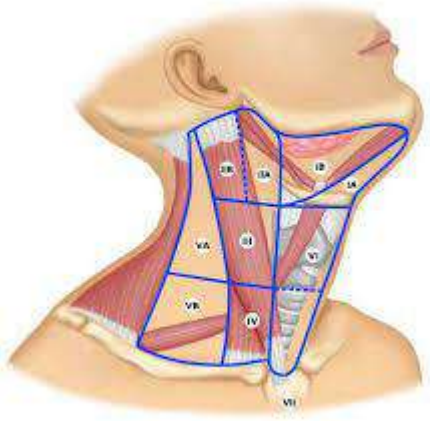
Prognosis

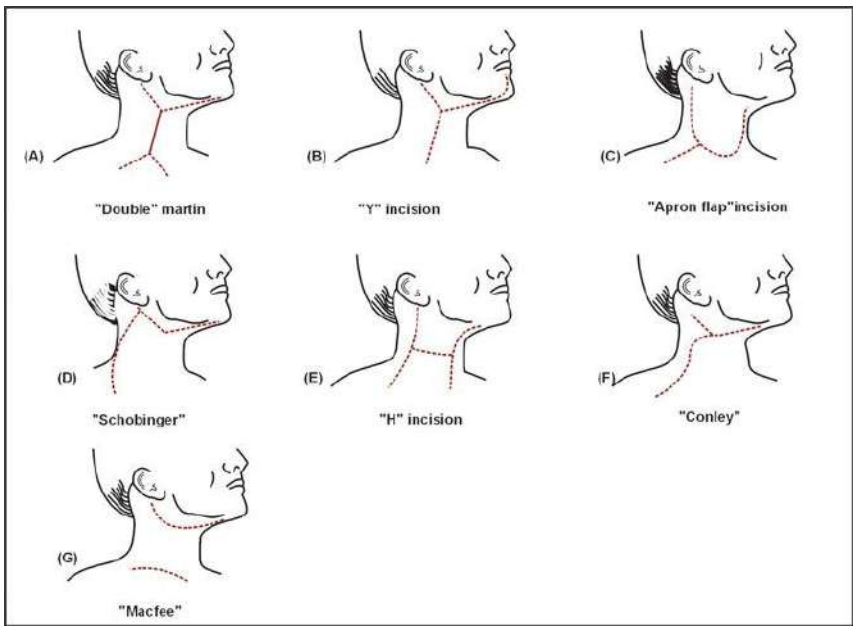
The survival rate of patients with oral carcinoma depends on a number of factors, but early diagnosis is by far the most important. It is influenced by the site of the lesion, and generally the further back in the mouth the tumour, then the worse the prognosis. This is probably because tumours at the back of the mouth tend not to be diagnosed at an early stage, but the rich lymphatic drainage around the base of the tongue may also favour early metastatic spread. Carcinomas in females have a better prognosis than carcinomas in males, possibly because they tend to be diagnosed and treated at an earlier stage. This probably reflects the fact that more females are regular dental attenders than males. Age affects prognosis, partly because with increasing age the patient becomes less well able to withstand extensive surgery or radiotherapy. Reduction in the effectiveness of cell-mediated immune responses may also be involved. Although the past few decades have seen major advances in the methods used to treat oral cancer, the overall 5-year mortality rates have not changed significantly. Local recurrence at the primary site, or within the neck in patients with metastatic disease, is the major cause of death. A few patients also develop new primary tumours. The major factors thought to influence prognosis have been incorporated into clinical staging systems which assess the extent of disease in the patient. The reduction in survival rates related to metastatic spread is well established. Several studies have shown 5-year survival rates of about 80 per cent for patients without lymph node metastases compared to between 45 per cent and 65 per cent for those with metastases, depending on their extent. In particular, the presence of extracapsular spread is an important indicator of poor prognosis.

Carcinoma in situ

This term is used to describe severe epithelial dysplasia in which the whole, or almost the whole, thickness of the epithelium is involved but the basement membrane is intact and there is no invasion of the lamina propria. Oral carcinoma in situ usually presents clinically as leukoplakia or erythroplakia. It is a precancerous (premalignant) lesion (see below) but its natural history is not well understood. In some patients the lesion may progress to invasive carcinoma but in others it remains static for long periods and, in some, the degree of dysplasia may regress or fluctuate with time. It is common to find histological changes of dysplasia, including carcinoma in situ, in the epithelium surrounding an invasive carcinoma, even though this may appear clinically healthy. This suggests that in some patients there may be a field of potentially precancerous change involving a wide area of mucosa. It is probable that some carcinomas thought to be recurrent tumours represent new primary lesions arising in such a field change.

Neck dissection





Radical (classical) neck dissection (RND): means removing all lymph nodes containing levels I-V with all three non-lymphatic structures (IJV, SCCM & AN)

Indications

1. significant operable palpable LN with primary (in continuity resection)
2. significant operable palpable LN with occult primary
3. significant operable palpable LN with primary well control

Contraindications

1. Distant metastesis
2. unfit for surgery
3. inoperable primary
4. inoperable neck
5. significant bilateral neck LN enlargement

Modified neck dissection (MND): preserving one or more of non lymphatic structures.

- Type I (preserve accessory nerve)
- Type II (preserve accessory nerve + IJV)
- Type III (preserve three non lymphatic structures)

Selective neck dissection (SND): preservation of one or more lymph node group + all three non lymphatic structures.

- Supra-omohyoid SND: remove I, II, III
- Extended (Anteriolateral) SND: remove (I, II, III, IV)
- Lateral SND: remove (II, III, IV)
- Posteriolateral SND: remove (II, III, IV, V)

Management of Neck

1. **N_0** : manage by

- Surgery by performing selective neck dissection (SND)
- Radiotherapy (DXT=deep x-ray therapy)

2. **N_1**: Modified neck dissection (MND).

   Note: some use DXT only when primary in nasopharynx.

3. **N_2** : manage as follow

- N_{2a} : Modified or Radical neck dissection.
- N_{2b}: Modified or Radical neck dissection.
- N_{2c}: Radical neck dissection.

4. **N_3** : often they are incurable, mostly treated by DXT and / or Chemotherapy (palliative therapy)

Suggested Reading

- Peter Ward Booth, Maxillofacial Surgery: Churchill Livingstone, 2006

AUTOIMMUNE DISEASES

The term *autoimmune disease* refers to a disorder in which there is evidence of an immune response against self. Autoimmune diseases may be primarily due to either antibodies (autoantibodies) or immune cells, but a common characteristic is the presence of a lymphocytic infiltration in the target organ. Examples include type 1 diabetes mellitus, autoimmune thyroiditis, Sjögren's syndrome, SLE, and multiple sclerosis.

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Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is the prototypical autoimmune disease characterized by the production of numerous autoantibodies. Organ injury is secondary to either the direct binding of autoantibodies to self-antigens or the deposition of immunocomplexes in vessels or tissues. In addition to systemic and isolated cutaneous lupus (chronic discoid lupus), a distinct syndrome of drug-induced lupus is recognized. Unlike SLE, drug-induced lupus rarely affects the kidney and is reversible on discontinuation of the offending agent.

Clinical Manifestations

Skin is affected in up to 85% of SLE patients. In addition, cutaneous lupus can occur without multisystem involvement. Skin lesions of lupus can be classified into l**upus-specific** (having diagnostic clinical or histopathological features) and **nonspecific lesions**.

Three subtypes of **lupus-specific skin lesions** have been described: **acute, subacute, and chronic.**

Acute cutaneous lupus : represented by the butterfly rash—mask-shaped erythematous eruptions involving the malar areas and bridge of the nose. Bullous lupus and localized erythematous papules also belong to the acute lupus category.

Chronic cutaneous lupus affects the skin of the face or scalp in about 80% of cases . The least common subtype, **subacute cutaneous lupus**, includes papulosquamous (psoriasiform) and annular-polycyclic eruptions usually on the trunk and arms.

Nonspecific but suggestive skin manifestations of lupus are common and include alopecia , photosensitivity, Raynaud's phenomenon, livedo reticularis, urticaria, erythema, telangiectases, and cutaneous vasculitis.

ETIOLOGY

The specific etiology of SLE is not known with certainty, but immunocomplexes, autoantibodies, and genetic, infectious, environmental, and endocrine factors play significant roles.

Renal Manifestations. Kidney involvement occurs in up to 50%–60% of patients with SLE and is a primary cause of morbidity and mortality in this population Clinically, renal disease in SLE can range anywhere from asymptomatic proteinuria to rapidly progressive glomerulonephritis with renal failure.

Musculoskeletal : Musculoskeletal manifestations occur in about 95% of patients with SLE, and arthralgia is the first presenting symptom in about 50% of cases. Nonersive symmetric arthritis most commonly affecting hands, wrists, and knees is typical of SLE.

Central Nervous System :occurs in about 20% of patients with SLE and is usually due to cerebral vasculitis or direct neuronal damage. CNS manifestations include psychosis, stroke, seizures and transverse myelitis and are associated with poor overall prognosis.

Cardiovascular Cardiovascular involvement in SLE is classically manifested by vasculitis and pericarditis.

Other Manifestations

Fatigue, depression, and fibromyalgia-like symptoms are commonly present and can be debilitating.

Oral Manifestations

The oral mucosa is affected in a significant percentage of lupus patients, with the predominant types of oral lesions being **ulcerations, erythematous lesions, and discoid lesions**. These ulcerations cannot be easily distinguished from other common oral conditions, such as aphthous ulcers, although they occur with increased frequency on the palate and in the oropharynx and are characteristically painless.

Discoid oral lesions are similar to those occurring on the skin and appear as whitish striae frequently radiating from the central erythematous area, giving a so-called *brush border*. Atrophy and telangiectases are also frequently present. Buccal mucosa, gingiva, and labial mucosa are the most commonly affected intraoral sites. **Isolated erythematous** areas are also common, especially on the palate.

Laboratory Findings

Anemia, leukopenia, and thrombocytopenia are among the most common manifestations of SLE. Elevation of erythrocyte sedimentation rate with normal C-reactive protein levels is characteristic of SLE.

Diagnosis

Diagnosis of SLE is based on the compatible symptoms and signs in the presence of suggestive laboratory abnormalities. Diagnostic criteria include:

1. Acute cutaneous lupus (e.g., malar rash or photosensitivity and other)
2. Chronic cutaneous lupus (e.g., classic discoid lupus and other)
3. Oral ulcers or nasal ulcers
4. Nonscarring alopecia
5. Synovitis involving 2 or more joints, characterized by swelling or effusion
OR tenderness in 2 or more joints and at least 30 minutes of morning stiffness
6. Serositis
7. Renal :Urine protein greater than or equal to 500 mg protein/24 hours
OR red blood cell casts
8. Neurologic disease (Seizures, psychosis, mononeuritis multiplex, myelitis, peripheral or cranial neuropathy, acute confused state).

9. Hemolytic anemia
10. Leukopenia (<4000/mm3 at least once)
OR Lymphopenia (<1000/mm3 at least once)
11. Thrombocytopenia (<100,000/mm3) at least once
Immunologic criteria
1. ANA level above laboratory reference range
2. Anti-dsDNA antibody level above laboratory reference range
3. Anti-Sm: presence of antibody to Sm nuclear antigen
4. Antiphospholipid antibody positivity
5. Low complement
Low C3
Low C4
Low CH50
6. Direct Coombs' test in the absence of hemolytic anemia

The proposed classification rule is as follows: classify a patient as having SLE if 4 of the clinical and immunologic criteria are satisfied, including at least one clinical and one immunologic criterions, OR if he or she has biopsy-proven nephritis compatible with SLE in the presence of antinuclear antibodies (ANAs) or anti-double-stranded DNA (dsDNA) antibodies.

TREATMENT

The oral ulcerations of SLE are transient, occurring with acute lupus flares. Symptomatic lesions can be treated with high-potency topical corticosteroids or intralesional steroid injections.

Dental Management

The dental management of the lupus patient should take into account the complex pathologic manifestations of the disease, including oral aspects and complications of immunosuppressive treatment.

Risk of Infection

Daily treatment with higher doses of prednisone (over 7.5– 10 mg/day) or other glucocorticoids, treatment with high doses of cyclophosphamide, and high disease activity are risk factors for infection in SLE patients. Impaired immune function that is part of this disease is also felt to contribute to their increased susceptibility to infection.

A baseline complete blood count should be obtained before dental treatment of SLE patients, as leukopenia, neutropenia, and/ or thrombocytopenia can occur. If possible, elective oral surgical procedures with the potential for bacteremia should be delayed until the absolute neutrophil count is over 1000 cells/mm3, as neutropenia may be transient and respond to treatment with glucocorticoids.

Risk of Bleeding

Traditionally, platelet transfusions have been recommended in surgical patients with platelet counts below 50,000 per mm3.

Adrenal Suppression/Secondary Adrenal Insufficiency

The surgical duration of an oral surgery procedure, the use of general anesthesia, the presence of infection, whether or not additional glucocorticoids are administered to reduce postoperative swelling, and the underlying health of the patient should be considered when deciding if it is necessary to prescribe supplemental glucocorticoids.

Oral Complications

SLE can be found in conjunction with Sjögren's syndrome, which is usually termed secondary Sjögren's syndrome. Sjögren's syndrome increases the risk of caries and other oral complications, which should be managed accordingly.

Scleroderma

Describes a group of clinical disorders characterized by thickening and fibrosis of the skin. The generalized form, systemic sclerosis, is a multisystem connective tissue disease in which the fibrosis extends to the internal organs, including the heart, lungs, kidney, and gastrointestinal tract. There are two main forms, **systemic sclerosis (SSc) and localized sclerodema**.

SSc is further divided into **limited cutaneous scleroderma** (previously called CREST syndrome for calcinosis cutis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia) and **diffuse cutaneous scleroderma**.

Patients with limited scleroderma often have a long history of Raynaud's phenomenon before the appearance of other symptoms. They have skin thickening limited to hands and frequently have problems with digital ulcers and esophageal dysmotility.

Diffuse scleroderma patients have a more acute onset, with constitutional symptoms, arthritis, carpal tunnel syndrome, and marked swelling of the hands and legs. They also characteristically develop widespread skin thickening (progressing from the fingers to the trunk), internal organ involvement (including gastrointestinal and pulmonary fibrosis), and potentially life-threatening cardiac and renal failure. Other possible variants are overlap syndromes with SLE, Sjögren's syndrome, RA, and dermatomyositis.

Localized scleroderma refers to scleroderma primarily involving the skin, with minimal systemic features. There are two major types of localized scleroderma: linear scleroderma and morphea.

Linear scleroderma is characterized by a band of sclerotic induration and hyperpigmentation occurring on one limb or side of the face.

Morphea is characterized by small violaceous skin patches or larger skin patches that indurate and lose hair and sweat gland function.

ETIOLOGY AND PATHOGENESIS

The etiology of SSc is unclear, but the pathogenesis is characterized by vascular damage and an accumulation of collagen and other extracellular matrix components at involved sites.

CLINICAL MANIFESTATIONS

PSS sclerosis is a chronic multisystem disorder characterized by intense fibrosis involving the skin, vasculature, synovium, skeletal muscles, and internal organs. The following is an overview of frequently encountered clinical manifestations.

Raynaud's Phenomenon. Raynaud's phenomenon, a paroxysmal vasospasm of the fingers that results in a change in the
colorof the fingertips as a response to cold or emotion, is the most common finding of PSS.

Cutaneous Manifestations. The thickening of the skin of PSS patients always begins in the fingers. Early skin changes, starting with pitting edema, often involve the whole hand and the extremities. In several months, the edema is replaced by a tightening and hardening of the skin, which results in difficulty in moving the affected parts.

Musculoskeletal Manifestations. Polyarthralgias and morning stiffness affecting both small and large joints are frequent in patients with scleroderma. Inflammatory joint pain with markedly swollen fingers often appears to be true synovitis and can lead to the premature diagnosis of rheumatoid arthritis.

Gastrointestinal Manifestations. Distal esophageal motor dysfunction is the most frequent gastrointestinal finding; it results from weakness and in coordination of esophageal smooth muscle and leads to distal dysphagia. Intestinal fibrosis leading to severe intestinal malabsorption can also occur.

Cardiac Manifestations. *Patchy fibrosis* is a term used to describe the myocardial lesions associated with SSc. Hypertension, dysrhythmias, conduction disturbances, and
left ventricular hypertrophy can develop.

Pulmonary Manifestations. Pulmonary interstitial fibrosis is now the most frequent cause of death in patients with scleroderma since renal disease has become a treatable complication.

Renal Manifestations. Until recently, renal involvement was the most dreaded and deadly complication of scleroderma. The use of high-dose corticosteroids for the treatment of scleroderma has been implicated in precipitating renal crisis in some patients.

Laboratory Evaluation and Diagnosis

The 2013 criteria include and apply various weights to the skin thickening, pulmonary manifestations, Raynaud's syndrome, telangiectases, and laboratory abnormalities (anticentromere, antitopoisomerase I and anti-RNA polymerase III).

Circulating antinuclear autoantibodies are present in >90% of scleroderma patients.
anticentromere, antitopoisomerase I and anti-RNA polymerase III are highly specific for the disease.

TREATMENT

The treatment of PSS depends on the extent and severity of skin and organ involvement. D-penicillamin has shown promise in the management of PSS by decreasing both skin thickening and organ involvement.

Oral Manifestations

The clinical signs of scleroderma of the mouth and jaws are consistent with findings elsewhere in the body. The lips become rigid, and the oral aperture narrows considerably. Skin folds are lost around the mouth, giving a masklike appearance to the face. The tongue can also become hard and rigid, making speaking and swallowing difficult. Involvement of the esophagus causes dysphagia. Oral telangiectasia is equally prevalent in both limited and diffuse forms of PSS and is most commonly observed on the hard palate and the lips. When the soft tissues around the temporomandibular joint are affected, they restrict movement of the mandible, causing pseudoankylosis.

The linear form of localized scleroderma may involve the face as well as underlying bone and teeth. Dental radiographic findings have been reported and widely described; these classic findings (which include uniform thickening of the periodontal membrane, especially around the posterior teeth) are found in less than 10% of patients.

Other characteristic radiographic findings include calcinosis of the soft tissues around the jaws.The areas of calcinosis will be detected by dental radiography and may be misinterpreted as radiographic intrabony lesions. A thorough clinical examination will demonstrate that the calcifications are present in the soft tissue.

Patients may also have oral disease secondary to drug therapy or xerostomia. Gingival hyperplasia can result from the use of calcium channel blockers; pemphigus, blood dyscrasias, or lichenoid reactions may result from penicillamine use.

Xerostomia results in an increased susceptibility to dental caries, *Candida* infections, and periodontal disease.

DENTAL MANAGEMENT

The most common problem in the dental treatment of scleroderma patients is the physical limitation caused by the narrowing of the oral aperture and rigidity of the tongue. Procedures such as molar endodontics, prosthetics, and restorative procedures in the posterior portions of the mouth become difficult, and the dental treatment plan may sometimes need to be altered because of the physical problem of access. The oral opening may be increased an average of 5 mm by stretching exercises. One particularly effective technique is the use of an increasing number of tongue blades between the posterior teeth to stretch the facial tissues. In addition, mechanical devices that assist the patient in performing the stretching exercises are available. If these approaches are insufficient, a bilateral commissurotomy may be necessary. When treating a patient with diffuse scleroderma, the extent of the heart, lung, or kidney involvement should be considered, and appropriate alterations should be made before, during, and after treatment. Patients with extensive resorption of the angle of the mandible are at risk for developing pathologic fractures from minor trauma, including dental extractions.

Patients with Sjögren's syndrome should have daily fluoride treatments and make frequent visits to the oral hygienist.

Rheumatoid Arthritis

RA is a disease characterized by symmetrical, inflammatory arthritis of small and large joints affect up to 2% of the population in the United States over the age of 60 years, with a higher prevalence in women.

SUBTYPES

Juvenile arthritis (JA) is a term to describe any arthritis in children. A subset, juvenile idiopathic arthritis (JIA) includes those children with chronic arthritis. Some clinicians refer to this subset as juvenile RA.

In general, symptoms of JIA include joint pain, swelling, tenderness, warmth, and stiffness for at least 6 weeks without another cause. Similar to RA in adults, these children may have severe joint and organ damage.

There are seven classifications of JIA: systemic arthritis, oligoarthritis, polyarthritis—rheumatoid factor (RF) negative, polyarthritis—RF positive, psoriatic arthritis, enthesitis-related arthritis, and undifferentiated arthritis. Felty's syndrome is characterized by neutropenia and splenomegaly in conjunction with RA. These patients have additional susceptibilities to bacterial infection if neutropenia is severe.

ETIOLOGY

The pathogenesis of RA is unknown, but it appears to be multifactorial, involving genetic,immune, and infectious etiologies.

CLINICAL MANIFESTATIONS

RA is a symmetric polyarthritis often involving the proximal interphalangeal joints of the fingers and metacarpophalangeal joints of the hands. ; the wrists, elbows, knees, and ankles also can be affected. In some patients, all joints may be involved, including the TMJ and the cricoarytenoid joint of the larynx. Affected joints develop redness, swelling, and warmth, with eventual atrophy of the muscle around the involved area. Cervical spine disease may cause C1–C2 subluxation and spinal cord compression. One long-term complication of RA is a marked increase in cardiovascular disease.

Oral Manifestations

The treatment of RA can cause oral manifestations. The long-term use of methotrexate and other antirheumatic agents such as D-penicillamine and NSAIDs can cause stomatitis. Cyclosporine may cause gingival overgrowth. Direct effects of the disease are also seen. Patients with long-standing active RA have an increased incidence of periodontal disease, including loss of alveolar bone and teeth. Although the increased dental and periodontal disease may be chiefly related to a decreased ability to maintain proper oral hygiene. Sjögren's syndrome is a common complication of RA.

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DIAGNOSIS AND LABORATORY EVALUATION

The initial diagnosis of RA is made primarily by observing clinical features. As with many autoimmune diseases, a list of diagnostic criteria is used to evaluate patients.

Rheumatoid arthritis: add score A through D; a score of ≥6 of 10 is needed for classification of a patient as having definite rheumatoid arthritis

Classification	**Score**
A. Joint involvement	
• 1 large joint (shoulders, elbows, hips, knees, ankles)	0
• 2–10 large joints	1
• 1–3 small joints (with or without large joints)	2
• 4–10 small joints (with or without large joints)	3
• >10 joints (at least one small joint)	5
B. Serology (at least 1 test result is needed for classification)	
• Negative rheumatoid factor (RF) and negative anticitrullinated protein antibody (ACPA)	0
• Low-positive RF or low-positive ACPA	2
• High-positive RF or high-positive ACPA	3
C. Acute-phase reactants (at least 1 test result is needed for classification)	
• Normal C-reactive protein (CRP) and normal erythrocyte sedimentation rate (ESR)	0
• Abnormal CRP or abnormal ESR	1
D. Duration of symptoms	
• <6 weeks	0
• ≥6 weeks	1

DENTAL MANAGEMENT

The most common complication that affects dental treatment relates to the toxicity of the drugs used to treat RA. It is imperative that the dentist knows the drugs the patient is currently taking and their possible side effects and interactions with other drugs. The most common adverse effects of NSAIDs involve the gastrointestinal (GI) tract and the kidneys. In addition, many patients take aspirin at dosages approaching 5 g per day or take an equivalent dosage of NSAIDs. These drugs affect platelet function, causing a prolongation of the bleeding time and possible hemorrhage after surgery. Patients with severe RA who have had joints surgically replaced with prosthetic joints may require prophylactic antibiotic therapy before invasive dental procedures.

Patients with cervical spine disease may have C1–C2 subluxation and spinal cord compression. Hyperextension of the neck must be avoided. Prolonged morning stiffness is common in RA, so later morning appointments may be best for patients. Patients with severe RA who have prosthetic joints may require prophylactic antibiotic therapy beforeinvasive dental procedures, though the evidence for the practice is very limited. Patients with Sjögren's syndrome may require additional instruction in personal oral care and instruction on diet and dietary modifications.

The dentist should determine if the RA patient has a form of the disease that affects the bone marrow (such as Felty's syndrome) since such patients have an increased risk of developing infection due to neutropenia and hemorrhage secondary to thrombocytopenia.

Mixed Connective-Tissue Disease

The term "mixed connective-tissue disease" (MCTD) was coined to denote a condition that has the combined clinical features of SLE, PSS, and DM. The cause of MCTD, as with other rheumatologic diseases is unknown.

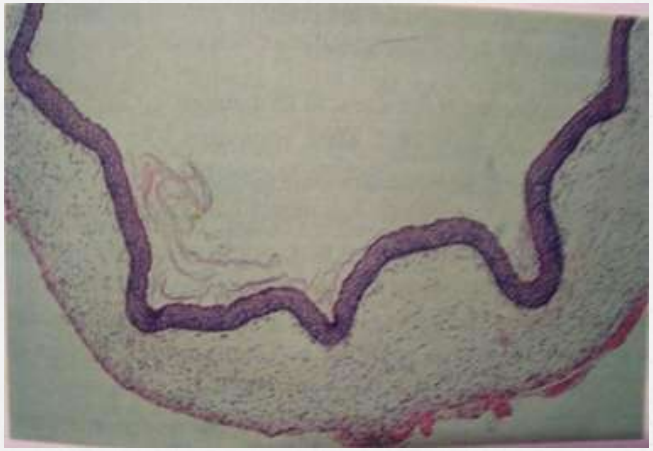
Common clinical features of MCTD include Raynaud's phenomenon, polyarthritis, sclerodactyly, and inflammatory myositis. Generalized lymphadenopathy has been observed in 50% of patients with MCTD. Pericarditis, renal disease, and pulmonary disease are also common. A prerequisite for the diagnosis of MCTD is the presence of high titers of autoantibodies against small nuclear ribonucleoprotein (SnRNP) antigen. Little has been reported concerning the oral manifestations of MCTD. The oral manifestations of conditions such as xerostomia and a decreased mandibular range of movement are possible features although few reports are available.

Cysts of jaw

Dr. Sabah Alheeti

Definition

- Cyst is a pathological cavity having fluid, semi-fluid or gaseous contents that are not created by the accumulation of pus, frequently but not always, is lined by epithelium



WHO Classification of jaw cysts

A. Developmental cysts

1. Odontogenic

a) Primordial cyst (keratocyst)

b) Gingival cyst

c) Eruption cyst

d) Dentigerous cyst (folicular)

2.Non-odontogenic

a) Nasopalatine (incisive canal)

b) Globulomaxillary

c) Nasolabial

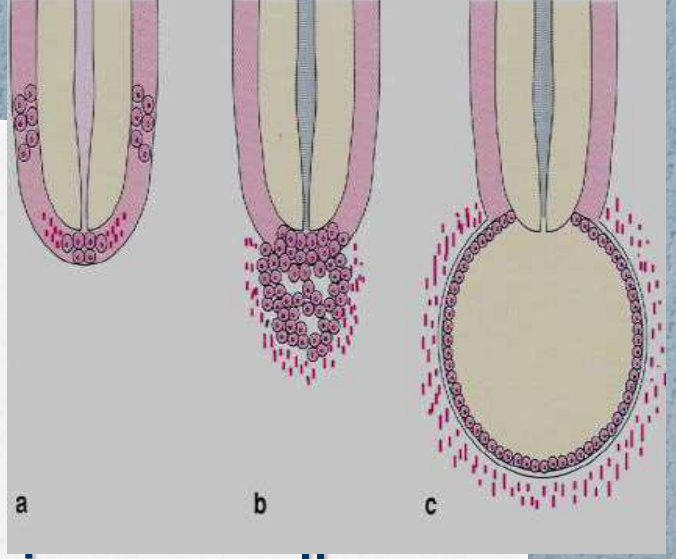
B- Inflammatory cysts:

- Radicular (periapical) cysts
- Residual
- Paradental

C- FALSE cysts:

- Solitary bone cyst
- Aneurysmal bone cyst

PATHOGENESIS



Steps in Cyst Formation

- The formation of a cyst takes place in generally three stages:
- 1. Initiation
- 2. Cyst formation
- 3. Enlargement or expansion of cyst cavity by 3 mechanisms:

A. Increase in the volume of its contents.

B. Increase in the surface area of the sac

C. Resorption of surrounding bones

Typical features of jaw cysts

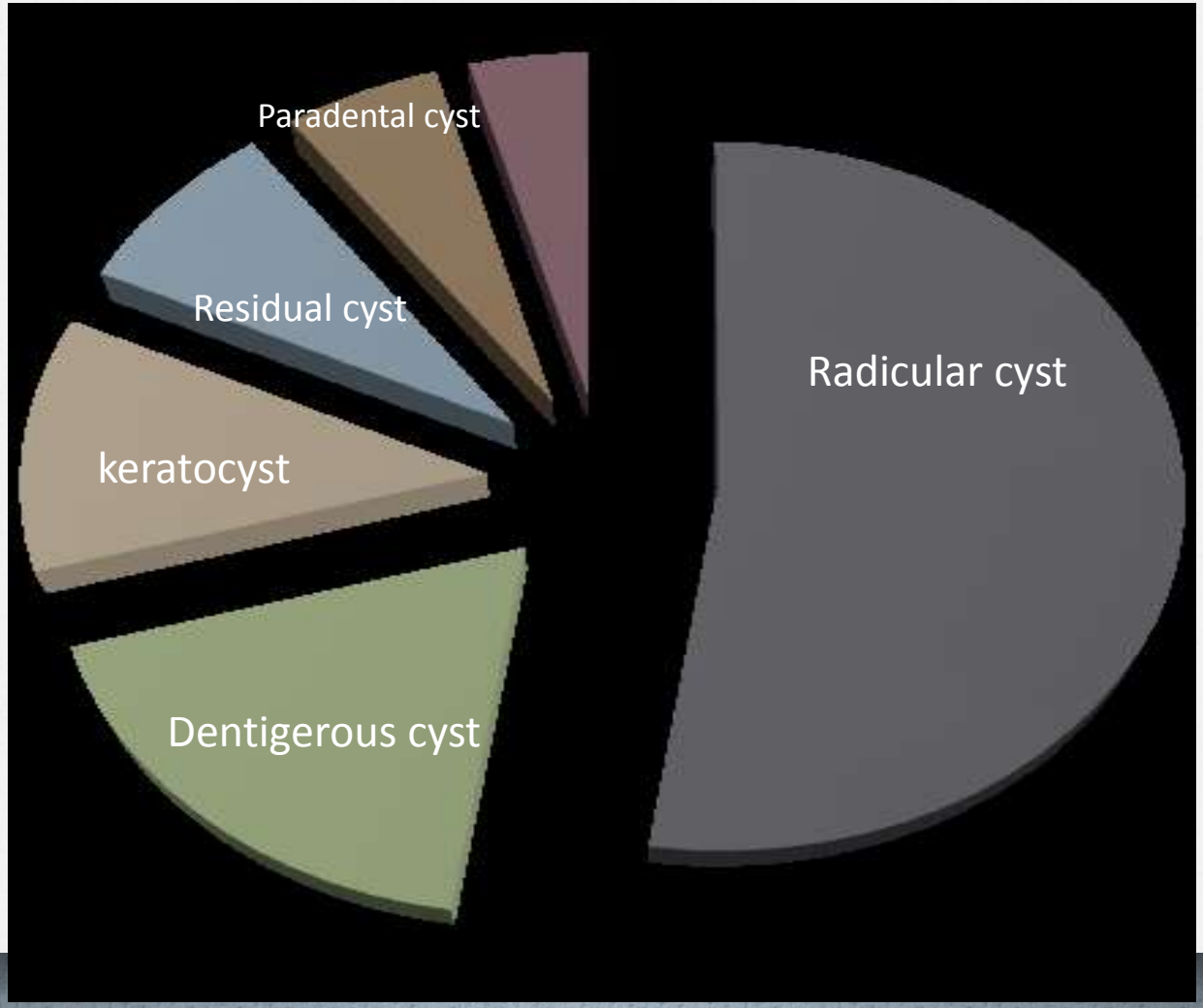
- Form sharply- defined radiolucencies with smooth borders.
- Fluid may be aspirated
- Grow slowly, displacing rather than resorbing teeth
- Symptomless unless infected and frequently chance radiographic findings
- Rarely large enough
- Eggshell cracking
- Form compressible and flactuant swellings if extending into soft tissue
- Appear bluish when close to mucosal surface.

Diagnostic work-up

- History
- Clinical features (mainly age, gender, site, tooth vitality)
- Radiograph (OPG)
- Aspiration
- Biopsy

Diagnosis

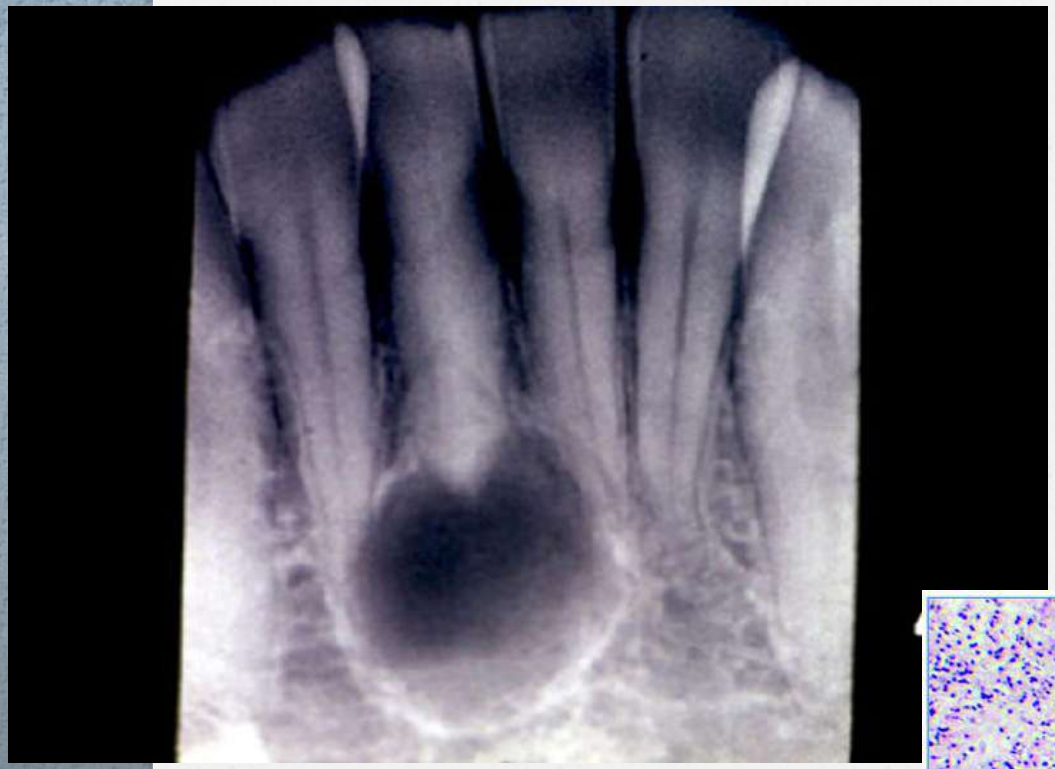
Frequency of epithelial cysts

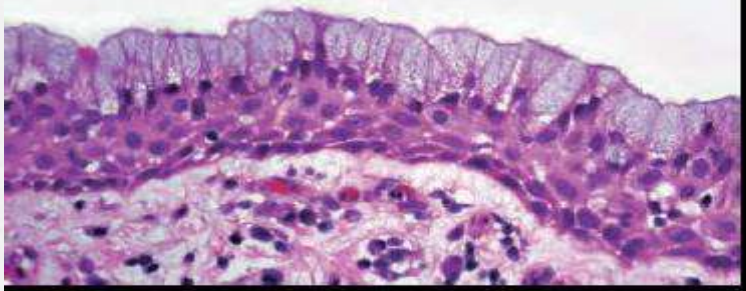


Radicular (Periapical) Cyst

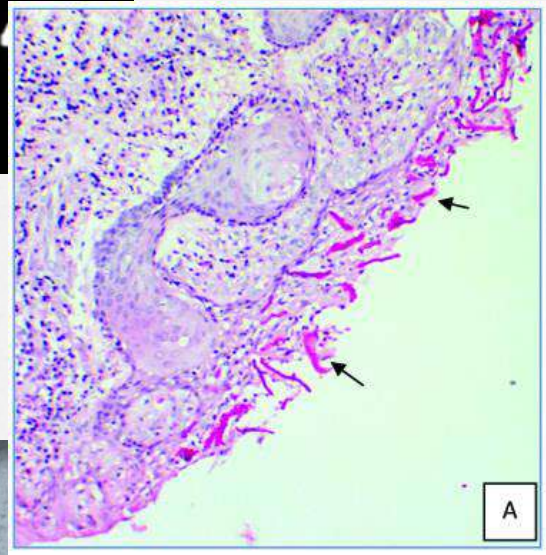
- Most common (65%)
- Epithelial cell rests of Malassez
- Age: peak in 3rd, 4th and 5th decades
- Gender: male bias
- Site : maxillary anterior region
- Radiographic findings
  - Pulpless, nonvital tooth
  - Small well-defined periapical radiolucency
- Histology
- Treatment – extraction, root canal

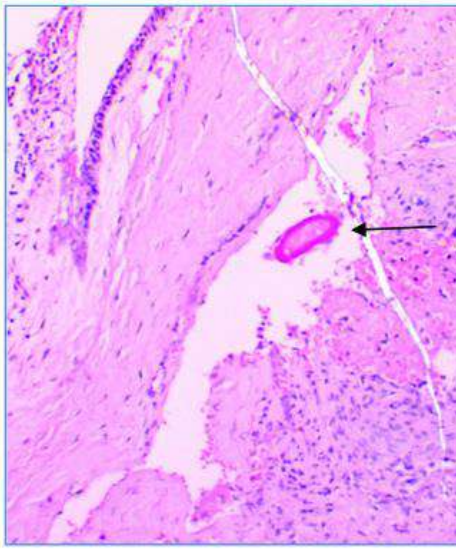
Radicular Cyst



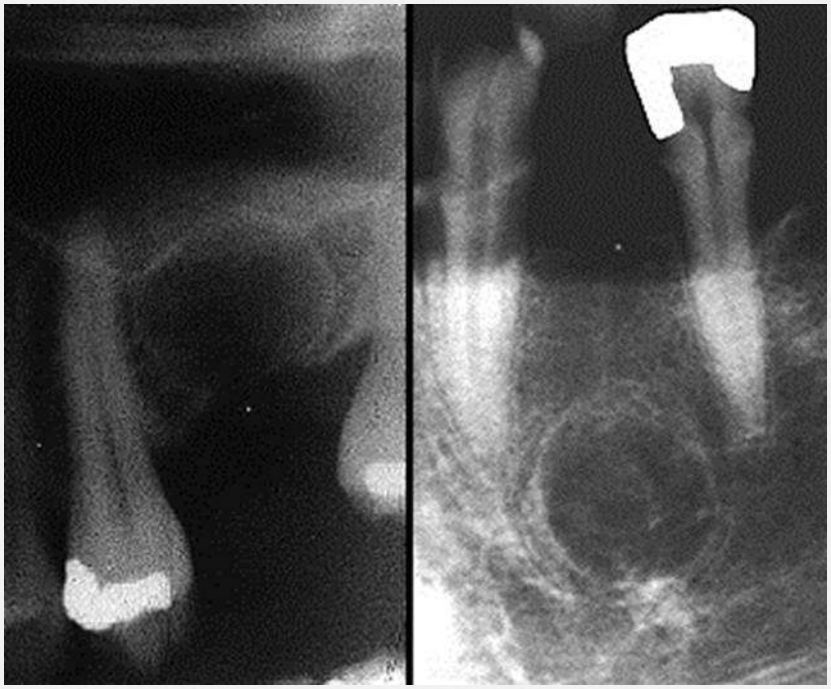


Mucous cells in the surface layer of the stratified squamous epithelial lining of a radicular cyst (H & E).



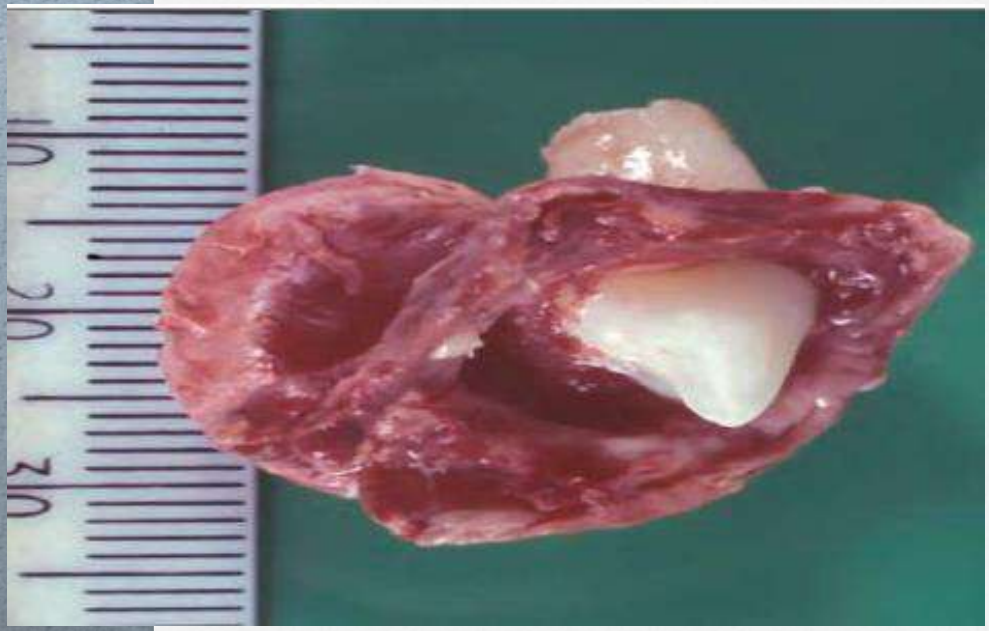


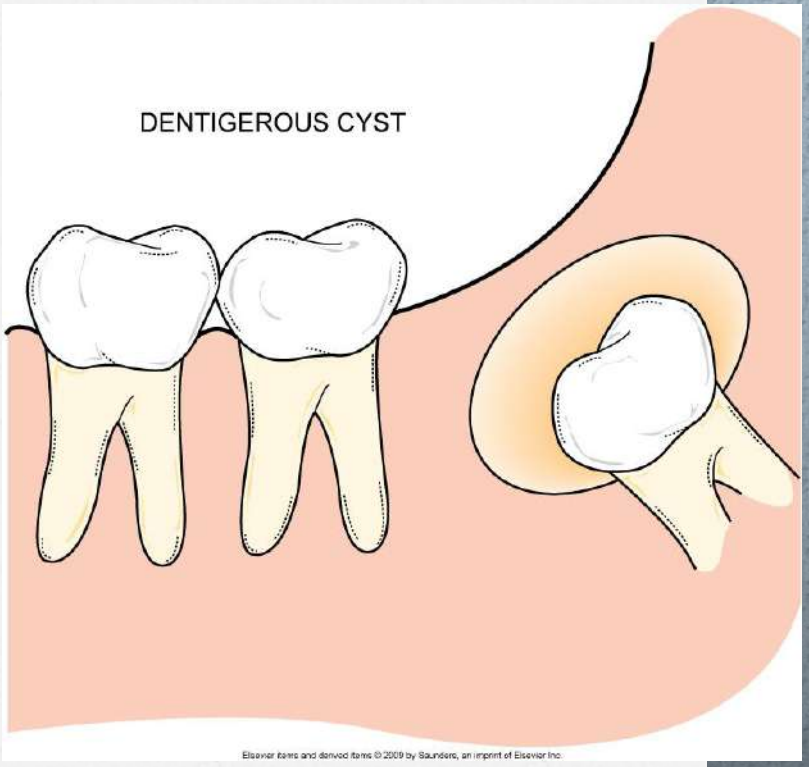
Residual Cyst



Dentigerous cyst

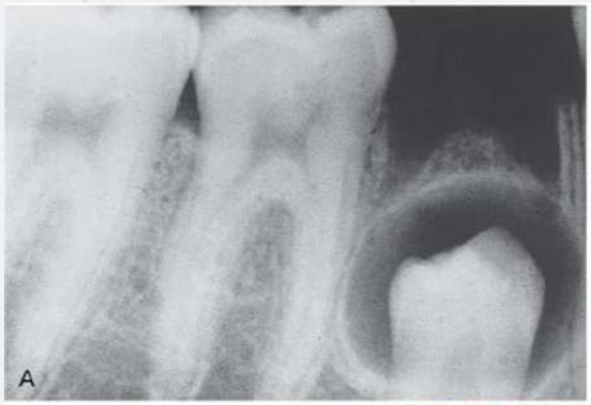
- The most common developmental odontogenic
- A dentigerous cyst occurs in association with an unerupted tooth, most commonly, mandibular third molars
- is attached to the tooth at the cementoenamel junction
- Age : 1st to 3rd decades. Male predilection, most commonly, mandibular third molars

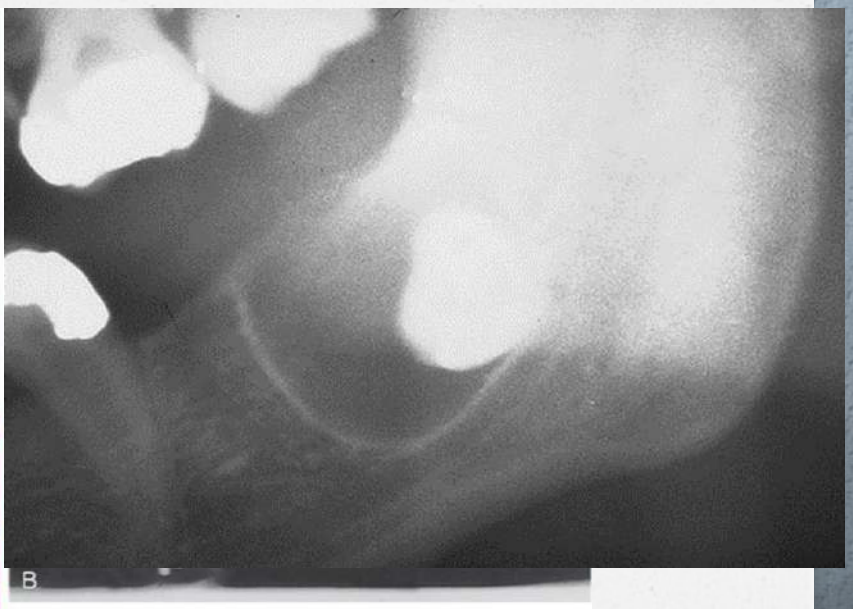


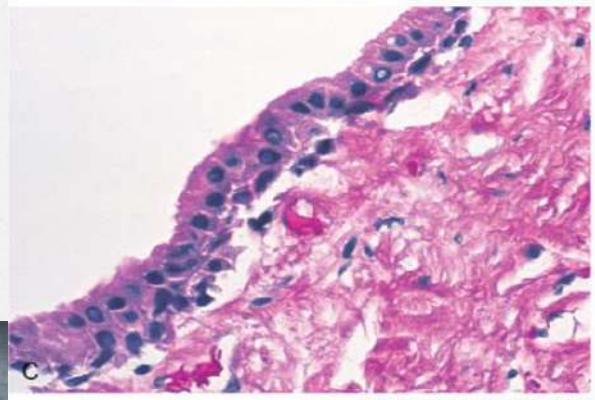

DENTIGEROUS CYST

Radiograph

- A well-defined, unilocular radiolucency around the crown of an unerupted or impacted tooth





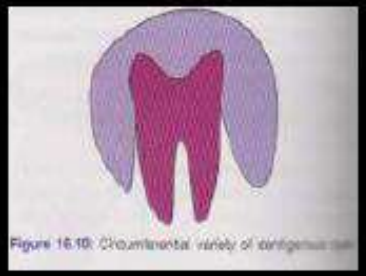


RADIOLOGICAL FEATURES

- CENTRAL TYPE:

- LATERAL TYPE :

- CIRCUMFERENTIAL TYPE :

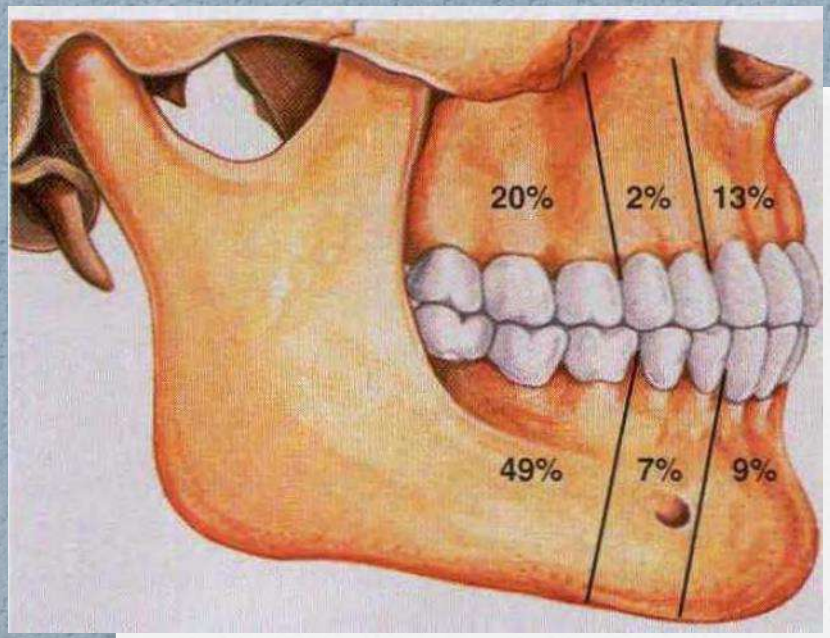


Treatment

- Enucleationtogether with removal of the unerupted tooth
- Marsupialization

Odontogenic Keratocyst (OKC)

- Age: 10-40 yrs, male predilection, mandibular posterior area (premolar-molar)
- Characterized by frequent recurrence after enucleation.
- Asymptomatic till reach large size or become infected.
- The lumen of the cyst contains perakeratin.
- Tend to grow in AP direction without cause obvious bony expansion.
- Well defined R.L. mostly multilocular , with scalloped margin+ may associated with unerupted tooth
- Dx. By clinical, radiographical and histopathological findings.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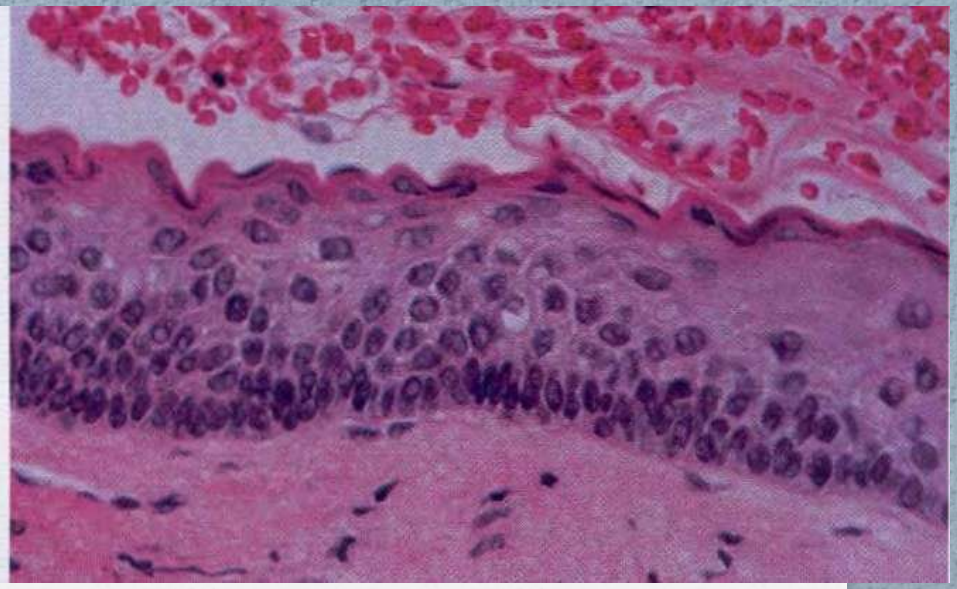
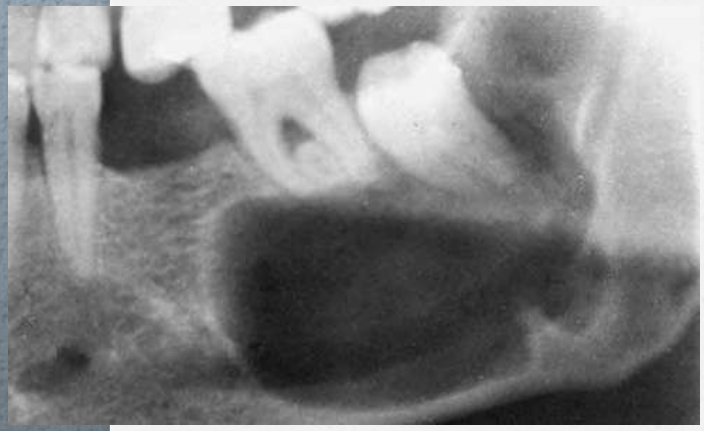
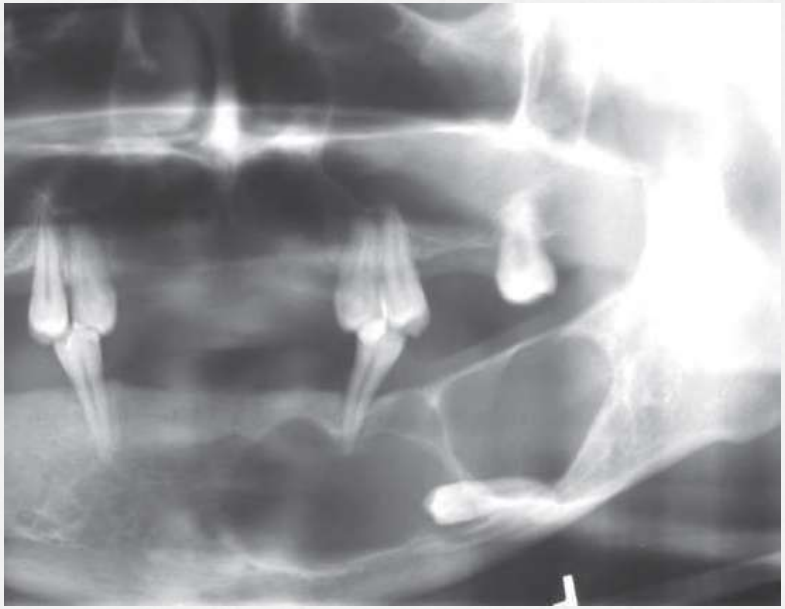






- Enucleation with adjuvant carnoy solution

- Complications:recurrence due to:

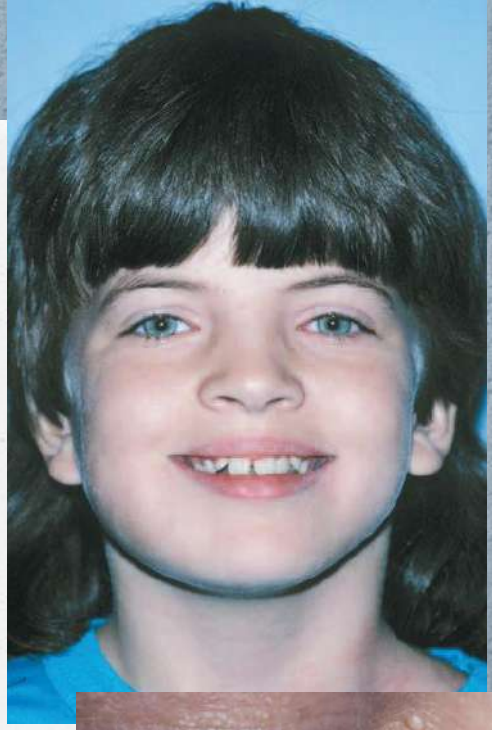
1.Thin, fragile lining .

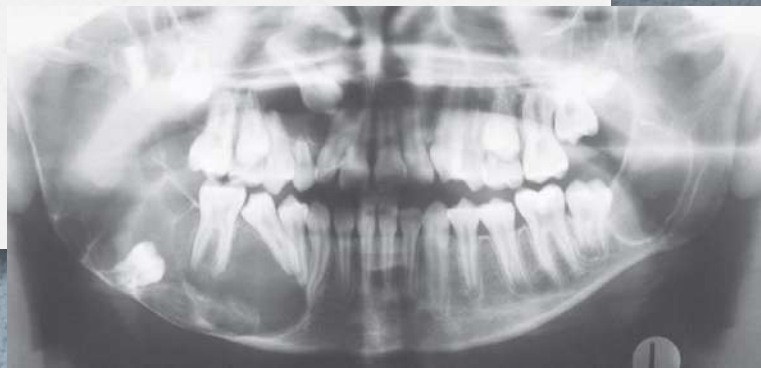
2. New cysts develop from satellite cysts left behind.

- 3. Some cysts may be left behind in cases of Gorlin – Gotz syndrome.
- 4. New cysts can also develop from basal cells of overlying oral epithelium

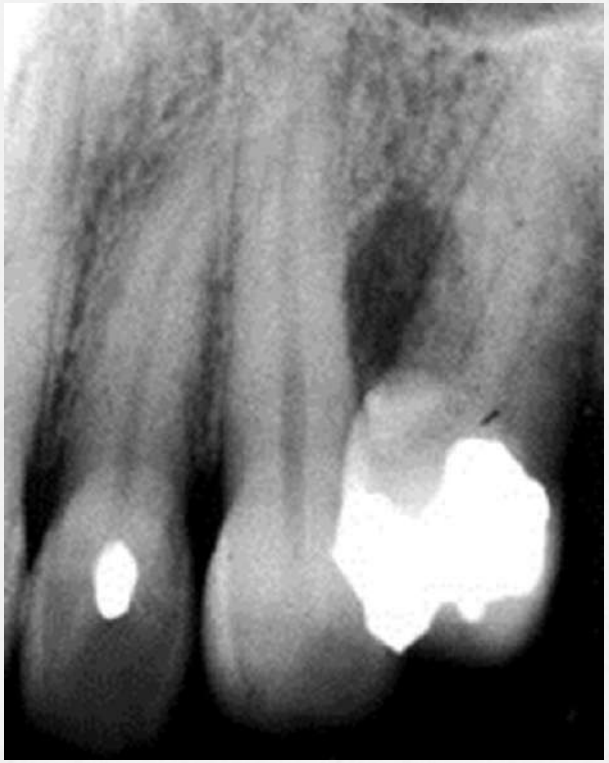
NEVOID BASAL CELL CARCINOMA SYNDROME (GORLIN SYNDROME)

- autosomal dominant inherited condition
- 50% OR GREATER FREQUENCY
- Multiple basal cell carcinomas
- Odontogenic keratocysts
- Epidermal cysts of the skin
- Palmar/plantar pits
- Calcifi ed falx cerebri
- Enlarged head circumference
- Rib anomalies (splayed, fused, partially missing bifi d)
- Mild ocular hypertelorism



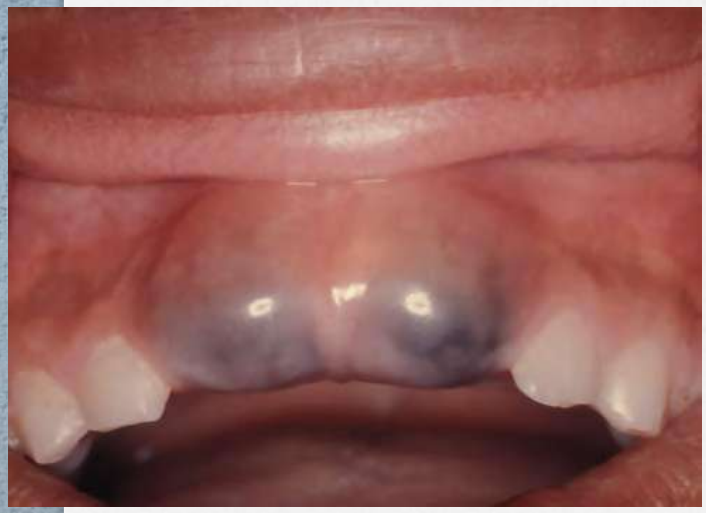


Lateral periodontal cyst

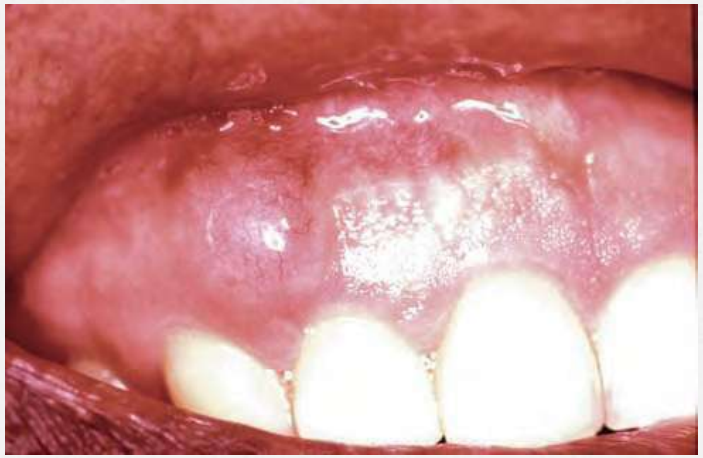


- peak in 6th decade, male, lateral side of PDL especially mandibular premolars
- Round to ovoid ‘lucency with sclerotic margins.
- Cyst can be present anywhere between cervical margin to root apex.

- Cysts rarely < 1cm in size, except for BOTRYOID VARIETY which is larger and also a multilocular lesion



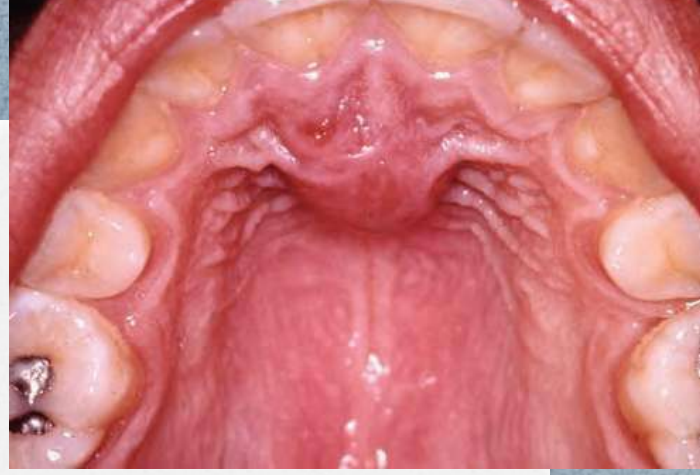
gingival cyst of an adult



Gingival cyst of an adult

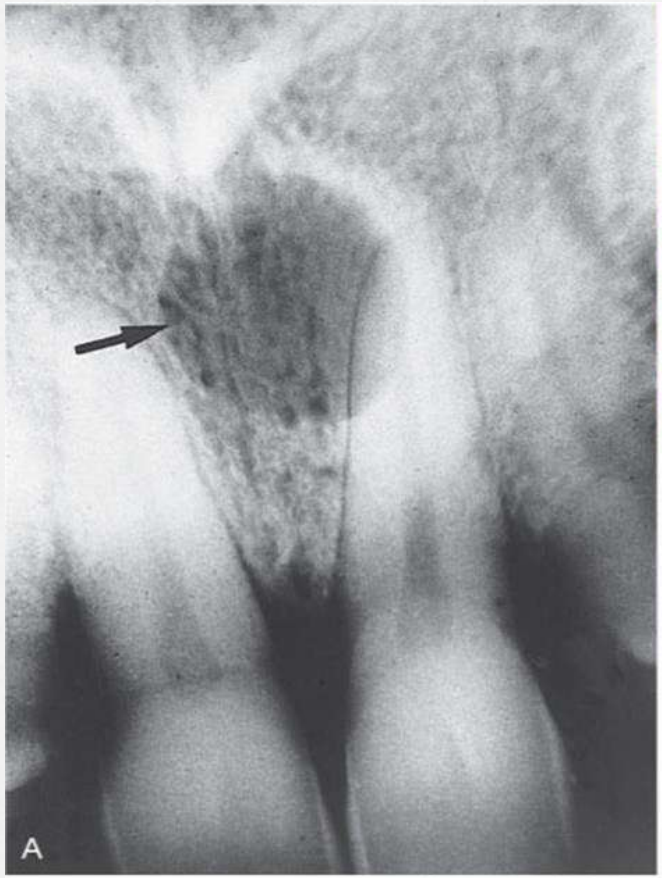
Developmental non-odontogenic cysts

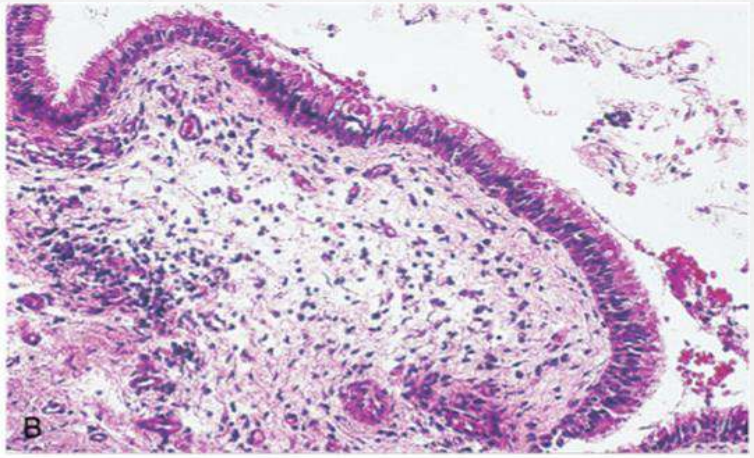
NASOPALATINE DUCT (INCISIVE CANAL) CYST



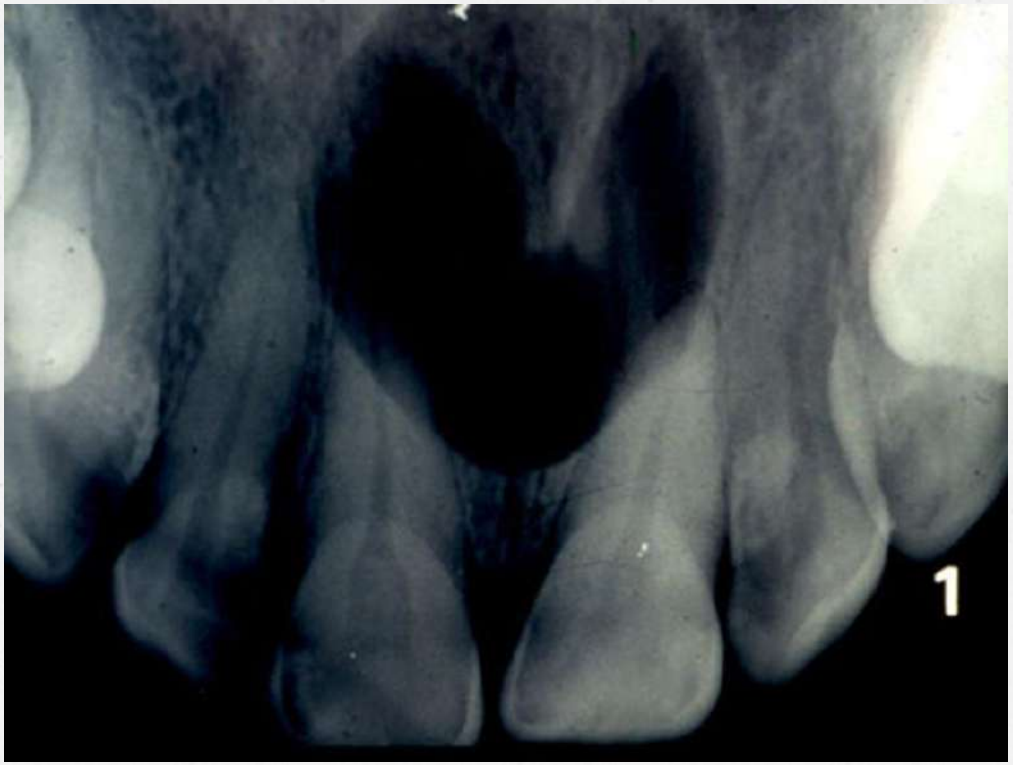
- Believed to be derived from epithelial remnants included during closure of embryonic facial processes.
- Site: Usually occurs within the nasopalatine canal or in soft tissue of palate at the opening of canal.
- 4th, 5th & 6th decades + More in females.
- Commonest symptom is swelling, usually in anterior region of mid palate.
- Swelling can also occur in midline on labial aspect of
- alveolar ridge.
- If pressure on NP nerves – pain

Nasopalatine duct cyst





The lucency appears well defined with sclerotic borders, in midline of palate between roots of incisors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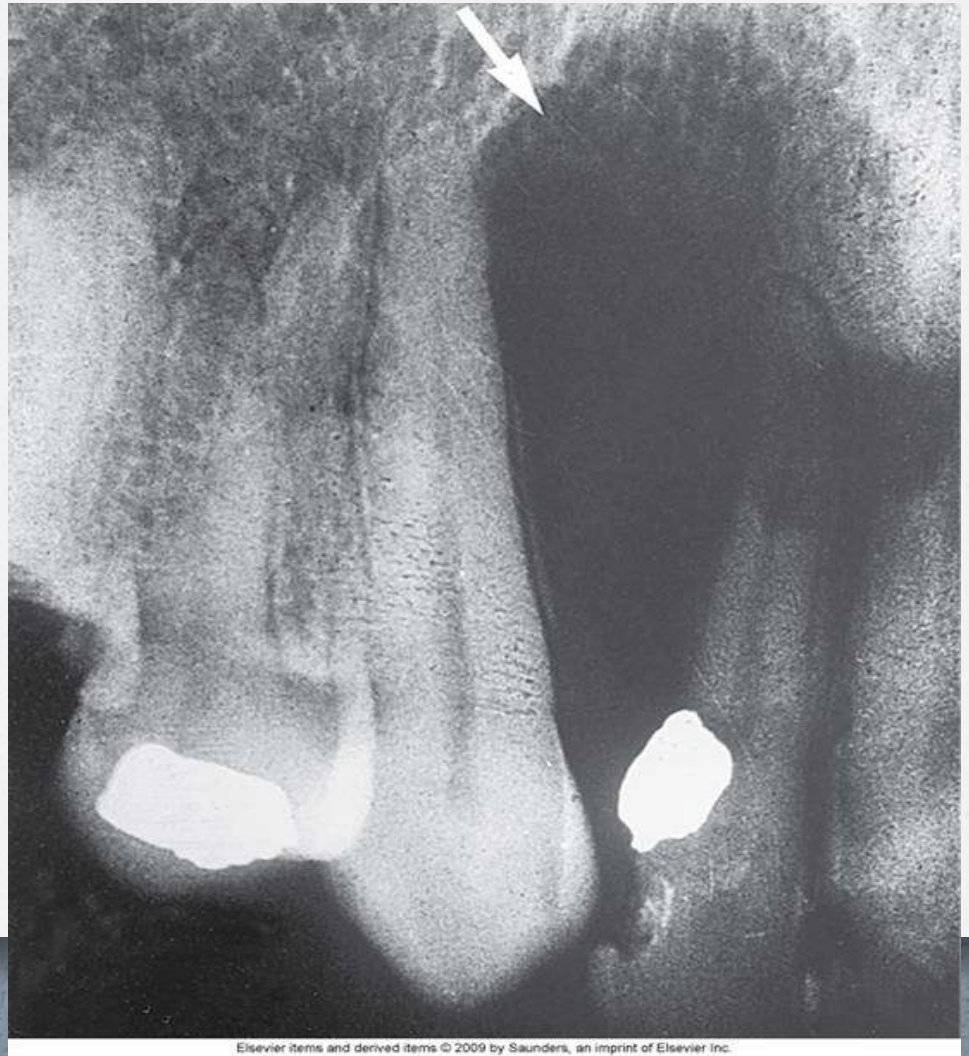
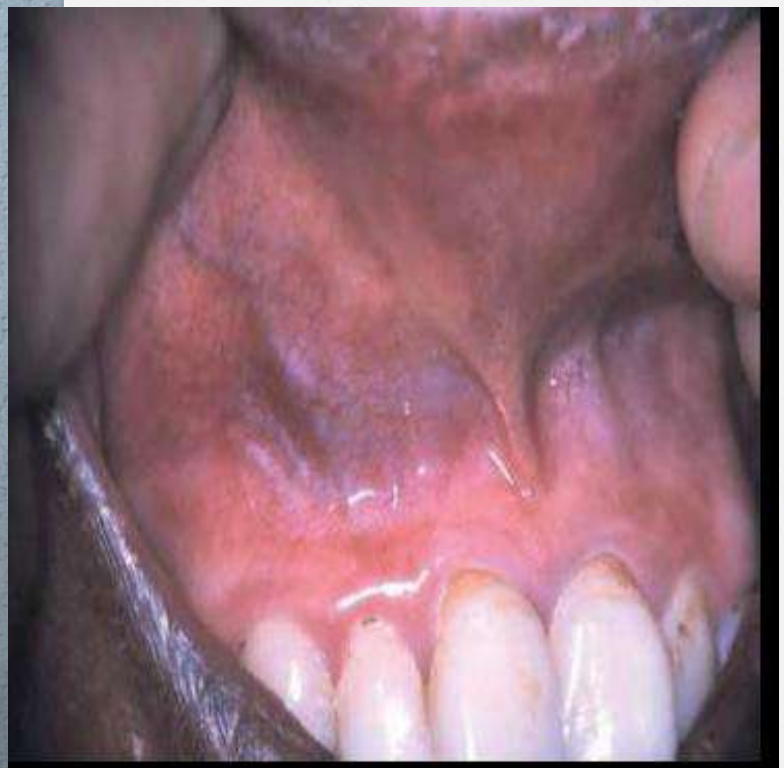
Globulomaxillary Cyst

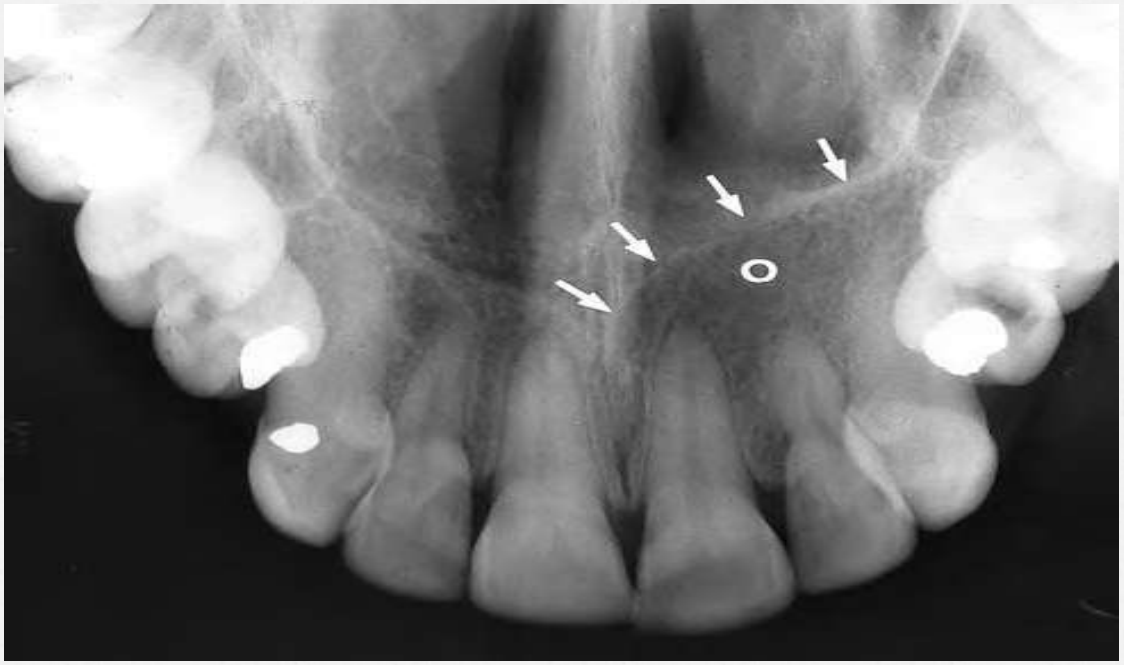


Nasolabial Cyst



Nasolabial cyst producing a swelling of the right upper lip, forming a bulge in the labial sulcus.

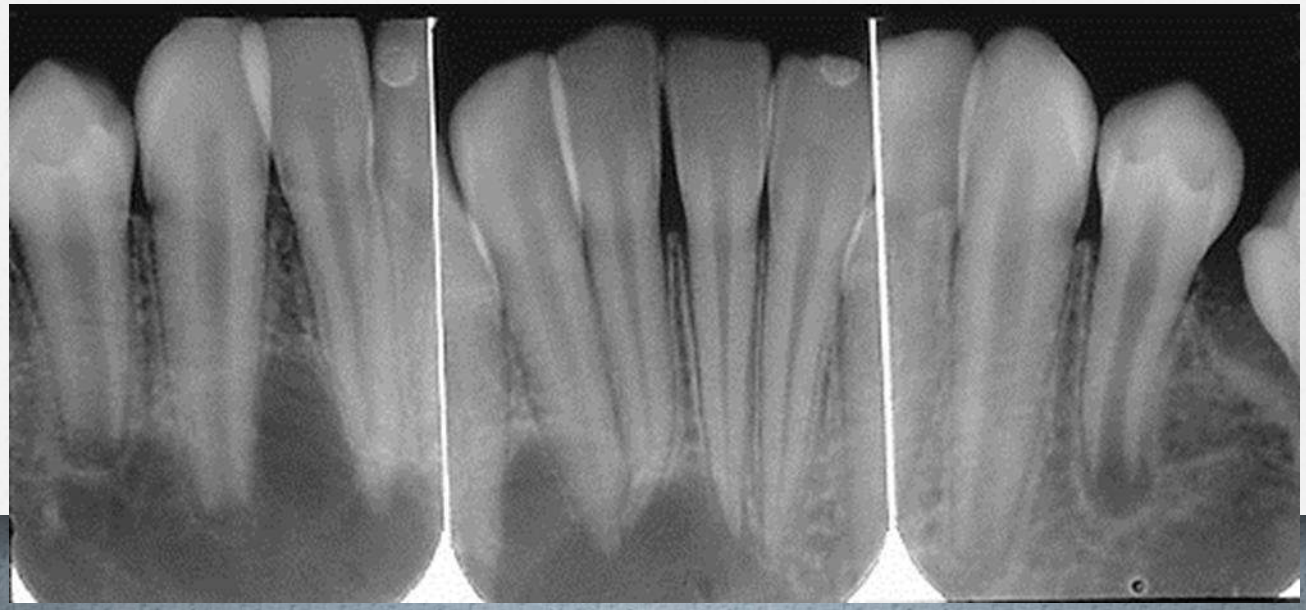
- Believed to develop from lower anterior portion of nasolacrimal duct.
- occurs outside the bone in the nasolabial folds below the alae nasi
- in 4th & 5th decades+ More in females.
- Extra orally – filling out of nasolabial fold and may lift ala nasi.



May be seen as localized increased lucency of alveolar process above apices of incisors

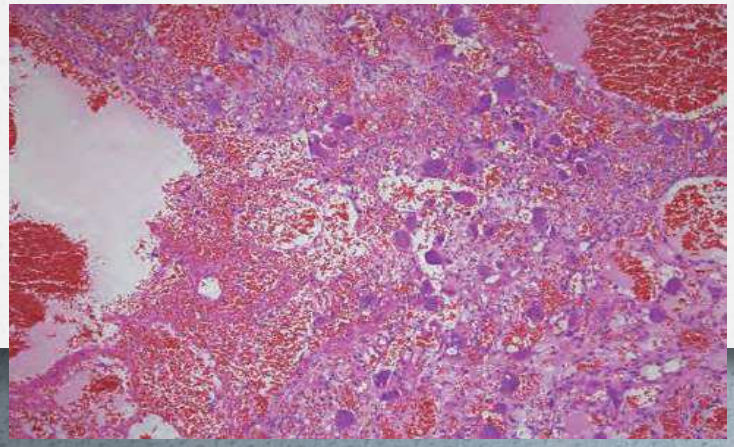
Solitary bone cyst
(haemorrhagic, traumatic)

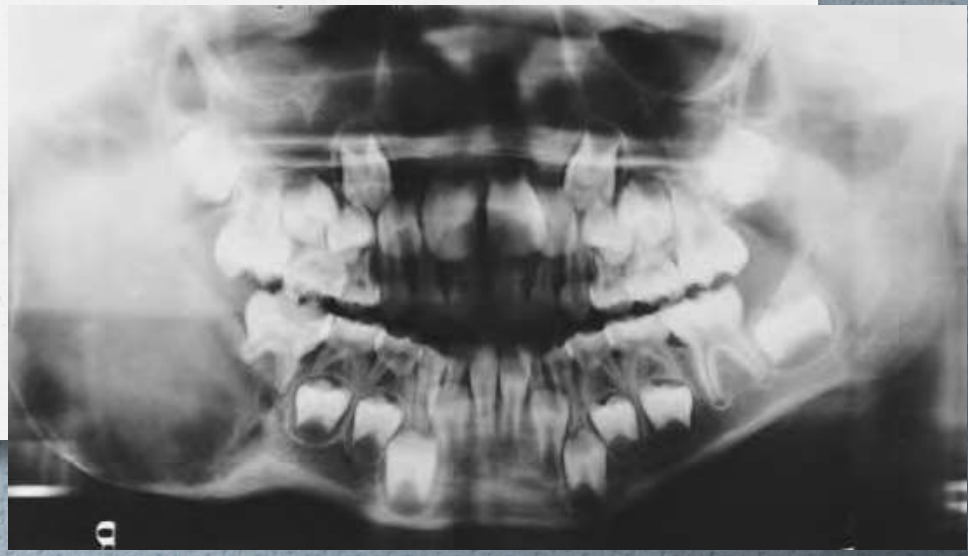
- Young individuals+ Equal gender+ Body and symphysis menti of mandible.
- Asymptomatic. Rarely, swelling and pain may be seen
- (dome- like projections upward)



Aneurysmal cyst

- Age : First 3 decades+Mainly females+ molar regions of mandible & maxilla.
- Hard, rapidly growing swelling which can cause malocclusion.
- If lesion perforates cortical plates, can cause “egg shell crackling”.
- ‘soap-bubble’R.L.+ballooning





- Any radiolucent lesion should be aspirated before surgical exploration.
- This provides the dentist with valuable diagnostic information regarding the nature of the lesion

PATHOLOGY	ASPIRATE	Other Findings of Aspirates
Dentigerous Cyst	Clear, pale straw colour fluid	Cholesterol crystals. Total protein in excess 4 g / 100ml. Resembles serum
Odontogenic Keratocyst	Dirty, creamy white viscoid suspension	Para keratinized squames. Total protein less than 4 g /100ml. Mostly albumin
Periodontal Cyst	Clear, pale yellow straw colour fluid	Cholesterol crystals. Total protein 5 — 11g / 100ml
Infected Cyst	Pus, brownish fluid	Polymorphonuclear leukocytes, ,Cholesterol clefts
Mucocele, Ranula	Mucus	-----
Gingival Cysts	Clear fluid	-----

Operative Procedures

- Marsupialisation
- Enucleation
- Enucleation after marsupialization
- Enucleation with curettage
- Resection

Marsupialisation or Partsch I Operation (cystotomy)

- • In this procedure, a window or a fenestration is made in the bone and the cystic contents are evacuated. The cyst lining is left behind.
- • Once the cyst contents are evacuated, the intracystic pressure reduces. The hollow cavity is then packed till it gets obliterated by bone slowly over a period of time.
- • The cystic lining then becomes continuous with the normal oral mucosa

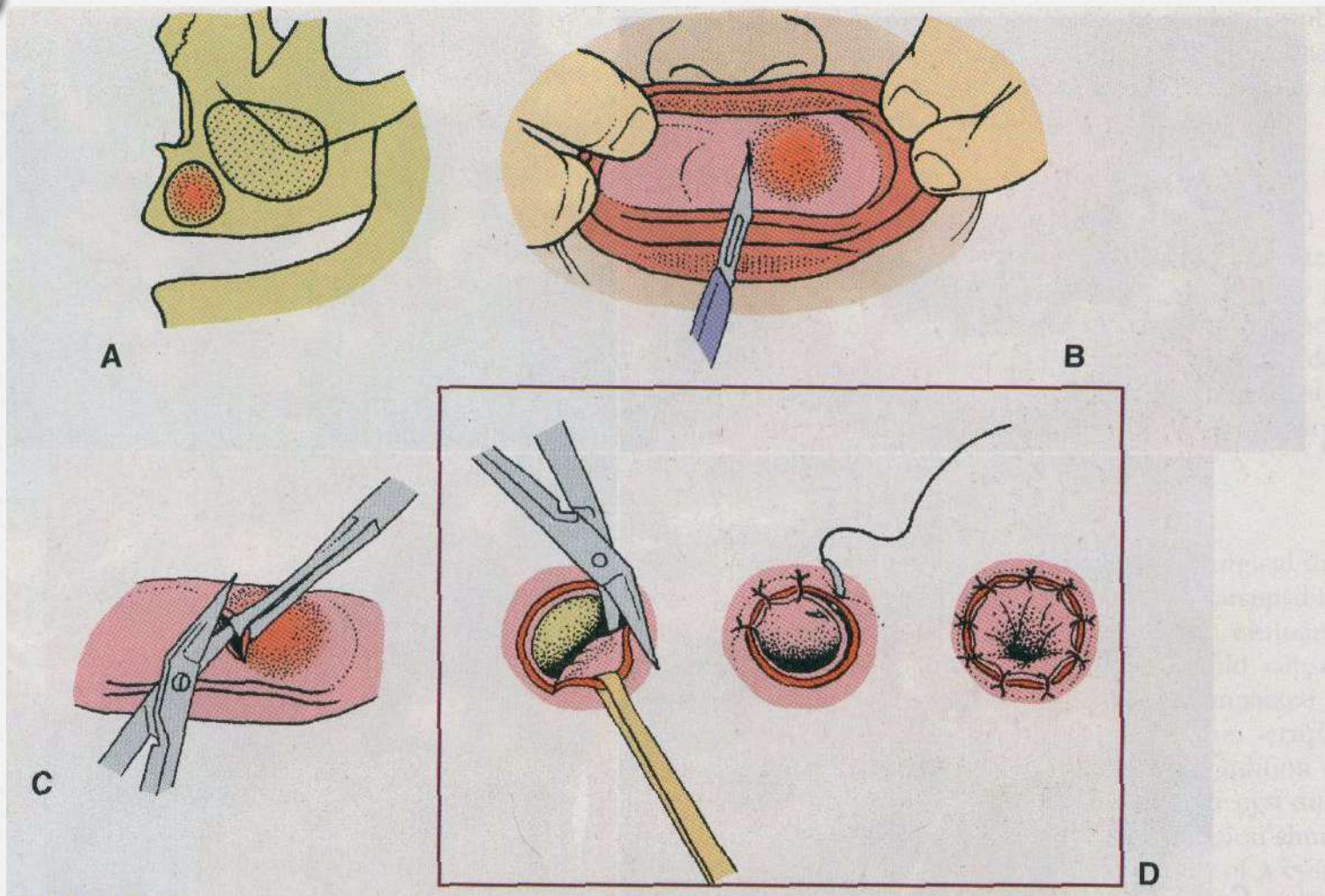


FIGURE 22-6 Marsupialization technique. A, Cyst within maxilla. B, Incision through oral mucosa and cystic wall into center of cyst. C, Scissors used to complete excision of window of mucosa and cystic wall. D, Oral mucosa and mucosa of cystic wall sutured together around periphery of opening.

Indications for Marsupialisation

- 1. Extremely large cyst to reduce pathological fracture
- 2. proximity to vital structures e.g. antrum, nerve, BVs, tooth.
- 3- access to all portions of cysts is difficult
- 4- assistance in tooth eruption
- 5- Extent of surgery

Advantages

- Simple procedure
- Spare vital structures from damage

Disadvantages

- *1.* Entire pathological tissue is left behind.
- 2. High chances of recurrence of the cyst.
- 3. As the bony cavity is large, healing and filling up with normal bone takes a long time.
- 4. Use of cyst plug is required with repeated cleansing.
- 5. Time consuming and repeated appointments for the patient

Enucleation (Partsch ll/cystectomy)

- Enucleation is the surgical removal of the entire cystic lining in toto.
- By definition, it means shelling out of the entire cystic lining without rupture.
- This surgical procedure leaves behind a hollow cavity in bone covered by oral mucoperiosteum. This gets filled up with blood clot which eventually organizes to form healthy bone.

Enucleation with primary closure

Enucleation open packing:-

- Gauze impregnated with bismuth iodoform parraffin paste (BIPP) or whitehead varnish.

Enucleation with bone grafting:-

- • Bone grafting with autogenous cancellous bone grafts can be done in case of large bony defects.
- • The bone graft obliterates the cavity and stimulates osteogenesis.
- • There is, however, a risk of wound break-down and infection of the bone graft which may lead to failure

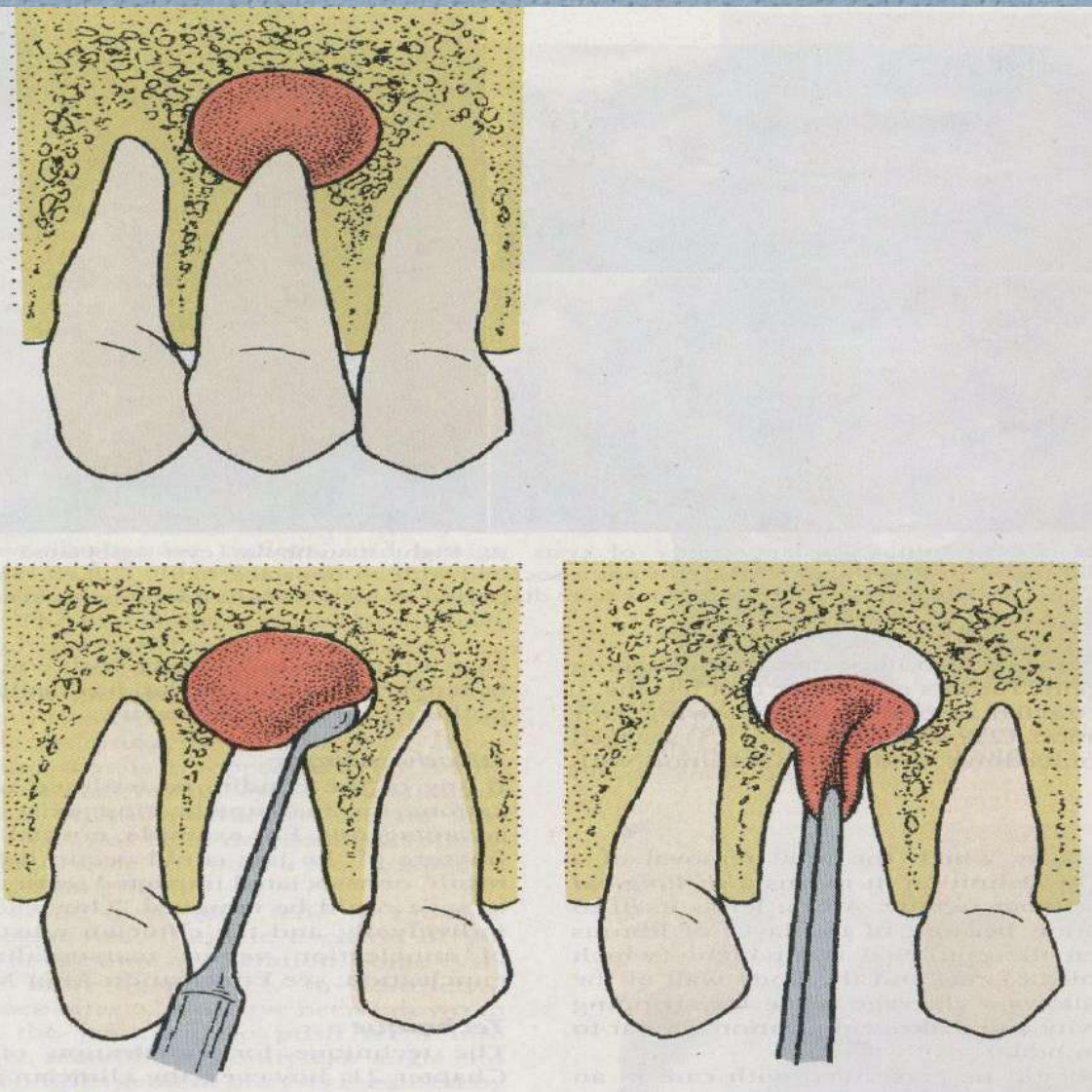


FIGURE 22-4 Apical cystectomy performed at time of tooth removal. A to C, Removal with curette via tooth socket is visualized. An apical cystectomy must be performed with care because of proximity of apices of teeth to other structures, such as maxillary sinus and inferior alveolar canal.

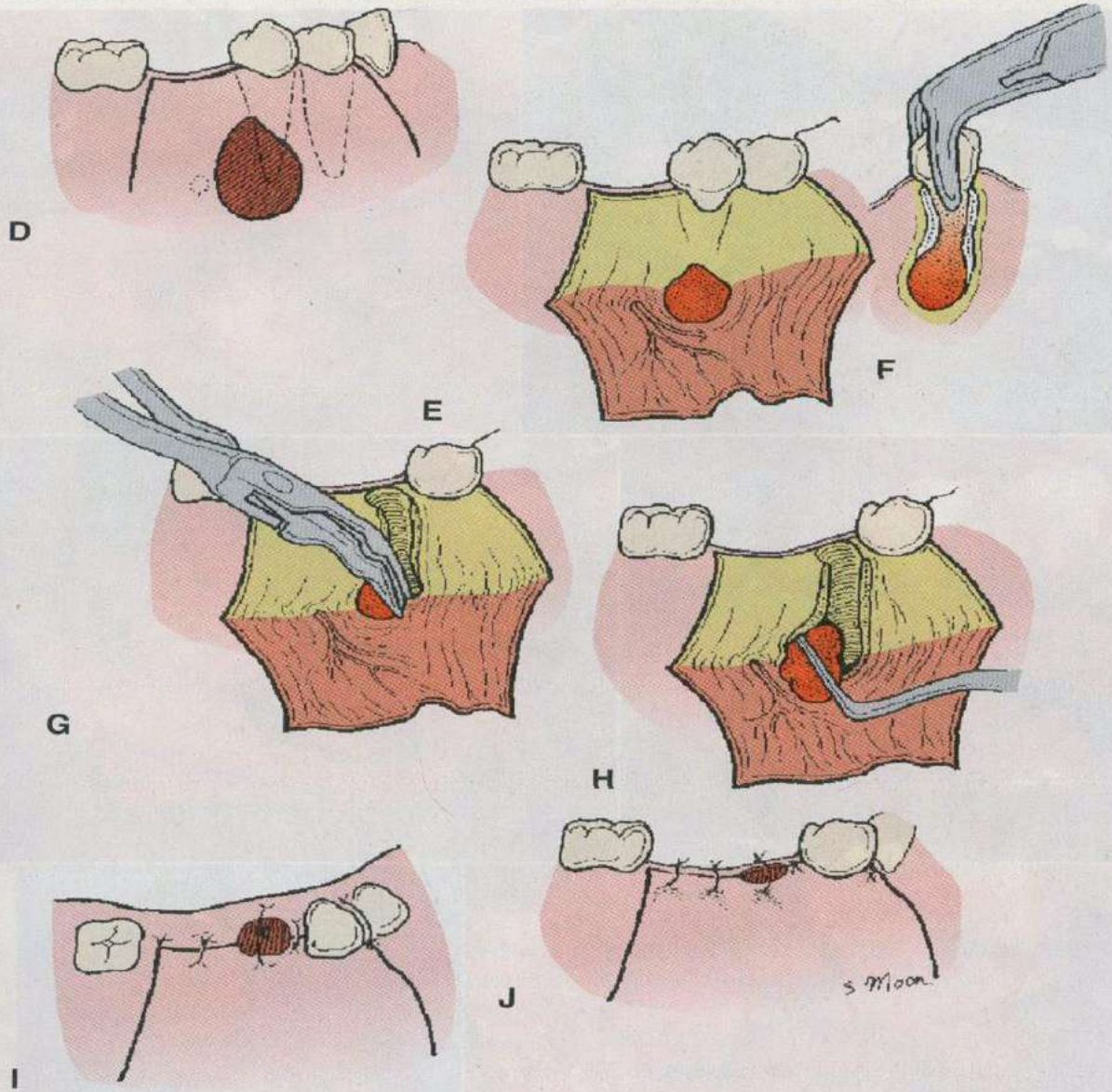


FIGURE 22-4, cont'd Apical cystectomy performed at time of tooth removal. D to J, Removal of apical cyst by flap reflection and creation of osseous window is demonstrated at the time of tooth removal.

Indications for Enucleation

- Enucleation is the treatment of choice for removal of cysts of the jaws and should be employed with any cyst of the jaw that can be safely removed without unduly sacrificing the underlying structures

Advantages

- 1. Entire pathological tissue is removed from the lesion.
- 2. Tissue available for histopathological examination.
- 3. Chances of recurrence are less.
- 4. Healing time is faster and less appointments for the patient
- 5. Enucleation with primary closure eliminates the need for repeated appointments for packing medicated gauze, irrigation, fabrication of plug etc.

Disadvantages

- 1. Relatively radical procedure
- 2. Chances of devitalising the adjacent teeth
- 3. Chances of fracture of the jaw
- 4. Risk of creation of oroantral/oronasal communication.

Enucleation after marsupialization

- Initial healing is rapid after marsupialization but the size of the cavity may not decrease appreciably past a certain point.
- Combination procedure will reduce morbidity and accelerate healing of the defect.

Indications

- Same of marsupialization. However, if the cyst cavity not obliterated totally after marsupialization.
- Cystic cavity that the patient is finding difficult to cleanse.

- The advantages are the same as those listed for marsupialization and enucleation.
- The disadvantages are the same those for marsupialization.

Enucleation with curettage

- After enucleation a curette or bur is used to remove 1-2 mm of bone around the entire periphery of the cystic cavity.
- This is done to remove any remaining epithelial cells that give recurrence.

Indications

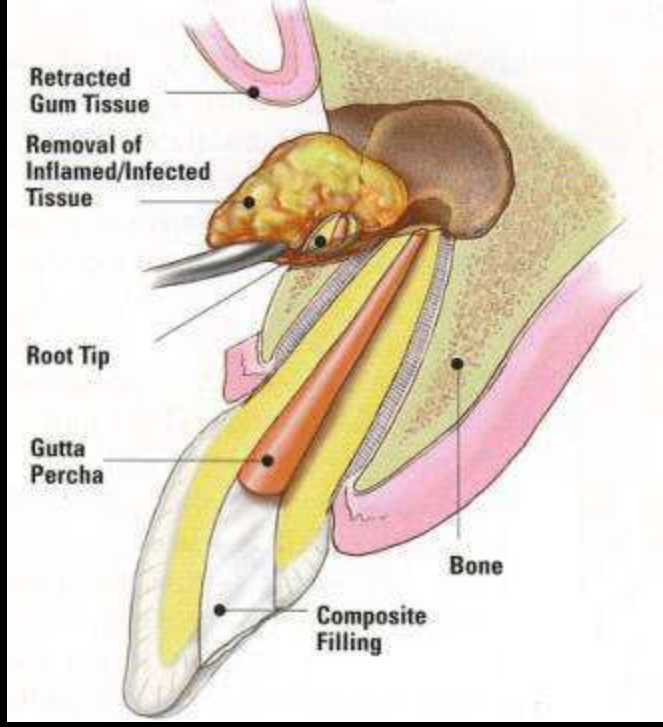
- 1- in case of odontogenic keratocyst.
- 2- in any cyst that recur after what was deemed a thorough removal.

- Advantages : reduce the likelihood of recurrence.
- Disadvantages : curettage is more destructive of adjacent bone and other tissues.

Complications of cyst management

1. Injury to inferior alveolar nerve
2. Injury to adjacent teeth
3. Fracture of jaw
4. Oro antral fistula communication
5. Hematoma formation
6. infection
7. Dead space
8. Incomplete removal
9. Recurrence
10. Malignant transformation

Endodontic surgery



By

Dr. Sabah Alheeti

Definition and classifcation

- Endodontic surgery is the management or prevention of periradicular pathosis by a surgical approach. this includes

Objectives

- To ensure the placement of a proper seal between the periodontium and the root canal foramina
- To remove the causative agents of periradicular pathology
- To restore the periodontium to a state of biologic and functional health.

Relative Contraindications

1. Patient's medical status
- Major system disorder – Cardiovascular, Respiratory, 2.

2. Anatomical considerations
- Nasal floor
- Maxillary sinus
- Proximity to neurovascular bundles of mandibular canal and
- mental foramen

3. Practitioner's skill and experience

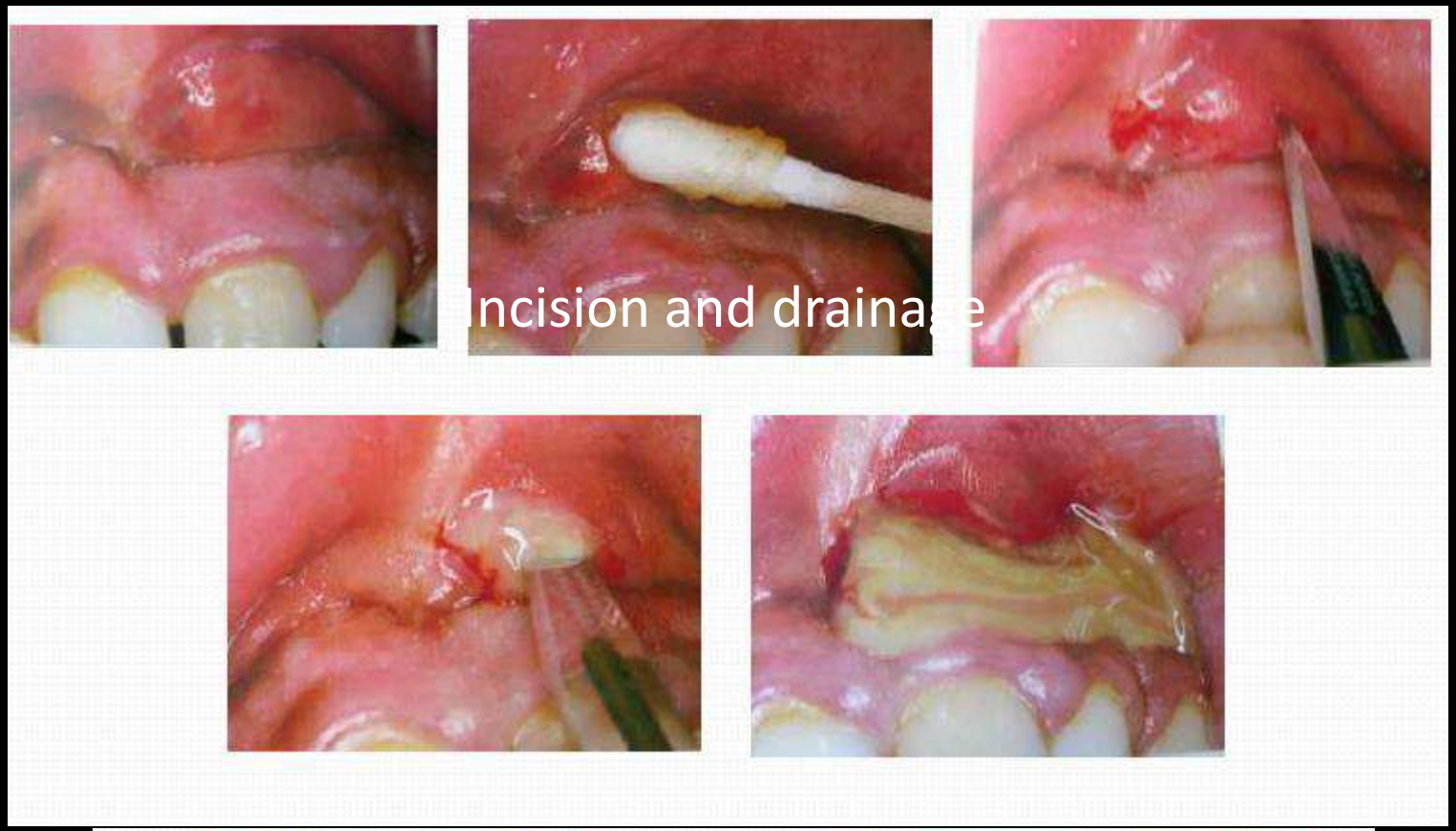
Surgical Drainage

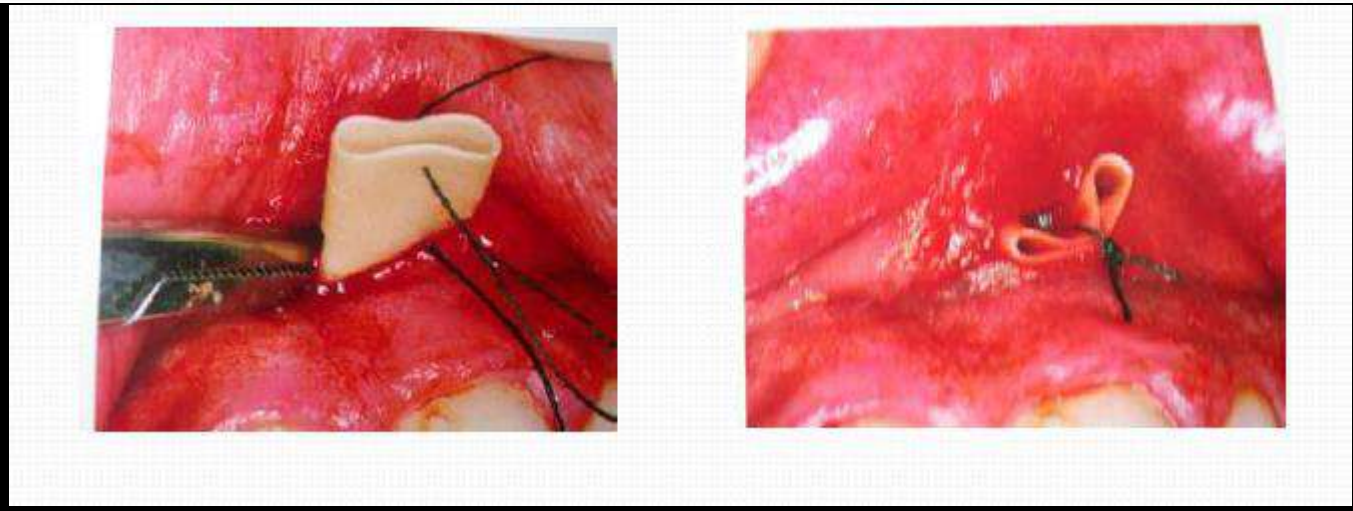
- indicated when purulent and/or hemorrhagic exudates forms within the soft tissue and the alveolar bone; a result of a symptomatic Periradicular abscess.

- Maybe accomplished by :

➢ Incision and drainage (I and D)

➢ cortical Trephination


Incision and drainage



Trephination

- procedure involving the perforation of the cortical plate to accomplish the release of pressure from the accumulation of exudate within the alveolar bone

PERIRADICULAR SURGERY

Periapical (i.e., periradicular) surgery

- Includes resection of a portion of the root that contains undebrided or unobturated (or both) canal space.
- It can also involve reverse filling and sealing of the canal when conventional root canal treatment is not feasible.
- It is often performed in conjunction with apical curettage

Indications

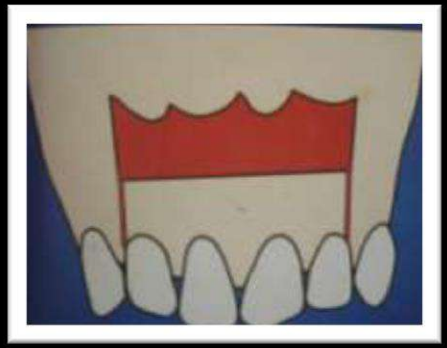
- Anatomic problems preventing complete débridement/obturation
- Restorative considerations that compromise treatment
- Horizontal root fracture with apical necrosis
- Irretrievable material preventing canal treatment or retreatment
- Procedural errors during treatment
- Large periapical lesions that do not resolve with root canal treatment

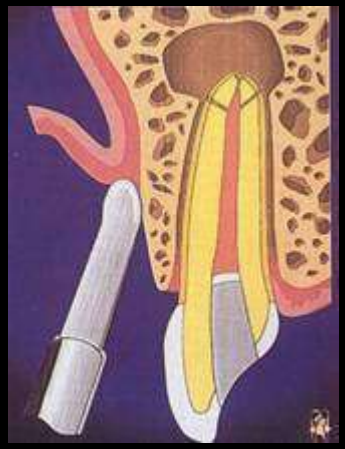
Treatment planning for periradicular surgery

- 1. Presurgical patient management
- 2. Need for profound local anesthesia and hemostasis
- 3. Management of soft tissue
- 4. Management of hard tissues
- 5. Surgical access, both visual and operative
- 6. Access to root structure
- 7. Periradicular curettage
- 8. Root-end resection
- 9. Root end preparation
- 10. Root-end restoration
- 11. Soft-tissue repositioning and suturing
- 12. Postsurgical care

Management of soft tissue (PRINCIPLES OF FLAP DESIGN)

1. Making sure base of the flap should be wider than the free end.
2. Avoiding the incision over a bony defect
3. Including the full extent of the lesion.
4. Avoiding sharp corners
5. Avoiding incision across a bony eminence
6. Avoiding incision in the mucogingival junction.
7. Taking care during retraction.
8. Incision should be made with firm, continuous firm stroke perpendicular to the cortical bone plate.

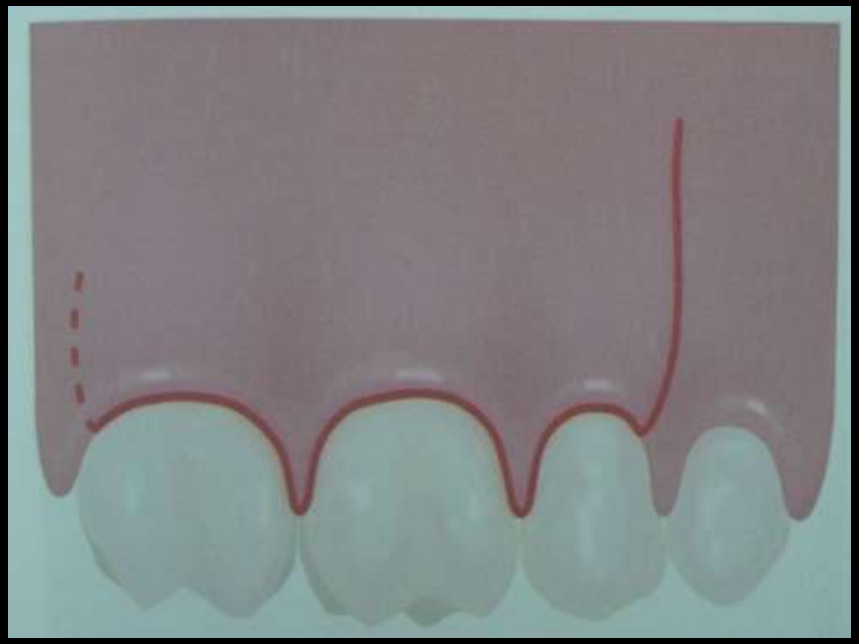


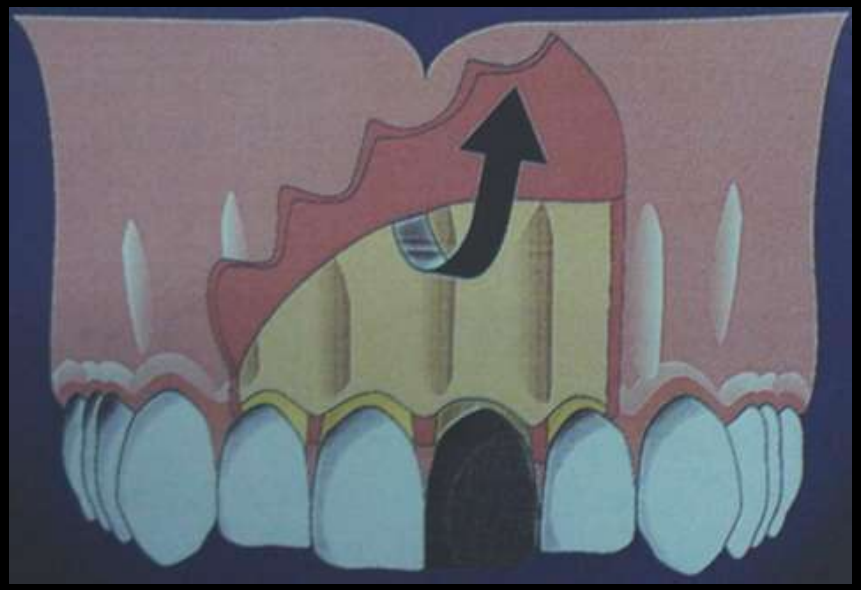


Full mucoperiosteal flap

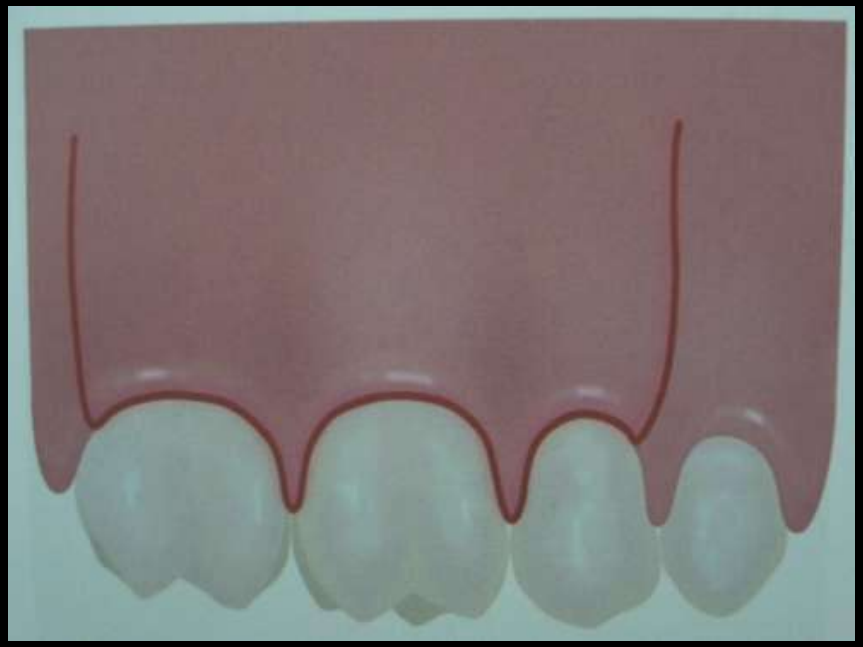
- Triangular
- Rectangular
- Trapezoidal
- Horizontal/Envelope
- SUBMARGINAL CURVED/SEMILUNAR FLAP

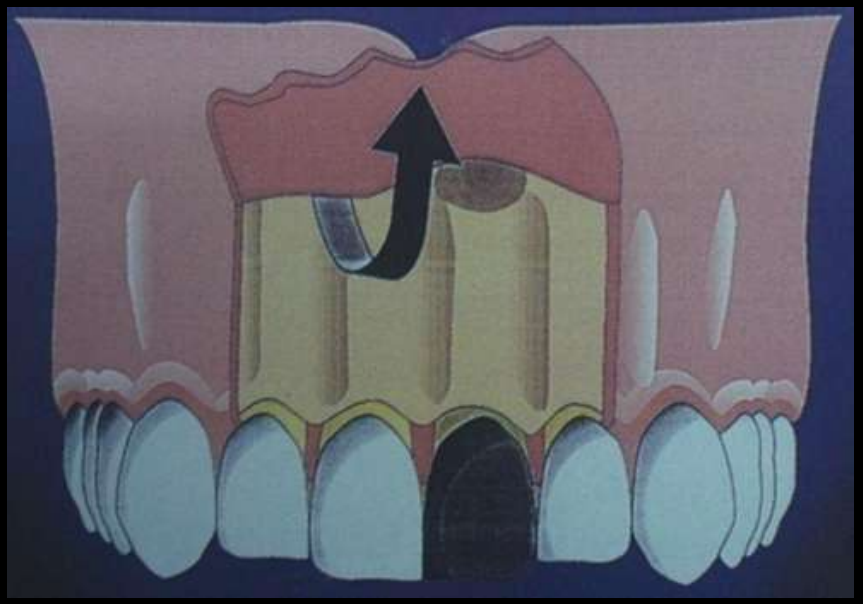
TRIANGULAR FLAP





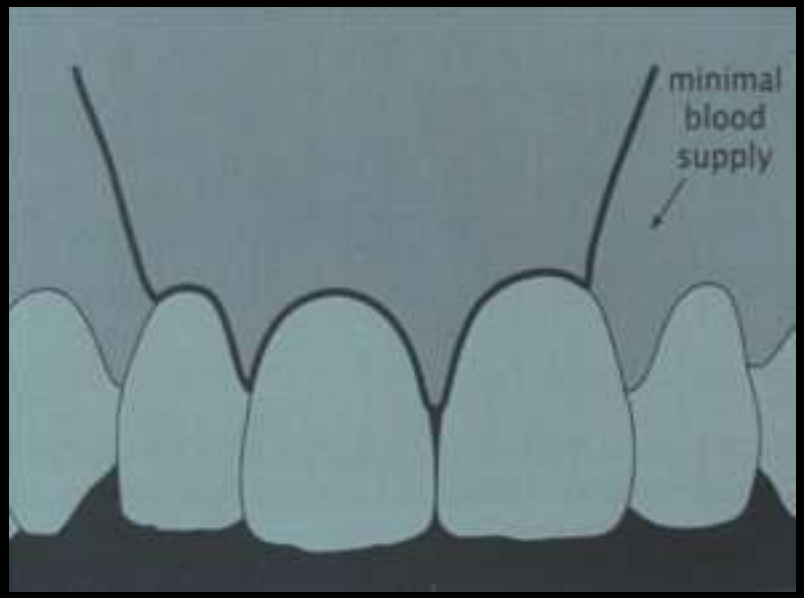
RECTANGULAR FLAP

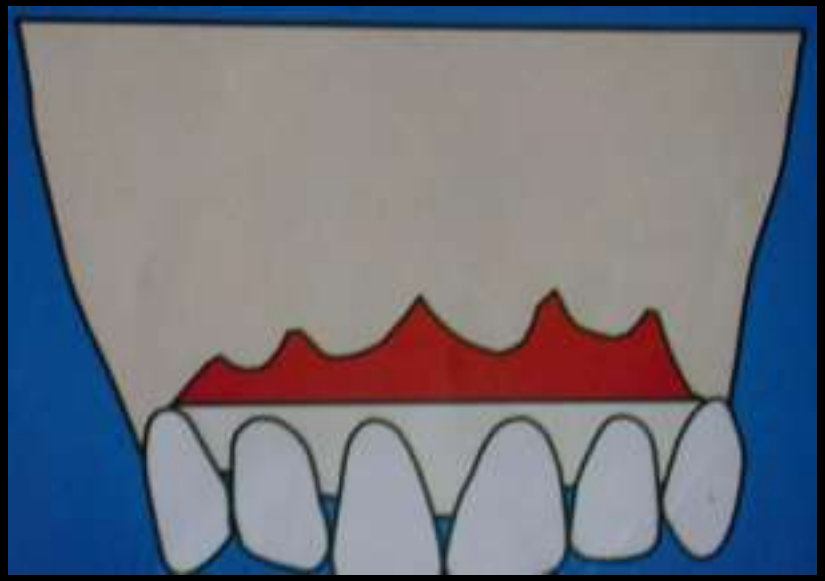




Trapezoidal

horizontal

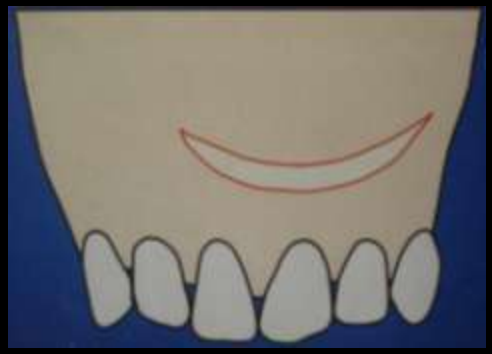




SUBMARGINAL CURVED/SEMILUNAR FLAP

- INDICATION

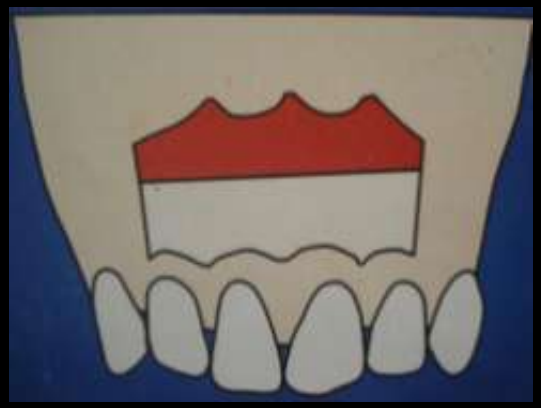
1. Esthetic crowns present
2. Trephination



- ADVANTAGES

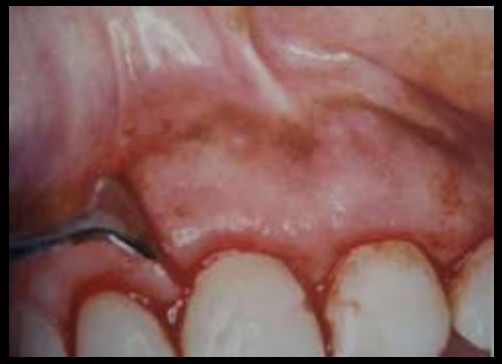
1. Reduces incision and reflection time
2. Maintain integrity of gingival attachment
3. Eliminates potential crestal bone loss

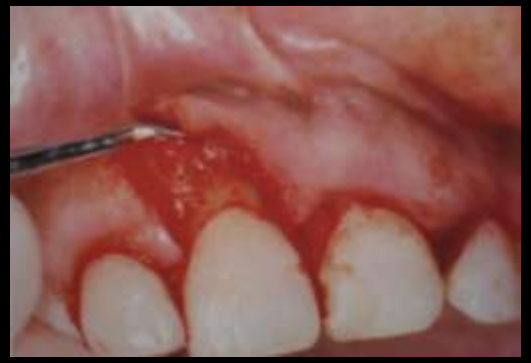
SUBMARGINAL SCALLOPED RECTANGULAR (LUEBKE OCHSENBEIN FLAP)



FLAP REFLECTION

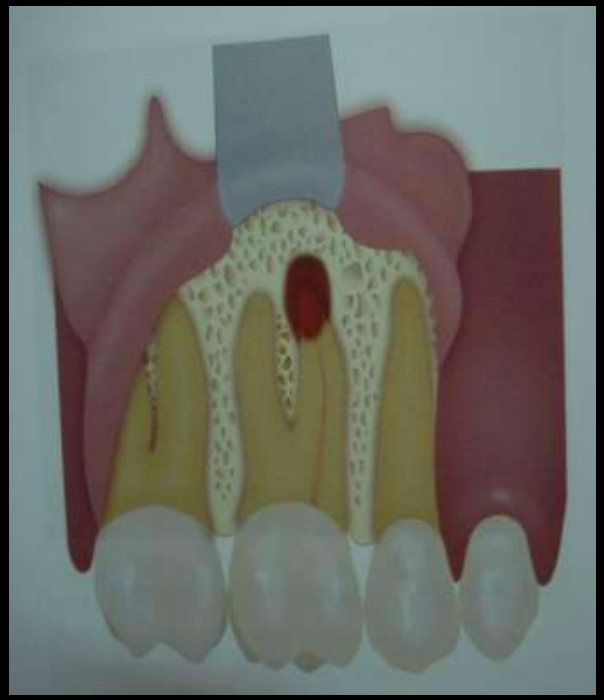
- Flap reflection is the process of separating the soft tissue (Gingivac, Mucosa and Periosteum) from the surface of the alveolar bone
- This process should begin in the vertical incision a few millimeter apical to the junction of the horizontal and vertical incisions.
- Performed Periosteal elevator e.g. Howarth





FLAP RETRACTION

- Process of holding in position the reflected soft tissues



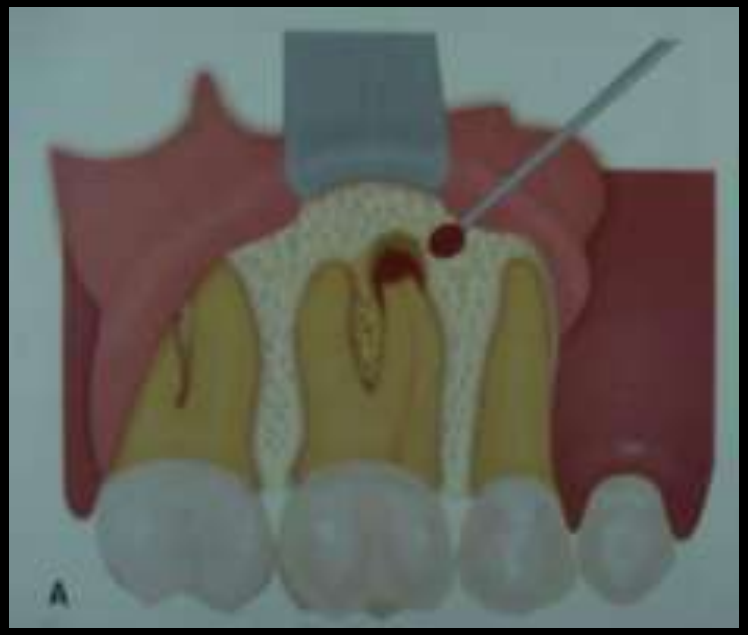
Osteotomy

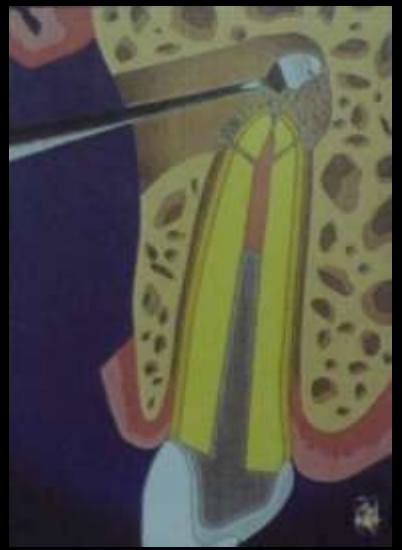
- surgical access must be made through the cortical bone to the roots of the teeth.
- Methods to locate the root apex

1. Visual and tactile method
2. Methylene blue dye

Periradicular curettage

A surgical procedure to remove diseased or reactive tissue from alveolar bone in the periradicular area or lateral region surrounding a pulpless tooth.

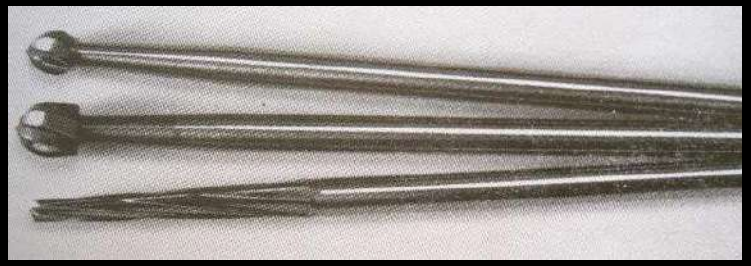




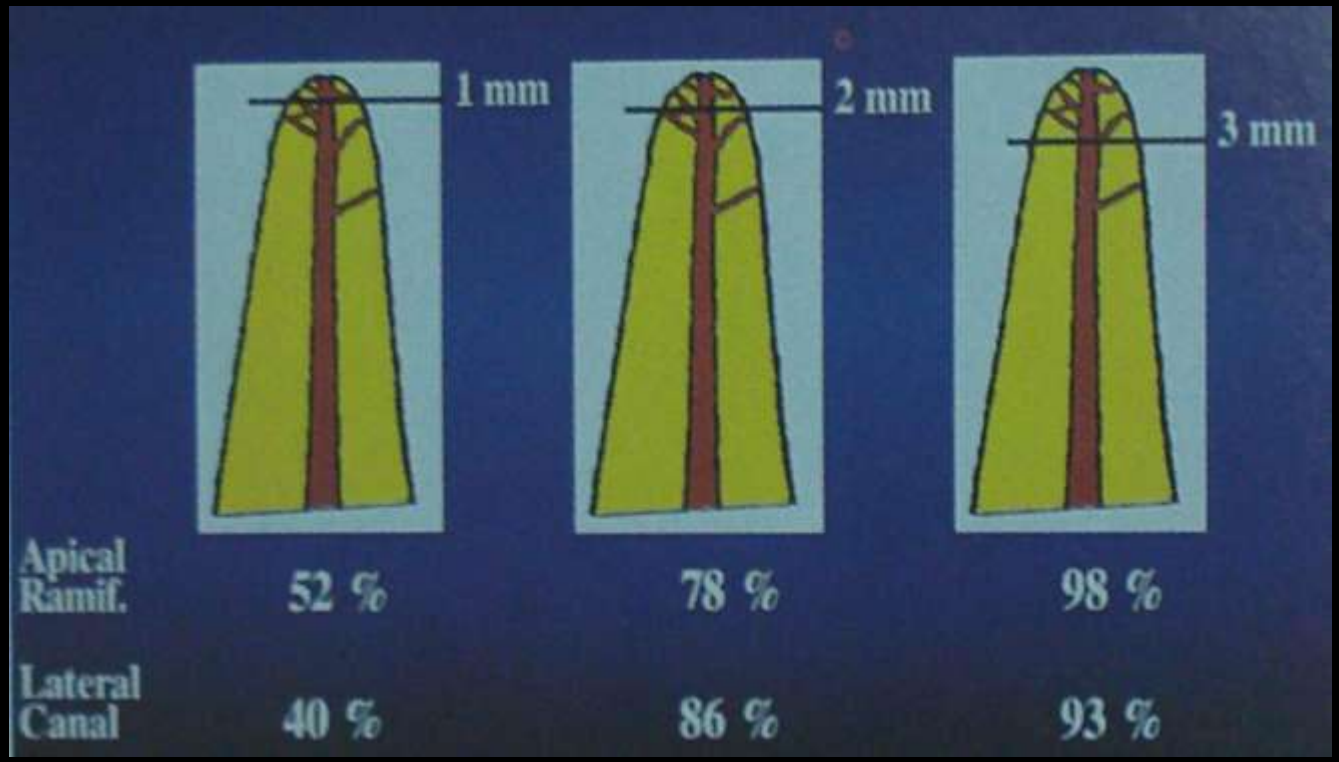
Root End Resection

Indications: Eliminating of

I. Anatomical variations
II. Ledges
III. Canal obstructions
IV. Resorptive defects
V. Perforation defects
VI. Separated instruments

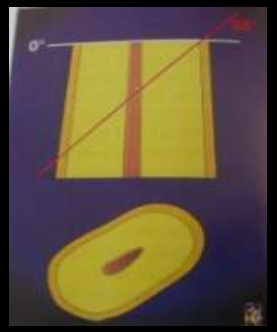


EXTENT OF APICAL RESECTION



BEVEL ANGLE

- Historically : 30-45° to gain visual and operating access to the root tip for resection, placement of retro filling materials, and inspection.



- Present : 90° Maximum= 10° degree bevel

Advantages:

Exposes fewer dentinal tubules, thus preventing excess leakage and contamination

ROOT END PREPARATION

- Purpose:
- To create a cavity to receive a root-end filling.

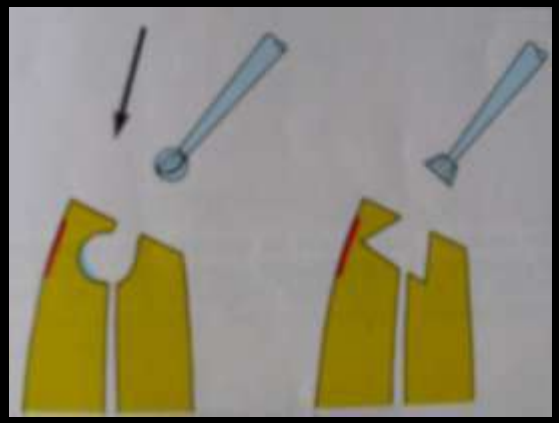
- Objective: It must be placed parallel to the long axis of the root.

- Instruments Used:

I. Small round or inverted cone burs
II. Ultrasonic tips

Traditional root-end cavity preparation technique

- Miniature contra-angle or straight hand piece Small round or inverted cone bur.
- Class I cavity preparation along the long axis of the root within the confines of the root canal.
- Recommended depth - 2 to 3 mm being the most commonly advocated.
- Disadvantage: Apical perforation due to difficulty in aligning the bur



RETROGRADE RESTORATIVE MATERIALS AND TECHNIQUES

- Purpose: To seal the apex so that no bacteria or bacterial by products can enter or leave from the canal
- **Root End filing materials :**
- Gutta percha
- Amalgam
- IRM
- Super EBA
- Glass Ionomer
- Composite resins
- Carboxylate cements
- Zinc phosphate cements
- Zinc oxide eugenol cements
- Mineral trioxide aggregation (MTA)

SOFT TISSUE REPOSITIONING AND COMPRESSION

- The elevated mucoperiosteum gently replaced to its original position with the incision lines approximated as closely as possible.
- Tissue compression: Using a surgical gauze moistened with sterile saline, gently apply firm pressure to the flapped tissue for 2 to 3 minutes (5 minutes for palatal tissue) before suturing

POST OPERATIVE INSTRUCTIONS AND CARE

- A little bleeding from surgical is normal. This should only last for a few hours.
- A little swelling and bruising face may be evident which may last for a few days.
- Do not drink alcohol or use tobacco (smoke or chew) for the next 3 days.
- Have a good, soft diet and drink lots of liquids for the first few days after surgery.

- Place an ice bag (cold) on face where the surgery was done. Leave it on for 20 minutes and take it off for 20 minutes. Continue this for 6 to 8 hours.
- Take the prescribed medicines as recommended.
- Rinse the mouth with 1 tablespoon of the chlorhexidine mouthwash twice daily for 5 days.
- Suture removal after 5-7 days by the dental personnel only.

Corrective surgery

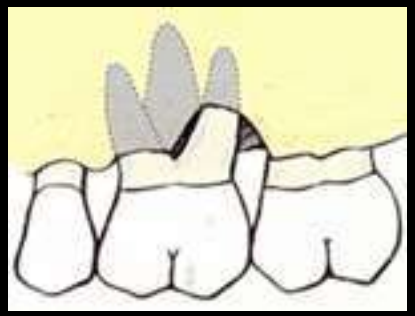
- Corrective surgery is categorized as surgery involving the correction of defects in the body of the root other than the apex.
- Corrective surgical procedure may be necessary as a result of procedural accidents, resorption (internal or external), root caries, root fracture, periodontal disease.

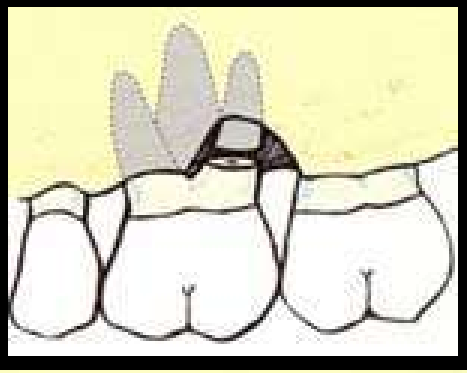
- Corrective surgery may involve

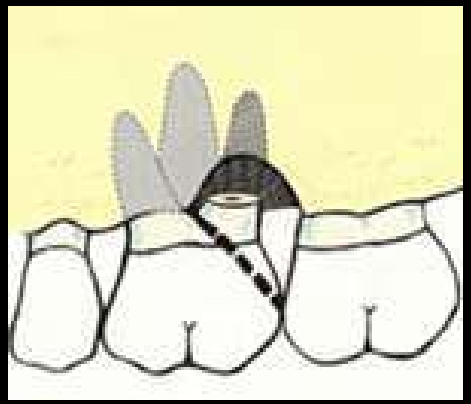
❑ Root resection.
❑ Hemi section.
❑ Intentional replantation.

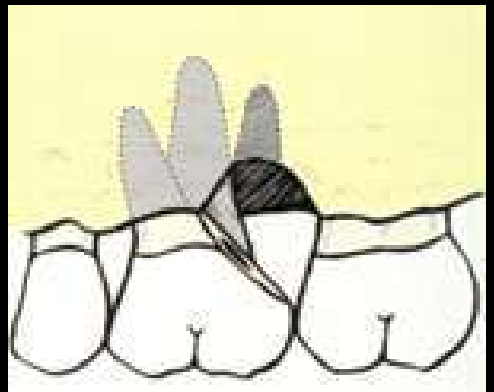
ROOT AMPUTATION

- Root amputation procedures are a logical way to eliminate a weak, diseased root to allow the stronger root(s) to survive when, if retained together, they would collectively fail.



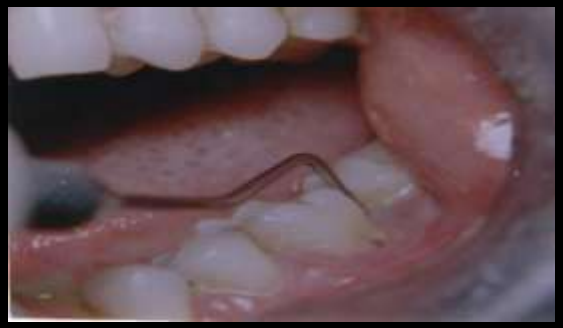


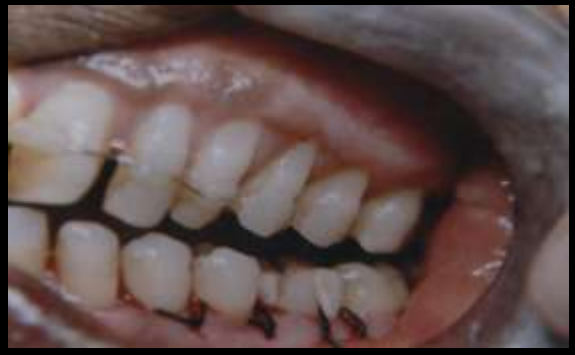




HEMISECTION

- Hemi section is defined as separation of a multi rooted tooth and the removal of a root and the associated portion of the clinical crown.

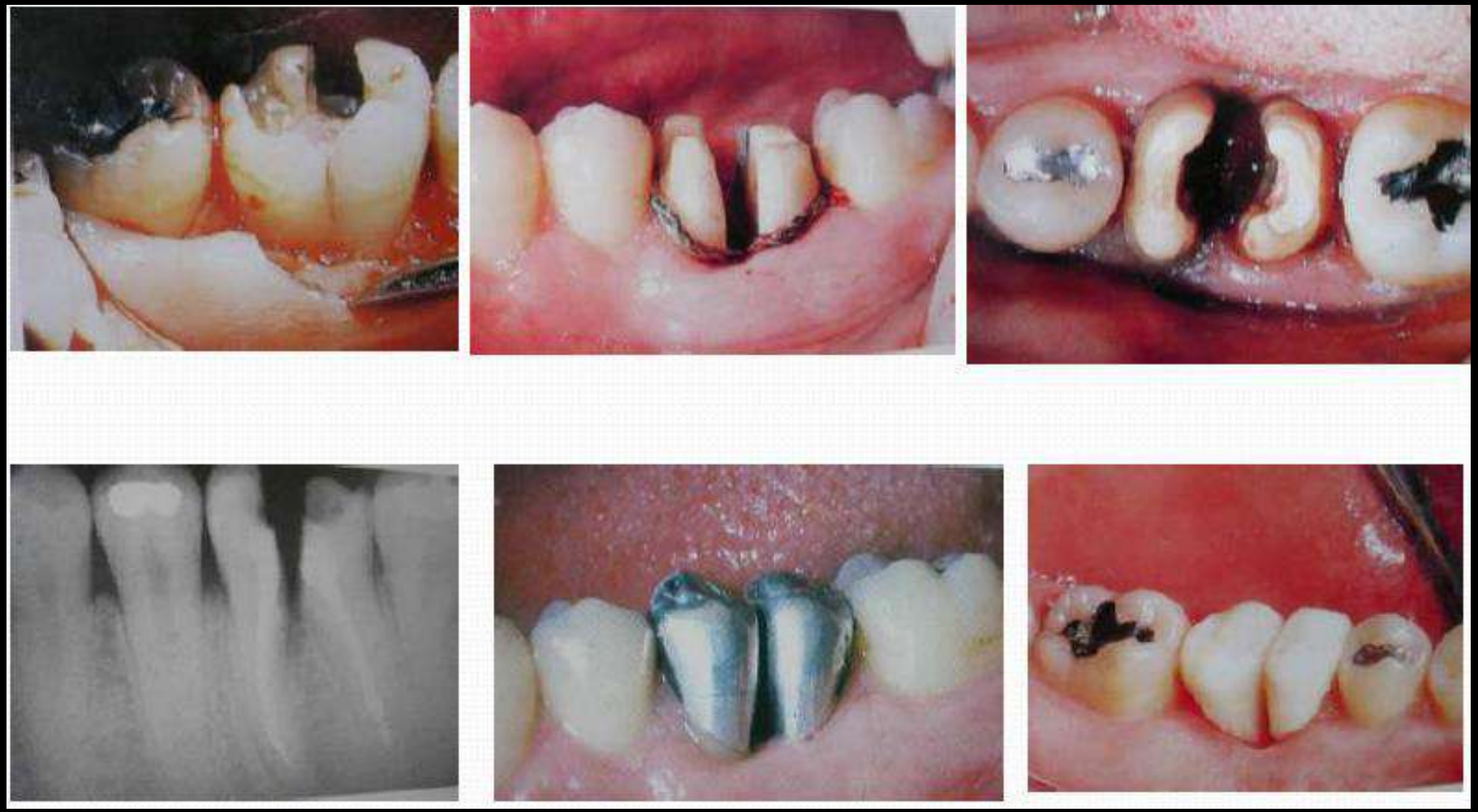




BISECTION OR “BICUSPIDIZATION”

- Refers to a division of a crown that leave the two halves and the respective roots.
- BS should be considered in mandibular molars in which periodontal disease has invaded the bifurcation and repair of internal furcation perforation has been unsuccessful

Bisection



INTENTIONAL REPLANTATION

- Defined as the act of deliberately removing a tooth and following examination, diagnosis, endodontic manipulation and repair returning the tooth into its original socket.

- INDICATIONS
- □ Difficult access
- □ Anatomic limitations
- □ Perforation in areas not accessible surgically.
- □ Failed apical surgery
- □ Apical surgery creating defect
- □ Accidental avulsion(unintentional replantation)

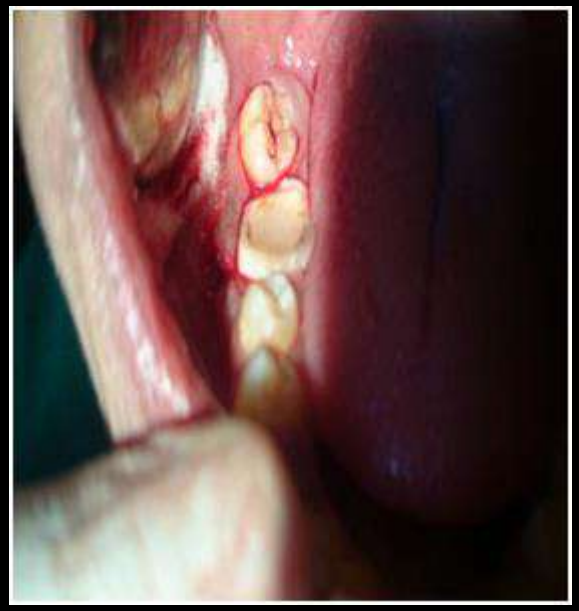
Contraindication

- Pre-existing moderate to severe periodontal disease
- Curved and flared roots
- Non restorable tooth
- Missing interseptal bone

1

2

3



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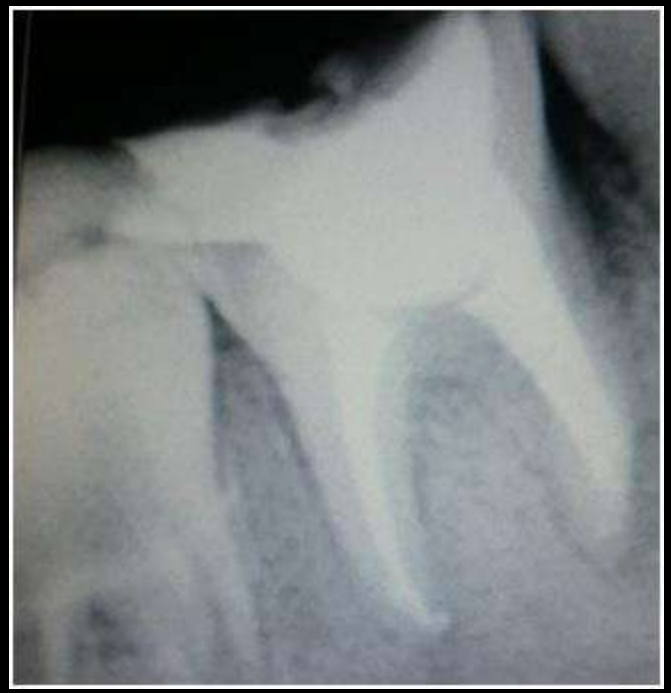



Thank you

Diagnostic imaging in maxillofacial surgery

By

Sabah Alheeti

Diagnostic imaging classified in to :

A-Non invasive imaging *which include*

1- plain radiographic (conventional)

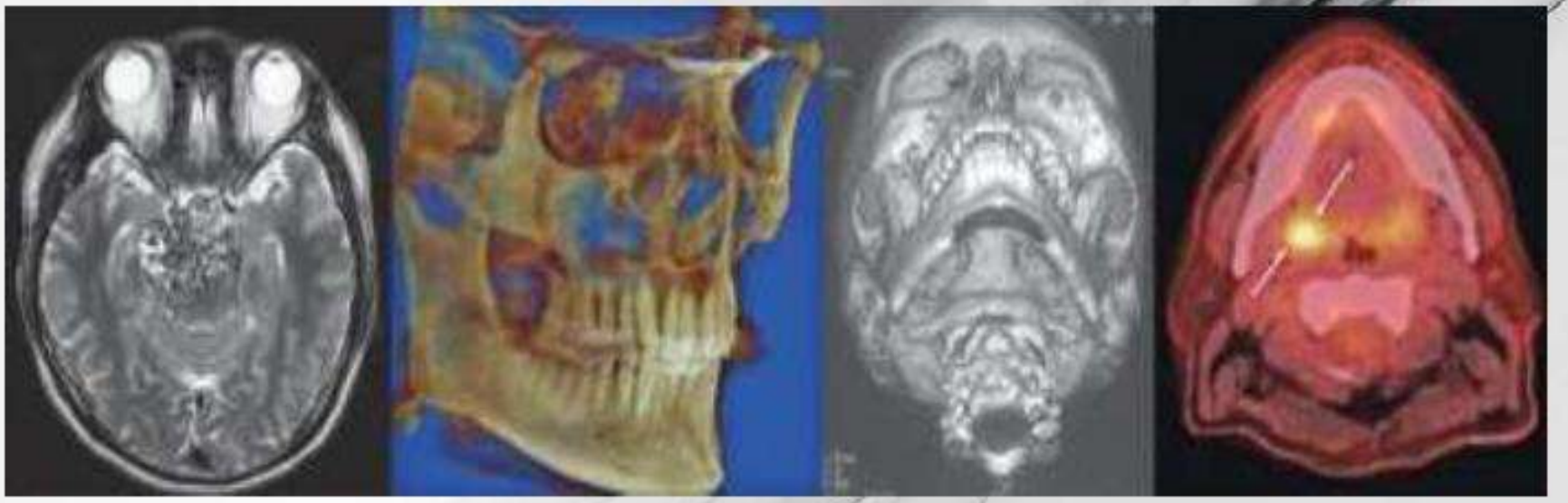
2-Computed tomography (CT)

3-Magnetic resonance imaging (MRI)

4-Contrast enhanced imaging (sialography ,arthrography)

5-Ultrasonography

6- Nuclear Imaging (SPECT, PET …)



B-Invasive imaging:

This is performed for diagnostic as well as therapeutic purpose

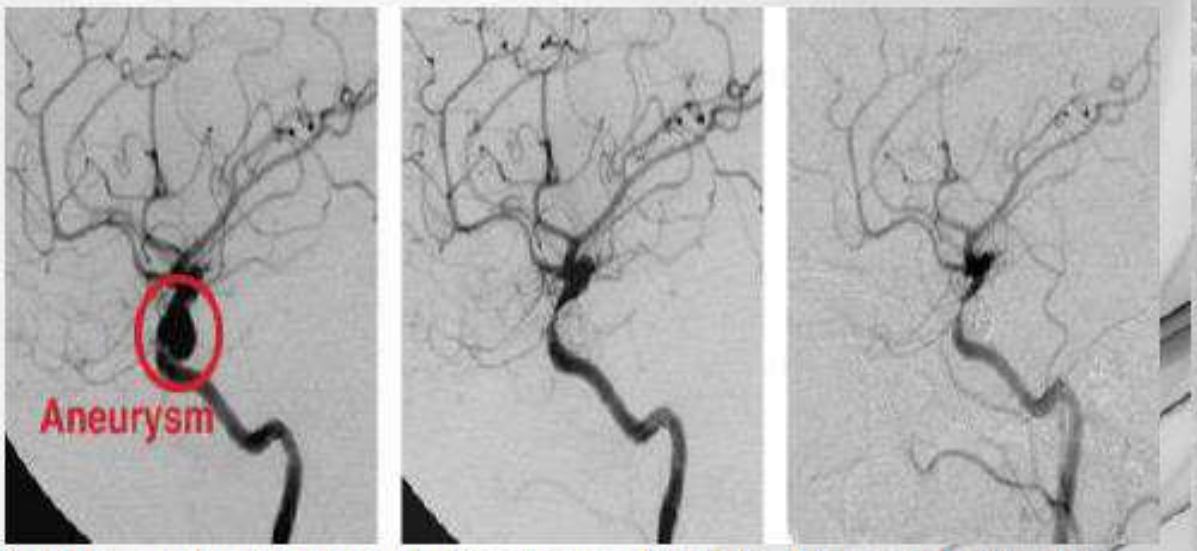
This include :

-Angiography

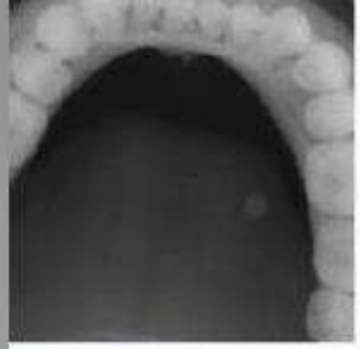
-Angioplasty

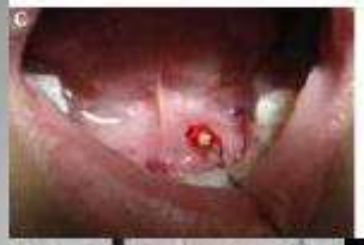
-Embolization

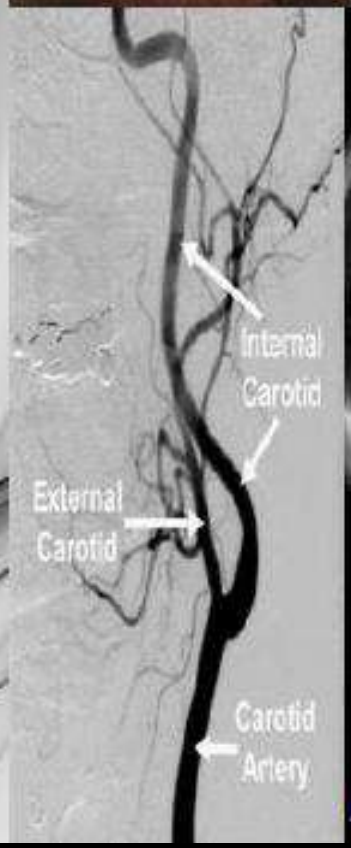
-Calculus destruction ,others…

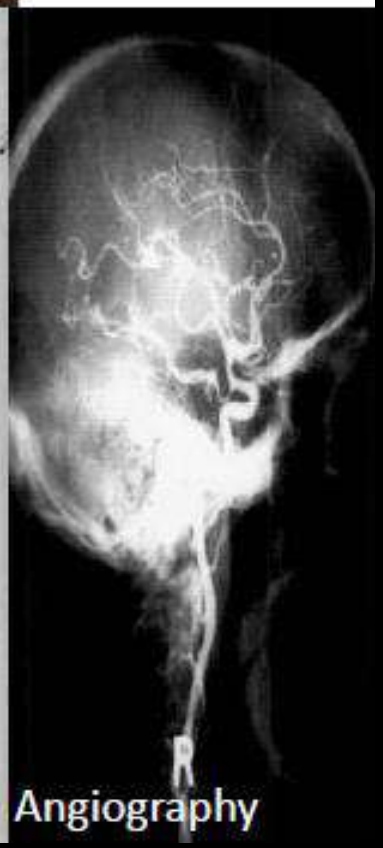


Before embolization (left); immediately after embolization (middle); and, 12 months following embolization (right).









Angiography

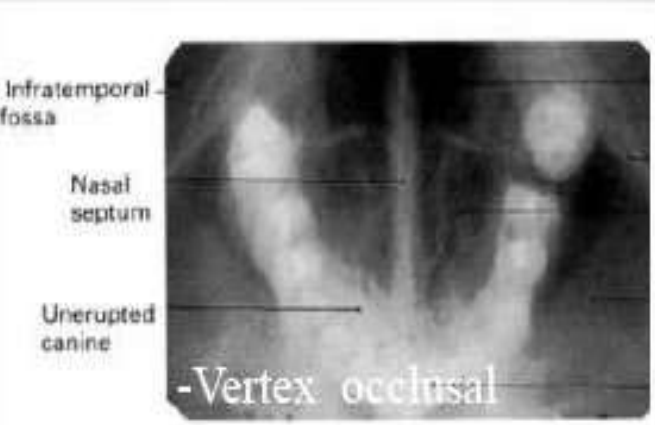
Plain Radiograph

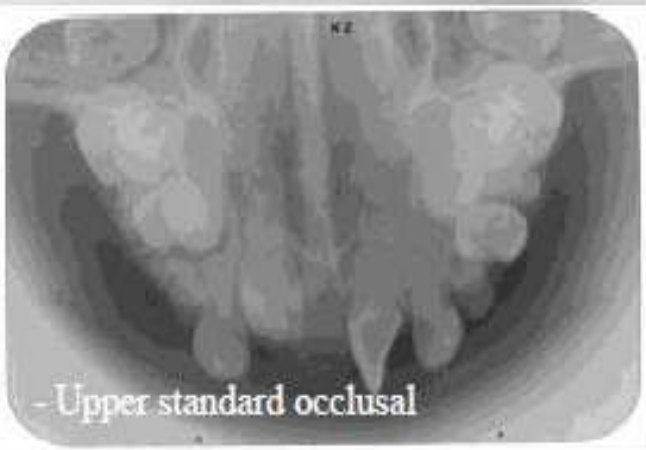
A-intra-oral radiograph :

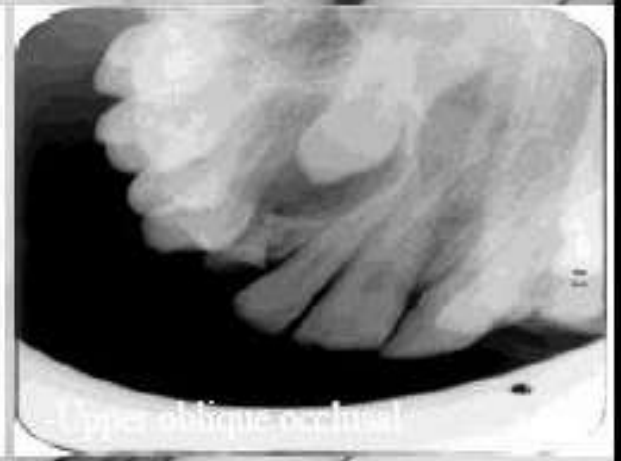
A-Periapical

B-Bite-wing

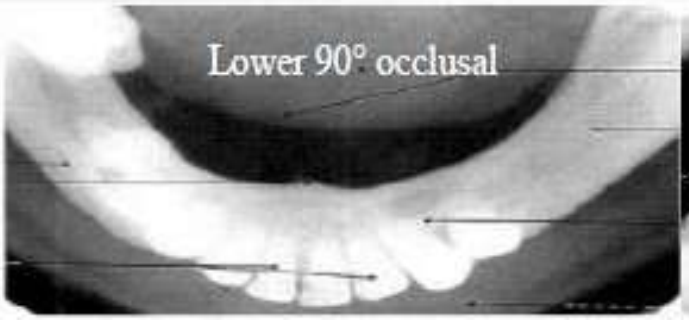
C-Occlusal include : >>Maxilla (occlusal view)

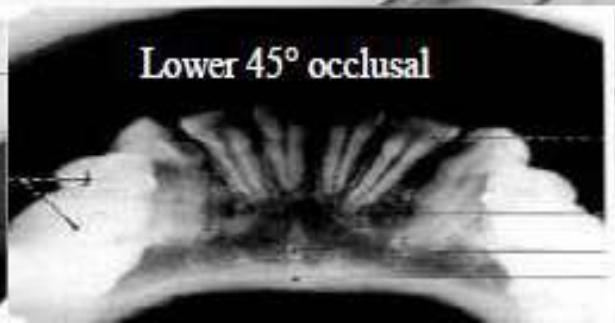


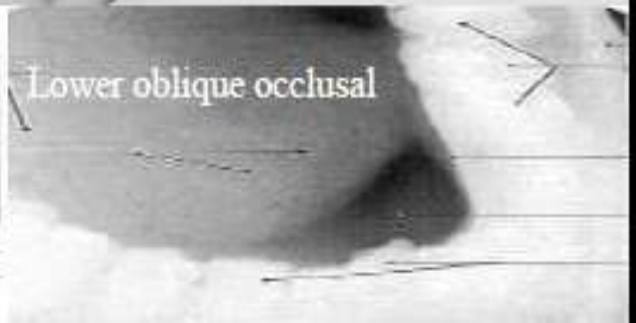




-Mandible (occlusal view)







Extraoral radiographs

- occipitomental (0°, 30° OM)
- Postero-anterior of the skull (PA skull)
- OPG
- Submento-vertex (SMV)
- Lateral skull view

Role of Plain Radiograph

- Trauma

1) Detect site and extension of fractures
2) Locating the foreign bodies

- Oncology
- Craniofacial anomalies

Oncology

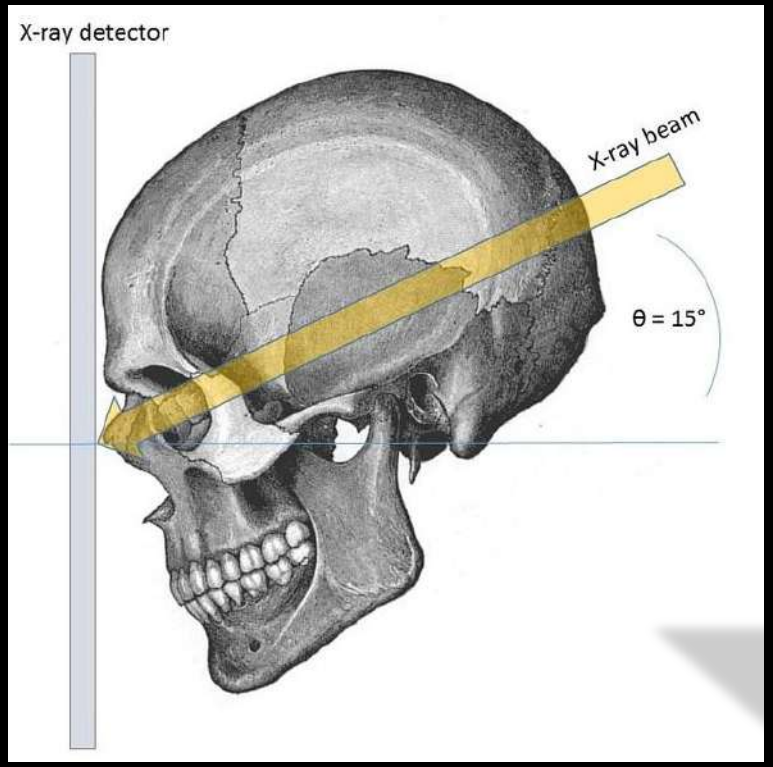
- Offer little in assessment of tumor extension.
- OPG is generally needed routinely
- Evaluation of :

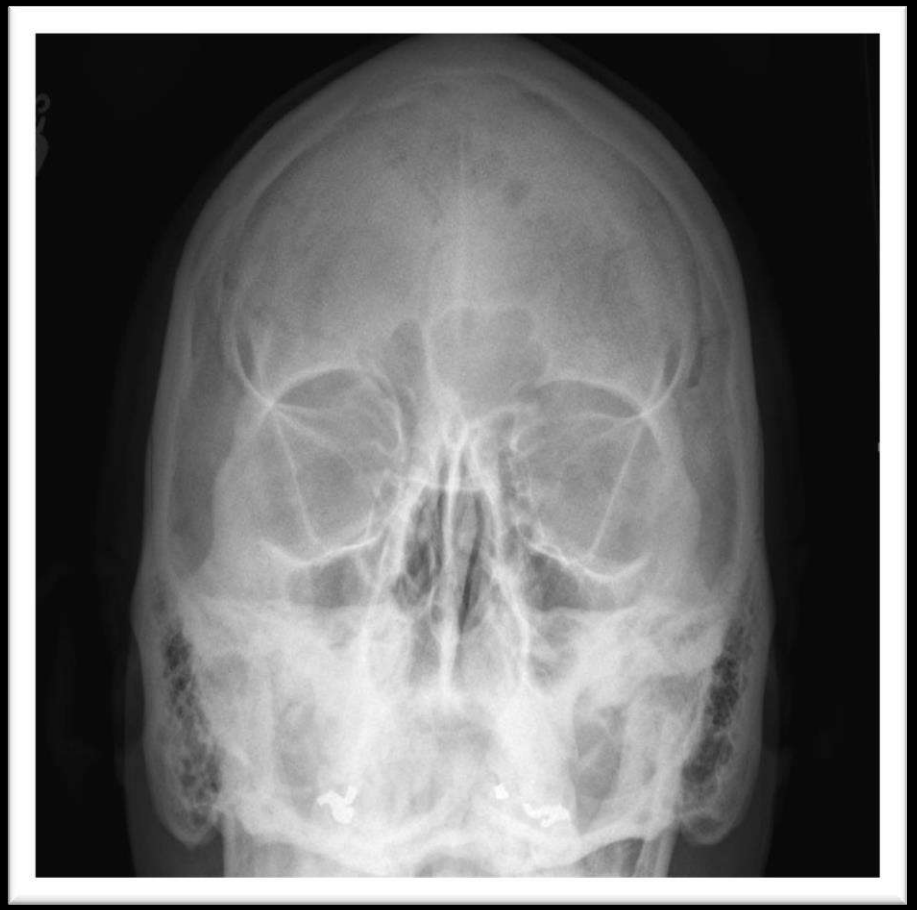
1) Intraosseous pathology
2) Demonstrate bone erosion or destruction
3) Assesment of dental diseases in patient who undergone surgery or RRx
4) CXR to look for distant metastasis

Plain radiograph projections

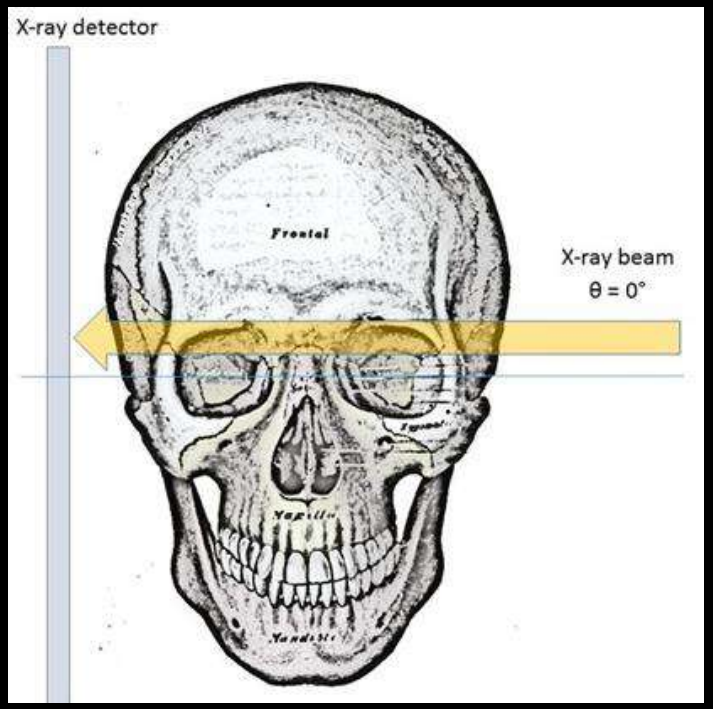
Upper third projections

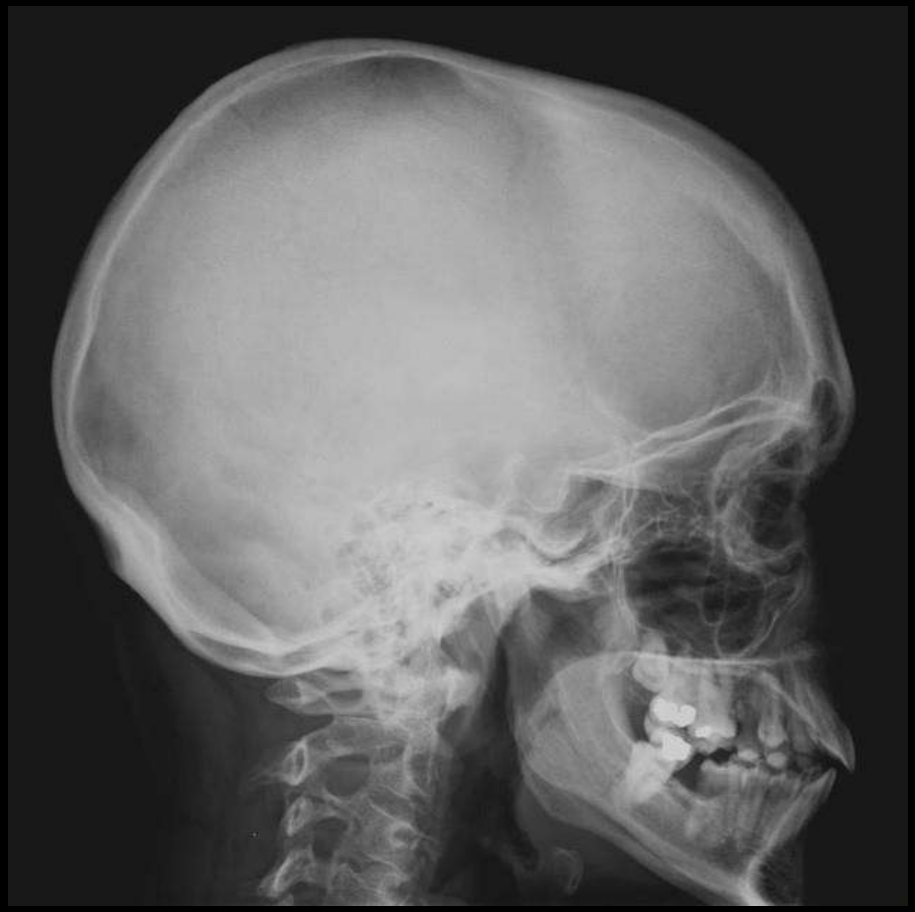
Caldwell view





Skull (lateral view)

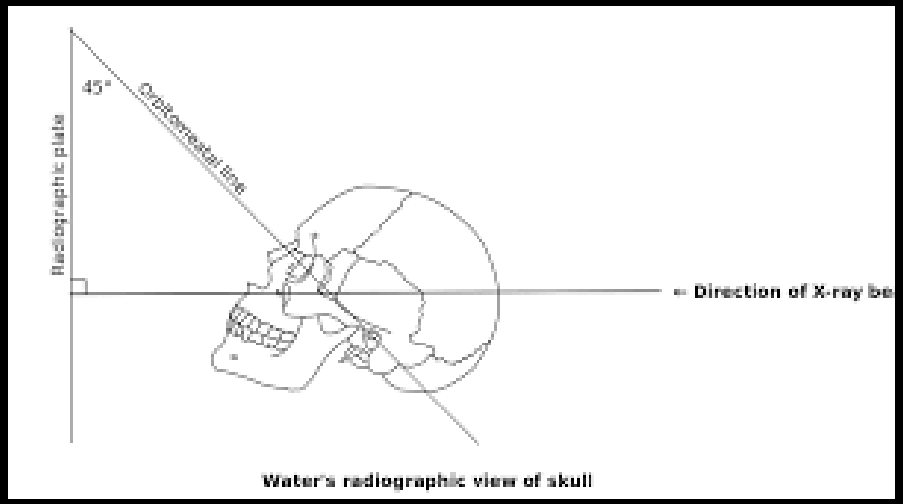




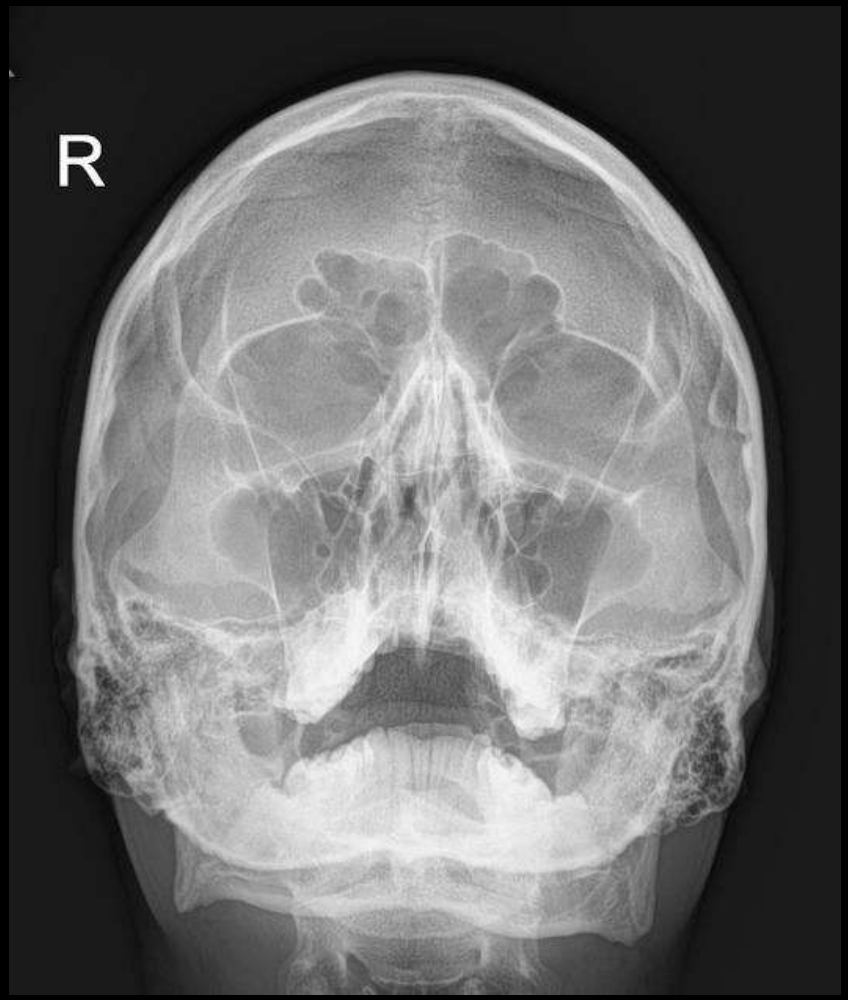
Middle third projections

- Occipitomental view (Water's view)
- Submentovertex
- Lateral view
- Upper occlusal projection: detect midline split palate

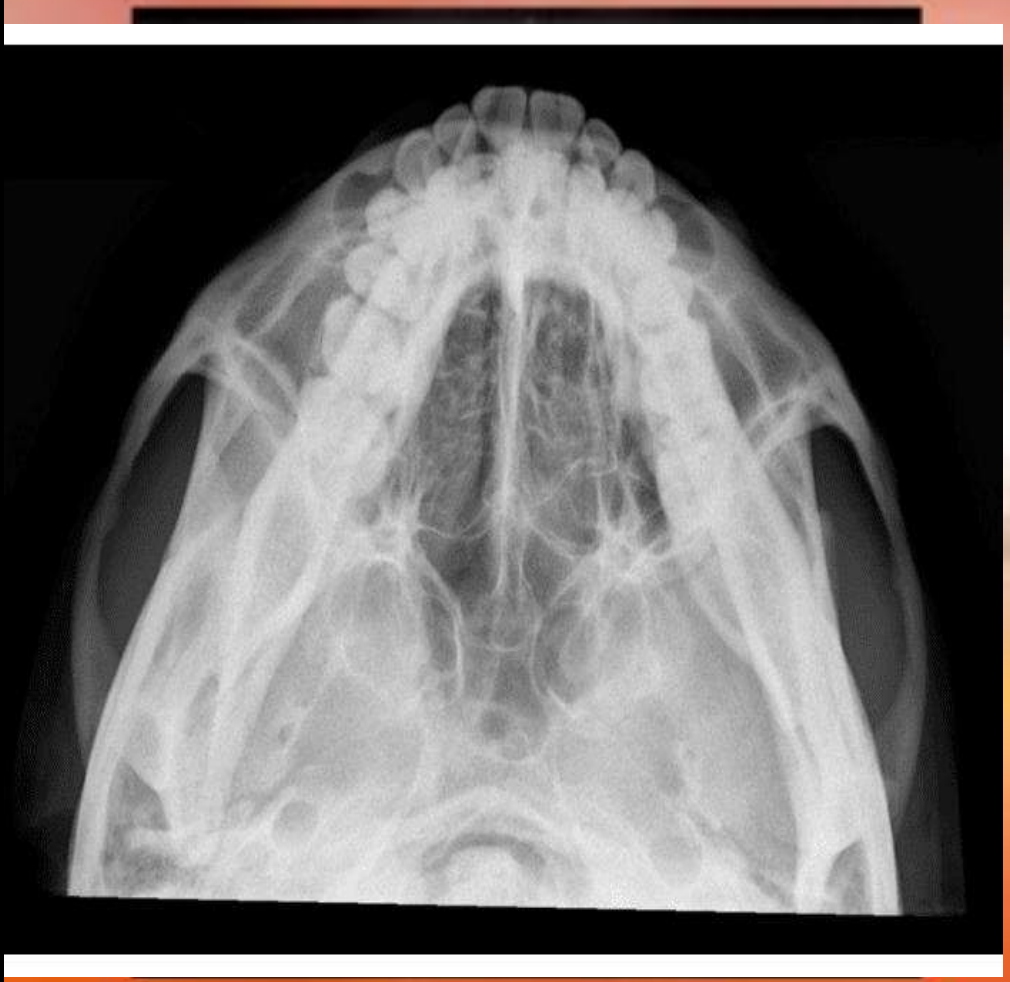
Waters view
(OM 37°)



Water's radiographic view of skull



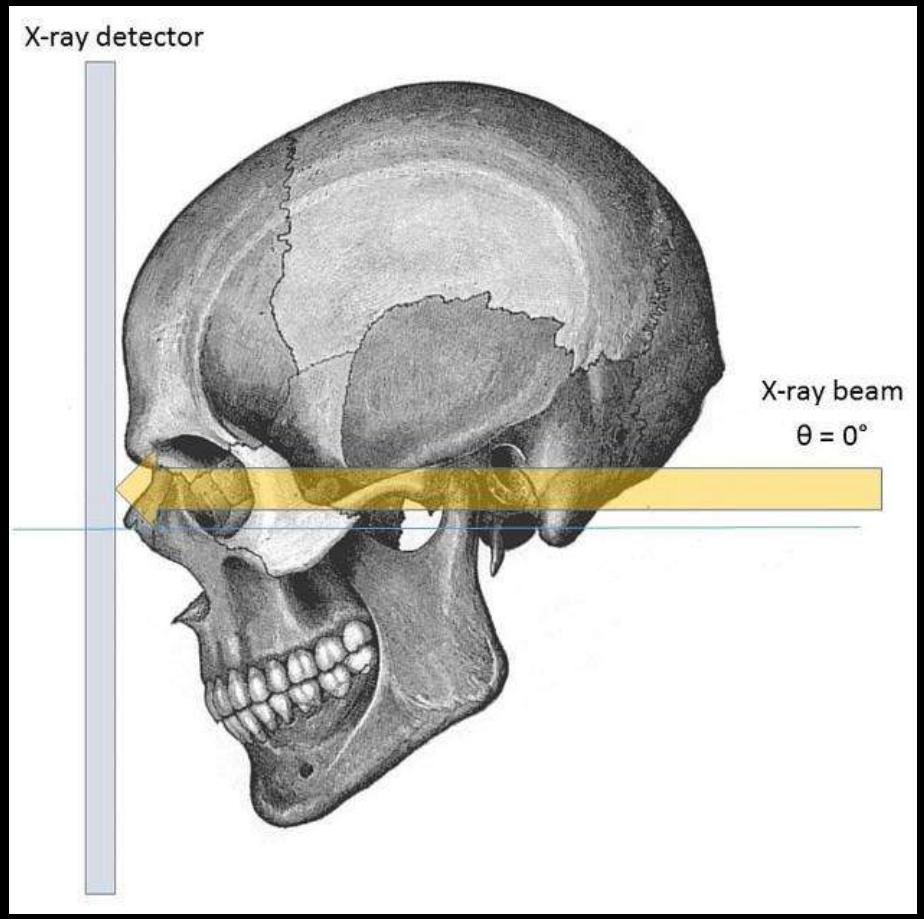
4-Submentovertex (SMV) view:

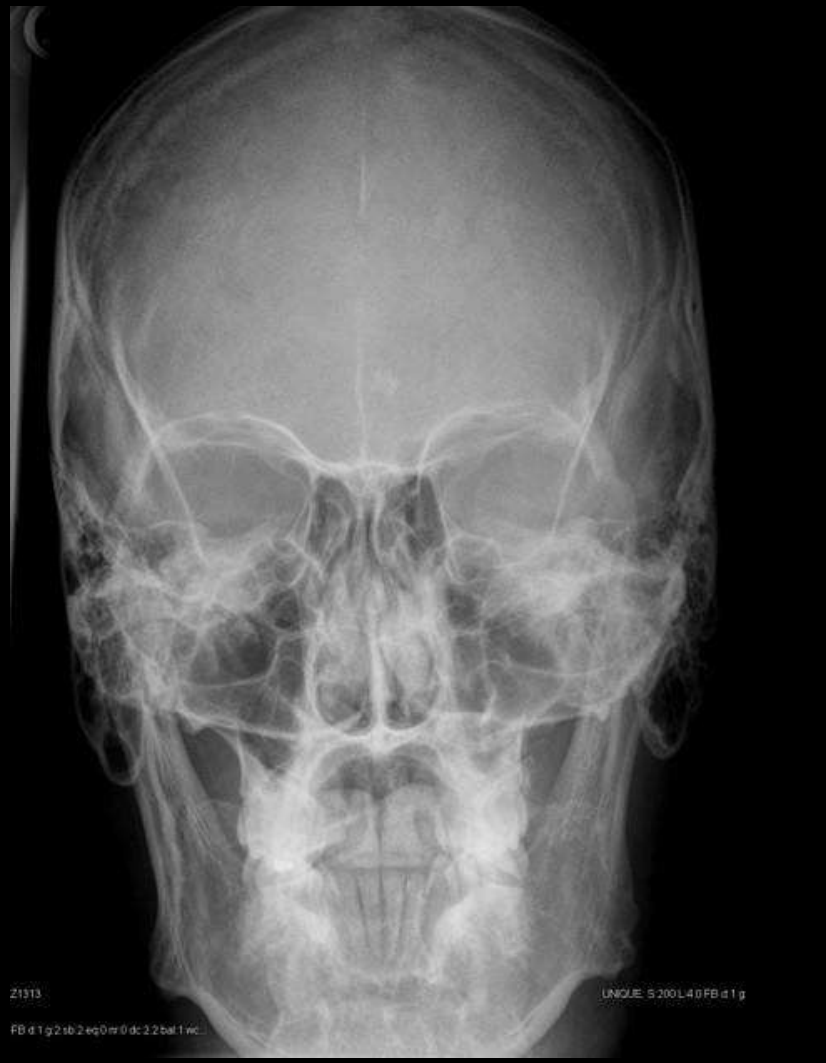


Lower third projections

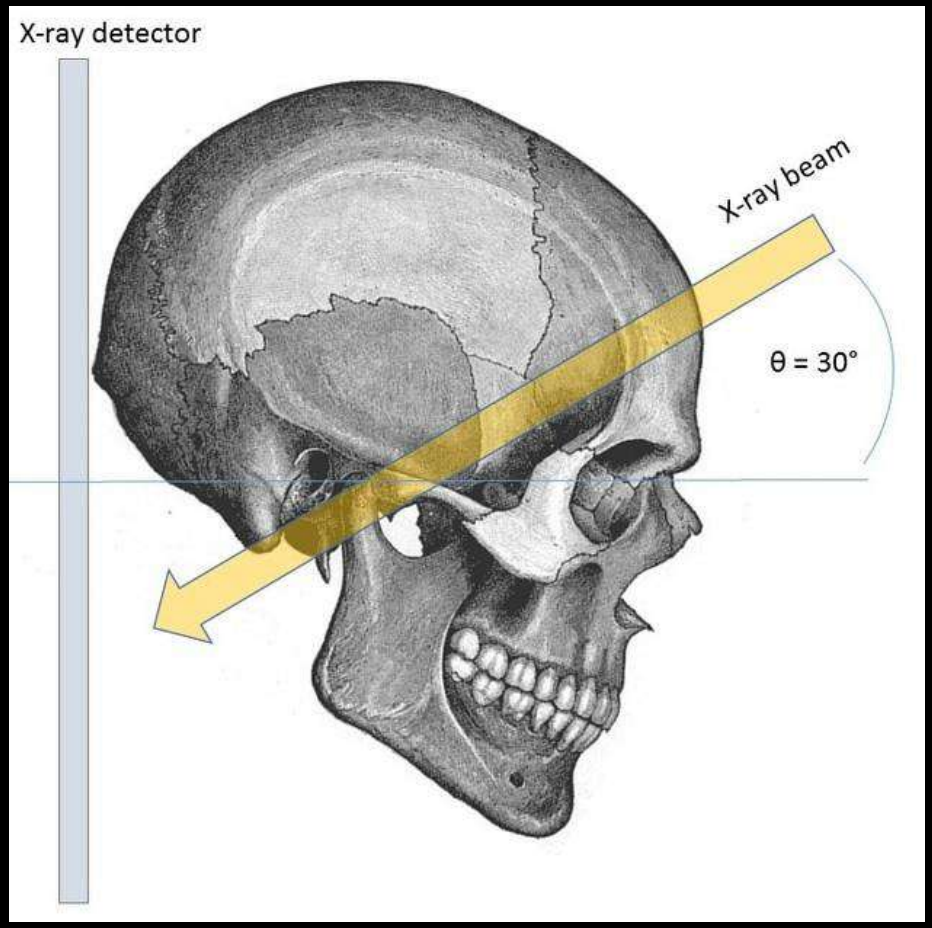
- Posterio-anterior view (PA)
- Townes view
- OPG
- Tomograph
- Others : lateral oblique, Cephalometry..

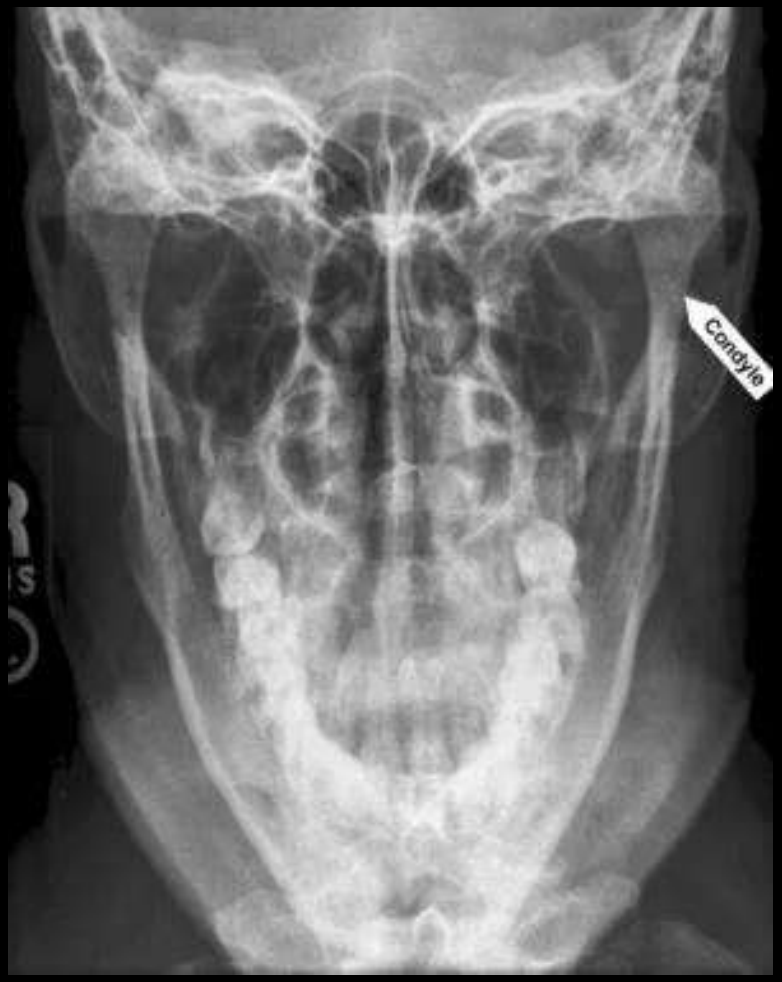
Skull (PA view)



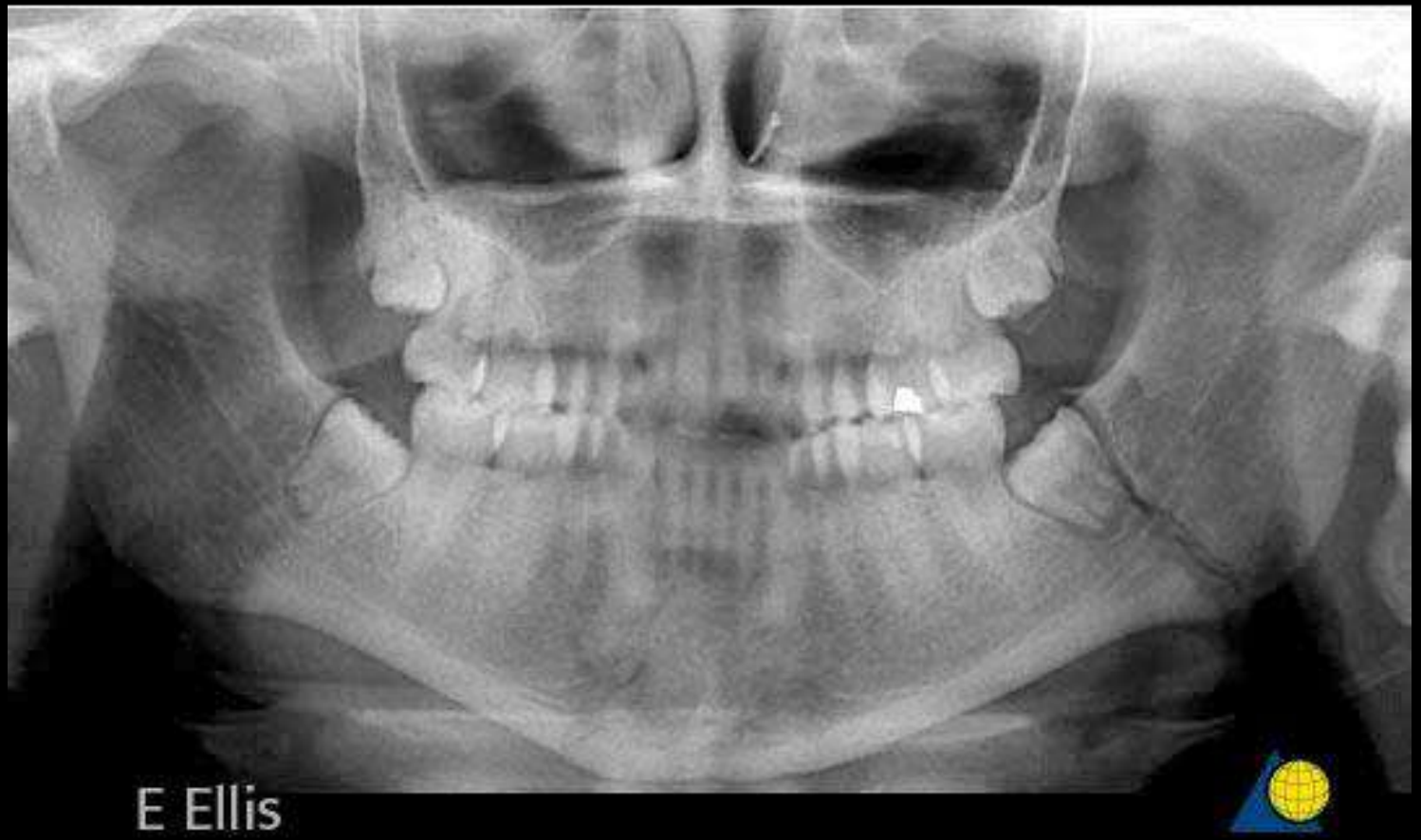


Towne view





OPG



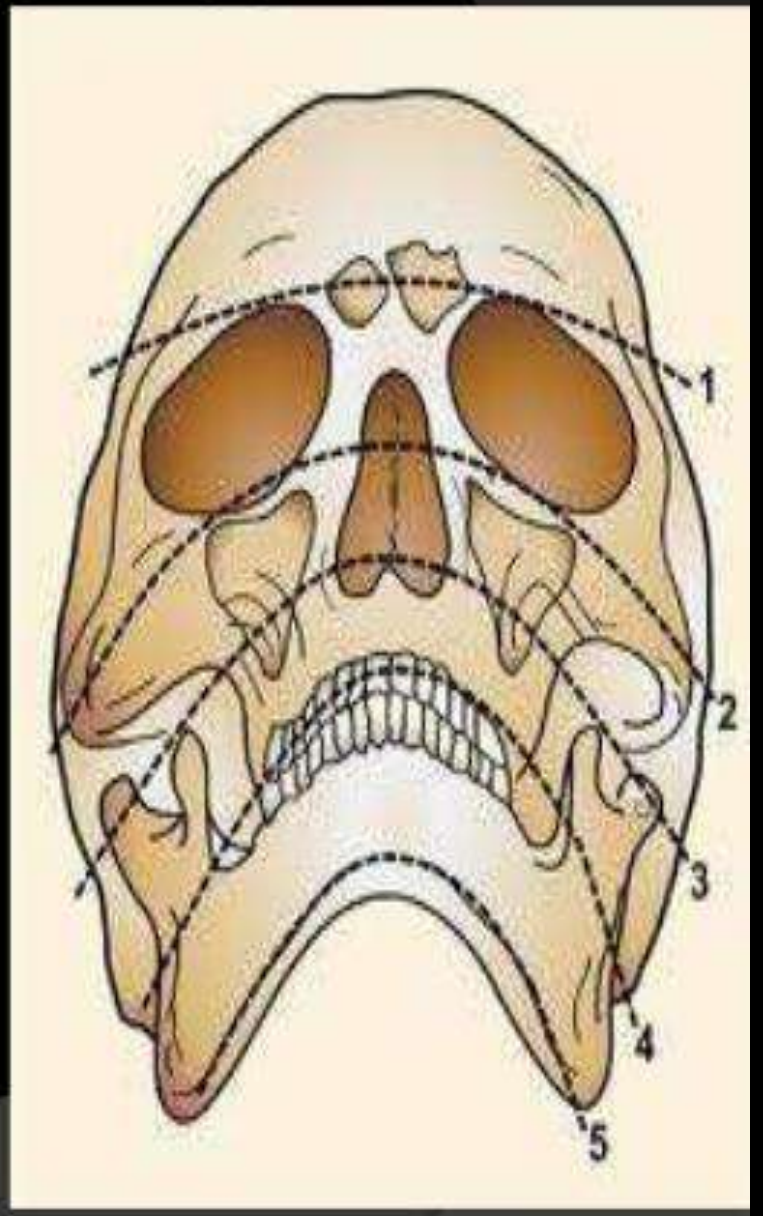
Radiographical interpretations

- There is methodical approach to searching the image for information.
- Campbell's and Trapnell's lines is useful guide in occipitomental view.

- Looking for hot sites where fractures most likely occur.

Campbell's and trapnell's lines

- 1- First line across the zygomaticofrontal, the superior margin of the orbit and the frontal sinus
- 2- Second line across the zygomatic arch, zygomatic body, inferior orbital margin and nasal bone
- 3- Third line across the condyles, coronoid process and the maxillary sinus
- 4- Fourth line across the mandibular ramus, occlusal plane
- 5- Fifth line (trapnell's line) across the inferior border of the mandible from angle to angle

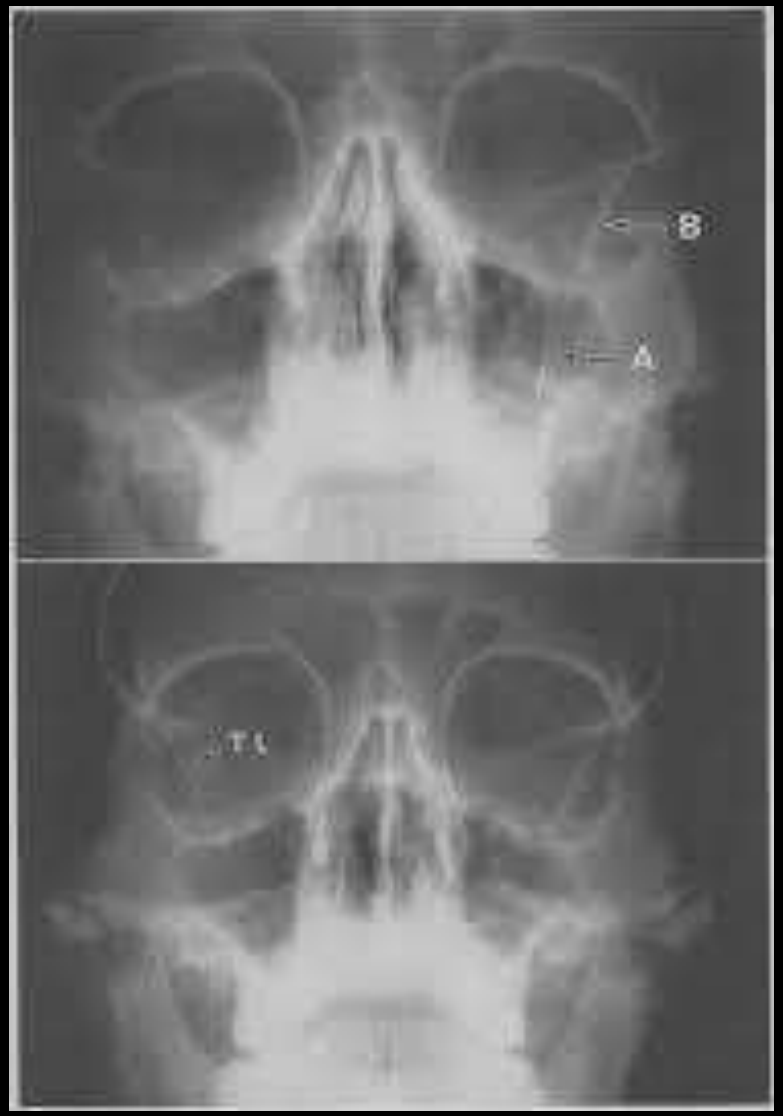


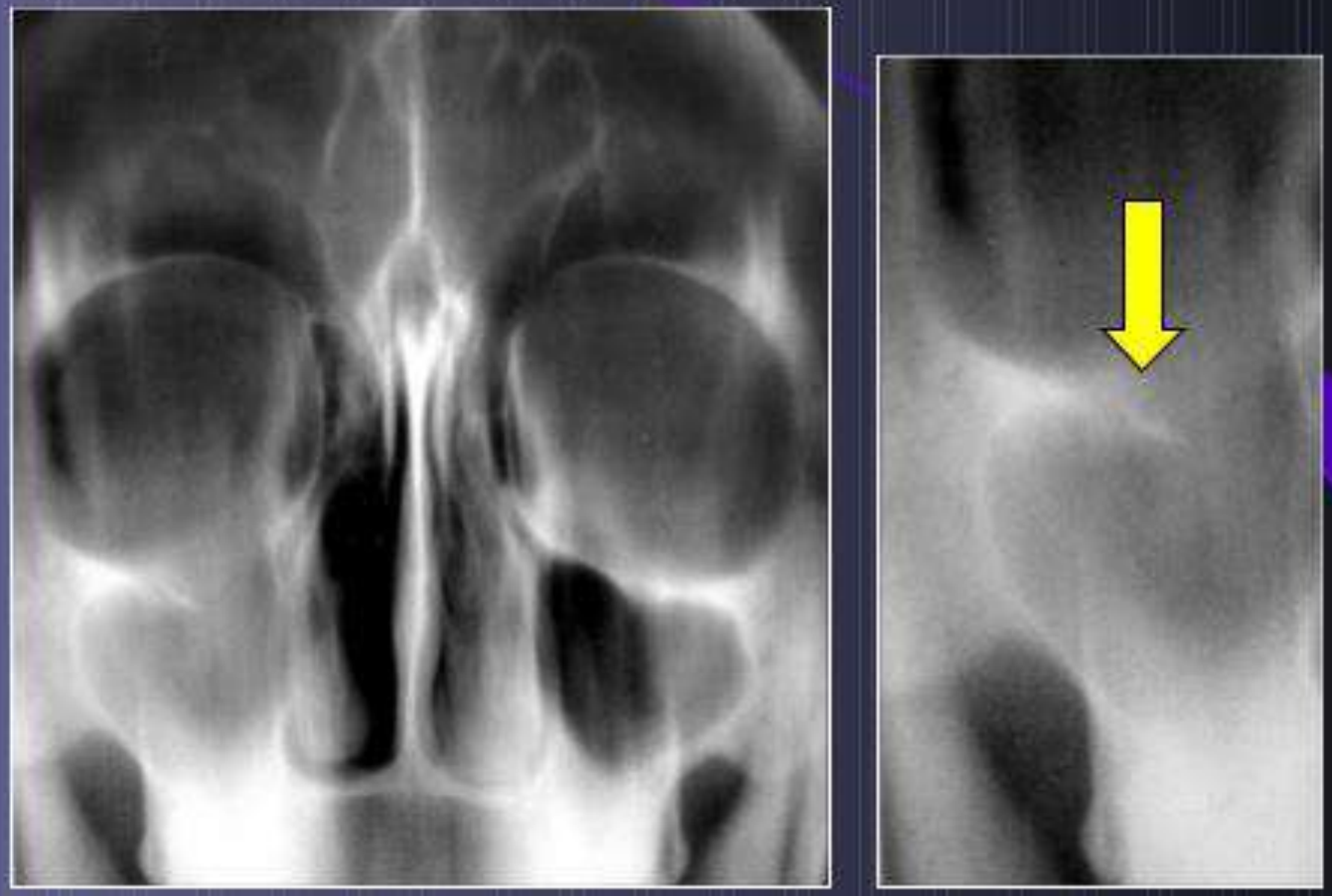
Radiographical interpretations of skull

- The ‘’4S’’ are good reminder of what to be looking for on radiographs.

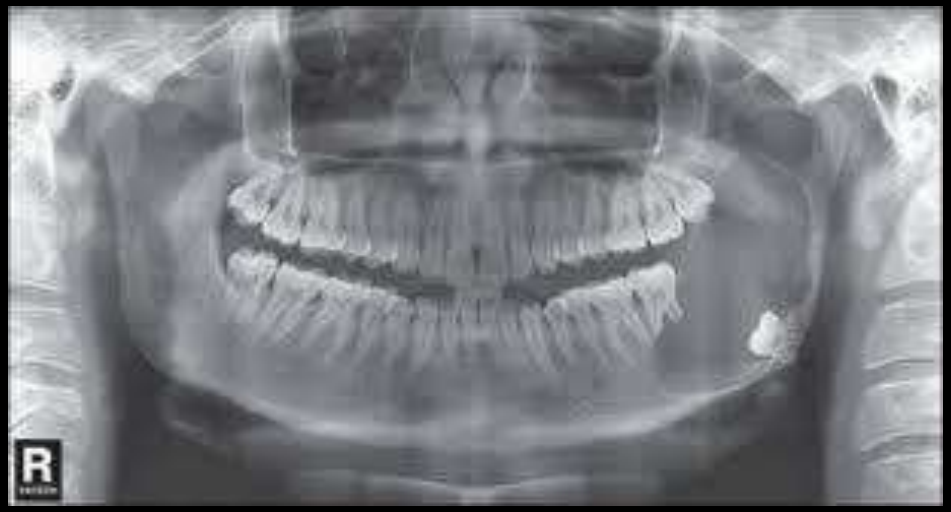
I. Symmetry
II. Sharpness: bright sign, trapdoor sign
III. Sinus
IV. Soft tissue

Bright sign





Blow-out fracture of right orbit shows typical "trap door" sign. There is opacification of the affected sinus (coronal tomograph).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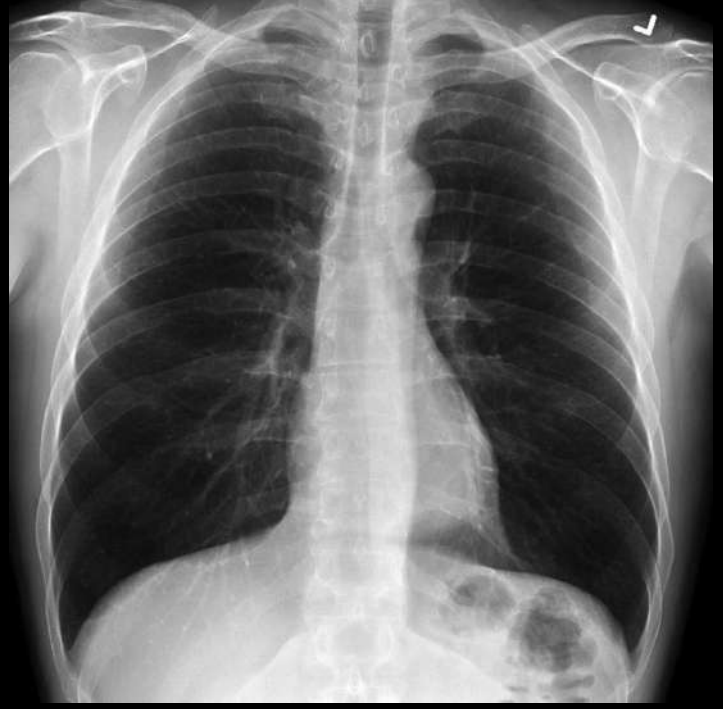
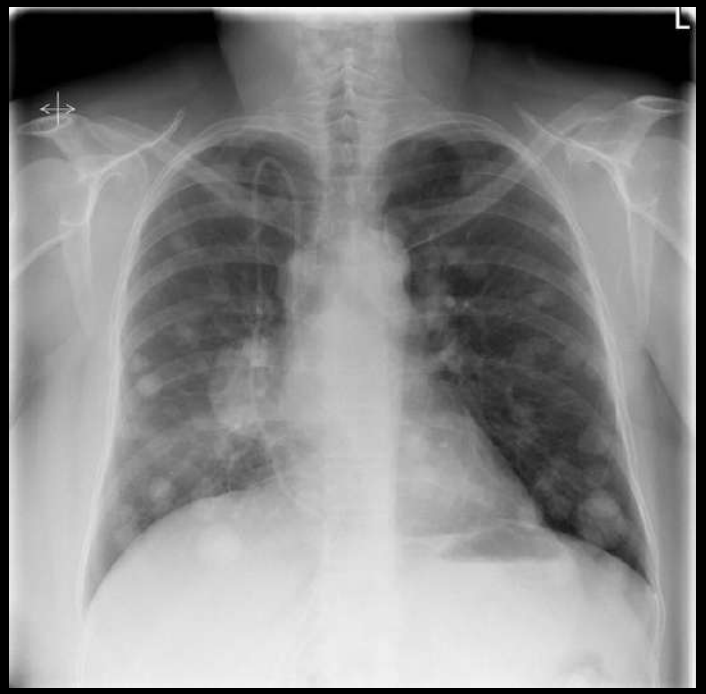


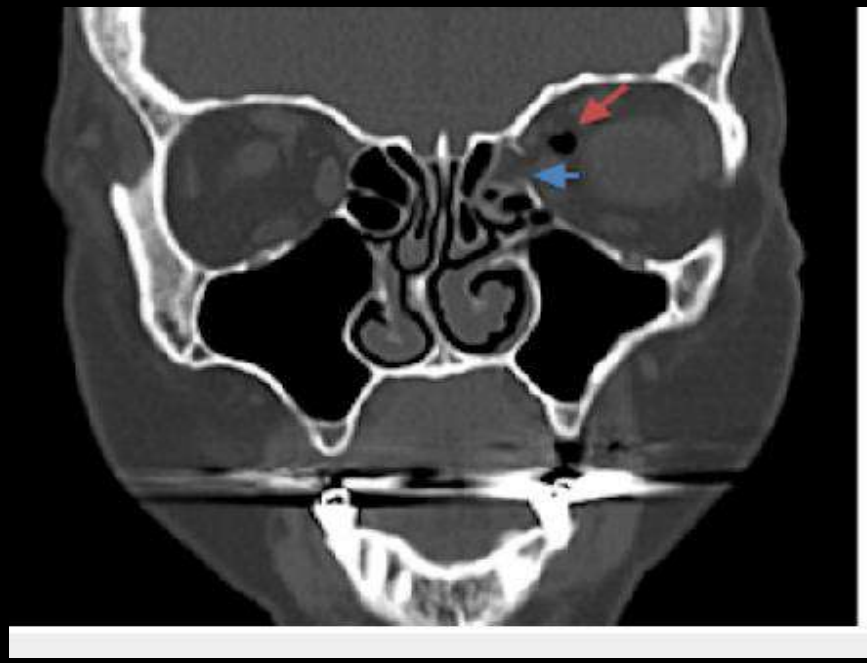


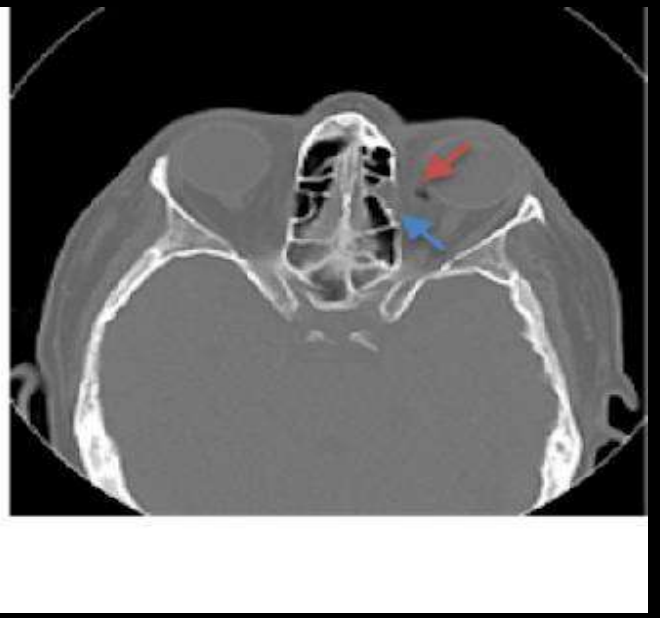
Computed tomography (CT)

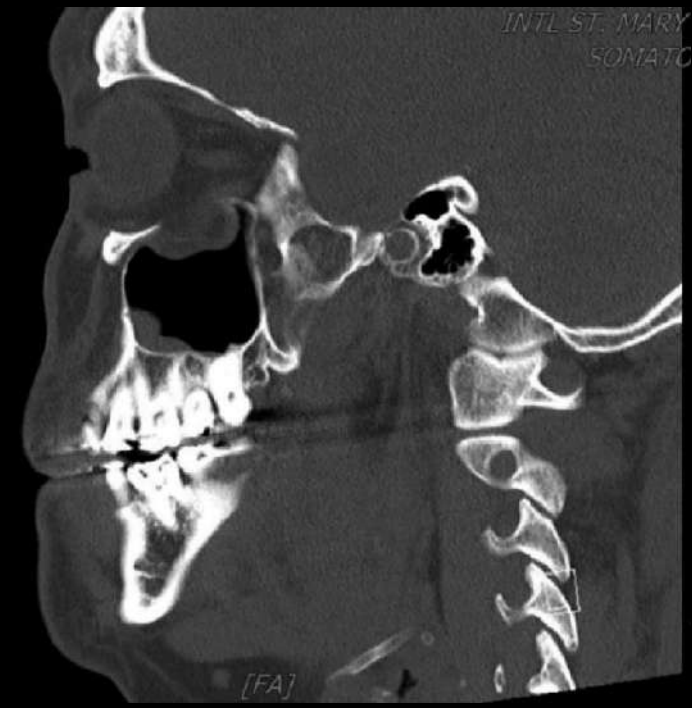
- Principle: depend on multiple x-ray projections through part of the body.
- The patient is exposed to burst of radiation as the x-ray source and bank detectors rotate around the patient.

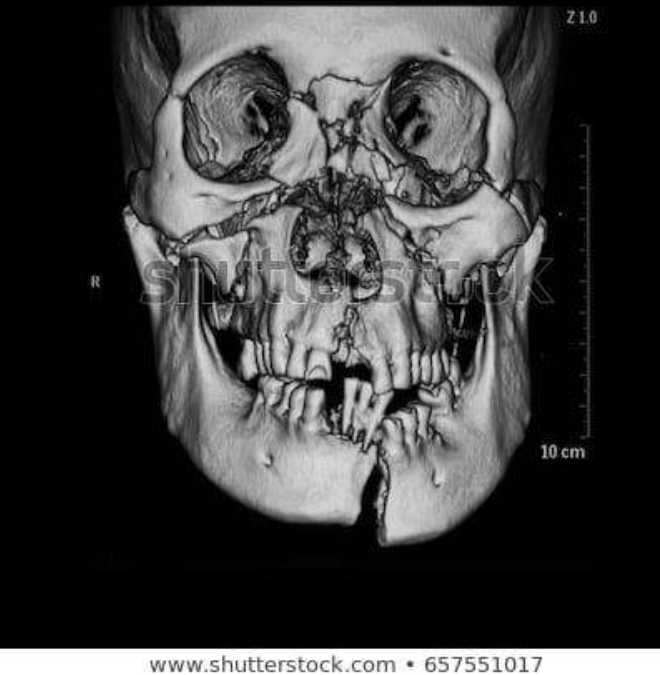
Disadvantage of conventional plain radiograph

- Ionizing radiation (X ray)
- -Two dimensional image of 3D object
- -Superimposition
- - It can't detect early pathology unless at least 30% of mineral is changed









What Is Bright on CT?

- Blood
- Contrast
- Bone
- Calcium
- Metal

What Is Dark on CT?

- Air
- H20
-

Role of CT in trauma

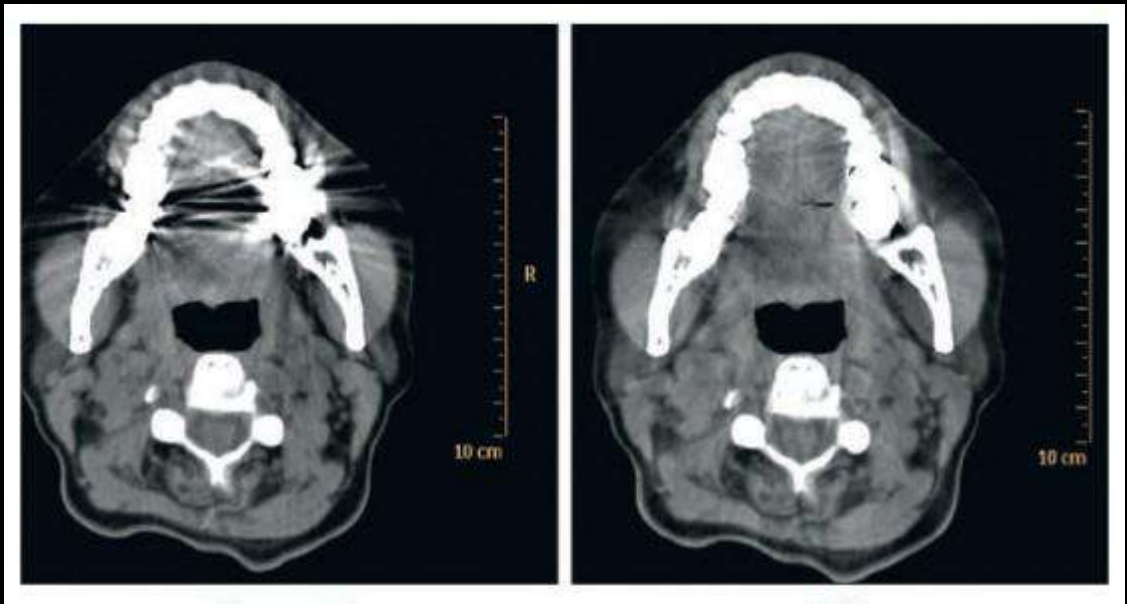
- The different contrast of various soft tissue e.g. muscle is easily demonstrated by CT.
- No superimposition of structures (unlike plain radiograph)
- Create 3D image of complex and comminuted trauma sites
- Rapid than MRI
- Can be used with contrast agent

Role of CT in oncology

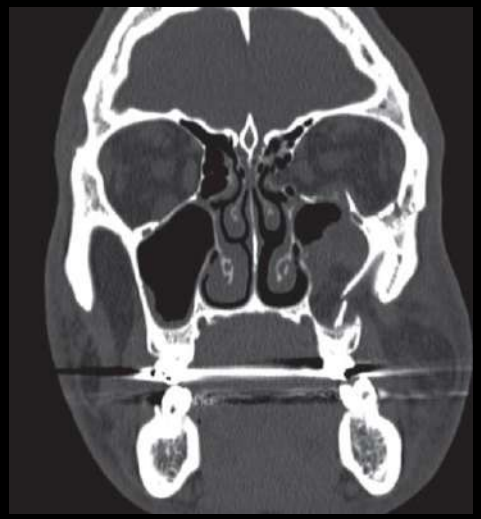
- Diagnosis of tumor (site, size and extension)
- Staging of carcinoma and detection any lymph node involvement
- Detect any recurrence of tumor
- Monitoring the therapy CCx OR RRx

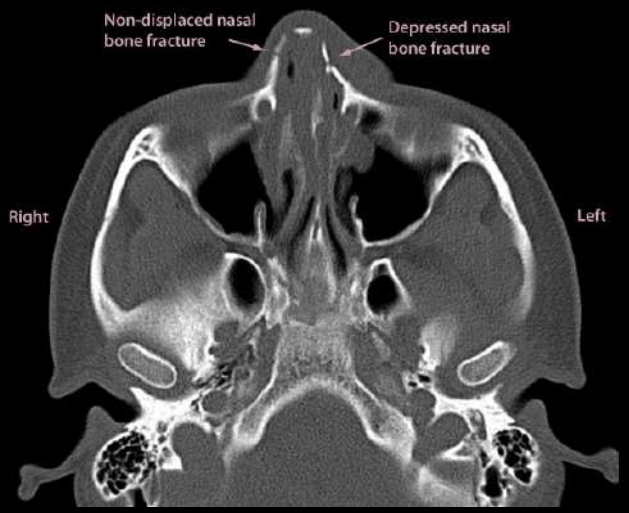
Disadvantages and limitations

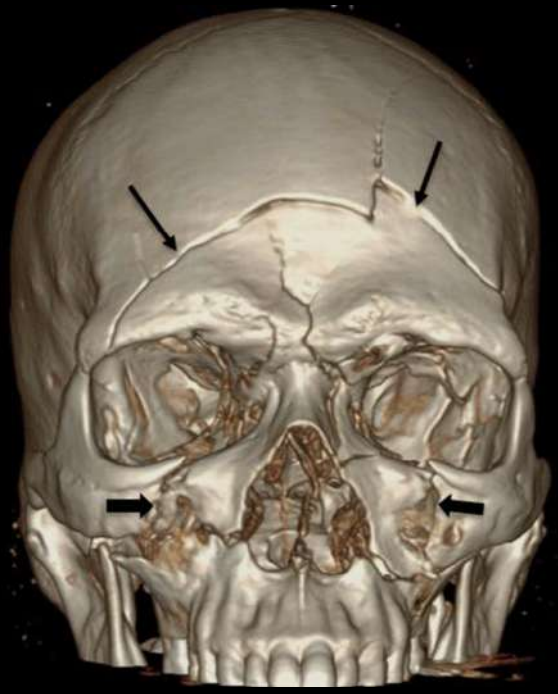
- Costy
- Radiation hazard
- Artifacts which result from metal e.g. amalgam
- Radiolucent bodies may be missed like wood



Examples of using CT in maxillofacial surgery

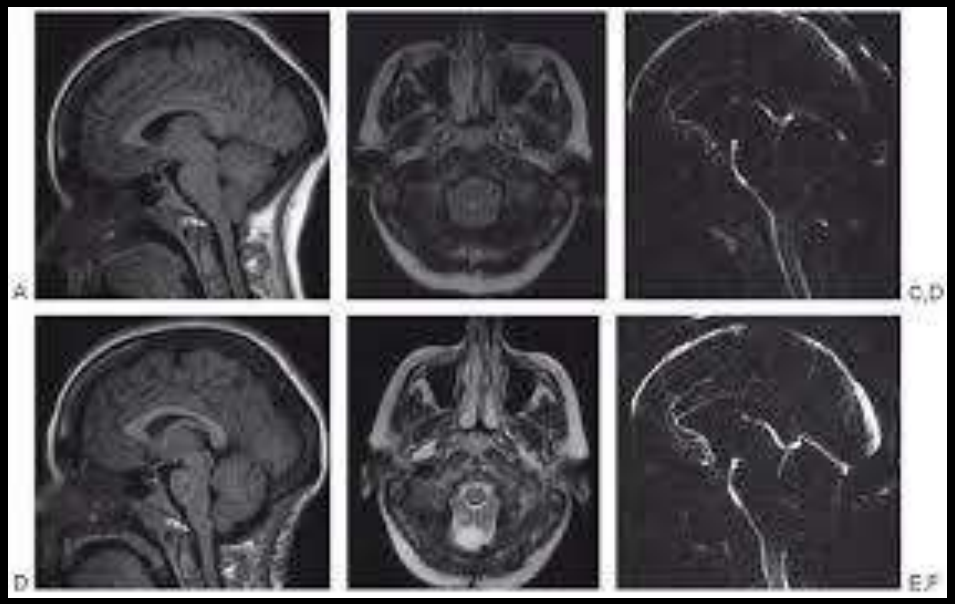



Non-displaced nasal bone fracture
Depressed nasal bone fracture
Right
Left



Magnetic resonance imaging (MRI)

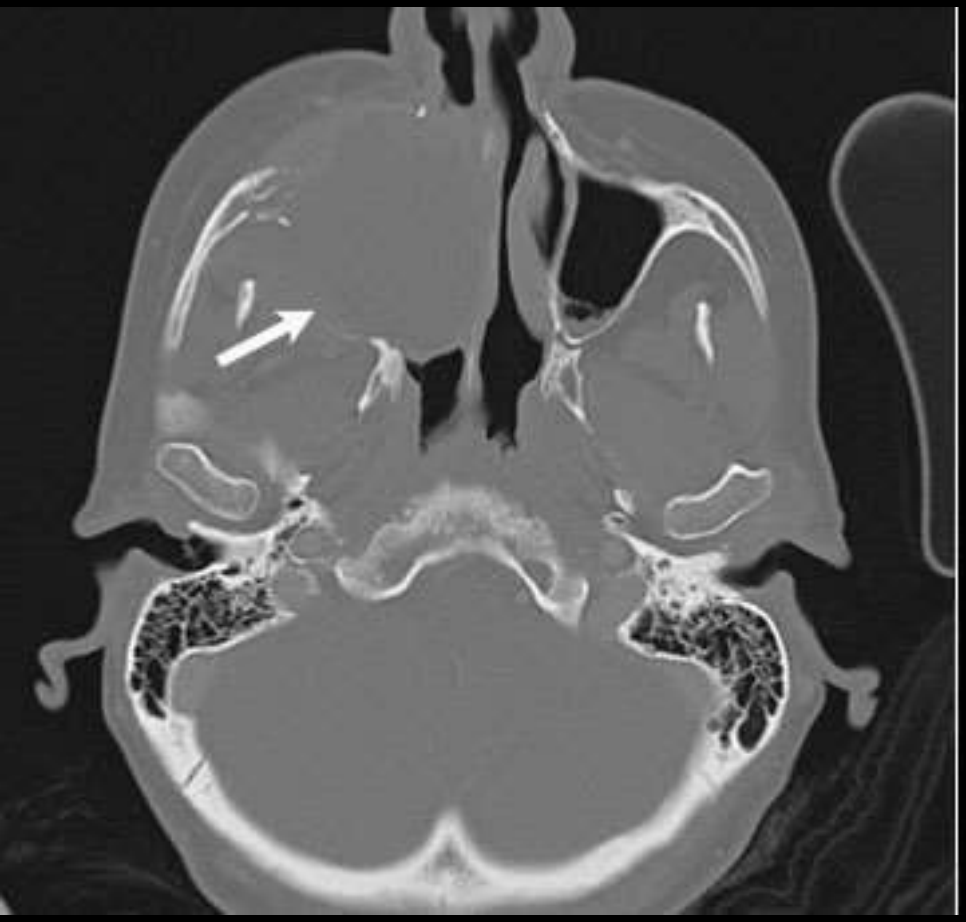
- *It use electrical and magnetic fields and radiofrequency (RF) pulses, rather than ionizing radiation to produce an image.*

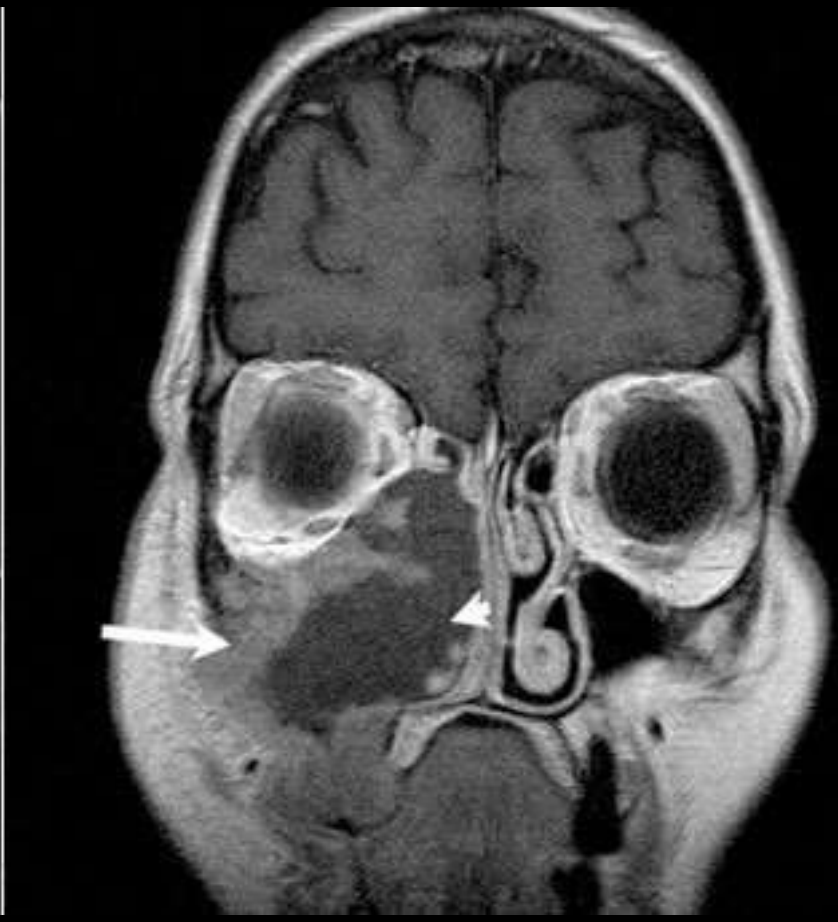


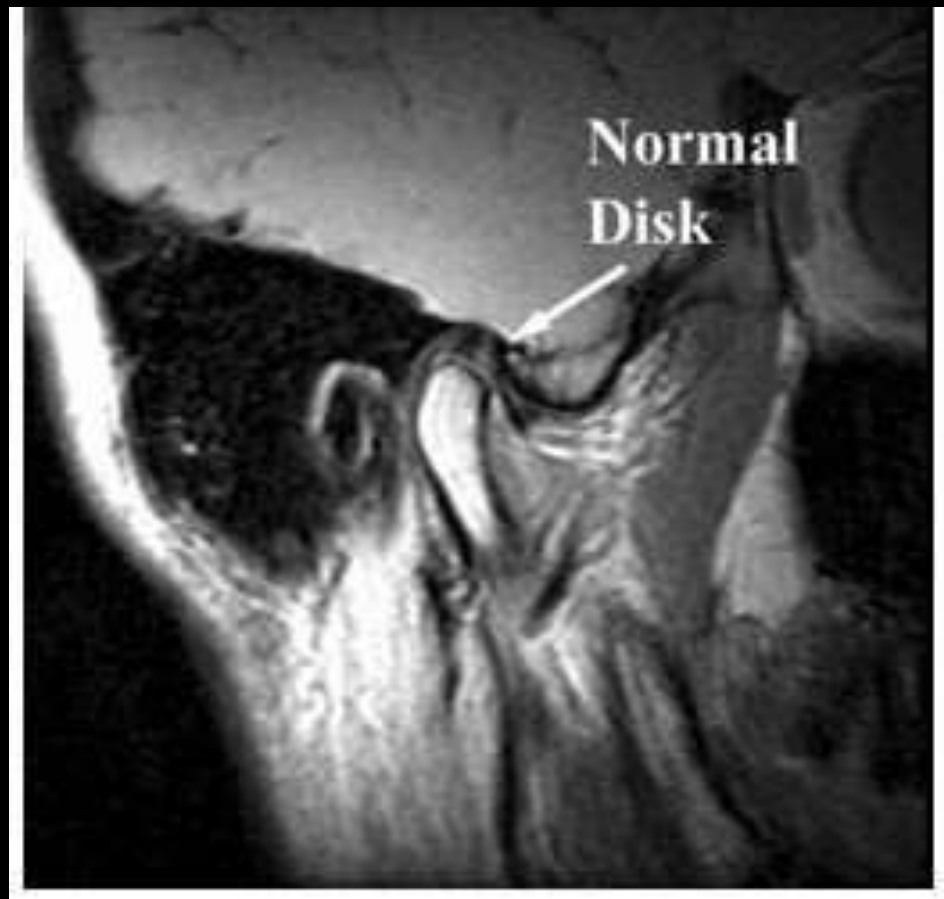
- Two distinct views are typically generated: T1 and T2.
- IN T1: fat appearing bright
  water appearing darker

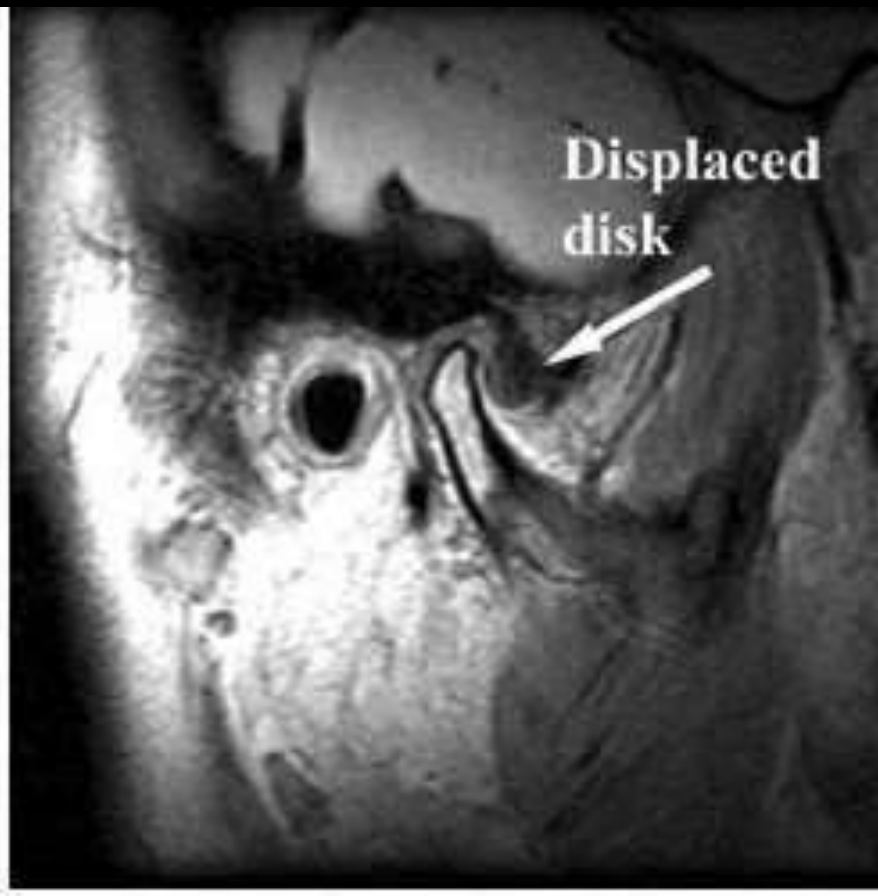
Role of MRI in dentistry

- In dentistry, the primary uses of MRI have been the evaluation of various pathologic lesions (such as tumors) and the assessment of the TMJ, salivary glands .
- To define the extension of soft T tumor
- To distinguish fluid from tumor in Paranasal sinus
- Relationship of major B.V to S.T tumor
- visualize the anatomy of cranial nerves



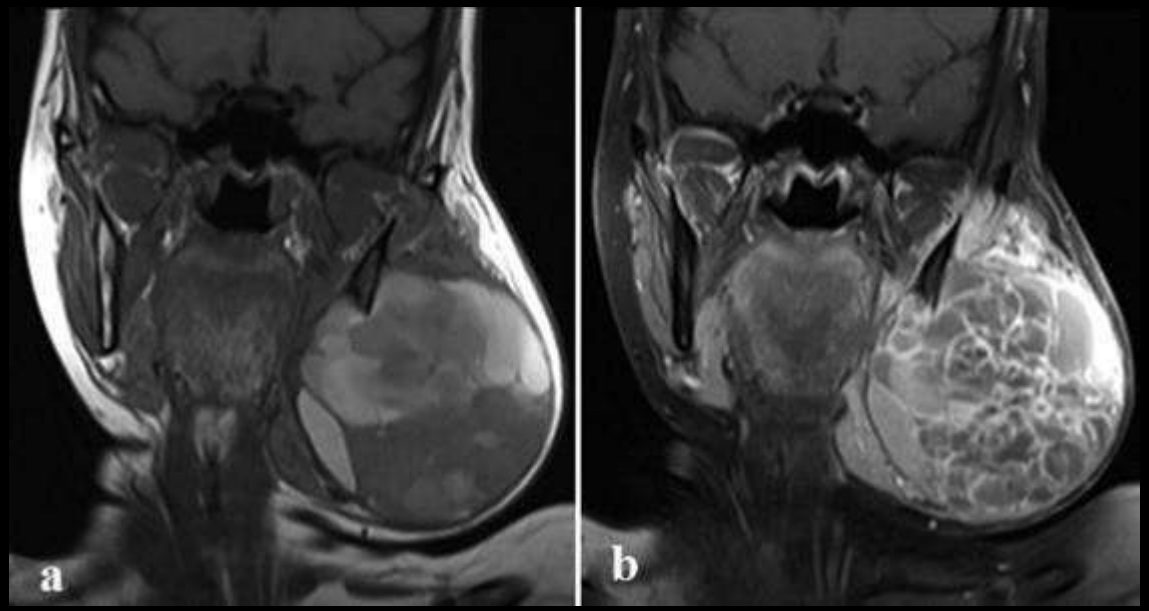



Normal
Disk

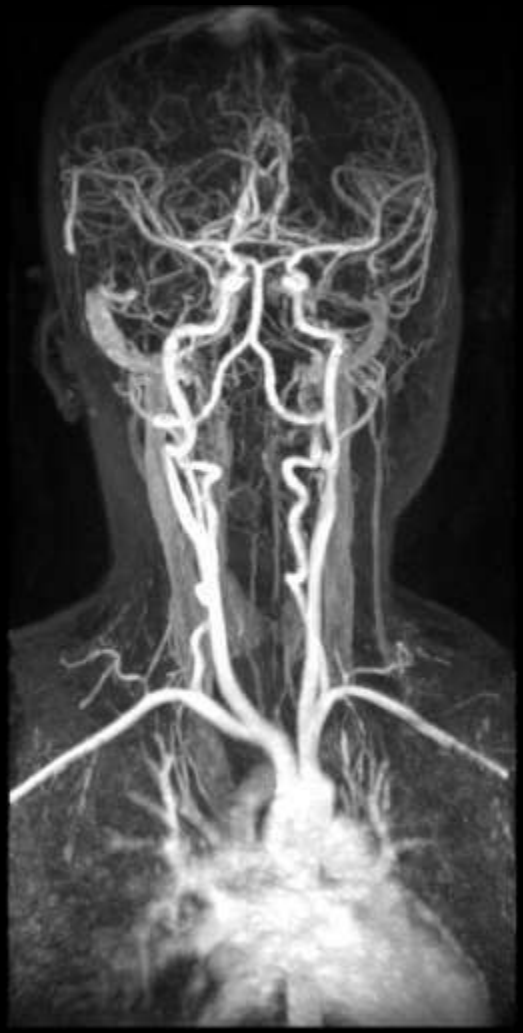

Displaced
disk

Contrast MRI

- Used for enhancement of vessels in MR angiography or for brain tumor.
- The most commonly used compounds for contrast enhancement are gadolinium



magnetic resonance (MR) angiography



The major disadvantage of MRI

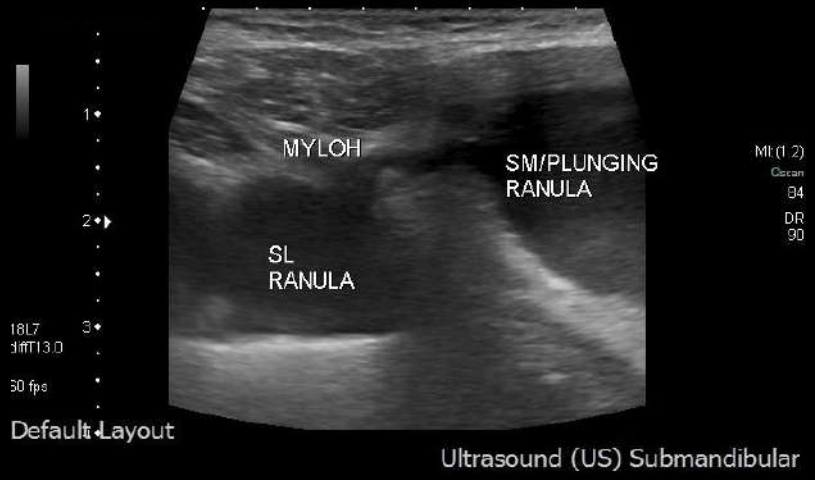
- - Long periods of time ... up to 90 minutes
- -Cost
- -Contraindicated for certain patients, Eg/patient with cardiac pacemakers, due to interference by the electrical and magnetic fields. Patients with ferromagnetic metallic objects in strategic places (such as aneurysm clips in the brain and metallic fragments in the eye) also should not be placed in the magnet.
- -Some patients feel claustrophobic inside the magnet and may need to be sedated for the procedure

Differences between CT and MRI

Table 3.2. Advantages of MRI versus CT scan

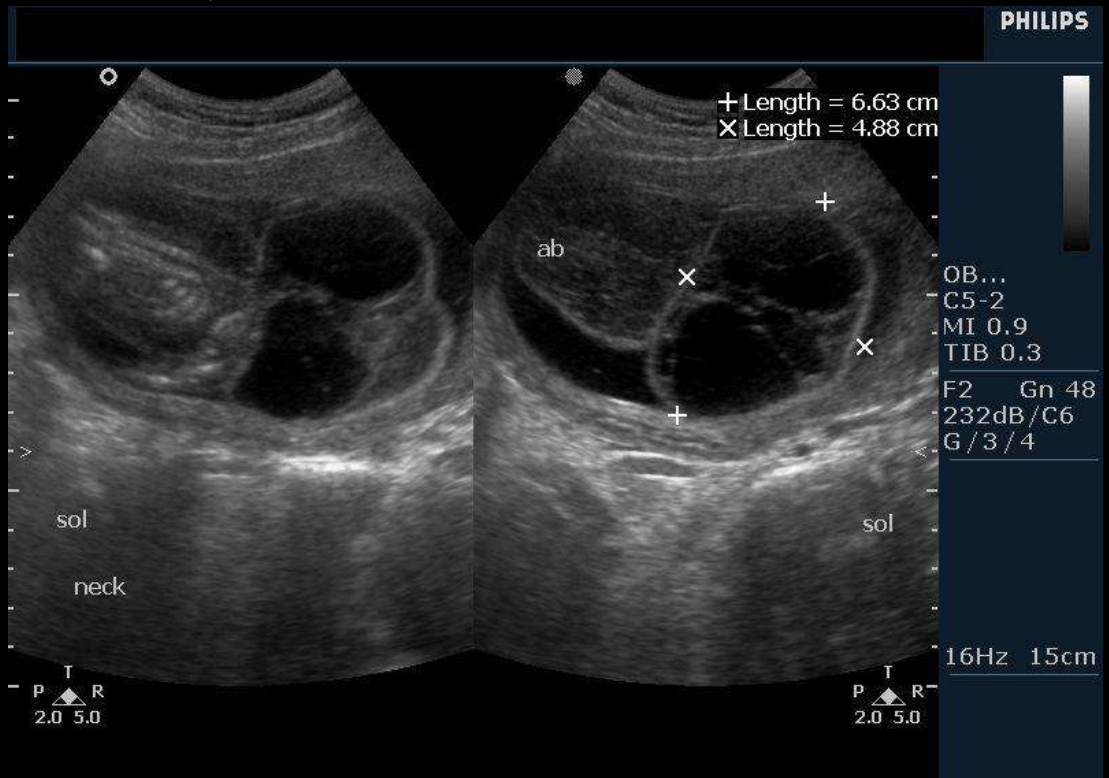
MRI	*CT scan*
• Noninvasive—no ionizing radiation	• Large radiation dose
• Vessels shown without IV contrast	• IV contrast necessary for soft tissue, blood vessels enhancement
• Direct multiplanar easy imaging	• Axial images reformatting requires large radiation
• High soft tissue contrast reduction	
• Safe procedure	
Can be repeated again following previous studies	

Ultrasonography

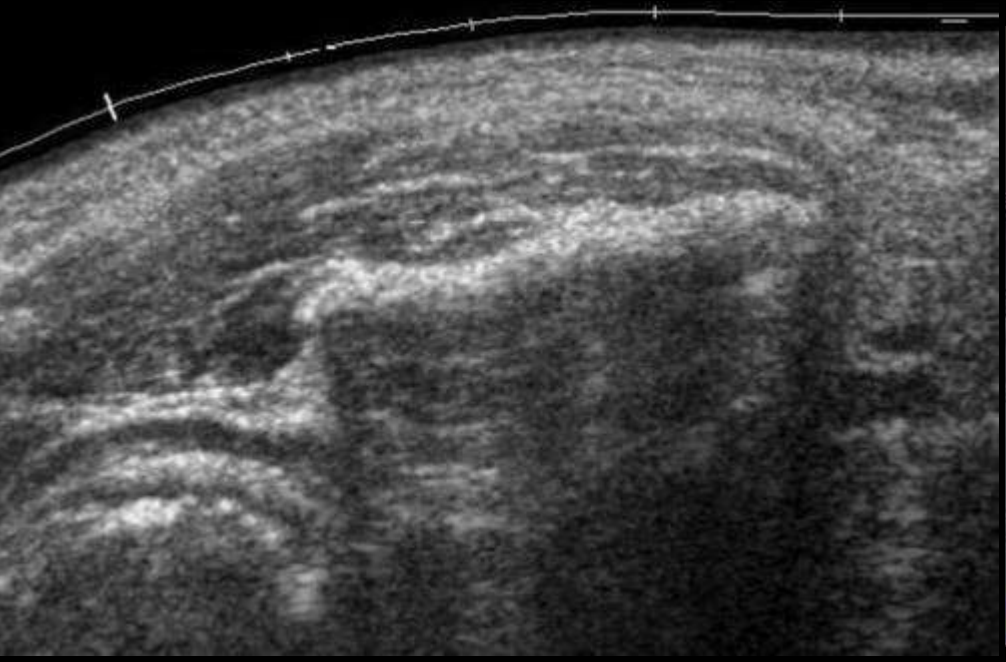


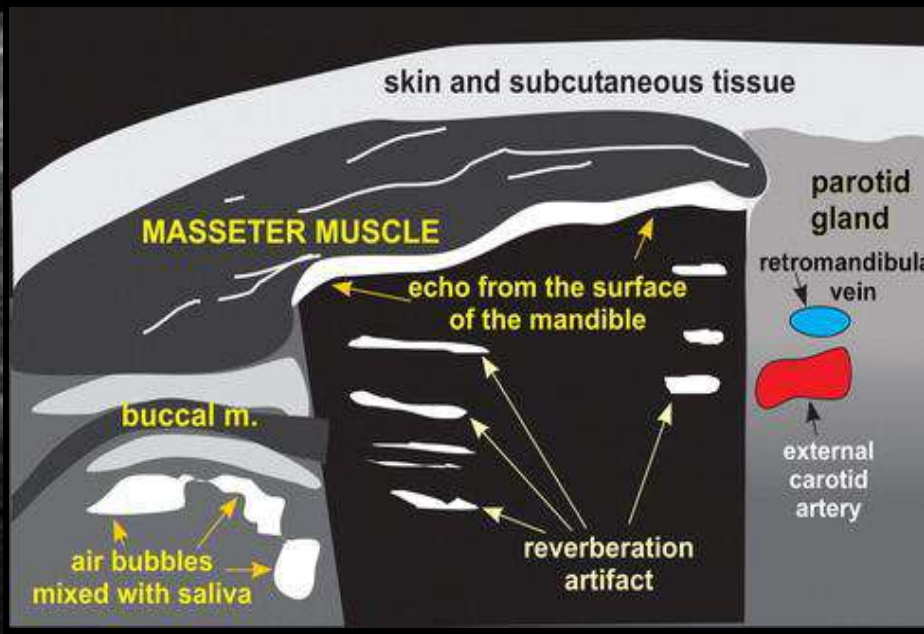
- This is a noninvasive and relatively inexpensive technique for imaging
- Principle of action: The probe converts electrical energy in to high frequency sound waves Which pass in to tissue of different densities
- The vibration energy of US is reflected back to the scanning transducer Where the sound converting in to image

- Fluid → pass without reflection → Hypoechoic
- bone & lung → All are reflected → Hyperechoic
- -Soft tissue → Waves are partly pass → Isoechoic

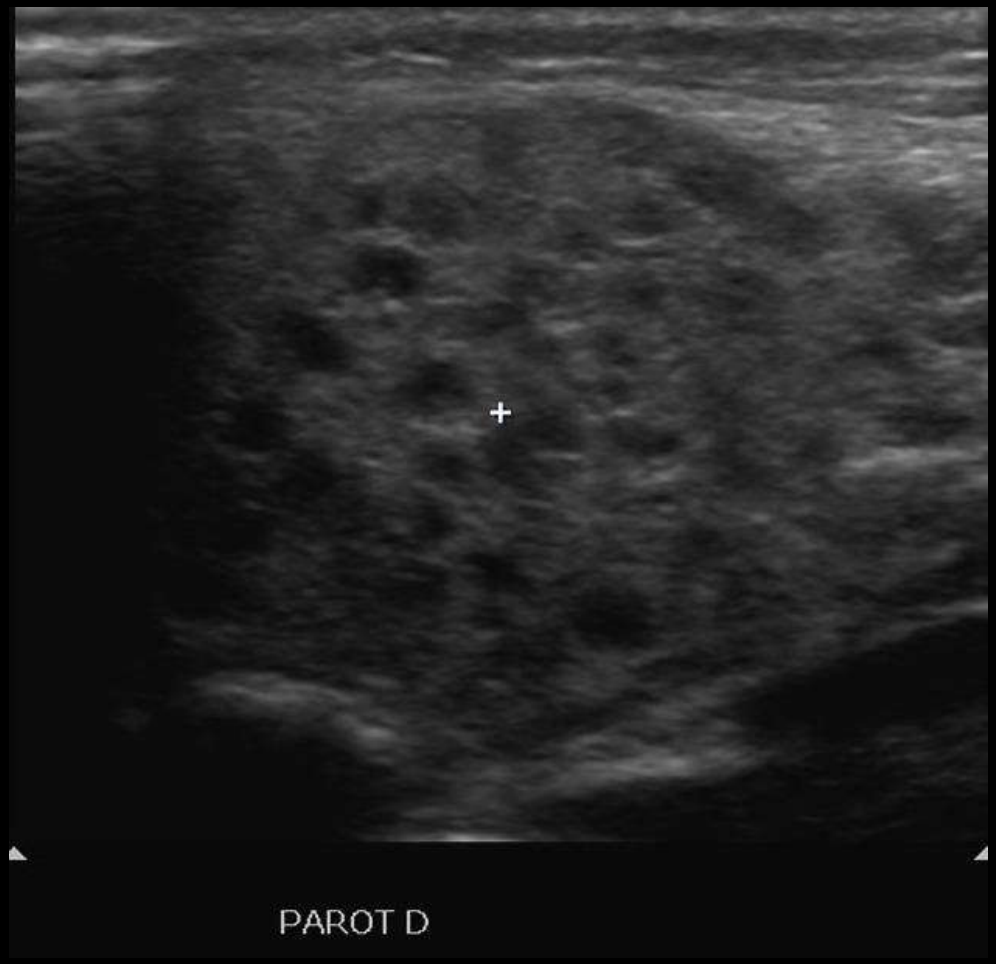


Normal parotid gland and muscles





Viral parotitis (Mump)



Indications of U/S in head and neck

A. Salivary glands;

B. Lymph nodes;

C. Congenital lesions;

D. Miscellaneous mass lesions (in soft tisssue)

E. Infection and trauma; and

F. Endocrine

Nuclear Medicine

- Radionuclide imaging is the technique of producing diagnostic images by analyzing the radiation emitted from a patient who has previously been given radioactive medications
- Nuclear medicine differs from most other imaging modalities in that the tests primarily show the physiological function
- This is done by injecting certain radioactive compounds into the patient that have an affinity for particular tissues — so-called *target tissues.*

Main indications for isotope imaging in the head and neck

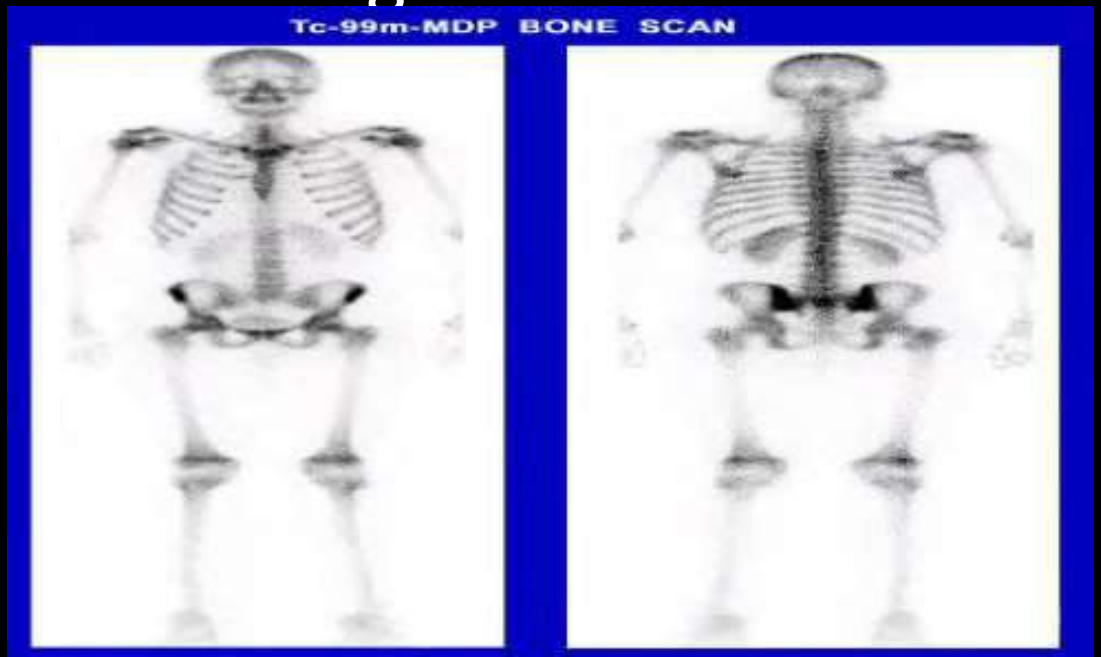
1. Tumour staging — the assessment of the sites and extent of bone metastases
2. Investigation of salivary gland function, particularly in Sjogren's syndrome
3. Evaluation of bone grafts
4. Assessment of continued growth in condylar hyperplasia
5. Investigation of the thyroid
6. Brain scans and assessment of a breakdown of the blood-brain barrier.

Types of nuclear imaging

- Scintigraphy
- Positron Emission Tomography (PET)

Scintigraphy

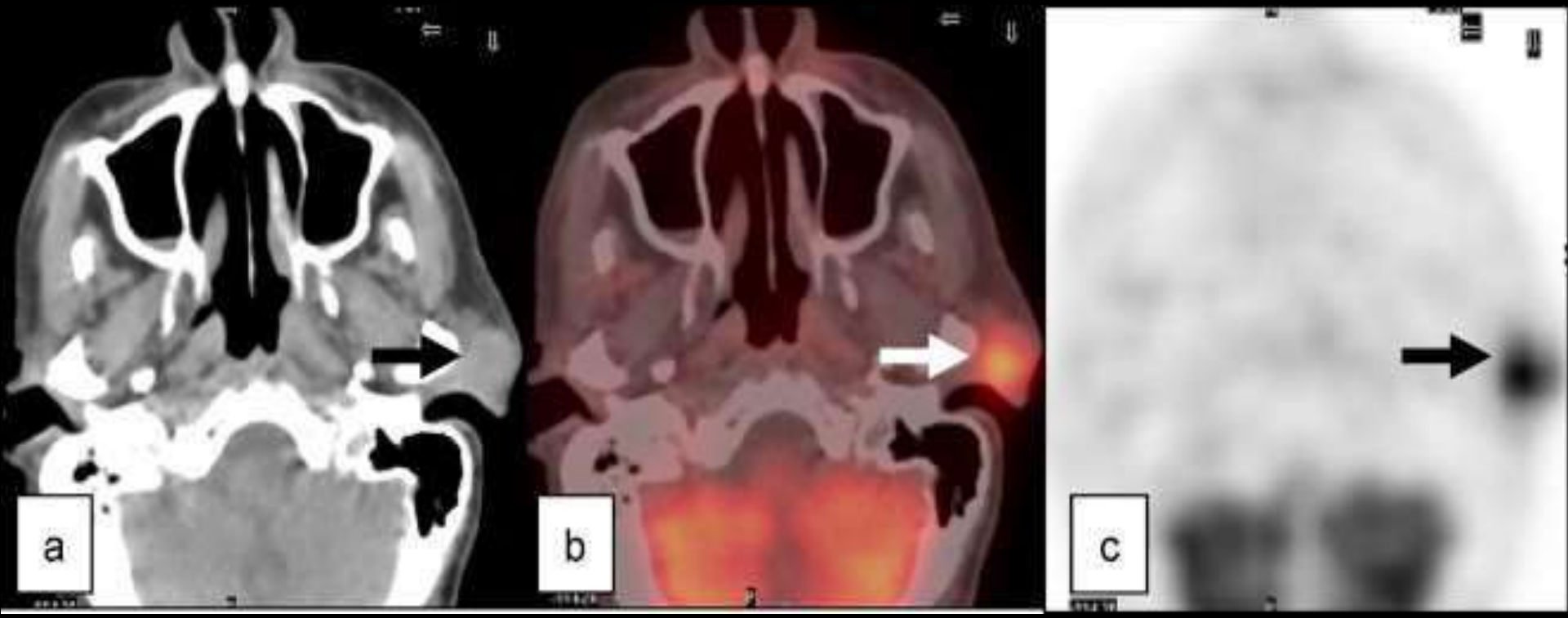
- a form of diagnostic imaging where the radioisotopes are taken internally, and the emitted radiation is captured by external detectors (gamma cameras) to form two-dimensional images



Positron Emission Tomography (PET)

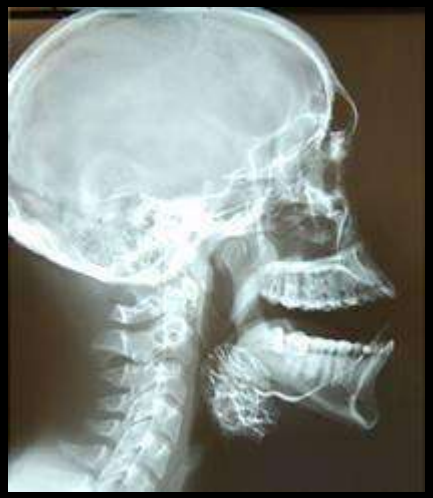
- Positron imaging radionuclide e,g, fluorine -18 can be bound to glucose which is used in human metabolic pathways.
- Used to detect malignancy because one of the hallmarks of malignant tissue is its increased glucose uptake compared with nonmalignant tissue.

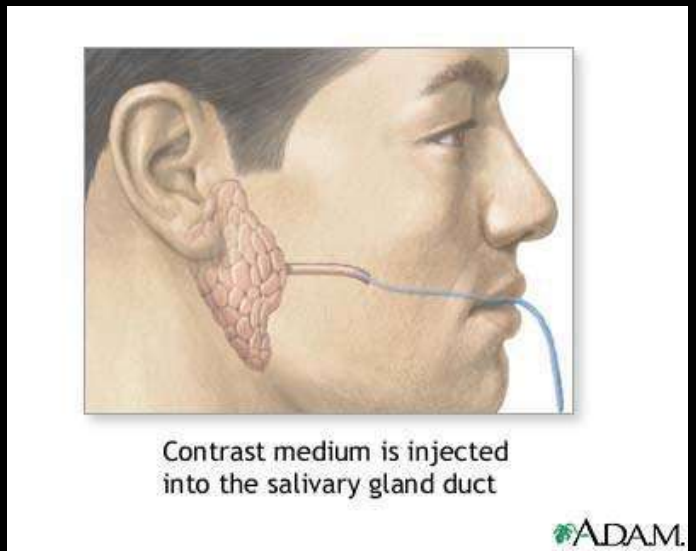
PET/CT: Intense FDG uptake due to a Warthin's tumour within the left parotid gland



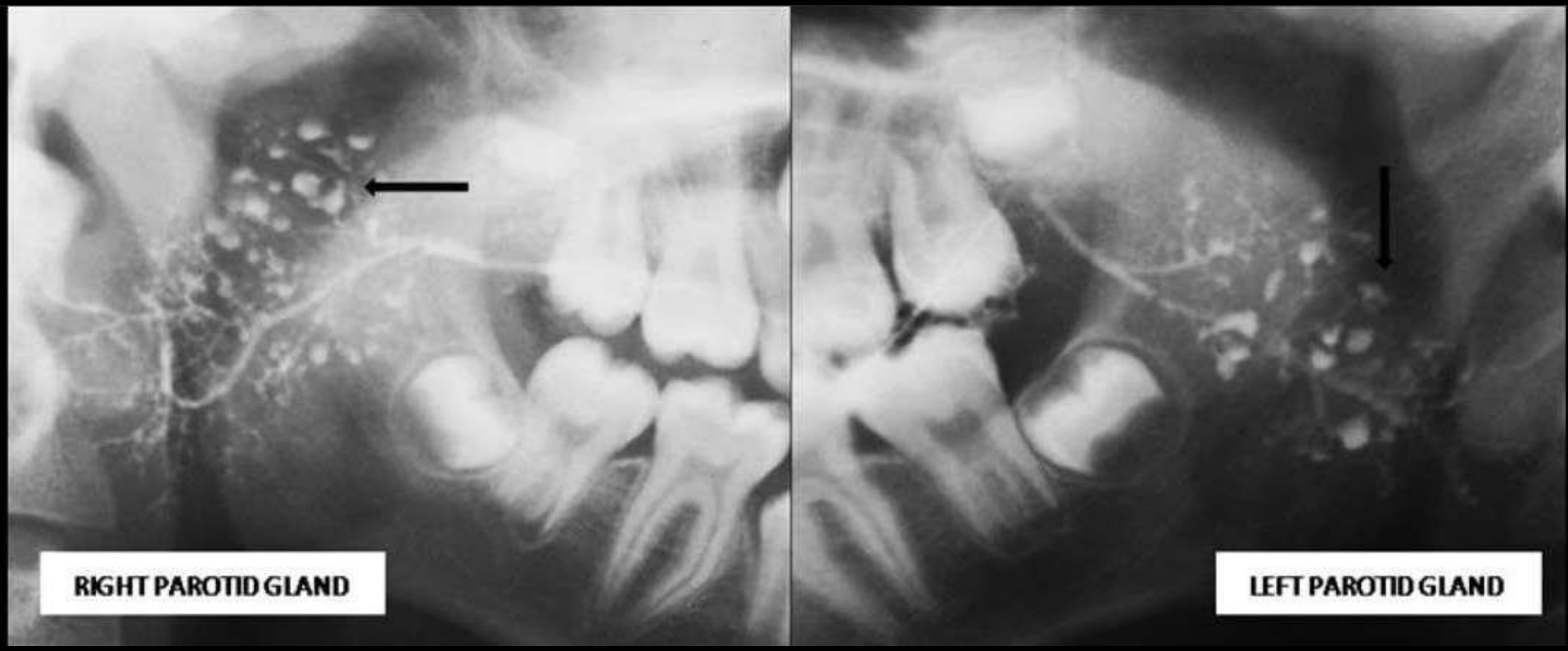
Sialography

- Used for evaluating the ductal system of the major salivary glands
- Contrast medium is injected into the major duct of the salivary gland of interest . Then radiograph is taken
- It is contraindicated during acute infection because of possible exacerbation.
- Can be used with CT sialography & MRI sialography

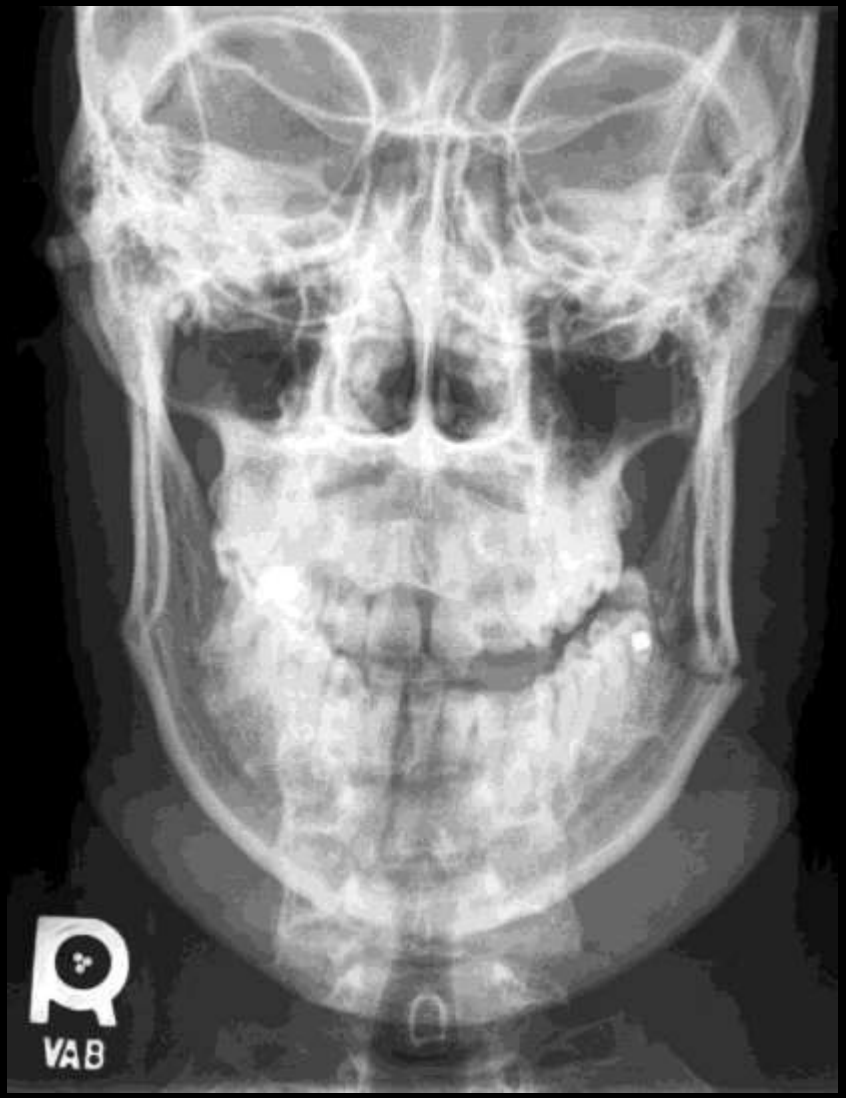


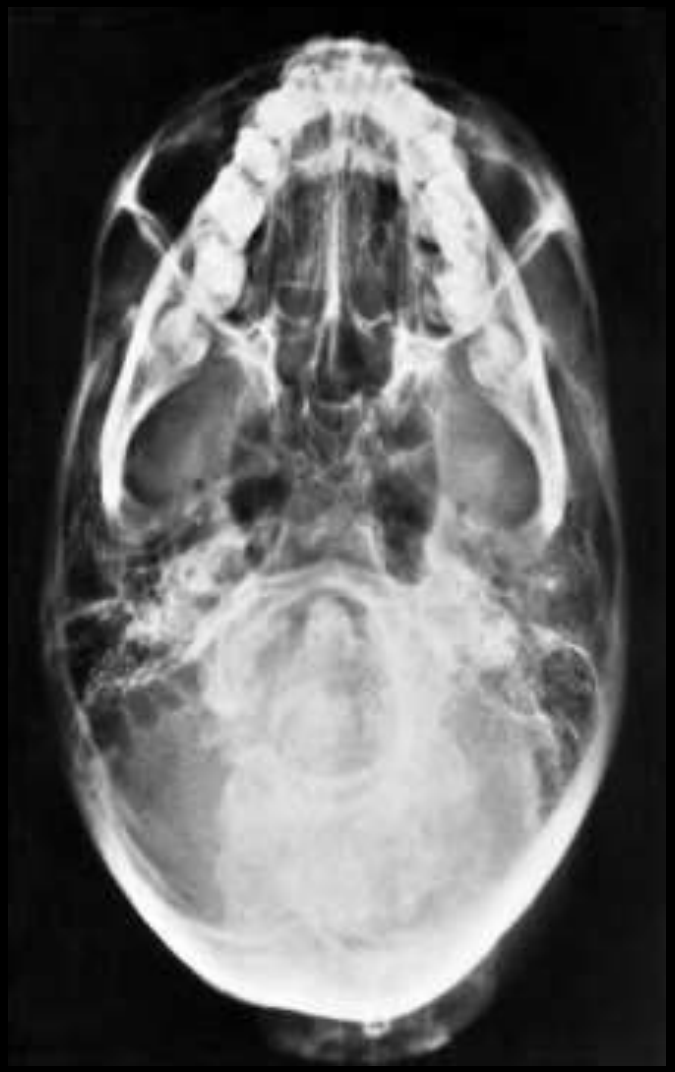


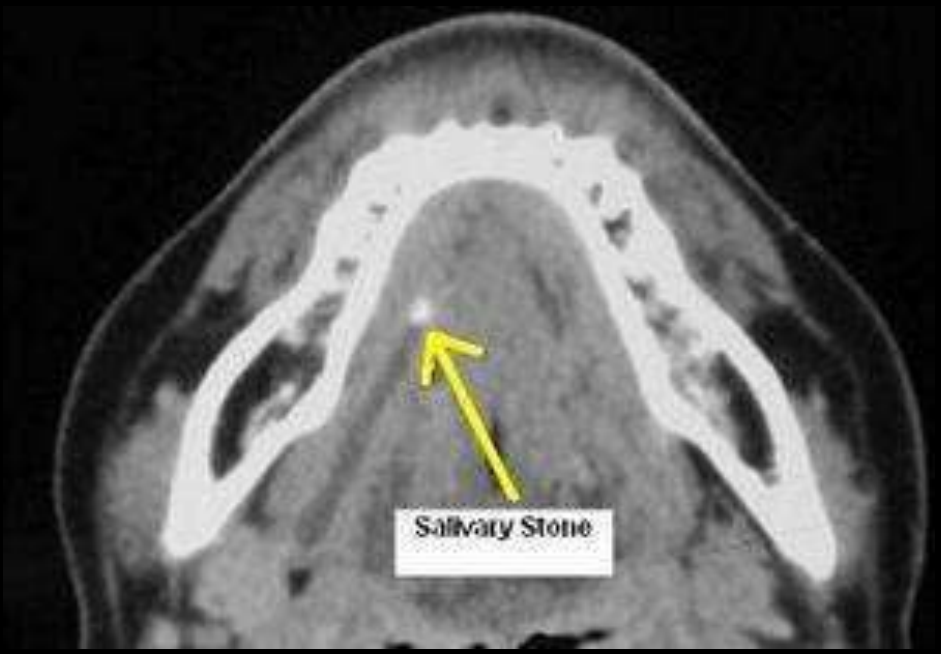
Sialogram of the right and left parotid glands with juvenile recurrent parotitis demonstrates sialectasis (arrow) on the panoramic radiograph

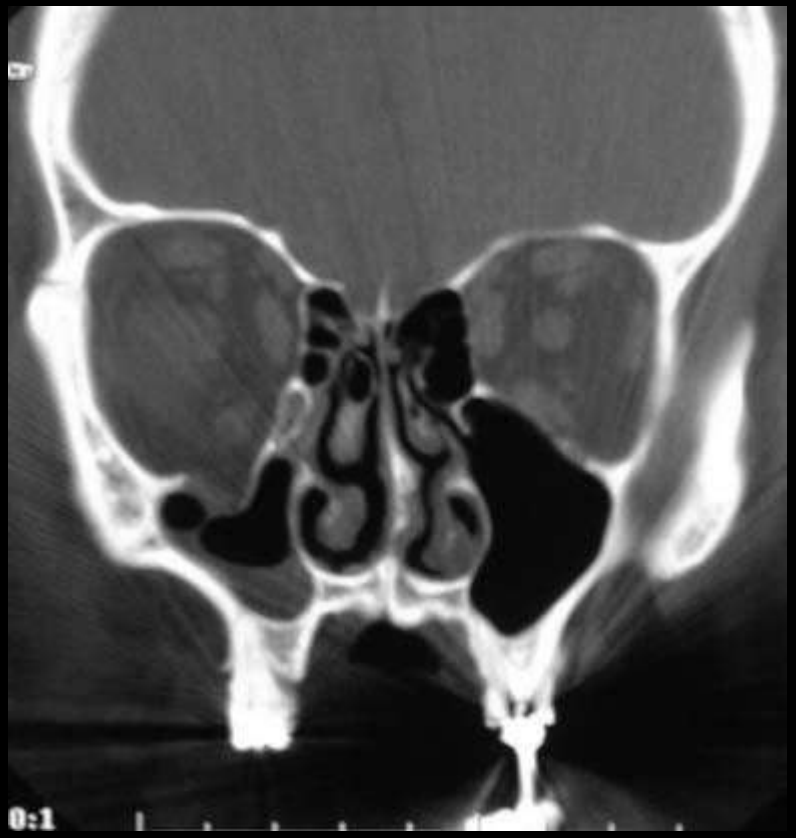


Self assessment


VAB




Salivary Stone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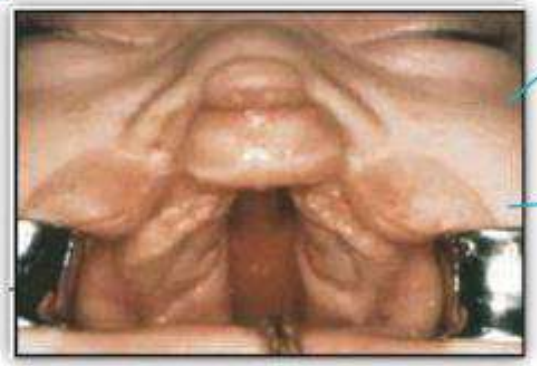
Thank you

Cleft lip and palate

Cleft lip and palate (CLP)

- The cleft is a congenital abnormal space or gap in the upper lip, alveolus, or palate.
- The general dentist will become involved in managing CLP special dental needs, because they may have partial anodontia and supernumerary teeth.
- Malocclusion is usually presents, and orthodontic therapy with or without corrective jaw surgery is frequently indicated.
- Treatment requires team approach

Management of cleft lip and palate



ENT surgeon

Social worker

Genetic scientist

Nursing support

Plastic and oral surgeon

Speech therapist

Orthodontist

Prosthodontist

psychiatrist

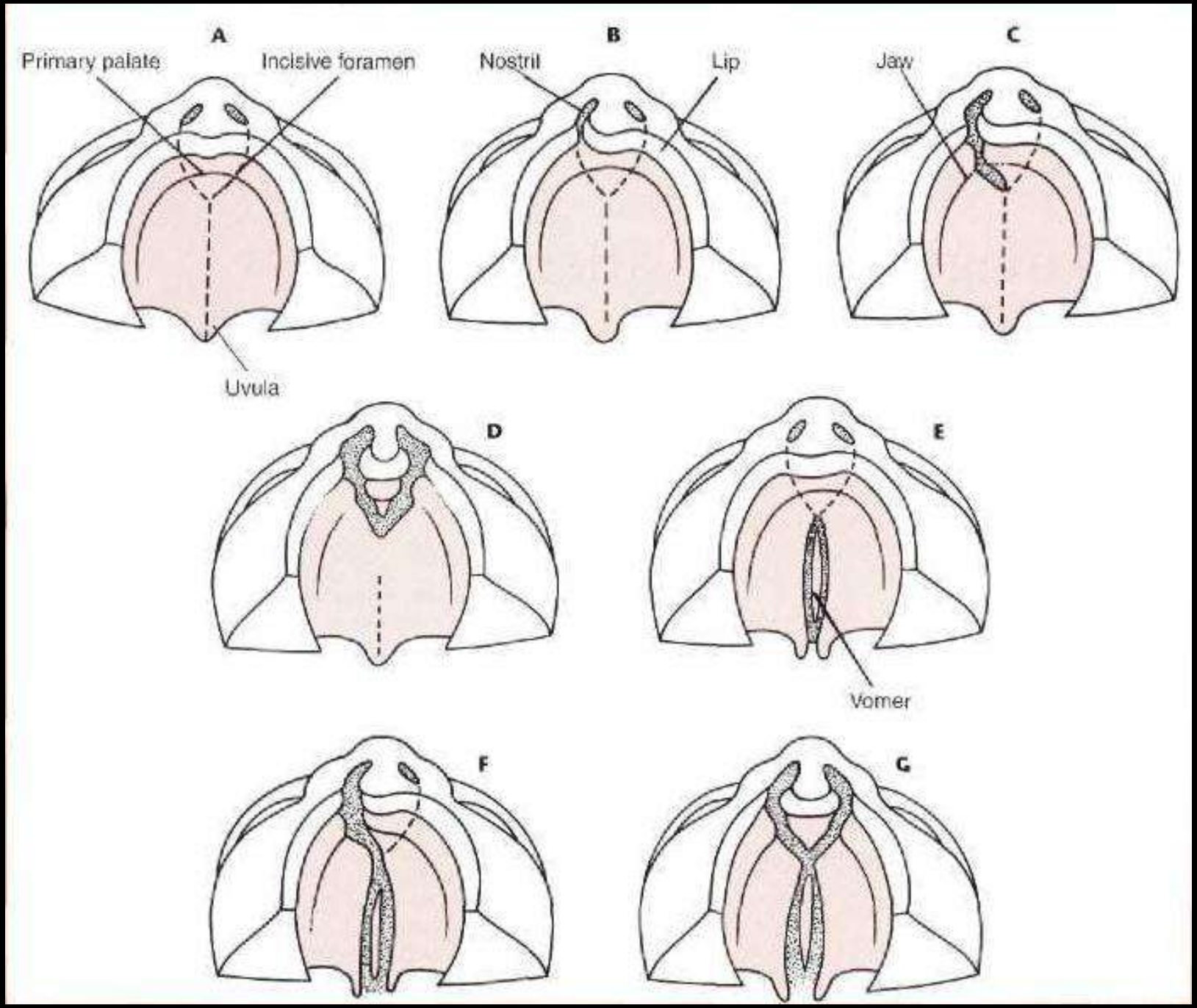
Ophthalmologist

Pediatric dentist

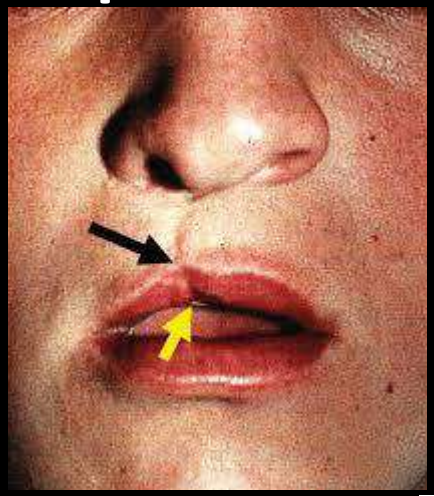
Multidisciplinary cleft palate team

Incidence and prevalence

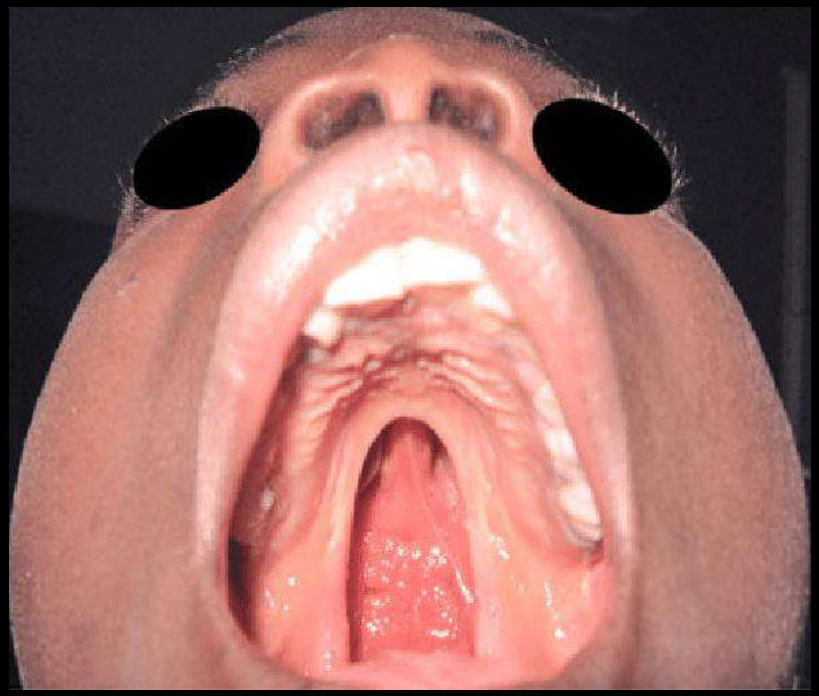
- 1 in 700 births.
- Negroids having least incidence (0.4/1000) and asians having the highest incidence.
- Cleft lip and palate (together) occurs about twice as often in boys as in girls, whereas isolated clefts of the palate (without cleft lip) occur slightly more often in girls.
- 75% are unilateral deformities; 25% are bilateral. The left side is involved more frequently than the right

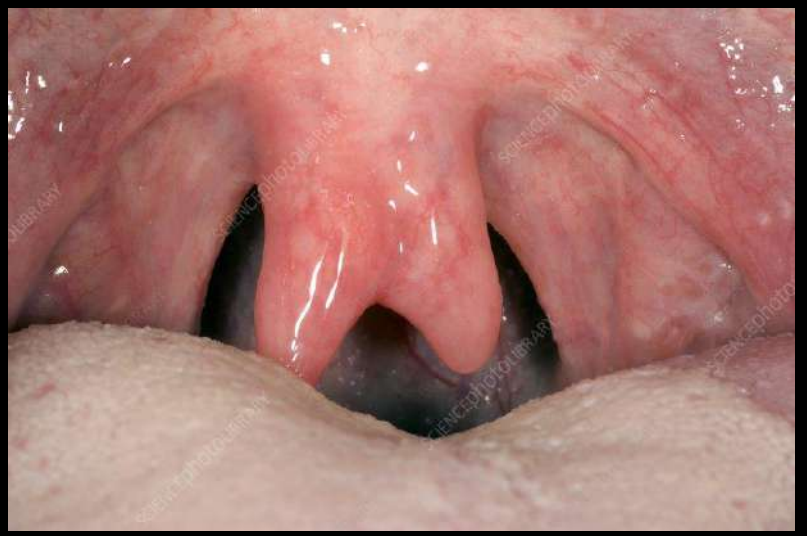

A
Primary palate
Incisive foramen
Uvula
B
Nostril
Lip
C
Jaw
D
E
Vomer
F
G

Cleft lip variations



- Clefts of the soft palate may also show wide variations from a bifid uvula to a wide inoperable cleft. The bifid uvula is the most minor form of cleft palate. In which only the uvula is clefted





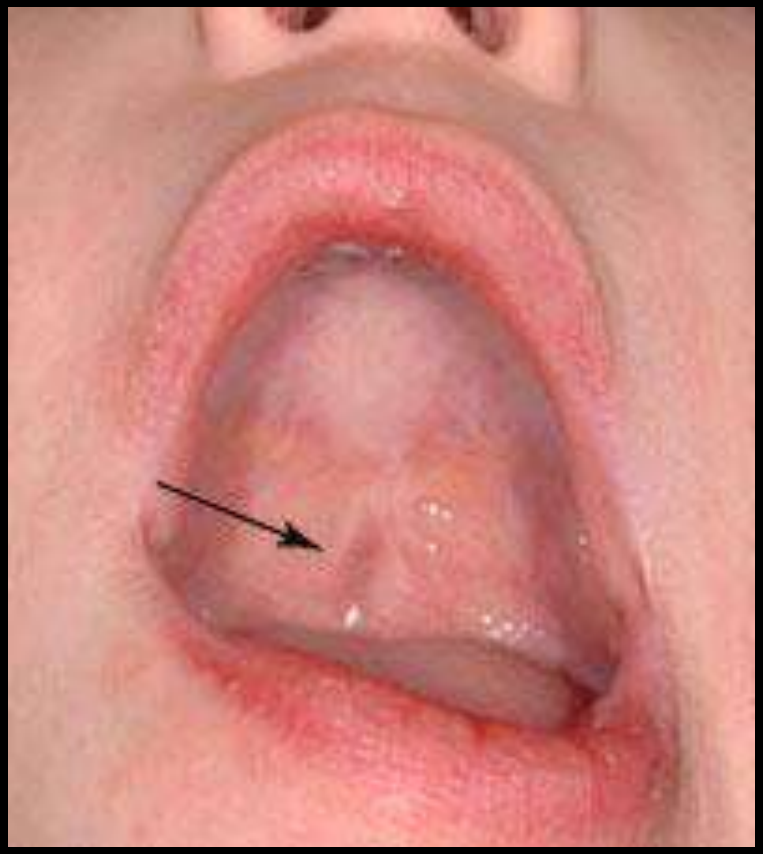
Submucosal clefts of the soft palate

- To diagnose such a defect,

I. the dentist inspects the soft palate while the patient says *"ah,“*

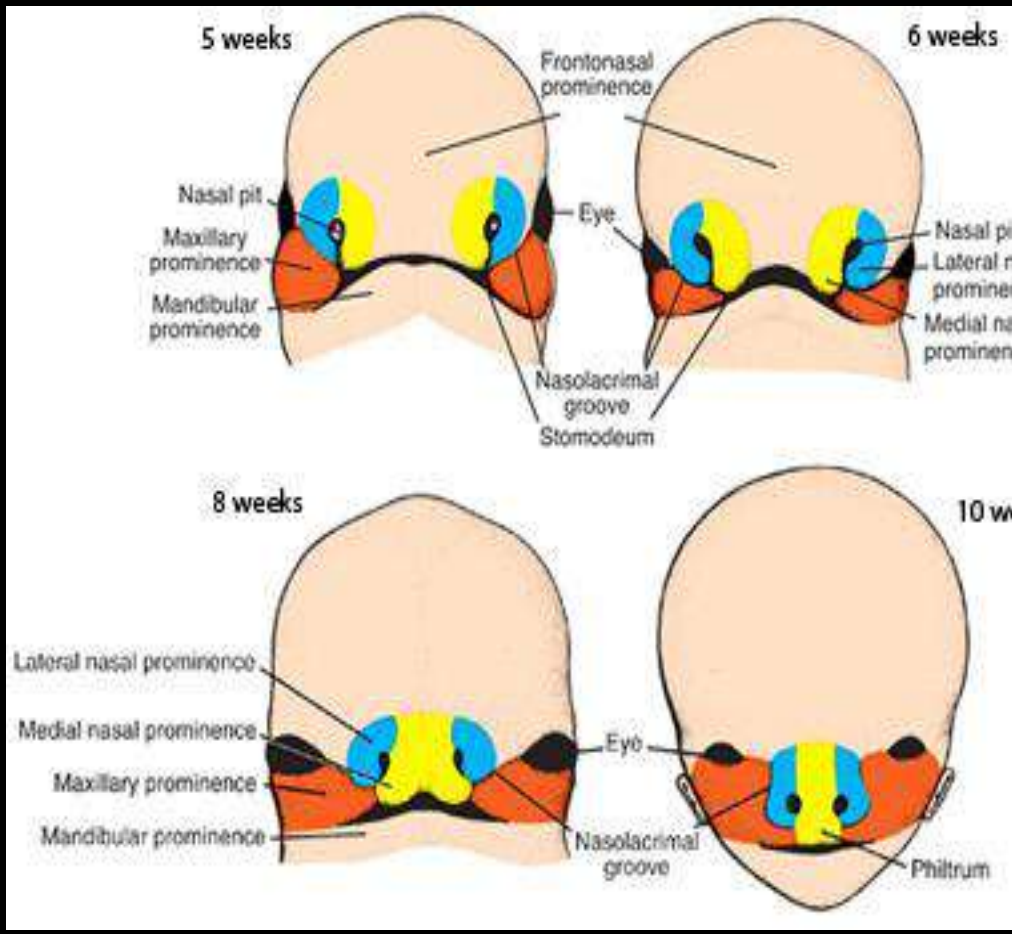
II. palpate the posterior aspect of the hard palate to detect the absence of the posterior nasal spine

III. Hypernasal speech



Embryology

- Failure of merging between the medial nasal and maxillary processes at 5 weeks' gestation,on one or both sides,results in cleft lip.

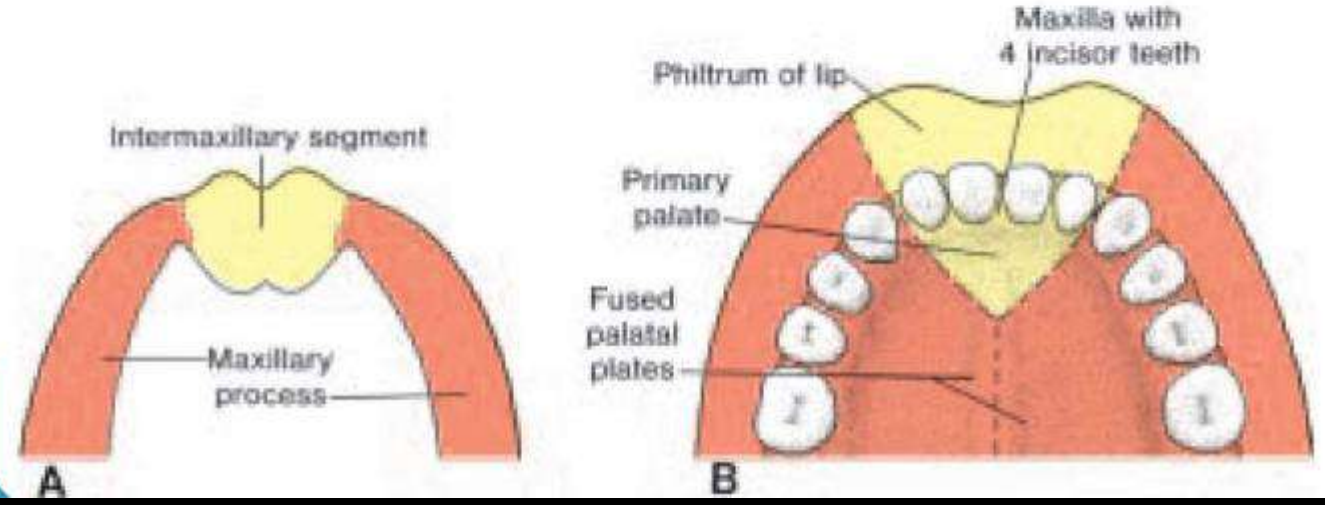


Development of palate

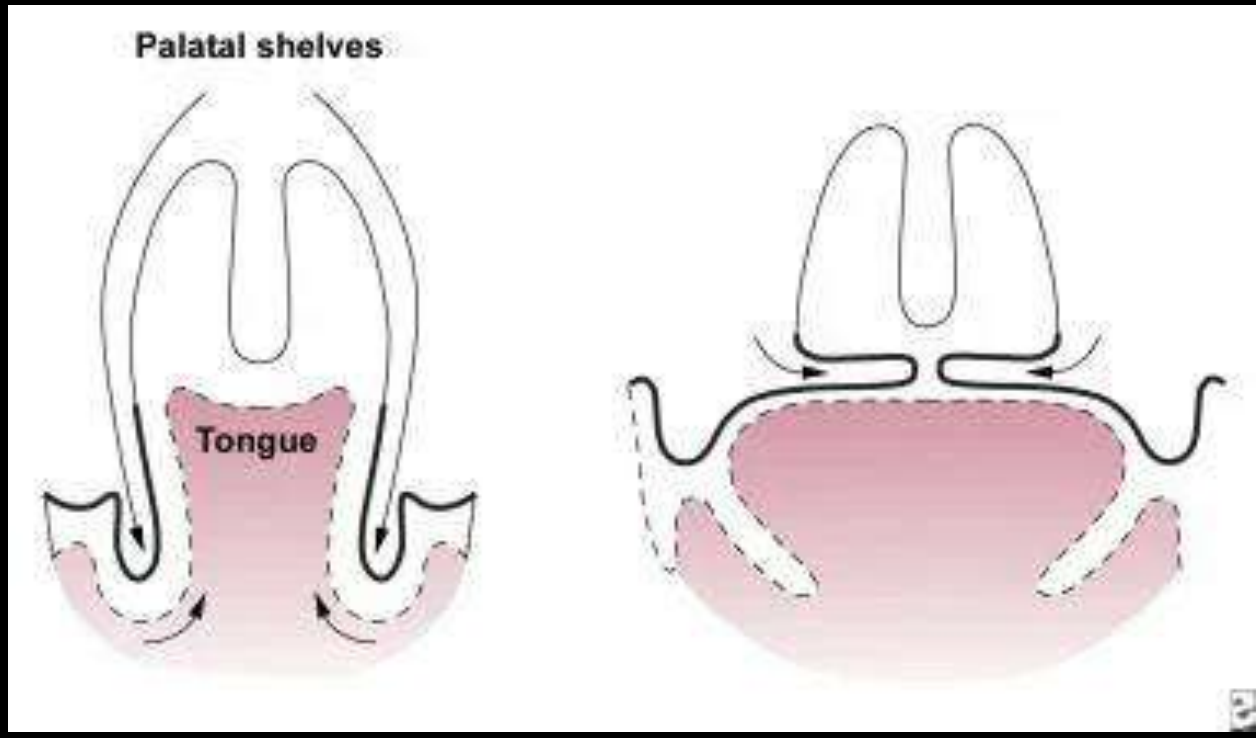
Development begins in 6th week

Develops from-

1. Primary palate(from medial nasal process)
2. Secondary palate(from maxillary process)



- Fusion of palatal shelves begin at 8th week which continues till 12/17th week



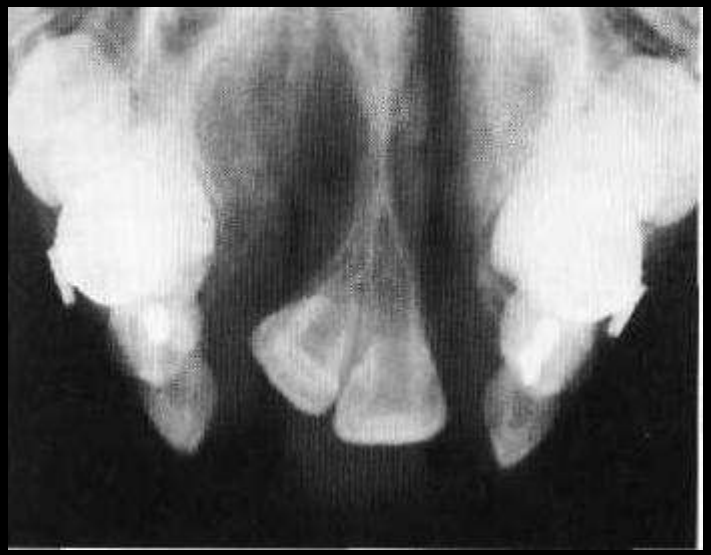
The causative factors

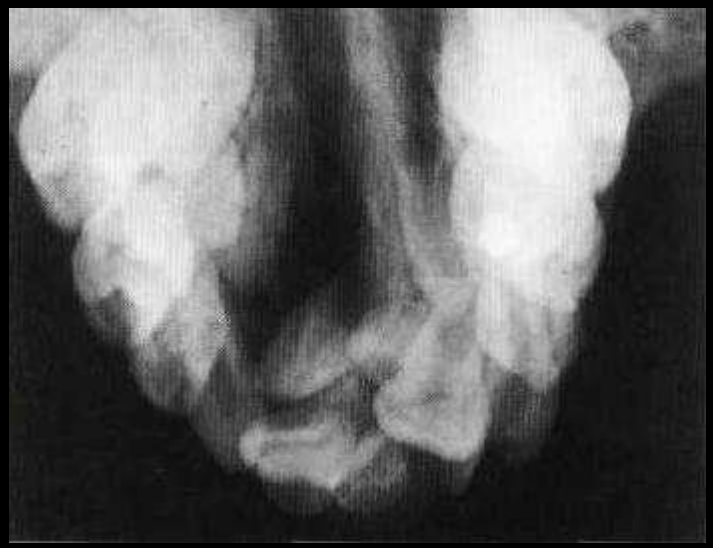
- it is important to distinguish between *isolated clefts* and *clefts associated with other birth disorders or syndromes.*
- *There is interaction between*

I. Hereditory
II. Environmental : Nutritional deficiencies, radiation, several drugs, hypoxia, viruses and vitamin excesses or deficiencies

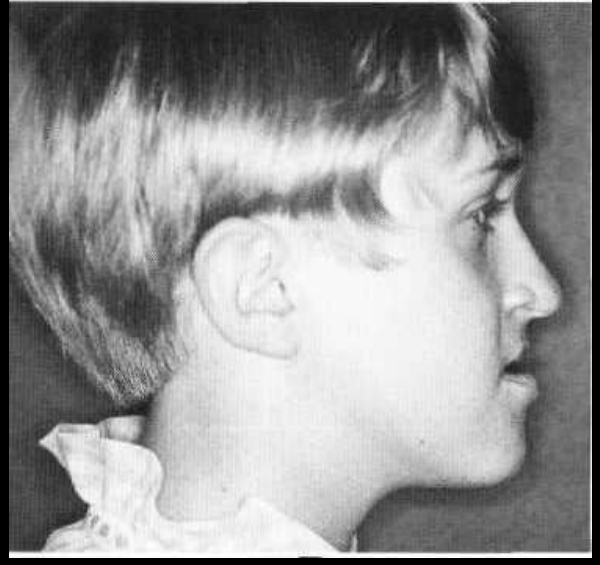
PROBLEMS OF CLEFT AFFLICTED INDIVIDUALS

- **1-Dental Problems**
- Congenital absence of teeth
- Supernumerary teeth



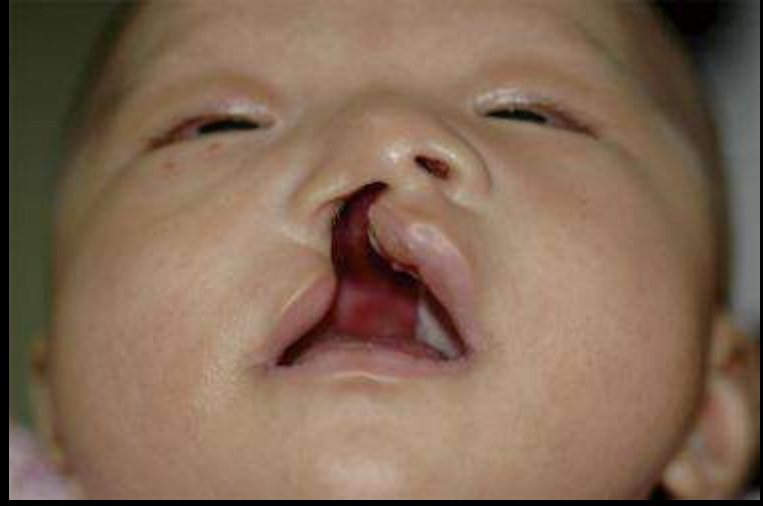


2-Malocclusion



- The maxilla may be deficient in all three planes of space, with retrusion, constriction, and vertical underdevelopment common.
- Class III malocclusion
- Orthodontic treatment may be necessary
- Space maintenance and control is instituted during childhood.
- Appliances to maintain or increase the width of the dental arch are frequently used. This treatment is usually begun with the eruption of the first maxillary permanent molars.

3-Nasal Deformity



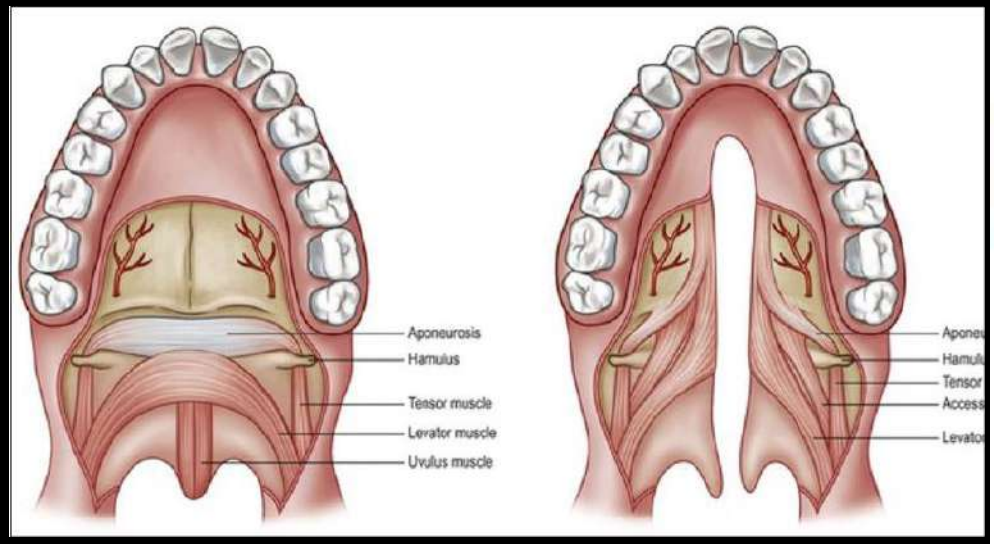
- the alar cartilage on that side is flared
- the columella of the nose is pulled toward the noncleft side
- Surgical correction of nasal deformities should usually be deferred till puberty

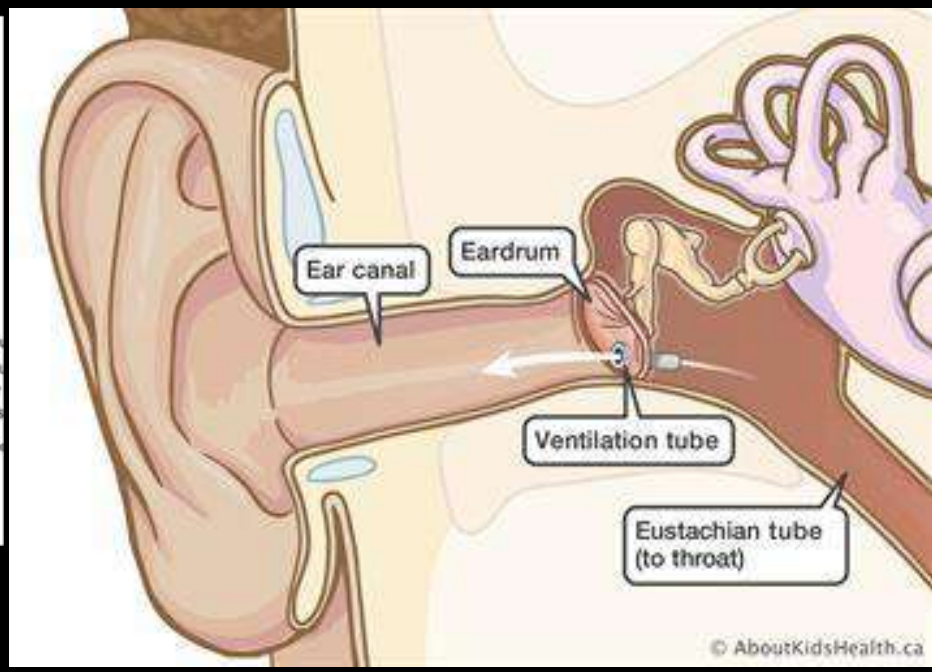
4-Feeding

- have extreme difficulty producing the necessary negative pressure in their mouth to allow sucking either breast or bottle milk.
- overcome through the use of specially designed nipples , eyedroppers or large syringes with rubber extension tubes

5-Ear Problems

- Children afflicted with a cleft of the soft palate are predisposed to middle ear infections
- Treated by (myringotomy)



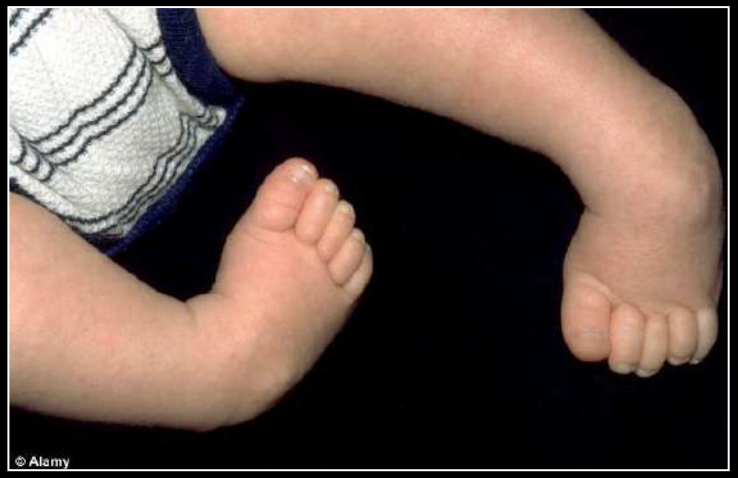


6-Speech Difficulties

- Four speech problems are usually created by cleft lip and palate deformity
- Retardation of consonant sounds
- Hypernasality is usual in the patient with a cleft of the soft palate and may remain after surgical correction.
- Articulation problems
- Hearing problems

7-Associated Anomalies

- club-foot, neurologic disturbances, heart diseases



TREATMENT OF CLEFT LIP AND PALATE

The aim of treatment of cleft lip and palate

- correct the cleft and associated problems surgically and thus hide the anomaly so that patients can lead normal lives.
- Aim of lip repair: (1) Restore esthetic and function

- Aim of cleft palate repair are (1) better palatal and pharyngeal muscle development once repaired, (2) ease of feeding, (3) better development of phonation skills, (4) better auditory tube function, (5) better hygiene when the oral and nasal partition is competent, and (6) improved psychologic state for parents and baby

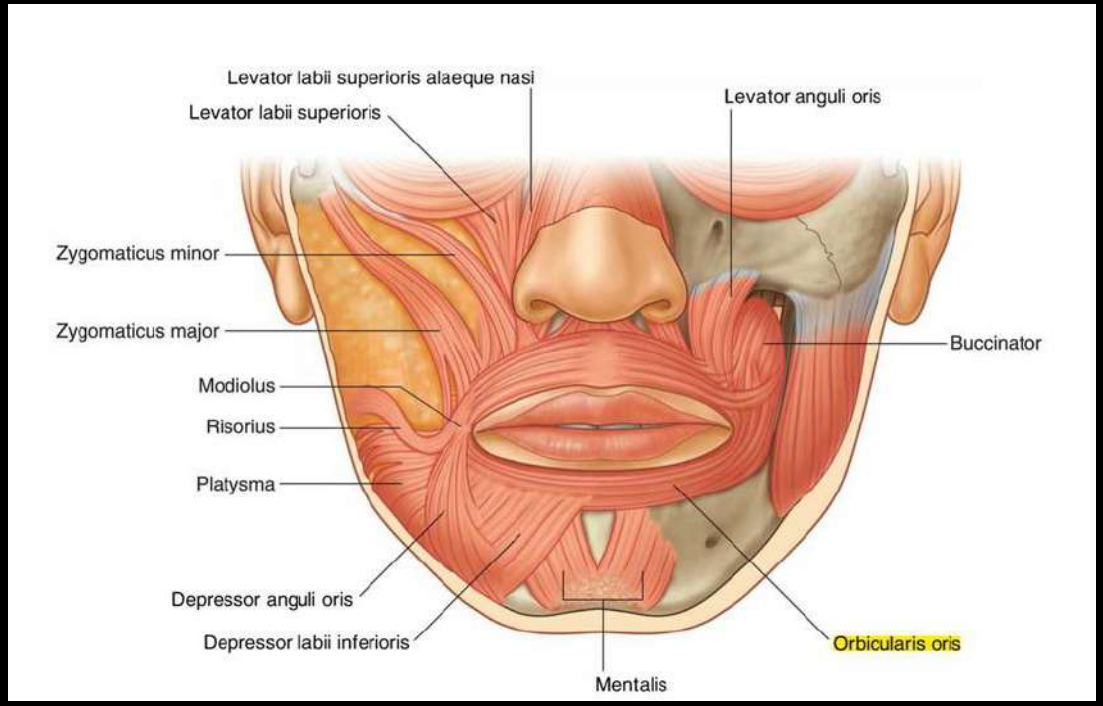
Time of repair

Table 42-1 Staged Reconstruction of Cleft Lip and Palate Deformities

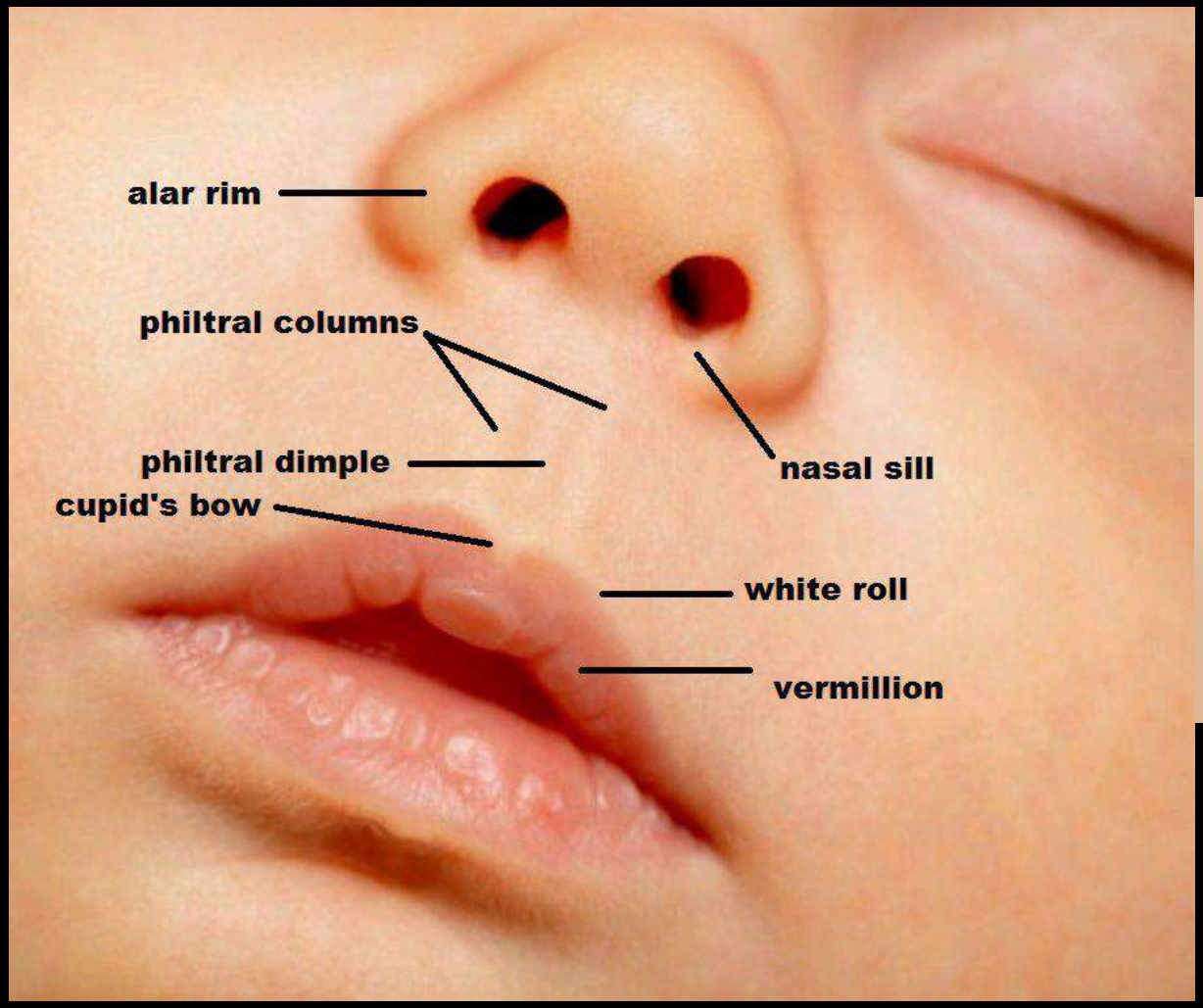
Procedure	*Timing*
Cleft lip repair	After 10 weeks
Cleft palate repair	9–18 months
Pharyngeal flap or pharyngoplasty	3–5 years or later based on speech development
Maxillary/alveolar reconstruction with bone grafting	6–9 years based on dental development
Cleft orthognathic surgery	14–16 years in girls, 16–18 years in boys
Cleft rhinoplasty	After age 5 years but preferably at skeletal maturity; after orthognathic surgery when possible
Cleft lip revision	Anytime once initial remodeling and scar maturation is complete but best performed after age 5 years

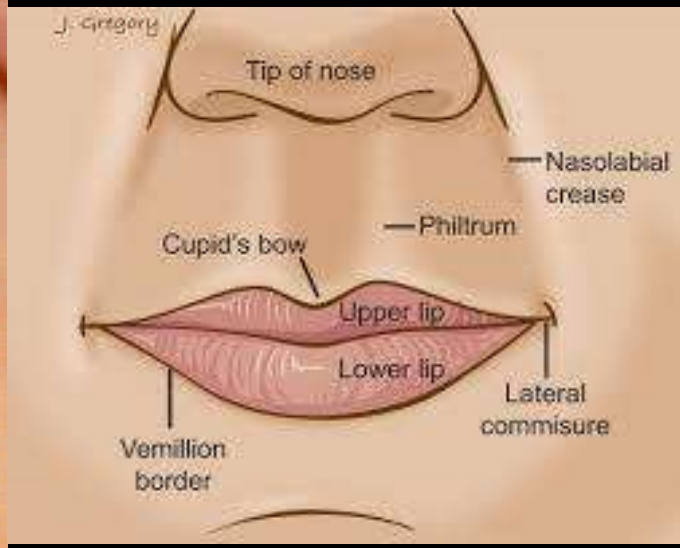
Cheilorrhaphy

- is the surgical correction of the cleft lip deformity.
- The objectives of cheilorrhaphy are twofold: (1) functional and (2) esthetic. The cheilorrhaphy should restore the functional arrangement of the orbicularis oris

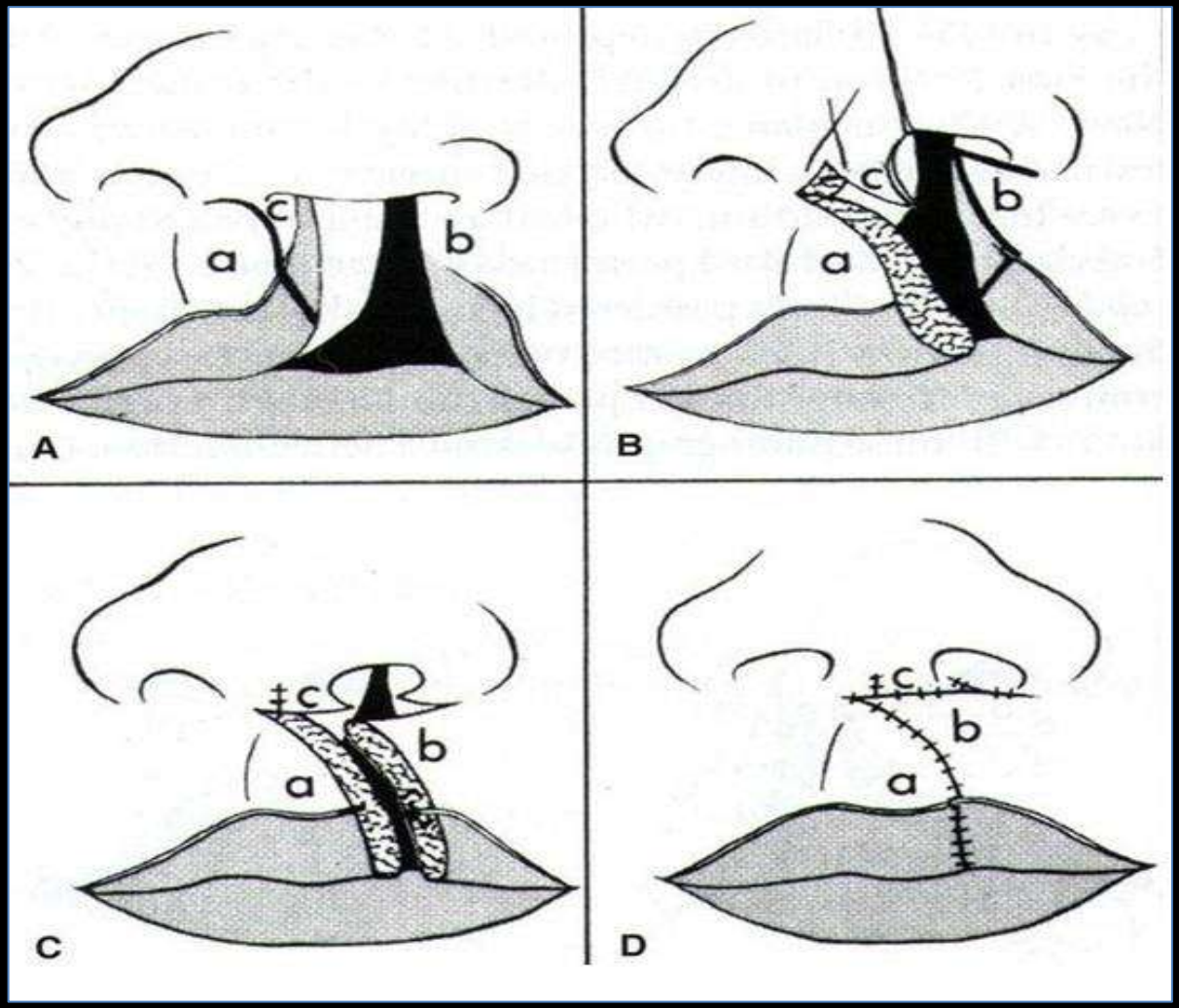


normal anatomic structures of upper lip



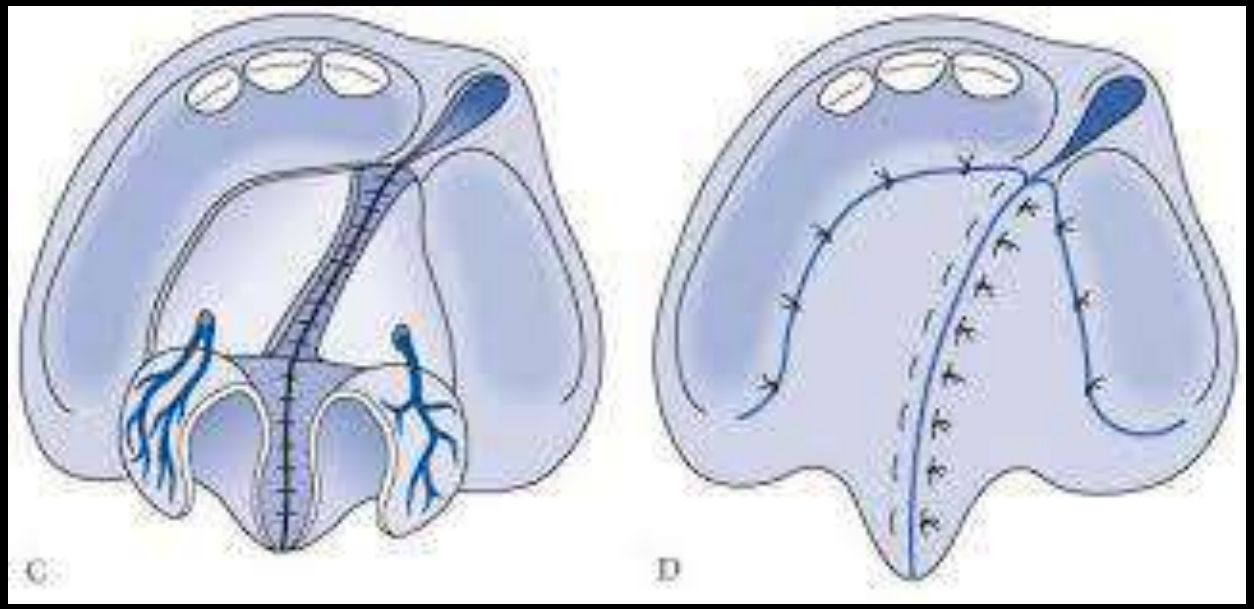


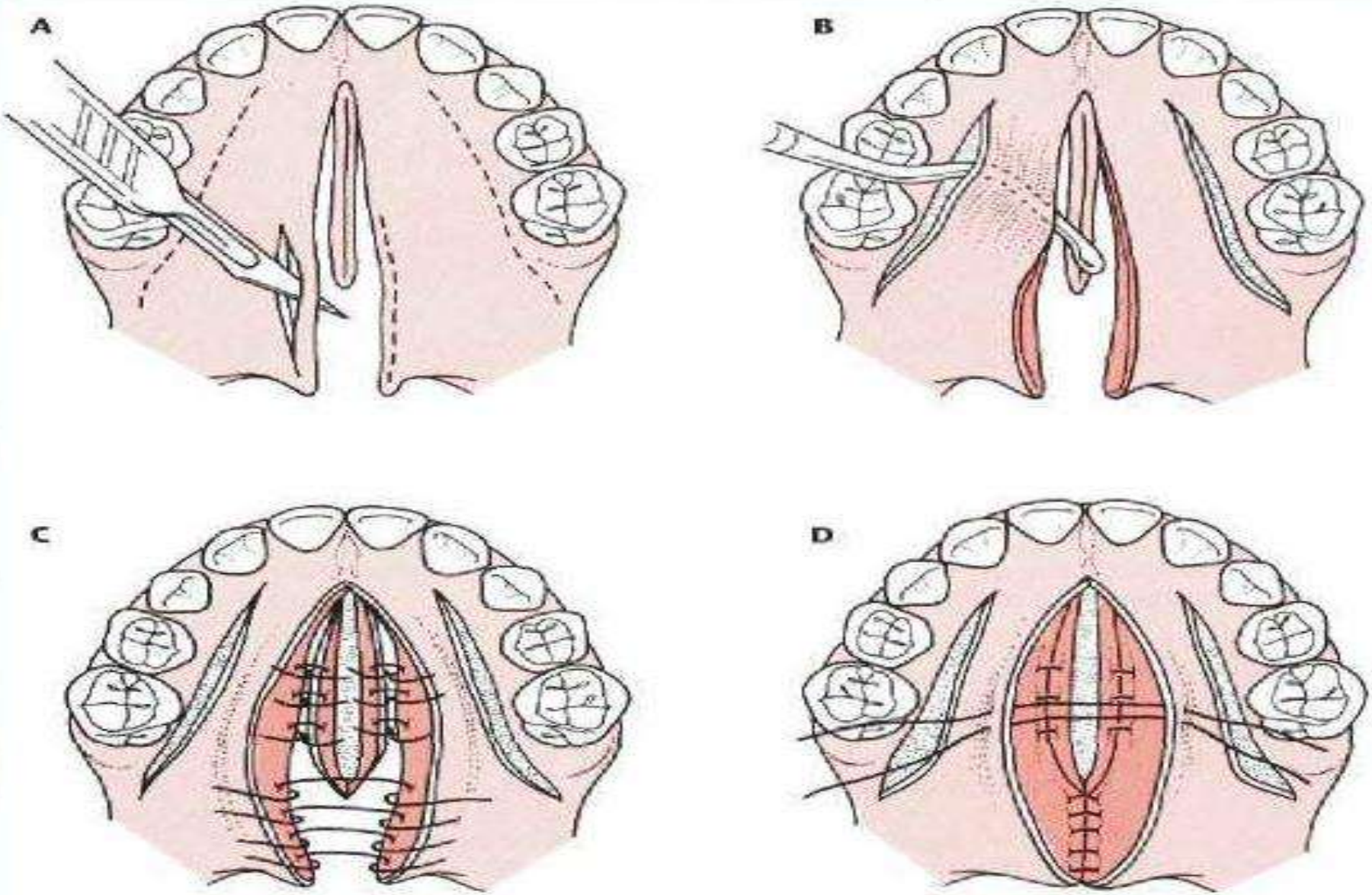
Millard (rotation-advancment)



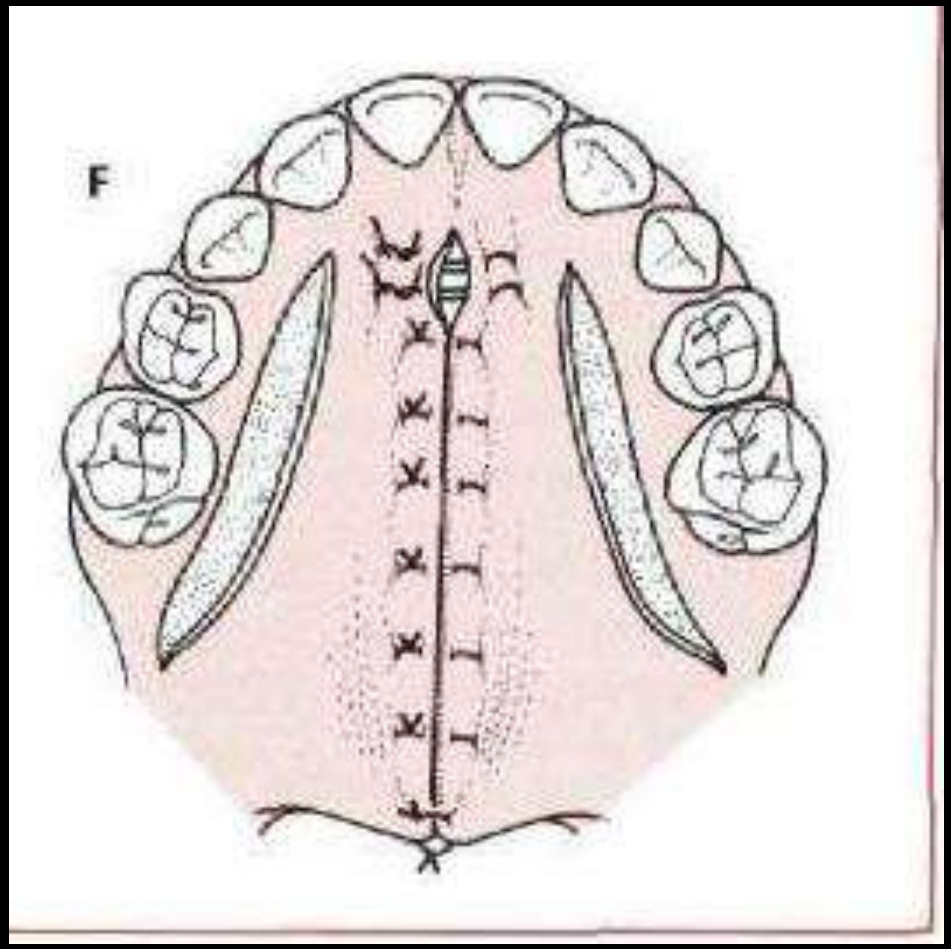
Palatorrhaphy

- Palatorrhaphy is usually performed in one operation, but occasionally it is performed in two.
- In two operations the soft palate closure (i.e., staphyloirhaphy) is usually performed first and. the hard palate closure (i.e., uranorrhaphy) is performed second




A
B
C
D

von Langenbeck operation



Alveolar Cleft Grafts

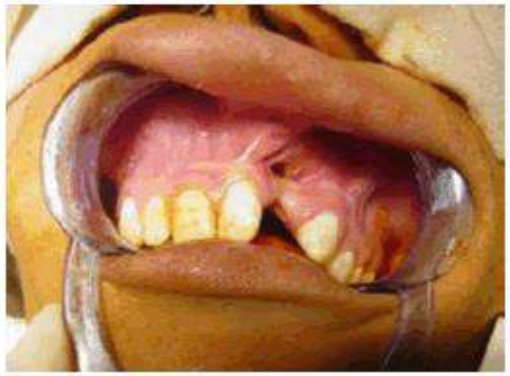
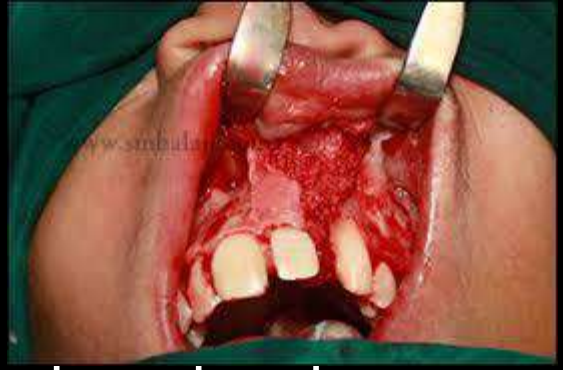


Figure 2: Preoperative intraoral view of the alveolar cleft in group 2.

- The alveolar cleft defect is usually not corrected in the original surgical correction of either the cleft lip or the cleft palate
- Five problems commonly occur in oronasal fistula

(1) oral fluids escape into the nasal cavity, (2) nasal secretion drains into the oral cavity, (3) teeth erupt into the alveolar cleft, (4) the alveolar segments, collapse, and (5) if the cleft is large, speech is adversely affected

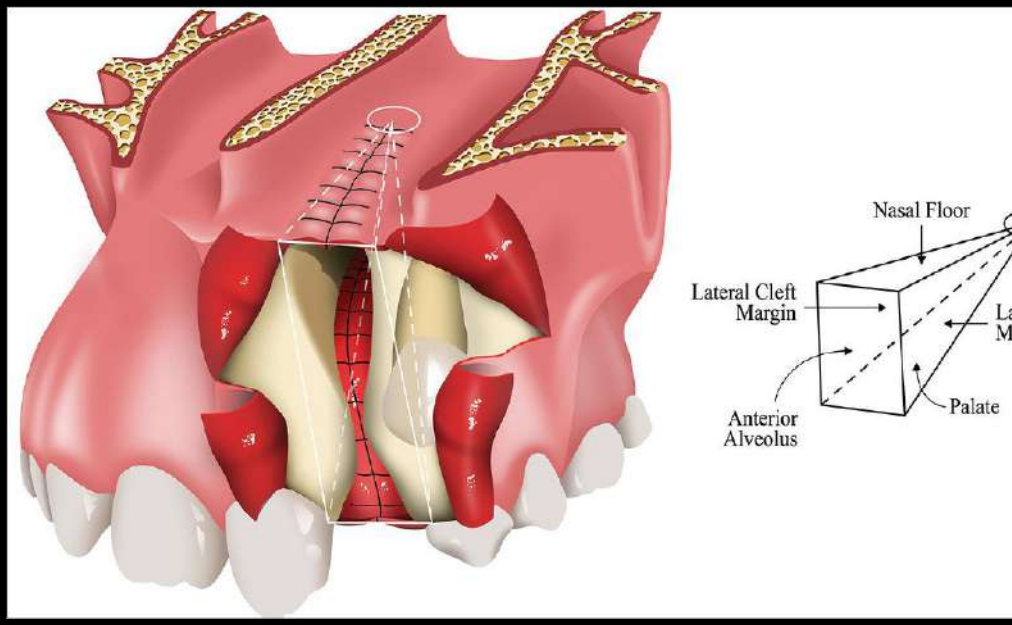
The aim of treatment



- To restore physiological continuity of the dental arch to enable oral and dental health to be maintained.
- To provide bone for stability of the dental arch, premaxilla, and adjacent teeth and to allow for eruption of unerupted permanent teeth(usually canine) or placement of dental implant.
- To allow for orthodontic alignment of the teeth
- To provide support for the lip and nose and to close a persistent oronasal fistula
- The treatment should cause minimal impairment of growth and development in the maxillofacial complex.

Timing of graft

- Between ages 7 and 10
- Ideally, when one half to two thirds of the unerupted canine root has formed



Management of alveolar cleft

1. Orthodontic expansion of the arch, why?
2. Surgery: The soft tissue incisions for alveolar cleft is performed so that flaps of nasal mucosa, palatal mucosa, and labial mucosa must all be developed and sutured in a tension-free, watertight manner.

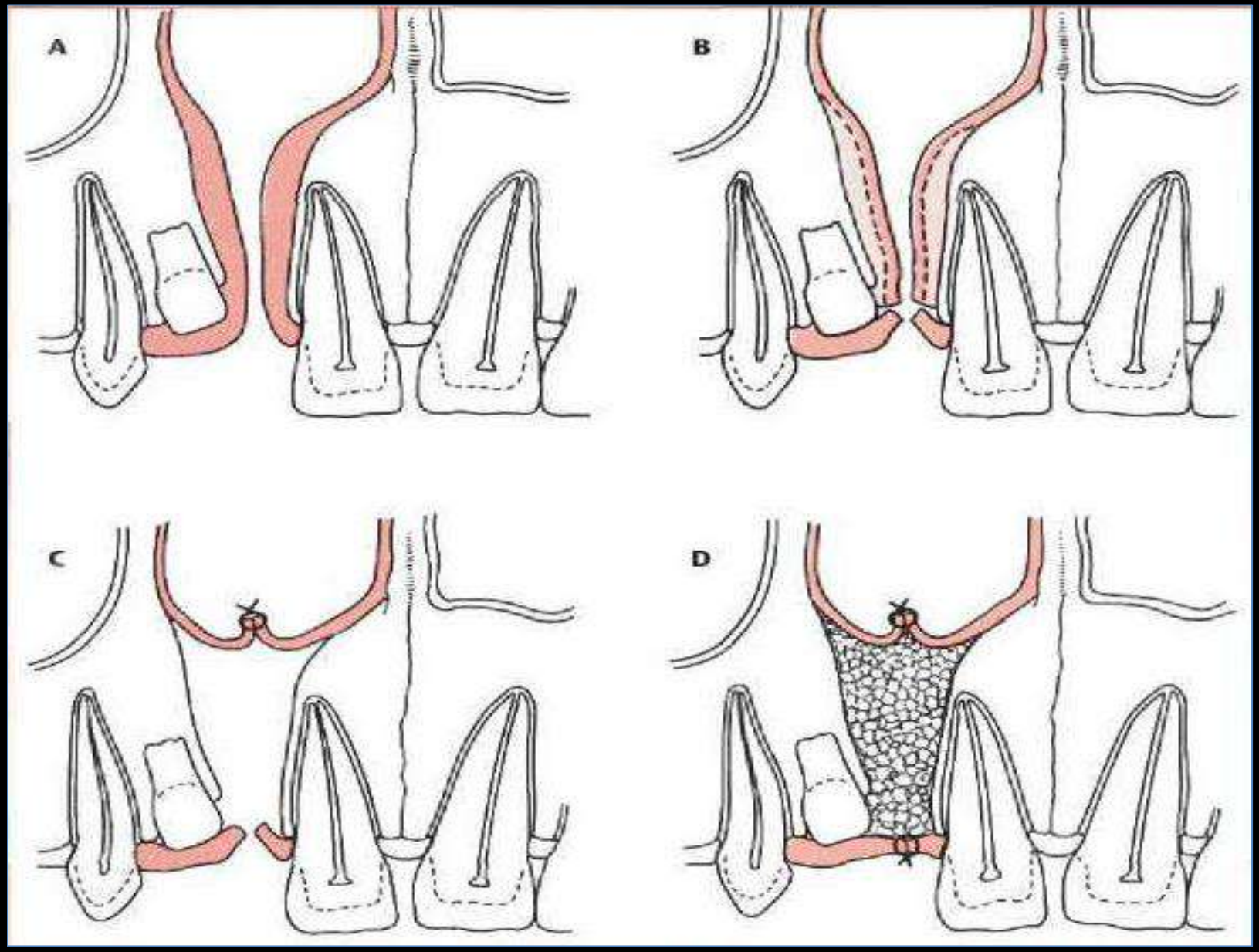
- Bone graft is used which may be autogenous cancellous or allogenic
- 3 layers in manner : nasal mucosa

↓

bone graft

↓

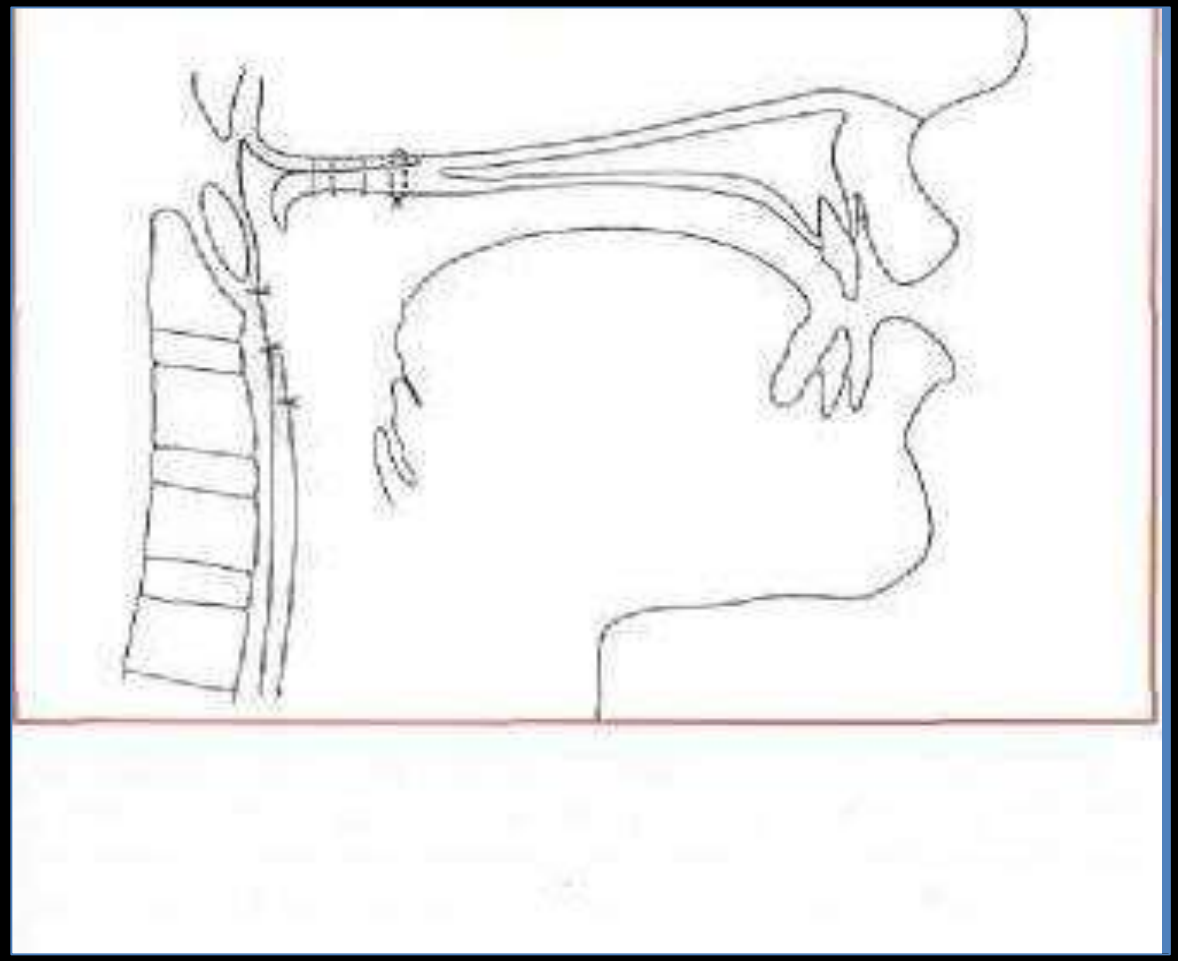
oral mucosa


A
B
C
D

Correction of Maxillomandibular Disharmonies

- The individual with a cleft deformity will usually exhibit maxillary retrusion and a transverse maxillary constriction resulting from the cicatricial contraction of previous surgeries.
- In these cases orthognathic surgery procedures are indicated to correct the underlying skeletal malrelationships (total maxillary osteotomies are necessary to advance and sometimes widen the maxilla).

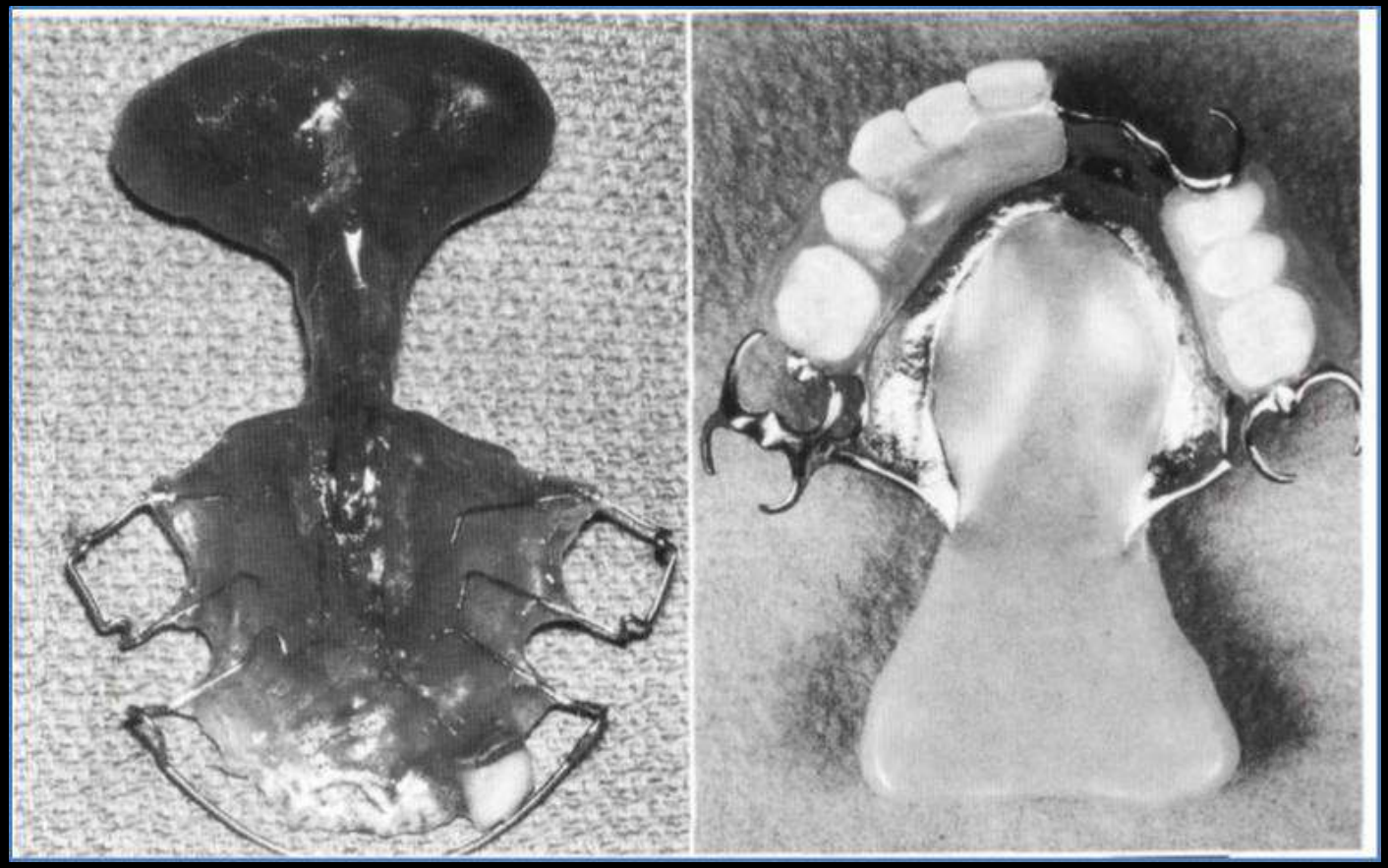
Other procedures commonly used in cleft patient is pharyngeal flap



Role of dentist in management of cleft patients

- it behooves the dentist to be aware of the overall treatment plan formulated by the cleft team for the patient‘ management.
- Avoid performance of any irreversible or costly procedures on teeth that may be charted for extraction in the future e.g. bridge for missing lateral incisor, Exo of supernumerary
- Oral hygienic measures may be difficult at cleft area .these individuals may need more frequent prophylaxis and special oral hygienic instructions with careful reinforcement
- Prosthetic Speech Aid Appliances

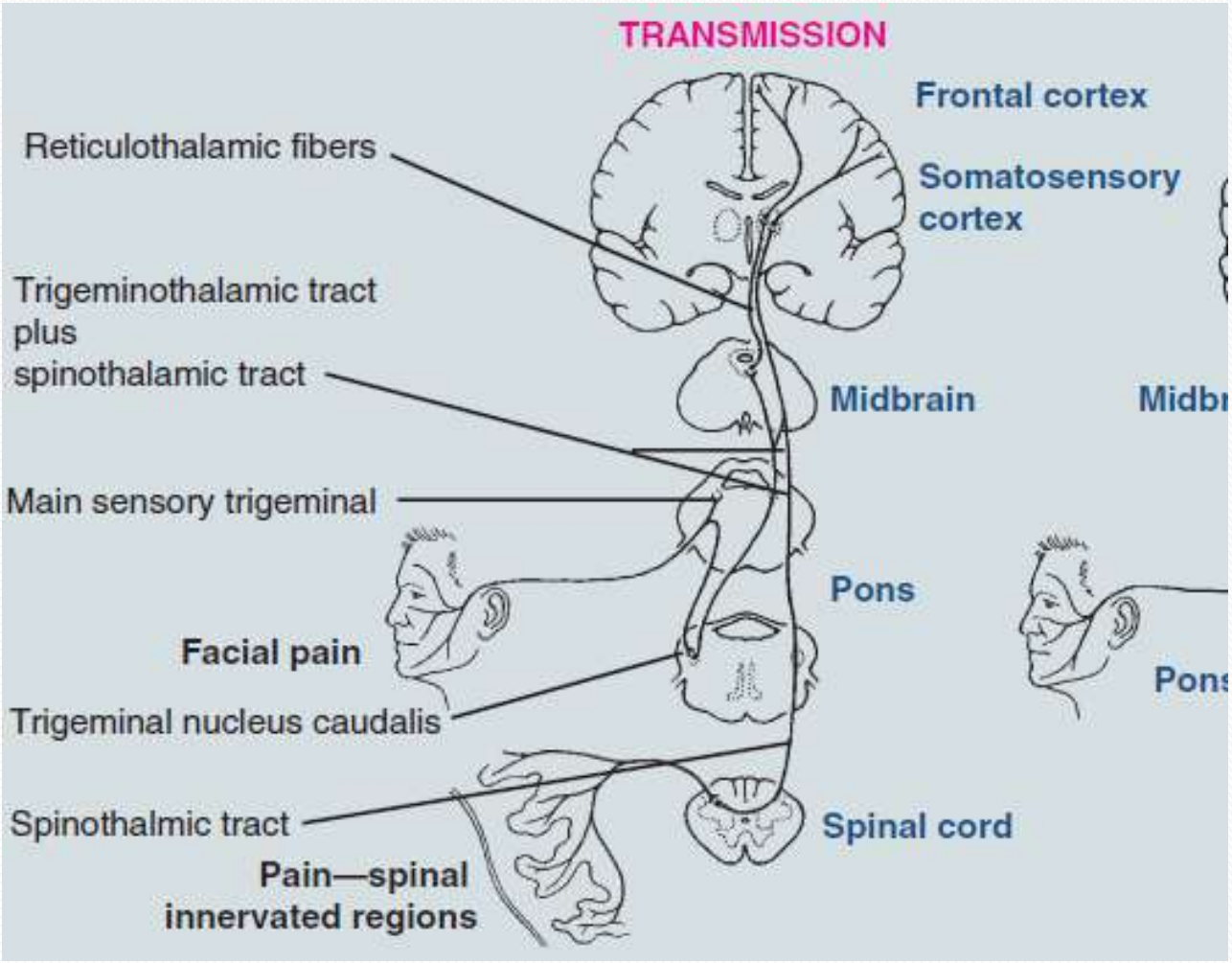
Prosthetic speech aid appliances




Thank you

Facial Pain

Pain Neurophysiology

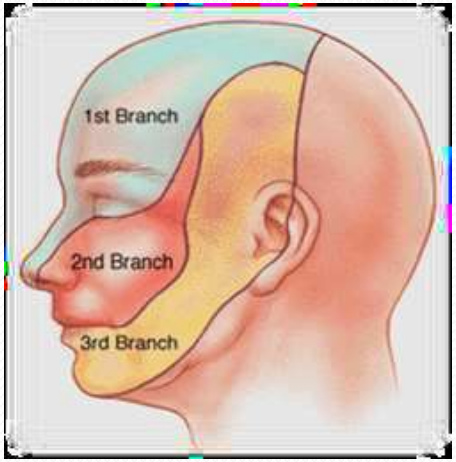


Category	Type of pain
Local	Dental (include pulpitis, dentin hypersensitivity, periapical periodontitis, cracked tooth syndrome)
	Gingival (e.g. primary herpetic gingivostomatitis, ulcerative gingivitis)
	Mucosal (e.g. ulceration)
	Salivary gland (e.g. sialadenitis)
	Temporomandibular joint (dysfunction and others)
	Maxillary sinuses (sinusitis, malignancy)
	Ear disease (otitis media, neoplasm)
Neurological	Trigeminal neuralgia
	Glossopharyngeal neuralgia
	Postherpetic neuralgia
	Ramsay- hunt syndrome
Vascular	Giant cell arteritis
	Migraine and variants
	Cluster headache, Tension headache
Psychogenic	Atypical facial pain
	Atypical odontalgia
	Burning mouth syndrome
Referred pain	Cardiac pain

Trigeminal neuralgia

- Trigeminal neuralgia is characterized by unilateral severe, brief, stabbing , recurrent pain in the distribution of one or more branches of the fifth cranial nerve.
- Features :

1. sharp, electric shock-like pain in the face or mouth
2. lasting for brief periods of seconds to 1 minute
3. Usually a "trigger zone" is present
4. Trigger factors present
5. No clinical neurological deficit



Characteristic features

- Incidence: 8 : 1,00,000
- Age: 5th – 6th decade of life
- Sex: Female > male
- Affliction for side: Right > left
- Division of trigeminal nerve involvement:V3 > V2 > V1

Trigger zones and trigger points

V2- skin of upper lip, ala, cheeks & upper gums

V3- lower lip, teeth or gums of lower jaw

V1- supraorbital ridge of affected side

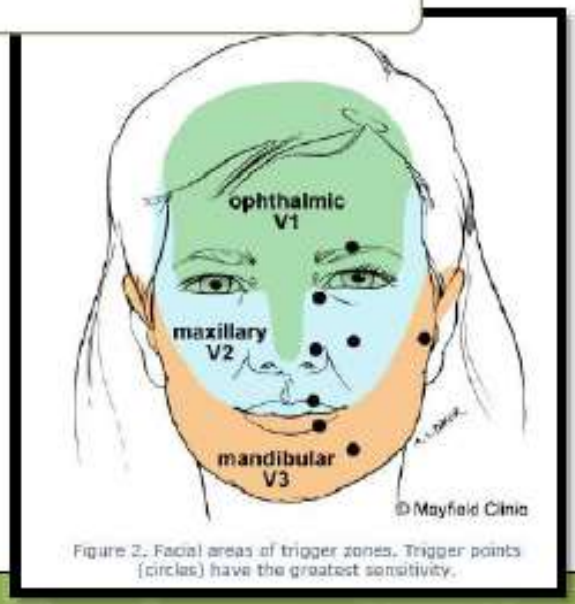
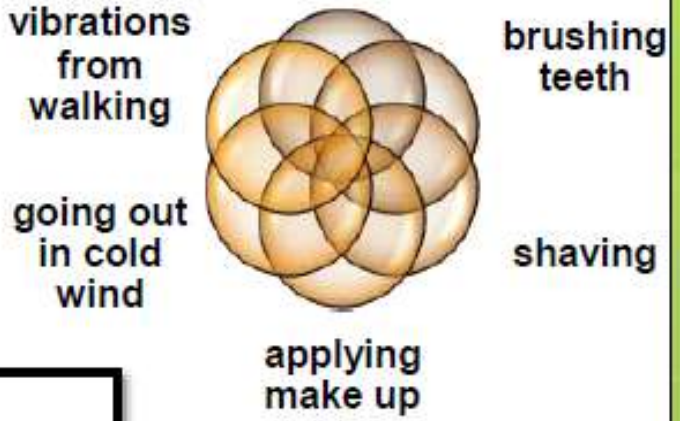


Figure 2. Facial areas of trigger zones. Trigger points (circles) have the greatest sensitivity.

Trigger factors

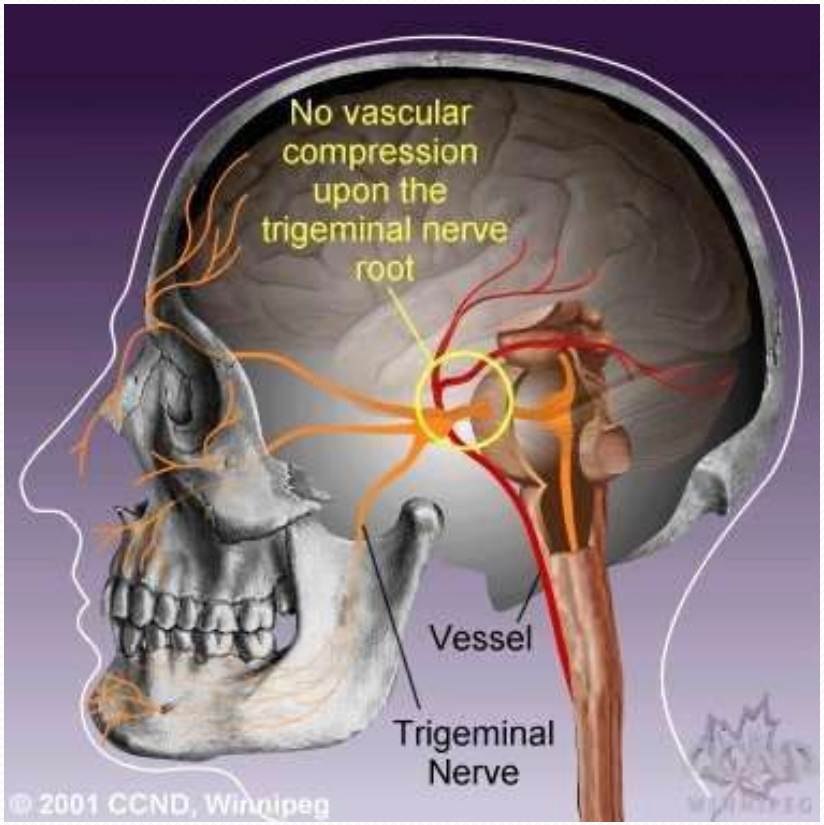


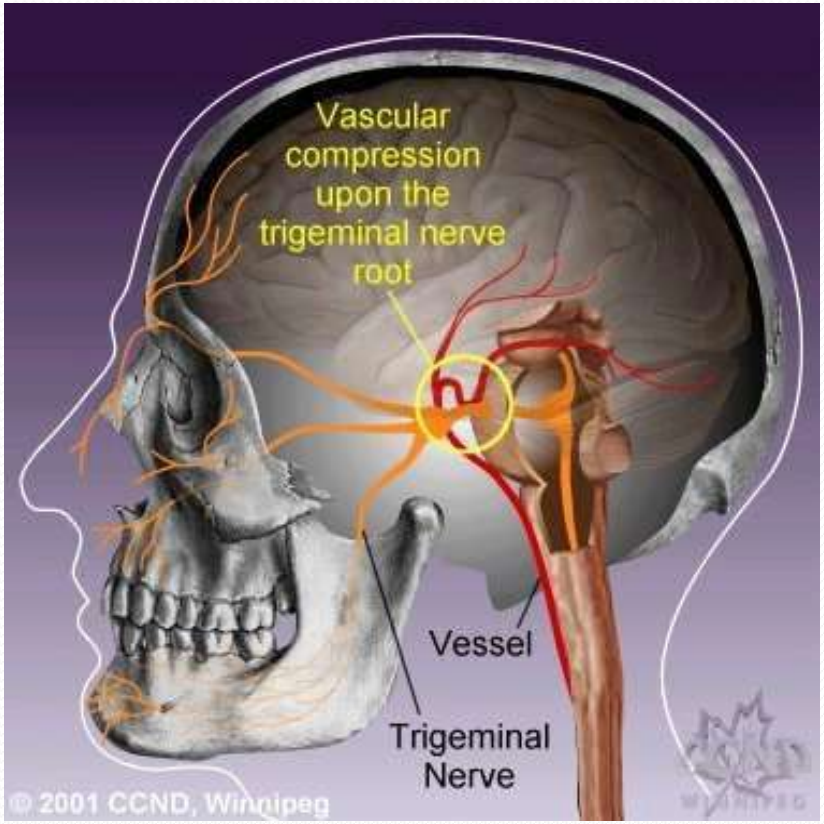
WHITE AND SWEET DIAGNOSTIC CRITERIA FOR TRIGEMINAL NEURALGIA

- Pain- paroxysmal
- Pain- provoked by light touch to trigger zones
- Pain- confined to trigeminal distribution
- Pain- unilateral
- Clinical sensory examination- normal

- Nearly all cases of typical trigeminal neuralgia are caused by blood vessel compressing the trigeminal nerve root. This in turn precipitates ectopic or hyperactive discharge of the nerve.

- Ix: MRI (detect any intracranial lesion).


No vascular compression upon the trigeminal nerve root
Vessel
Trigeminal Nerve
© 2001 CCND, Winnipeg


Vascular compression upon the trigeminal nerve root
Vessel
Trigeminal Nerve
© 2001 CCND, Winnipeg

Management

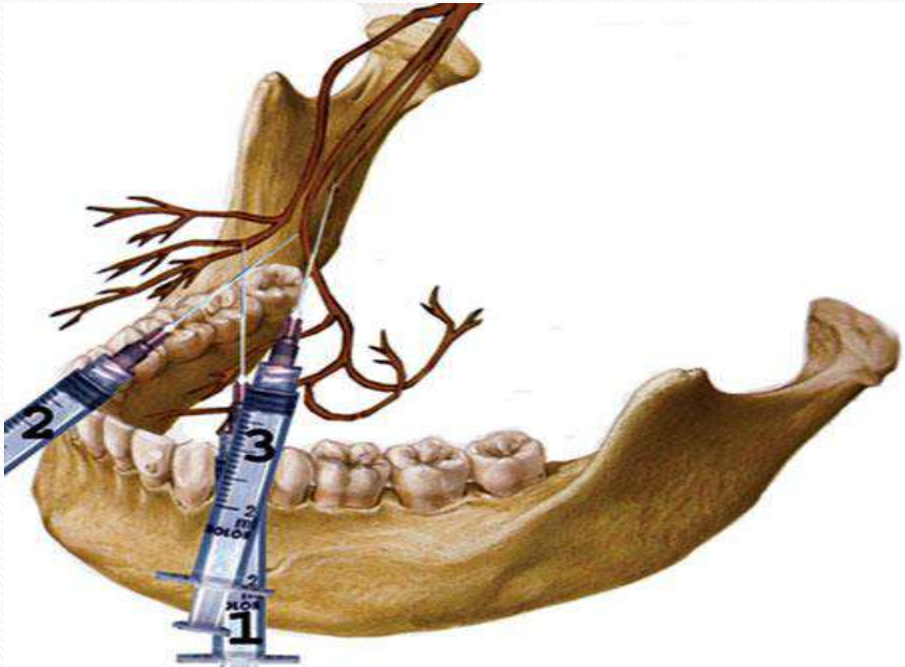
- Medical : carbamazepine (Tegretol)
  - 200 mg/ day for the first week
  - 400 mg/ day for 2nd week and so on till pain controlled.
  - Not exceed 1200 mg/day

Drug	Initial Dose (mg)	Target or Maximal Dose (mg)*	Dose Increase (Titration)*	Schedule
Carbamazepine	100-200	1200	100-200 mg/2 days	×3-4/day
Gabapentin	300	900-2400	300 mg/1-2 days	×3/day

Surgical

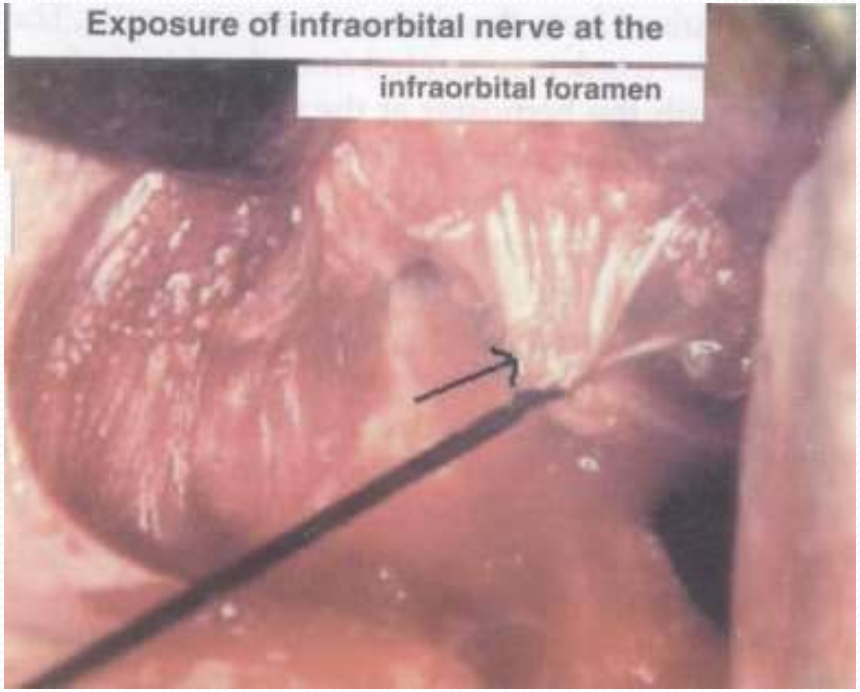
1) PERIPHERAL INJECTION:

It has been known that injection of destructive substance into peripheral branches of the trigeminal nerve, produces anaesthesia in the trigger zones or in areas of distribution of spontaneous pain



2.PERIPHERAL NEURECTOMY (NERVE AVULSION)

- involving the severing or removal of a nerve..
- Performed commonly on infraorbital, inferior alveolar, mental nerves.



3. CRYOTHERAPHY

- Direct application of cryotheraphy probe at temperatures colder than -60 C are known to produce Wallerian degeneration without destroying the nerve sheath itself.

GASSERIAN GANGLION PROCEDURES: (rhizotomy)

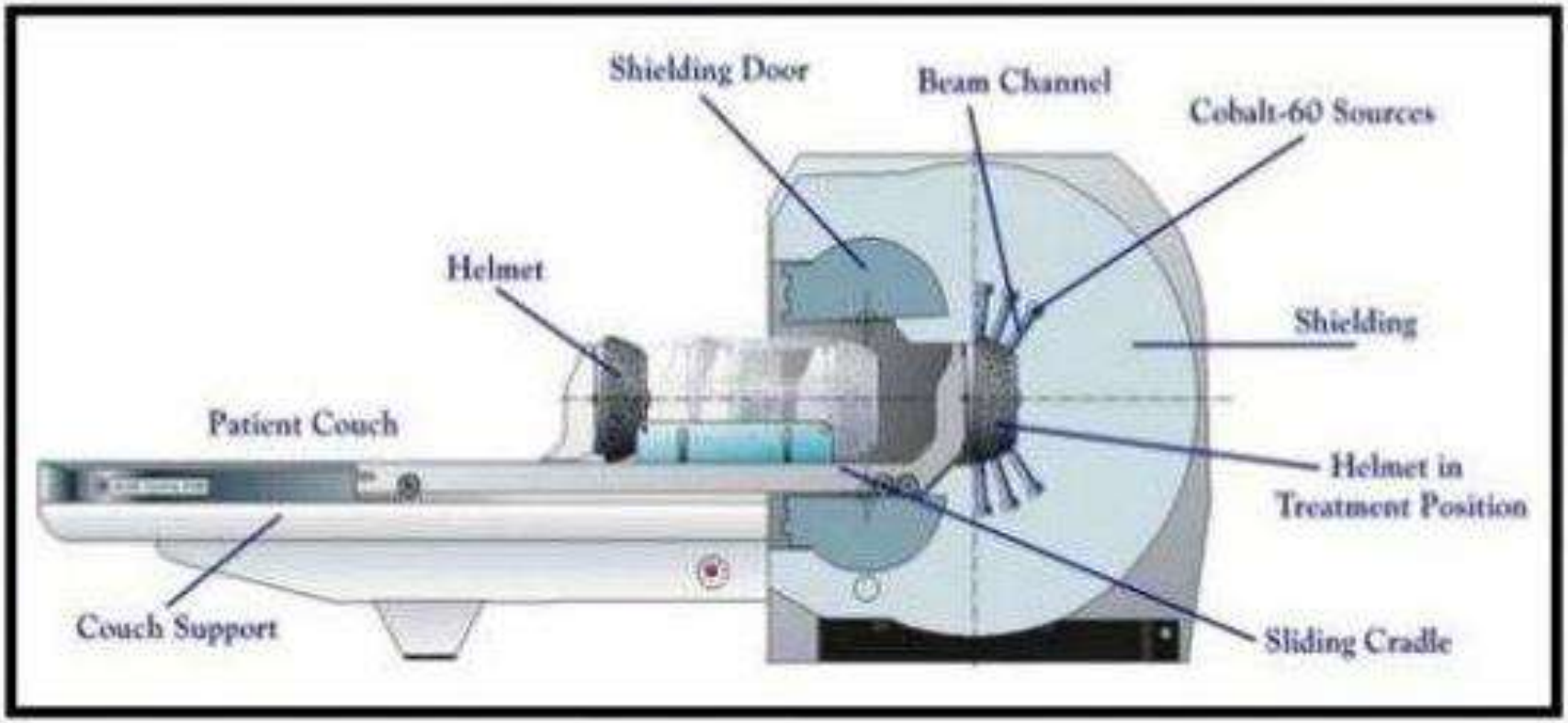
- a surgical procedure to sever trigeminal nerve roots.

1. Percutaneous rhizotomy
2. Thermocoagulation
3. **balloon compression**

OPEN PROCEDURES (INTRACRANIAL PROCEDURES):

- Microvascular decompression of the trigeminal nerve sensory root.
- Trigeminal root section:

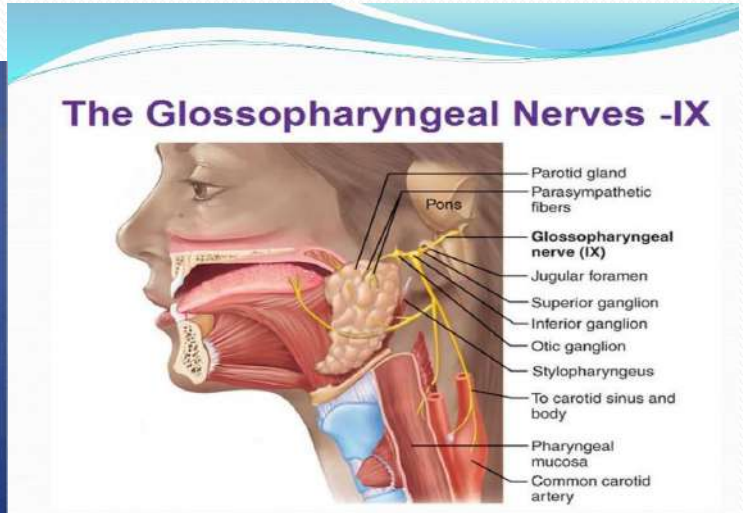
- The Gamma Knife is not a knife ; it is a complex machine that uses **Cobalt – 60** as the energy source in the radiosurgery



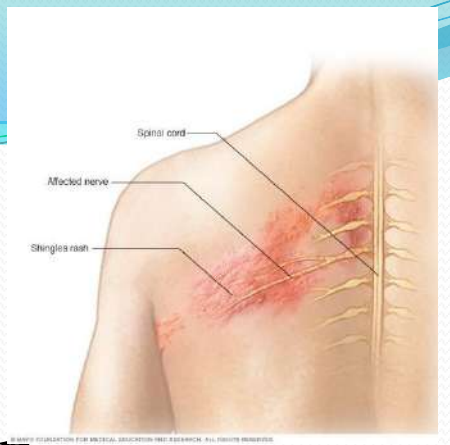
Gamma knife radiosurgery machine

Glossopharyngeal neuralgia

- characterized by lancinating pain in oropharynx (tongue base, lateral pharyngeal wall) or neck,
- sometimes triggered by swallowing, coughing, or talking.
- The pain is usually unilateral and may radiate to ear and mouth, lasting weeks to months. Syncope can be a feature due to vagal stimulation. The management of GN typically parallels that of TN.

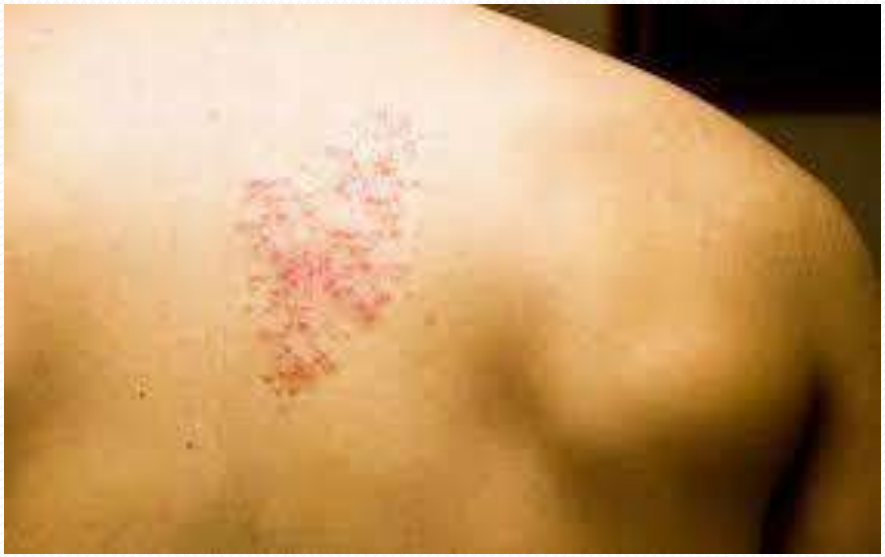


Postherpetic neuralgia



- potential sequelae of herpes zoster infection
- PHN occurs after reactivation of varicella zoster virus, which can lay dormant in the ganglia of a peripheral nerve.
- Most commonly this is a thoracic nerve, but approximately 10% to 15% of the time the trigeminal nerve is involved, with the V1 dermatome affected in approximately 80% of cases
- V1 dermatome is outlined by rash. In the V2 or V3 distribution, both intraoral and cutaneous expression is commonly seen. The acute phase is quite painful.

PHN



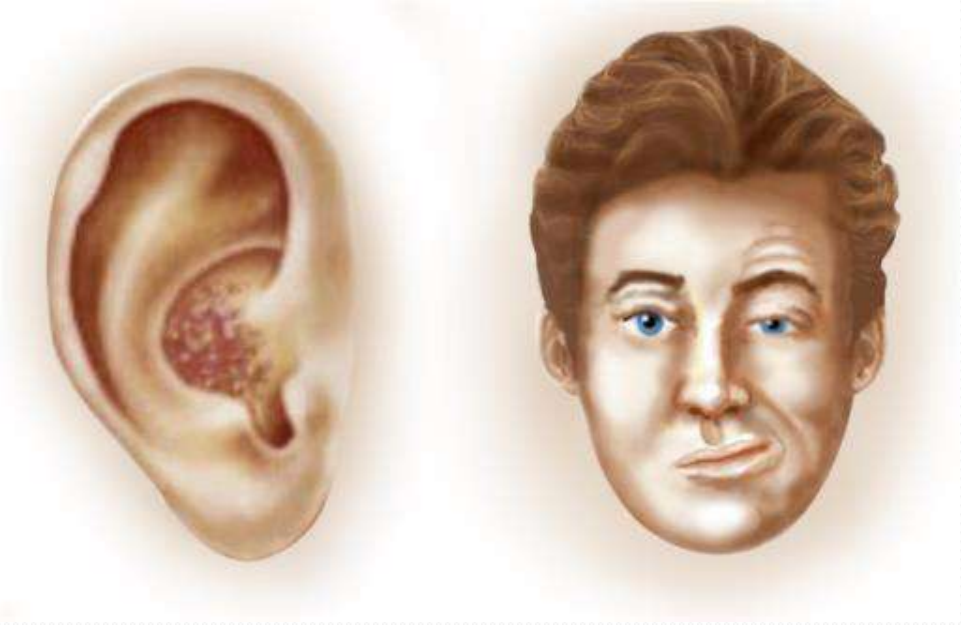
Treatment

- Most importantly, preventive treatment of PHN with antivirals, analgesics and frequently corticosteroids very early after rash presentation can significantly reduce the expression of PHN.
- Treatment:

TAD	**ex: Nortriptyline**
Anticonvulsants	Gabapentine
Local anesthesia	Lidocaine
Surgery	Destruction of peripheral nerve

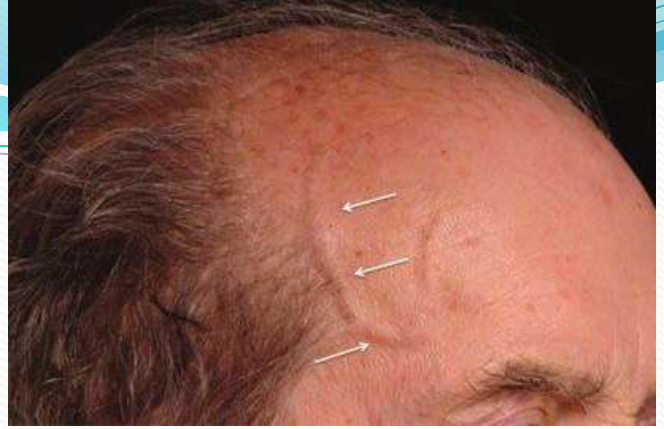
Ramsay Hunt Syndrome

- a herpes zoster infection of the sensory and motor branches of the facial nerve (VII) and in some cases the auditory nerve (VIII). Symptoms include facial paralysis, vertigo, deafness, and herpetic eruption in the external auditory meatus



Vascular facial pain

Temporal Arteritis



- an inflammation (i.e., vasculitis) of the cranial arterial tree that can affect any or all vessels of the aortic arch and its branches.
- Dull aching or throbbing temporal or head pain is a common complaint affecting 70% of patients
- Jaw claudication may lead the patient to visit the dentist for diagnosis (may resemble TMDs)
- Ix.: elevated ESR(over 100) +CPR+ biopsy
- Treatment: dose corticosteroids

Any older patient reporting jaw or face pain not obviously of odontogenic origin and whose symptoms suggest temporal arteritis should be referred for an erythrocyte sedimentation rate (ESR) test

Chronic headache

- When headaches recur regularly, the majority will be diagnosed as one of the primary (no other cause) headaches: migraine, tension-type headache, or cluster headache
- Although most headaches are centered in the Orbits and temples, many may present in the lower half of the face, teeth, or jaws.

Migraine

• BOX 30.5 International Headache Society Criteria for Migraine Headache Without Aura

A. At least five attacks fulfilling criteria B through D
B. Headache attacks lasting 4–72 h (untreated or unsuccessfully treated)
C. Headache has two or more of the following characteristics:
    1. Unilateral location
    2. Pulsating quality
    3. Moderate or severe pain intensity
    4. Aggravation by or causing avoidance of routine physical activity (e.g., walking, climbing stairs)
D. During headache ≥1 of the following:
    1. nausea and/or vomiting
    2. photophobia and phonophobia
E. Not attributed to another disorder

Prevention

- Preventive treatment is directed at normalizing neurotransmitter imbalance with antidepressants, anticonvulsants, beta-blockers

Treatment

- Treatment of acute attacks is with the "triptans" (e.g., sumatriptan 2.5mg and repeated 2 hrs after), ergots (ergotamine) (NSAIDs), opioid analgesics, antiemetics

Cluster Headache

Common Cluster Headache Features

Sex: Mainly male
Frequency: Up to 8 per day
Quality: Throbbing/stabbing
Intensity: Severe

BOX 29-9

IHS Criteria for Cluster Headache

A. Severe unilateral orbital, supraorbital, or temporal pain (or a combination) lasting 15 to 180 minutes *(Note: Frequently in posterior maxillary dentoalveolar region as well)*
B. At least one of the following on the headache side:
   - Conjunctival injection
   - Facial sweating
   - Lacrimation
   - Miosis
   - Nasal congestion
   - Ptosis
   - Rhinorrhea
   - Eyelid edema
C. No evidence of organic disease

- Dentists must be aware that cluster headache frequently produces pain in the posterior maxilla, mimicking severe dentoalveolar pain in the posterior maxillary teeth
- Unnecessary dental therapy is, unfortunately, common.
- Common features can distinguish a toothache resulting from cluster headache from a toothache produced by a dental problem:

A. Rapid emergence and discontinuation of symptoms unlike typical toothache
B. Toothache precipitated by alcohol ingestion
C. Toothache accompanied by unilateral rhinorrhea or other parasympathetic symptoms
D. Toothache that occurs with periodicity

Tension headache

IHS Criteria for Episodic Tension-Type Headache

A. Headache pain accompanied by two of the following symptoms:
- Pressing/tightening (nonpulsating) quality
- Mild-to-moderate intensity
- Bilateral location
- Not aggravated by routine physical activity

B. Headache pain accompanied by both of the following symptoms:
- No nausea or vomiting
- Photophobia and phonophobia not present or only one present

C. Fewer than 15 days per month with headache
(Note: If more than 15 days/month, termed chronic tension-type headache)

D. No evidence of organic disease

Psychogenic facial pain

Atypical Facial Pain

- AFP is persistent pain in maxillofacial region that doesn't fit any diagnostic criteria of any orofacial pain and has no identifable cause.
- Diagnosis occurs by exclusion other diseases

❖T3: Trauma, tumor, Tmj disorder

❖I3: Infection, inflammation, impingement

❖MN: Myofacial pain, neuralgia

❖ACH: Allergy, cracked tooth syndrome, headache

Features

- Mostly occurs in 4-6th decade of life
- frequently involves most quadrant and extend to neck and temple. The patient has difficulty to describe the pain as continuous, dull ache with intermittent severe episodes. The pain may be bilateral and often present for several years
- Pain is initiated or enhanced by oral surgery.
- Analgesics are ineffective
- Almost all patients with AFP complain of other symptoms including headache, back pain, dermatitis, IBS.

Management

- Psychogenic therapy, behavior modification
- Opoids (codeine, fentanyl..) but effectiveness decrease with time
- Tricyclic antidepressants such as amitriptyline, nortriptyline
- Surgical destruction of portion of CN V nerve or sympathetic nerve block

Atypical odontalgia

- pain in a tooth or site of dental extraction in the absence of clinical or radiographical evidence of pathological dental condition.
- Its most common in the 5^{th} decade with female bias. Molar and premolar sites are involved more frequently, and maxillary teeth are more likely.

Management

The treatment of atypical odontalgia remains unsatisfactory. The treatment includes:

- Topical application of capsaicin (0.025%) and EMLA 5% (mixture of lidocaine and prilocaine)
- TAD
- Anxiolytics such as clonazepam
- Gabapentin

Burning mouth syndrome.

- It is burning and painful sensation of clinically normal mucosa.
- The tongue is the most frequently involved site. The onset is usually abrupt and spontaneous starts at morning and increase in intensity to reach maximum intensity at evening.
- The pain lessens or disappears during sleeping.
- Perceived dry mouth and altered taste is common

- Ix: CBP, Swab biopsy, hematinics
- Mx: correction of underlying cause like fungal infection + psychogenic-based therapy (TAD, DIAZEPAM, Chlorediazepoxide)

EVALUATION OF OROFACIAL PAIN PATIENT

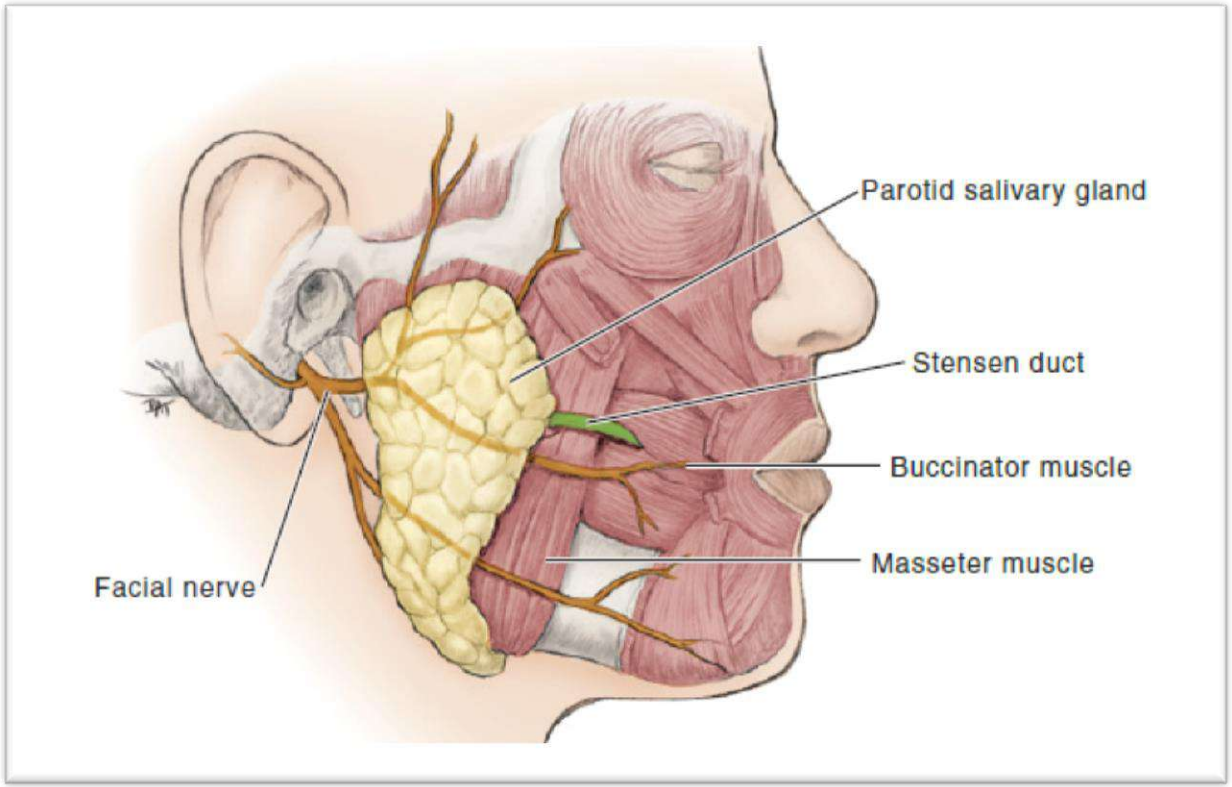
Evaluation of the dental patient who presents with jaw or face pain of nonodontogenic origin is an important skill for the dentist to master

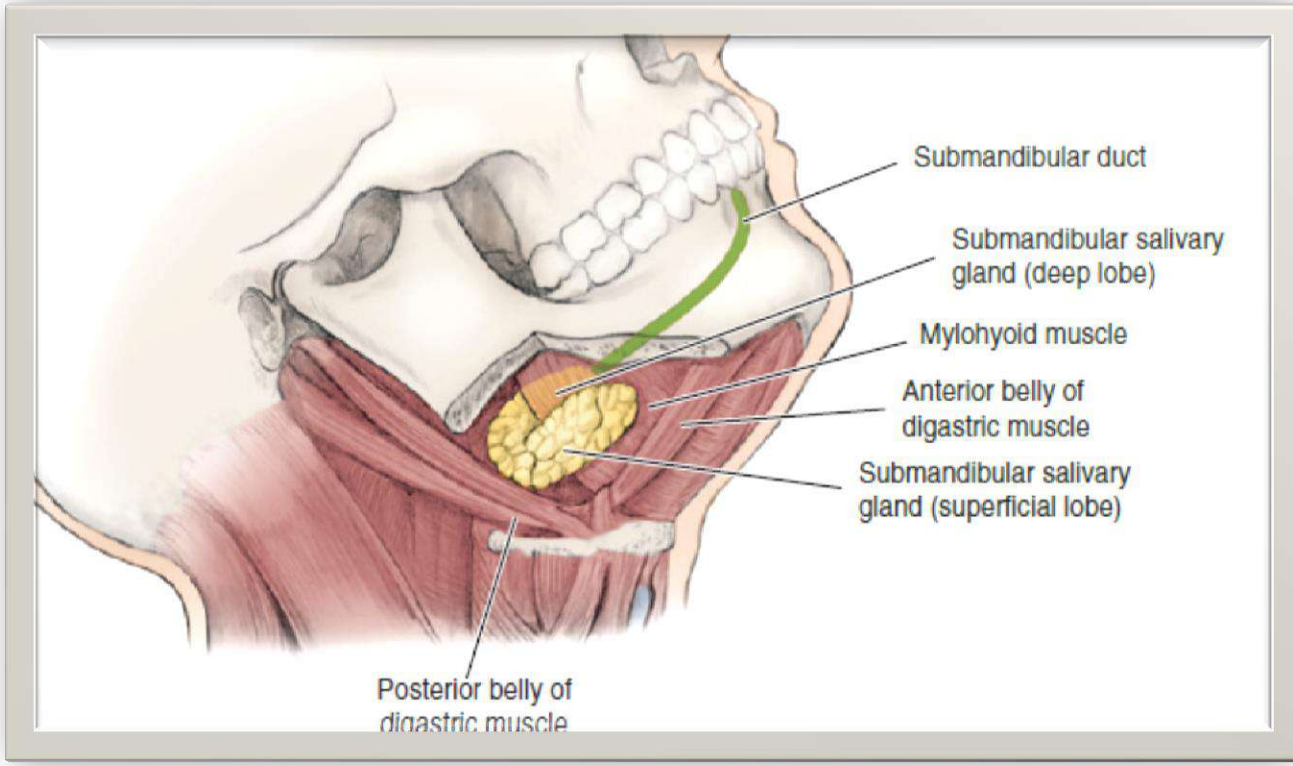
- Obtaining an accurate history is the most important component of information gathering.
- chief complaint, including the current description of pain (SOCRATE)
- a comprehensive medical and dental history
- Physical examination (head and neck, LNs, cranial nerve exam.+ LA test)
- Imaging (For most neuropathic and headache disorders, CT or MRI is recommended)

Glossary of Pain Terms

Allodynia	Pain caused by a stimulus that does not normally provoke pain
Analgesia	Absence of pain in response to stimulation that would normally be painful
Anesthesia	Absence of all sensation
Deafferentation pain	Pain caused by loss of sensory input into the central nervous system (CNS)
Dysesthesia	Unpleasant abnormal sensation, whether spontaneous or evoked *(Note: Dysesthesia includes paresthesia but not vice versa.)*
Hyperalgesia	Increased sensitivity to noxious stimulation
Hyperesthesia	Increased sensitivity to all stimulation, excluding special senses *(Note: When the sensation is painful, the terms allodynia and hyperalgesia may be appropriate.)*
Hypoalgesia	Diminished sensitivity to noxious stimulation
Hypoesthesia	Diminished sensitivity to all stimulation, excluding the special senses *(Note: When the sensation is pain, the terms hypoalgesia and analgesia may be appropriate.)*
Neuralgia	Pain in the distribution of a nerve or nerves
Neuropathy	Disturbance of function or pathologic change in a nerve
Paresthesia	Abnormal sensation, whether spontaneous or evoked

Management of Salivary Gland Disorders


Parotid salivary gland
Stensen duct
Buccinator muscle
Masseter muscle
Facial nerve


Submandibular duct
Submandibular salivary gland (deep lobe)
Mylohyoid muscle
Anterior belly of digastric muscle
Submandibular salivary gland (superficial lobe)
Posterior belly of digastric muscle

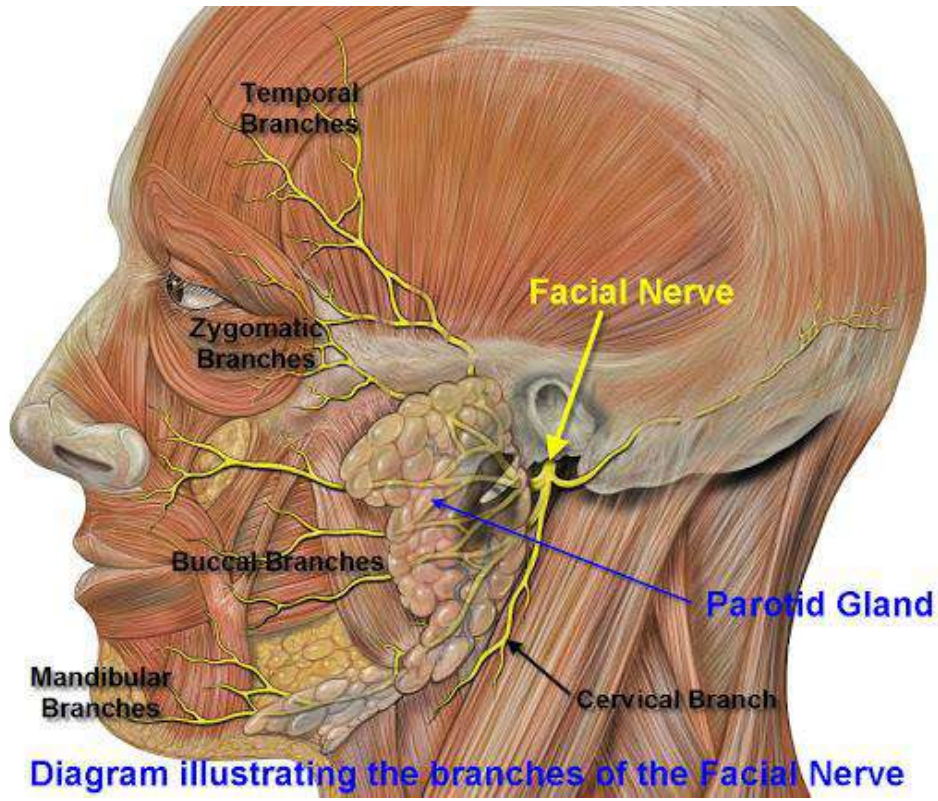
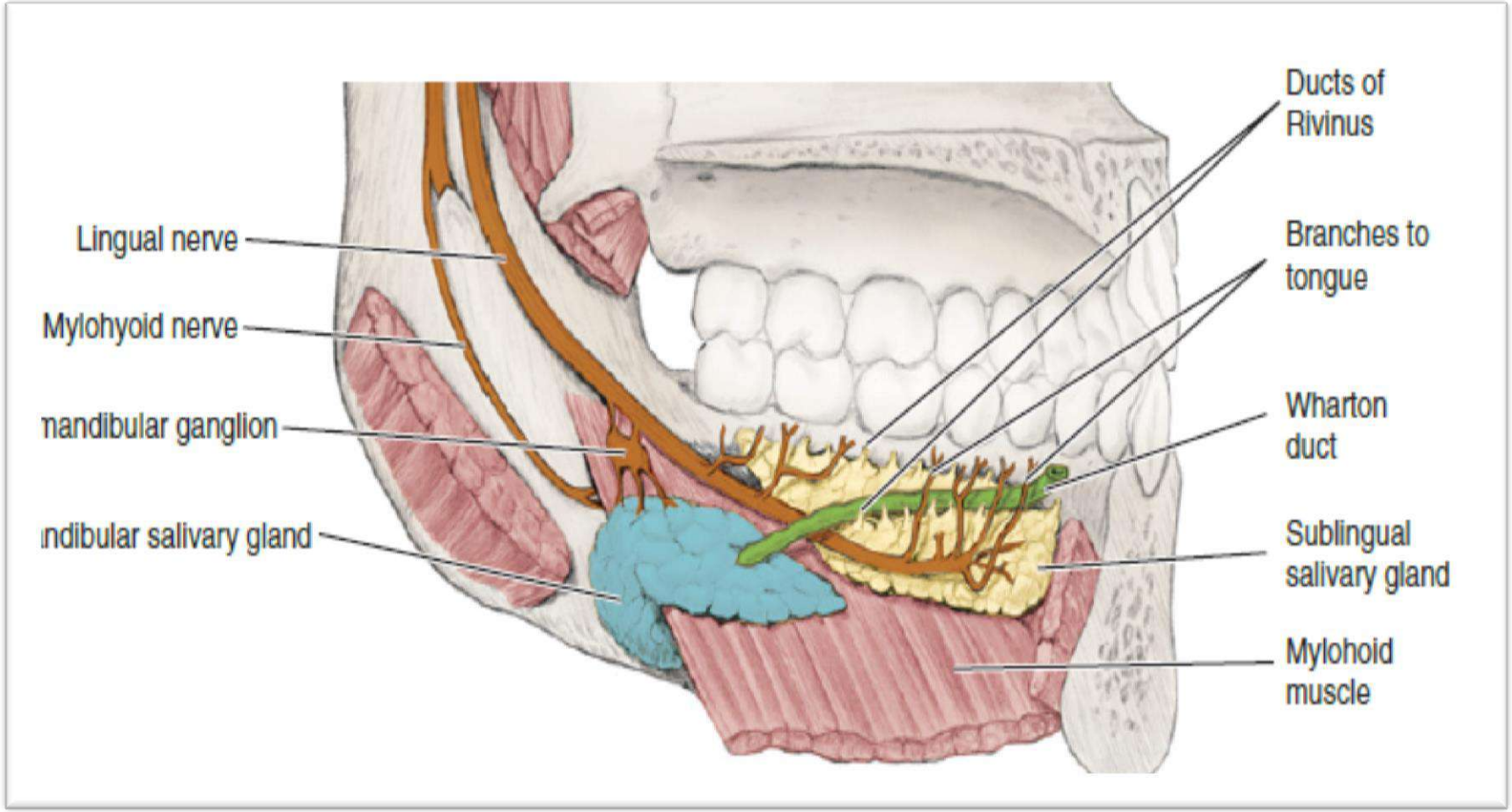


Diagram illustrating the branches of the Facial Nerve


Ducts of Rivinus
Lingual nerve
Branches to tongue
Mylohyoid nerve
nandibular ganglion
Wharton duct
ndibular salivary gland
Sublingual salivary gland
Mylohoid muscle

History and Clinical Examination

- Chief complaint(s),
- Mealtimes which may indicate an obstructive phenomenon
- if comorbidities may have contributed to the salivary gland complaints (e.g., autoimmune disease)
- whether trauma has occurred (e.g., lip biting resulting in a mucocele).

Inspection

bimanual palpation

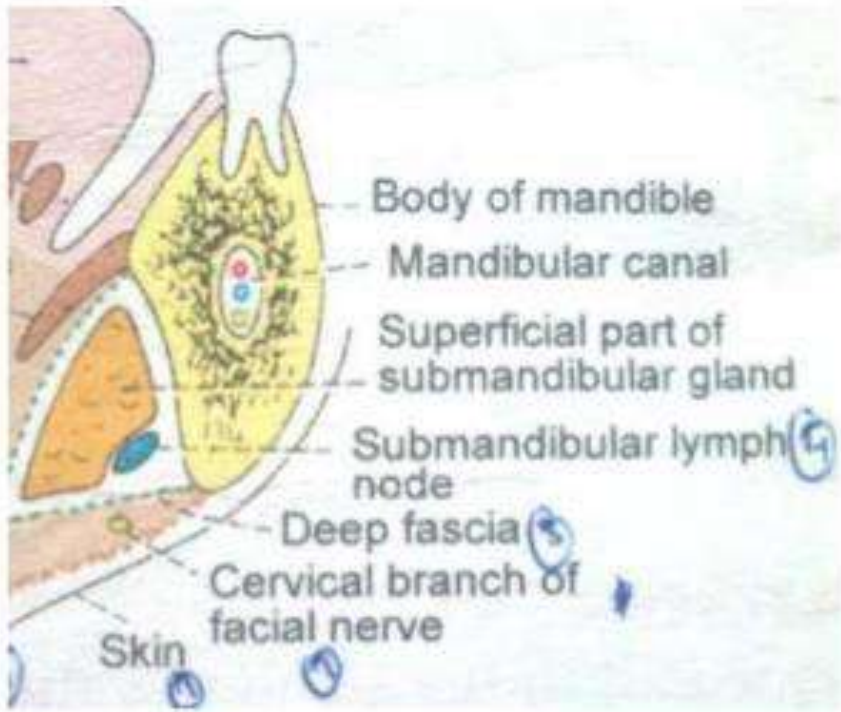
Saliva flow (reduced, pus discharge)

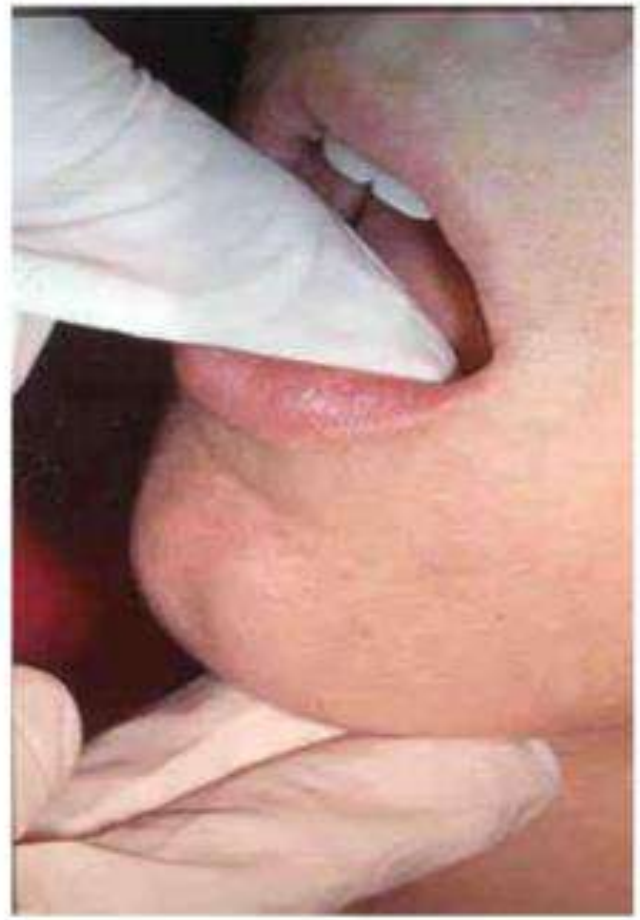
Orifices of ducts

Submandibular gland

Bimanual palpation – gland

Extraoral palpation – lymphnode





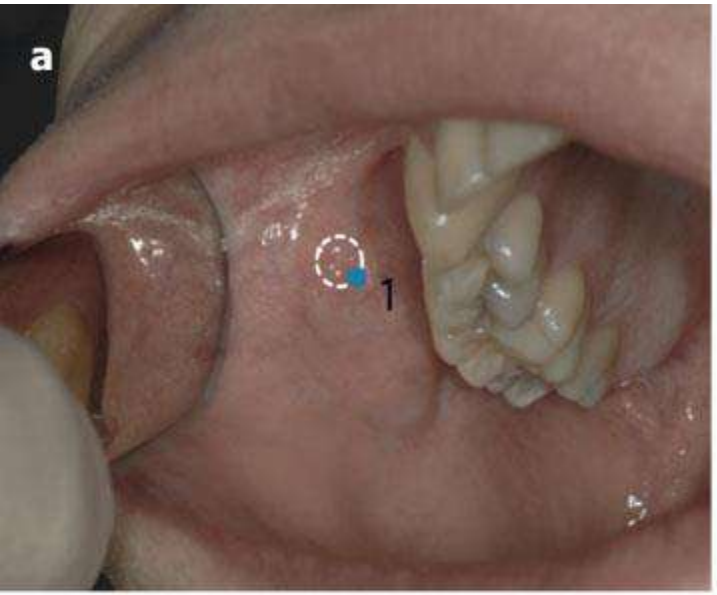
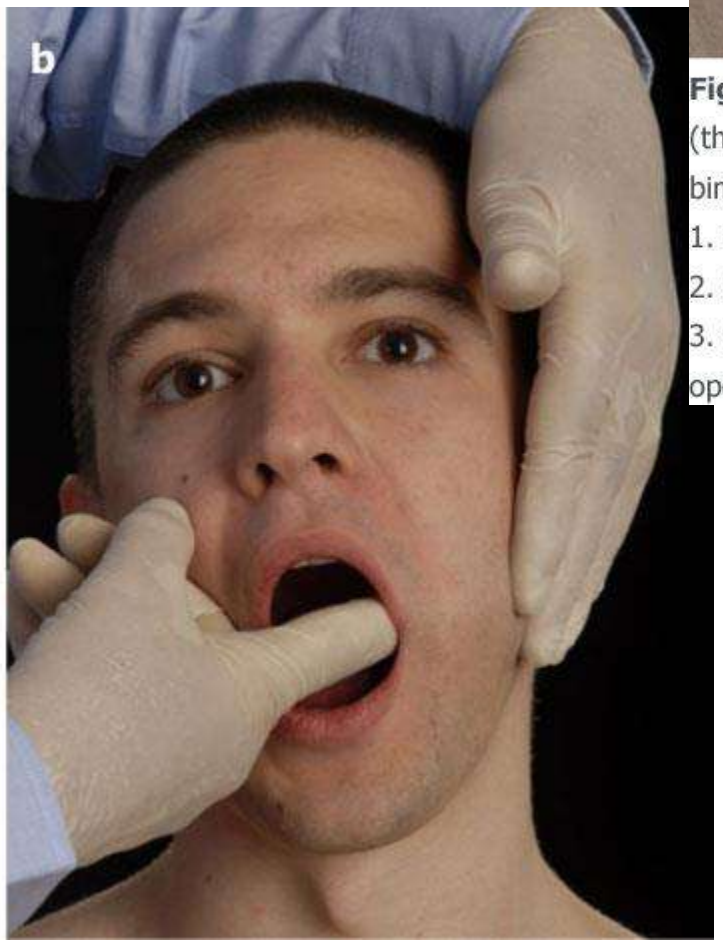
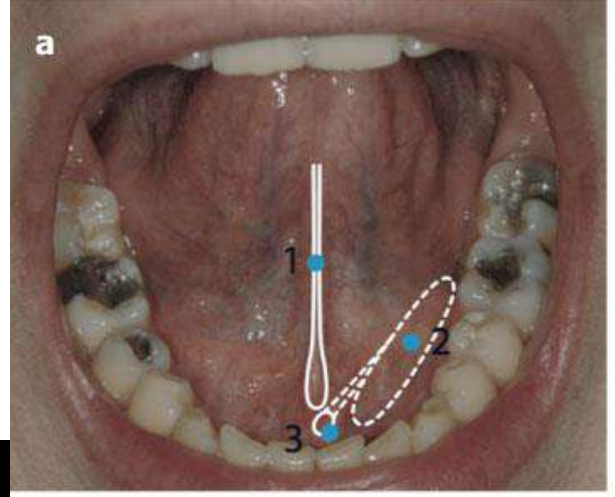


Figure 12 a. Opening of parotid duct opposite second upper molar tooth; b. bimanual examination of gland.





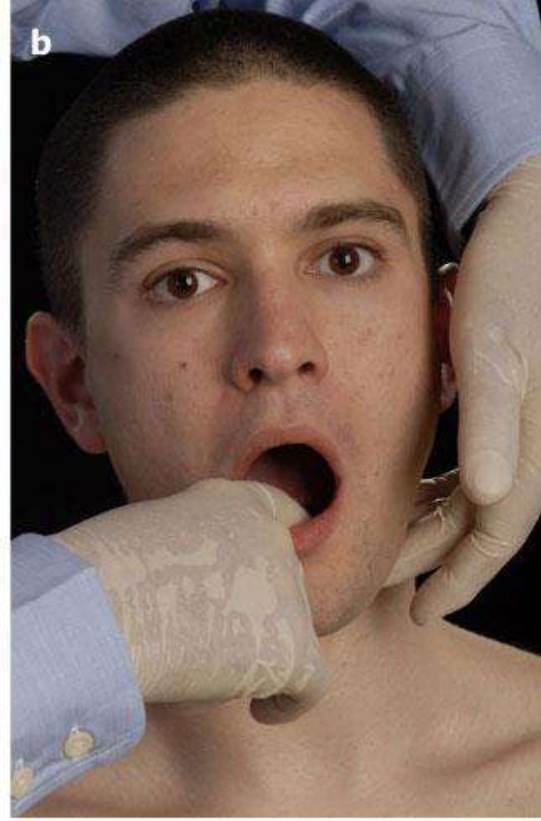
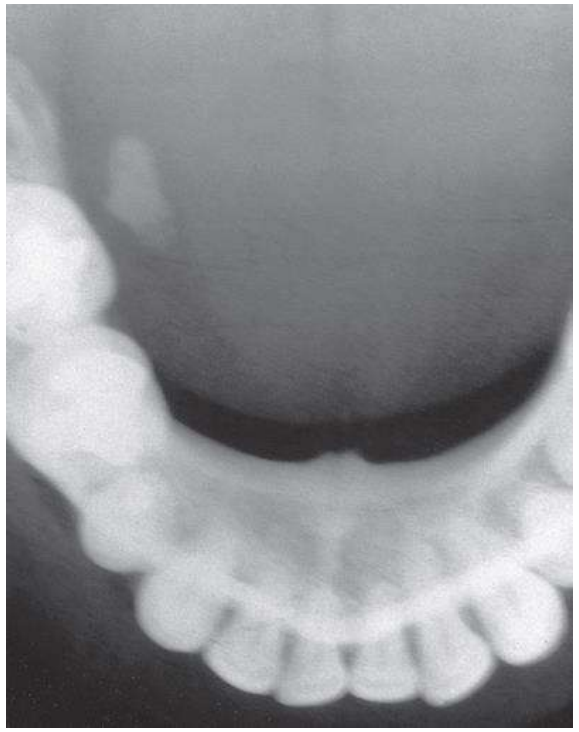


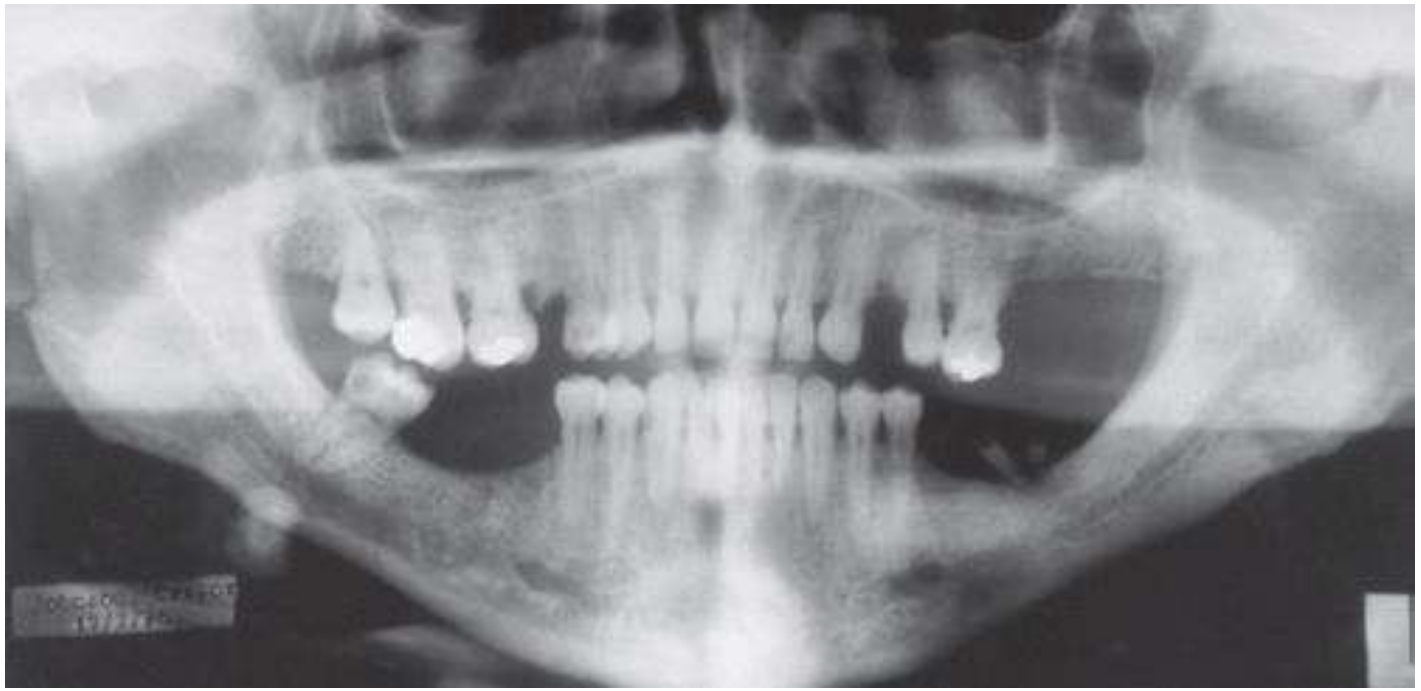
Figure 14 a. Submandibular duct opening (the sublingual gland is outlined); b. bimanual examination of gland.

1. Frenulum
2. Sublinguinal gland
3. Submandibular papilla, containing opening of submandibular duct

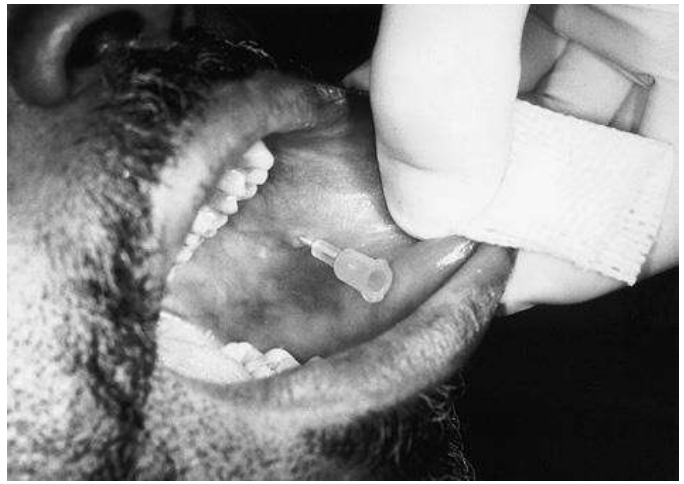
Salivary Gland Radiology

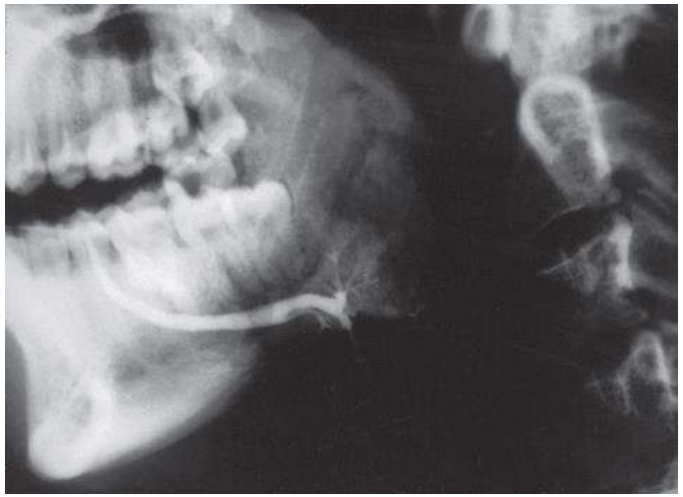
Plain-Film Radiograph

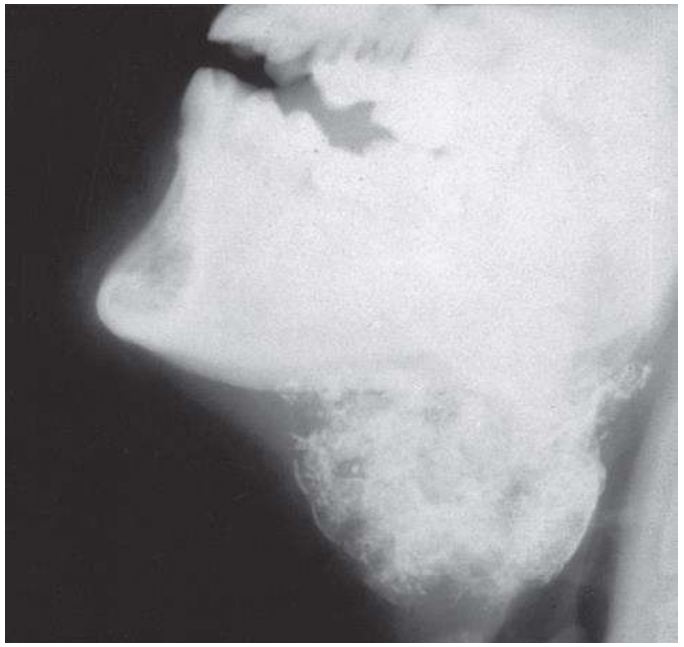


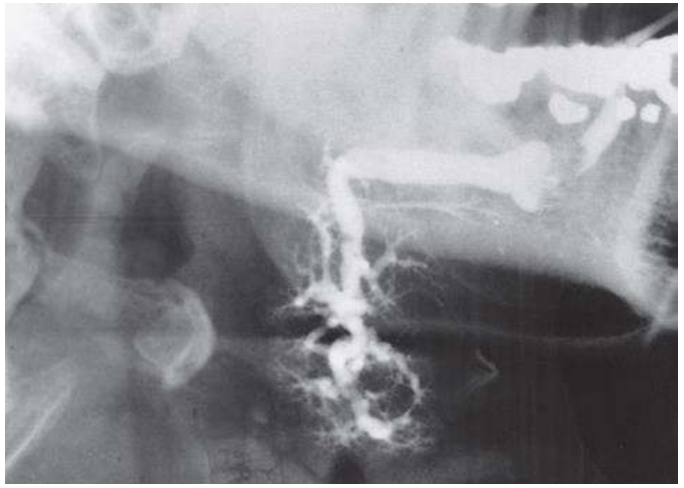


Sialography

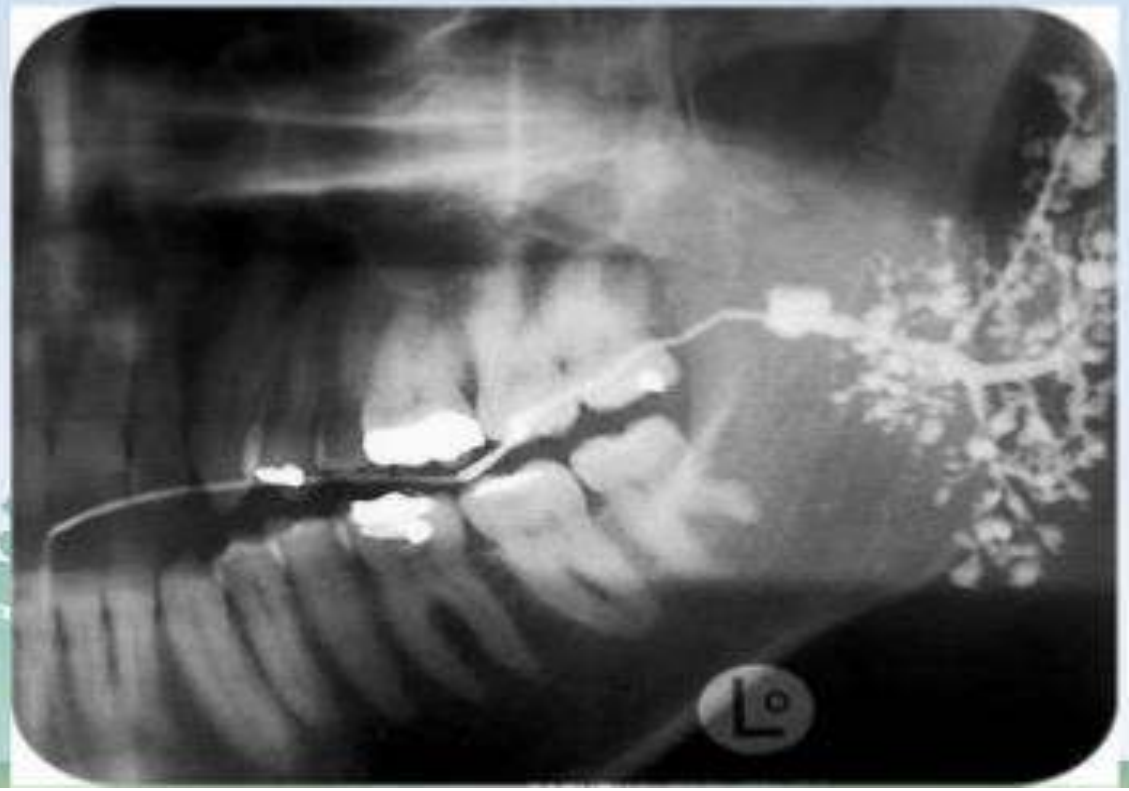


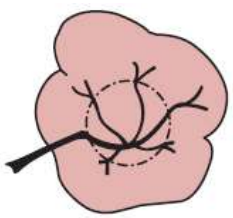


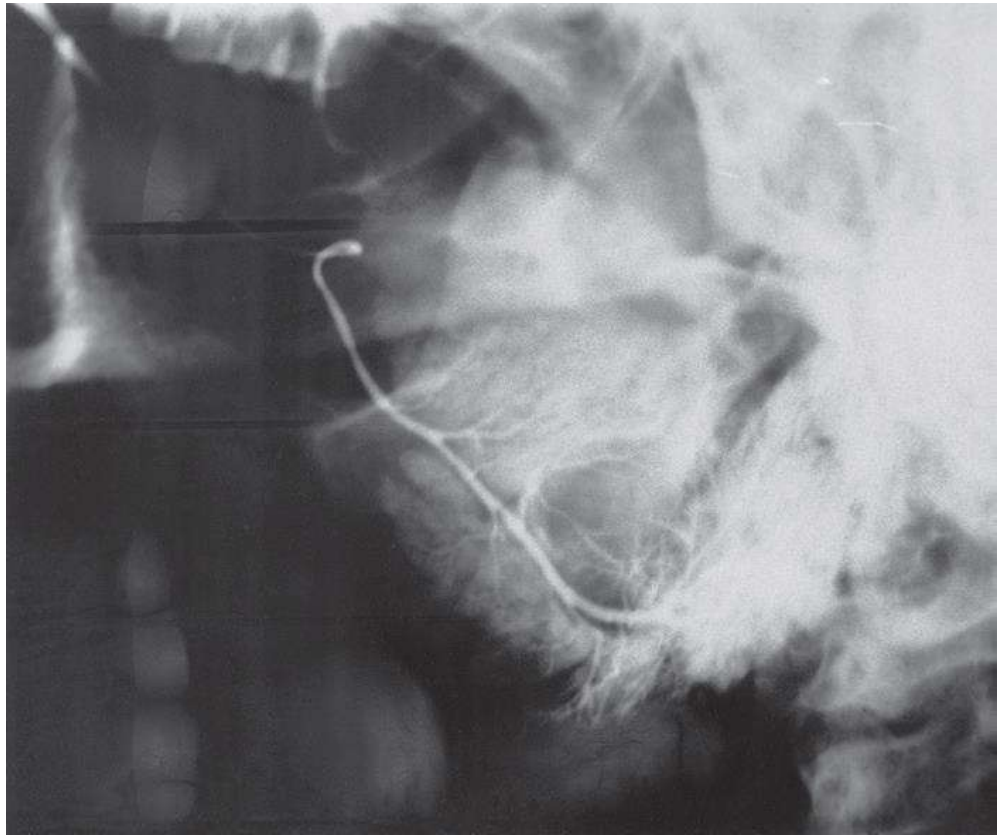




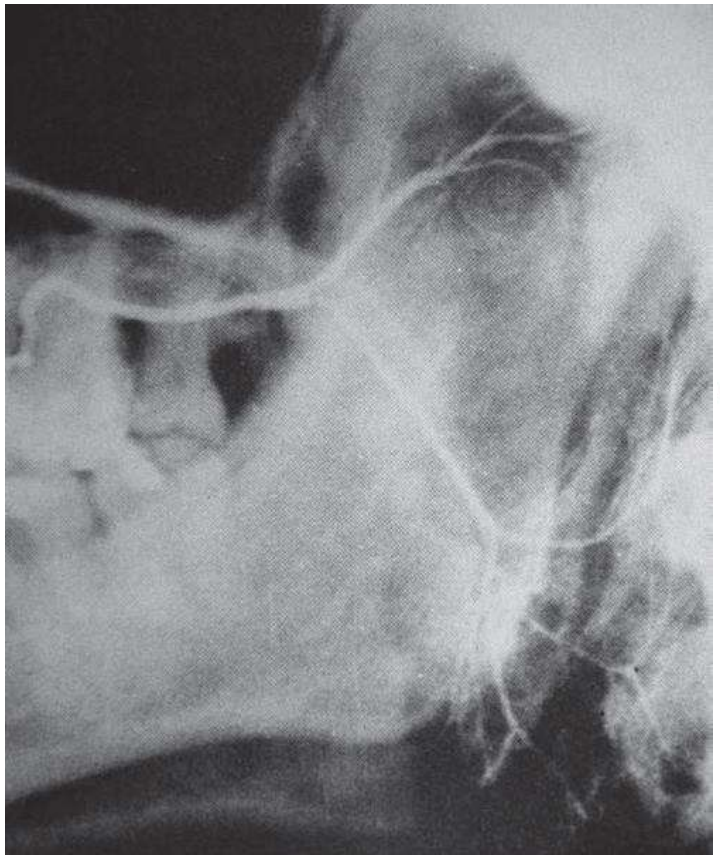
- 1. Multiple sialectasias (snow storm app/ branchless fruit laden tree/ cherry blossom) in sialogram with atrophy of ductal system delayed emptying of dye.
- 2. Impaired salivary activity seen in salivary scintiscanning

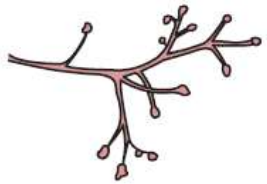




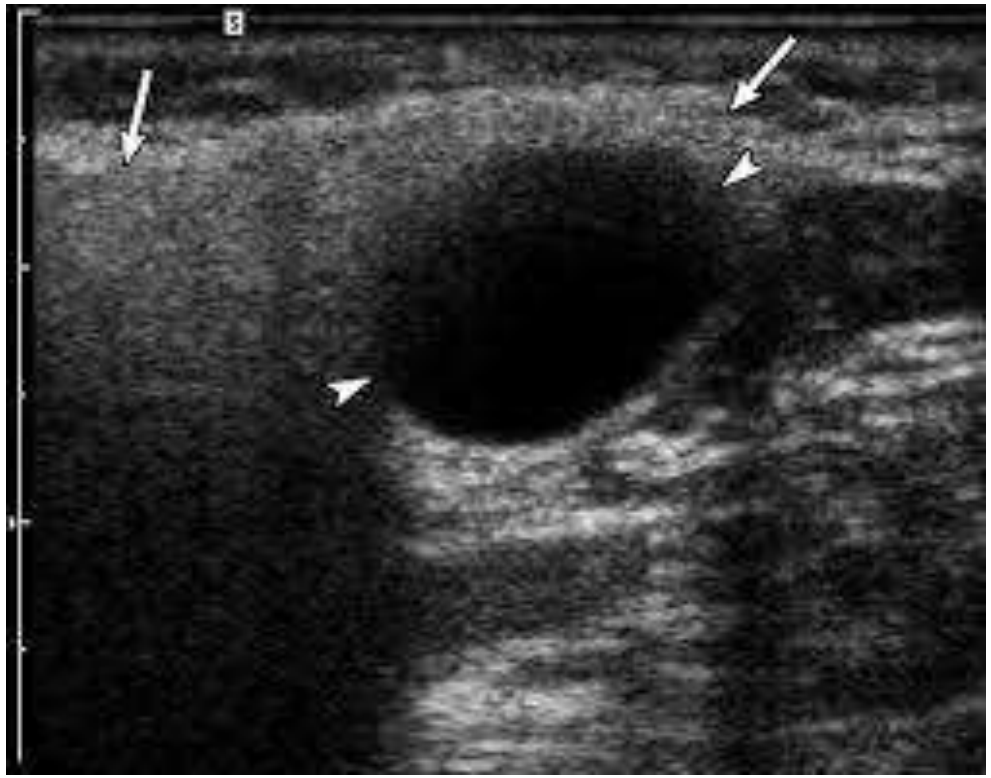


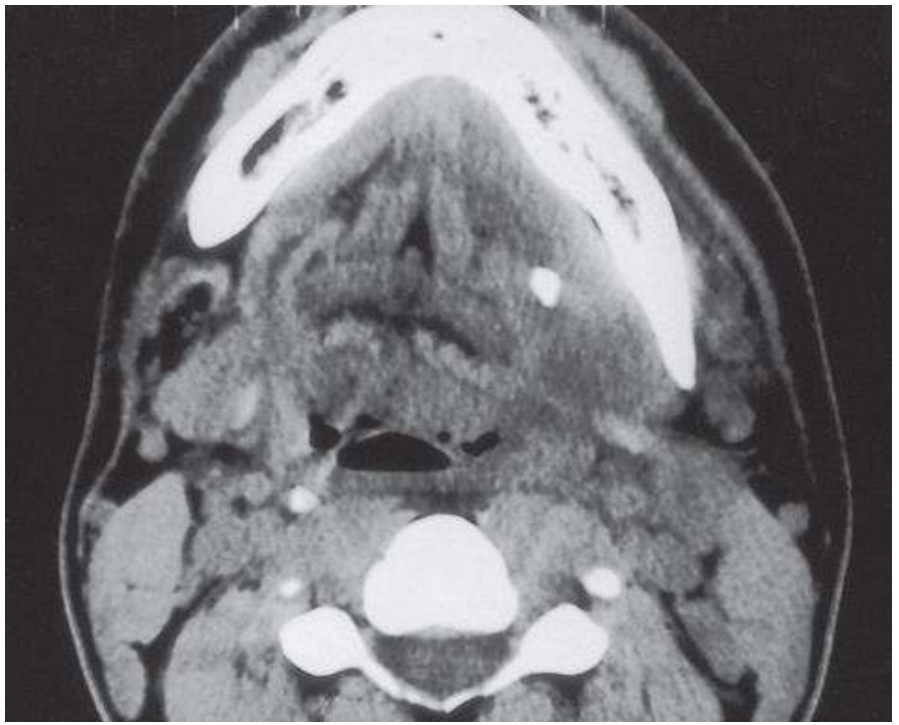
ball-in-hand” phenomenon caused by tumor displacement of the gland.

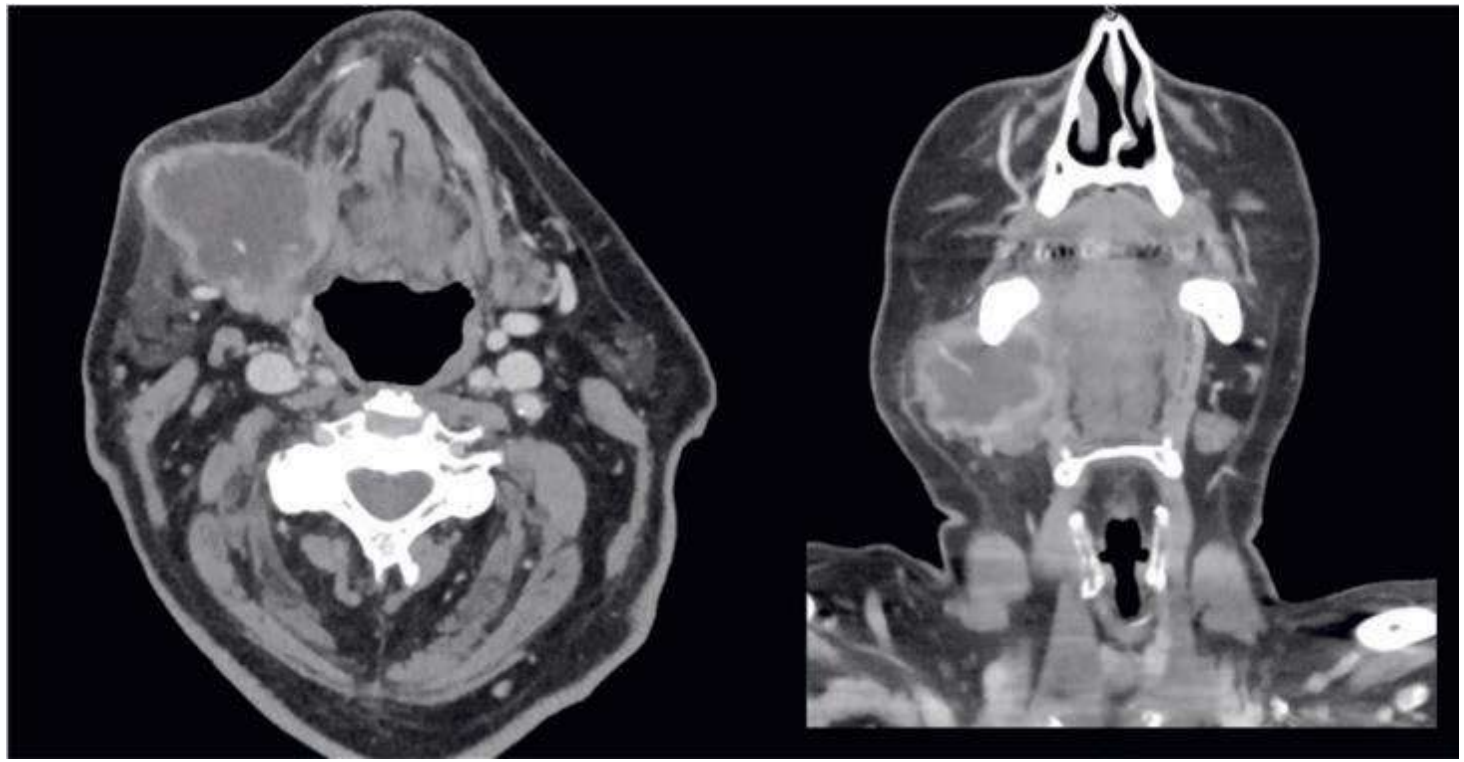




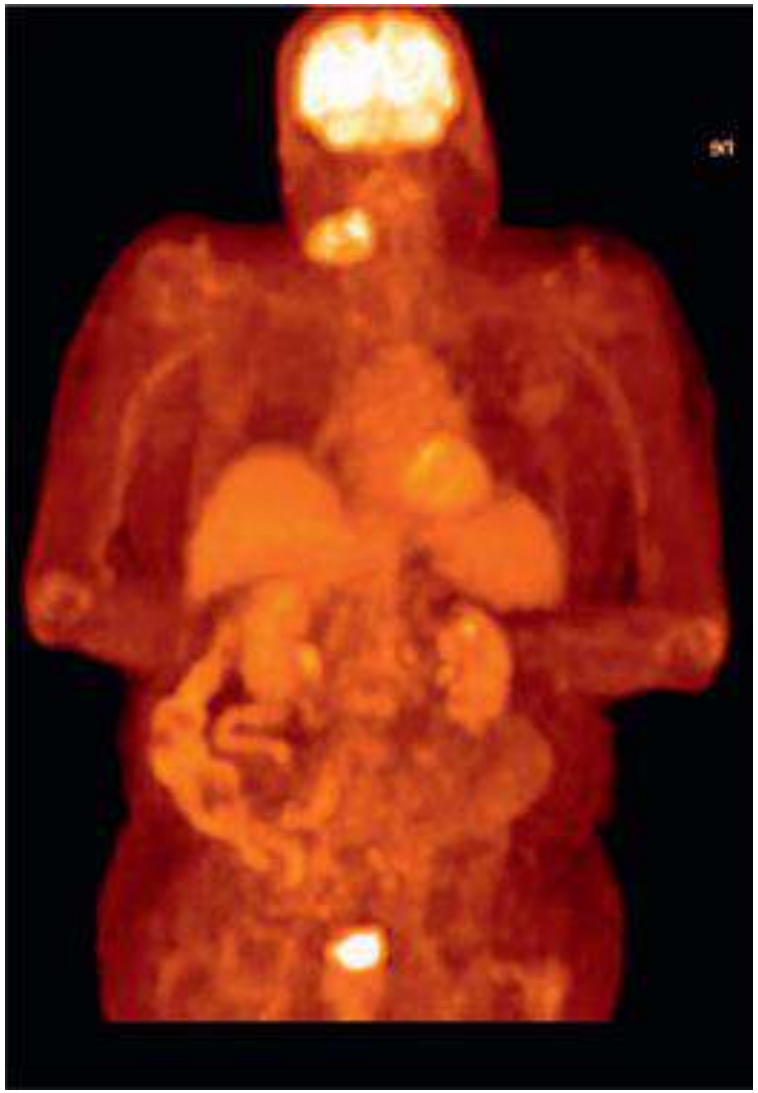
Parotid sialadenitis with acinar destruction from chronic disease. *Inset* shows “pruning of the tree” caused by acinar destruction



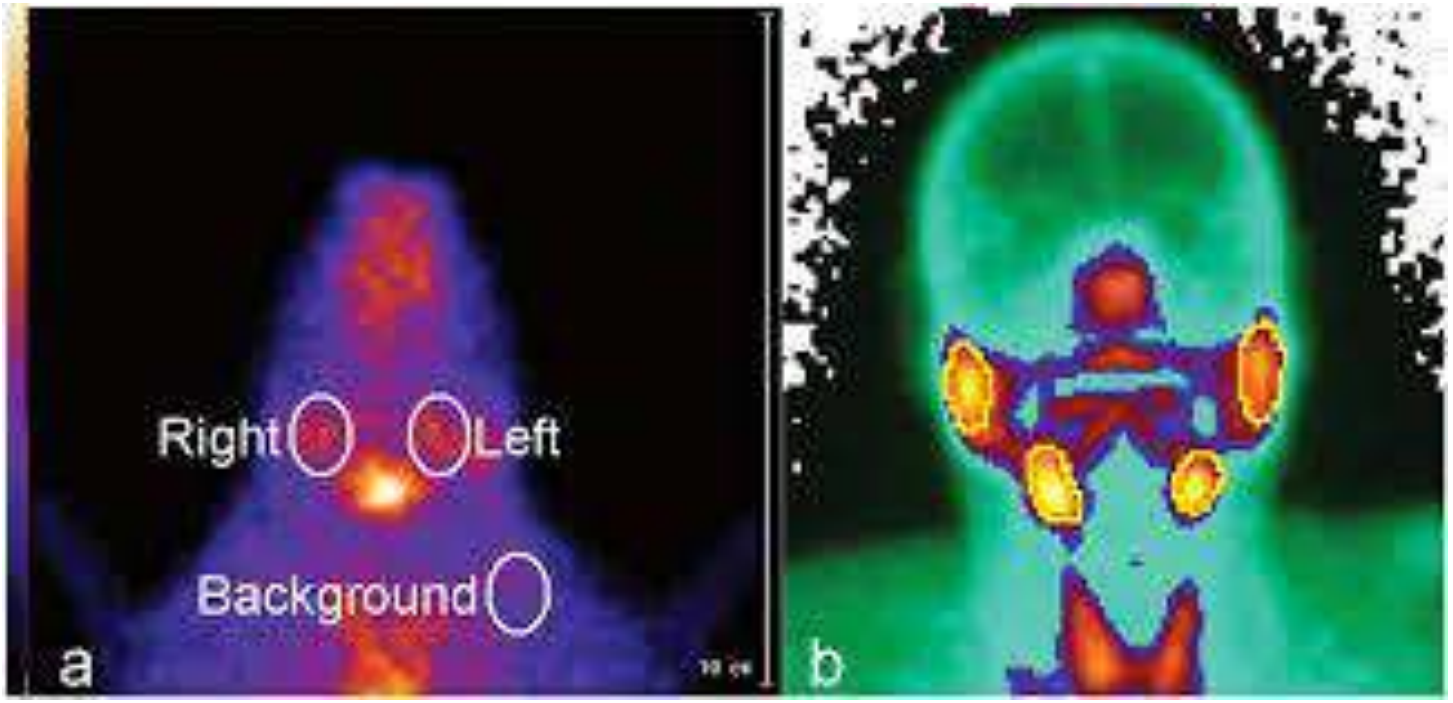




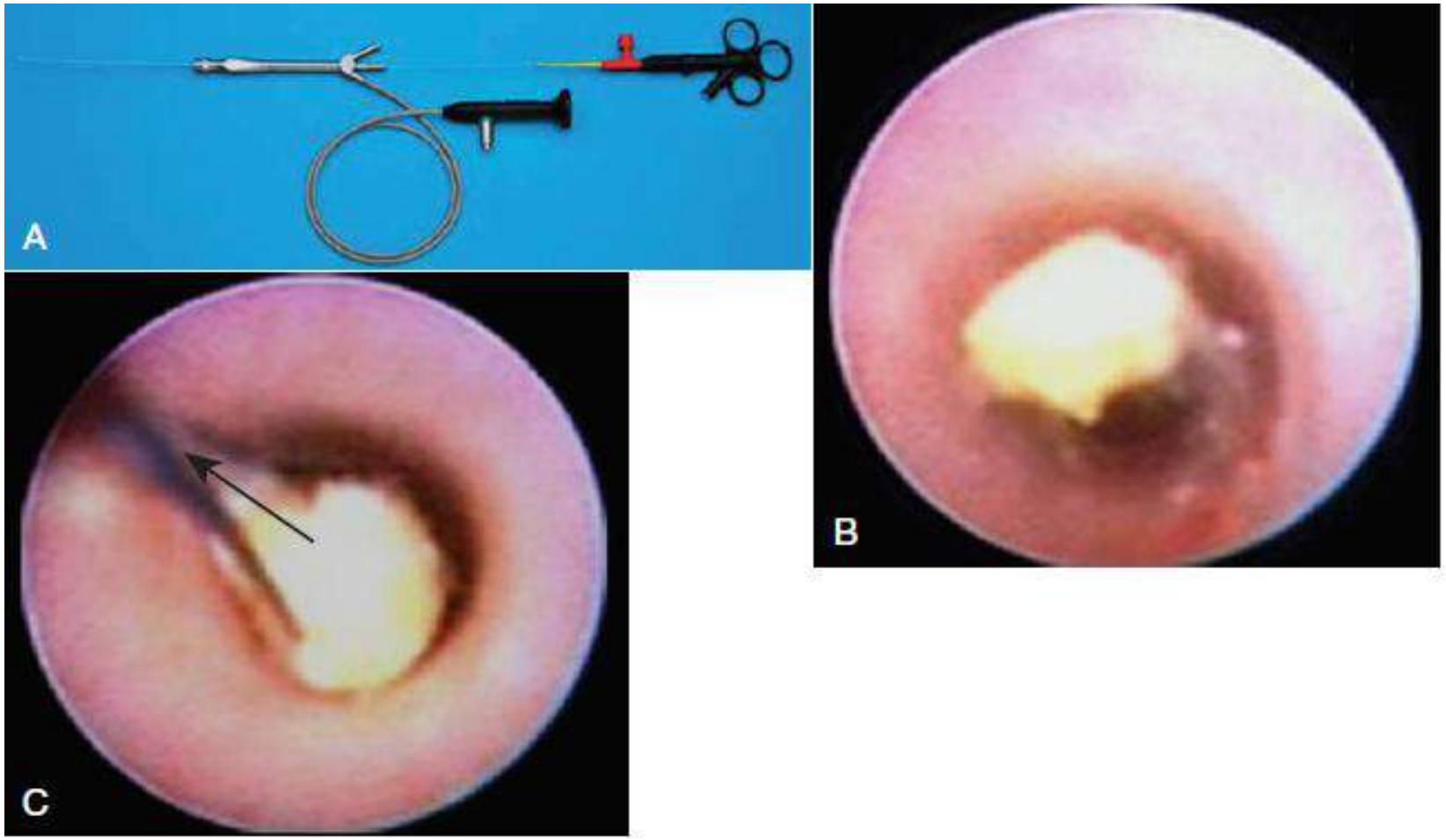
FDG-PET



Salivary Scintigraphy



Sialoendoscopy

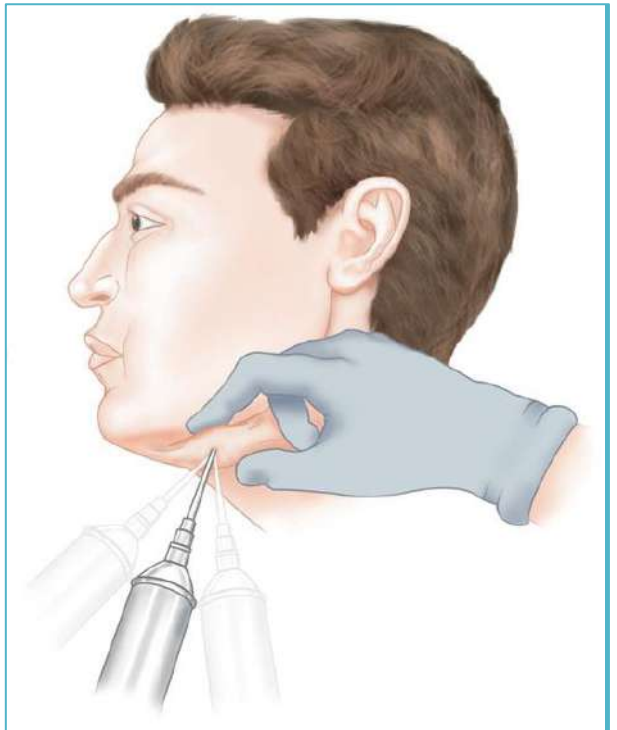


Other investigations of salivary glands

1-Sialochemistry

2-Fine-Needle Aspiration Biopsy

3- Salivary Gland Biopsy: *incisional or excisional, may be used to diagnose a masses in salivary glands and also in Sjogren syndrome*



Obstructive Salivary Gland Disease: Sialolithiasis

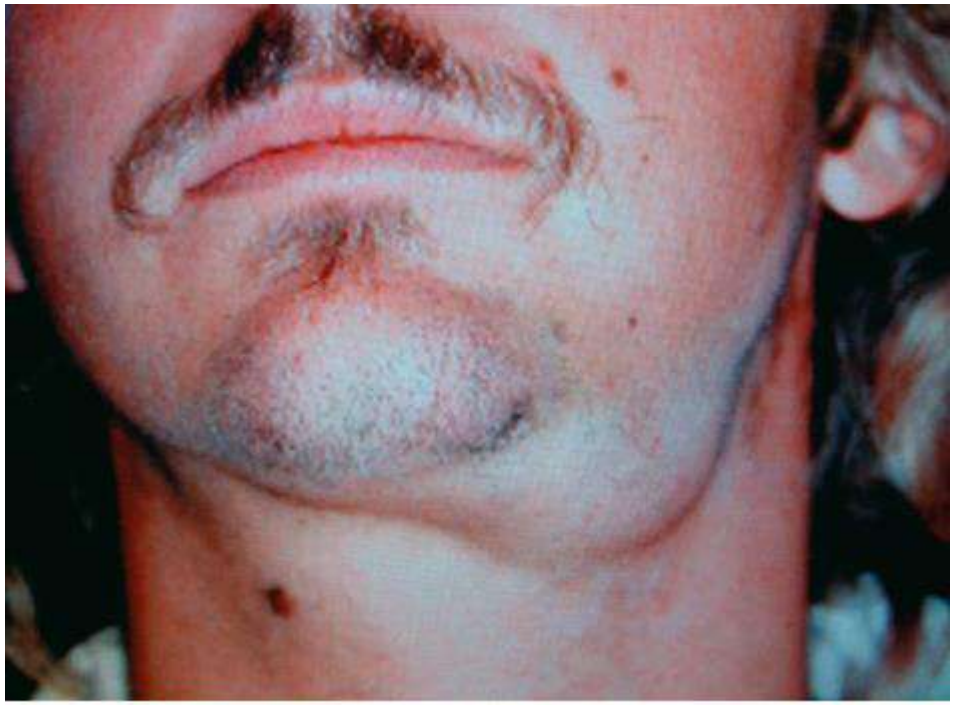
Sialolithiasis

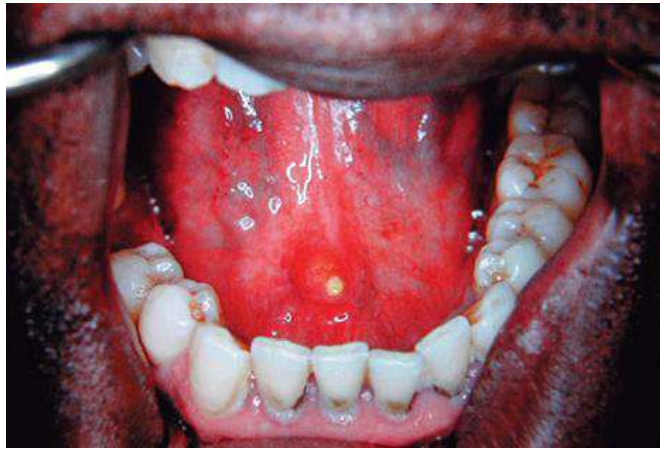
- The formation of stones *is twice as common in men*
- *30 and 50 years*
- *obstruction, reduced salivary flow rate, dehydration, change in salivary pH associated with oropharyngeal sepsis, and impaired crystalloid solubility*

Location	Incidence
Submandibular gland	85%
Parotid gland	10%
Sublingual gland	5%
Minor glands	Rare

Signs and Symptoms

- Pain and swelling of the submandibular gland at mealtimes
- Tenderness to palpation of submandibular gland
- Cervical lymphadenopathy
- Constitutional symptoms (e.g., fever, malaise)
- Reduced salivary flow, or purulence, from Wharton duct
- Palpable stone at orifice of Wharton or Stensen duct
- Occlusal film, Panorex, or cone-beam computed tomography may show stone in duct





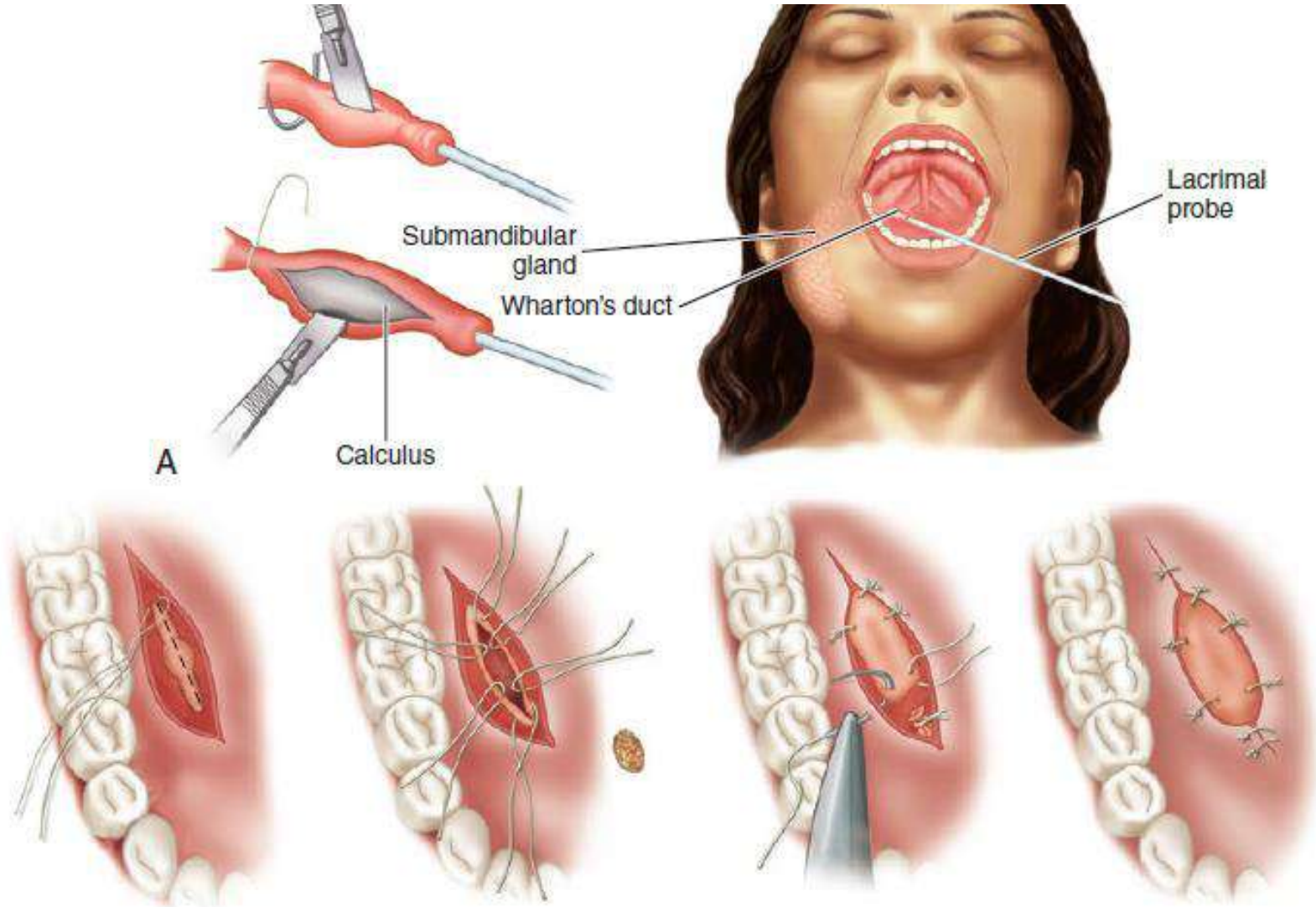
The management of submandibular gland calculi

For anterior stones of submandibular gland :

1. Small anteriorly located stones may be retrieved through the ductal opening after dilation of the orifice.
2. an incision made in the floor of the mouth to expose the duct and the stone (*sialodochotomy)*

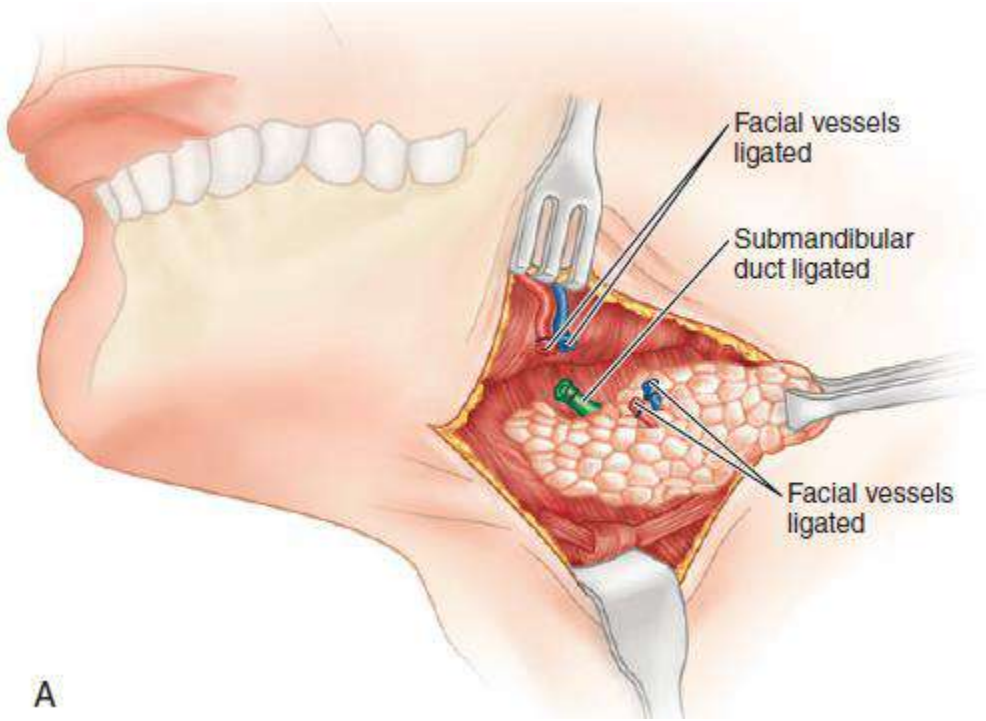
For posterior stones:

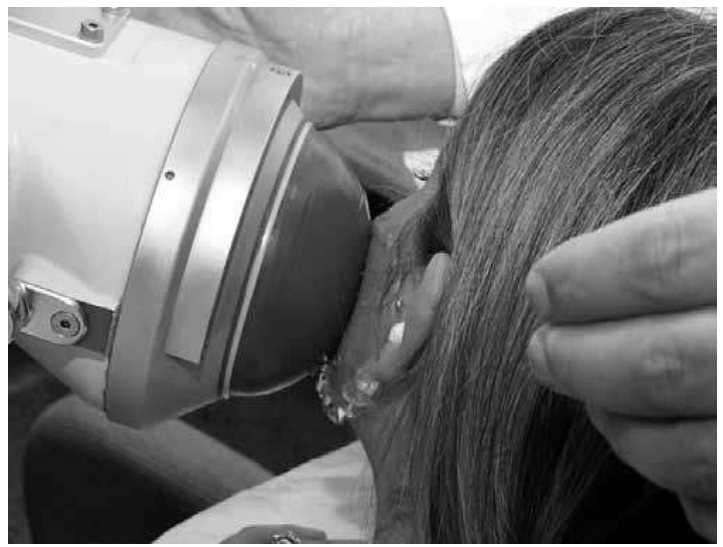
Excision of gland



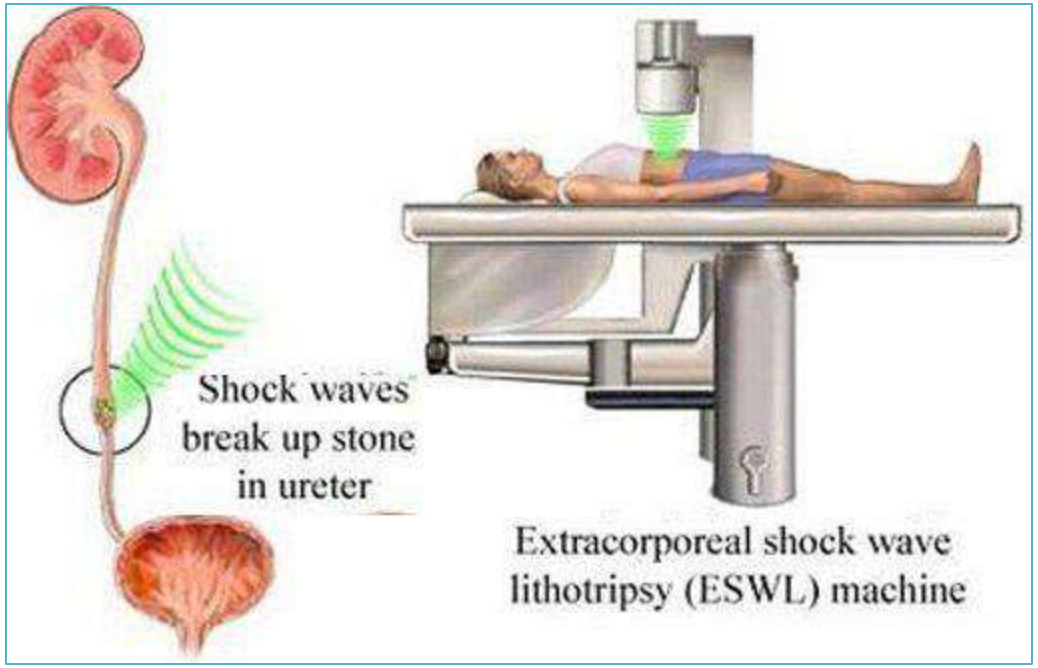
• **Fig. 21.28** (A) Surgical opening of the submandibular duct (sialodochotomy) and removal of the stone (sialolithectomy). Note the lacrimal probe in the orifice of the Wharton duct and the suture passed proximal to stone to prevent displacement. (B) Duct revision occurs by suturing the duct to the floor of mouth mucosa (sialodochoplasty) and tying off the distal end of the duct.

Submandibulectomy

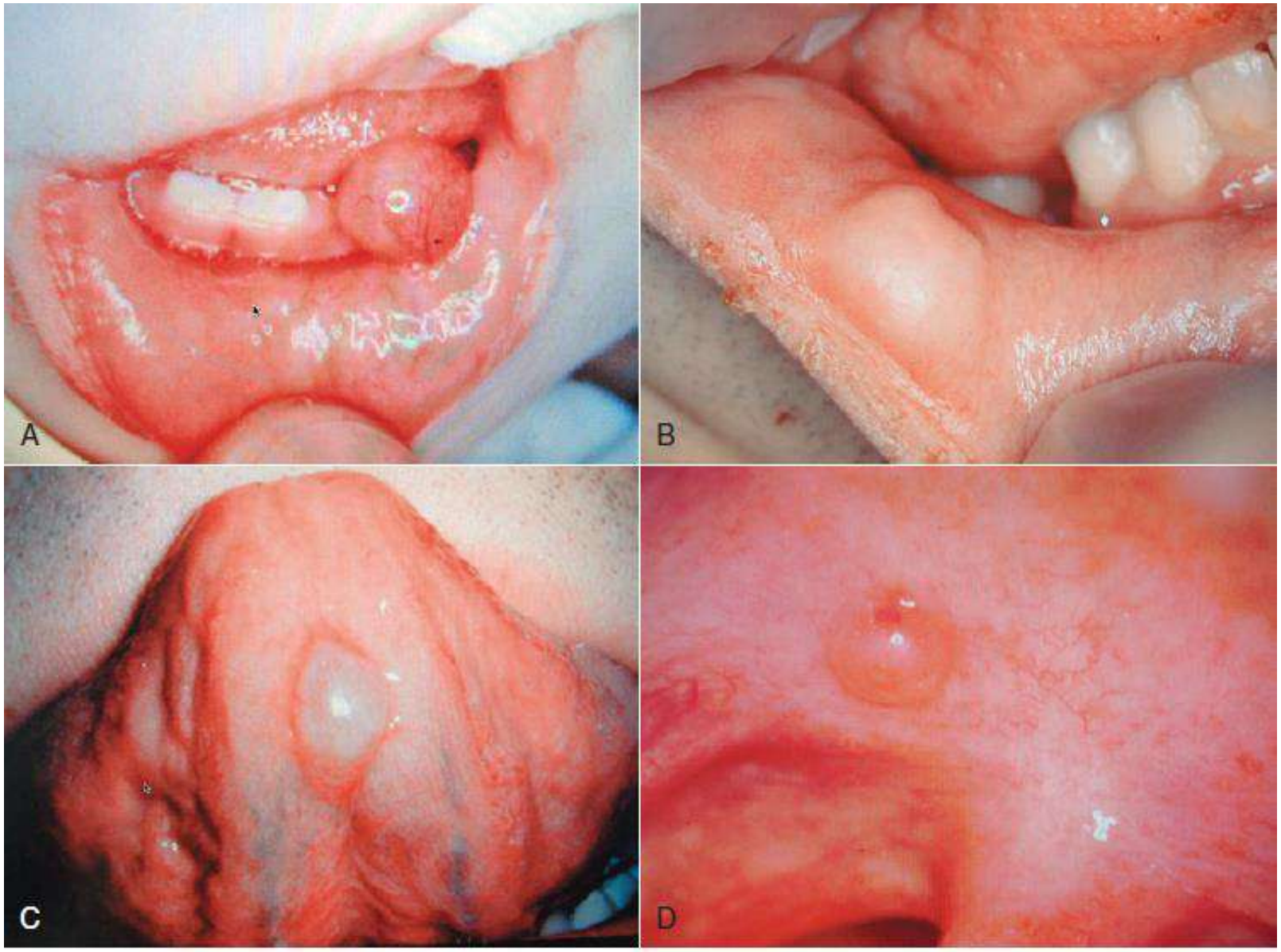




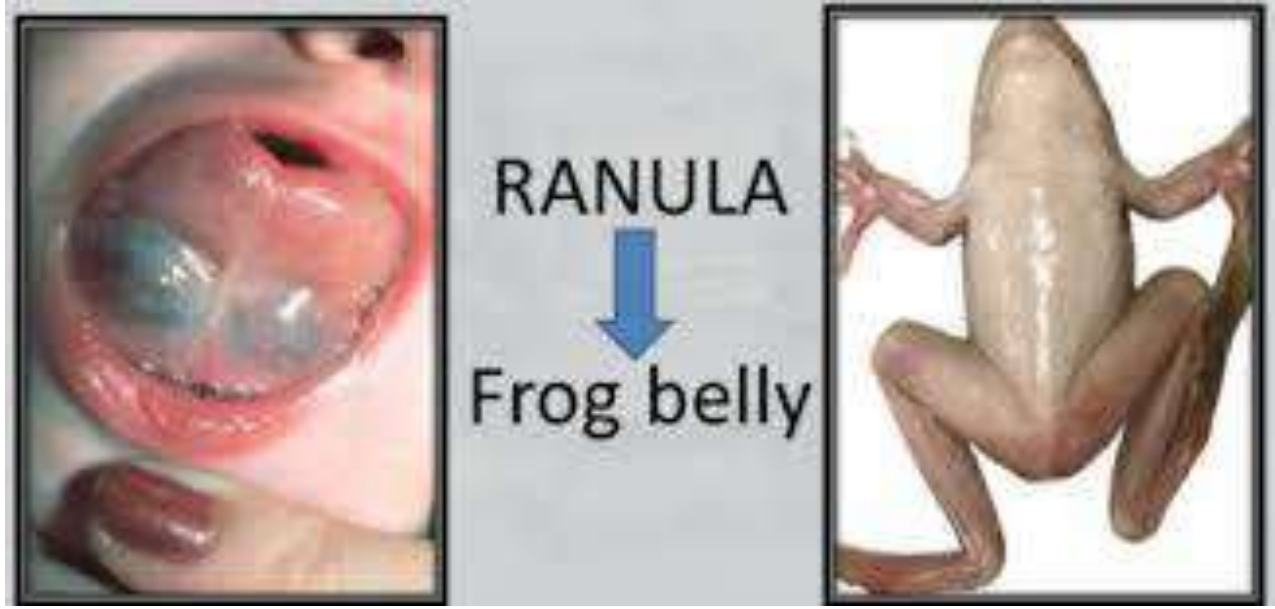
Extracorporeal shockwave lithotripsy

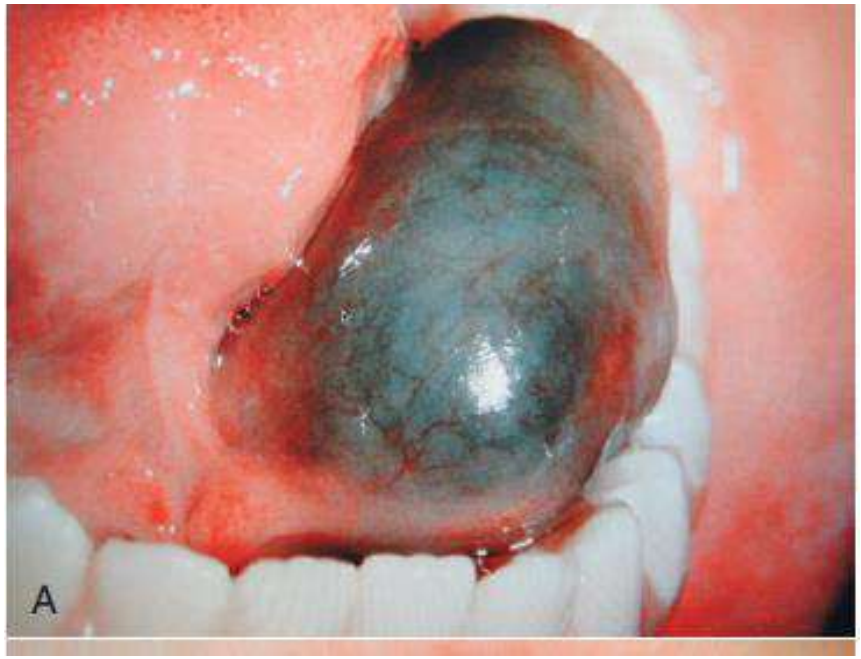


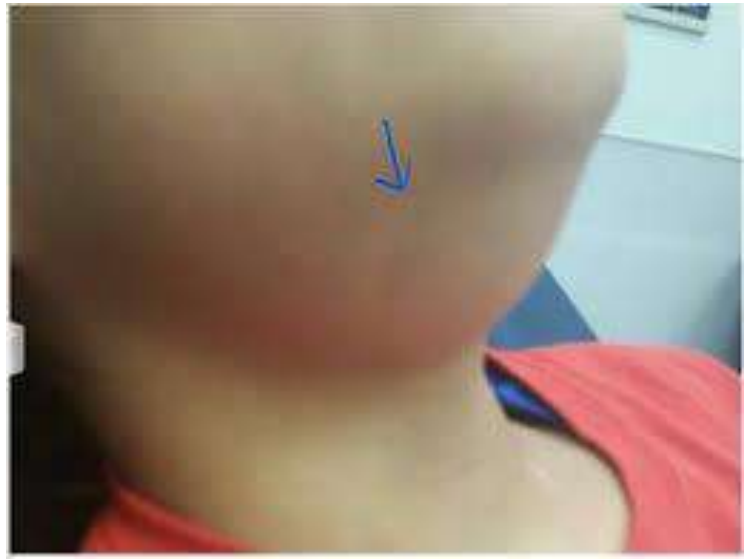
Mucocele



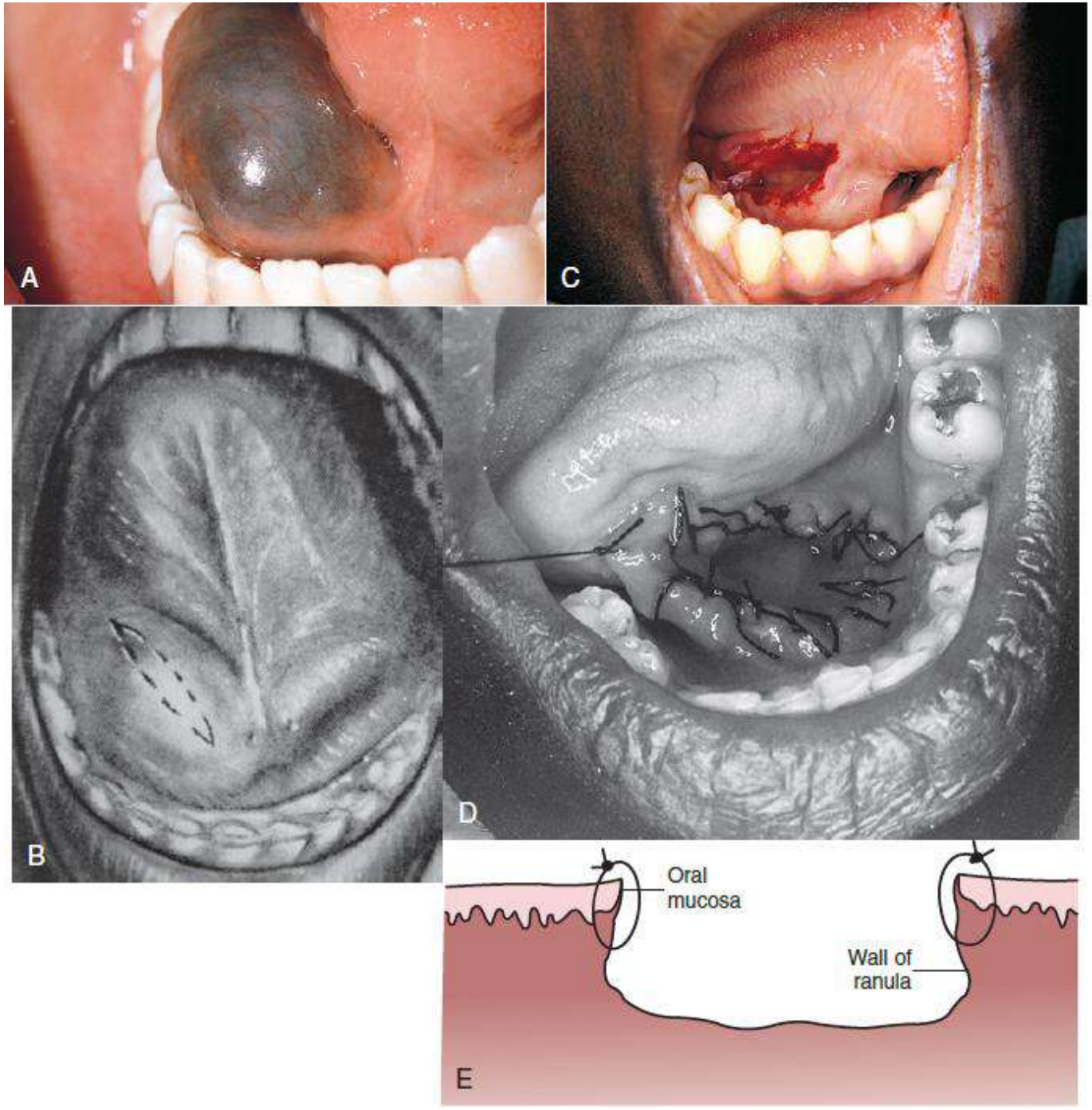
Ranula



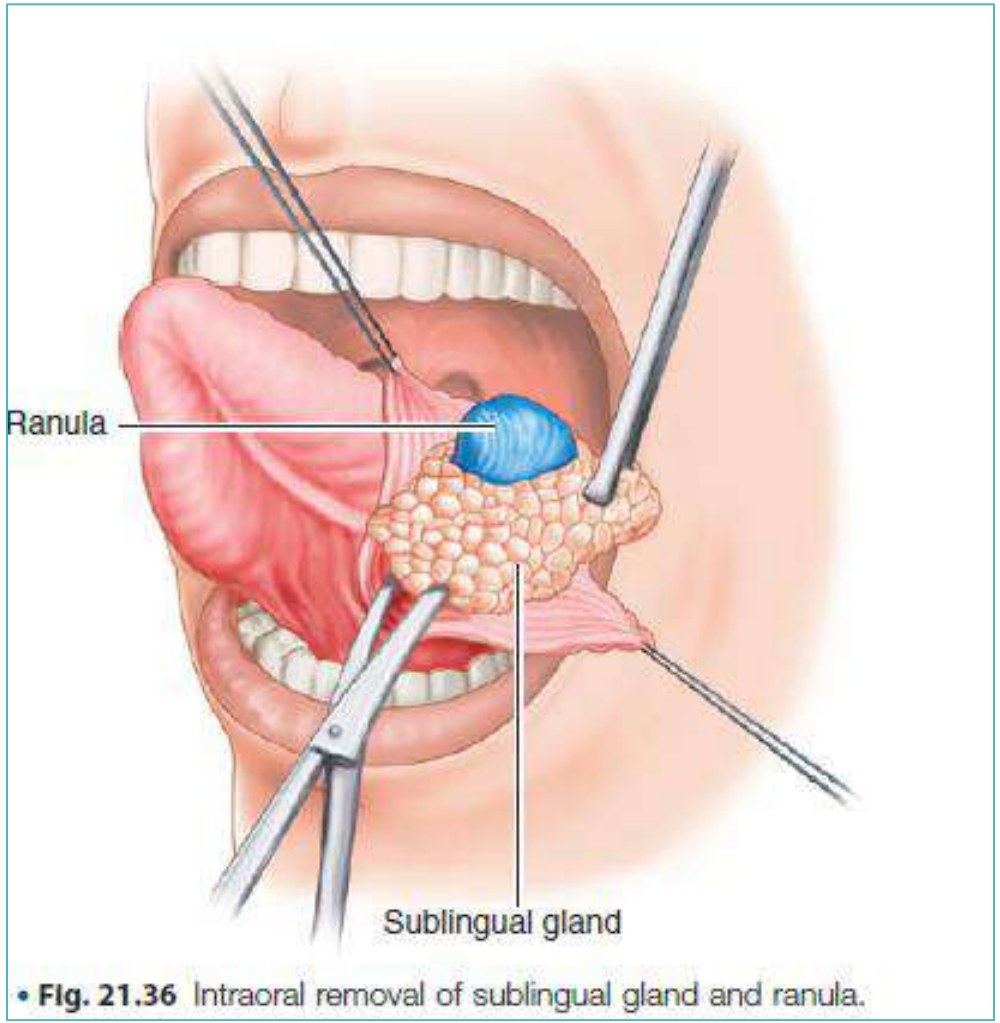




Marsupialization

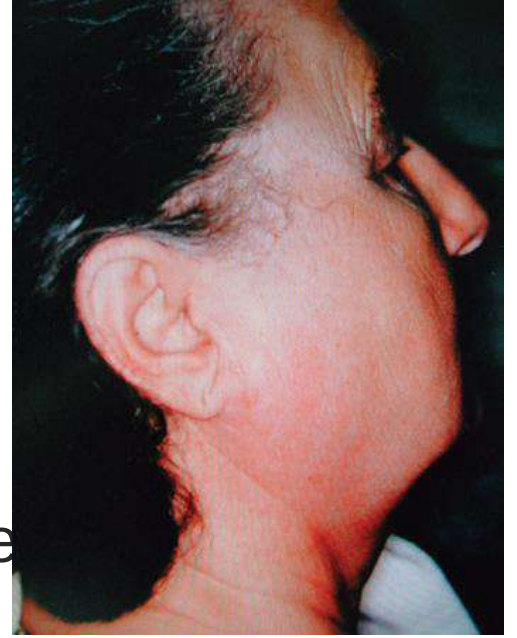


Excision of sublingual gland and ranula



• **Fig. 21.36** Intraoral removal of sublingual gland and ranula.

Salivary Gland Infections



- ❖Acute or chronic
- ❖If occurs in submandibular gland, it is usually obstructive
- ❖If parotids The cause of *acute suppurative sialadenitis* usually due

change in fluid balance that is likely to occur in older patients;
debilitated,
malnourished,
Dehydrated
chronic illness or significant comorbidities.

- Usually bilateral.
- Age is 60 years, with a slight male predilection
- Mixed infection
- rapid onset of swelling in the preauricular (parotid gland) or submandibular regions, with associated erythema and pain .
- Palpation of the involved gland reveals no flow or elicits a thick, purulent discharge from the orifice of the duct (in this case, “infection leads to obstruction

Treatment of acute suppurative sialadenitis

1) intravenous fluid hydration,

2) antibiotics, and analgesics

3) salivary flow stimulants :by using of sialogogues such as lemon drops+massage of glands +discontinue of antisialogogue such as antihistamine

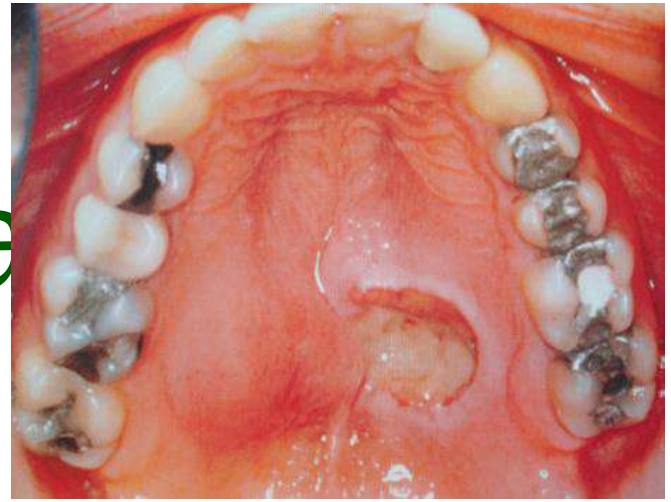
4) incision and drainage: if there abscess

5) (sialoadenectomy) may be indicated.

Parotitis

Bacterial	viral
1)Unilateral	***bilateral***
2)No prodromal s.& S.	***Preceded by prodromal signs and symptoms*** *of 1 to 2 days' duration, including fever, malaise, loss of appetite, chills, headache, sore throat, and preauricular tenderness.*
3)Purulent discharge	***Purulent discharge from Stensen's duct is rare***
4)Lab. Investigations: -ve	***Lab investigations*** *reveal elevated serum titers for mumps or influenza virus, leukopenia, relative lymphocytosis, and high levels of serum amylase*

Necrotizing Sialometaplasia



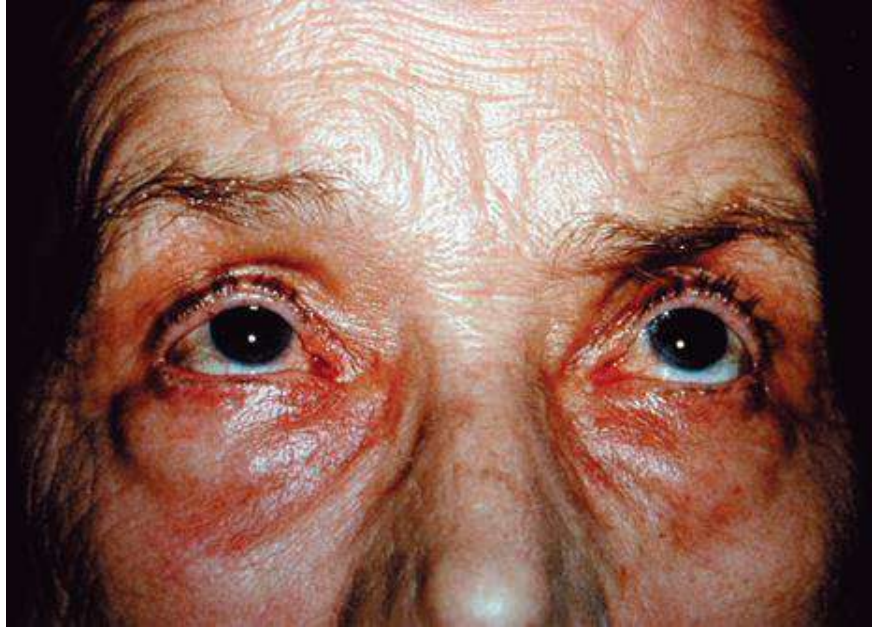
reactive, nonneoplastic inflammatory process that usually affects the minor salivary glands of the palate.

Lesions usually appear as large (1 to 4 cm), painless or painful, deeply ulcerated areas lateral to the palatal midline and near the junction of the hard and soft palates. This condition is of considerable concern because, clinically and histologically, it resembles a malignant carcinoma.

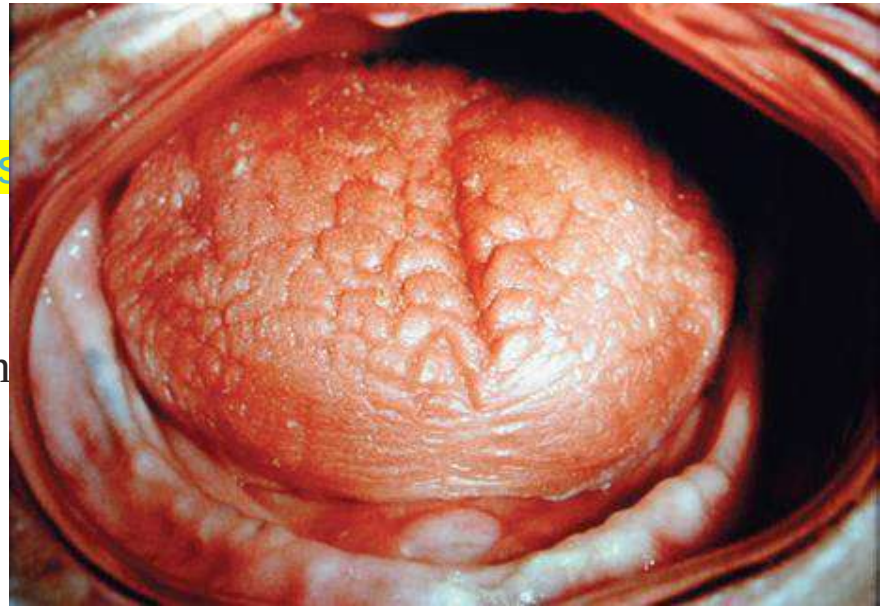
The ulcerations of necrotizing sialometaplasia usually heal spontaneously within 6 to 10 weeks after onset and require no surgical management

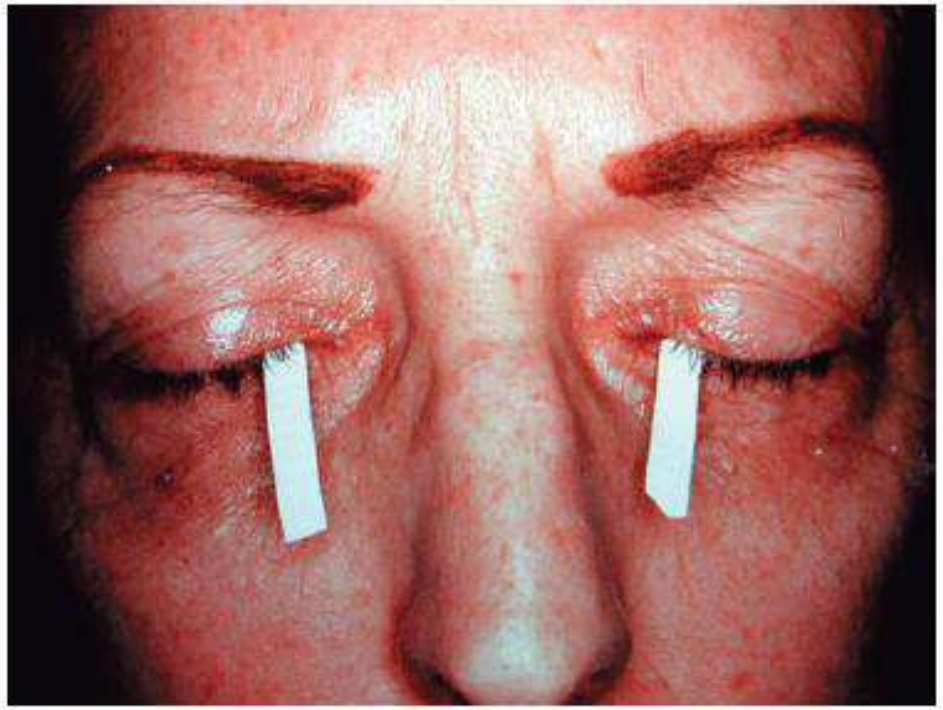
- # Sjögren Syndrome

Sjogren syndrome is a multisystem disease process with a variable presentation.
The two types of Sjogren syndrome are (1) *primary*
Sjogren syndrome, or *sicca syndrome*, characterized by *xerostomia*
(dry mouth) and *keratoconjunctivitis sicca* (dry eyes)
and (2) *secondary Sjogren syndrome.*

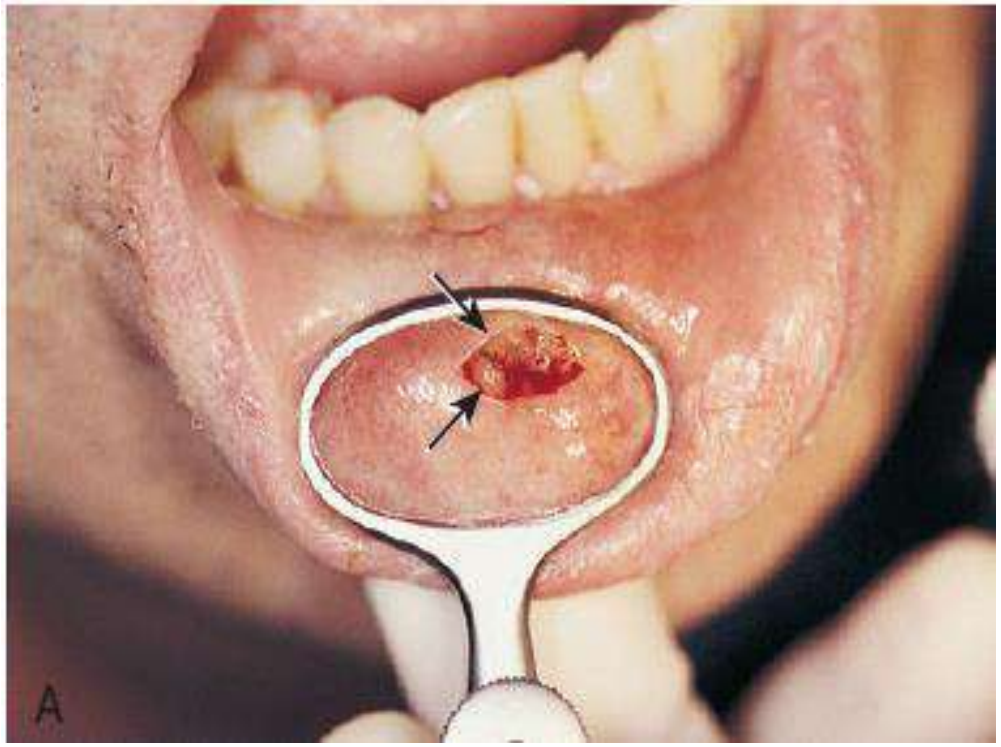


female predilection of 9 : 1, with a mean age of 50 years.
In general, the first symptoms to appear are arthritic complaints,
followed by ocular symptoms and, late in the disease process, salivary gland symptoms
Painless, bilateral, diffuse persistent or intermittent swelling of major salivary gland.
Sialography show"fruit-laden, branchless tree" pattern
Schirmer test for lacrimal flow can be performed to quantify the degree of lacrimal flow reduction
labial minor salivary gland biopsy
laboratory tests: elevated ESR, autoantibodies such as SS-A and SS-B antibodies





• **Fig. 21.41** Schirmer test for dry eyes in a patient with Sjögren syndrome. Filter paper is placed in the ocular fornix and observed for wetting (15 mm within 5 minutes is normal; <5 mm in 5 minutes is severe and may indicate Sjögren syndrome).



Salivary gland tumors

(80% to 85%) of tumors occur in major glands, opposed to the minor glands (15% to 20%.

75% to 80% of major gland tumors are benign, and 50% to 55% of minor gland tumors are benign.

Majority of salivary tumors occur in the *parotid gland*, and the majority of those are benign (most commonly *pleomorphic adenoma.*

Benign Tumors

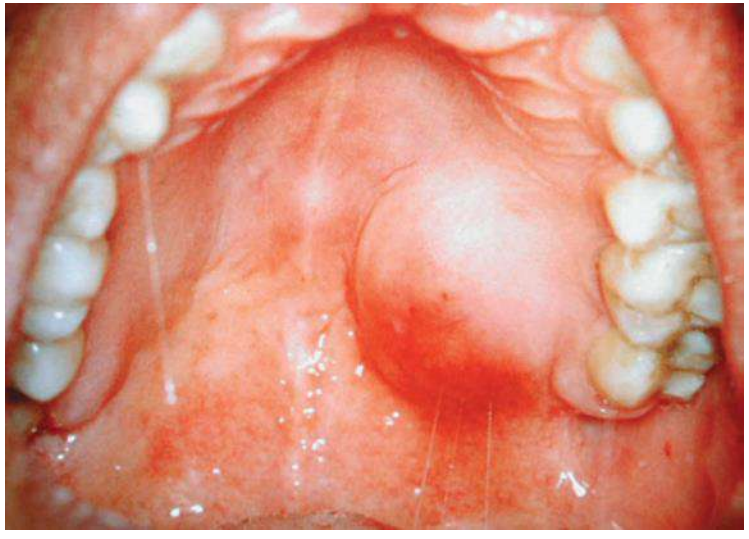
Pleomorphic adenoma

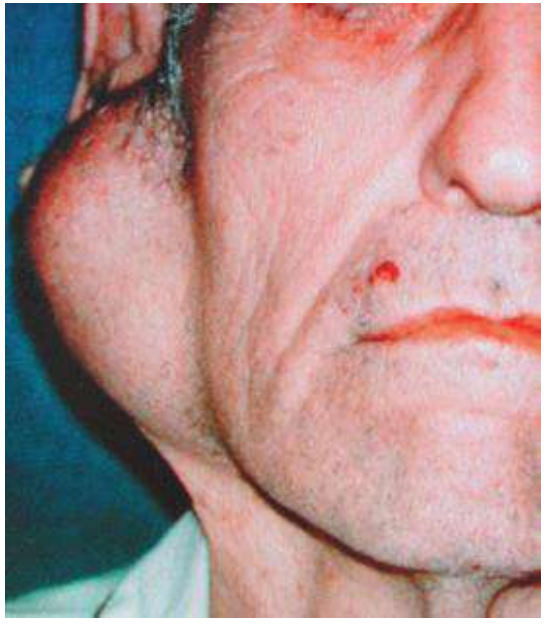
Warthin tumor

Monomorphic adenoma

Pleomorphic adenoma

- The pleomorphic adenoma is the most common benign salivary tumor at all sites. Approximately 80% of all pleomorphic adenomas occur in the parotid. While the palate is the most site for minor glands.
- Pleomorphic adenomas are usually slow-growing, firm, painless swelling.
- It derives its name from the architectural Pleomorphism (variable appearance)
- pseudo capsule





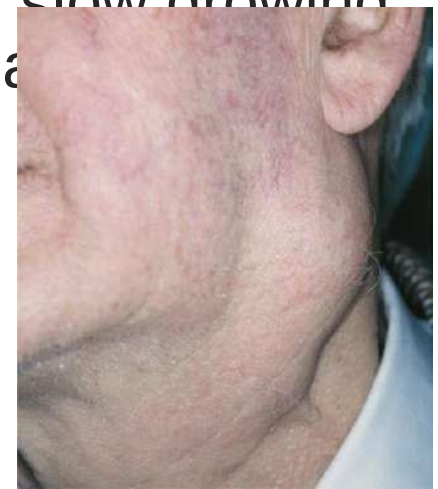
Warthin's Tumors adenoma

almost exclusively found in the parotid.

mostly in men and is more common in smokers.

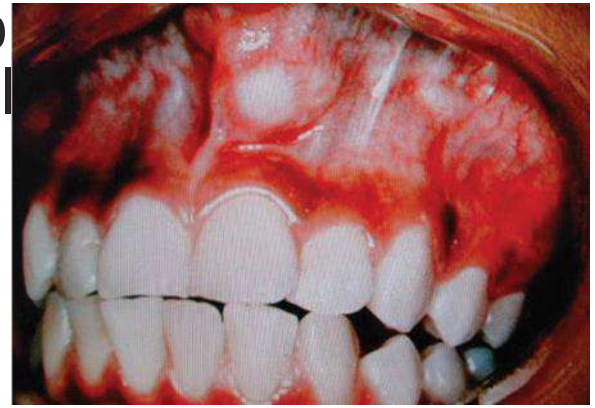
derive from salivary duct cells that are entrapped in lymph nodes during embryonic development.

Painless, slow growing movable a



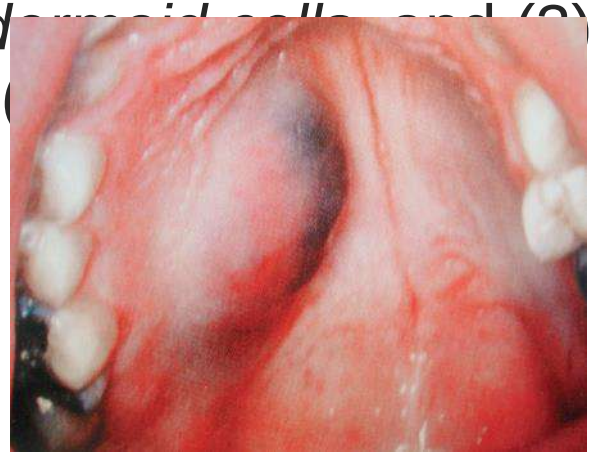
monomorphic

- uncommon solitary lesion composed of one cell type
- affecting predominantly the upper lip minor glands (*canalicular adenoma*) and the parotid gland *(basal cell adenoma)*.
- Histopathologic examination reveals an encapsulated lesion composed of one type (monomo ductal epithelial cel



The *mucoepidermoid carcinoma*
adenocarcinoma

- the most common malignant salivary gland tumor
- mostly occur in parotid and palatal.
- The clinical presentation is a submucosal mass that may be painful or ulcerated. The mass may appear to have a bluish tinge.
- Histopathologic examination shows three cell types: (1) *mucous cells*, (2) *epid* [illegible] *intermediate* (

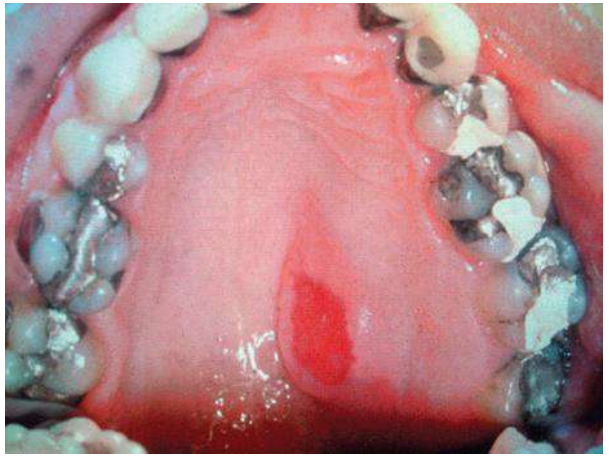


Polymorphous low-grade

second most common salivary gland malignancy.

The most common site is the *junction of the hard and soft palate.*

The *adenoid cystic carcinoma*



is the third most common intraoral salivary gland malignancy, with a mean age of 53 years and a male-to-female ratio of 3 : 2

50% of these tumors occur in the parotid gland, whereas the other 50% occur in the minor glands of the palate.

nonulcerated masses, with an associated chronic dull pain. Occasionally parotid lesions may result in facial paralysis

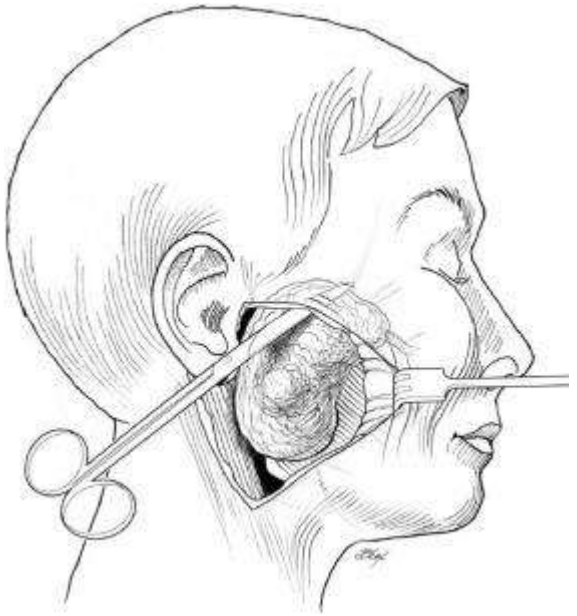
The treatment is wide surgical excision, followed, in some cases, by radiation therapy

Parotidectomy

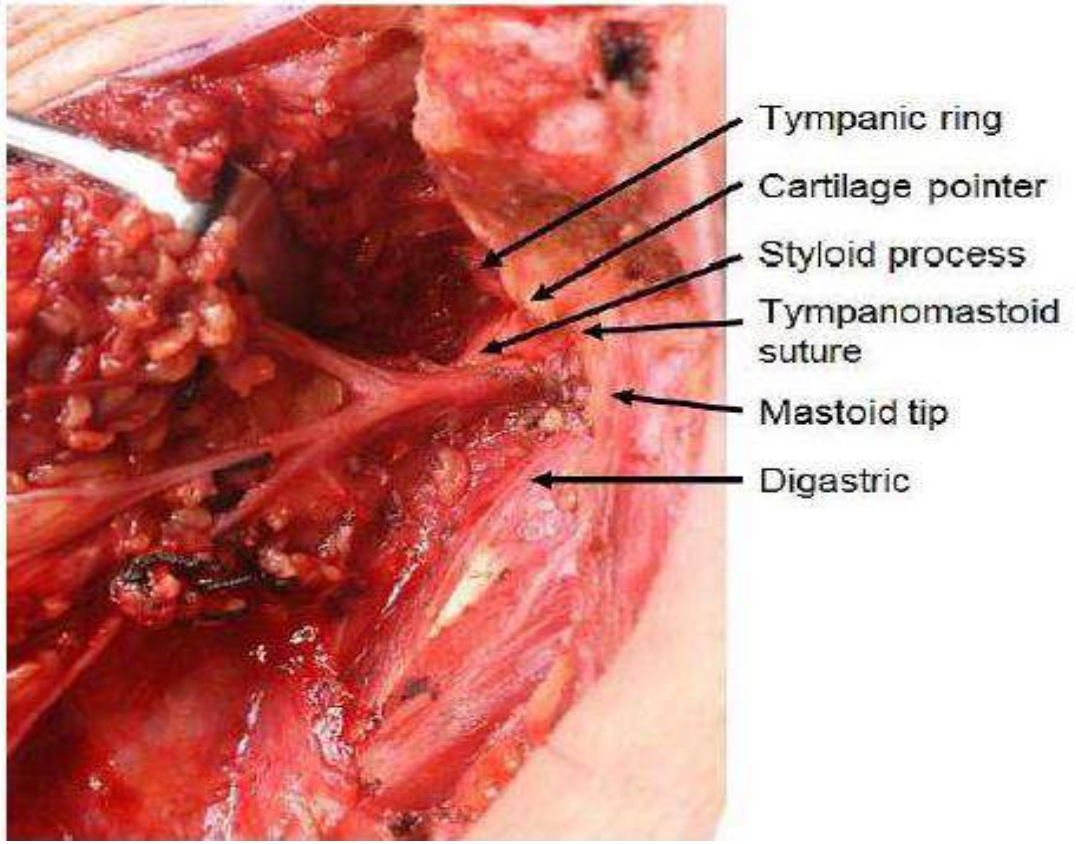
Incision and Flap Elevation

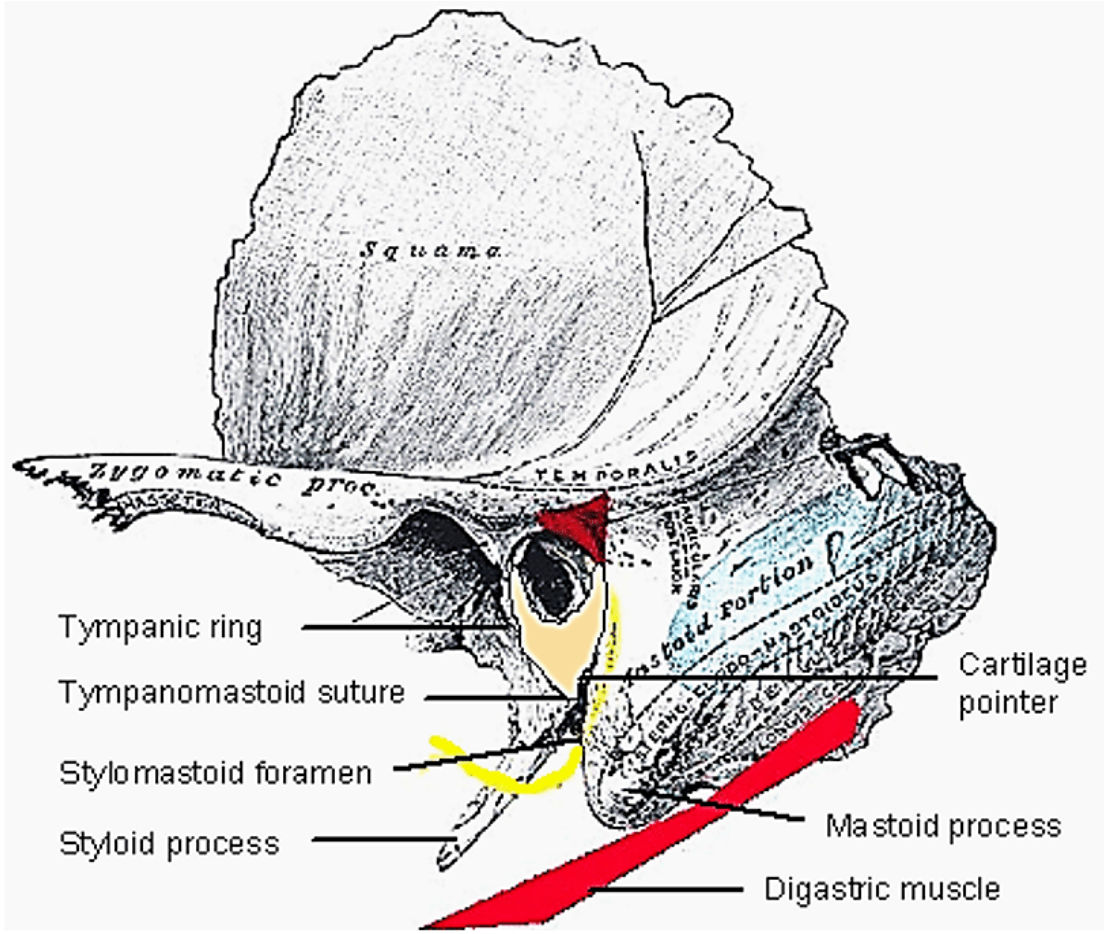
Mobilization of Parotid in Preparation for VIIth Nerve Dissection

identify the facial nerve after obtaining this wide exposure employing landmarks



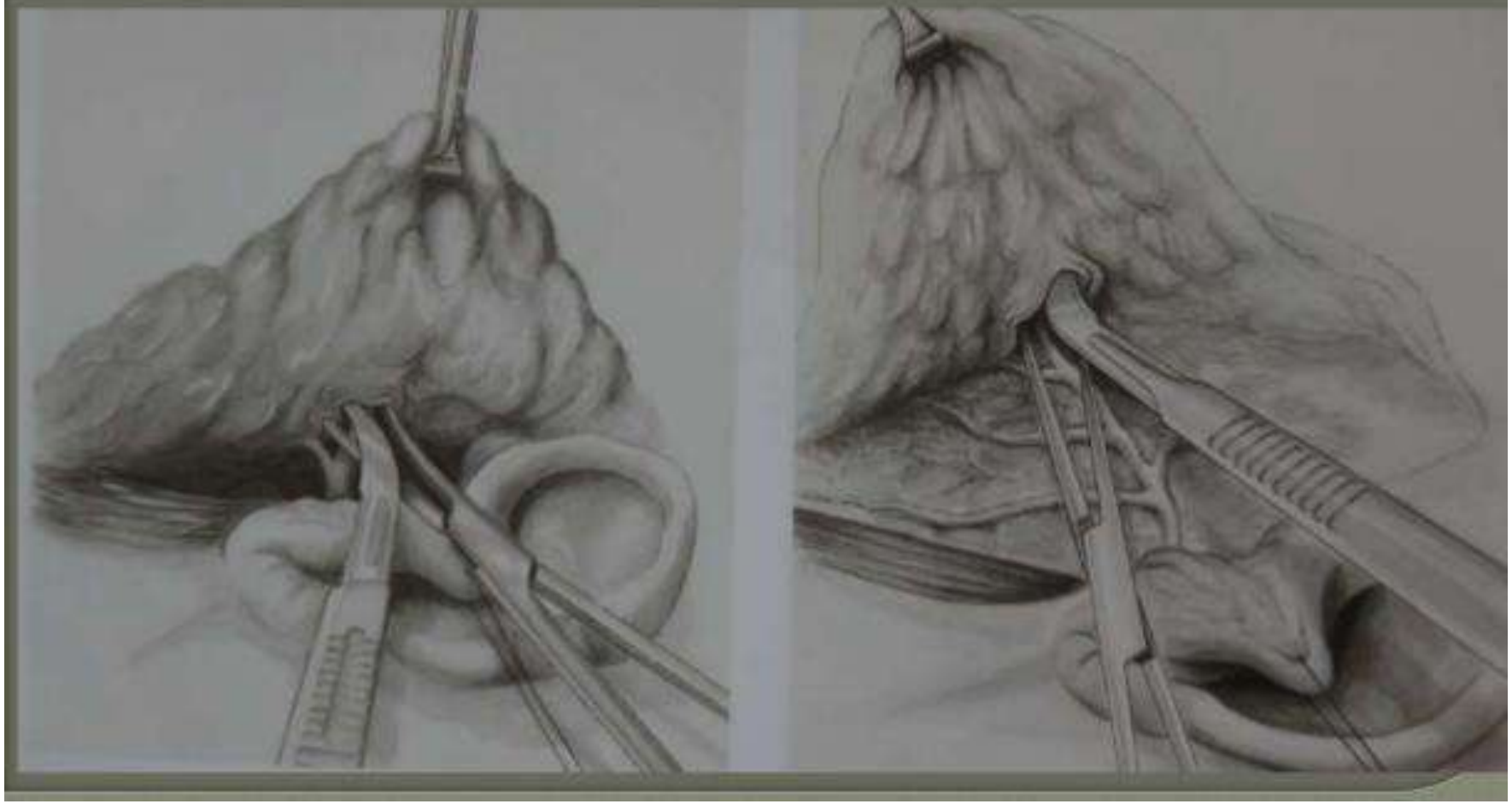
Landmarks for Identify the facial nerve





(cont.)

"Slide, lift, spread & cut technique" in Horizontal plane parallel to nerve

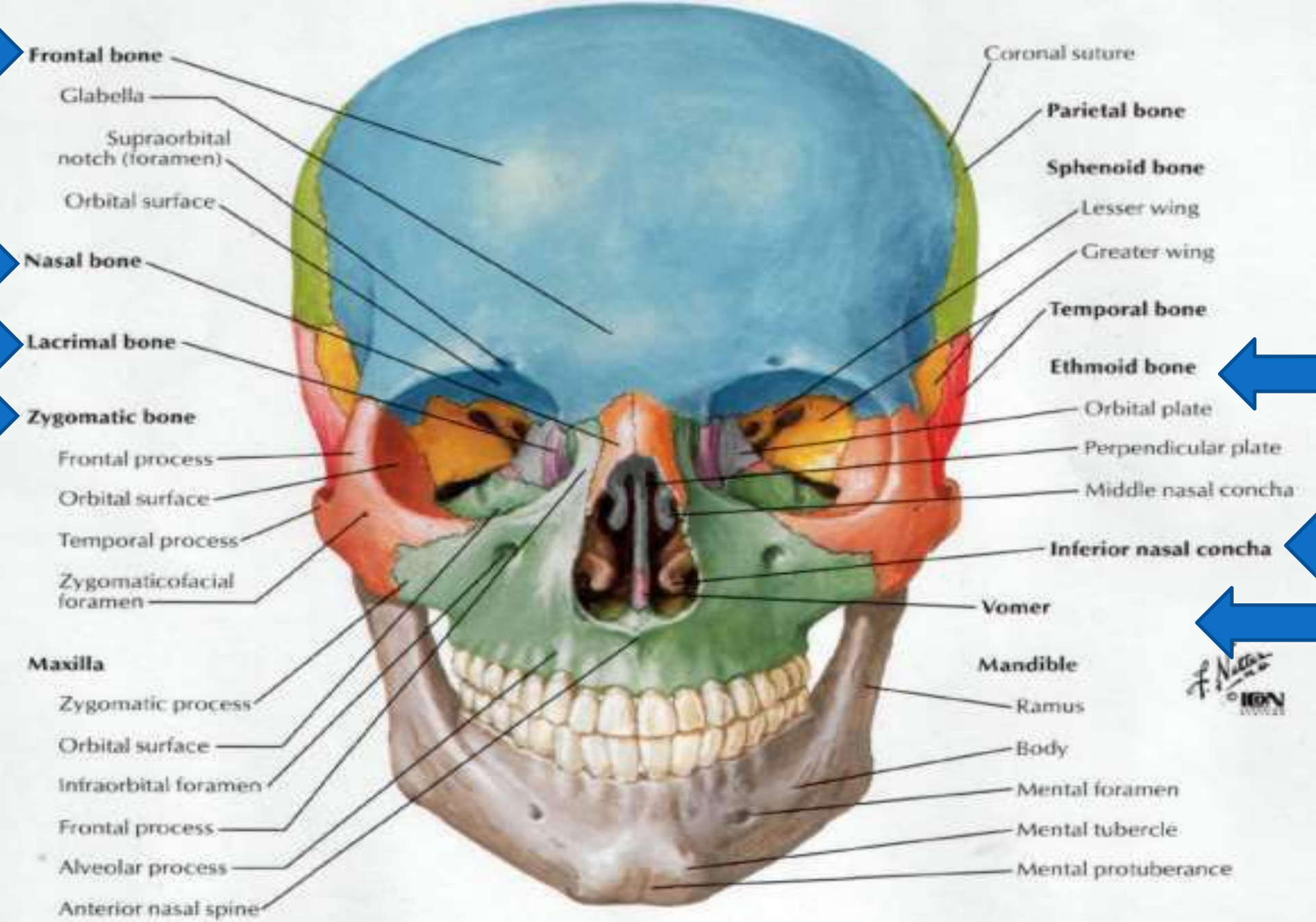


Maxillary fractures

By
Dr. Sabah Alheeti
CABMS., FIBMS

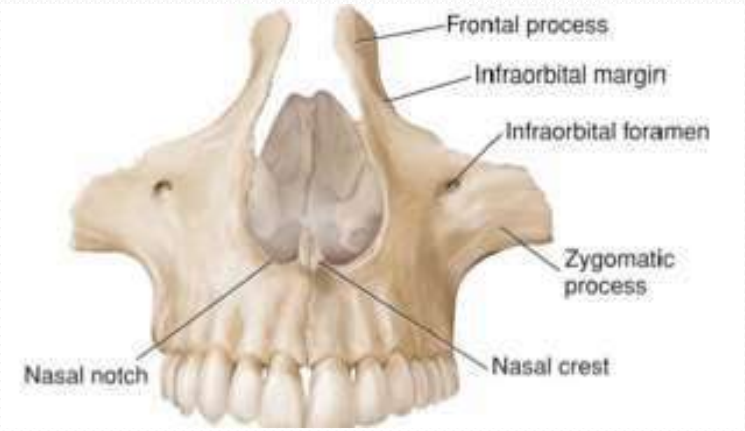
- Objective of lecture : Understanding the basic principles, diagnosis and management of maxillary fracture .
- Lecture outline the following

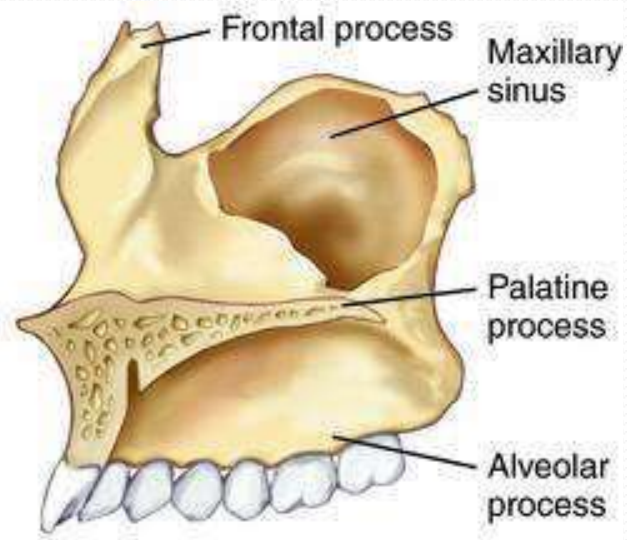
I. Anatomy
II. Clinical examination
III. Imaging
IV. Treatment
V. Complications


Frontal bone
Glabella
Supraorbital notch (foramen)
Orbital surface
Nasal bone
Lacrimal bone
Zygomatic bone
Frontal process
Orbital surface
Temporal process
Zygomaticofacial foramen
Maxilla
Zygomatic process
Orbital surface
Infraorbital foramen
Frontal process
Alveolar process
Anterior nasal spine
Coronal suture
Parietal bone
Sphenoid bone
Lesser wing
Greater wing
Temporal bone
Ethmoid bone
Orbital plate
Perpendicular plate
Middle nasal concha
Inferior nasal concha
Vomer
Mandible
Ramus
Body
Mental foramen
Mental tubercle
Mental protuberance

Four Processes

1. **Zygomatic**
2. **Frontal**
3. **Alveolar**
4. **Palatine**

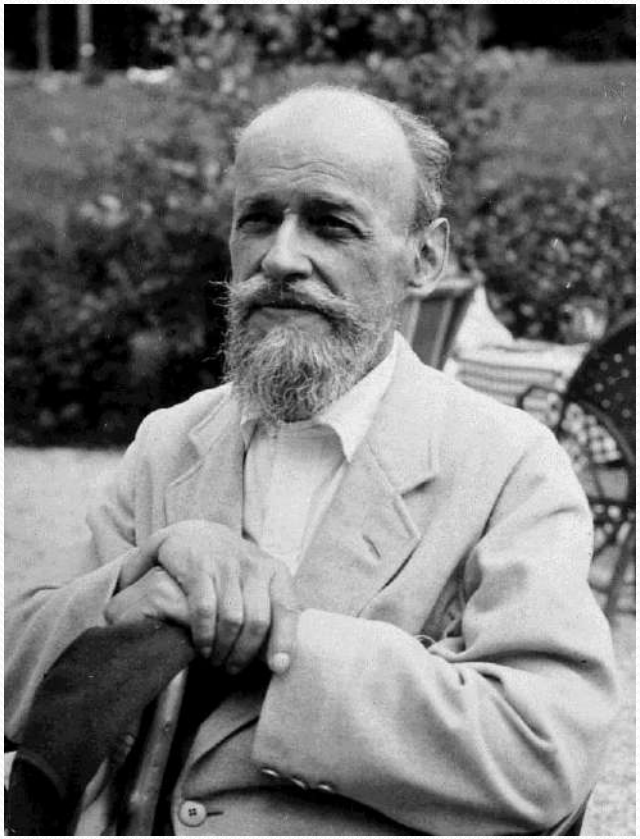


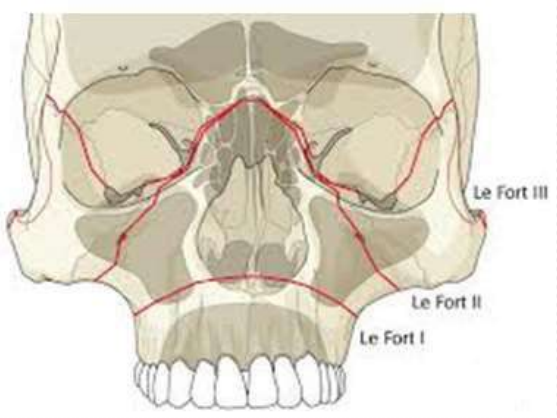


Classification of maxillary fractures

Rene Le Fort classification (1901) of maxillary fractures:

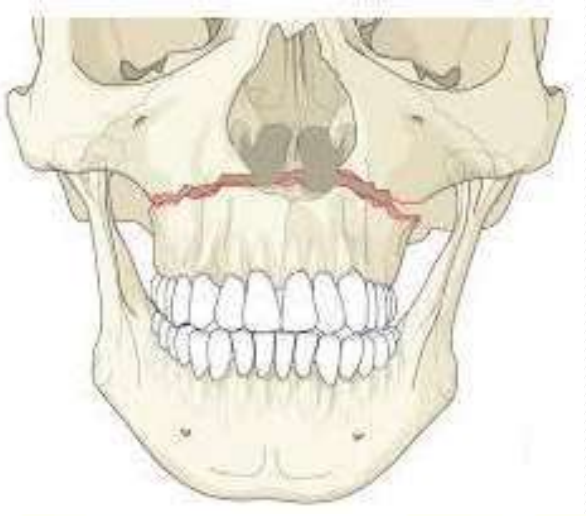
- **Le Fort I**
- **Le Fort II**
- **Le Fort III**

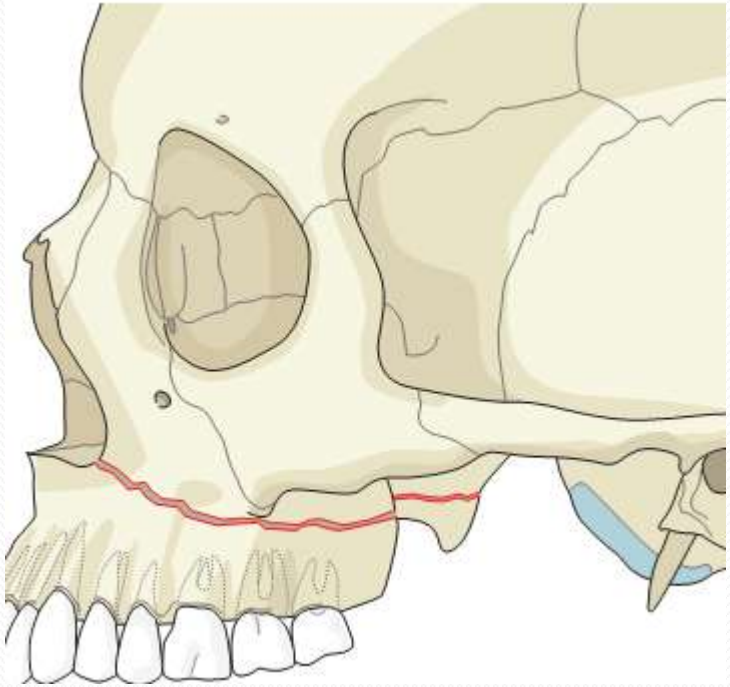




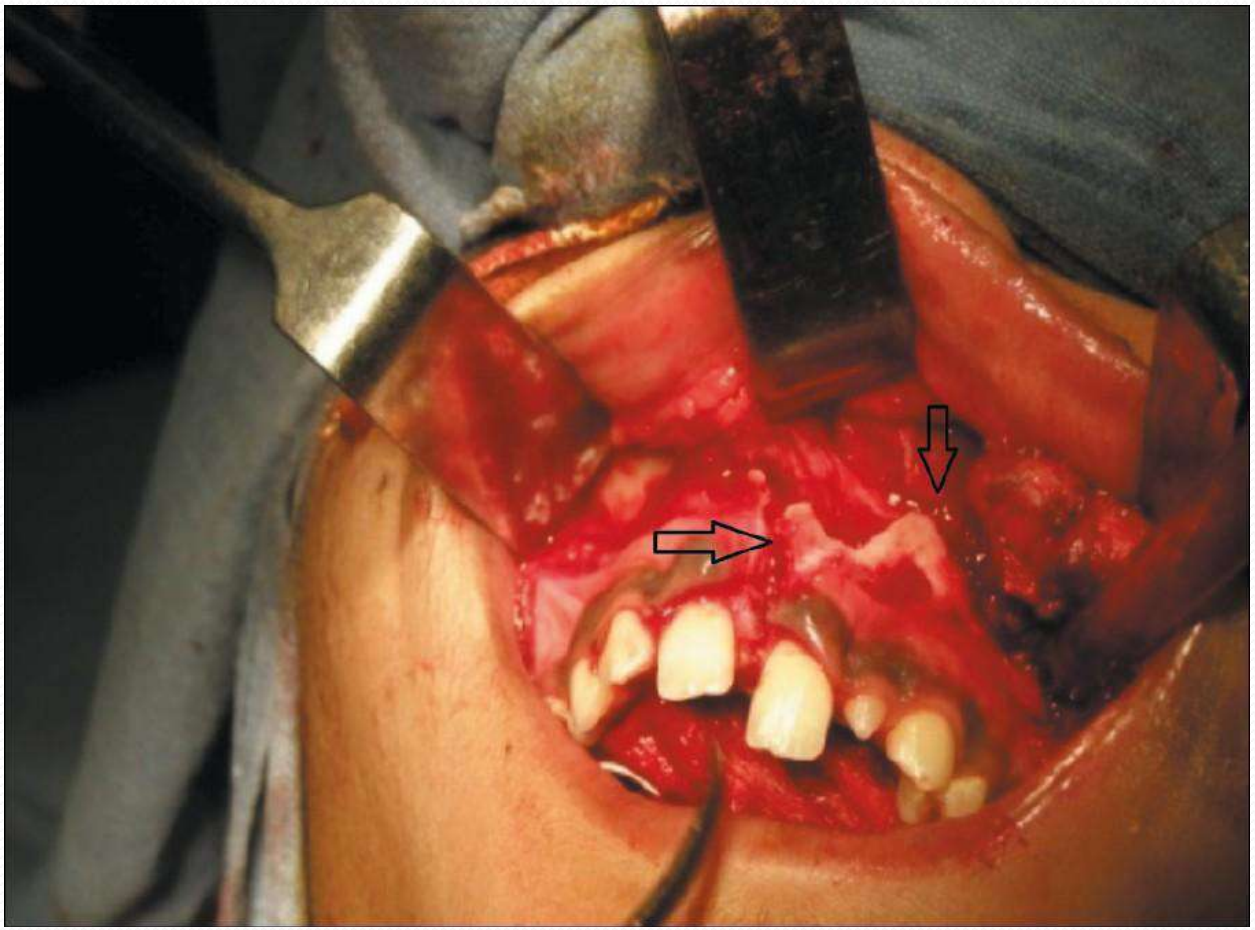
Le Fort I fracture

- ***The fracture line extends from* lower third of nasal septum** to the **lateral pyriform rims**,
- Travels horizontally above the teeth apices, crosses below the **zygomaticomaxillary** junction.
- Traverses the **pterygomaxillary** junction to interrupt the **pterygoid plates** (lower 1/3).





Le Fort I



Clinical features of Le Fort I fracture

- Slight Swelling of upper lip
- Ecchymosis–seen in maxillary buccal sulcus beneath each zygomatic arch
- Guerin sign: –Echymosis in palate at greater palatine foramen bilaterally
- Occlusion

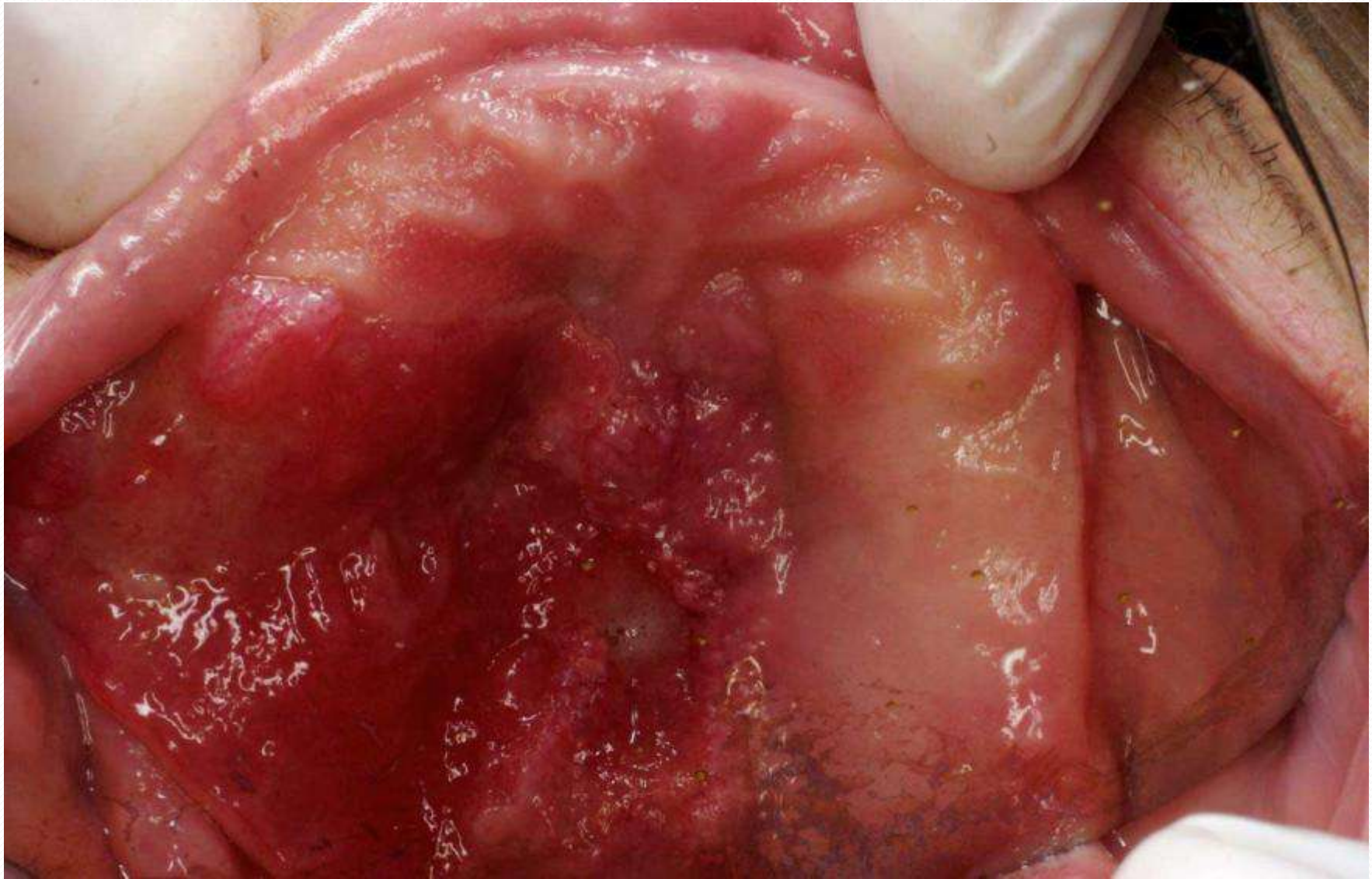
–Undisplaced incomplete fracture –no disturbance to occlussion

–Displaced occlusion

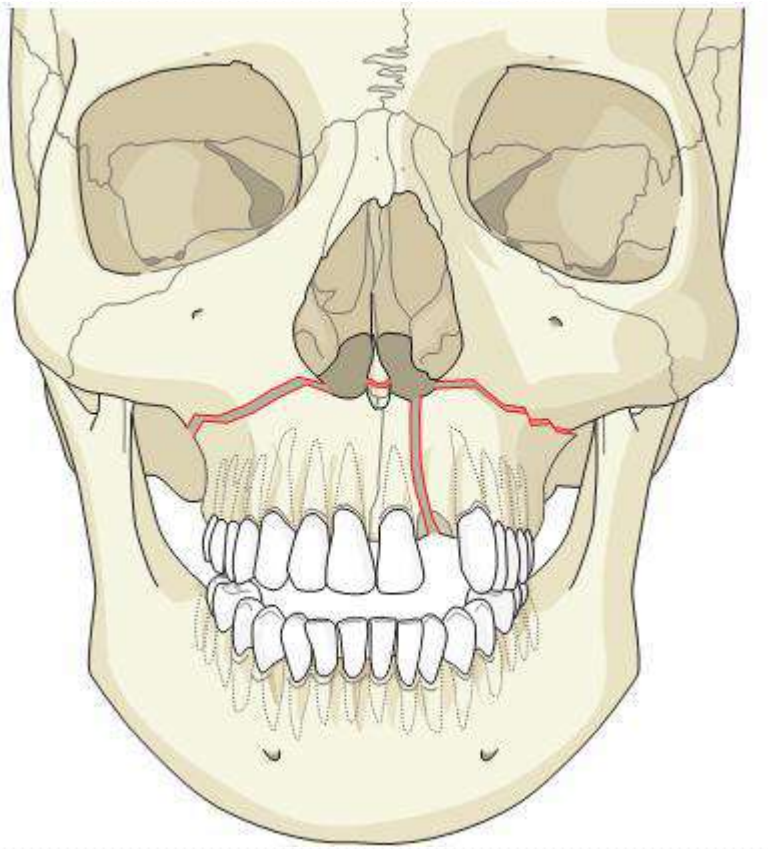
Anterior open bite : backward and downward distraction of posterior maxilla due to inferior and medial traction from medial and lateral pterygoid muscles.

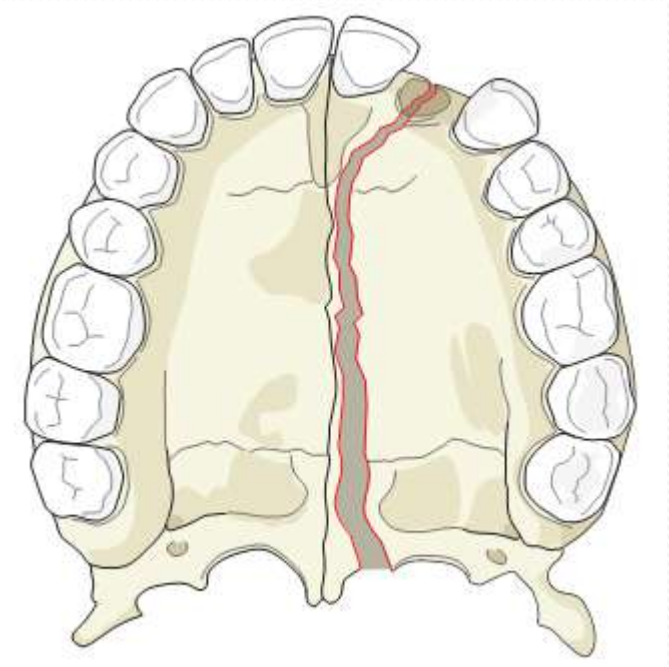
Posterior gagging of occlusion –threat to airway

Guerin sign



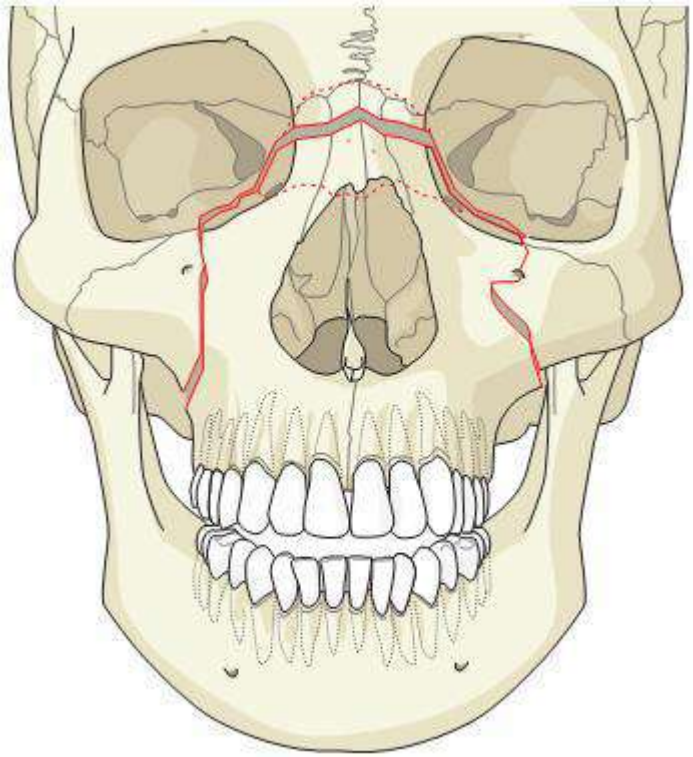
Palatal fracture

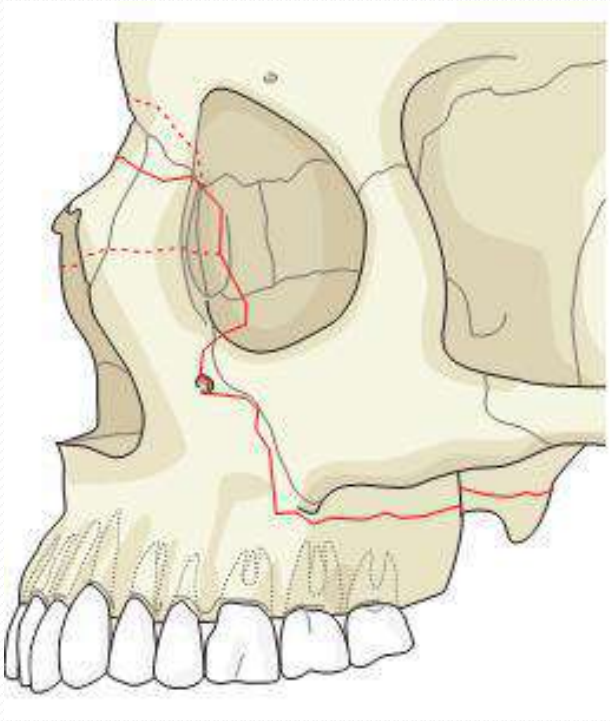




- Due to impaction of mandibular teeth against the maxillary counterpart, damage to cusp of individual maxillary tooth is seen (mostly in premolar region).
- Percussion of maxillary teeth results in distinctive" Cracked pot sound" similar to the sound produced when a cracked China pot is tapped with a spoon.
- The teeth and maxilla will move but the upper face and nose will stay fixed
- Sometimes fracture of palate may be associated with Le Fort I fracture.
- No tenderness over, or disorganization and mobility of zygomatic arch and bone

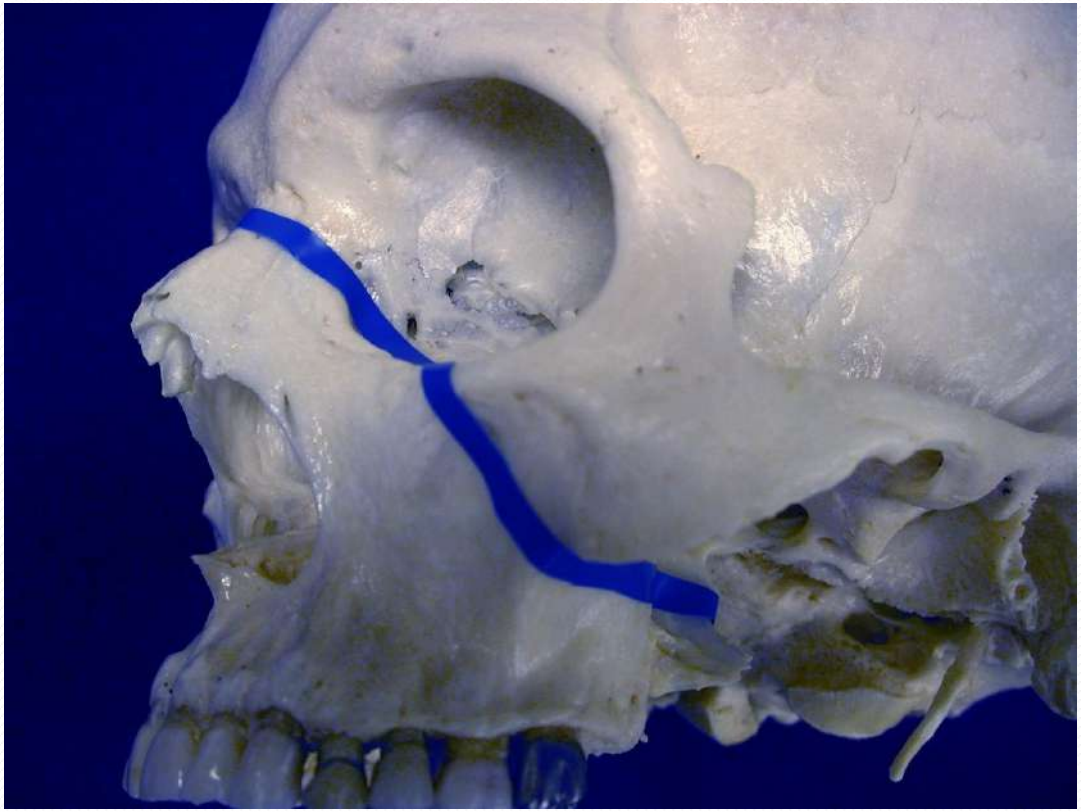
Le Fort II/Pyramidal fracture





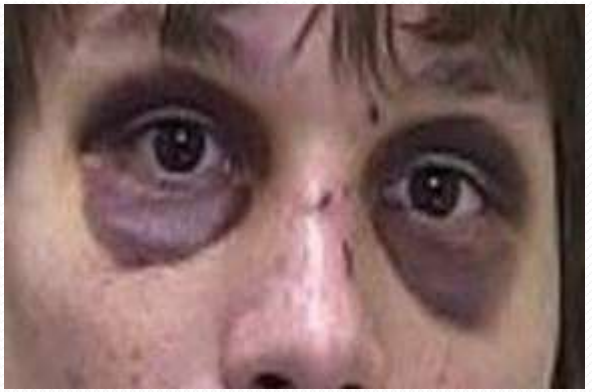
Le Fort II Level

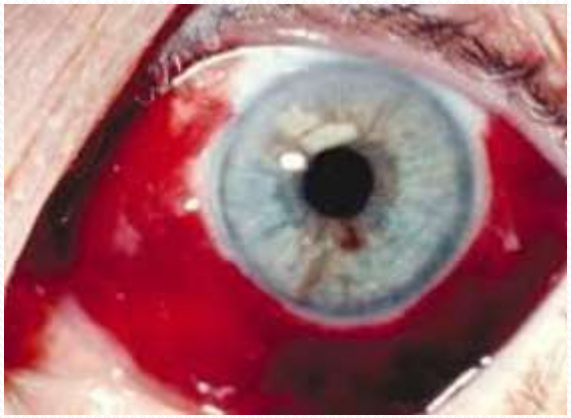
- Starts from **nasal bridge** at or below the nasofrontal suture through the frontal processes of the maxilla,
- then Inferolaterally through the **lacrimal bones** and **inferior orbital floor** and **rim** through or near the **inferior orbital foramen**,
- and inferiorly through the **anterior wall of the maxillary sinus**; It then travels under the **zygoma**, across the **pterygomaxillary fissure**, and through the **pterygoid plates**.



Clinical features of Le Fort II fracture

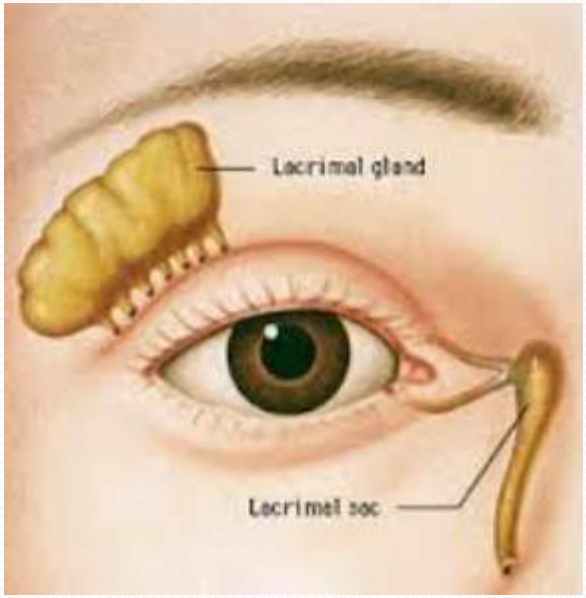
- Gross edema of the middle third of the face gives the appearance of **moon face** to the patient.
- Bilateral circumorbital edema and ecchymosis giving the appearance of '**raccon eye**' (**Black eye**)
- Bilateral subconjunctival hemorrhage -medial half of eye.





- '**Dish- face**' deformity with lengthening of face due to separation of middle third from the skull base.
- Pseudotelecanthus: swelling over the nasal bridge illusion of telecanthus, true telecanthus on the involvement of NOE complex.
- Diplopia may be seen in case of orbital floor fracture.
- Pupils are at level unless there is gross unilateral enophthalmos
- When maxillary teeth are grasped, the mid-facial skeleton moves as a pyramid and the movement can be detected at the infraorbital margin and nasal bridge.
- CSF leak may be present
- Step deformity at the infraorbital rims or nasofrontal junction is noticed.
- Anesthesia and/or paresthesia of the cheek is noted

Epiphora



Enophthalmos

- Posterior recession of eye.
- Due to volume changes

of the bony orbit relative to globe and soft tissues

Telecanthus

Patient with Lefort II fracture (note telecanthus and face lengthening)

Le Fort II

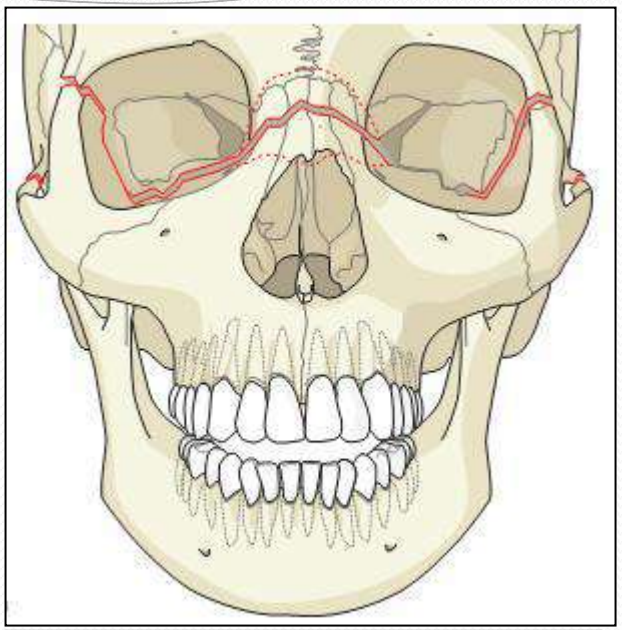
- Displacement of maxilla = malocclusion

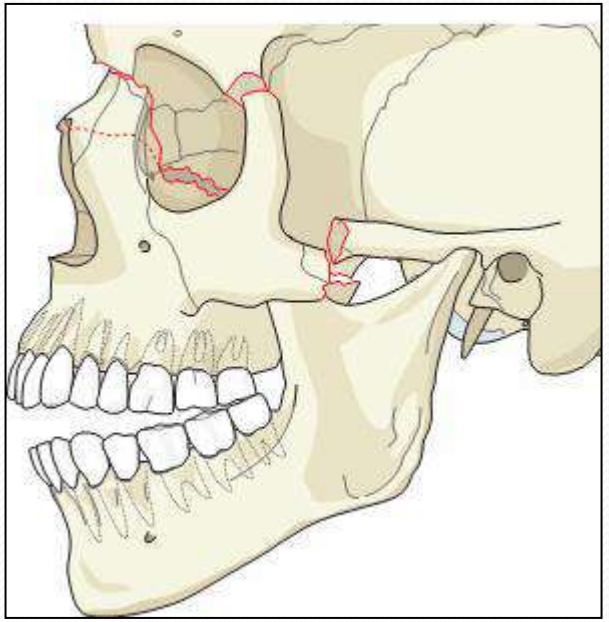
Most common abnormality is an open-bite deformity

- Dish face deformity from blow to the front

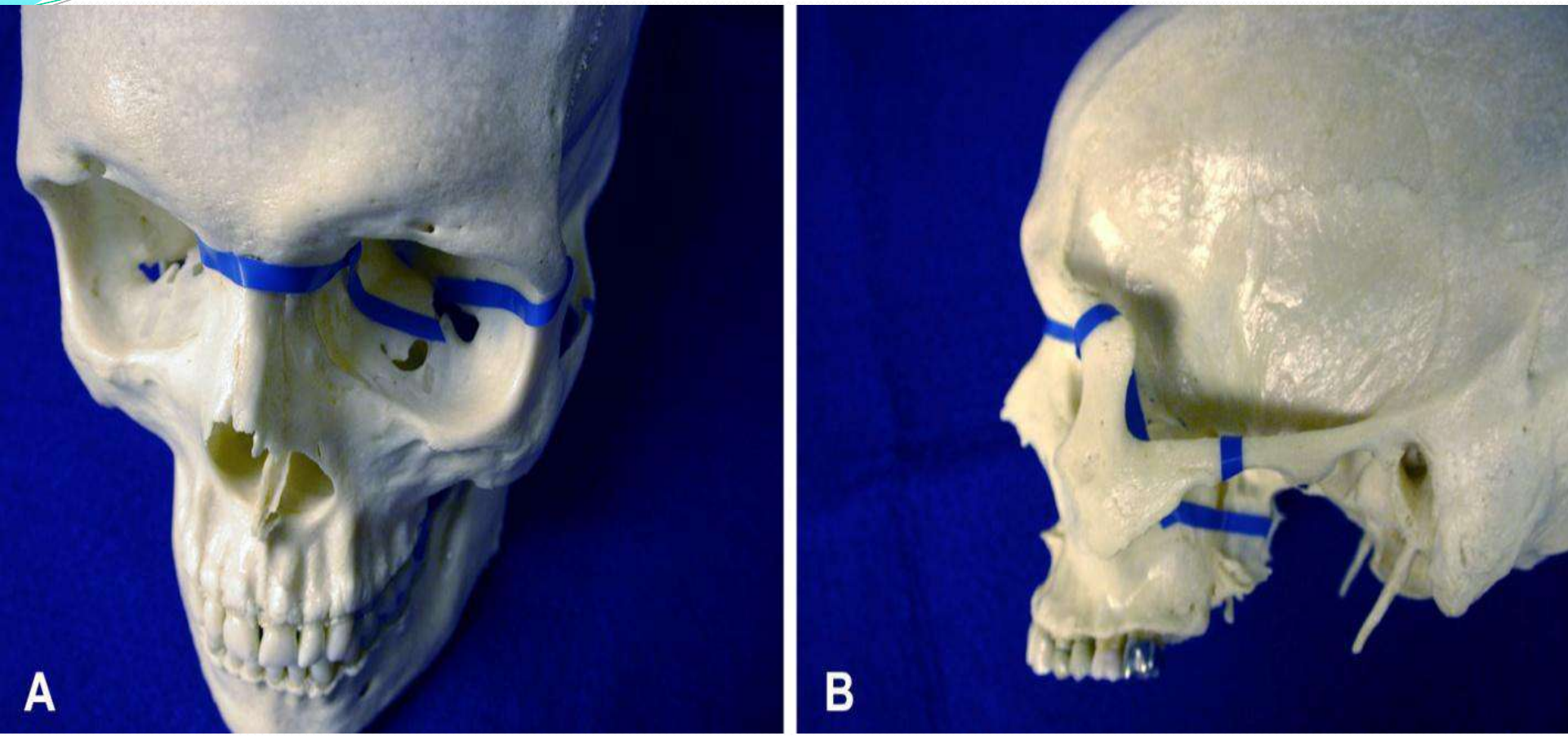
Le Fort III

- The fracture line extends from the nasofrontal suture along the medial wall of the orbit through the superior orbital fissure.
- It then extends along the inferior orbital fissure and the lateral orbital wall to the zygomaticofrontal suture.
- The zygomaticotemporal suture is also separated.
- The fracture then extends along the sphenoid bone, separating the pterygoid plates.





Le Fort III



Clinical features of Le Fort III fracture

- Mobility of whole facial skeleton as a single block can be felt
- Gross edema of the face.
- Bilateral circumorbital/periorbital ecchymosis and gross edema 'Racoon eye
- Bilateral subconjunctival hemorrhage

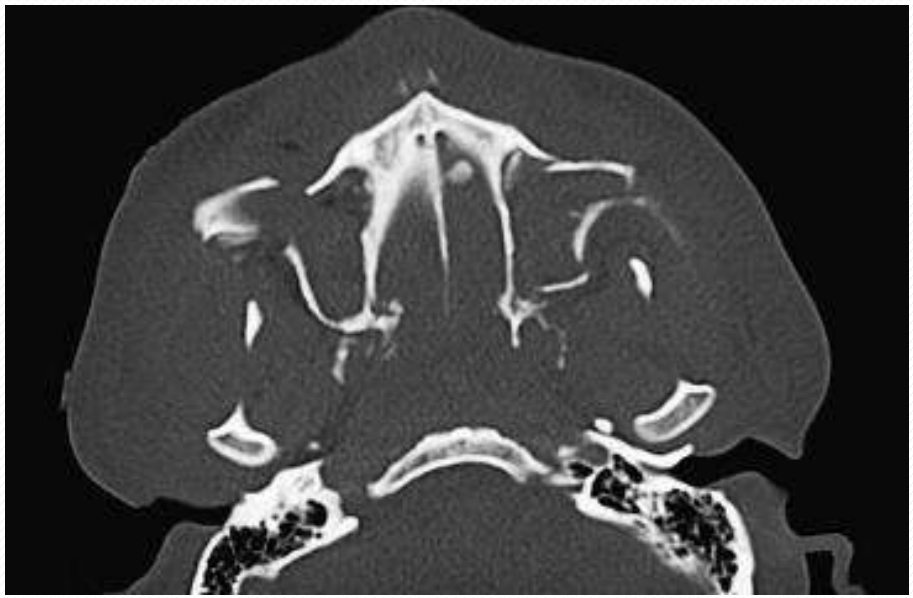
- Tenderness and separation of the bones at the frontozygomatic sutures
- Characteristic ‘dish face’ deformity with lengthening of the face.
- 'Hooding of the eyes' may be seen due to separation of the frontozygomatic sutures
- When lateral displacement has taken place tilting of occlusal plane and gagging of one side is seen.

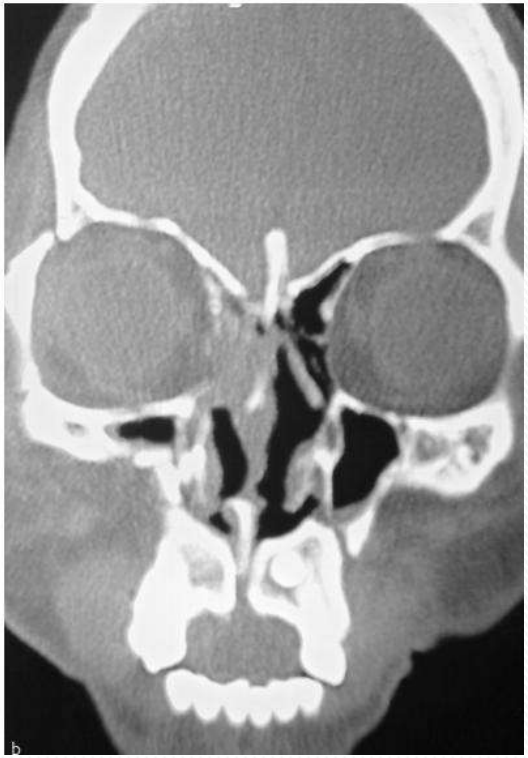
- Deformity of the zygomatic arches.
- Disorganization and lengthening of the nasal skeleton
- Epistaxis, CSF rhinorrhea
- Depression of ocular level
- Difficulty in opening the mouth, inability to move the lower jaw.

HOODING OF EYE (PTOSIS)

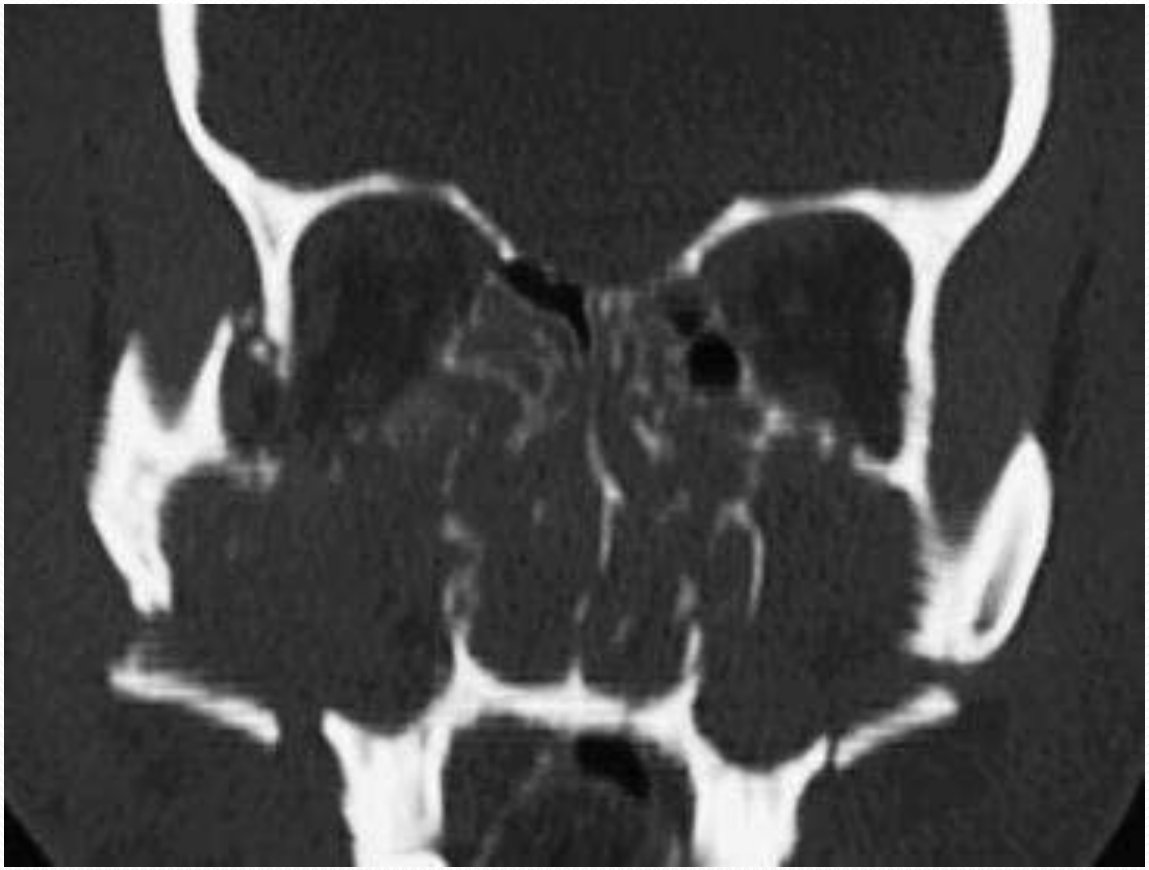
Imaging

- **CT has become the standard to delineate extent and severity of midface fractures** (Axial and coronal views)

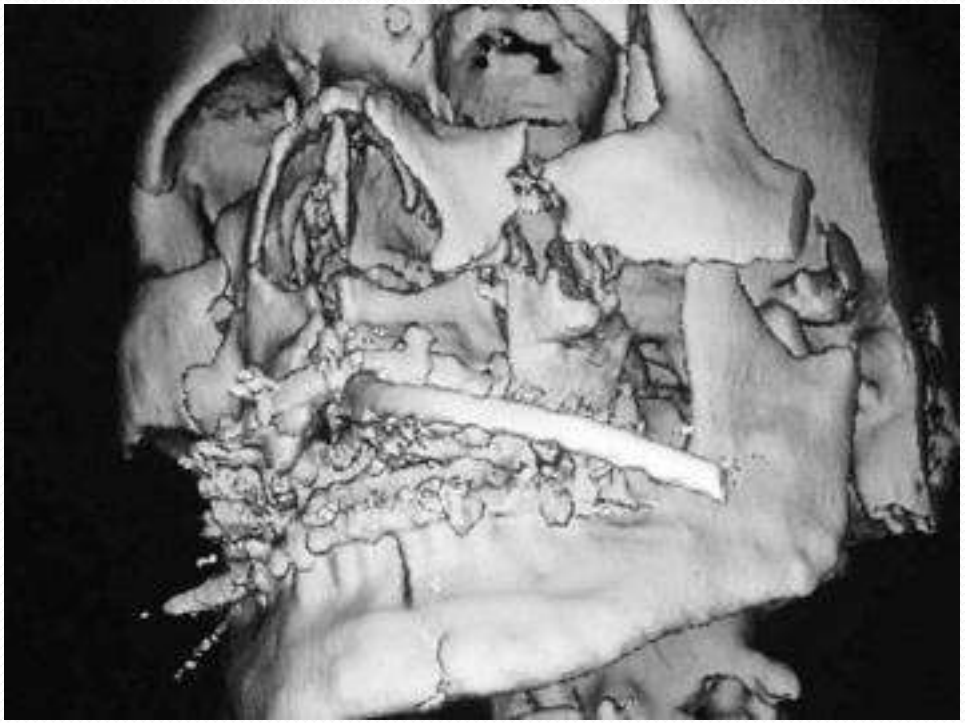




Le Fort III on right side
LE Fort II Bilateral

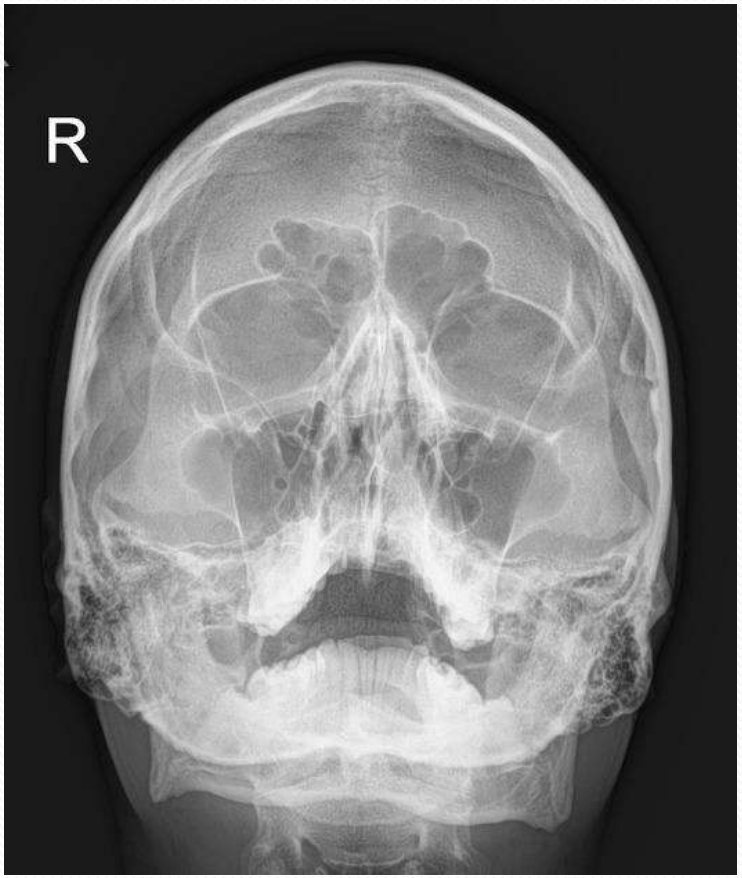


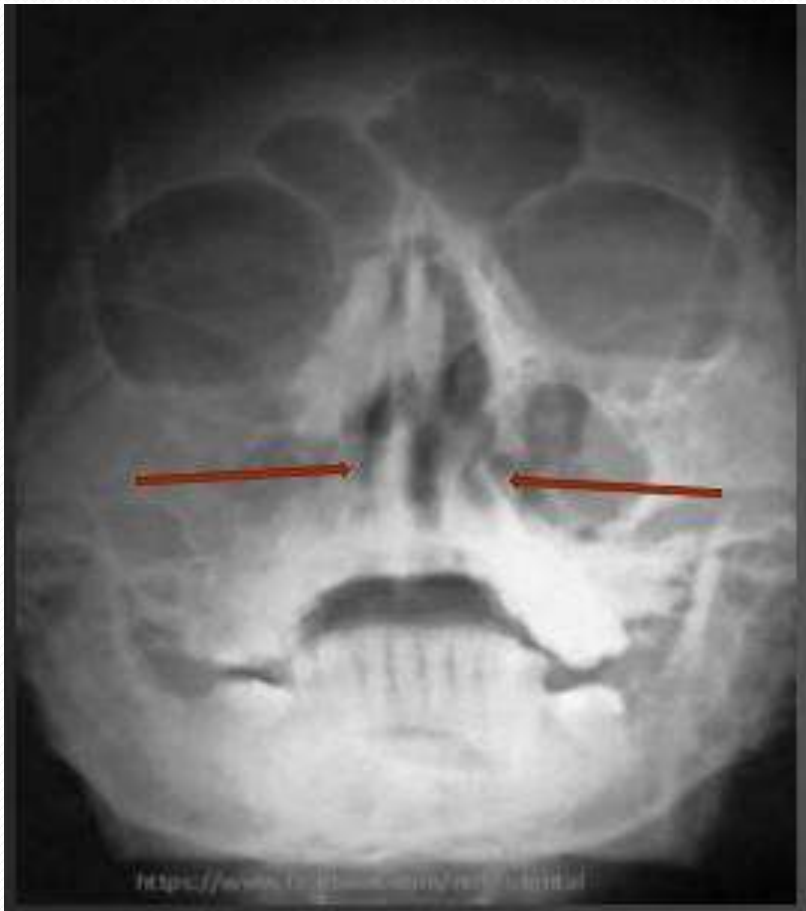
CT (3D)



Plain radiography

- Occipito-mental view 30 degrees (waters view)





LEFORT I – Waters view

Water's View

The most comprehensive single projection display

Excellent view of

- Maxilla
- Maxillary sinuses
- Zygoma
- Zygomatic arches
- Rims of orbits, esp. floor
- Nasal bones

Surgical treatment planning for maxillary fractures

Management of Facial fractures

1. Resuscitation of patient
2. Reduction of fracture
3. Fixation of fracture

Timing

(A) Emergency treatment- resuscitate patient

1- Maintain airway (need for tracheostomy, stabilize the mobile fractures…)

2- Arrest the haemorrhage and transfuse if necessary

3- Monitor the vital signs

(B) Within 24 hours

1- Repair deep laceration

2- Impression of teeth

3- Treat less severe maxillary fractures if no other major injuries

(C) Definitive treatment (2-8 days): is optimal time to allow

1- Improvement in medical condition of patient

2- Careful clinical assessment and planning

3- Reduction of soft tissue oedema and easier manipulation of bones

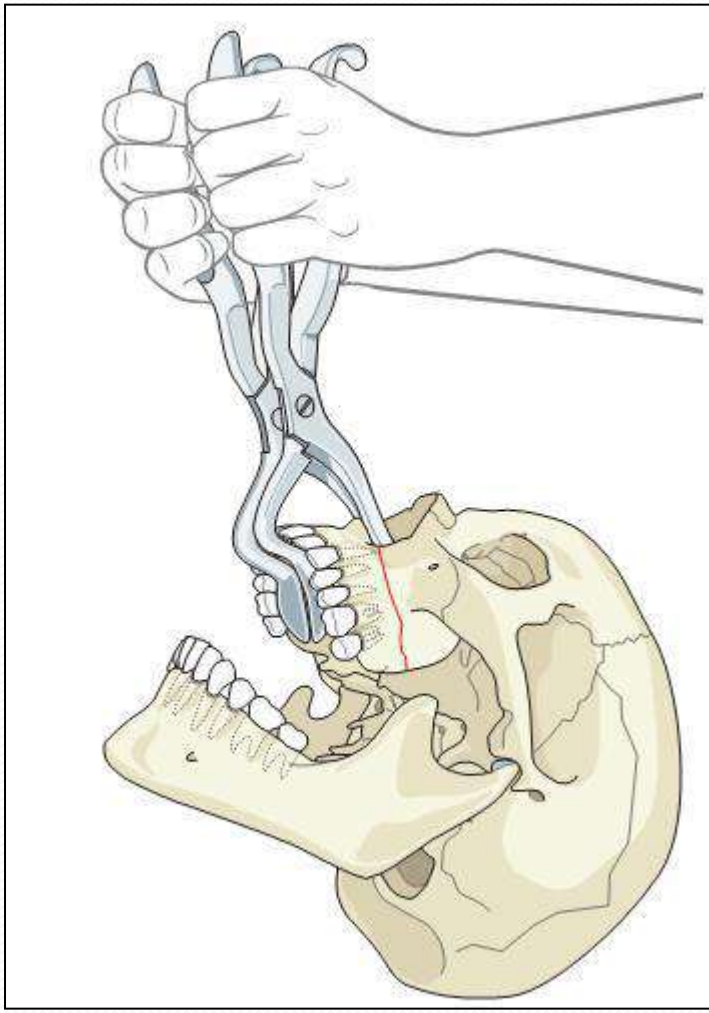
Reduction

- **Loosely mobile** : Finger manipulation
- **Impacted**: Rowe's William forceps

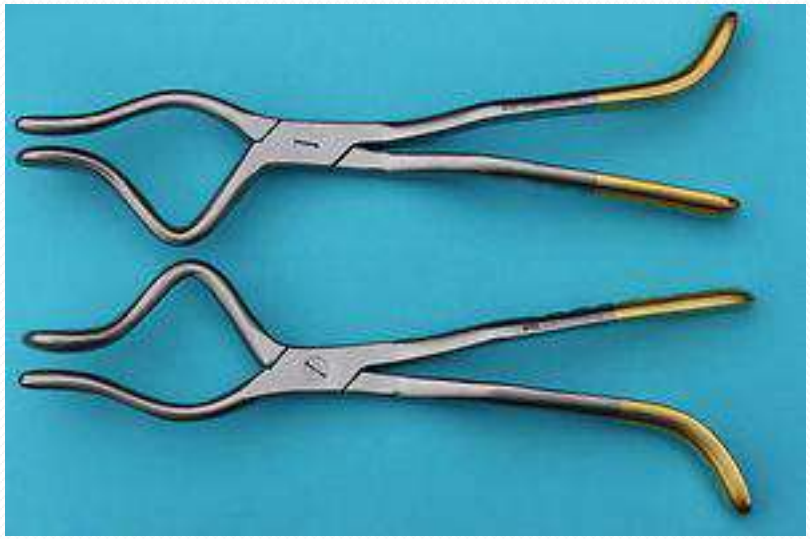
–Padded blade is inserted inside the mouth and unpadded in nostril

–Standing from behind, grasping the two forceps, fracture segment is manipulated

- **Firmly impacted** : fracture line should be exposed and mobilised using osteotome and disimpacted forceps



Rowe disimpaction forceps



Hayton Williams forceps

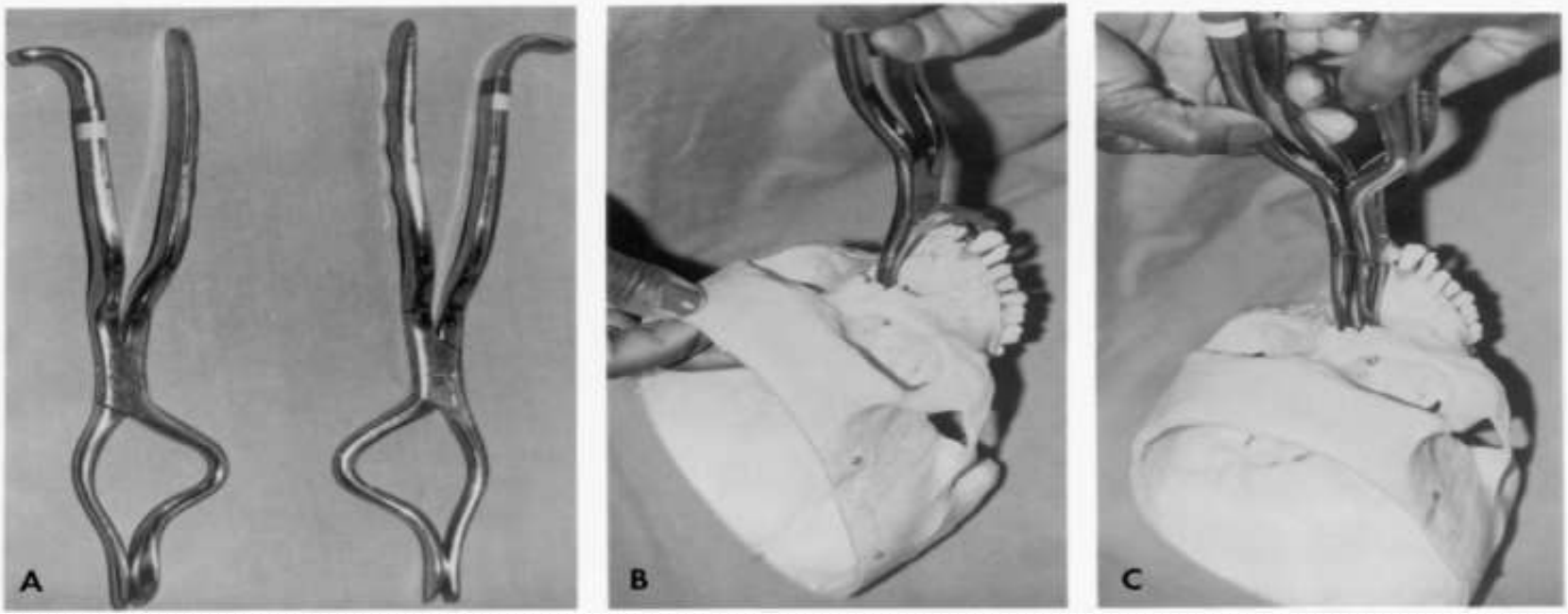
Displaced fractures may require disimpaction before placement in intermaxillary fixation

- Rowe disimpaction forceps

I. Straight blade placed along nasal floor

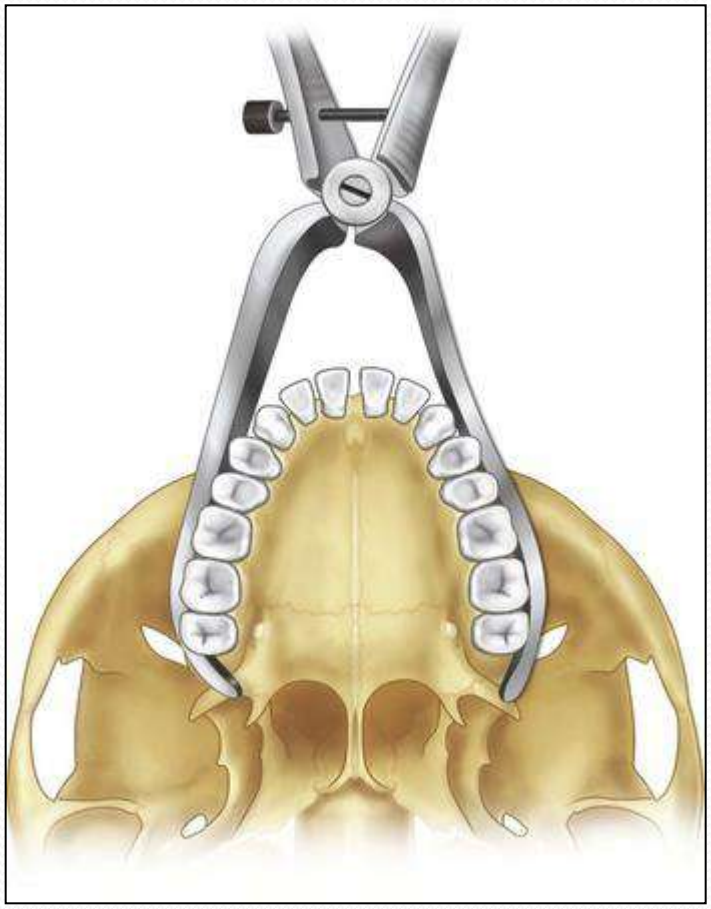
II. Curved blade along palate

Rocking motion with constant anterior traction to disimpact the fracture.



Hayton Williams forceps

- Alternative to Rowe disimpaction forceps
- In case of palatal split, two palatal halves are approximated by traction applied by HAYTON-WILLIAM forceps and then with Rowe's William forceps



Reduction of Le Fort II

- Disimpaction is carried out in a similar manner as in Le Fort I fracture using Rowe's disimpaction forceps. Extreme care should be taken because this fracture usually involves base of skull.

Reduction of Le Fort III

- Le Fort III fractures usually occur in association with other fractures of facial skeleton like orbital and nasoethmoidal fractures.

Table 28-2 Sequence B: Top Down and Outside In*

1. Tracheostomy
2. Repair of frontal sinus fracture
3. Repair of bilateral zygomati-comaxillary complex (including arch) fracture
4. Repair of naso-orbitoethmoid fracture
5. Repair of Le Fort fracture (including midpalatal split)
6. Maxillomandibular fixation
7. Repair of bilateral subcondylar fractures
8. Repair of mandibular fracture (symphysis/body/ramus)

Fixation

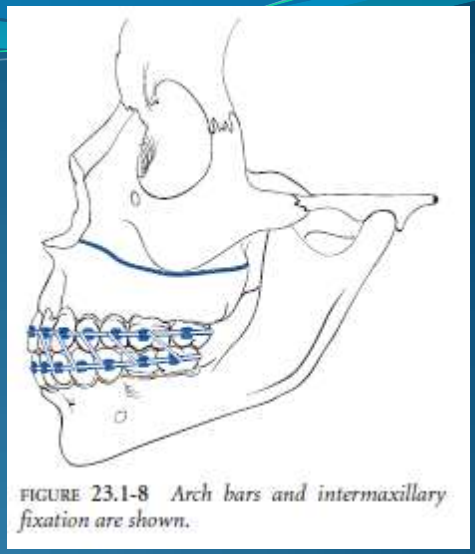
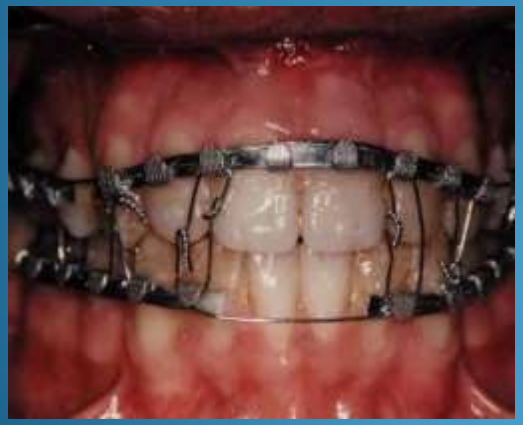


FIGURE 23.1-8 *Arch bars and intermaxillary fixation are shown.*



1- Internal skeletal fixation

a- Direct osteosynthesis (preferred method of treatment)

- Miniplates and screws
- Transosseous wires

b- Transfixation : K- wire

c- Wire suspension (ancillary method of treatment)

i. Frontal
ii. Circumzygomatic
iii. Infraorbital
iv. Pyriform aperture
v. Peralveolar

2- External skeletal fixation (less frequently used method of treatment)

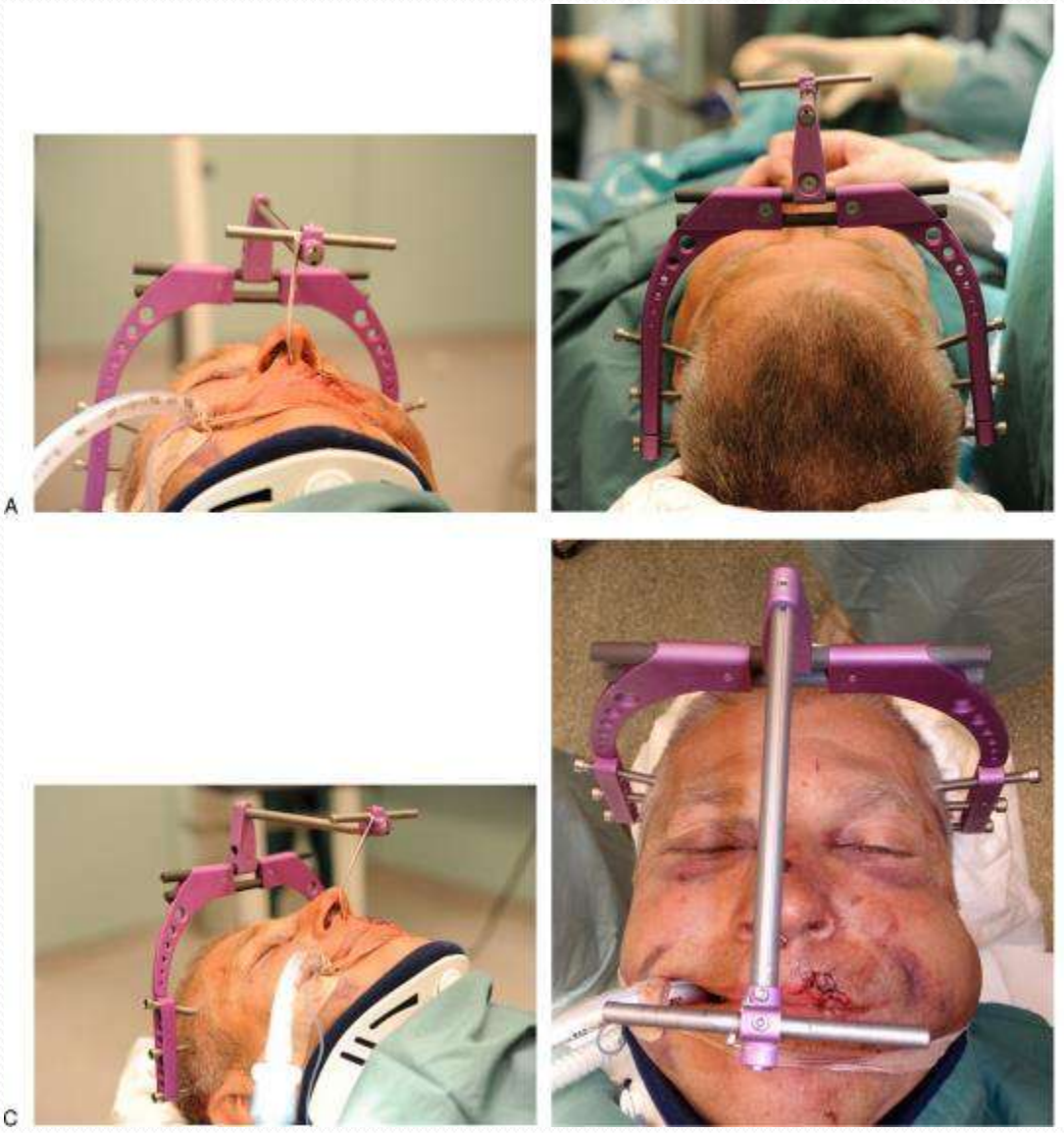
a- Craniomandibular

i. Haloframe
ii. Box frame
iii. Plaster of paris

b- Craniomaxillary

i. Pin fixation
ii. Haloframe
iii. Plaster of paris

External skeletal fixation



A. Craniomandibular fixation: Mandible is fixed by means of rods and universal joints to the cranial vault and the fractured middle third is sandwiched in between.
B. Craniomaxillary fixation: after establishing the occlusion, the upper jaw is attached by means of rods and universal joints to the cranial vault.

2. External fixation

a. Craniomandibular

i. box frame system

ii. halo frame

iii. Plaster of paris head cap

b. Craniomaxillary

i. Supraorbital pins

ii. Zygomatic pins

iii. Halo frame

BOX FRAME

HALO FRAME

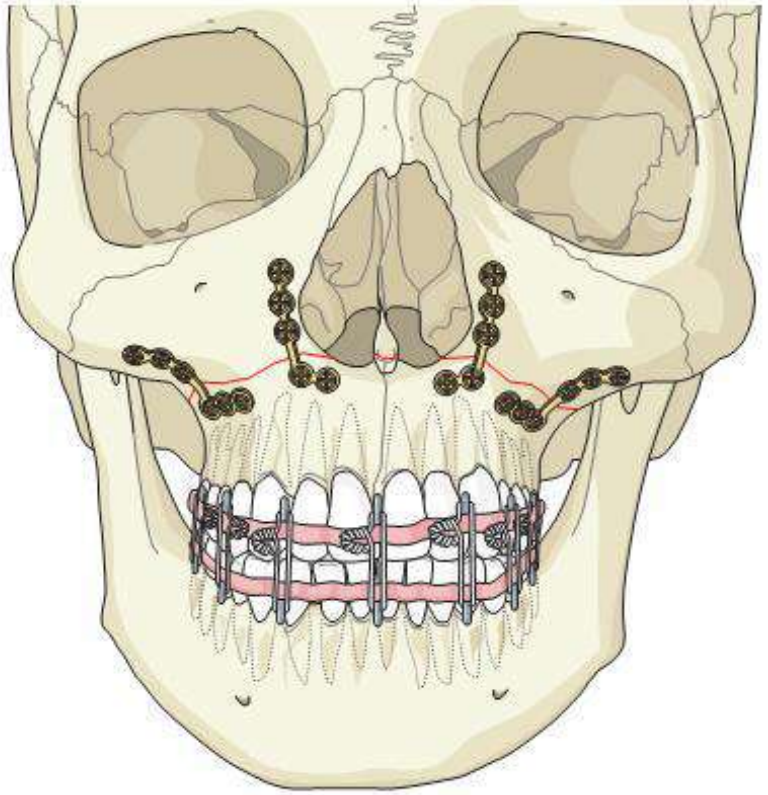
POP HEAD CAP WITH METAL FRAME

Internal skeletal fixation

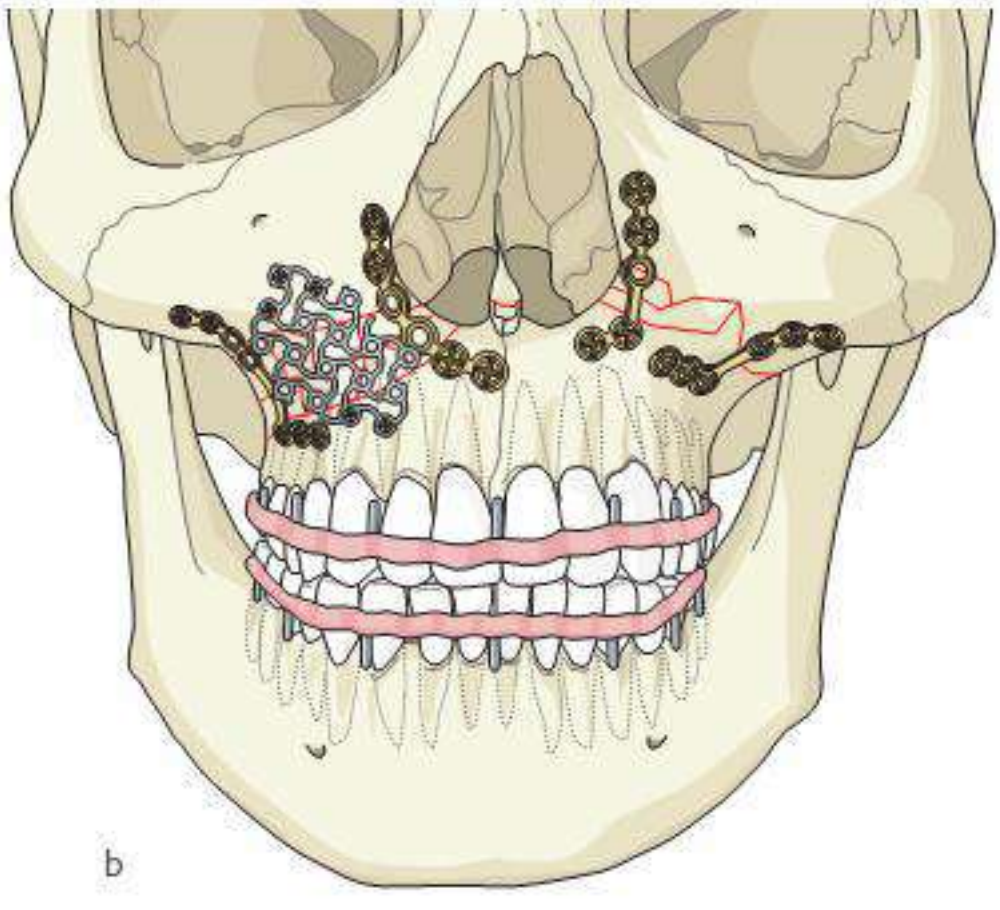
Direct
–Miniplate: Stabilization with L-shaped miniplates(1.5 or 2.0), Fixation with at least two screws on either side of the fracture line in order to avoid rotational instability.

Advantages

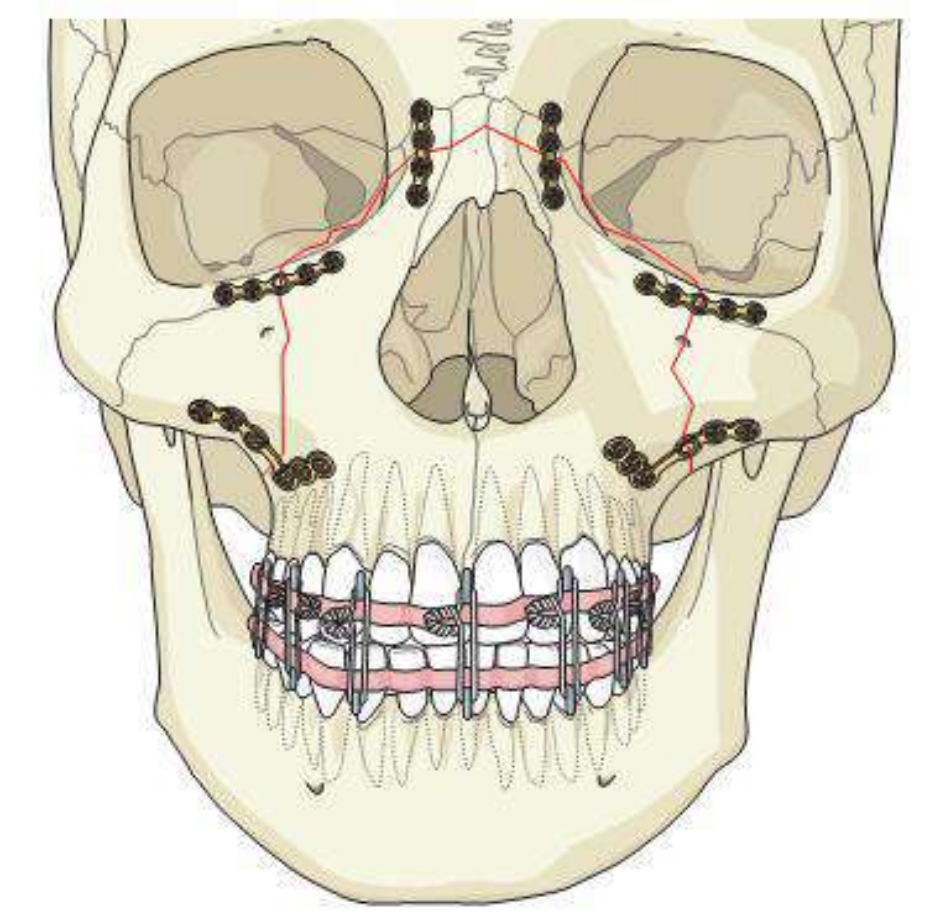
- ❑ Permit primary bone healing
- ❑ Increased three - dimentional, mechanical and functional stability.
- ❑ Precise anatomical reduction and enhanced bone healing
- ❑ No additional fixation required
- ❑ Greater patient comfort- function restore early
- ❑ Intermaxillary fixation not required- airway maintained and patient can eat



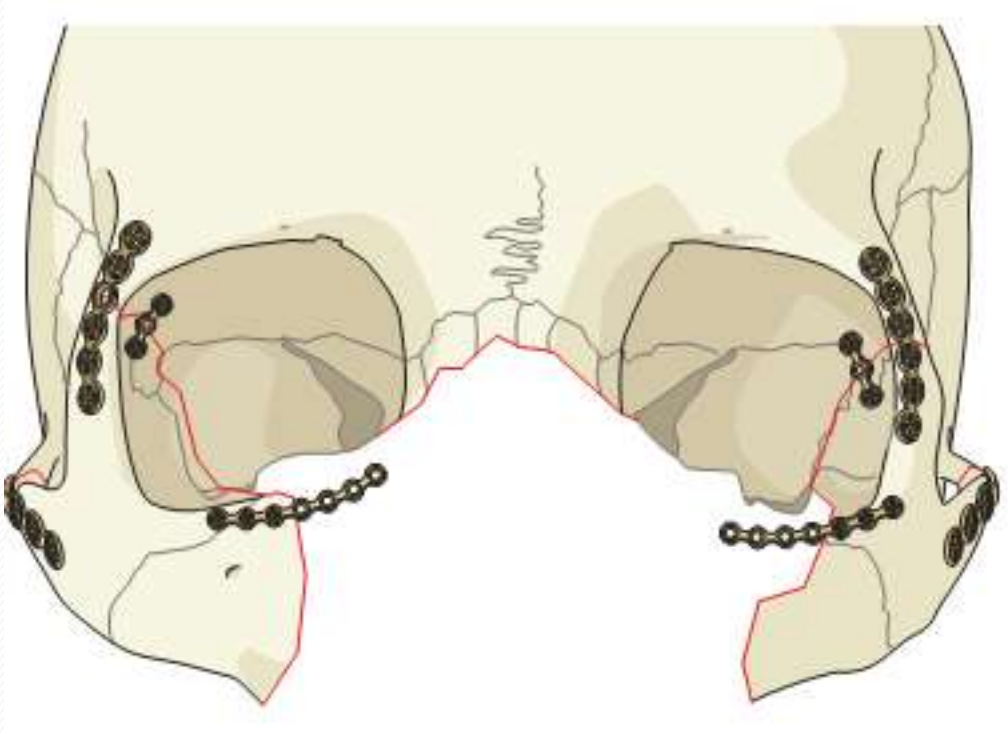
Stabilization with longer miniplates bridging the areas of comminution by titanium mesh



Fixation of Le Fort II



Le Fort III

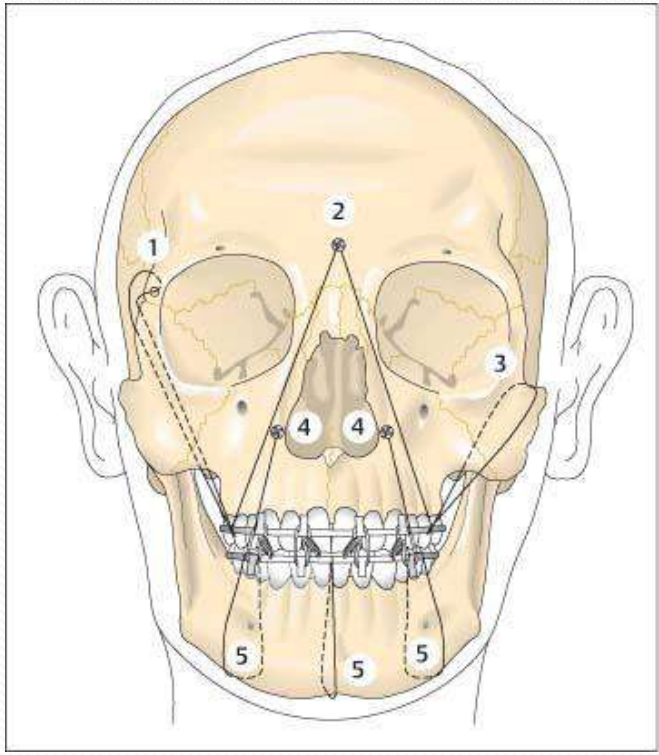


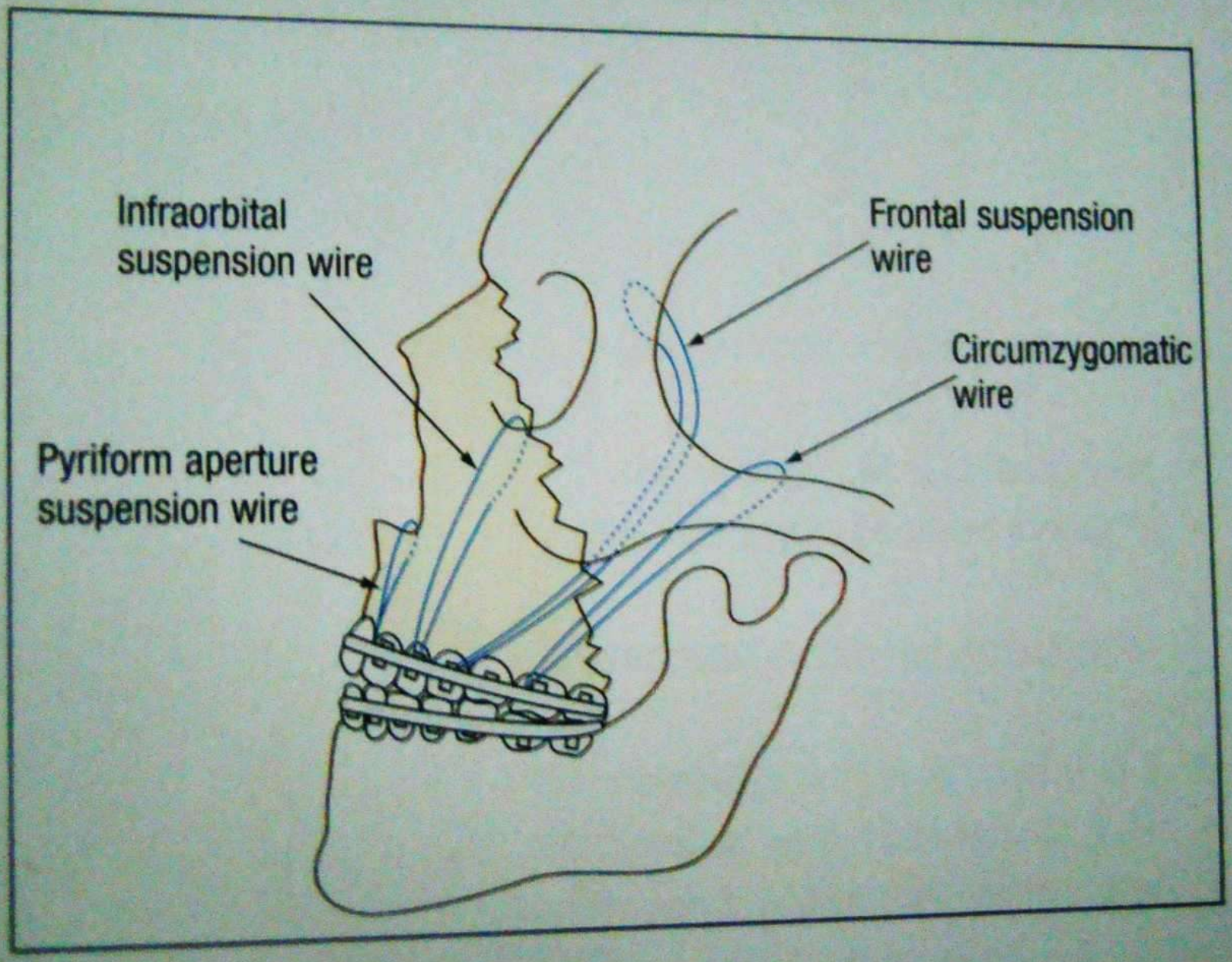
Disadvantages

- Stress- shielding
- Expense
- May interfere with CT scans and radiotherapy
- Risk of screws damaging teeth or nearby vital structures
- Infection may necessitate removal of plate
- Wound dehiscence

Wire suspension

- *Was used to reduce and suspend a mobile fragment below to a firm stable fragment above the fracture by means of a 0.5 mm stainless steel wire.*
- *Frontal suspension*
- *infraorbital suspension*
- *Circumzygomatic*
- *Piriform aperture*
- *Peralveolar*

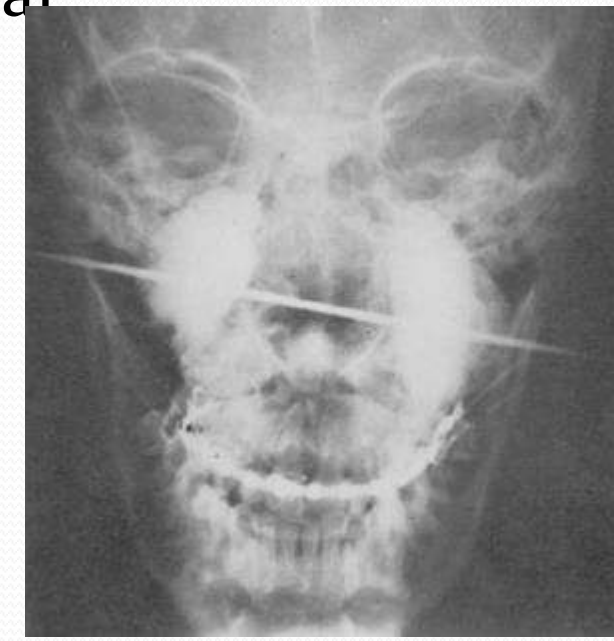




sta-

Transfixation via K wire

- For Transfixing the Maxilla. Pass the wire from one zygoma to the other. Palpate the bone about I inch behind and below the outer canthus and introduce the wire here; no starting hole is required. Wires introduced in this way rarely leave a scar

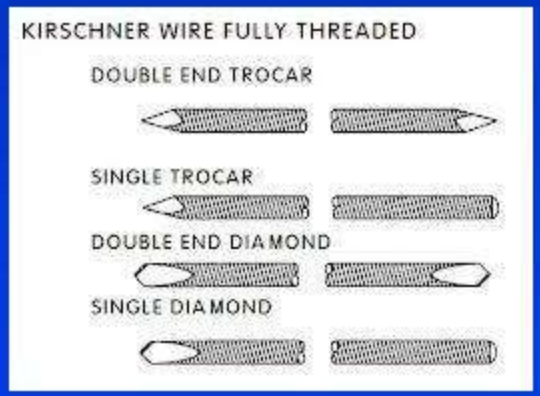


K-WIRES AND STEINMANN PINS

Trocar or diamond end

Smooth or threaded

Single or double



Complications of maxillary fractures

Table 23.1-1 Complications Associated with Maxillary Fractures
Infraorbital nerve paresthesia
Enophthalmos
Infection
Exposed hardware
Deviated septum
Nasal obstruction
Altered vision
Nonunion
Malunion or malocclusion
Epiphora
Foreign body reactions
Scarring
Sinusitis

Thank you

Neuromuscular Diseases

Definition

Are diseases that affect both nerve and muscle tissue .Neuromuscular disorders represent a spectrum of nerve related diseases and conditions that affect the body's voluntary muscles cause weakening of muscles in the body because of interrupted communication between the nervous system and the muscles it controls. Typically, these diseases can be managed to improve quality and length of life, but are incurable.

Classification of neuromuscular disorders:

- CEREBROVASCULAR DISEASE
- MULTIPLE SCLEROSIS
- ALZHEIMER'S DISEASE
- SEIZURE DISORDERS
- PARKINSON DISEASE
- MYASTHENIA GRAVIS

Cerebrovascular Disease

Cerebrovascular disease refers to disorders that result in damage to the cerebral blood vessels leading to impaired cerebral circulation**.**

A cerebrovascular accident (CVA), or complete stroke, is a sudden impairment in cerebral circulation resulting in death or a focal neurologic deficit lasting more than 24 hours.

Transient ischemic attack (TIA), defined as a reversible, acute, short-duration, focal neurologic deficit ("mini stroke") resulting from transient (reversible within 24 hours) and localized cerebral ischemia;

Reversible ischemic neurologic defect (RIND), defined as a reversible, acute, focal neurologic deficit due to transient and localized cerebral ischemia but resulting in neurologic deficits that last more than 24 hours; and

Stroke in evolution, defined as progressive worsening of stroke symptoms.

Clinical Manifestations

The clinical manifestations of stroke vary depending on the size and location of the affected brain region. The most common signs and symptoms include sensory and motor deficits, changes (paresis) in extra ocular muscles and eye movements, visual defects, sudden headache, altered mental status, dizziness, nausea, seizures, impaired speech or hearing, and neurocognitive deficits such as impaired memory, reasoning, and concentration.

General symptoms following stroke:-

- variable motor paralysis
- sensory loss
- visual difficulties
- speech impairment

Types of cerebrovascular diseases

Cerebrovascular Accident (CVA) or Stroke either due to:-

1- Atherosclerosis(85%) leading to cerebral ischemia and infarction result from ischemia due to atherosclerotic disease, thromboembolic events, and occlusion of cerebral blood vessels, with neurologic deficits related to the loss of neural function in tissues distal to the event.

2- Cerebral hemorrhage (15%) result from hemorrhagic events leading to infarction, most often related to hypertension, trauma, substance abuse, or aneurysmal rupture.

Three major types of ischemic stroke syndromes have been described:

1- small vessel (lacunar),

2- large vessel (cerebral infarction)

3- Brainstem stroke

Lacunar strokes:- result from obstruction of the small (<5 mm diameter) penetrating arterioles.

Age and uncontrolled hypertension are the greatest predisposing factors. Symptoms usually include unilateral motor or sensory deficit without visual field changes or disturbances of consciousness or language. The prognosis for recovery from lacunar infarction is fair to good, with partial or complete resolution usually occurring over four to six weeks.

Cerebral infarction (large vessel):- is characterized by extensive downstream ischemia, usually due to a thromboembolic event along the distribution of the internal carotid artery and cerebral arteries. Emboli often originate from the heart after acute myocardial infarction or in hyperdynamic conditions such as chronic atrial fibrillation.

Hypertension is an important risk factor in the development of thrombosis, particularly at the carotid bifurcation, and treatment of severe hypertension is essential for the prevention of stroke. High level brain functions are affected, and the prognosis is poor.

Brainstem infarction: - results from occlusion of small or large vessels supplying the brainstem, resulting in variable deficits ranging from motor and sensory deficits to death when respiratory centers are affected.

D.D. of CVA

Seizures, hypoglycemia, intracranial tumors, trauma, infection, encephalitis, multiple sclerosis (MS), and prolonged migrainous Aura.

Diagnosis

Stroke should be considered whenever a patient experiences the clinical manifestations. Laboratory evaluation of the stroke patient includes compete blood count, comprehensive metabolic panel, urinalysis, coagulation profile, and, when indicated, blood culture, echocardiography, and lumbar puncture.

Treatment

The outcome of stroke and related TIAs and RIND is significantly affected by the timeliness of treatment. Early intervention is critical to prevention, treatment, and recovery.

TIAs and RIND are treated by reduction in hypertension (lifestyle changes such as diet, exercise, smoking cessation, and stress reduction; medical therapy for hypertension; and anticoagulant or antiplatelet medications).

Once intracranial hemorrhage has been excluded as the source of acute cerebral ischemia, thrombolysis with intravenous tissue plasminogen activator (t-PA) can improve reperfusion, minimize infarction, and reduce disability.

After a completed stroke, treatment focuses on:

1. The prevention of further neurological damage, through the reduction of underlying risk factors.

2. Rehabilitation procedures, including speech and physical therapy.

3. An intracranial hemorrhage should also be treated as a medical emergency of airway maintenance and requires the transfer of the patient to an intensive care unit with close monitoring.

4. The surgical treatment of a hemorrhaging aneurysm consists of closing off the blood vessels that supply the area and removing the abnormality.

Oral Health Considerations

Following stroke, patients may experience several oral problems, including masticatory and facial muscle paralysis, impaired or lost touch and taste sensation, diminished protective gag reflex, and dysphagia. These problems can lead to impairment of food intake, poor nutrition, and weight loss due to diminished taste satisfaction, chewing capacity, and swallowing; choking; and gagging. Diminished motor function of masticatory and facial muscles may also reduce food clearance from the mouth and teeth and alone or combined with the presence of diminished dexterity of the arms or hands may adversely affect oral hygiene and increase the risk for caries and periodontal disease. Creative and effective use of adjuvant oral hygiene techniques

and devices (oral antimicrobial rinse, oral irrigation, floss holders) represents an important approach to oral health promotion and disease prevention, supported by frequent recall examination and prophylaxis. Replacement of missing teeth and adequacy of removable and fixed prostheses are essential to effective chewing and diet.

Multiple Sclerosis

MS is characterized by multiple areas of central nervous system (CNS) white matter inflammation, demyelination, and gliosis (scarring). Myelin is critical for propagation of nerve impulses, and when it is destroyed in MS, slowing and/or complete block of impulse propagation are manifested by abnormal muscular and neurologic signs and symptoms, associated with the myelination of axons within the central nervous system.

The disease occurs more frequently among women. The average age of onset is during the fourth decade of life, but MS may occur at any age. The disease presents in the form of recurrent attacks

Etiology

1- An immunologic (autoimmune disease) basis is strongly suggested by the presence of activated T lymphocytes and autoantibodies to glycoproteins detected in MS lesions.

2- Environmental exposure in MS , and two common infectious agents to be implicated in the pathogenesis of this disease are Epstein–Barr virus and human herpesvirus 6. Other viruses that have been implicated in the pathogenesis of MS include measles, mumps, rubella, parainfluenza, vaccin, and human T-lymphotropic virus

3- increased antibody titers against measles virus, rubella virus, mumps virus, Epstein-Barr virus, herpes simplex viruses 1 and 2, and human herpes virus 6 (HHV-6) have been found in the cerebrospinal fluid and serum.

4- Genetic influences also appear to play a significant role in the development of MS.

Clinical Manifestations

The most common symptoms following an acute exacerbation include impairment of vision, muscular incoordination, and bladder dysfunction.

1. The clinical signs and symptoms of MS depend on the site of the demyelinating lesion of the CNS involved, and frequently affected areas include the optic chiasm, brainstem, cerebellum, and spinal cord.

2. More than 60% of individuals with MS have visual disturbances caused by demyelinating lesions of the second cranial nerve. The loss of vision usually occurs over a period of several days, with partial recovery within 1 month.
3. Other ophthalmic symptoms include ―color blindness‖ and diplopia caused by involvement of the third, fourth, and sixth cranial nerves.
4. Uhthoff's sign, found in MS, is characterized by rapid vision loss following a body temperature increase that is associated with strenuous exercise.
5. MS patients frequently complain of electric shock–like sensations that are evoked by neck flexion and radiate down the back and into the legs. This is referred to as **Lhermitte's symptom** and is generally self-limiting but may persist for Years
6. Weakness or paresthesia of the extremities, with an increase in the deep tendon reflexes, is another common early finding in cases of MS.
7. Bladder dysfunction, euphoria, ataxia, vertigo, and generalized incoordination
8. The majorities of cases of MS are chronic and are characterized by exacerbations and remissions over a period of many years.
9. During acute episodes, severe neurologic involvement is evident. This slowly resolves, but some permanent neurologic involvement remains after each episode.

Diagnosis

1. Clinical and is based on the age of the patient, the presence of neurologic signs that cannot be explained by a single lesion, the progressive nature of the disease, and a history of exacerbations and remissions.
2. There are no definitive laboratory tests for MS, but demyelinating changes can be seen on (MRI) in more than 90% of cases. MRI demonstrates characteristic abnormalities of MS in >95% of patients. MS plaques are visible as hyperintense focal areas.
3. Evoked potentials measure CNS electrical potentials, and abnormalities are detected in up to 90% of patients with MS.
4. CSF is often analyzed in patients suspected of having MS, and positive findings include an increase in total protein and mononuclear white blood cells.

Treatment

1- High doses of **intravenous corticosteroids** may arrest the progress of MS; about 85% of patients with relapsing-remitting MS show objective signs of neurologic improvement during treatment with intravenous corticosteroids. Glucocorticoids are used to manage both initial attacks and acute exacerbations of MS. Intra-

venous methylprednisolone is typically administered at a dose between 500 and 1000 mg/d for three to five days to reduce the severity and length of an attacks

2- Long-term treatment with immunosuppressants may reduce the frequency of relapse in patients with MS. Azathioprine is probably the safest drug in this category and has reduced relapse to 70% of study patients in 3 years. Administration of methotrexate appears to be the best therapy for slowing deterioration in patients with chronic progressive MS.
3- The use of interferon-γ-1b and -1a has shown promise; both have been shown to reduce clinical attacks and lesions

Oral Health Considerations

Individuals may present with signs and symptoms of MS.
1- Trigeminal neuralgia (TGN), which is characterized by electric shock–like pain, may be an initial manifestation of MS in up to 3% of cases. MS-related TGN is similar to idiopathic TGN . Features of MS-related TGN include possible absence of trigger zones and continuous pain with lower intensity.
2- Medications often used to manage TGN are similar to those used for treatment of idiopathic TGN.
3- Patients with MS may also demonstrate neuropathy of the maxillary (V2) and mandibular branches (V3) of the trigeminal nerve, which may include burning, tingling, and/ or reduced sensation.
4- Neuropathy of the mental nerve can cause numbness of the lower lip and chin.
5- **Myokymia** may be seen in patients with MS and consists of rapid, flickering contractions of the facial musculature secondary to MS lesions affecting the facial nerve.
6- Facial weakness and paralysis may also be evident in MS patients.
7- Dysarthria that results in a scanning speech pattern is often seen in patients with MS.
8- Temporomandibular disorder and headache.
Evaluate cranial nerve function, if cranial nerve abnormalities are detected, the individual should be referred to a neurologist for further evaluation.
It is recommended to avoid elective dental treatment in MS patients during acute exacerbations of the disease due to limited mobility and possible airway compromise.

Patients with significant dysfunction may require dental treatment in an operating room under general anesthesia due to the inability to tolerate treatment in an outpatient setting.
In addition, electric toothbrushes and oral hygiene products with larger handles may be necessary for completing oral hygiene in patients with significant motor impairment. Be aware of possible interactions of these medications with those commonly used and prescribed in dentistry, as well as oral and systemic side effects of these agents.

ALZHEIMER'S DISEASE (AD)

Dementia is defined as an acquired deterioration in cognitive abilities that impairs the successful performance of activities of daily living. Memory is the most common cognitive ability lost with dementia; other mental faculties affected include problem-solving skills, judgment, visuospatial ability, and language.
The genetic basis of AD has been studied extensively, and specific genetic mutations have been implicated in both the familial and sporadic forms of the disease. Familial AD is an autosomal dominant disorder with onset typically prior to age 65 year.

Clinical Manifestations

AD is a slowly progressive disorder represented by a continuum recognizes three stages of AD: (1) preclinical AD, (2) mild cognitive impairment due to AD, and (3) dementia due to AD.
Preclinical AD occurs before changes in cognition, and everyday activities are observed and primarily used for research purposes.
Cognitive impairment (CI) due to AD is characterized by mild changes in memory and other cognitive abilities that are noticeable to patients and families but are not sufficient to interfere with day to-day activities.
Dementia due to AD is characterized by changes in two or more aspects of cognition and behavior that interfere with the ability to function in everyday life. The initial signs of AD involve retrograde amnesia from progressive declines in episodic memory. This may initially go unrecognized or be viewed; however, as the disease progresses, memory loss begins to affect performance of daily activities, including following instructions, driving, and normal decision making.
As AD progresses, the individual is often unable to work, gets confused and lost easily, and may require daily supervision. Also language impairment, loss of ab-

stract reasoning and skills. Advanced AD is characterized by loss of cognitive abilities, agitation, delusions, and psychotic behavior.
Patients may develop muscle rigidity associated with gait disturbances and often wander aimlessly.
In end-stage AD, patients often become rigid, mute, incontinent, and bedridden. Help is needed for basic functions, such as eating and dressing, and patients may experience generalized seizure activity. Death often results from malnutrition, heart disease, pulmonary emboli, or secondary infections.

Diagnosis

Diagnosis of preclinical AD primarily utilizes biomarker assessment, including markers of Aβ protein deposition in the brain, and markers of downstream neurodegeneration (elevated CSF tau protein and brain atrophy on MRI.
Clinical diagnosis of AD is based on an individual's medical history together with the clinical and neurologic examination findings.
Criteria include a history of progressive deterioration in cognitive ability in the absence of other known neurologic or medical problems.
Possible AD refers to those who meet the criteria for dementia but have another illness that may contribute to the neurologic status, such as:- hypothyroidism or cerebrovascular disease, vitamin deficiency, depression, delerium, side effects of drugs and toxicity and excessive use of alcohol .
Diagnostic analysis of CSF may show a slight increase in tau protein and a lower concentration of Aβ peptide compared with healthy individuals or those with other dementias.
Electroencephalographic (EEG) studies typically demonstrate generalized slowing without focal features. Neuroimaging is important in evaluating suspected AD to exclude alternative causes of dementia, such as cerebrovascular disease, subdural hematoma, or brain tumor.
MRI and CT typically reveal dilatation of the lateral ventricles and widening of the cortical sulci, particularly in the temporal regions.
Volumetric MRI uniformly demonstrates shrinkage in vulnerable brain regions (brain atrophy).

Treatment

There is no cure for AD, and therapy is aimed at slowing the progression of the disease. **Cholinesterase inhibitors** are approved to treat mild to moderate cases of AD and are considered the standard of care.
Memantine, a noncompetitive *N*-methyl-d-aspartate receptor antagonist believed to protect neurons from glutamate-mediated excitotoxicity, is used for treatment of moderate to severe AD.

Studies have demonstrated greater cognitive and functional improvement when memantine is used in conjunction with cholinesterase inhibitors compared to monotherapy.
Antidepressants, such as selective serotonin reuptake inhibitors, are commonly used to treat depression, which is often seen in the mild to moderate stages of AD.
Antipsychotic agents are used for those patients who display aggressive behavior and psychosis, especially in the later stages of the disease.
Other agents that have been reported to be of clinical value in the treatment of AD include antioxidants, such as α-tocopherol (vitamin E), cholesterol-lowering drugs, anti-inflammatories, and herbal

Oral Health Considerations

Oral and dental health is a major issue in patients with AD because significant deterioration in oral health status is commonly observed with advancing disease.
Patients with AD appear to be at higher risk for developing coronal and root caries, periodontal infections, temporomandibularn joint abnormalities, and orofacial pain compared to healthy subjects.
Patients with AD should be placed on an aggressive preventive dentistry program, including an oral examination, oral hygiene education, prosthesis adjustment, and a three-month recall.
It is recommended to complete restoration of oral health-care function in the earliest stages of AD because the patient's ability to cooperate diminishes as cognitive function declines. Time-consuming and complex dental treatment should be avoided in persons with severe AD.
Medications used to treat AD can cause a variety of orofacial reactions and potentially interact with drugs commonly used in dentistry. Cholinesterase inhibitors may cause sialorrhea, whereas antidepressants and antipsychotics are often associated with xerostomia. In addition, dysgeusia and stomatitis have been reported with use of antipsychotic agents. Antimicrobials, such as clarithromycin, erythromycin, and ketoconazole, may significantly impair the metabolism of galantamine, resulting in central or peripheral cholinergic effects.
Anticholinesterases may increase the possibility of gastrointestinal irritation and bleeding when used concomitantly with NSAIDs.
Local anesthetics with adrenergic vasoconstrictors should be used with caution in AD patients taking tricyclic antidepressants due to potential risk of cardiovascular effects, such as hypertensive events or dysrhythmias.

SEIZURE DISORDERS & Epilepsy

A seizure is a paroxysmal event due to abnormal, excessive, hypersynchronous discharges from neuronal aggregates in the CNS. The term *epilepsy* describes a group of neurologic disorders characterized by recurrent seizure activity.

1- Focal, 2- generalized and 3- unknown seizures are currently the three major categories of seizure activity used in clinical practice.

1-The focal seizure category (Partial Seizures)

Includes partial seizures; this type of seizure activity originates within networks limited to one hemisphere and clinical manifestations of these seizures depend on the site of origin. Simple partial seizures reflect neuronal discharge from a discrete cortical locus, such as the motor cortex of the frontal lobe, or in subcortical structures, and generally not associated with impaired consciousness.

Simple partial seizures consist of clonic activity, which are rapid jerks that also can be accompanied by somato-sensory phenomena, visual changes/distortions, and auditory, olfactory, and gustatory

2- Generalized seizures arise from both cerebral hemispheres simultaneously and have distinctive clinical features that facilitate diagnosis. The underlying pathophysiology of generalized seizures is attributed to abnormal neuronal excitability.

a- Absence seizures (petit mal) are a type of generalized seizure that is characterized by sudden, brief lapses of consciousness without loss of body tone and may be attributed to abnormal oscillatory rhythms generated during sleep by circuits connecting the thalamus and cortex.

b- **Tonic-clonic (grand mal)** seizures are generalized seizures that present with dramatic clinical features, most notably, tonic contracture and uncoordinated clonic muscular movements.

Other types of generalized seizures include atypical absence, atonic, and myoclonic seizures. 3- Those seizures that cannot be classified as either focal or generalized are termed **unknown seizures**.

Etiology usually varies according to patient,s age.

The most common seizures arising in late infancy and early childhood are febrile seizures without evidence of associated CNS infection; these usually occur between 3 months and 5 years of age and have a peak incidence between 18 and 24 months.

Isolated, non recurrent, generalized seizures among adults are caused by multiple etiologies, including metabolic disturbances, toxins, drug effects, hypotension, hypoglycemia, hyponatremia, uremia, hepatic encephalopathy, drug overdoses, and drug withdrawal.

Cerebrovascular disease may account for approximately 50% of new cases of epilepsy in patients older than 65 years. Other etiologies for epilepsy include degenerative CNS disease, developmental disabilities, and familial/genetic factors. Epilepsy occurs more frequently in individuals who have neurologic-based disabilities, such as cerebral palsy and autism.

Epilepsy

Epilepsy is a condition characterized by abnormal, recurrent, and excessive neuronal discharges precipitated by many different disturbances within the central nervous system.

These aberrant discharges may cause episodes of sensory and motor abnormalities as well as loss of consciousness.

Common causes of epilepsy:

1-Infants are much more likely to suffer from epilepsy after complications at birth, such as anoxia lack of oxygen traumatic brain injury during delivery, intracranial injury, metabolic disorders, abnormal brain development and congenital malformations.

2- Predominant causes in children and adolescents include head trauma and acute or febrile infections, fever, brain tumors, genetic disorders and brain scarring.

3- Young adults with alcohol or drug abuse commonly suffer from generalized seizures after periods of severe abuse.

4- Epilepsy in older adults occurs as a complication of any of the previously mentioned causes but is more often associated with cerebrovascular diseases such as stroke, brain scarring, abnormal brain development, head trauma and brain tumors.

Diagnosis:

1. History & physical examination are critical because the diagnosis may based on clinical findings.

2. A complete neurological examination (testing of cranial nerves)

3. Blood studies: complete blood count, mg, calcium , glucose to identify metabolic cause

4. Toxins screen: to identify seizure due to drugs, lumber puncture to exclude any infectious cause

5. Brain imaging: underlying CNS structural abnormalities or pathology

MRI and CT

6. EEG (to classify the seizure & to determine the type of anticonvulsant)

Treatment

Antiepileptic drugs (AEDs)

*phenytoine: long half-life, dosed less frequently cause gingival over growth, hirsutism, coarsening of facial features.

*carbamazepine: hepatotoxicity, leukopenia,aplastic anemia.

*Lamotrigine: skin rash.

*Valproic acid: treatment of G.tonic clonic,can cause bone marrow suppressison & hepatotoxicity.

*Additional drugs as topiramate, gabapentin & oxcarbazepine,

*surgical procedures.

*Vagus nerve stimulation.

Discontinuation of pharmacologic therapy is considered when seizure control has been achieved. The following patient characteristics yield the greatest chance of remaining seizure free after discontinuation of drug therapy:

(1) Complete medical control of seizures for one to five years;

(2) Single seizure type; (3) normal neurologic examination, including intelligence; and (4) a normal EEG.

Many patients are often withdrawn successfully from medication after an interval of two to four years without seizures who meet the above criteria and who clearly understand the risks and benefits.

Patients may use three or more drugs to successfully treat refractory epilepsy; however, up to 30% of patients are resistant to all medical therapies. Surgical procedures may be indicated for these patients.

Deep brain stimulation (DBS) and responsive neurostimulation systems are also currently used for treatment of refractory epilepsy.

Gene therapy is currently being investigated as an alternative treatment modality for epilepsy refractory to standard therapies.

Oral health consideration

* Uncontrolled Patients should be referred to a hospital

*Patient with implanted vagus nerve stimulator do not require antibiotic prophylaxis

*we must avoid any triggers of the patient seizures activity

*Placement of fixed prosthesis is recommended rather than removable prosthesis.

*Patient taking the medication mentioned requires laboratory evaluation prior to dental treatment

*Aspirin& NSAD should be avoided in patient taking valproic acid

*Gingival over growth intraoral lesion & lips enlargement

*Xerostomia:

Reduced salivary flow may result from the use of AEDs, may observe increased dental caries and oral candidiasis in patients using these agents.

Topical fluoride should be considered for patients with seizure disorders who are at increased risk of developing dental caries, and antifungal agents should be prescribed if oral candidiasis develops. Additional oral findings in patients taking AEDs may include stomatitis, glossitis, and ulcerations.

Parkinson disease

Parkinson disease (PD) is a chronic, progressive, neurodegenerative disorder characterized historically by its cardinal motor symptoms of resting tremor, rigidity, gait disturbance, and bradykinesia.
The American Academy of Neurology has developed diagnostic, assessment, and treatment guidelines to distinguish idiopathic PD from "parkinsonian syndromes" such as corticobasal degeneration, progressive nuclear palsy, dementia with Lewy bodies (LBs) and PD dementia, which share similar symptoms but have different risk factors, pathological processes, and clinical courses.

PD results from degeneration of the dopaminergic cells in the pars compacta of the substantia nigra (SN), leading to depletion of the neurotransmitter dopamine in the basal ganglia.

Clinical Manifestations

PD usually affects people older than 50 years, although it can occur at any age, and earlier cases occur more commonly in the familial forms of PD. Early signs of PD, particularly nonmotor signs, can subtle. The four cardinal motor signs of PD are resting tremor (in hands, arms, legs, jaw, and face); rigidity or stiffness (limbs and trunk), bradykinesia (slowness of movement), and postural instability or impaired balance and coordination. Between 30% and 50% of individuals with PD develop dementia and the majority also exhibit behavioral/psychiatric symptoms including depression, anxiety, apathy, and irritability.

Autonomic dysfunction is common and can develop early, including orthostatic hypotension, constipation, urinary frequency and urgency, and abnormal sweating. As symptoms become more pronounced, patients become increasingly impaired. Though rate of decline varies widely, PD is inevitably progressive and destructive.

Diagnosis

Currently, there are no laboratory tests specific for idiopathic (classic) PD.Clinical genetic markers are available for risk assessment where hereditary patterns of PD exist. Therefore, the diagnosis is based on the health history, neurologic examination, and response to levodopa therapy. When symptoms are subtle and the presentation is incomplete, the diagnosis can be difficult. Differentiating classic PD from a variety of parkinsonian syndromes characterized by motor decline and/or dementia can be challenging. Anatomic and functional brain imaging, CSF evaluation, and laboratory testing are often necessary to exclude other diagnoses.

Treatment

Currently, there is no cure for PD, but a variety of medications and procedures provide dramatic relief from the symptoms. Dopamine replacement therapy using levodopa (used by neurons to synthesize dopamine) combined with carbidopa remains the initial gold standard.

Oral Health Considerations

Patients with PD present several challenges to the dental health-care team and to the patient related to both the illness and its treatment. Patients with PD often must be treated in a relatively upright position, making complex dental procedures in the maxillary arch or posterior oral cavity a challenge. Resting tremors and drug-related dyskinesia can complicate procedures, and behavioral techniques to reduce anxiety as well as gentle cradling techniques can help. Dysphagia and impaired gag reflex increase the risk for aspiration of oral and irrigation fluids, and high-speed evacuation of fluids is important in reducing the risk for aspiration pneumonia. Some patients experience sialorrhea, making maintenance of a dry field difficult for some operative and surgical procedures.

Myasthenia gravis

Myasthenia gravis (MG) is a chronic neuromuscular disease caused by autoimmune destruction of the skeletal neuromuscular junction resulting in impaired neurotransmission and muscle weakness. Pathogenic antibodies directed against components of the postsynaptic membrane of the neuromuscular junction disrupt neurotransmission. MG is characterized by episodic weakness of the skeletal muscles that increases during periods of activity and improves after periods of rest. Prior to effective therapy death commonly resulted from respiratory failure and pneumonia.

Ocular weakness presenting as fluctuating ptosis and/or diplopia is the most common initial presentation of MG, occurring in 85% of patients. Muscles of facial expression, masticatory, and swallowing muscles are also affected early resulting in facial asymmetry, dysarthria, and dysphagia.

Clinical Manifestations

Eighty-five percent of MG patients present with ocular symptoms characterized by diplopia and/or ptosis.Oropharyngeal, facial, and masticatory muscle weakness is common and results in dysphagia, asymmetry, and dysarthria, and are the initial symptoms in one-sixth of patients.

Severity of weakness typically fluctuates on a daily and use basis, but tends to worsen as the day progresses. The clinical course of disease is variable but usually progressive.

In general, MG patients experience an initial period of one to two years during which the disease reaches a maximum level of severity, followed by improvement for the majority.

Diagnosis

The clinical examination and history are highly suggestive of MG. Diagnosis is confirmed by a variety of bedside, electrophysiological and immunological tests. Serologic evidence of autoantibodies to the AChR, although there are reports of MG without anti-AChR. CT or MRI of the chest is highly accurate in detecting thymoma, and every patient with MG should be screened for thymoma.

Treatment

1. Anticholinesterase drugs such as neostigmine and pyridostigmine bromide
2. thymectomy
3. Long-term cortico-steroids and immunosuppressive drugs are necessary.

Oral Health Considerations

Orofacial signs and symptoms are prominent presenting features of MG, and the dental provider may be in the position to recognize and refer for diagnosis. Difficulty with prolonged opening and swallowing presents challenges in dental treatment delivery and the ability to tolerate treatment, and difficulty in chewing can affect diet and the design of prostheses. Implant retained removable or fixed prosthesis may be preferable to tissue-supported for improved chewing efficacy.

Aspiration risks can be high and can be reduced by adequate suction, the use of a rubber dam, and avoiding bilateral mandibular anesthetic block. The MG patient may also be at risk for a respiratory crisis from the disease itself or from overmedication; if this is a substantial risk and the dental treatment is necessary, dental treatment in a hospital should be considered where endotracheal intubation can be performed. Avoid prescribing drugs that may affect the neuromuscular junction, such as narcotics, tranquilizers, and barbiturates.

Reference: Burket's Oral Medicine,12th edition ,2015

Non Odontogenic tumors of jaw

Classification

Benign	Malignant
Ossifying fibroma	Fibrosarcoma
Lesions containing giant cells	
Langerhans cell disease	
Neurogenic tumors	Malignant peripheral nerve sheath tumor
Osteogenic tumor (osteoma..)	Osteosarcoma
Chondroma	Chondrosarcoma
Vascular lesions (hemangioma, vascular malformation)	Ewing's sarcoma Burkitt's lymphoma Multiple myeloma
	Metastatic carcinoma

Giant cell lesions

1. Central giant cell granuloma
2. Giant cell tumor
3. Hyperparathyroidism
4. Cherubism
5. Aneurysmal bone cyst

- The lesions in this group are similar, if not identical, histologically, and they usually cannot be distinguished from one another solely on the basis of light microscopy.
- Clinical history, physical and radiographical examination, and serum biochemistry may be used to differentiate these lesions.

Hyperparathyroidism

- Characterized by the overproduction of parathyroid hormone (PTH).
- Three types :

1) Primary hyperparathyroidism is the uncontrolled production of PTH as the result of a parathyroid adenoma, hyperplasia, or rarely an adenocarcinoma. PTH↑, Ca↑, P↓

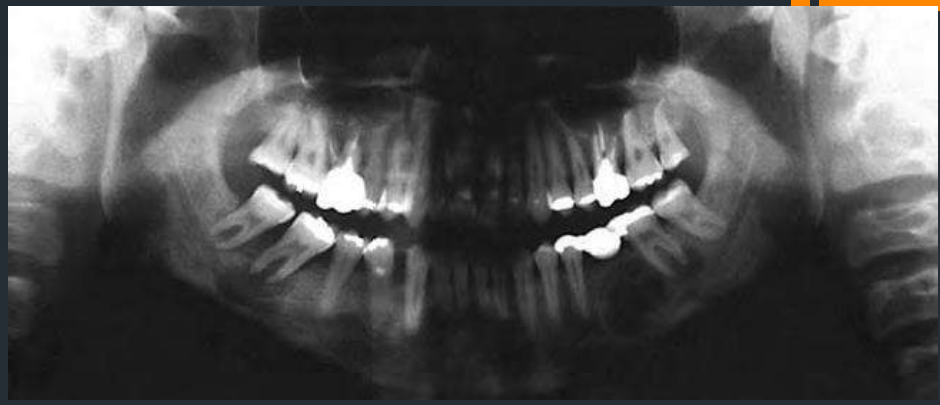
2) Secondary hyperparathyroidism occurs in response to hypocalcemia, most often as a result of chronic renal failure.

↑PTH, ↓Ca, ↑P

3) Tertiary Hyperparathyroidism: occurs after long standing 2°Hyperpara. And return of Ca level within normal or elevated.

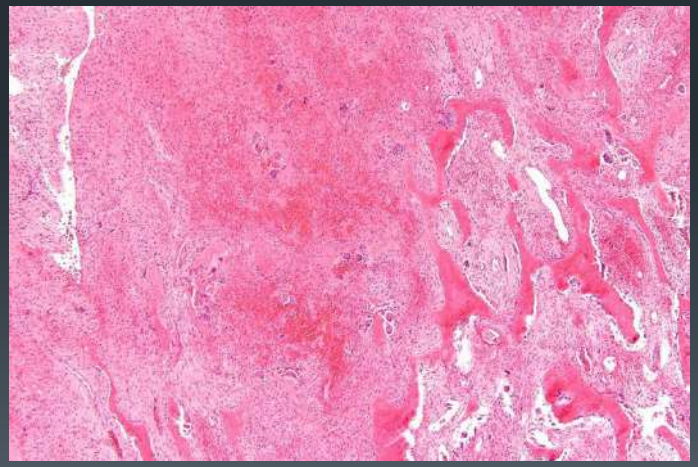
↑PTH, ↑Ca, ↑P

Brown tumor



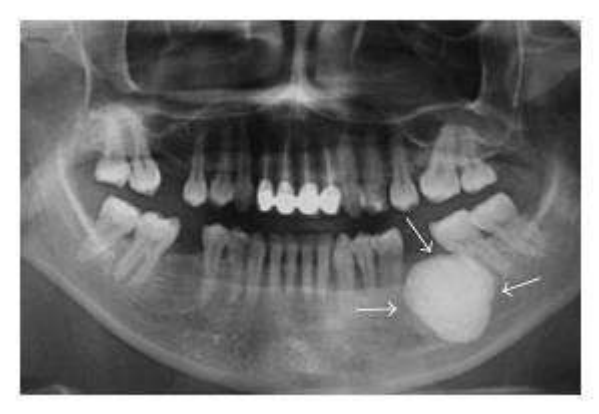
- Excess PTH levels stimulate bone resorption, which may produce a focal bone lesion known as a brown tumor of hyperparathyroidism.

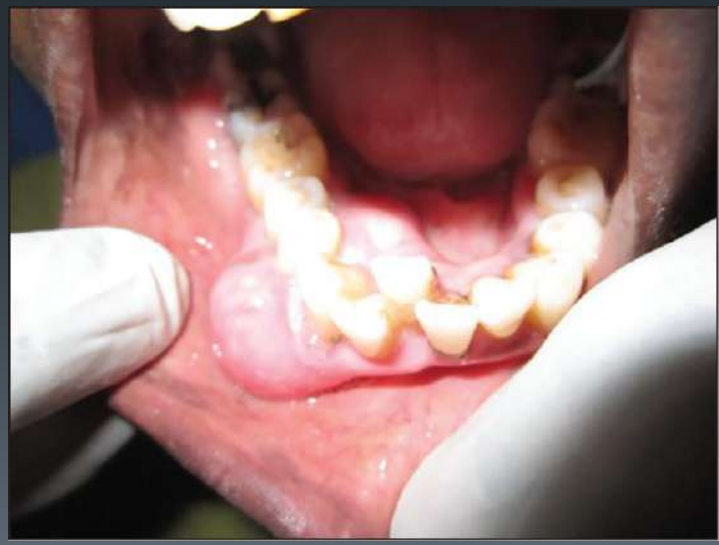
- The lesion derives its name from the color of the tissue as seen on surgical exploration, which is a result of the erythrocyte extravasation and hemosiderin deposition within the lesion



Osteomas

- Osteomas are benign tumors composed of mature compact or cancellous bone. They are distinguished from the common palatal and mandibular tori, as well as buccal exostoses, despite identical histopathology.
- Osteomas may arise from the surface of bone (periosteal osteoma), or they may be located in the medullary bone (endosteal osteoma)
- Radiographically, osteomas appear as well-circumscribed, sclerotic masses.
- Osteomas are treated by local excision.





Gardner syndrome.

- Osteomas are usually solitary, except in cases of Gardner syndrome.
- This syndrome is an autosomal dominant condition in which patients have intestinal polyposis, multiple osteomas, fibromas of the skin, epidermal cysts, impacted permanent and supernumerary teeth, and odontomas.
- The countless polyps in the colon predispose to the development of colon cancer.
- There is no cure at this time, and in its more advanced forms, it is considered a terminal diagnosis with a life expectancy of 35–45 years; treatments are surgery and palliative care.

Langerhans Cell Histiocytosis (histiocytosis X)

- Divided into three separate types:

I. Eosinophilic granuloma (chronic localized): bone only

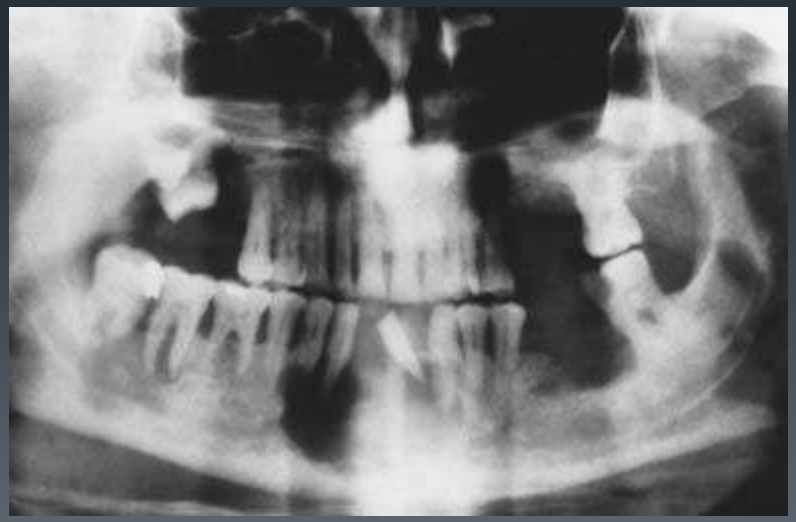
II. Hand-Schüller-Christian disease(Chronic disseminated) involve bone, skin and viscera

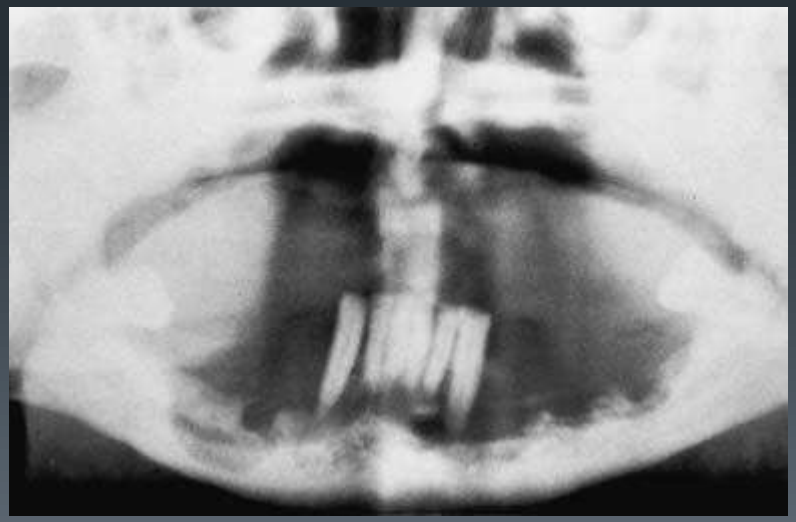
III. Letterer-Siwe disease(acute disseminated) involves skin, viscera and bone in infants.

- The common factor was the presence of histiocytes which are dendritic cells in the skin and mucosa that have a macrophage-like function
- Bone lesion: soliatry or multiple, dull pain, posterior region of mandible, causing loosening of teeth (resemble periodontitis)

Radiographically,

- jaw lesions usually appear as well-defined, punched-out radiolucencies, although they may be ill defined.
- Lesions often involve the alveolar bone, producing the classic appearance of “floating teeth.” The involved teeth remain vital





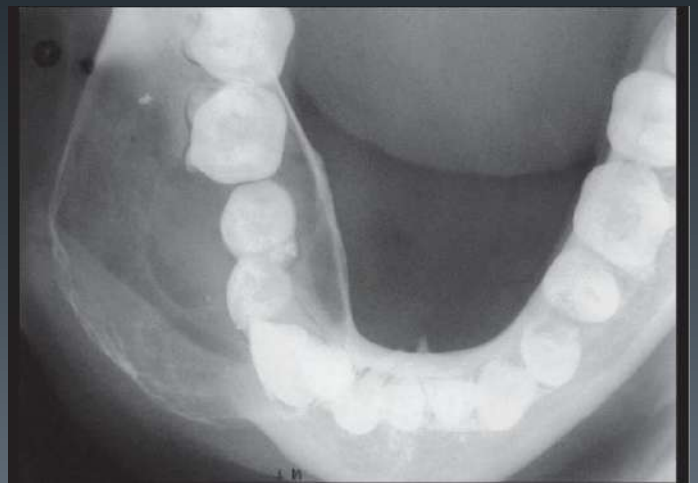
Management

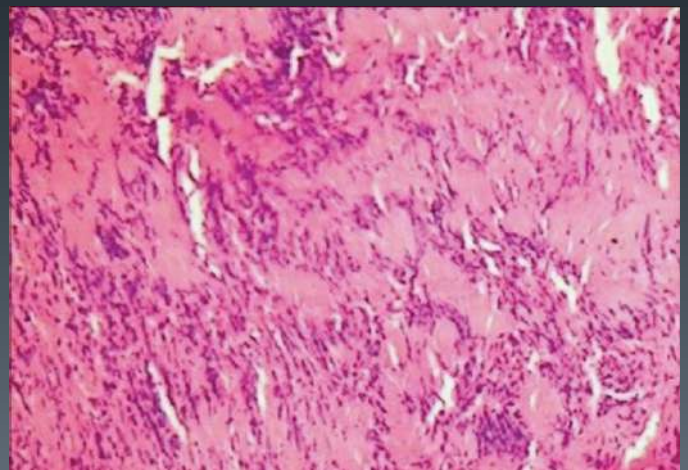
- Curettage or resection with 5mm safe margin for accessible bone lesions
- Intralesional steroid injections or RRx for inaccessible bone lesion
- CRx for acute disseminated disease.

Neurogenic Tumors

1. ***Schwannoma***

- The schwannoma is a slowly growing, benign neoplasm arising from Schwann cells of the nerve sheath (neurilemma).
- As this encapsulated tumor enlarges, it pushes the involved nerve aside without enveloping it.
- Bony lesions may be asymptomatic or produce expansion, pain, paresthesia, tooth mobility, and tooth displacement
- well-defined, unilocular radiolucency with a thin, sclerotic border.
- Histologically, this is an encapsulated spindle cell tumor that consists of variable amounts of two types of tissue, Antoni A and Antoni B.



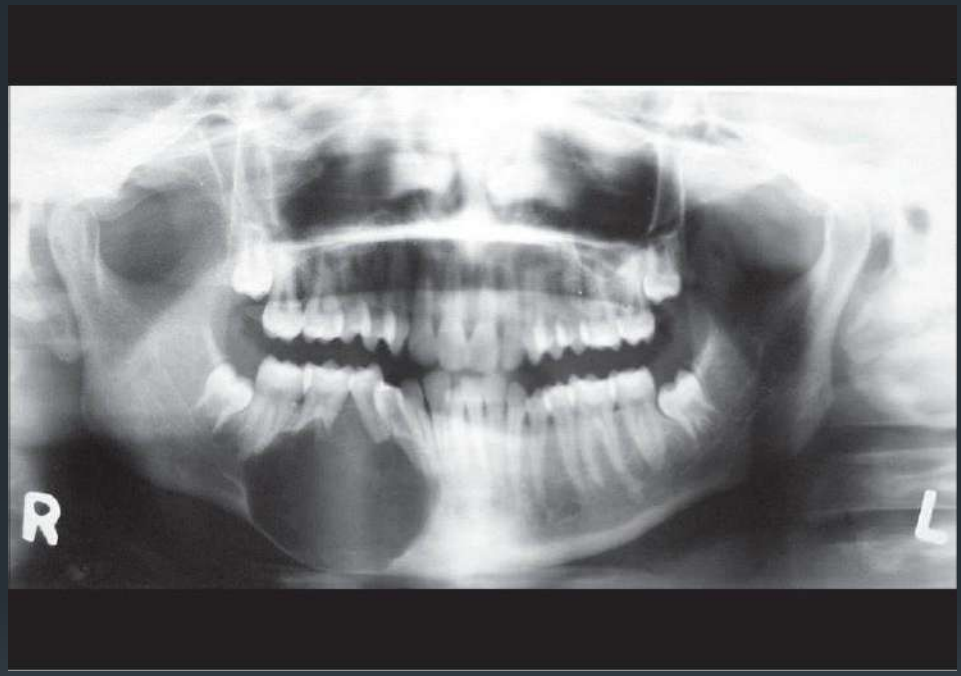


Treatment

Intraosseous schwannomas can be treated by enucleation and curettage.

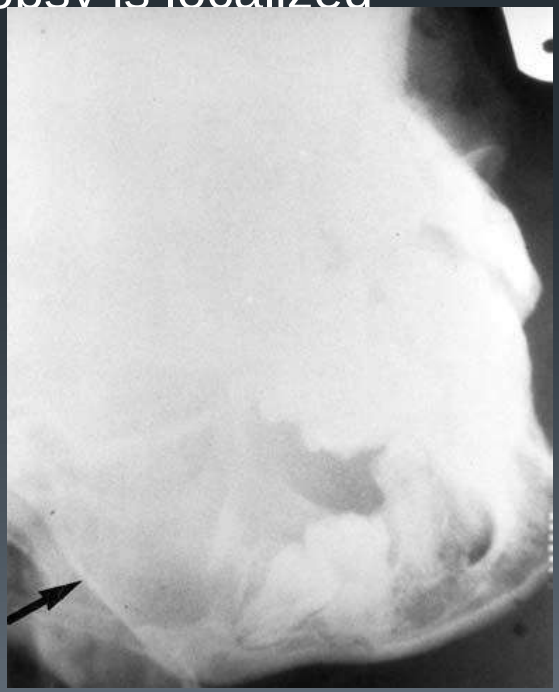
When the lesion arises from an identifiable nerve such as the inferior alveolar nerve, it

can be excised from the nerve while preserving the integrity of the nerve. Recurrences are rare



2- Neurofibroma

- Arise from a mixture of cell types including Schwann cells and perineural fibroblasts.
- They may occur as solitary lesions or in association with neurofibromatosis (von Recklinghausen's disease of skin)
- Pain, paresthesia and cortical expansion may result from lesions of the inferior alveolar nerve
- Intraosseous lesions may produce a well-demarcated or poorly defined unilocular or multilocular R.L.
- The normally recommended treatment following biopsy is localized excision



Vascular lesions of bone

- Vascular anomalies are divided into

1. vascular neoplasms (e.g. hemangioma) and
2. vascular malformations. (venous, arterial, capillary, lymphatic)

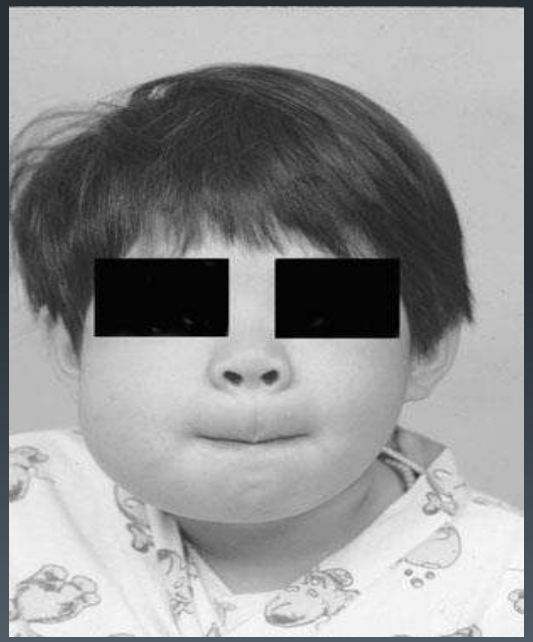
- The major distinction between them is the existence of an increased endothelial cell turnover (vascular neoplasms). Vascular malformations do not increase the endothelial cell turnover, since they represent structural abnormalities of the capillary, venous, lymphatic and arterial system.

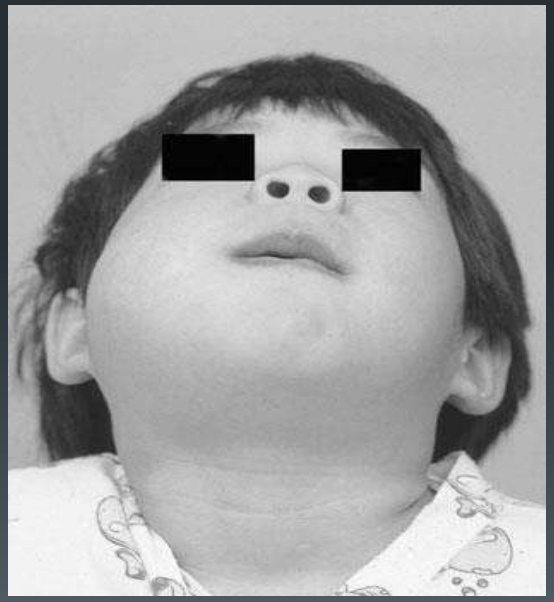
- Intraosseous vascular anomalies are either:

I. Venous malformation (VMF)
II. Arterial malformation (AMF)

Clinical features

- Female predilection, 10-20 years old, Mandible >Maxilla
- Usually asymptomatic (slow-growing asymmetric expansile lesion of the jaw) or may cause painful swelling, teeth mobility, or bleeding from gingival sulcus
- Bruit or pulsations is present in Arterial malformation only.

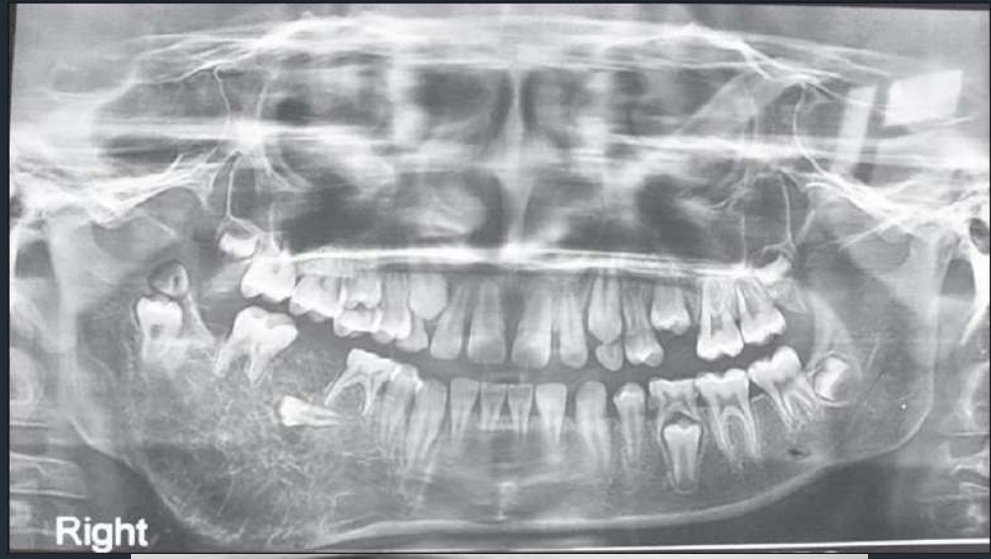


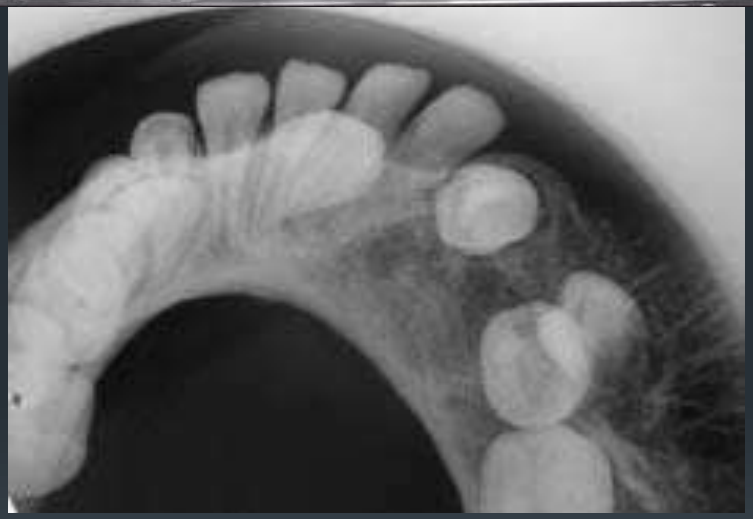


Radiographically

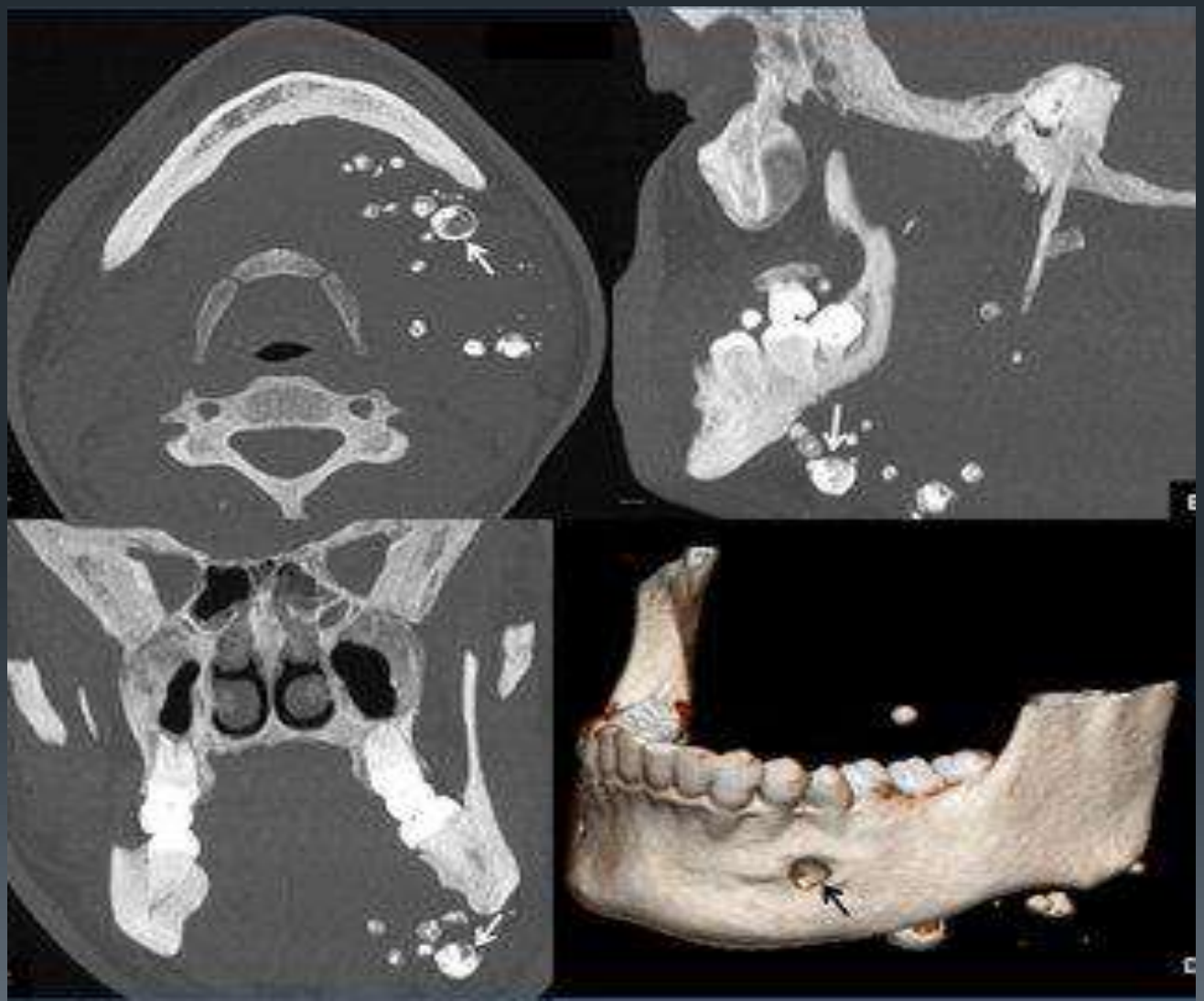
- a high-flow malformation = irregular poorly defined soap bubble Radiolucency which may cause resorption of roots

- Low- flow malformations are similar but are often somewhat better defined and may contain calcifications or phleboliths within them.





Phlebolith in VMF



Diagnosis of jaw vascular lesion

- To avoid the possibility of inadvertently carrying out a tooth removal or a biopsy in the presence of a high-flow malformation,
- a diagnostic needle aspiration should be carried out preoperatively.
- If bright red blood under pressure is encountered, surgery should be abandoned

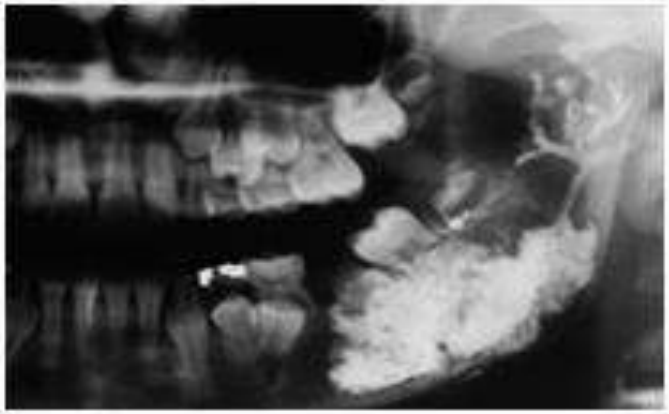
- When a vascular malformation is suspected or diagnosed,

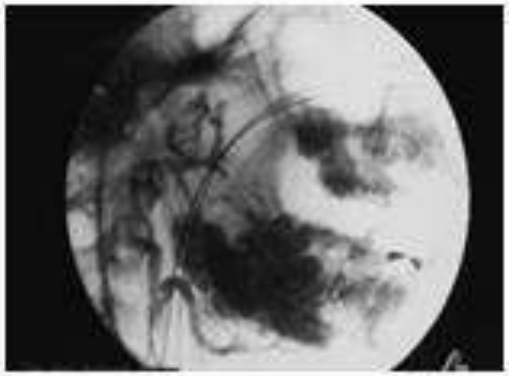
selective angiography is normally performed via a femoral approach

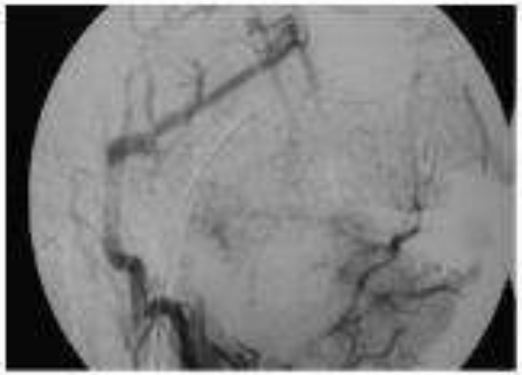
- If a high-flow vascular malformation is diagnosed, treatment is normally preoperative embolization followed by wide resective surgery

- LVM are not as life-threatening and are normally treated with direct puncture and an

attempt to thrombose the lesion by intralesional injection of a variety of agents, including sclerosing agents, an absorbable gelatin sponge, and platinum coils







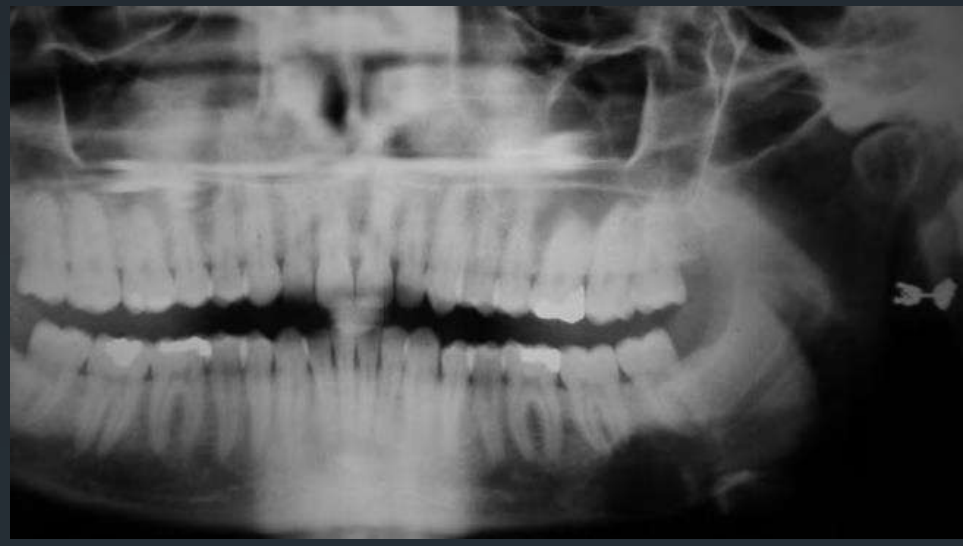
Malignant tumors of jaw

OSTEOSARCOMA

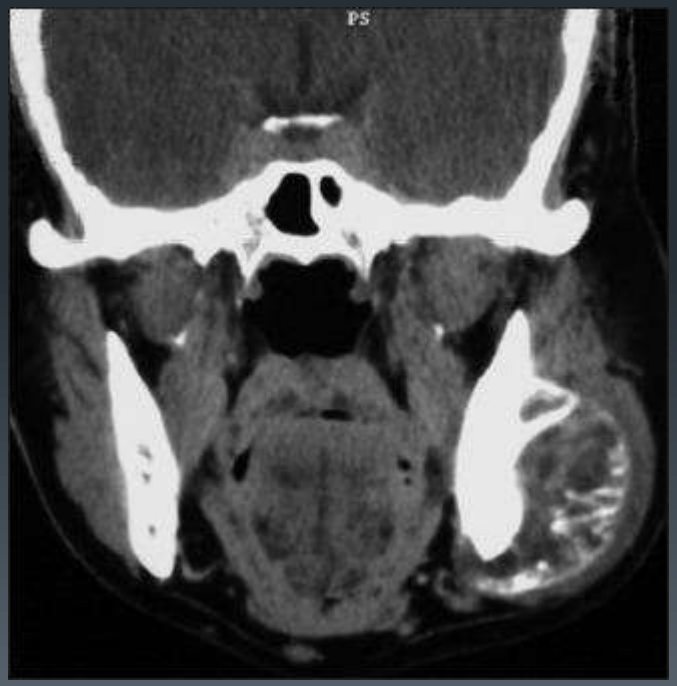
- Osteosarcoma is a malignant tumor characterized by the direct production of osteoid by a sarcomatous stroma
- It is the most common primary sarcoma of bone and second only to plasma cell neoplasms as the most common primary tumor of bone.
- It can develop in previously irradiated bone, as well as preexisting bone abnormalities such as Paget's disease, fibrous dysplasia, and giant cell tumors.
- Osteosarcomas may be classified into the more common central type, which arises from the medullary portion of the bone, and the less common peripheral (juxtacortical) type, which originates on the surface of the bone and initially grows outward.
- Most often involve the distal femur and proximal tibia of patients in their second decade of life.
- Lesions involving the jaws account for 5% to 7% of all osteosarcomas and most commonly affect patients in their third and fourth decades of life, with a mean age of approximately 35 years.
- There is a slight male predilection. The mandible is affected more frequently than the maxilla

Signs and symptoms

- swelling
- pain
- paresthesia,
- loosening of teeth,
- nasal obstruction, epistaxis, proptosis, or diplopia

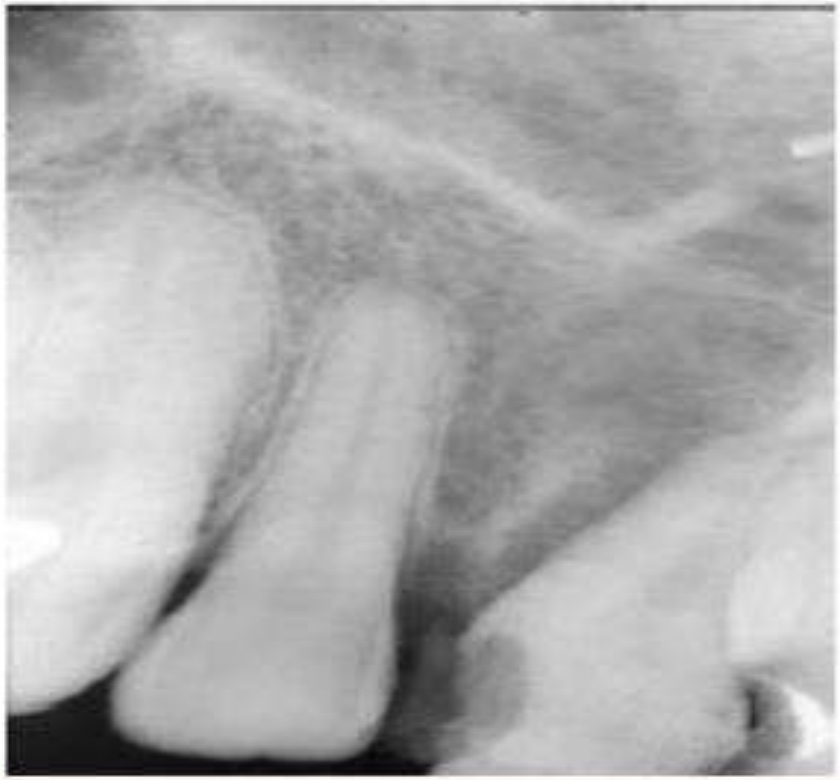


- dense radiopaque area to a mixed radiopaque and radiolucent lesion to a radiolucent process.
- margins are usually irregular and poorly defined.
- Symmetric widening of the periodontal ligament
- extracortical bone producing a “sunburst” appearance



➢ Radiographically

• Symmetric widening of periodontal ligament space: Due to tumour infiltration.



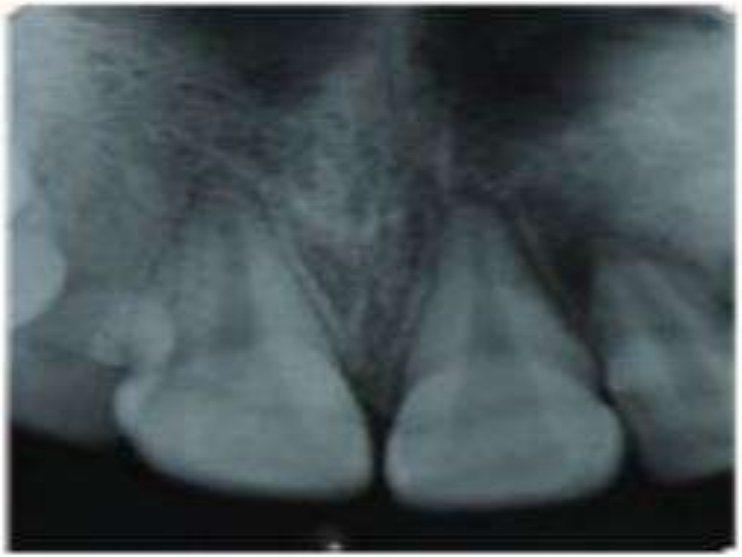
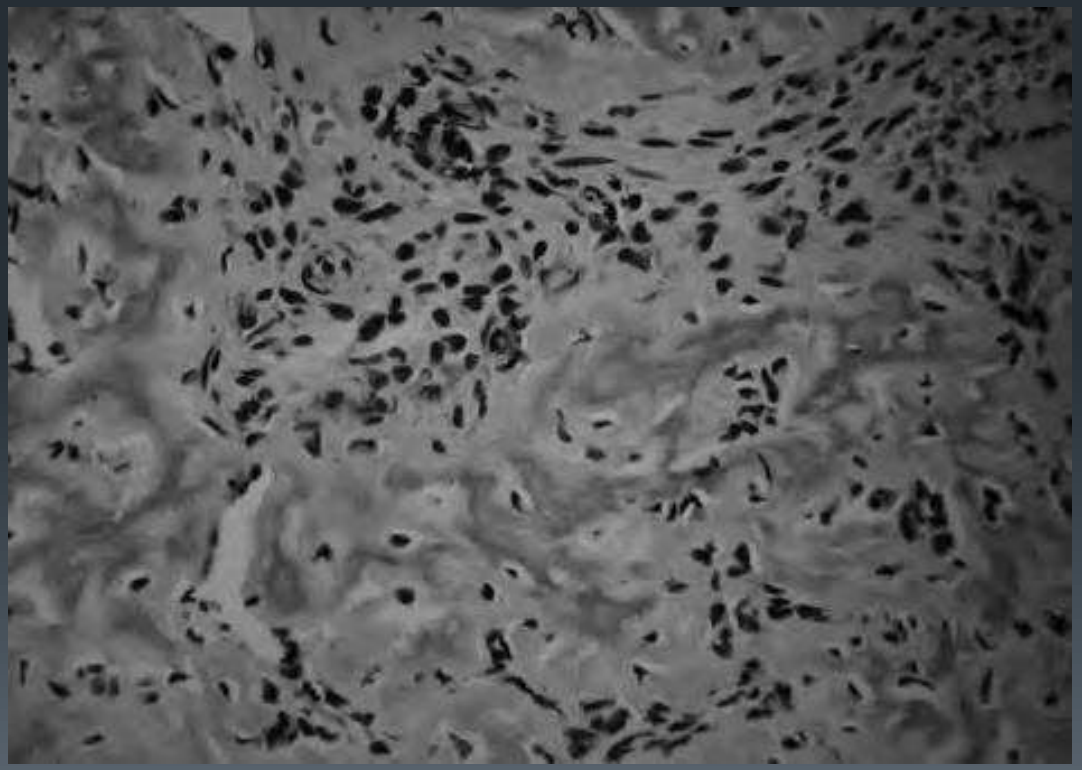


Fig. 4. Irregular widening of the periodontal ligament of 11, suggestive of osteosaroma.

Histologically

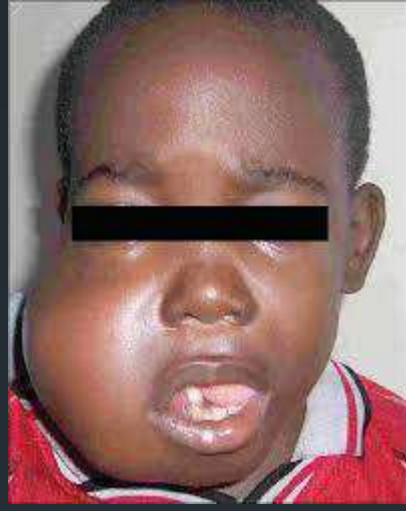
- Lesions are histologically subdivided into osteoblastic, chondroblastic, and fibroblastic subtypes, depending on the relative amounts of osteoid, cartilage, or collagen produced by the stroma



Treatment

- Wide surgical resection with negative margins is the only treatment that conclusively leads to increased survival. A bone margin of 3 cm from the radiographical margin is recommended
- At present, treatment protocols used in the treatment of osteosarcoma of the long bones, which include preoperative and postoperative chemotherapy and wide surgical resection, are commonly used to treat osteosarcomas of the jaws.
- 5-year survival rate for head and neck osteosarcomas of between 40% and 70%

Burkitt lymphoma



- Burkitt's lymphoma is a high-grade, non-Hodgkin's B-cell lymphoma that occurs in several clinical forms.

1. The endemic (African)
2. sporadic (American) forms.
3. HIV-associated form

- Clinically, jaw lesions of Burkitt's lymphoma can progress rapidly, appearing as a :
- Facial swelling or exophytic mass. These tumors may be associated with mobility of teeth, pain, and paresthesia

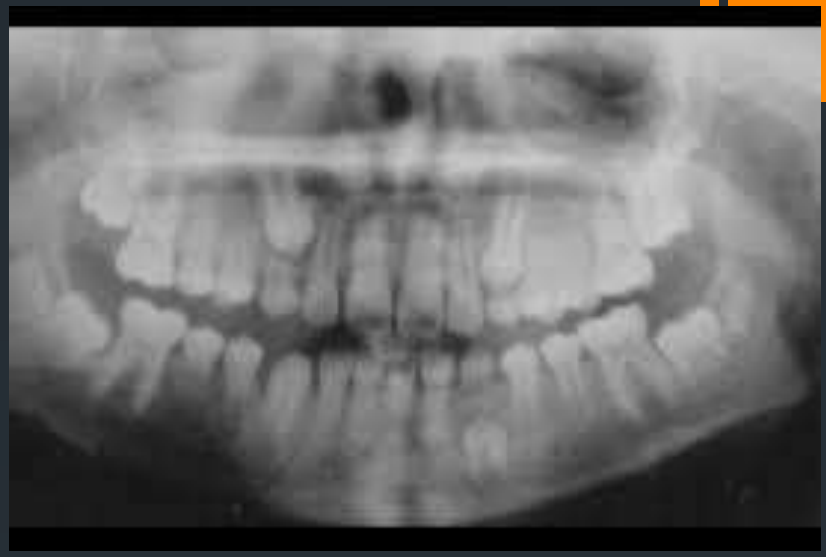
Endemic

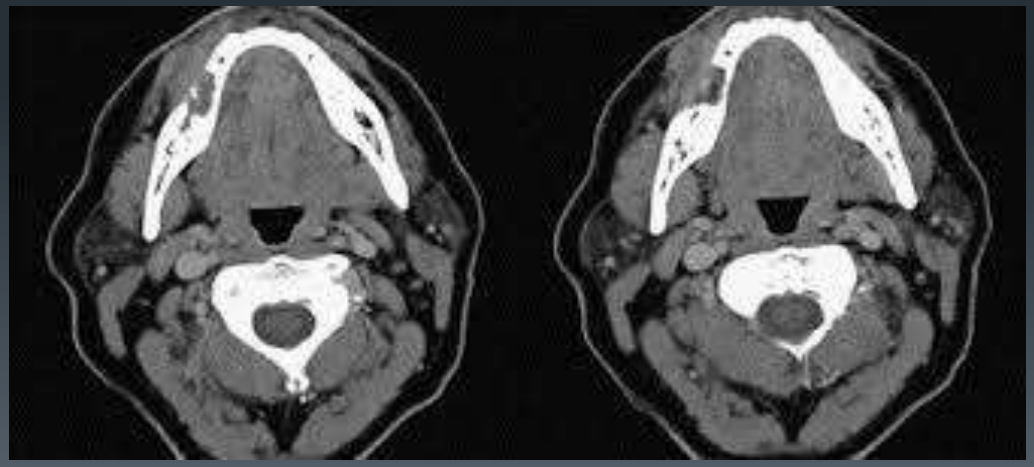
- associated with Epstein-Barr virus (EBV)
- peak incidence between 3 and 8 years of age.
- Jaw involvement is common
- The maxilla is involved more frequently than the mandible, although all four quadrants may be involved.

sporadic

- mostly EBV negative
- 10 and 12 years of age
- the jaws are involved in just 16% of cases
- Mandible >maxilla
- most commonly involving one quadrant;

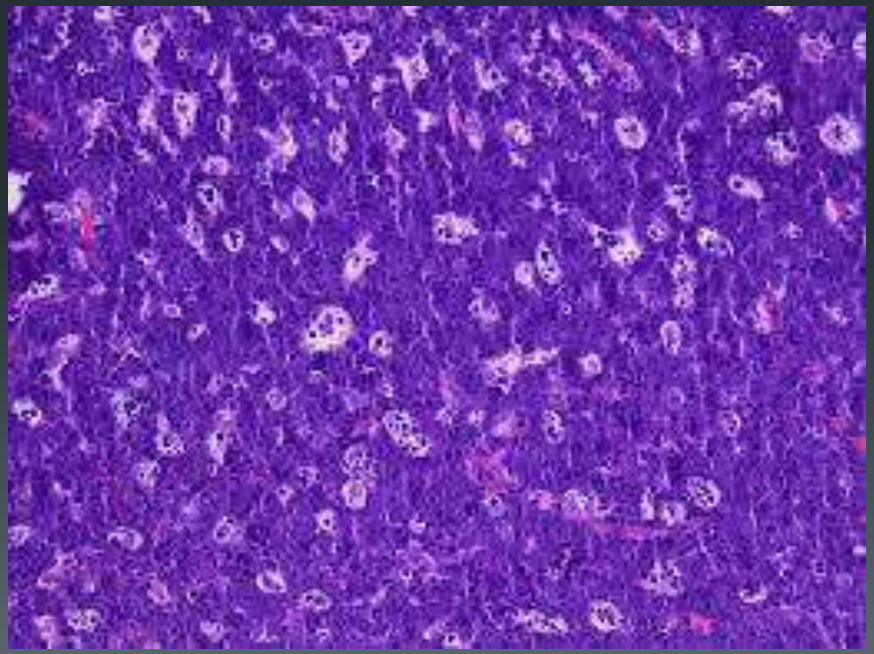
- Radiographically, an osteolytic process with ragged, ill-defined margins is seen





Histology

- The tumor is composed of sheets of medium-sized B cells with round nuclei and multiple nucleoli. Interspersed throughout the tumor cells are macrophages that stain less intensely than the hyperchromatic neoplastic B cells, resulting in a “starry sky” appearance



Management and prognosis

- This aggressive malignancy, if untreated, results in death within 4 to 6 months of diagnosis.
- Intensive chemotherapy has resulted in a dramatic improvement in the prognosis of Burkitt's lymphoma.
- Current treatment involves intensive, short-term, multi-agent chemotherapy

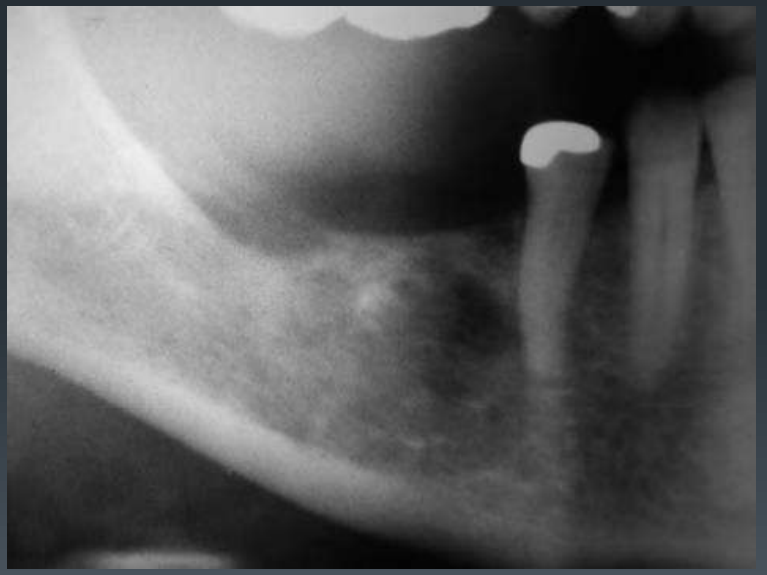
METASTATIC CARCINOMA

- Metastatic carcinoma is the most common form of malignancy affecting bone
- Bones with active marrow such as the vertebrae, ribs, pelvis, and skull are the preferential sites for metastasis.
- The most common sites of the primary carcinoma are the breast and lung, followed by the kidney, prostate,
- fifth to seventh decades, with a mean age of 45 years.
- Within the jaws, approximately 80% of the metastases are to the mandible (molar/premolar)
- C/P:

A. Swelling, pain paresthesia are the most common presenting symptoms.
B. Tooth mobility, trismus, and pathological fracture may seen

Radiographically

- , a metastatic lesion in the jaws usually appears as an irregular radiolucency



- The histological appearance of metastatic carcinoma is highly variable depending on the tumor type and the degree of differentiation.
- Renal and thyroid CA. → Well differentiated

Others → poorly differentiated

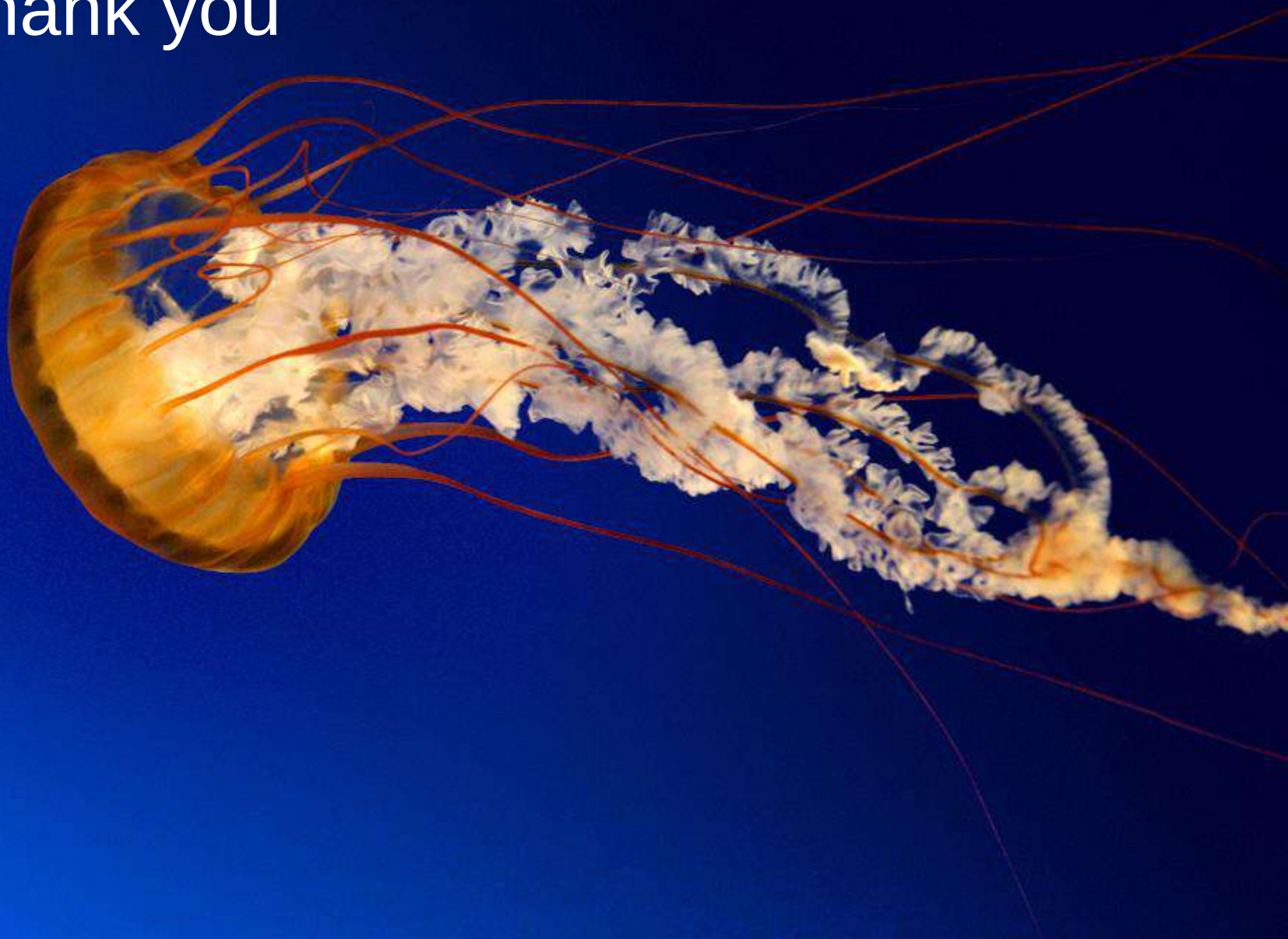
Q: HOW can you determine primary site of tumor?

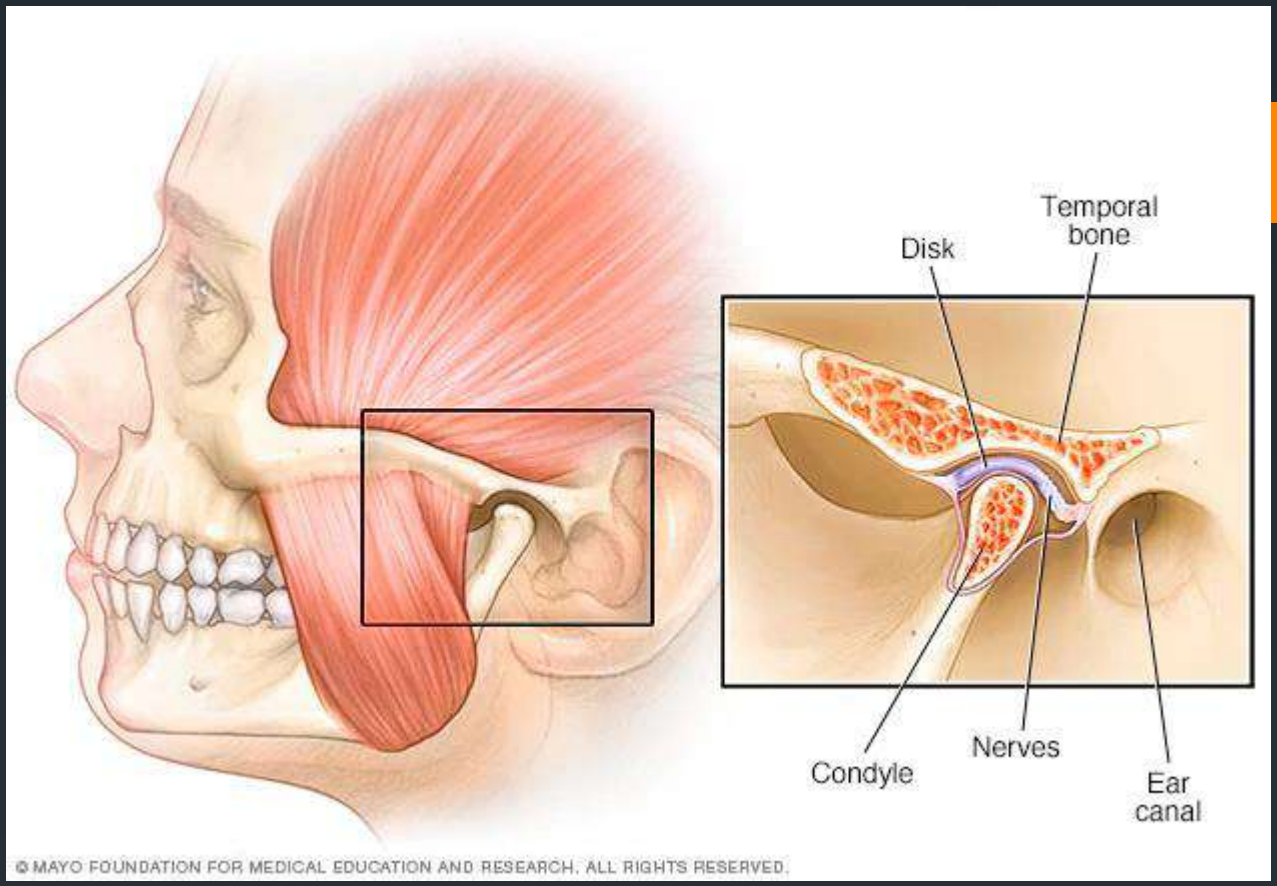
- Answer: immunohistochemistry can aid in determining the site of the primary carcinoma

Management of metastatic carcinoma to the jaws

- Management of metastatic carcinoma to the jaws begins with identification of the primary site and determining the extent of metastatic involvement.
- Jaw metastases are usually evidence of widely disseminated disease, and palliative treatment should be aimed.
- If the metastatic lesion of the jaw represents the only site of metastasis, adequate surgical treatment or chemoradiotherapy may improve the prognosis

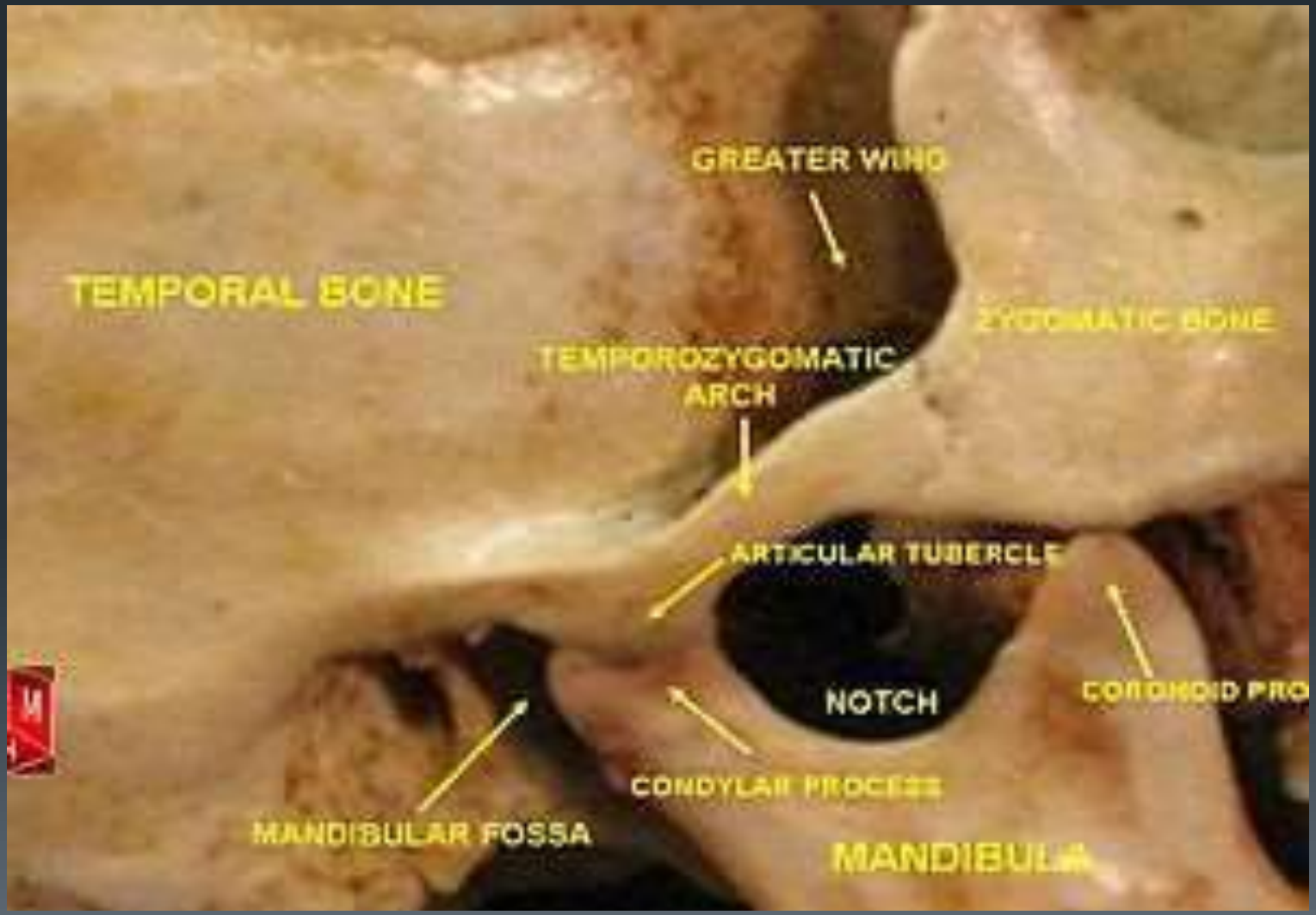
Thank you

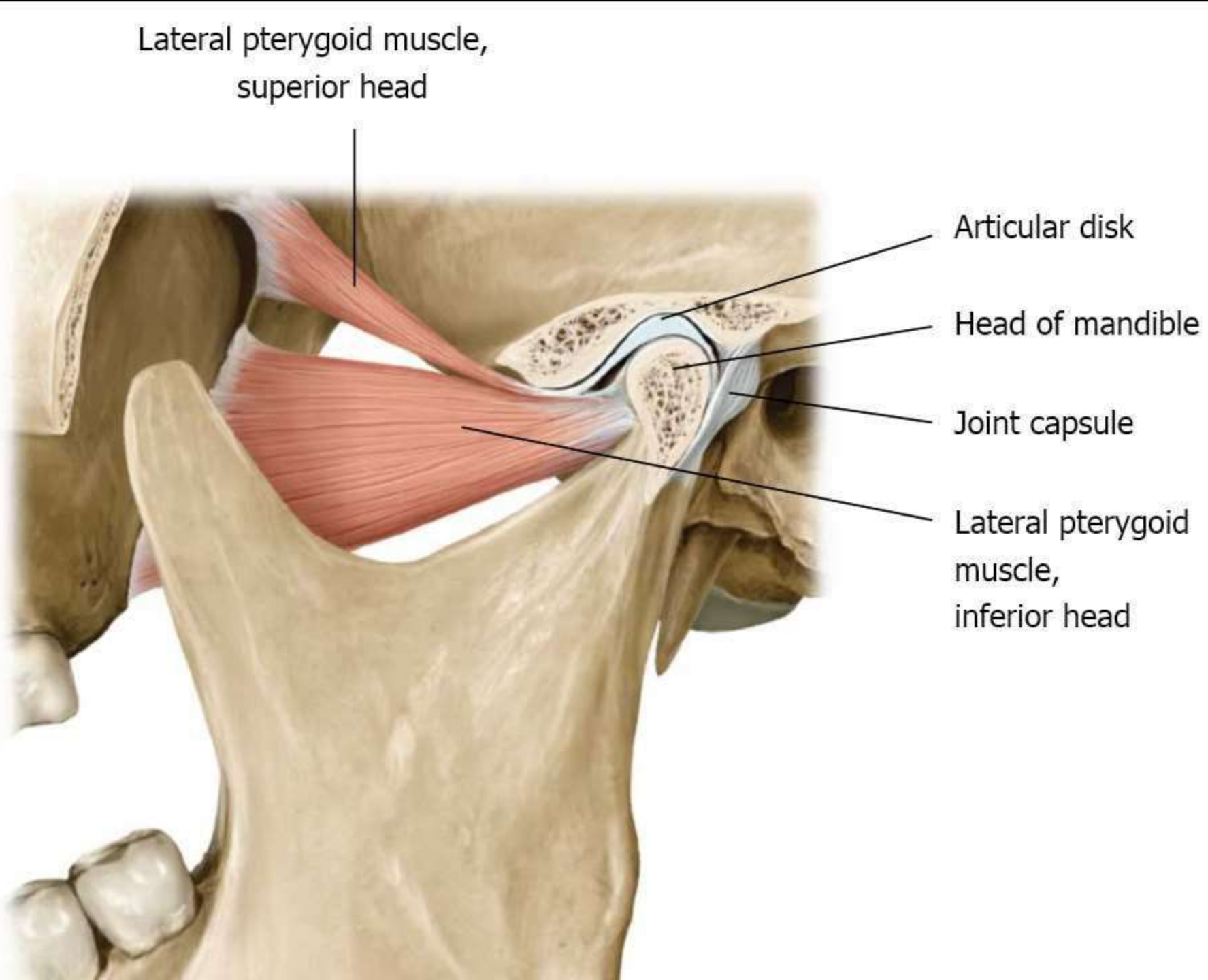


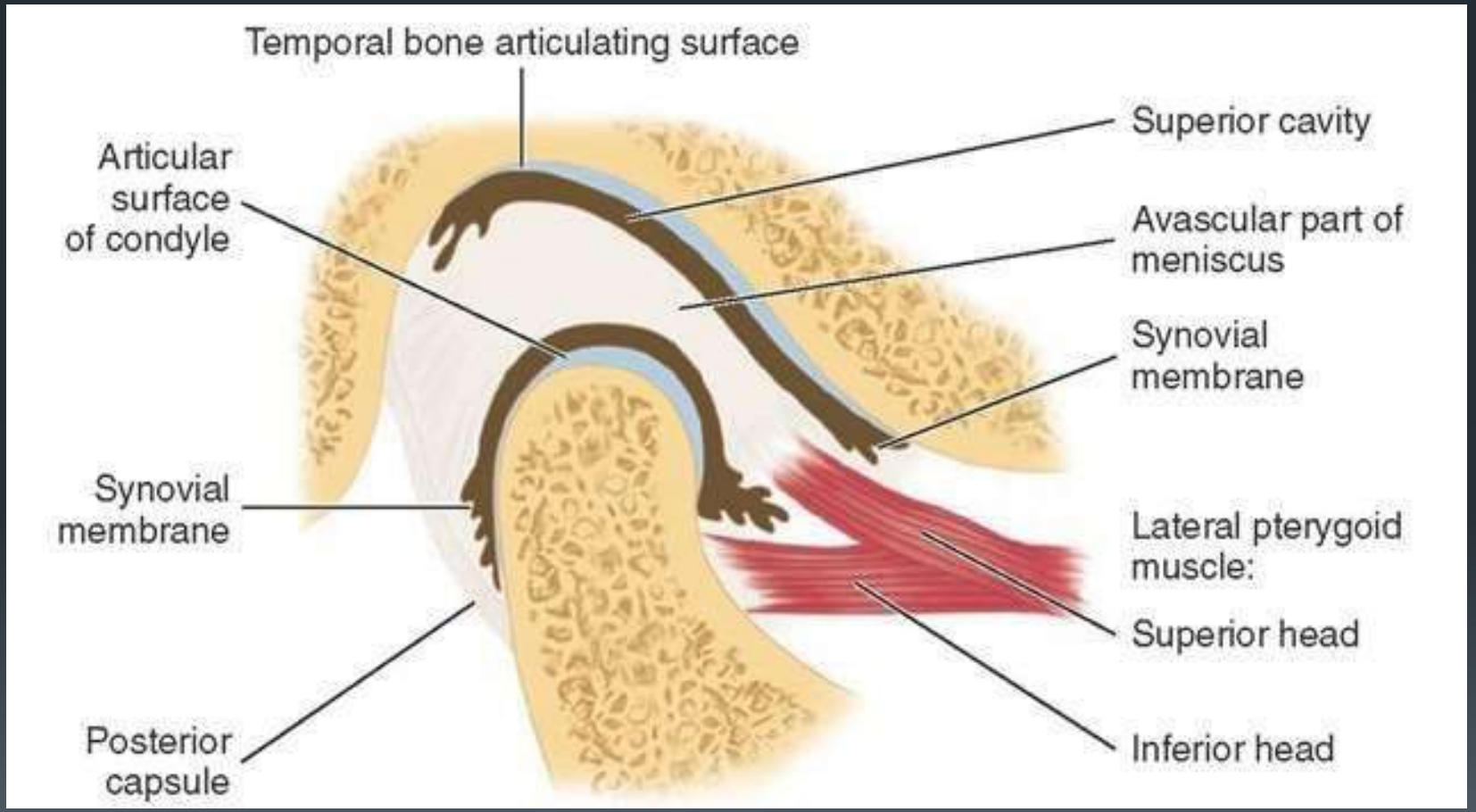


Temporomandibular Disorders

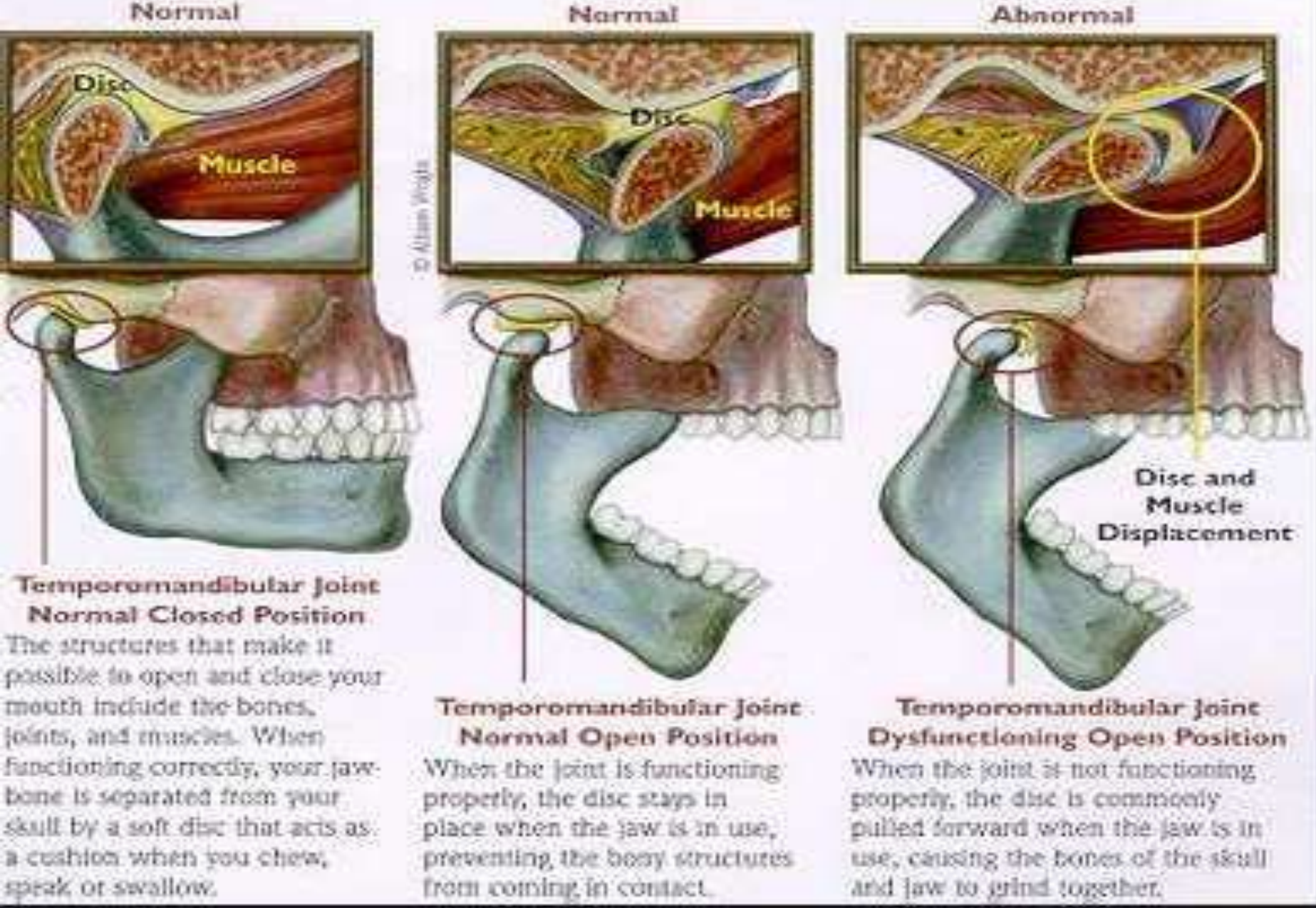
Anatomy of TMJ




Lateral pterygoid muscle, superior head
Articular disk
Head of mandible
Joint capsule
Lateral pterygoid muscle, inferior head


Temporal bone articulating surface
Articular surface of condyle
Synovial membrane
Posterior capsule
Superior cavity
Avascular part of meniscus
Synovial membrane
Lateral pterygoid muscle:
Superior head
Inferior head

Function of the Temporomandibular Joint



Temporomandibular Joint Normal Closed Position

The structures that make it possible to open and close your mouth include the bones, joints, and muscles. When functioning correctly, your jawbone is separated from your skull by a soft disc that acts as a cushion when you chew, speak or swallow.

Temporomandibular Joint Normal Open Position

When the joint is functioning properly, the disc stays in place when the jaw is in use, preventing the bony structures from coming in contact.

Temporomandibular Joint Dysfunctioning Open Position

When the joint is not functioning properly, the disc is commonly pulled forward when the jaw is in use, causing the bones of the skull and jaw to grind together.

The evaluation of the patient with temporomandibular pain, dysfunction

- History
- physical examination of the masticatory system
- TMJ radiography.
- Special diagnostic studies should be performed only as indicated and not as routine studies.

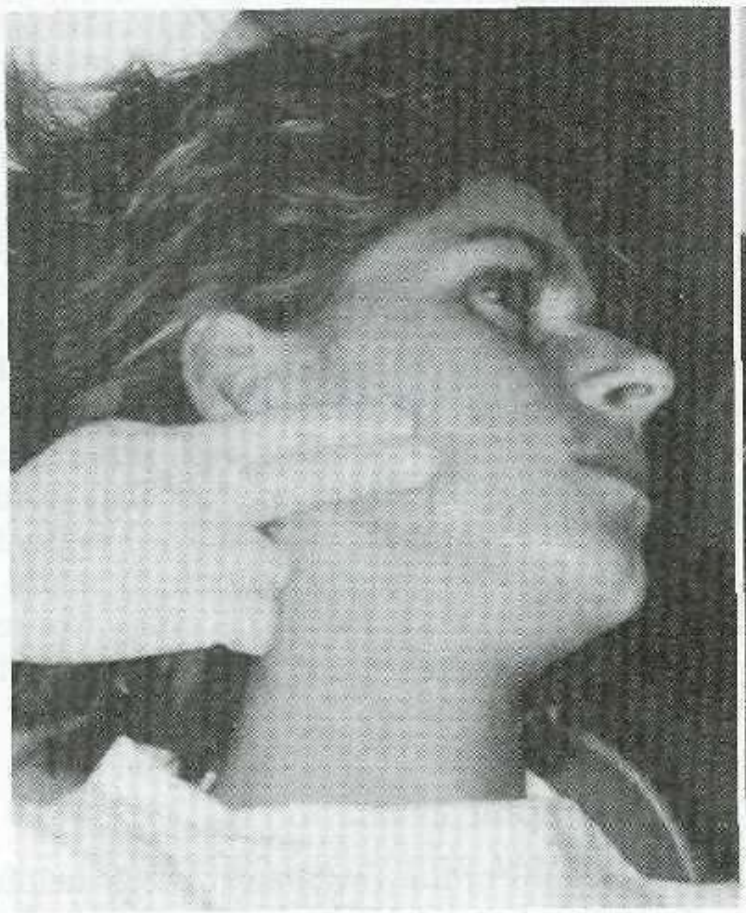
physical examination

1- Evaluation of the entire masticatory system

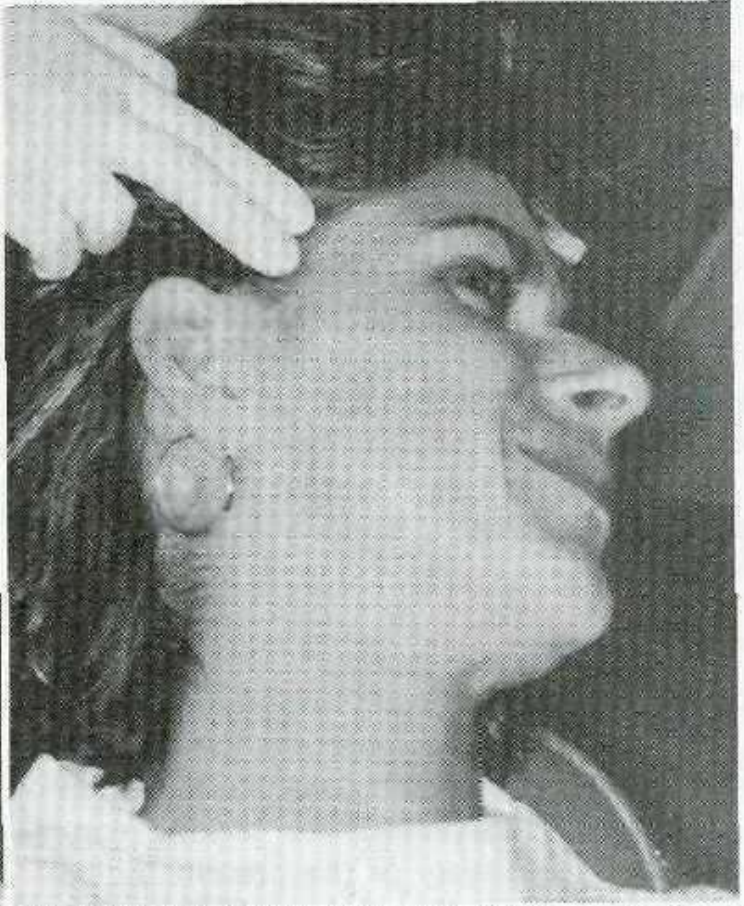
- On inspection: Asymmetry , muscular hypertrophy. The patient should be observed for signs of jaw clenching or other habits.

- On palpation: The muscles should be palpated for the presence of

I. Tenderness,
II. Fasciculations,
III. Spasm
IV. Trigger points



A



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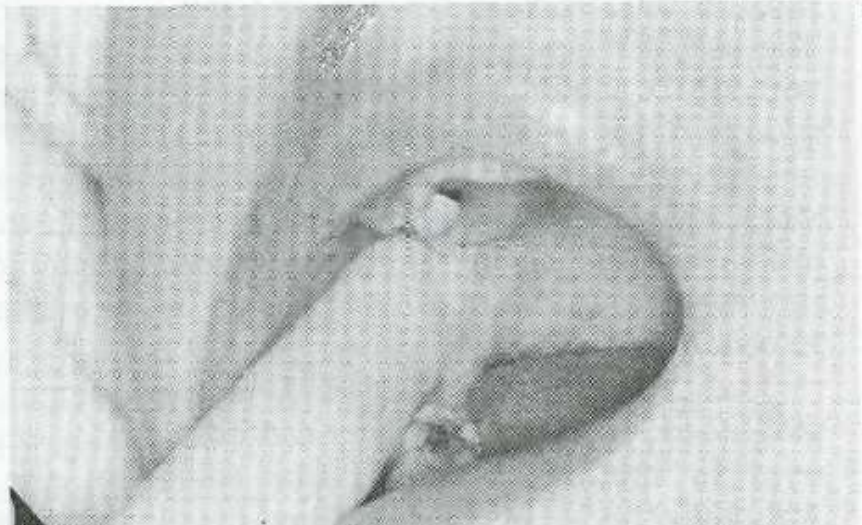

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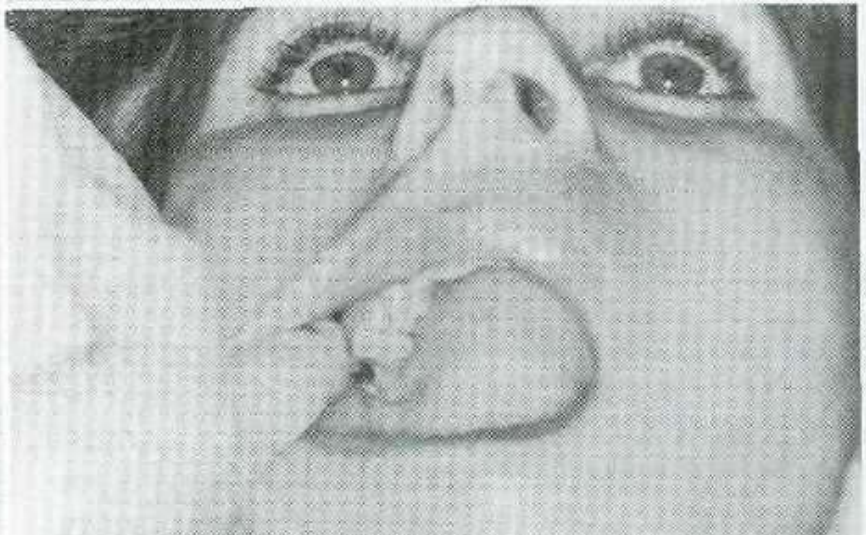
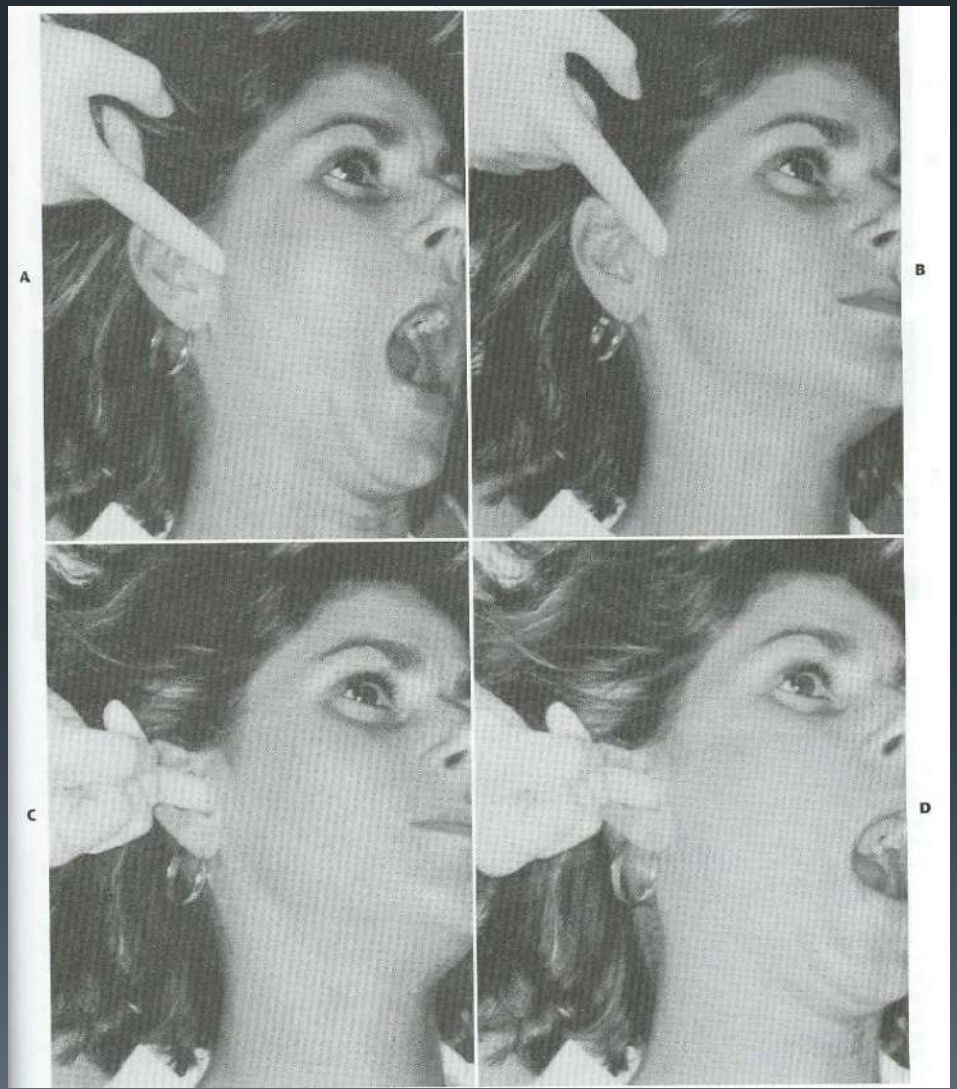


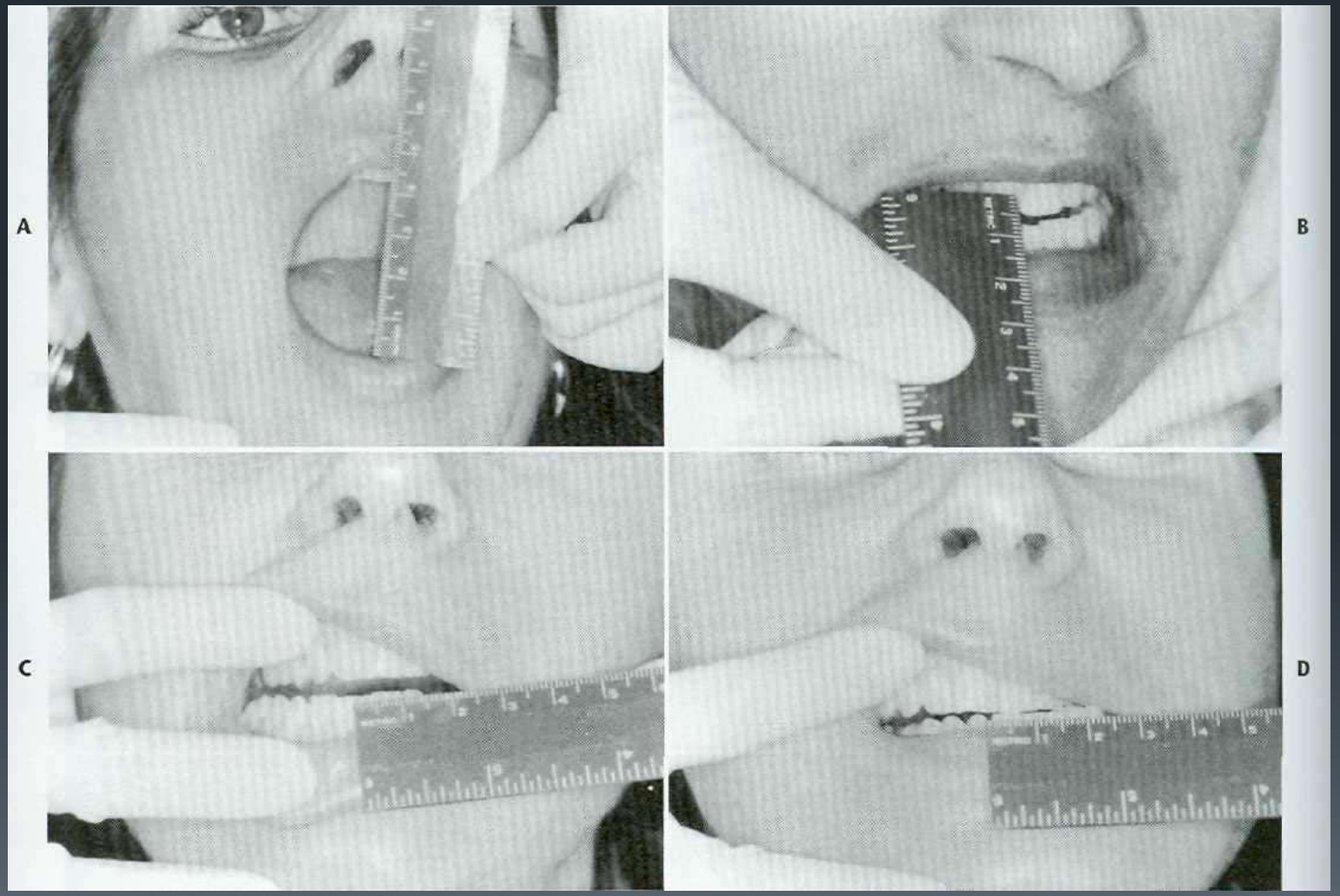
D

2- Examination of joint for noise and tenderness



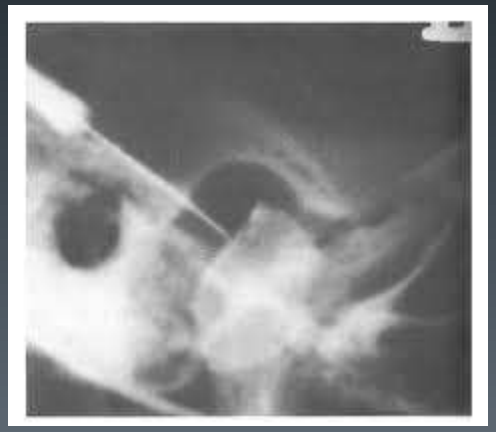
3- Examination: The mandibular range of motion should be determined.

- Normal range of movement of an adult's mandible is about 45 mm vertically (i.e., interincisally) and 10 mm protrusively and laterally

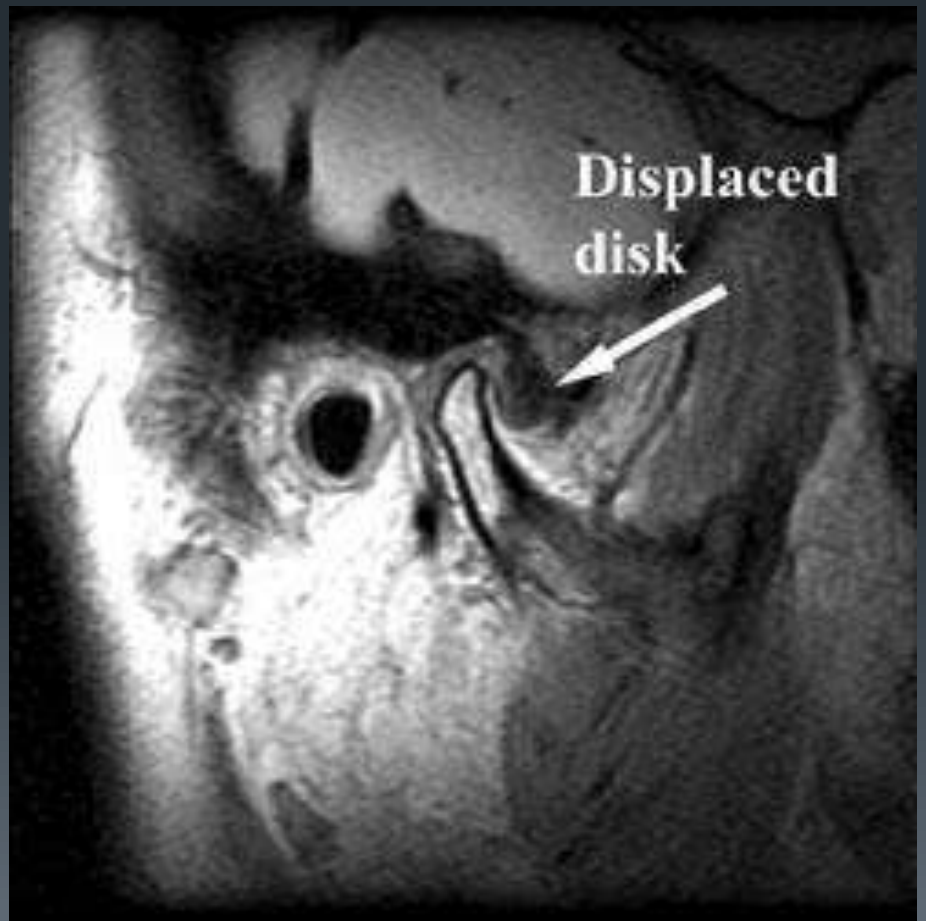


Radiographic Evaluation

1. ***Panoramic radiography:.***
2. ***Temporomandibular joint arthrography.*** Arthrography involves the injection of contrast material into the inferior or superior spaces of a joint, after which the joint is radiographed. This technique also demonstrates the presence of perforations and adhesions of the disk or its attachments.
3. ***Computed tomography.*** provide the most accurate radiographic assessment of the bony components of the joint .
4. ***Magnetic resonance imaging.*** The most effective diagnostic imaging technique to evaluate TMJ soft tissues is magnetic resonance imaging **(MRI)** (disc morphology and position)
5. ***Nuclear imaging***



MRI



CLASSIFICATION OF TEMPOROMANDIBULAR DISORDERS

- Myofascial Pain
- Disk Displacement Disorders
- Degenerative Joint Disease
- Systemic Arthritic Conditions
- Chronic Recurrent Dislocation
- Ankylosis
- Neoplasia
- infections

Myofascial pain and dysfunction (MPD)

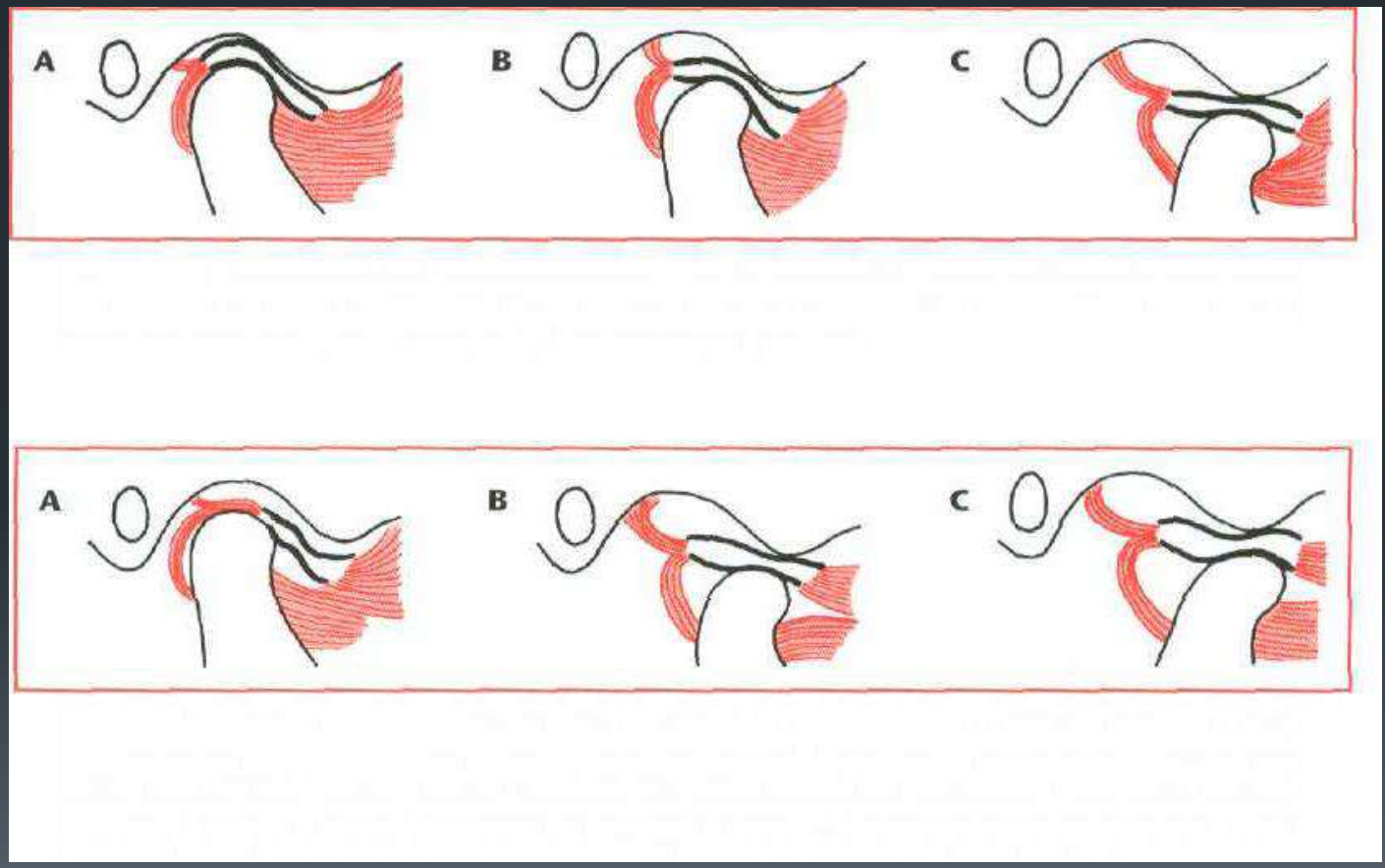
- Is the most common cause of masticatory pain and limited function
- The source of the pain and dysfunction is muscular
- Frequently but not always associated clenching or bruxism.
- Causes: stress, malocclusion, disc displacement
- C/P:

I. diffuse, poorly localized, preauricular pain
II. Tenderness muscles of mastication
III. decreased jaw opening with pain during functions such as chewing
IV. Headaches
V. joint noises are usually not present
VI. Radiographs of the TMJs are usually normal

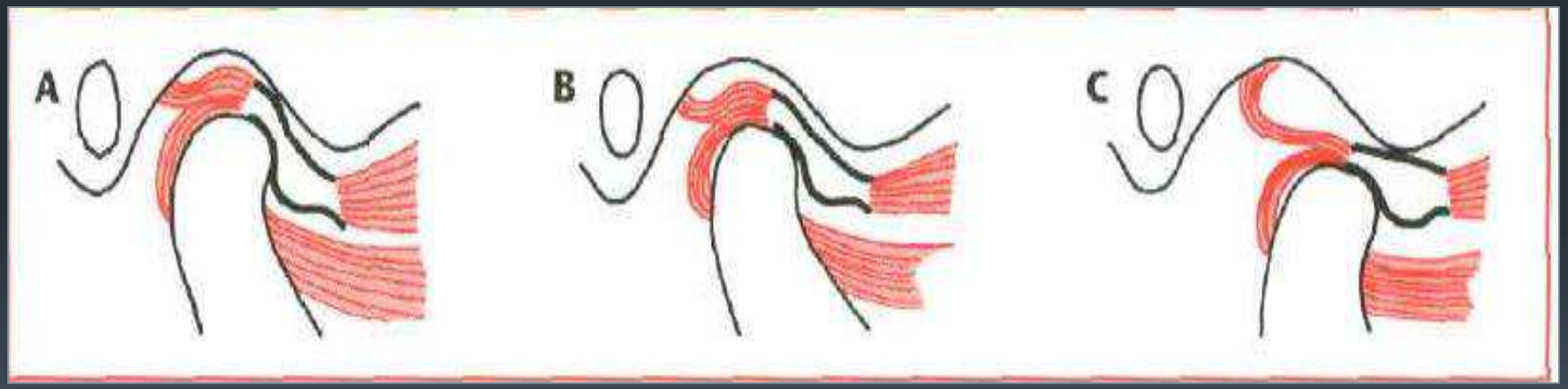
Disk Displacement Disorders

Normal TMJ

Disc displacement with reduction



Non reducing disc displacement



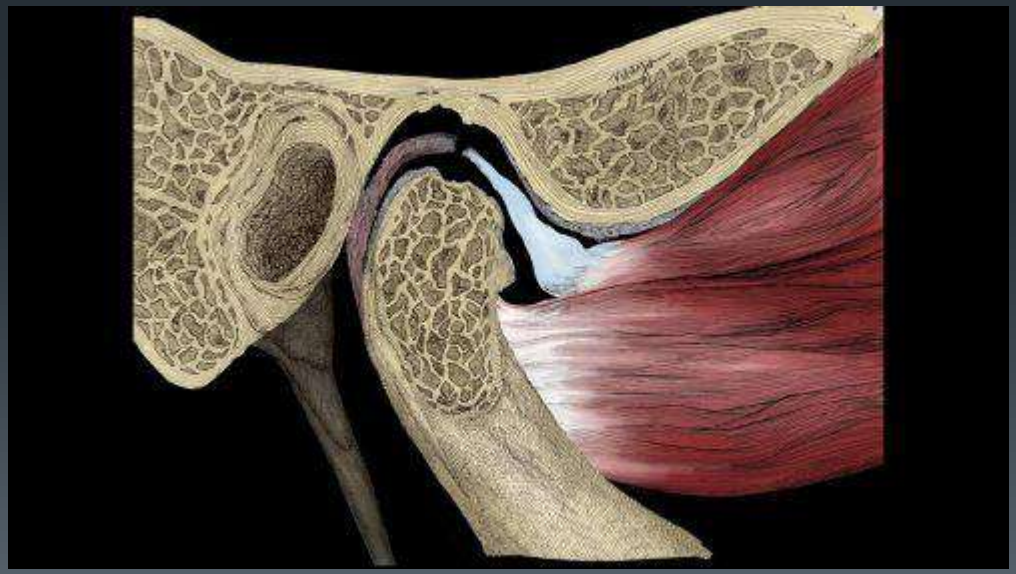
1. Joint noise: Clicking during opening the mouth. If it occurs during opening and closing, it is called reciprocal clicking
2. Painless but Joint tenderness and muscle tenderness may be seen
3. Maximal opening can be normal or slightly limited
4. Plain TMJ radiography: normal

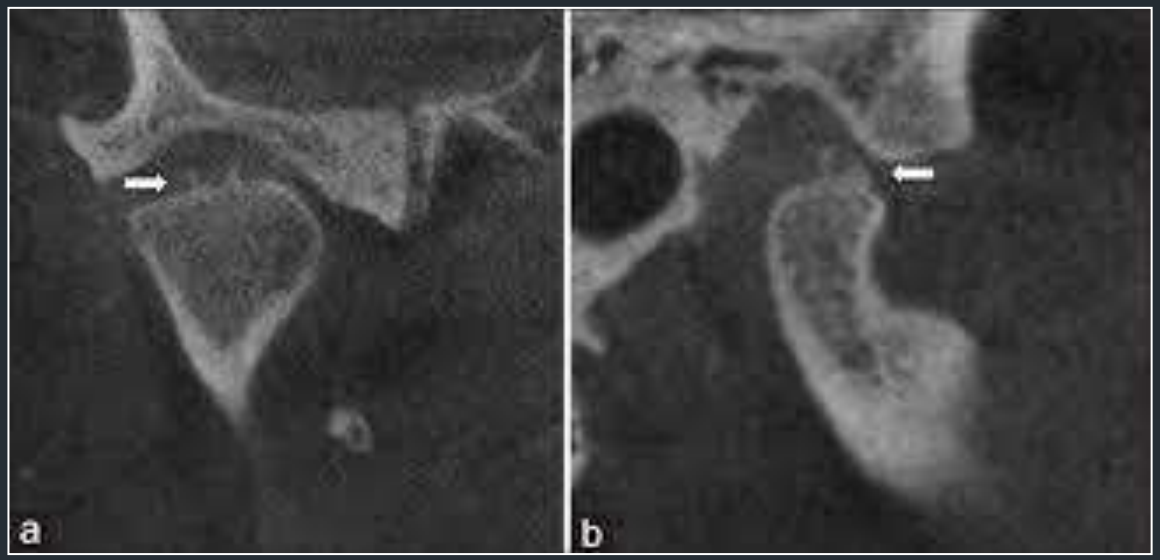
1. No clicking occurs, because they are unable to translate the condyle over the posterior aspect of the disk.
2. Joint tenderness and muscle tenderness
3. Restricted opening, deviation to the affected side, and decreased lateral excursions to the contralateral side
4. Plain Radiographic evaluation: normal

Degenerative Joint Disease (Arthrosis, Osteoarthritis

- includes a variety of anatomic findings, including

1. irregular, perforated, or severely damaged disks
2. articular surface abnormalities, such as articular surface flattening, erosions, or osteophyte formation

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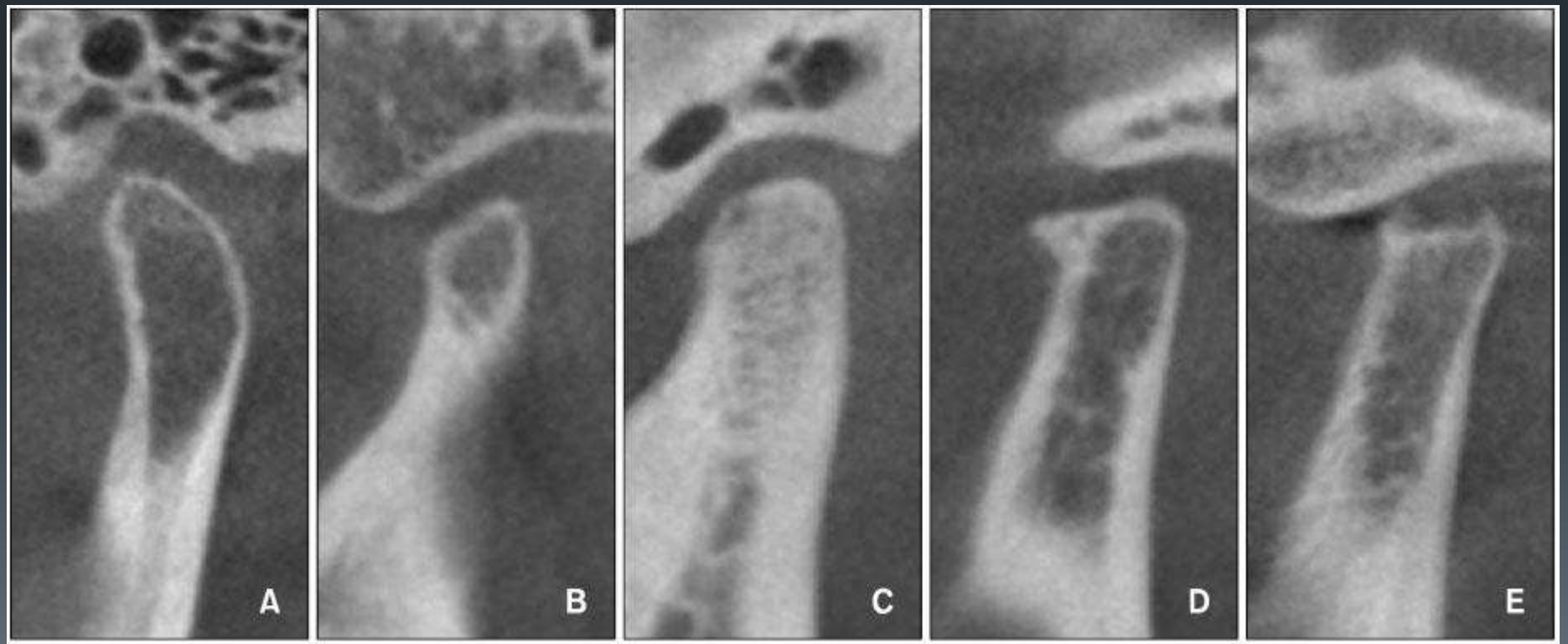
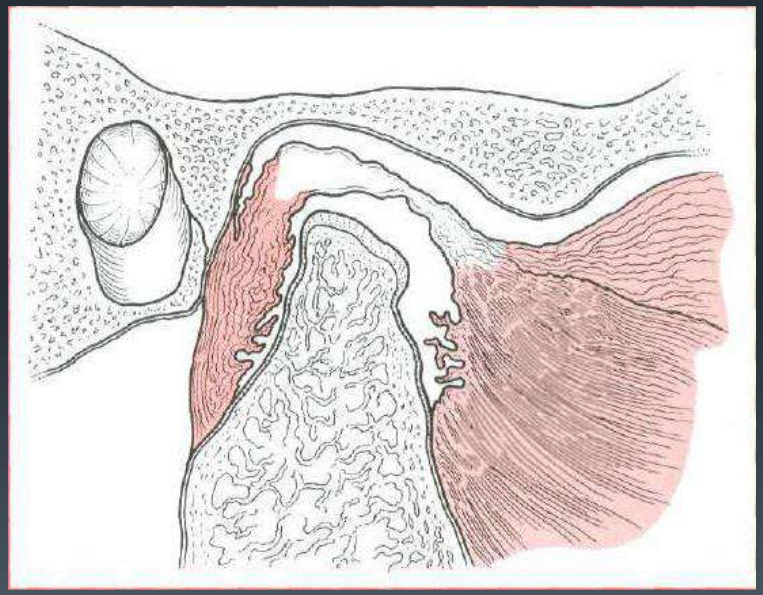

A
B
C
D
E

DJD

- pain associated with
- clicking or crepitus, located directly over the TMJ.
- Usually, an obvious limitation of opening is present

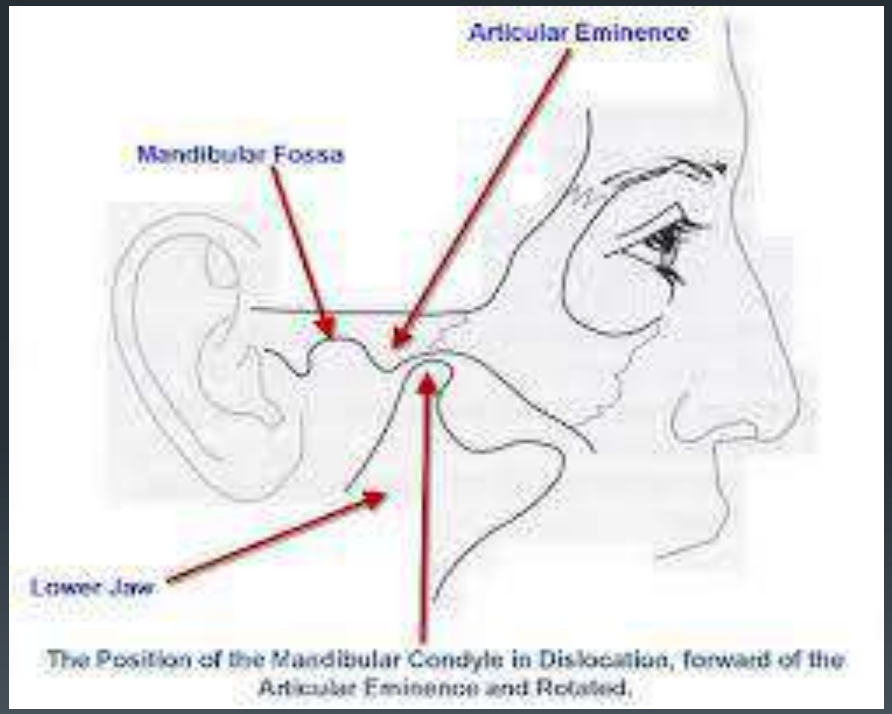
Rheumatoid arthritis.



- inflammatory process results in abnormal proliferation of synovial tissue in a so-called pannus formation.
- Same DJD Features but it is bilateral and associated with systemic manifestations
- ESR, RF level are elevated

Chronic Recurrent Dislocation

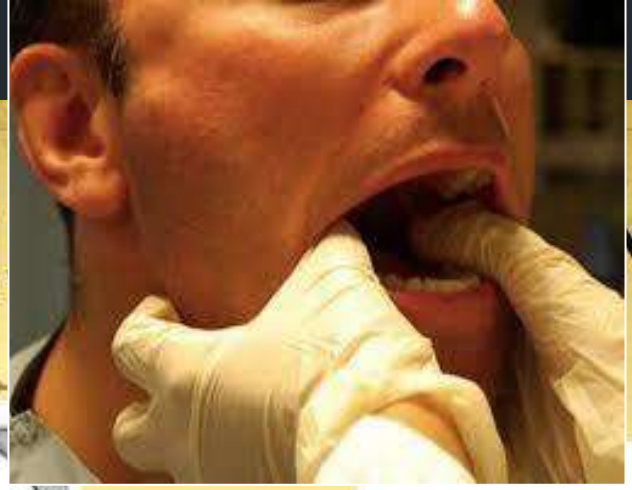
- Characterized by the condyle sliding over the articular eminence and becomes locked in that position .

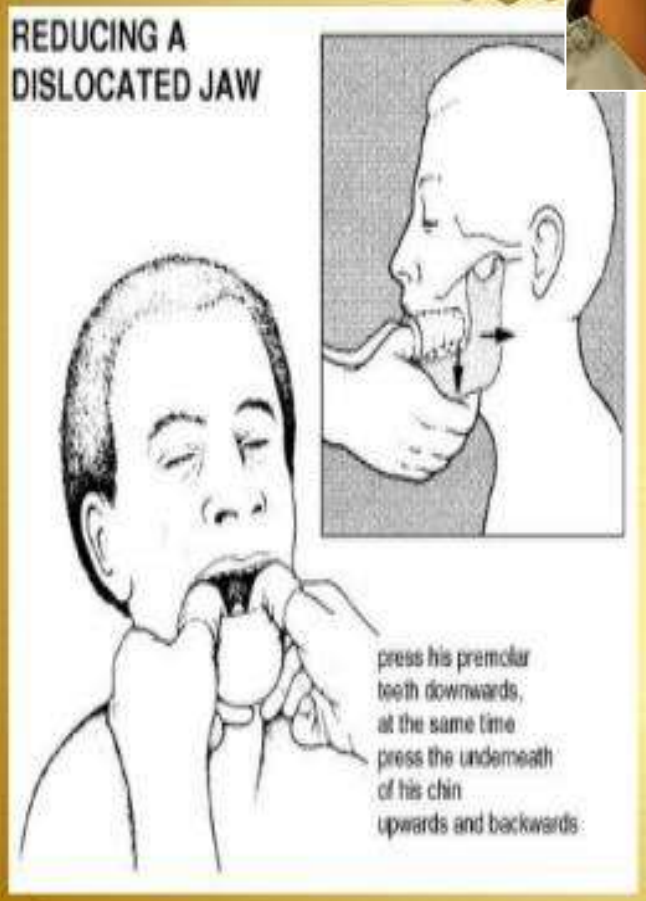


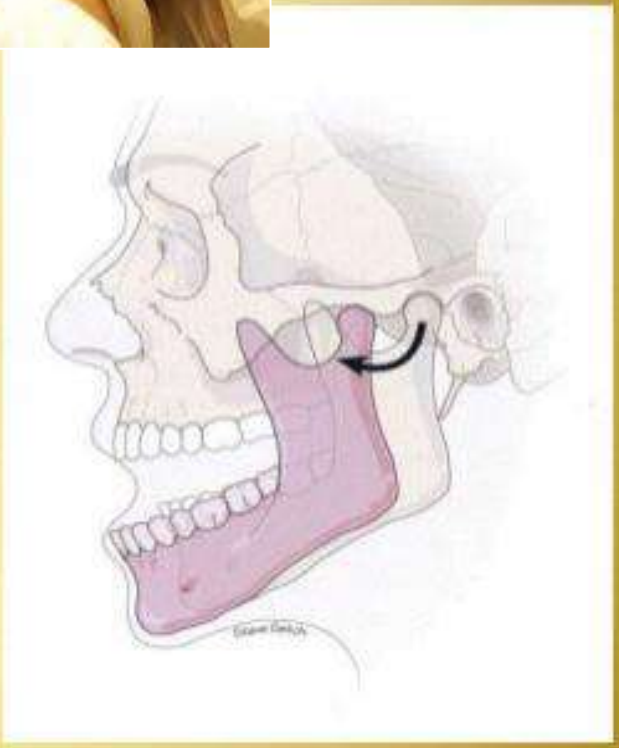
The Position of the Mandibular Condyle in Dislocation, forward of the Articular Eminence and Rotated.

- Dislocation may be unilateral or bilateral and may occur spontaneously after opening the mouth widely, such as when yawning, eating, or during a dental procedure
- painful and is often associated with severe muscular spasms
- Dislocations should be reduced as soon as possible.

TMJ DI…N….

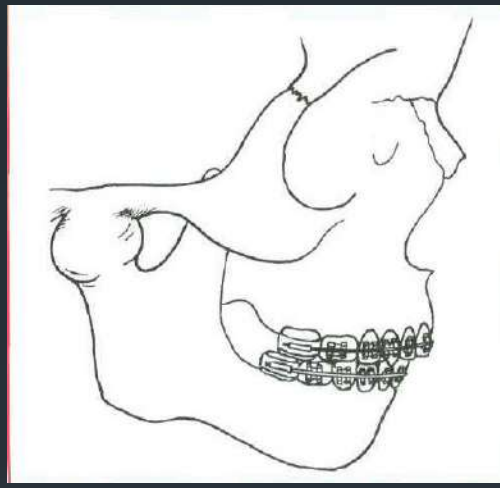



REDUCING A
DISLOCATED JAW
press his premolar
teeth downwards,
at the same time
press the underneath
of his chin
upwards and backwards



Ankylosis

- Bony fusion between the mandibular condyle and skull base involves the TMJ.
- MAY BE BONY, fibrous or fibro-osseous
- May be intracapsular or extracapsular ankylosis



- Trauma
- Infection
- Previous surgery

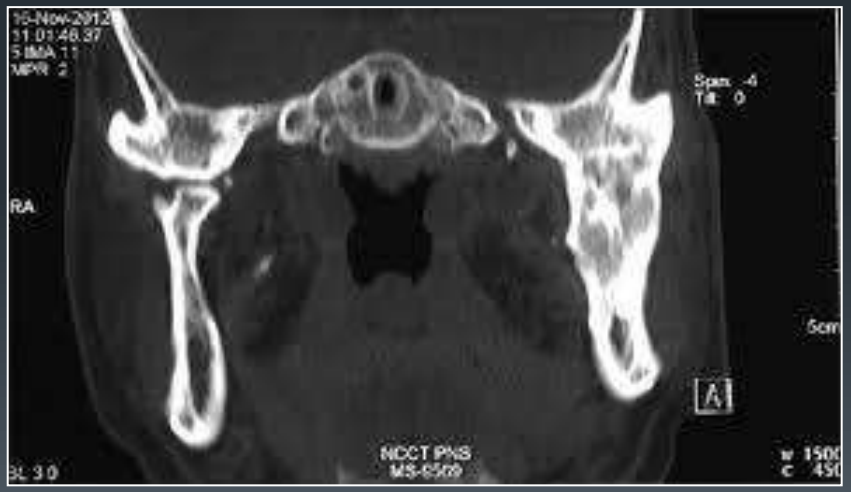
coronoid HP

infection around temporalis

zygomatic arch fracture

Features

- severe restriction of maximal opening,
- deviation to the affected side, and
- decreased lateral excursions to the contralateral side



Treatment of TMJ disorders

- **Non -surgical Treatment: must be applied to all patient**
- **Surgery**

Non-surgical treatment: Education and behavior modification
medications
splints
physical therapy

Diet

- Decreases muscle activity and loading forces on temporomandibular joints
- Controls range of motion—hinge and sliding
- Ranges from liquid diet to elimination of hard chewy food; involves cutting food into small pieces
- Eliminates gum chewing

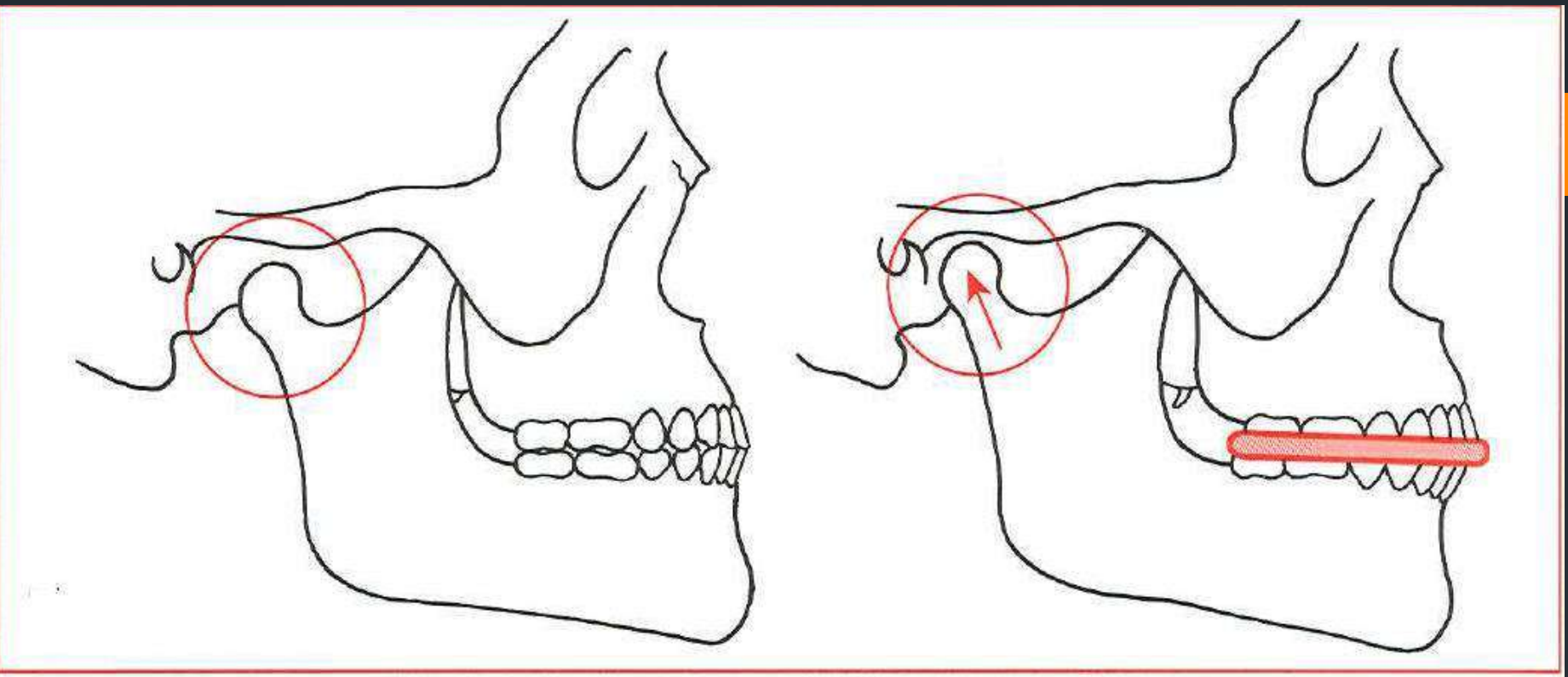
Medication

- (1) NSAIDs: meloxicam , piroxicam
- (2) analgesics: panadol, codeine
- (3) muscular relaxants: diazepam, cyclobenzaprine, myogesic
- (4) tricyclic antidepressants: amitryptaline
- .(5) anxiolytics.
- (6) local anesthetics

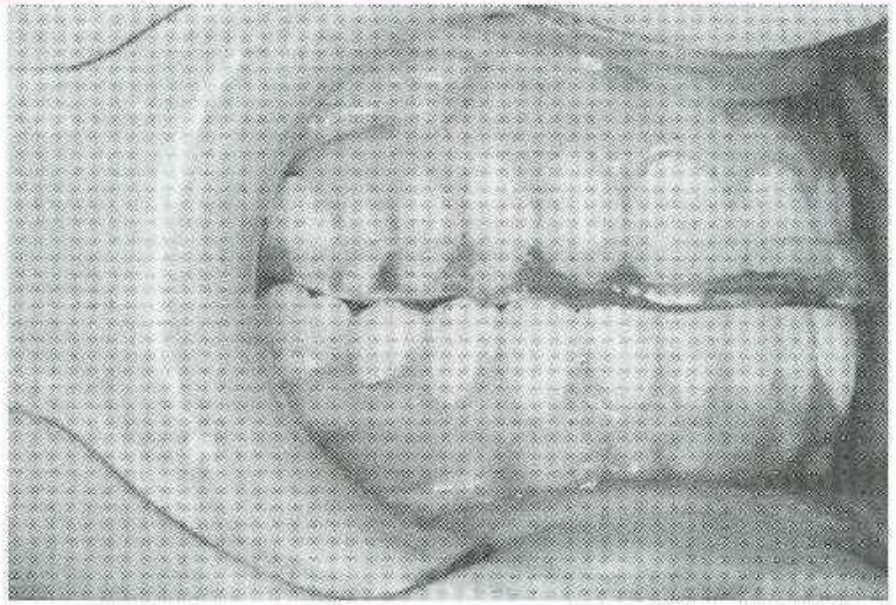
Table 48-13 Physical Therapy
Home Treatment Program (good for mild acute symptoms)
Soft diet
Decrease function
Heat/ice packs
Jaw/tongue posture opening exercise
Lateral jaw movement
Control passive motion (ie, Therabite)
Office Treatment (reduction of pain and inflammation)
Ultrasonography
Transcutaneous electrical nerve stimulation
Range of motion
Soft tissue manipulation
Trigger point injections
Acupuncture (reestablishing proper energy flow by adding electric current or heat to the placed acupuncture needle)

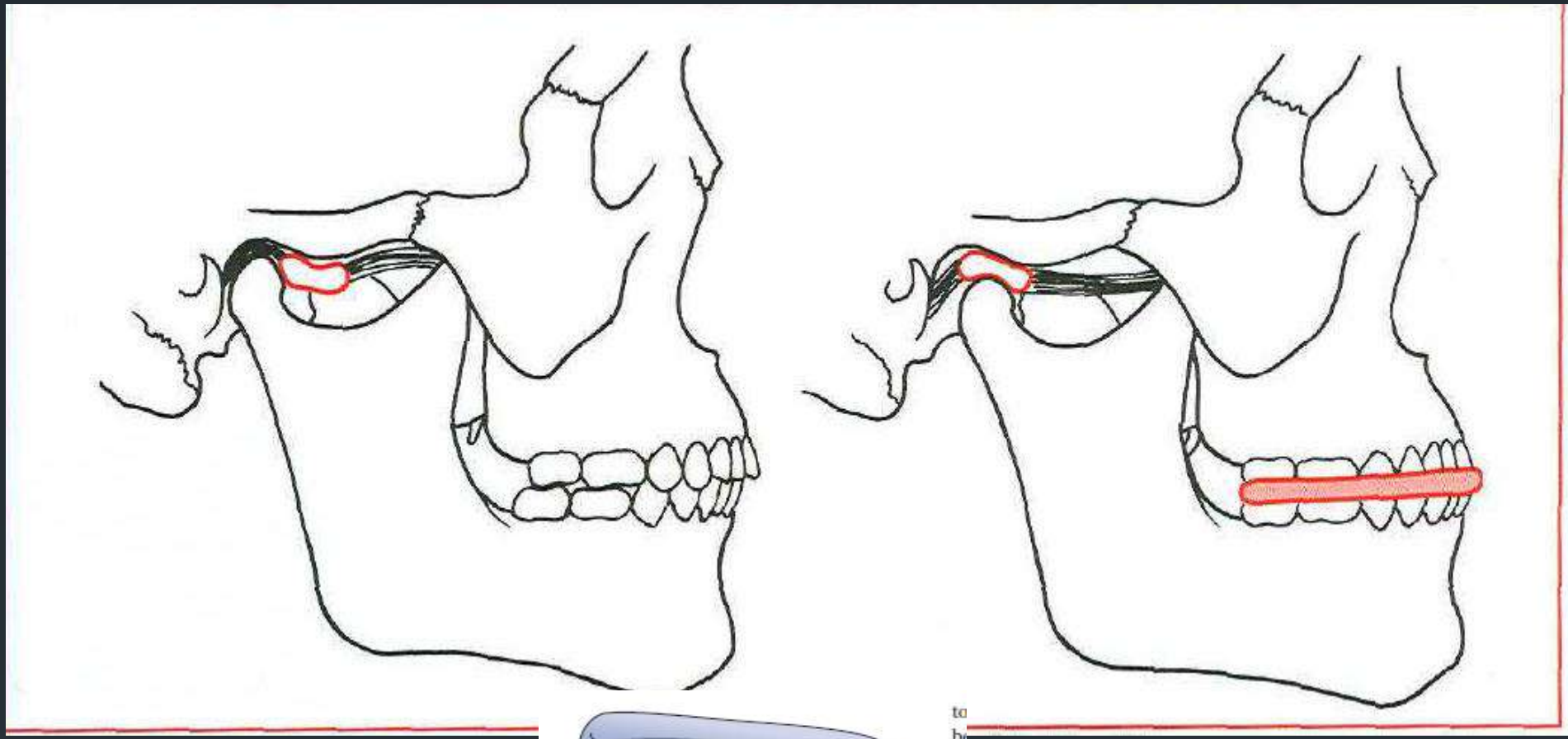
FIGURE 48-2 *Patient using passive jaw exercise device to improve interincisal opening and to break any fibrous bands. (This Ther-*

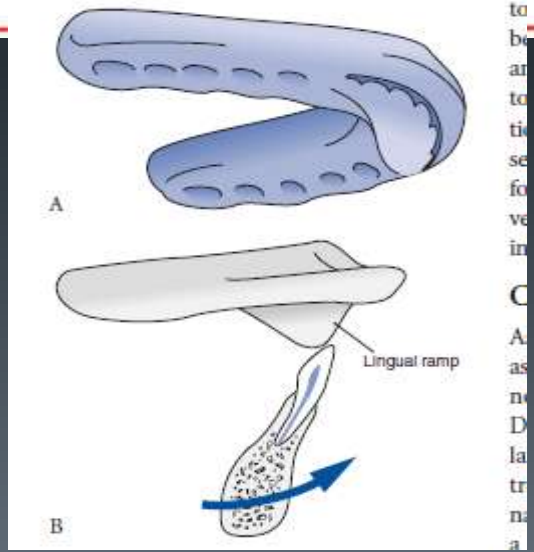
Splints



c






A
Lingual ramp
B

TEMPOROMADIBULAR JOINT SURGERY

1- Arthrocentesis:.

2-Arthroscopy:

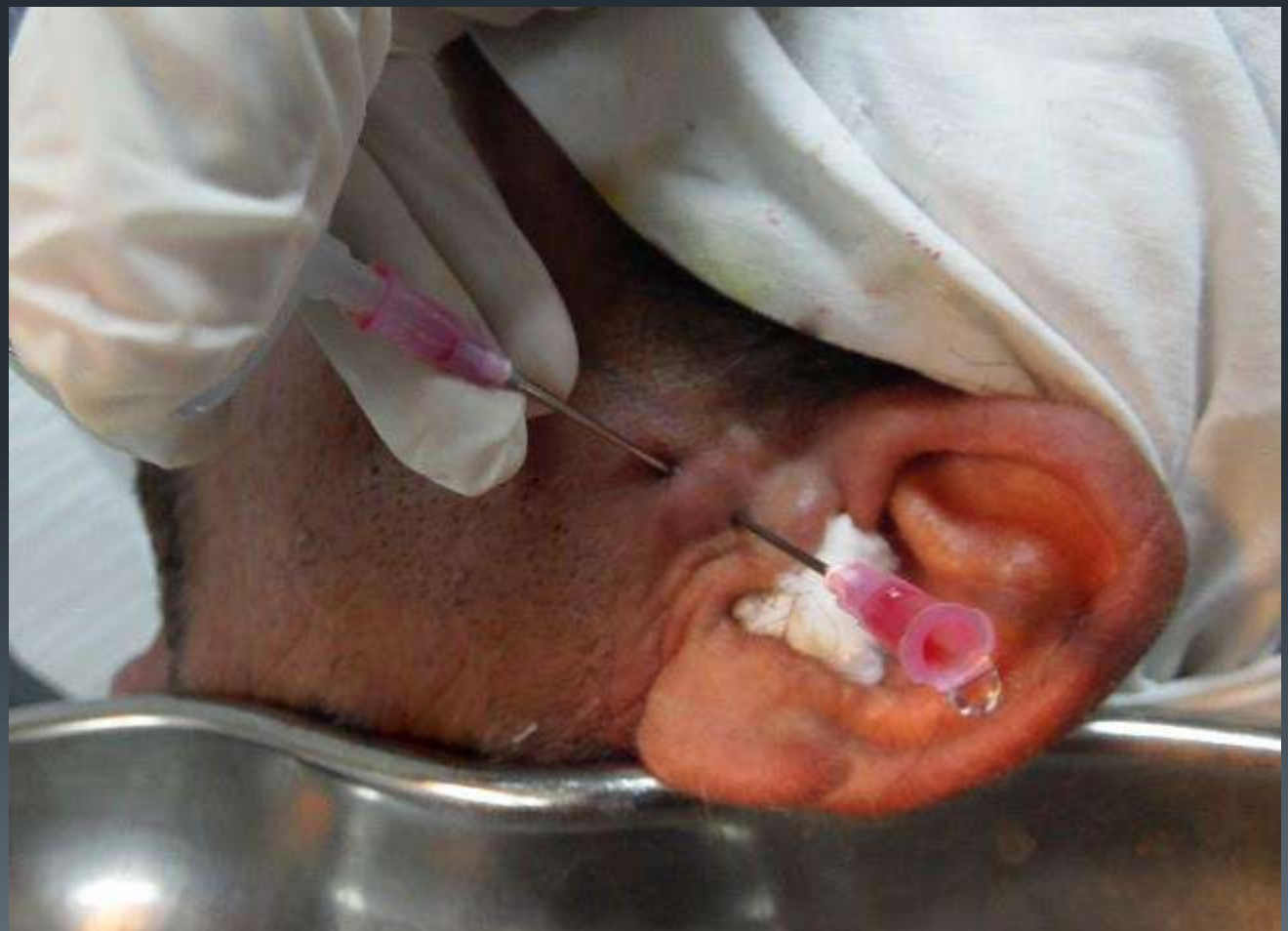
3-Disk-Repositioning Surgery

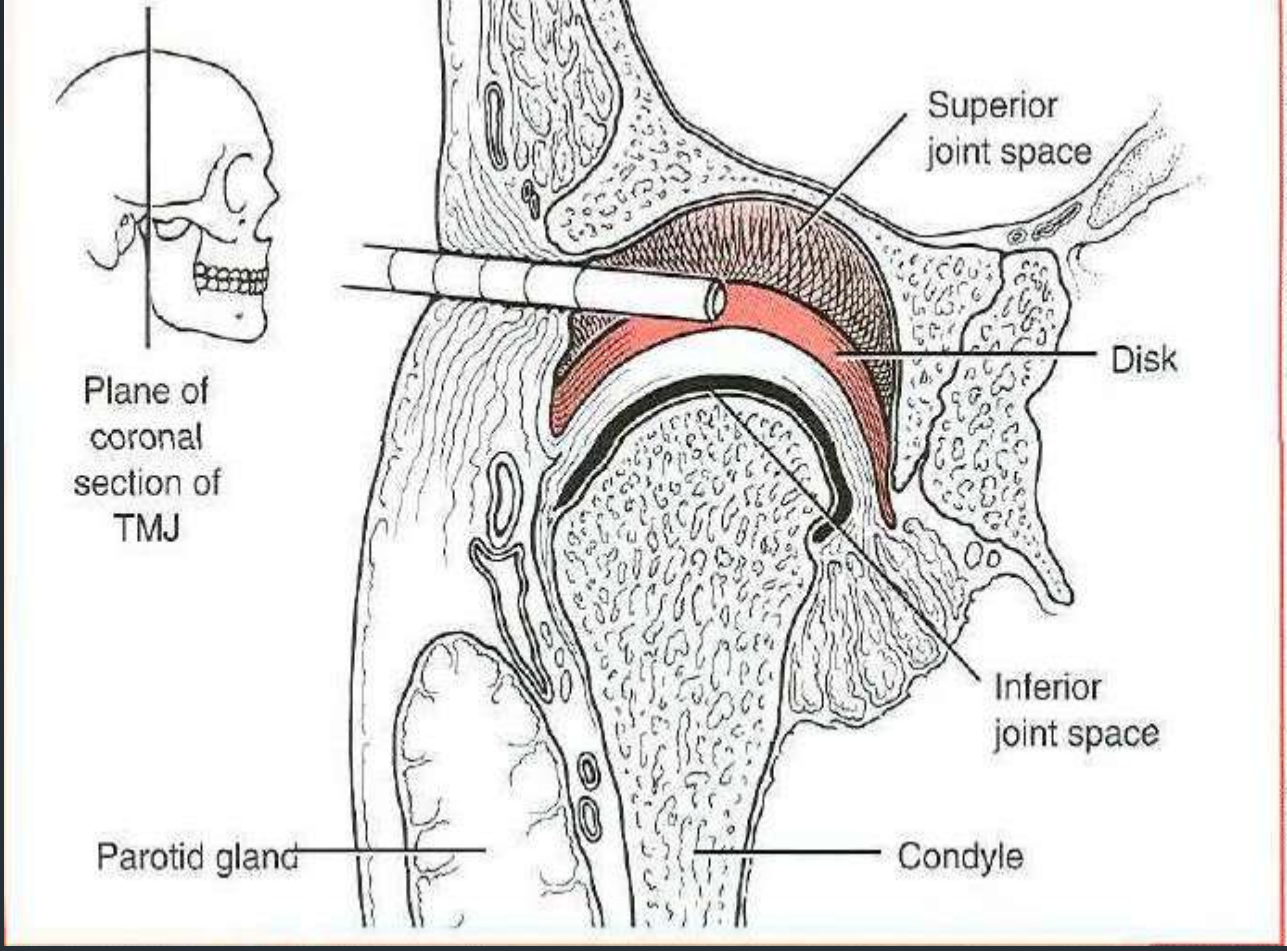
4-Disk Repair or Removal

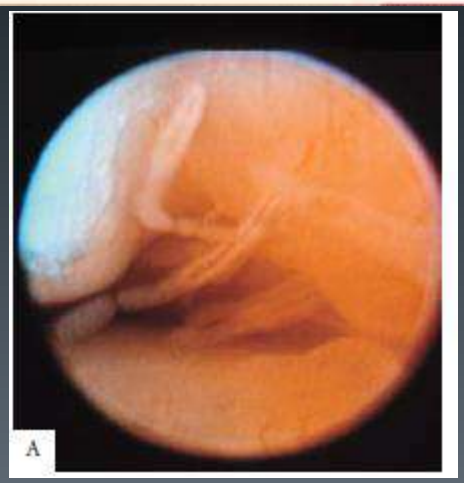
5-Condylotomy

6-Total Joint Replacement

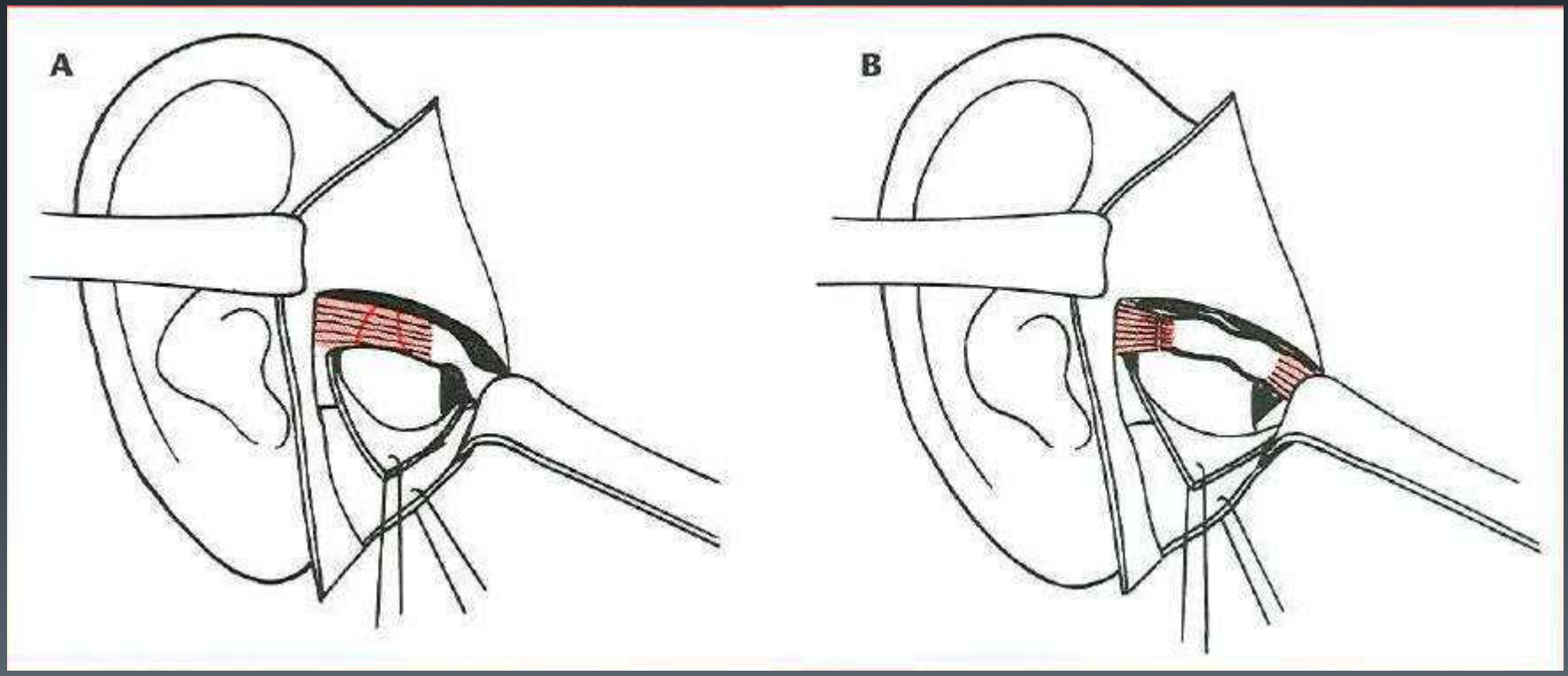
Arthrocentesis



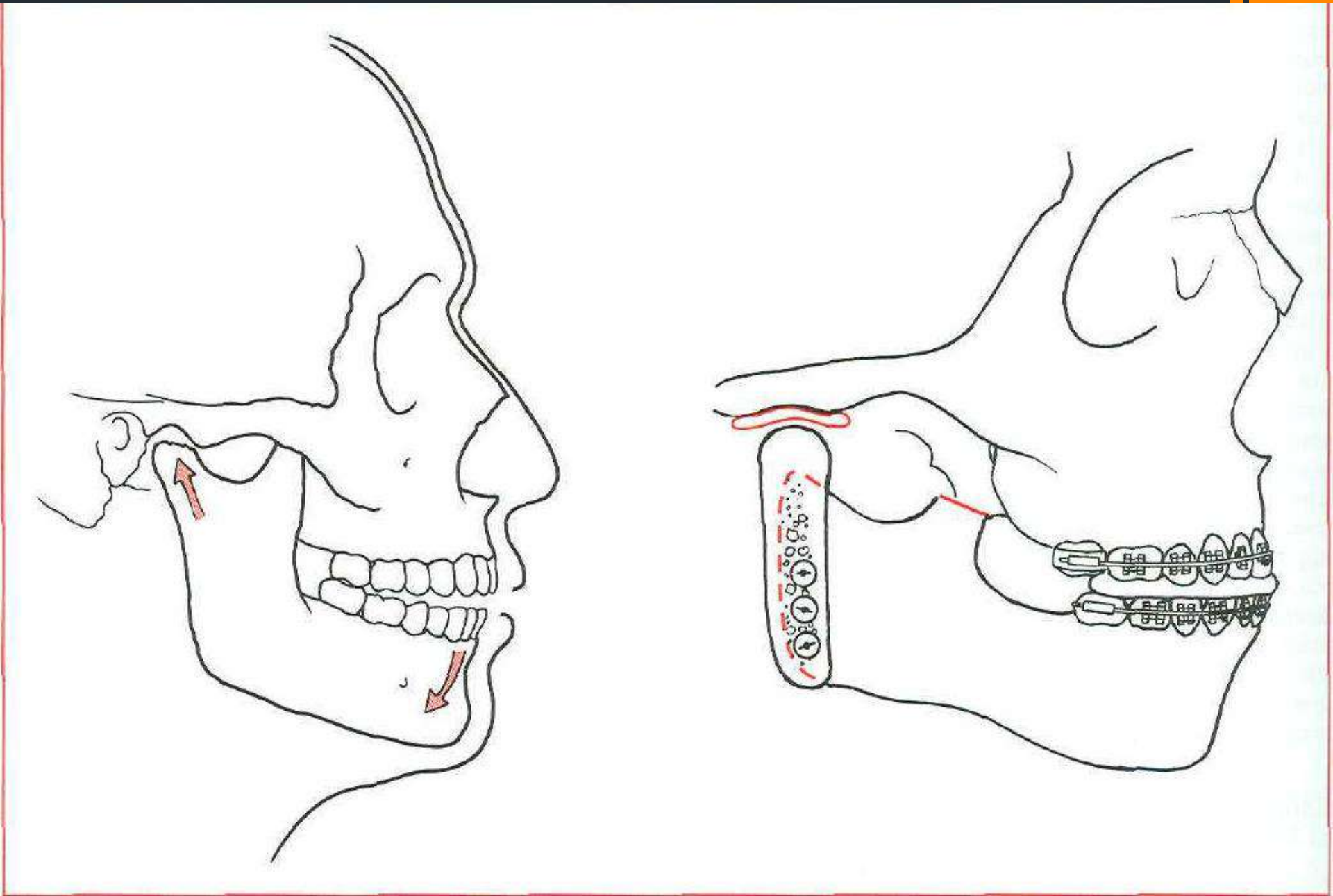

Plane of coronal section of TMJ
Superior joint space
Disk
Inferior joint space
Parotid gland
Condyle


A

Disk reposition surgery



Total joint replacement



Surgical aids to orthodontics

- Surgical interventions that may be required in orthodontic treatment include:

1. Extraction of erupted teeth,
2. Management of impacted,
3. Supernumerary and dilacerated teeth,
4. Corticotomy
5. Excision of labial frenum.

Management of Impacted teeth

- Impacted tooth is one that has failed to erupt into normal functional position beyond the time usually expected for such appearance. It occurs where there is prevention of complete eruption due to:

I. Lack of space in the dental arch (main cause).
II. Obstruction by another tooth.
III. Development in abnormal position.
IV. Dense overlying bone.
V. Thick fibrous tissue.
VI. Odontogenic cysts or tumors.

Impacted third molars

- History : Cc, HPI, medical and dental Hx
- Clinical examination:

General exam → Size of patient, attitude, General fitness is essential.

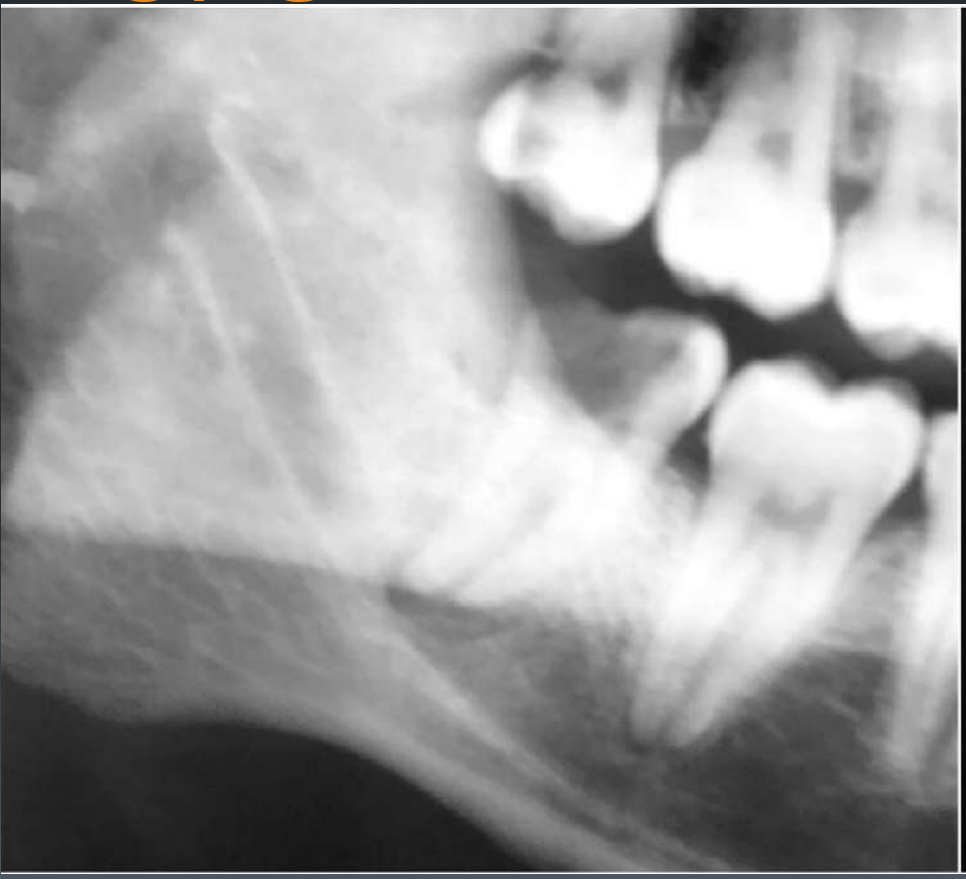
Local examination → EOE

→ IOE

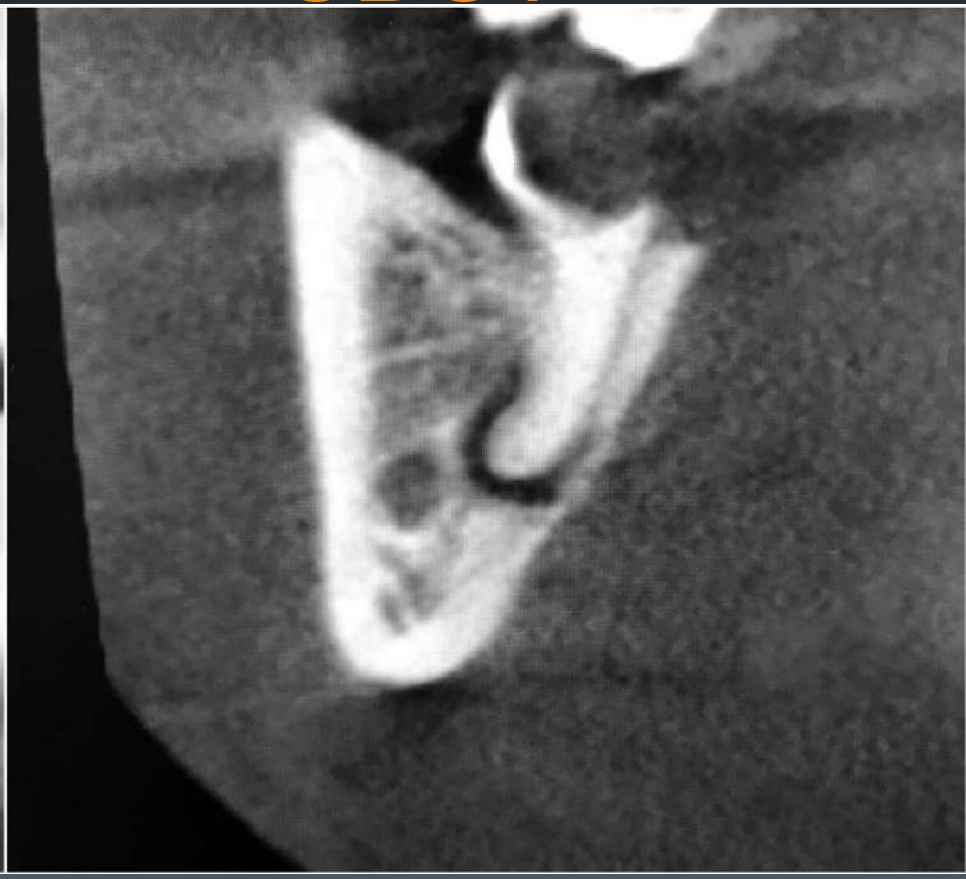
Radiographic examination

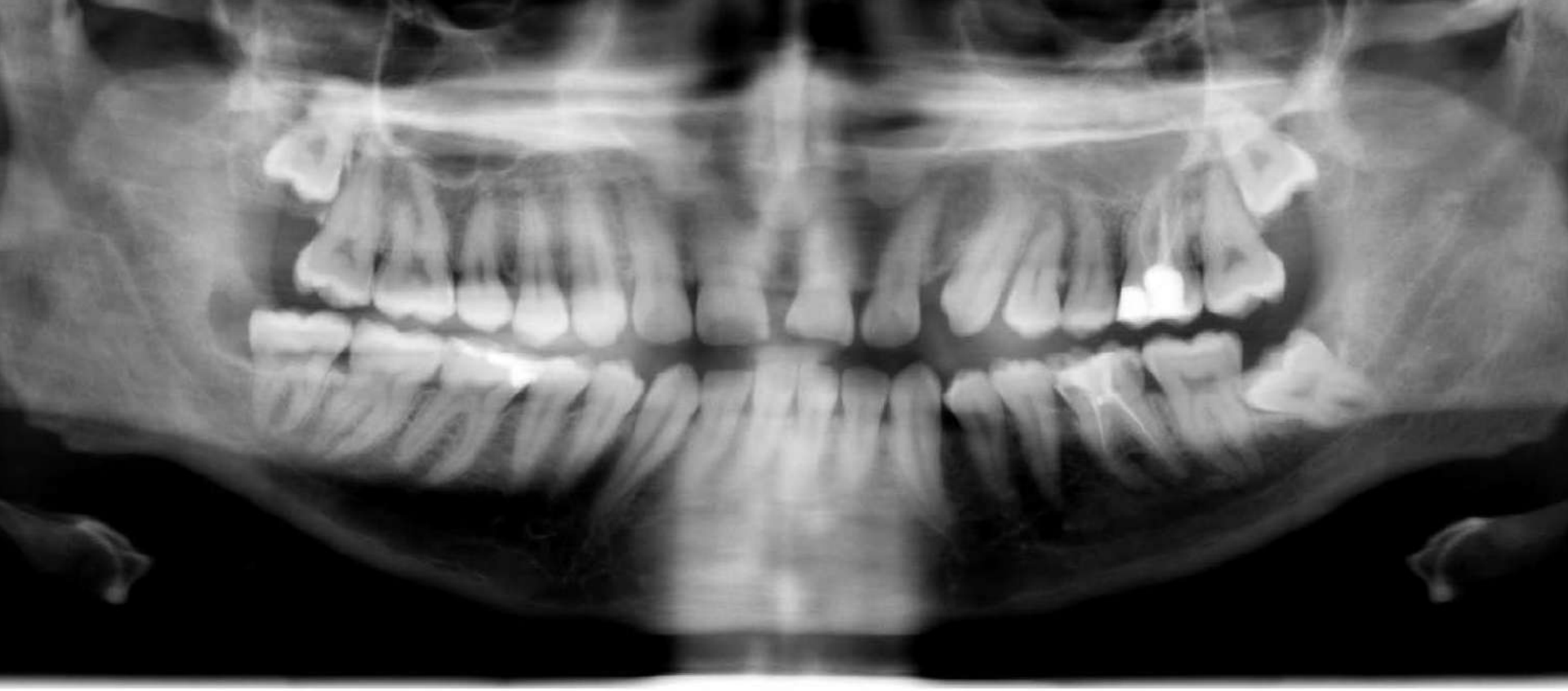
- Periapical radiograph offers the highest definition and it should be used whenever possible,
- Orthopantomogram (OPG) is regarded as the radiograph of choice,
- Occlusal films are used in difficult cases especially in unerupted teeth in conjunction with another view at right angle, this is necessary to understand the problem in 3 dimensions.
- Cone beam CT (CBCT) is indicated when on OPG there is a suggestion of a relationship between the roots of the impacted tooth and the inferior alveolar canal or the maxillary sinus or when the impacted tooth is associated with pathology.

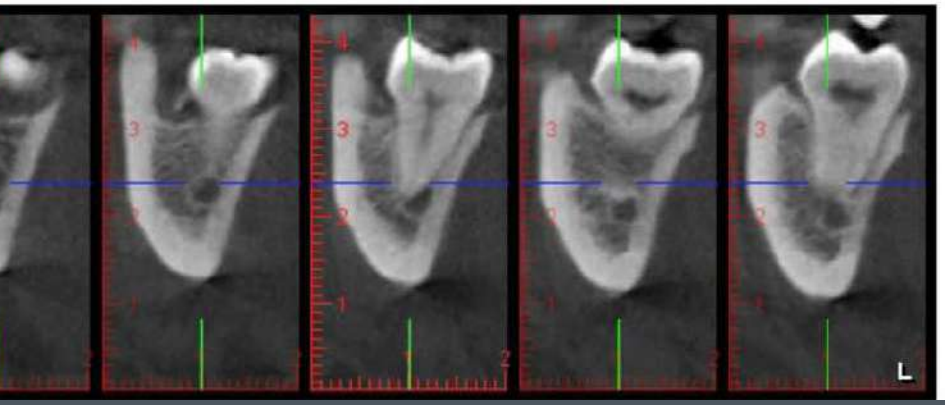
OPG

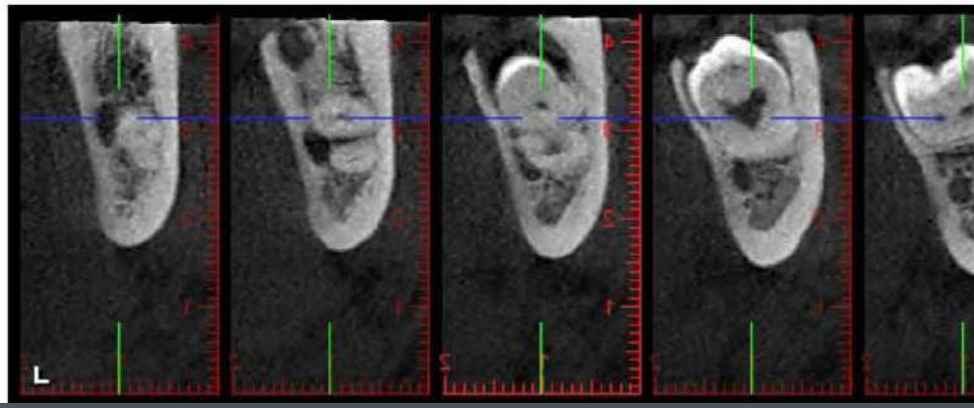


CBCT



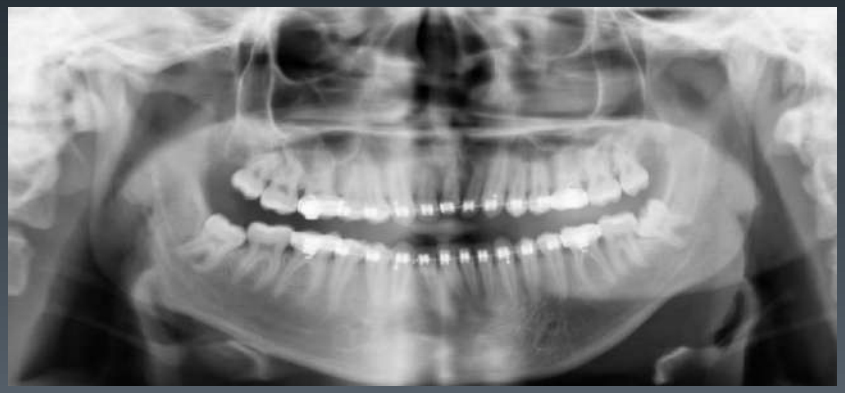



L

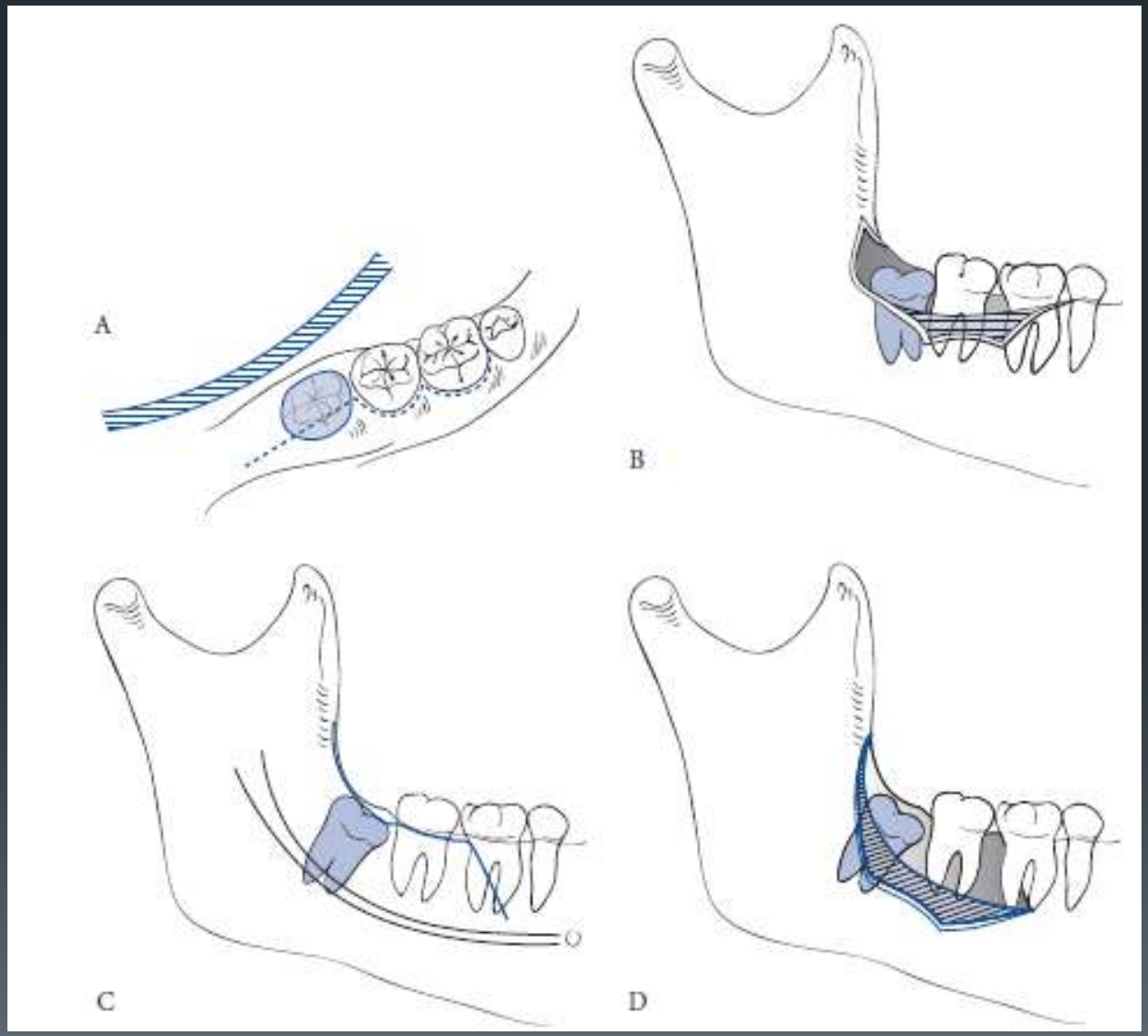

L

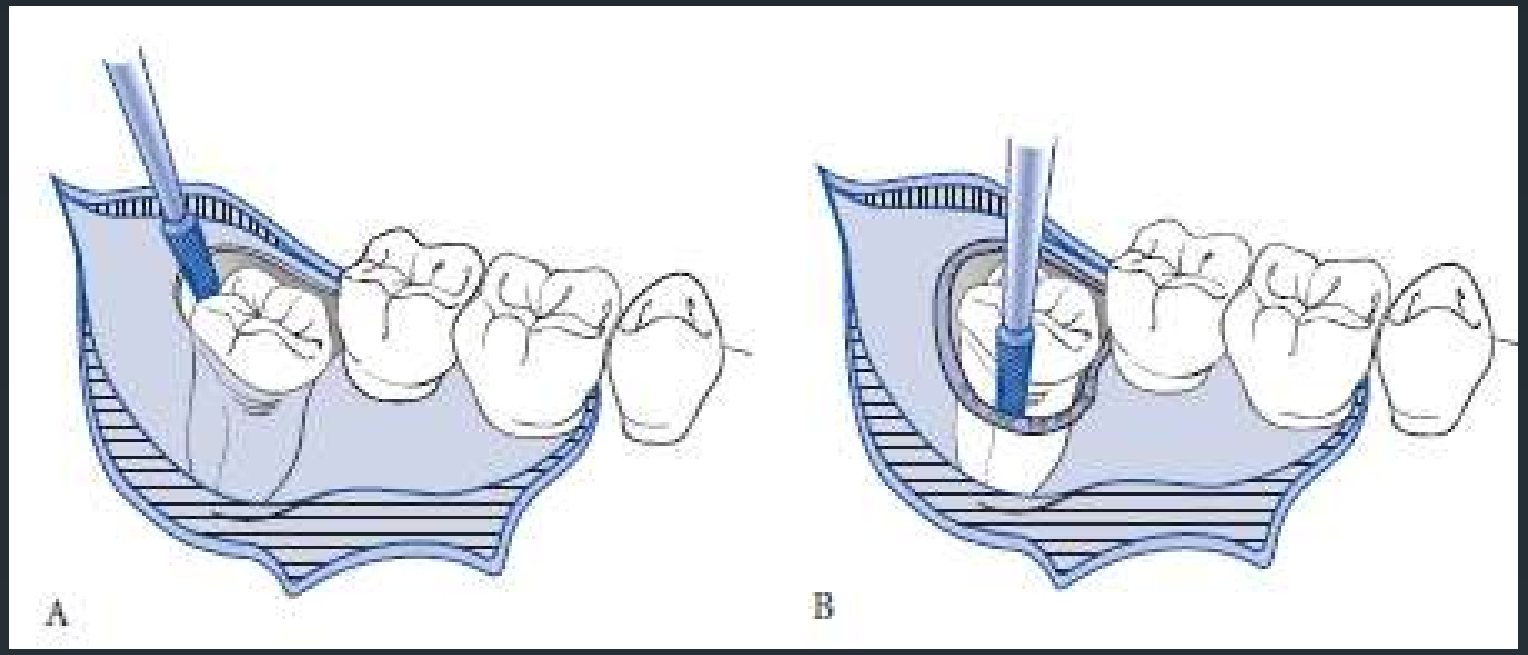
In radiographic assessment the following features should be considered

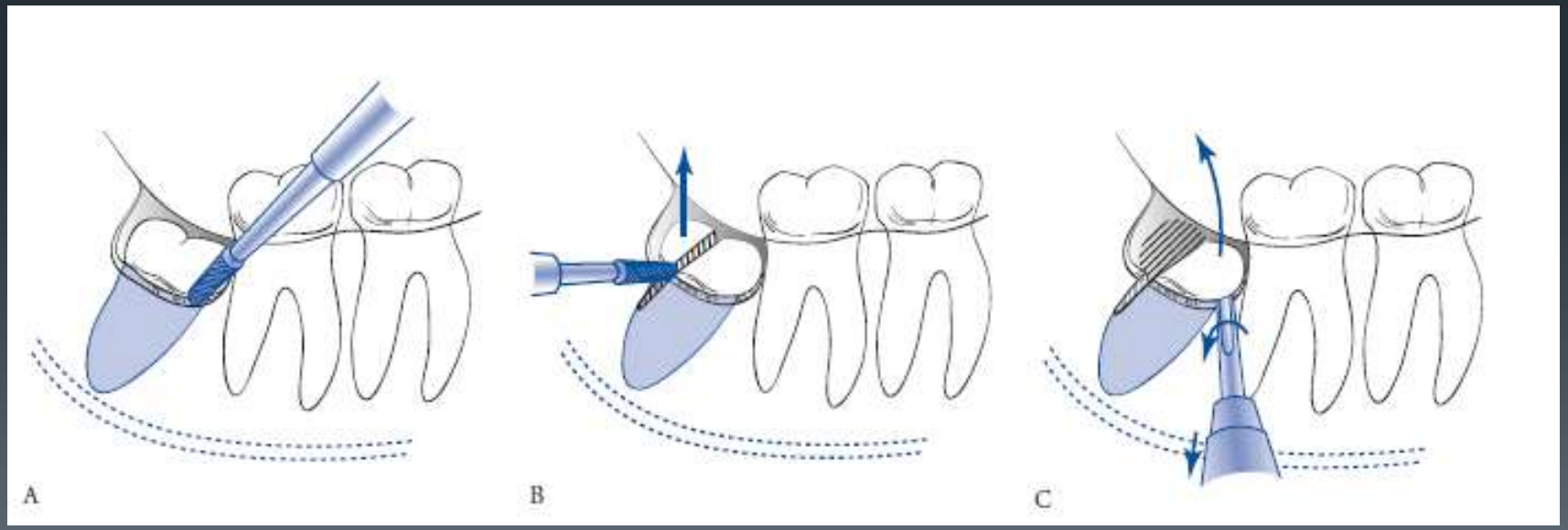
- Angulation of the impacted tooth.
- The depth of the impacted tooth.
- Crown features.
- Root morphology, e.g. fused roots, curved roots ...etc.
- Bone density.
- Any associated pathology like cysts or tumors.
- The state of the second molar (root morphology, caries, restoration or resorption ...etc.)



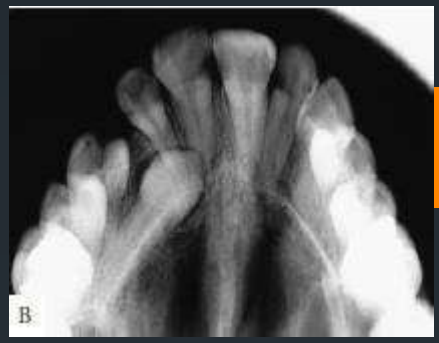
Surgical extraction of impacted third molars




A
B


A
B
C

Impacted maxillary canine

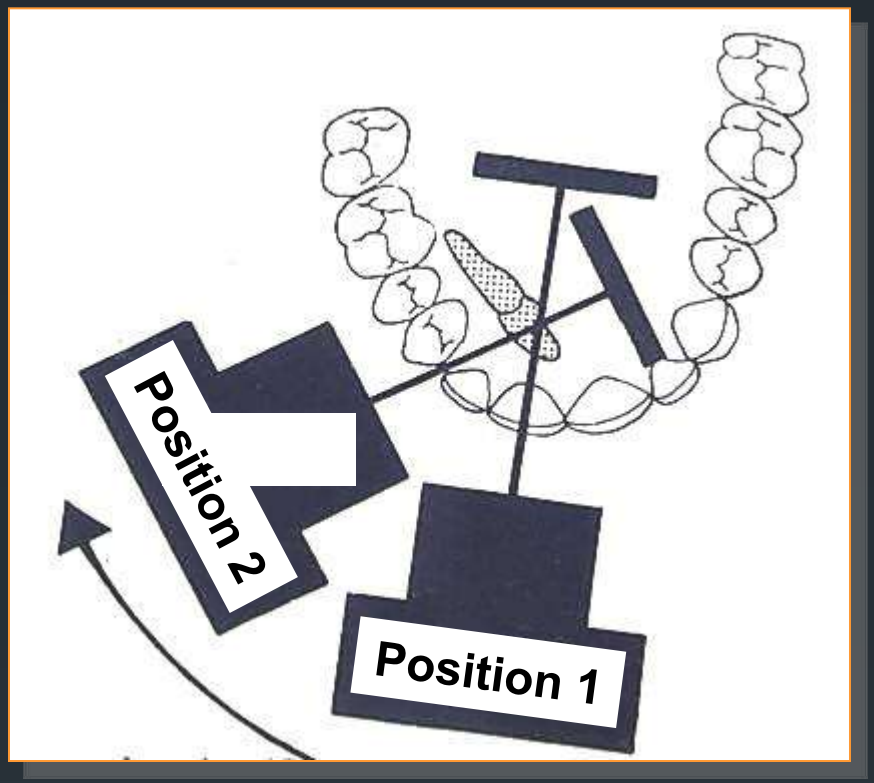


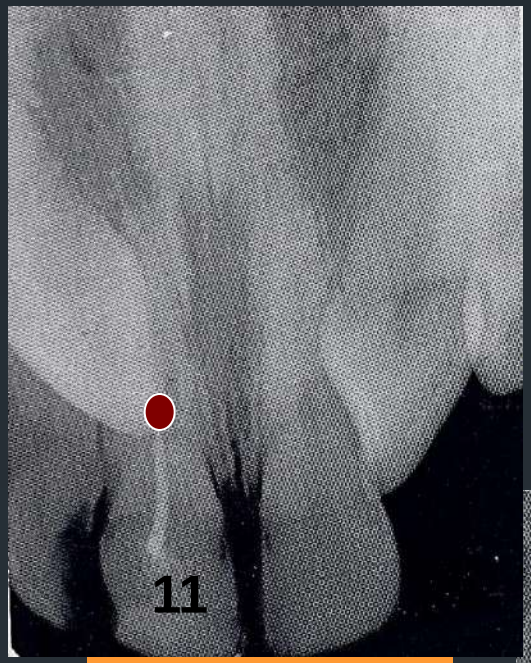
- If the maxillary canine fails to erupt by the age of 13 years, its position should be investigated, the orthodontist should be consulted to determine if the tooth can be brought to normal occlusion.
- The impacted maxillary canine can be located; palatally, buccally, within the alveolar ridge, or located labially and palatally; crown on one side and the root on the other side.
- It can also be horizontally, vertically or semivertically oriented.
- The position of the tooth may be obvious by the presence of a bulge either palatally or buccally
- The radiographic views that can be used include; periapical, occlusal, OPG, lateral skull view, and cone beam CT (CBCT)

Methods of localization include

- Buccal object rule (parallax method, tube shift technique)
- Vertex occlusal projection
- Periapical-occlusal method
- OPG
- Lateral skull view or cephalometric.
- CBCT.

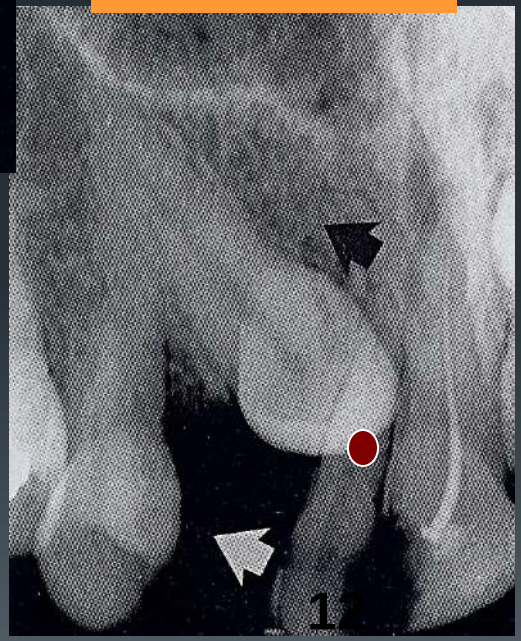
SLOB technique





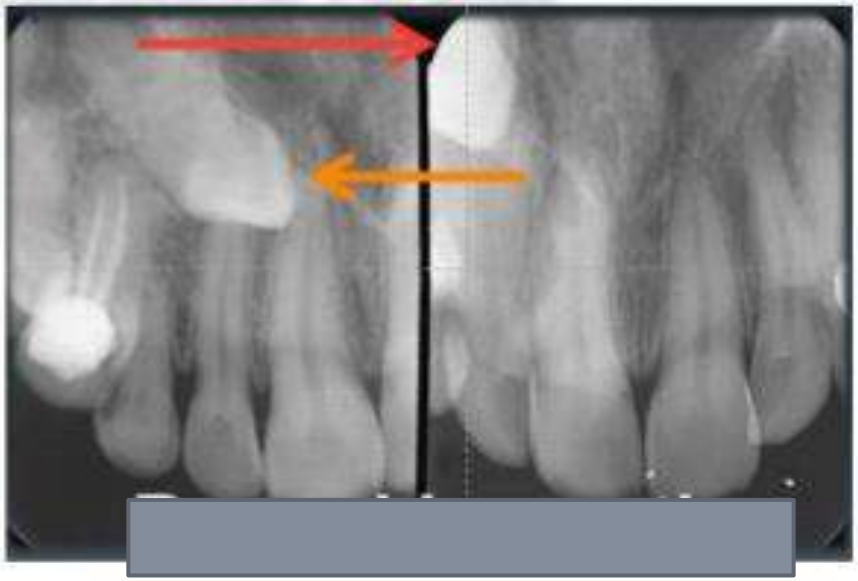
Position 1- centrally oriented

Position 2- distal shift



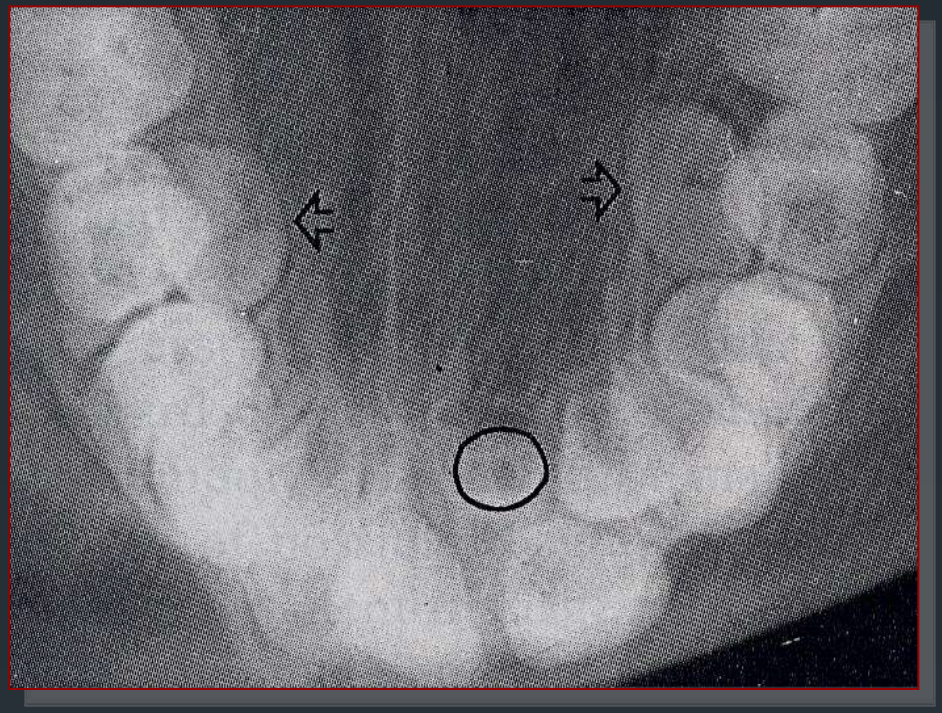
Same ⟶ Lingual (palatal side)

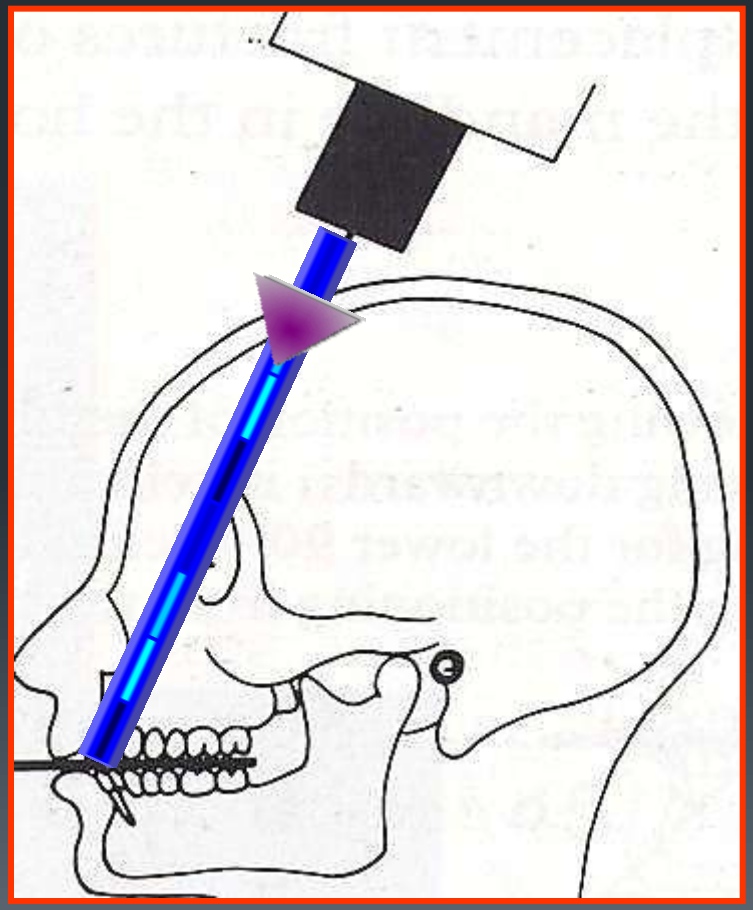
Example of Horizontal Parallax



- In the second film, when the tube was shifted MESIALLY, the crown of the impacted tooth moved DISTALLY

A vertex occlusal





Options of treatment

1- Retention or leave in situ; indicated when:

- The canine is asymptomatic and its extraction may lead to damage to the adjacent teeth.
- There is absence of any pathology like infection, abnormal widening of the follicle, resorption of the adjacent roots or any other associated pathology.
- Aesthetically acceptable

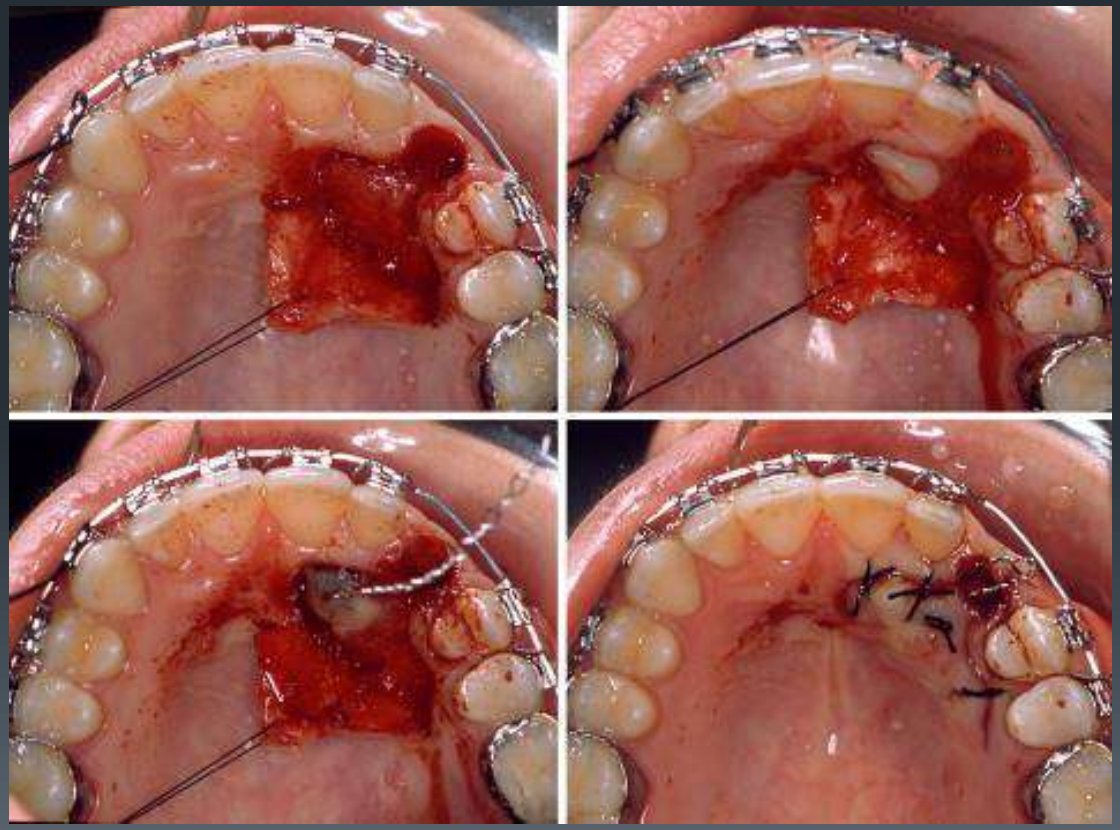
2- Surgical exposure and orthodontic traction; is the procedure that allows natural or orthodontically guided eruption of the impacted teeth.

  - There should be adequate space in the arch to accommodate the tooth.
  - There should be an unobstructed path of eruption.
  - After eruption the tooth should be in near to normal position in all planes.
- The timing of the procedure should be as close as possible to the normal eruption time

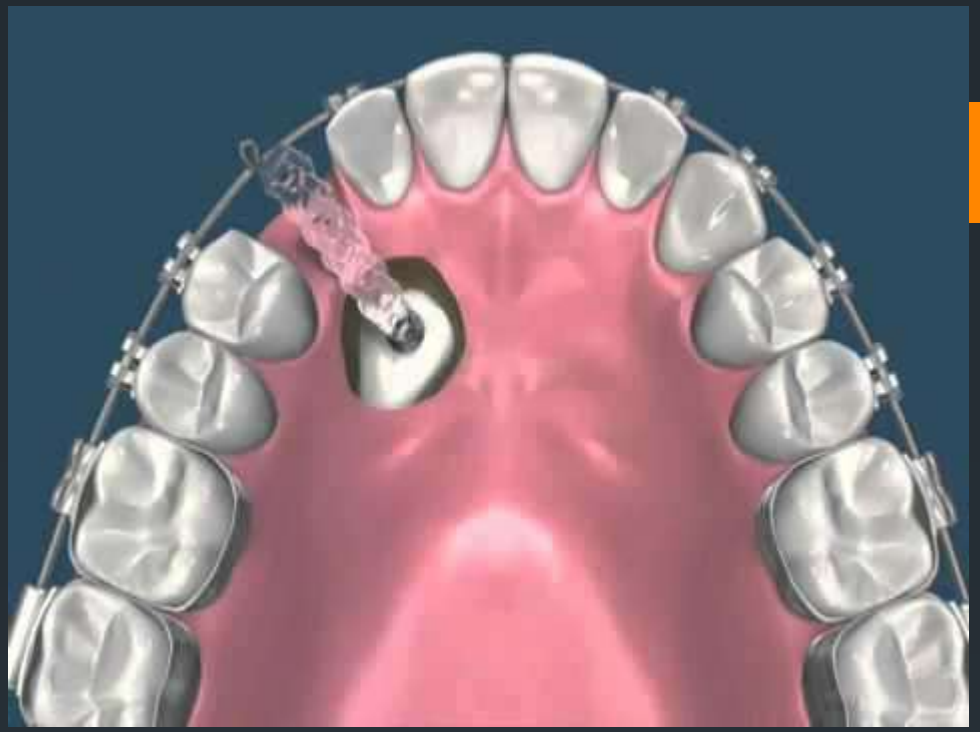
Procedure of exposure

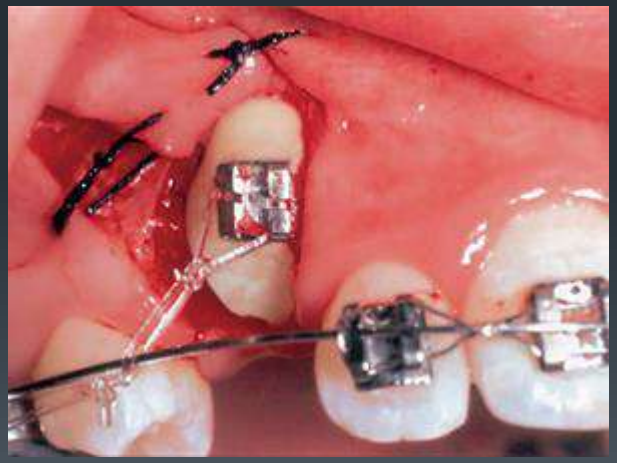
1- Approach:

- palatal envelope flap, for palatal impaction
- Buccally impacted teeth are approached through a 3-sided buccal flap, depending on its location.

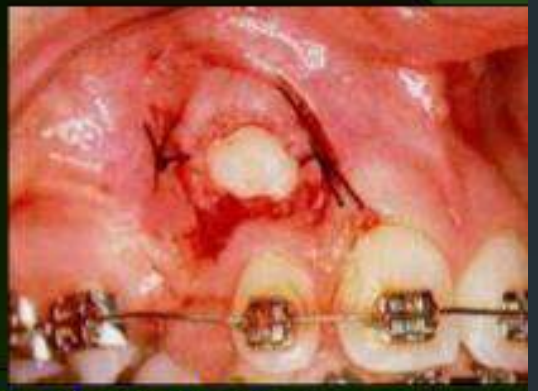



Surgery: Dr. Ioannis Vergoullis
Orthodontics: Dr. Elias Kathopoulis





APICALLY POSITIONED FLAP



- split thickness pedicle reflected from edentulous area
- bone covering the enamel is removed
- flap sutured back to periosteum, leaving 1/2 - 2/3 of the crown exposed
- surgical dressing placed for 1 week post-op to prevent tissue overgrowth
- 2 weeks post-op: bond an attachment on the tooth

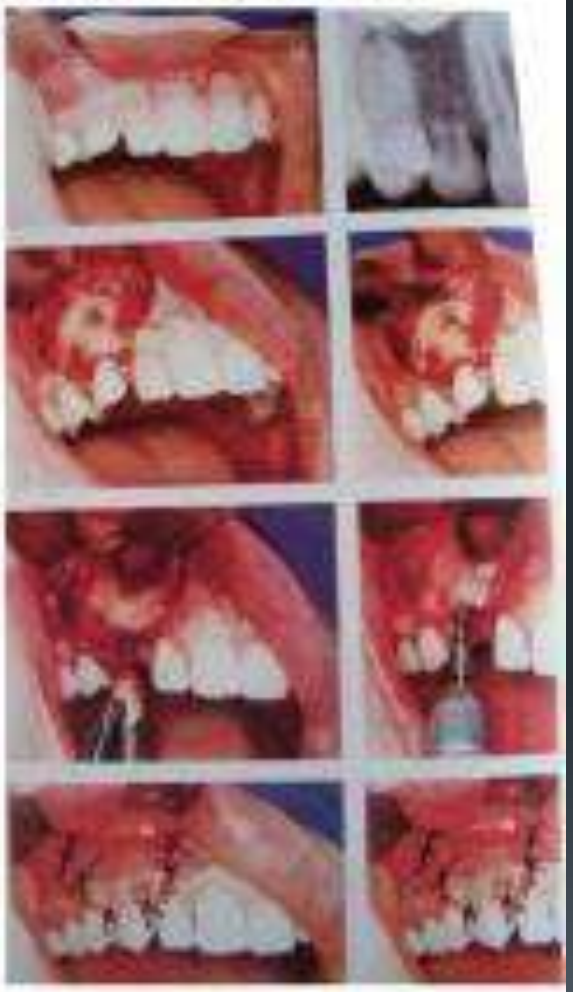
3-Transplantation; in this procedure the canine is carefully extracted and transferred to a surgically prepared socket in the dental arch with minimum delay.

- The transplanted tooth should be splinted in its new position for about a month with an orthodontic appliance.
- It is essential to have sufficient space to accommodate the crown of the canine.
- Success rate is increased by → open apex of root
- → minimum handling of root

- Endodontic treatment should be performed as soon as possible after surgery (about 6-8 weeks

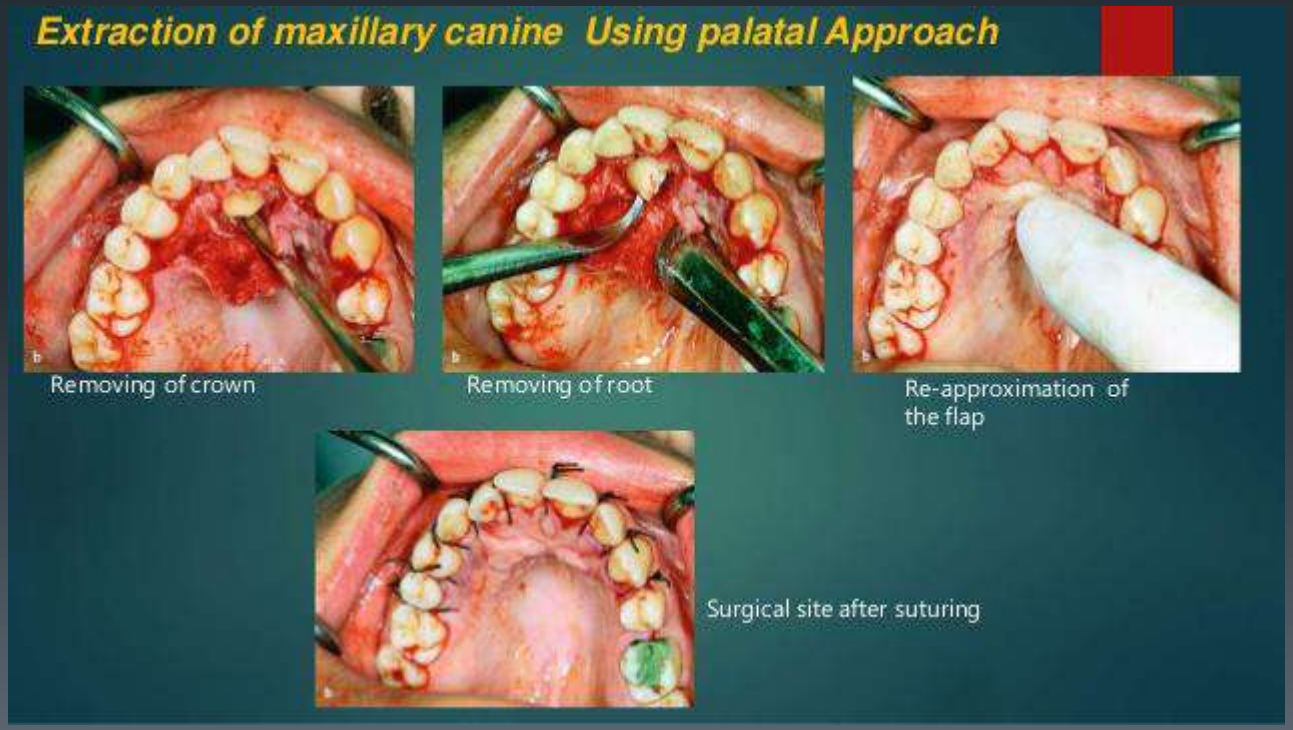
Transplantation of Labially Positioned Canine

- Transplantation of maxillary canine with labial position
- Exposure of the crown
- Preparation of a new socket
- Splinting



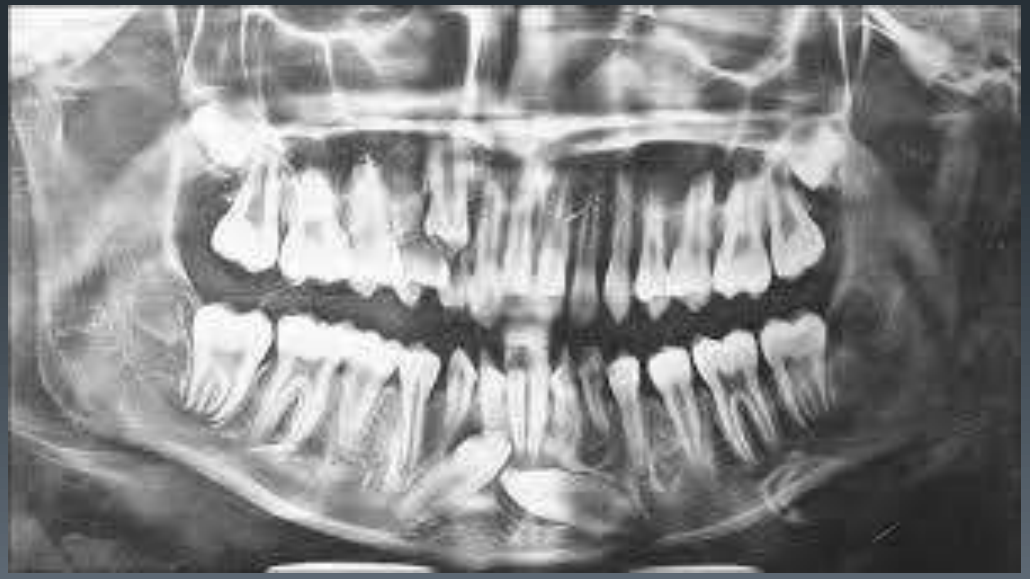
4- Removal; surgical extraction maybe performed when the other options are unavailable. Indications are

- Before construction of a dental prosthesis.
- To permit orthodontic alignment of other anterior teeth.
- When there is resorption of the roots of adjacent teeth.
- When a follicular cyst has developed.
- Infection although uncommon



Impacted mandibular canines

- mostly buccally located, partially erupted teeth can be removed easily using elevators or forceps
- Localization of the unerupted teeth is by periapical film, OPG, occlusal view or CBCT
- Deeply impacted teeth or those located lingually can be left in situ if they do not cause any damage to the adjacent roots or not associated with other pathologies
- Orthodontic traction is difficult



Impacted lower premolars

- It occurs mostly due to loss of space by drifting forward of the first permanent molar after early extraction of the second deciduous molar
- Same management protocol for impacted canine

raising a buccal flap with preservation of the mental nerve

↓

bone removal

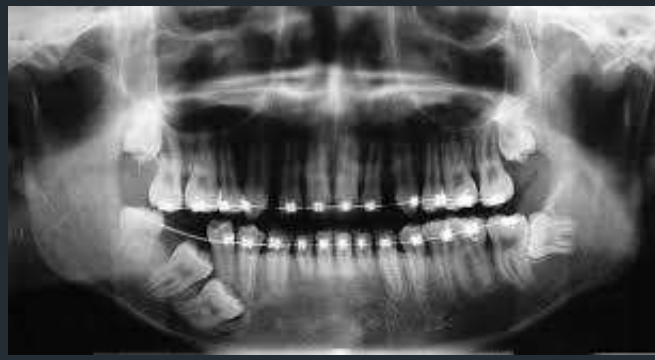
↓

sectioning of the tooth if needed and extraction of the tooth.

↓

Consultation with orthodontist before extraction

Impacted first and second molars



- Treatment either

1. surgical extraction through a buccal flap or
2. surgical uprighting, especially of the impacted second molar

- Done through buccal approach, the tooth is exposed carefully without exposing the CEJ,
  - if the third molar is present it needs to be removed, if not,
  - bone posterior to the second molar is removed, followed by
- tipping the tooth slightly posteriorly and superiorly, and the tooth can be allowed to erupt spontaneously.

Supernumerary teeth

- Supernumerary teeth can be classified according to their position into:

1. **Mesiodens**; is situated in the premaxilla in the midline and it is commonly conical, it can have a horizontal or inverted position. Supplemental teeth may also occur in the anterior maxillary region.
2. **Paramolar**; appear in the premolar or molar region and is situated buccally to the teeth, they can be conical or supplemental.
3. **Distomolar**; appear as a fourth molar usually distal to the standing molars and they are either normal or smaller in size

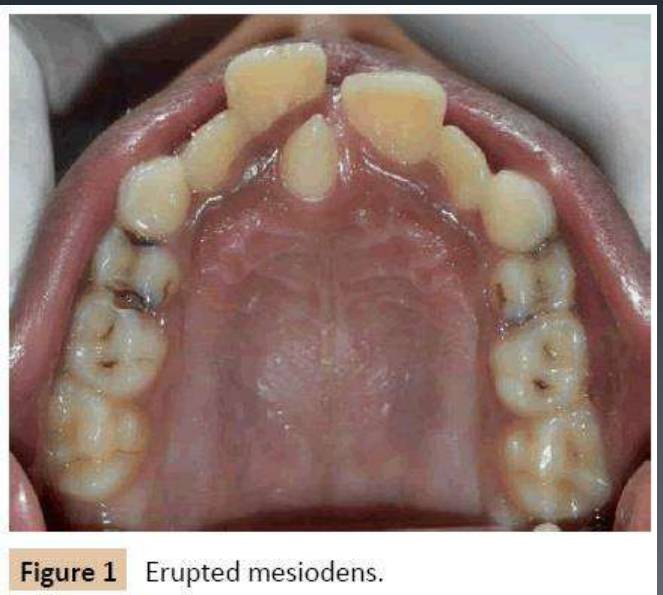
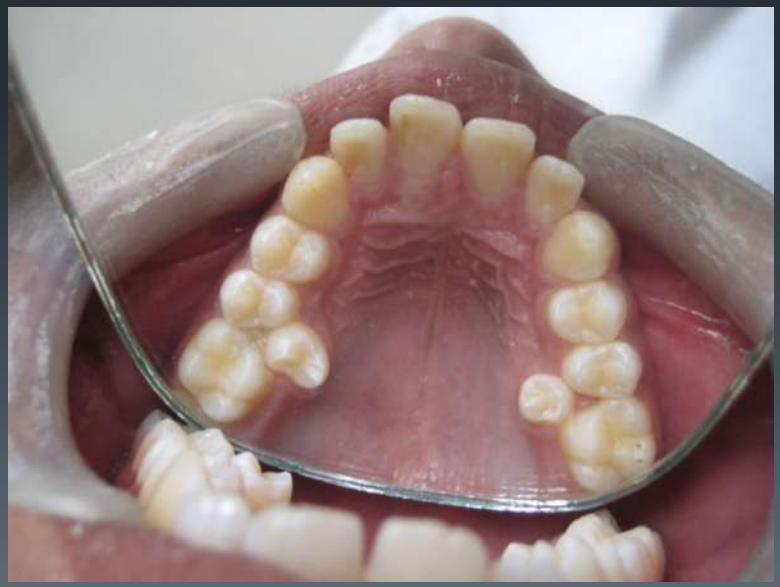
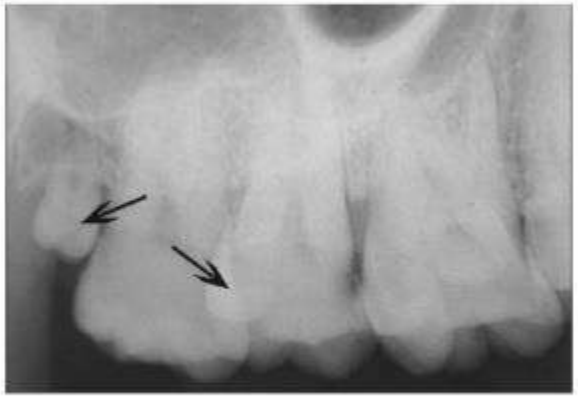


Figure 1 Erupted mesiodens.



Distomolar/Distodens

- molar located distal to molar



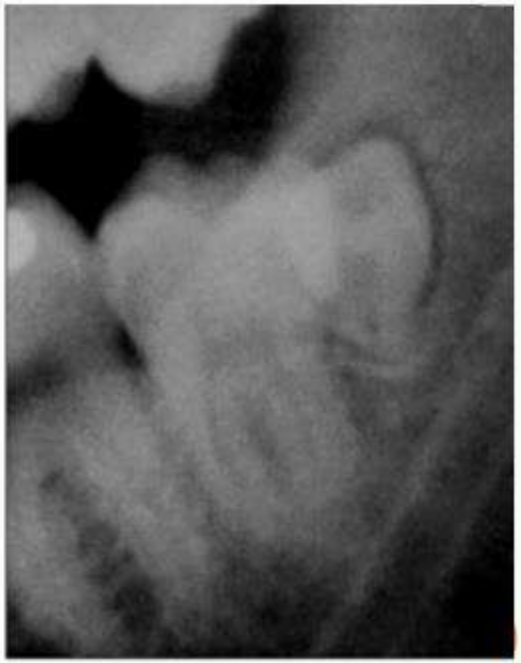
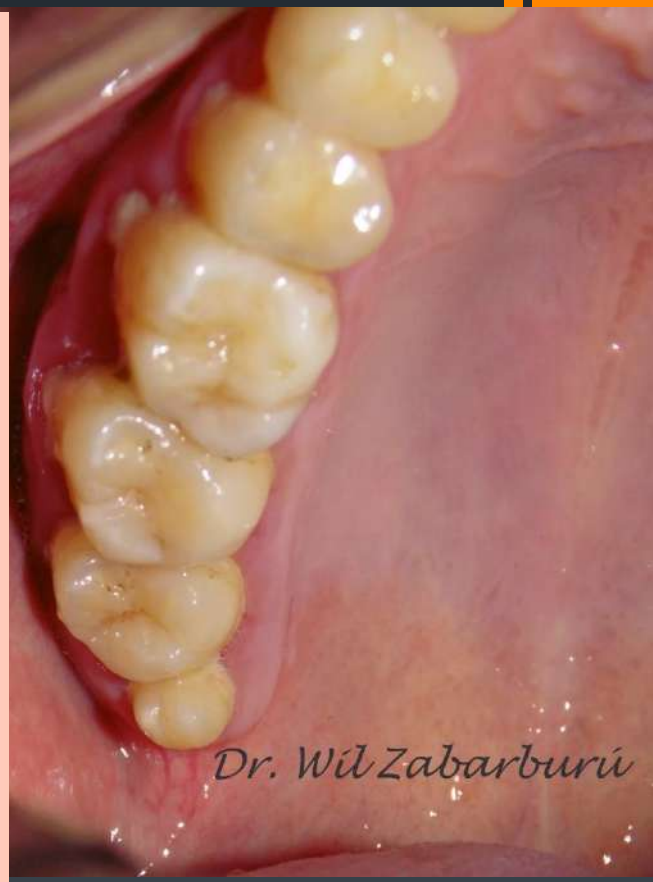


Fig. 5. Distomolar 3.9.



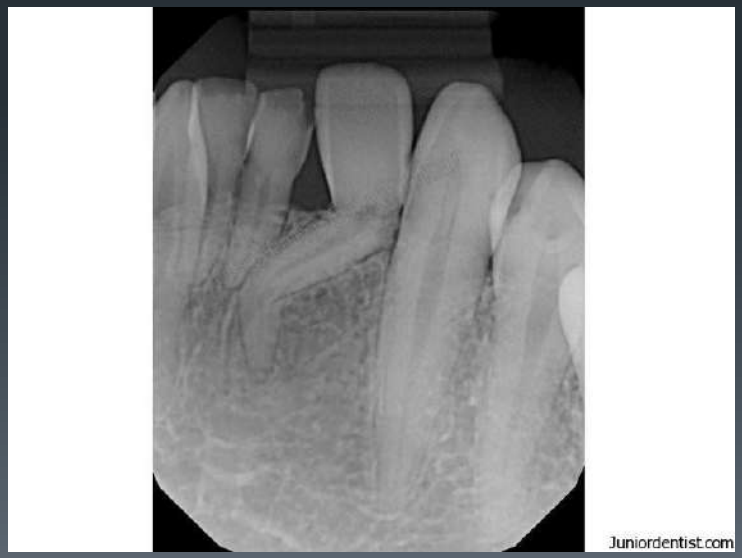
Management of supernumerary teeth

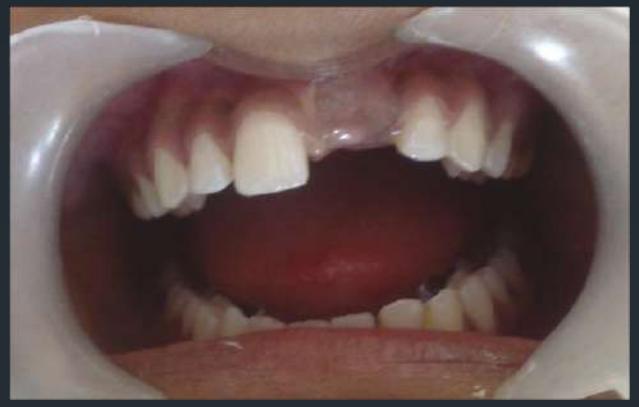
- Supernumerary teeth can have no effect on other standing teeth or they can cause failure of eruption of the other teeth, crowding, malposition or misalignment, resorption of the roots of the adjacent teeth or they can be associated with other pathologies (e.g. cysts).

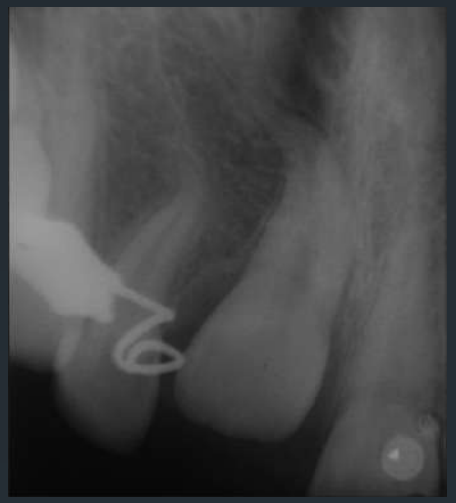
- If they need extraction, they should be localized accurately by periapical films (buccal object rule may be applied), vertex occlusal view or CBCT.
- Buccal, palatal or combined approach may be used

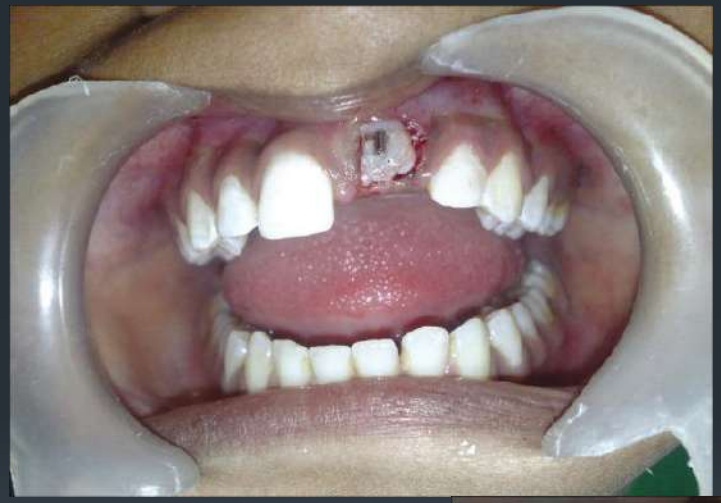
Dilacerated incisors

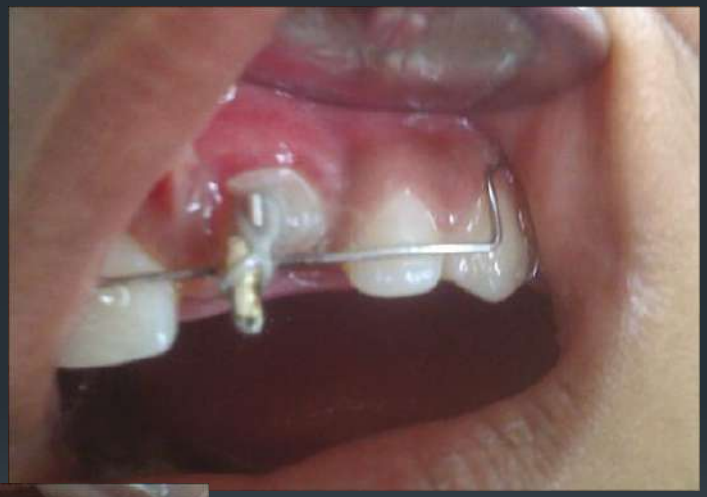
- Trauma to the deciduous incisors especially in the 2-3 years of age can cause damage to the underlying permanent incisor tooth germ causing root development to take place at an angle.
- Exposure and orthodontic traction can be performed if possible, but if not, these teeth should be removed and the lateral incisors allowed filling their space.

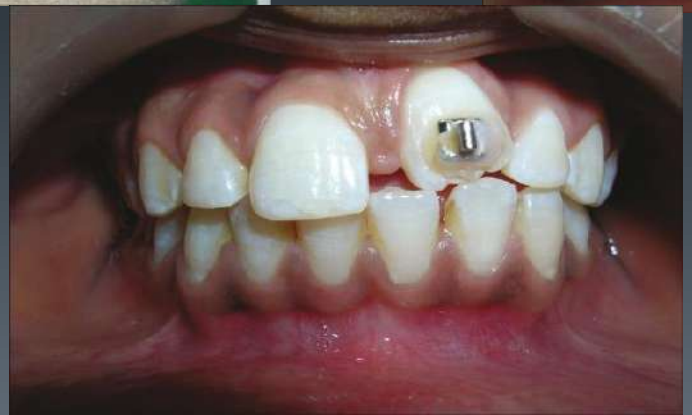












Corticotomy

- It can be defined as a linear cutting technique in the cortical plates (only) surrounding the teeth to produce accelerated tooth movement.
- Differ from osteotomy
- The biological mechanism behind the acceleratory effect of corticotomies**:**

regional acceleratory phenomenon in which an injury to bone results in the acceleration of all processes involved in healing which allows teeth roots to move rapidly through the alveolar bone

Indications

- ✓Treatment of crowding.
- ✓Canine Retraction after Premolar Extraction.
- ✓Enhance post orthodontic stability.
- ✓Facilitate eruption of impacted teeth.
- ✓Facilitate orthodontic expansion.
- ✓Molar intrusion and open bite correction

- ❖Corticotomy is contraindicated in patients with active periodontal disease or gingival recession

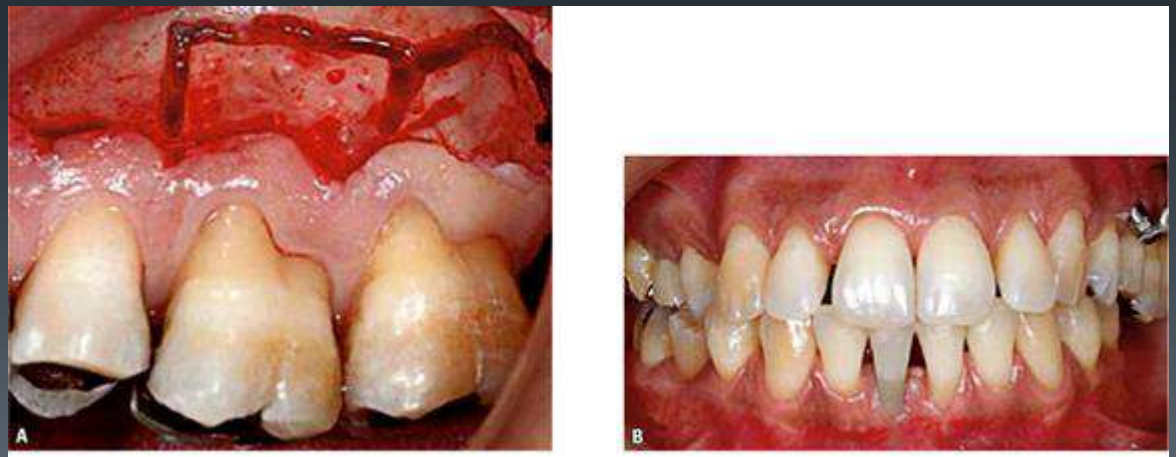
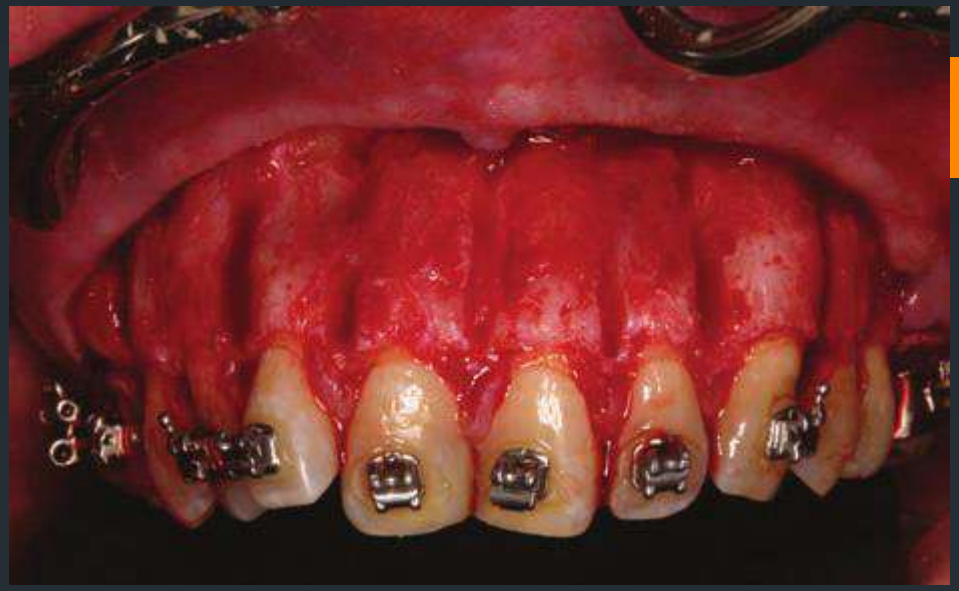


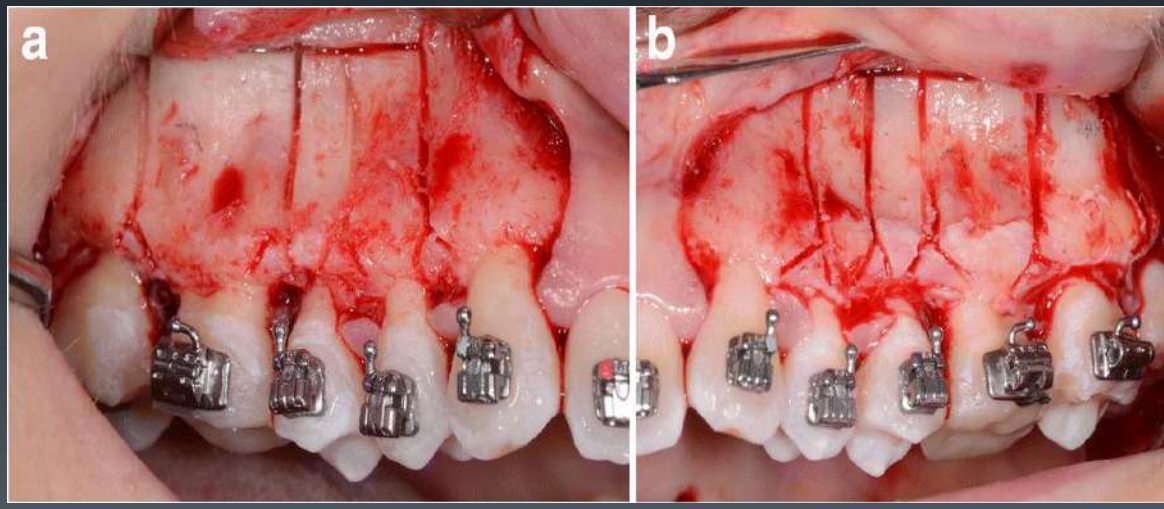
FIGURE 3 - Transoperative photographs. **A)** Corticotomies circumscribing the roots of the teeth to be intruded. **B)** Buccal mini-implants to support the cast metal bars.

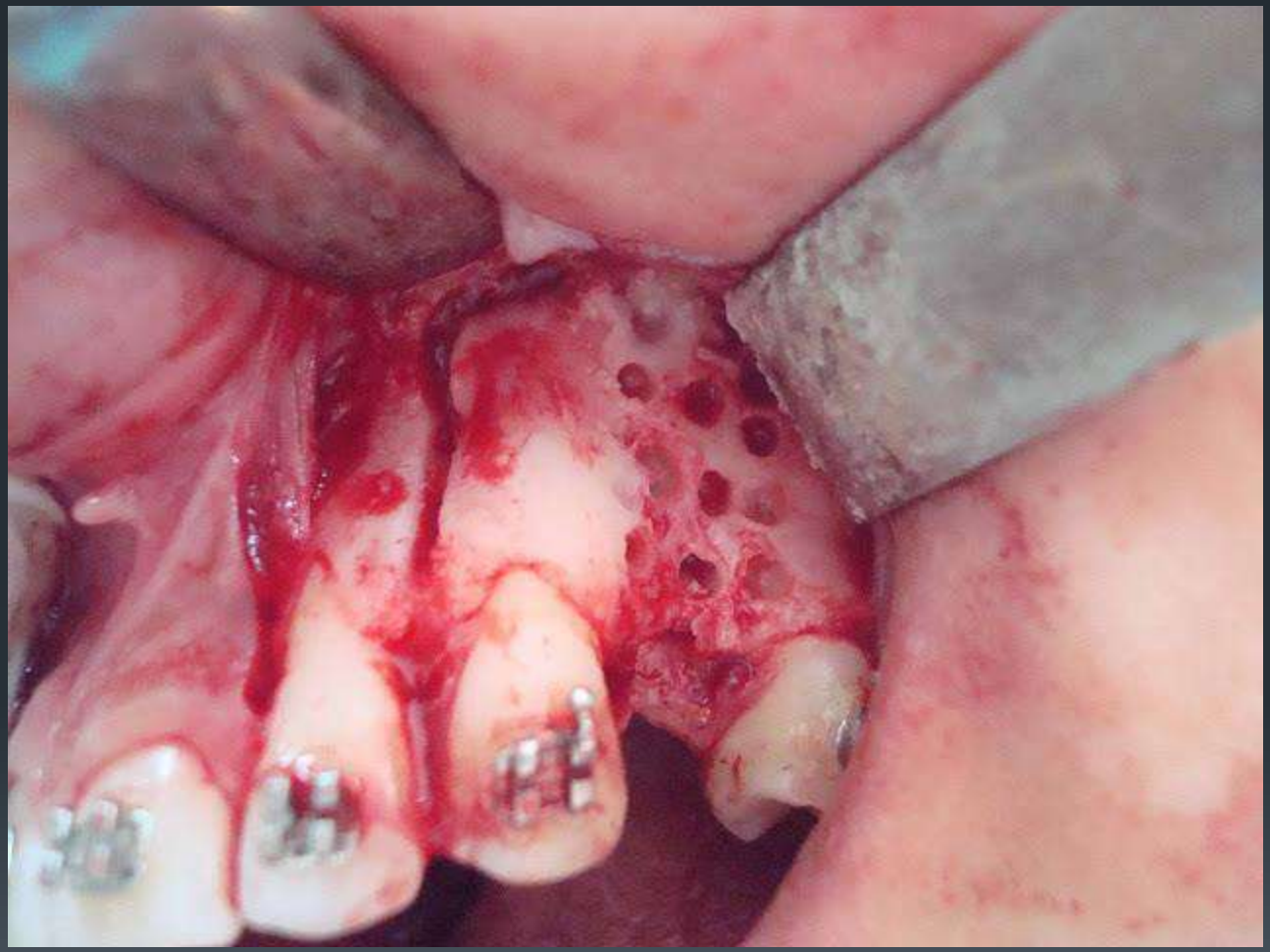
Surgical procedure



The traditional corticotomy procedure :

- raising full-thickness buccal or buccal and palatal/lingual mucoperiosteal flaps and
- performing vertical linear interradicular corticotomy cuts (about 0.5 mm in depth), with or without joining horizontal subapical corticotomy cuts, or
- by drilling multiple holes that penetrated the cortical plate instead of linear cuts





- making vertical interproximal soft tissue incisions without raising soft tissue flaps and making corticotomy cuts through these incisions using piezosurgical instrument or a hard tissue laser

Labial frenectomy

- labial frenum inhibits orthodontic closure necessitating surgical removal before the end of orthodontic therapy although surgical removal of the frenum before orthodontic treatment is reported to lead to a more rapid crown approximation
- Various surgical techniques have been proposed:

I. The simple excision technique.
II. The Z-plasty technique.
III. Localized vestibuloplasty with secondary epithelialization.
IV. The laser-assisted frenectomy

The simple excision technique

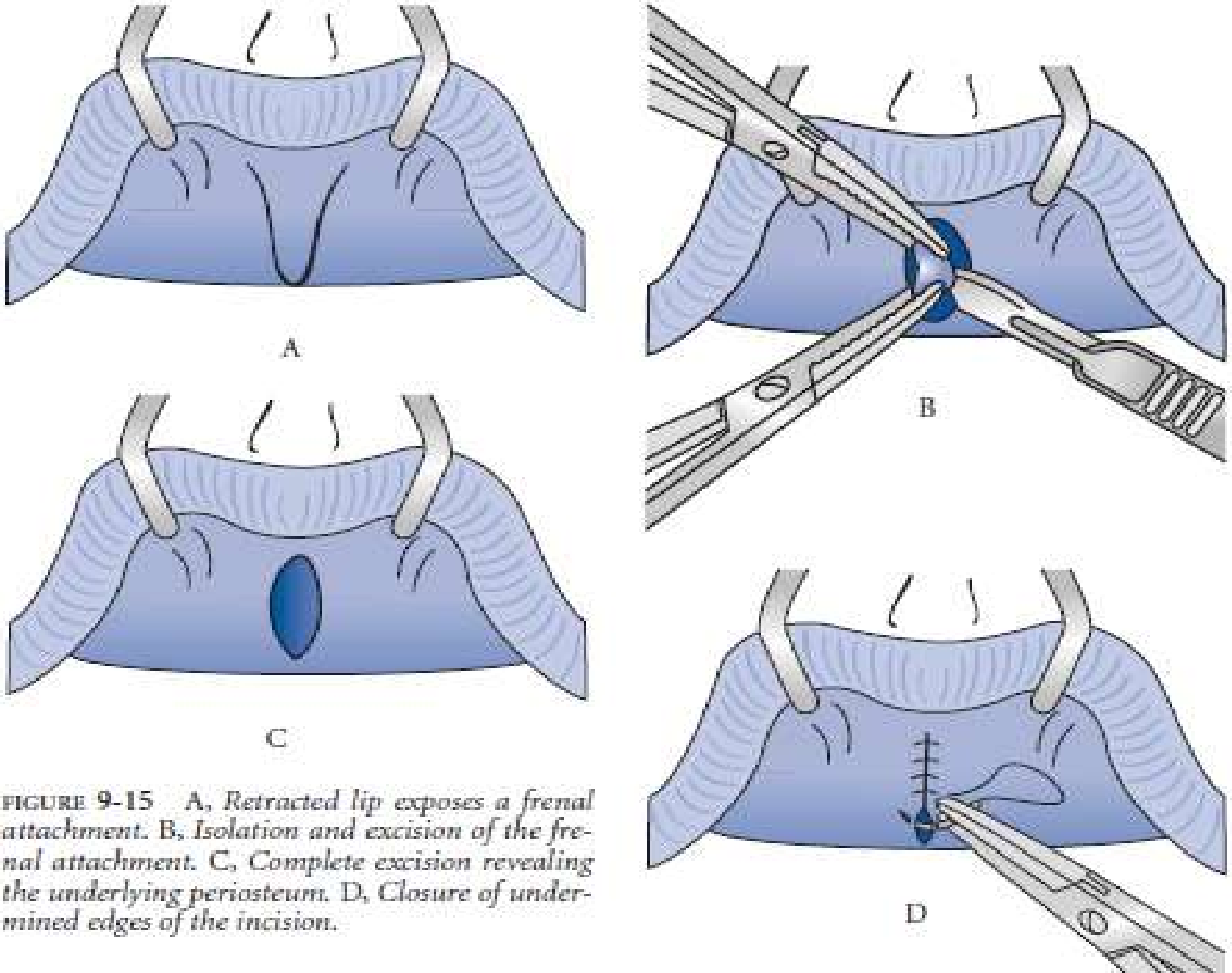
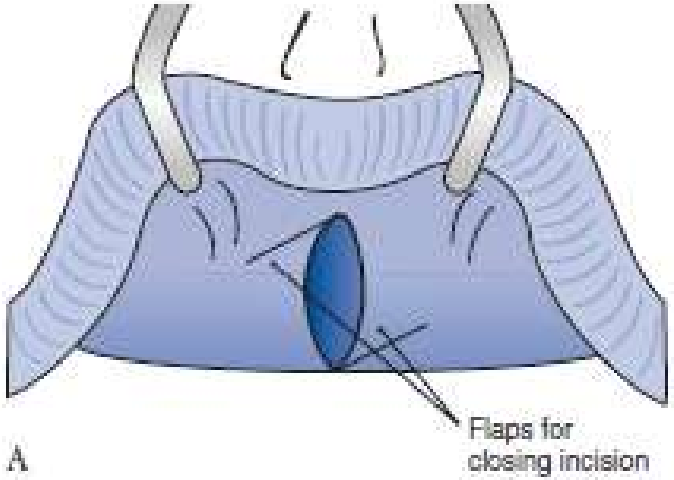
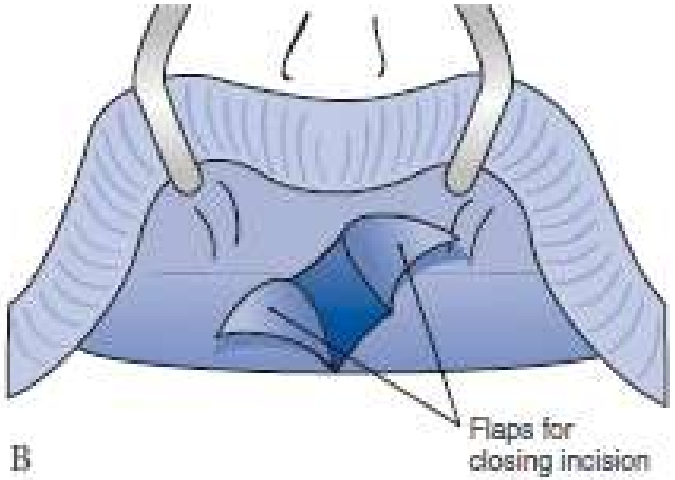


FIGURE 9-15 A, *Retracted lip exposes a frenal attachment.* B, *Isolation and excision of the frenal attachment.* C, *Complete excision revealing the underlying periosteum.* D, *Closure of undermined edges of the incision.*

Z-plasty





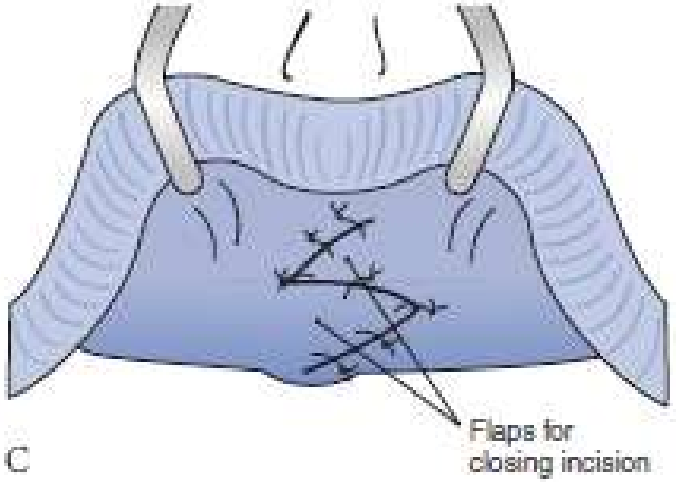
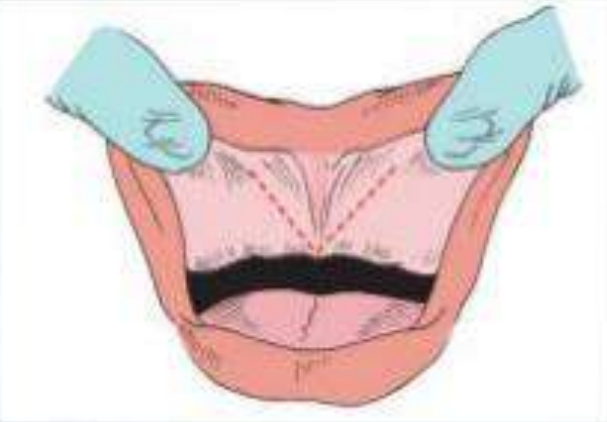
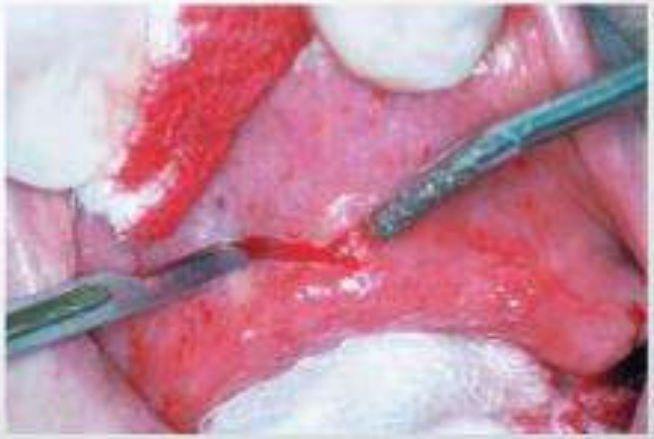


FIGURE 9-16 A, *Excision of a frenum with proposed Z-plasty incisions.* B, *Undermined flaps of the Z-plasty.* C, *Transposed flaps lengthening the incision and lip attachment.*

3. localized vestibuloplasty with secondary epithelialization





Wide V-type of incision made at most inferior portion of frenal attachments in area

Odontogenic tumors

By

Dr. Sabah Alheeti

Odontogenic tumors

- Comprises a complex group of lesions arise from odontogenic tissue.
- Many of these lesions are true tumors, whereas some are hamartomas (malformation).
- Clinically, they are typically asymptomatic, although they may cause jaw expansion, movement of teeth, root resorption, and bone loss.

- Knowledge of age, location, and radiographic appearance of the tumors can be extremely valuable in developing a clinical differential diagnosis

Classification of Odontogenic Tumors

I. Tumors of odontogenic epithelium
   A. Ameloblastoma
      1. Malignant ameloblastoma
      2. Ameloblastic carcinoma
   B. Clear cell odontogenic carcinoma
   C. Adenomatoid odontogenic tumor
   D. Calcifying epithelial odontogenic tumor
   E. Squamous odontogenic tumor

II. Mixed odontogenic tumors
   A. Ameloblastic fibroma
   B. Ameloblastic fibro-odontoma
   C. Ameloblastic fibrosarcoma
   D. Odontoameloblastoma
   E. Compound odontoma
   F. Complex odontoma

III. Tumors of odontogenic ectomesenchyme
   A. Odontogenic fibroma
   B. Granular cell odontogenic tumor
   C. Odontogenic myxoma
   D. Cementoblastoma

Tumors from odontogenic epithelium

Ameloblastoma

- The ameloblastoma is the most common clinically significant and potentially lethal odontogenic tumor
- These tumors may arise from rests of the dental lamina
- occurs in three different variants, each with specific implications for treatment and a unique prognosis

I. Solid or multicystic
II. Unicystic
III. Peripheral

Clinical Features of Multicystic Ameloblastoma

- **Age:** 4th- 5th decade.
- **Sex:** male = female.
-
- **Site:** ↑mand. Molar area.
- **Signs & symptoms:**

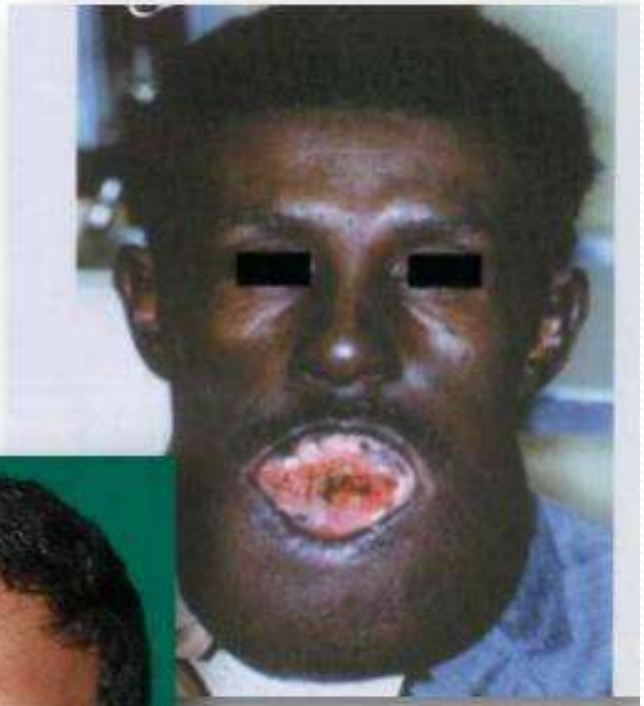
-painless

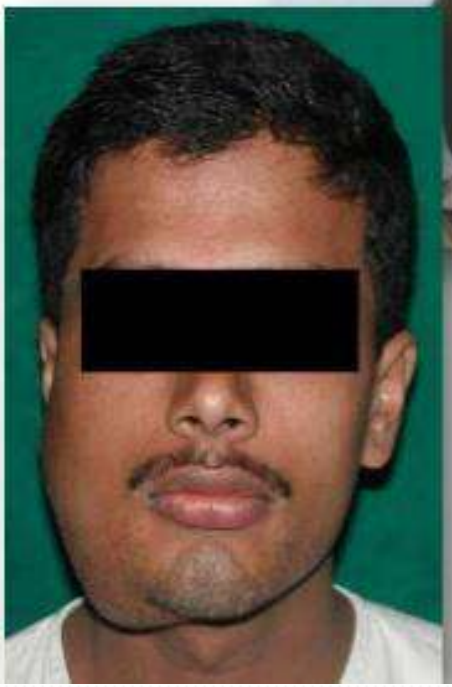
-Slowly growing.

-gradula facial asymmetry.

-Looseness of teeth.

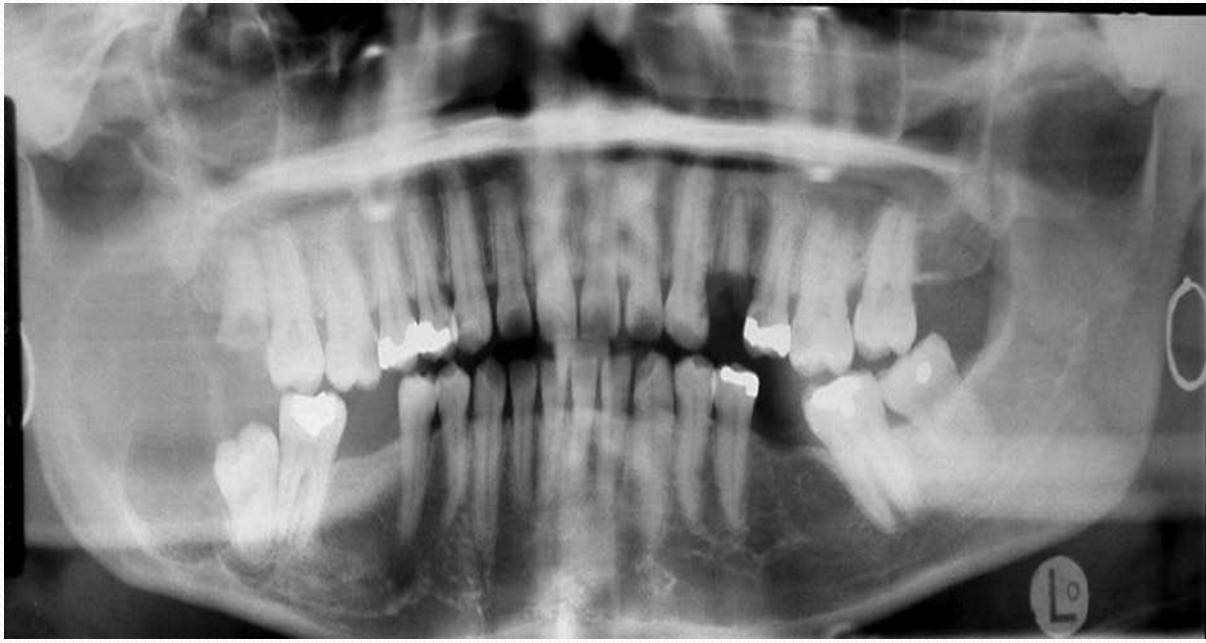
-NO metastasis (benign).





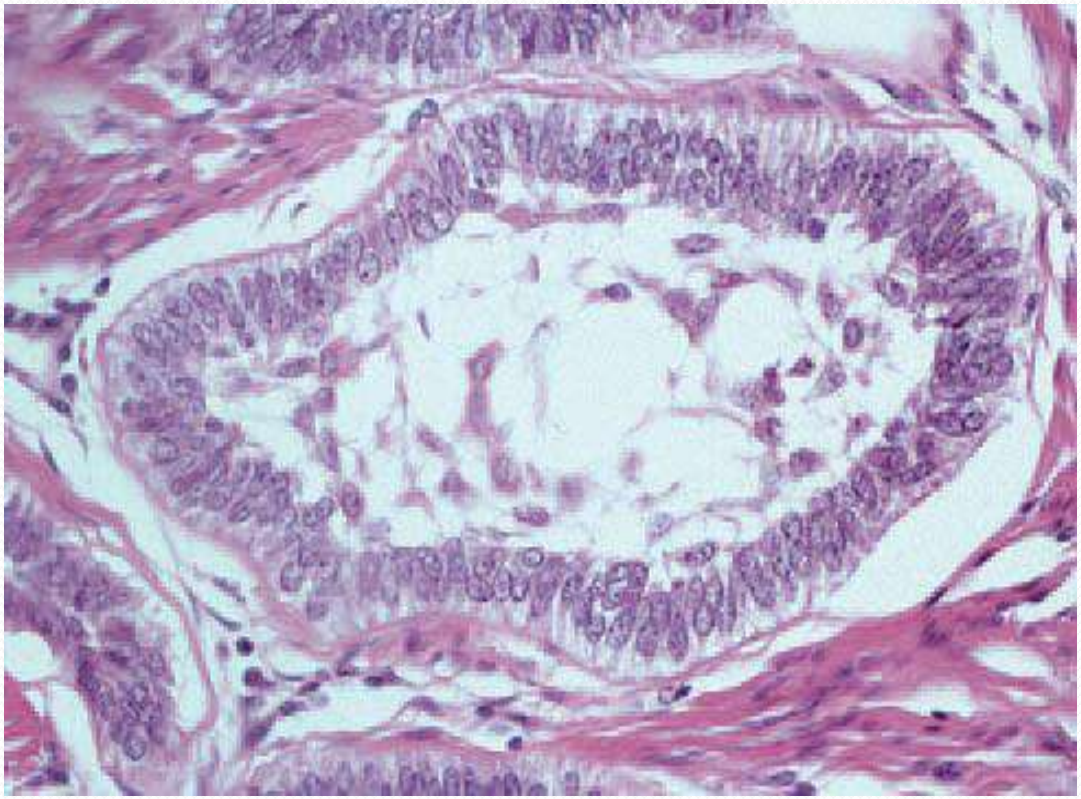
Radiographically

- Multilocular radiolucency.
- Buccal and lingual cortical expansion is common, frequently to the point of perforation.
- Resorption of adjacent tooth roots is common.



Histologic patterns

I. Follicular
II. *Plexiform*
III. *Acanthomatous*
IV. *granular cell*
V. *Desmoplastic*
VI. *Basal cell*



Treatment and Prognosis

- Resection with 1 cm safe margin is the recommended treatment , why?
- The solid or multicystic ameloblastoma tends to infiltrate between intact cancellous bone trabeculae at the periphery of the tumor before bone resorption becomes radiographically evident.
- Therefore, the actual margin of the tumor often extends beyond its apparent radiographic or clinical margin.

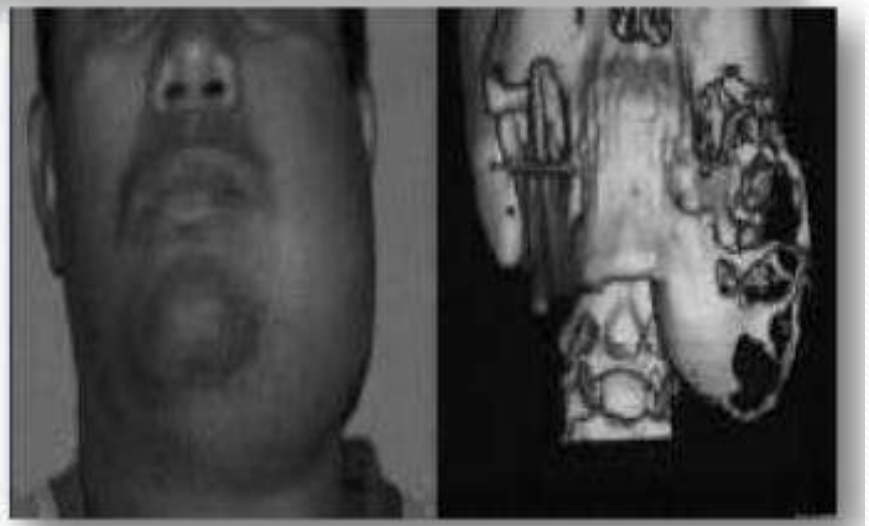
Unicystic Ameloblastoma

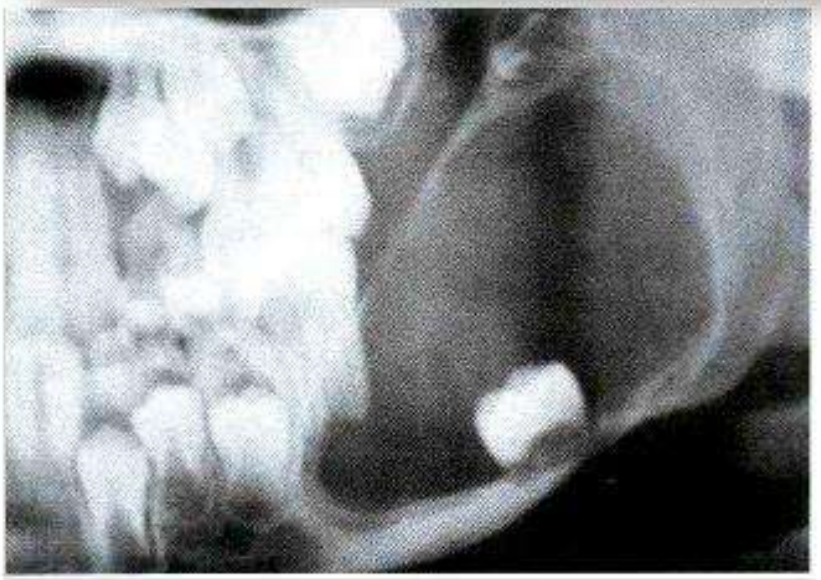
- **Def.**: Locally invasive tumor consists of a central large cystic cavity.
- **Age:** ? 20 years.
- **Sex:** male= female.
- **Site:** r **90% cases in mandible**
- **Signs & symptoms:**

- ↓ aggressive.
- ↓ recurrence rate.
- Painless &Slowly growing.

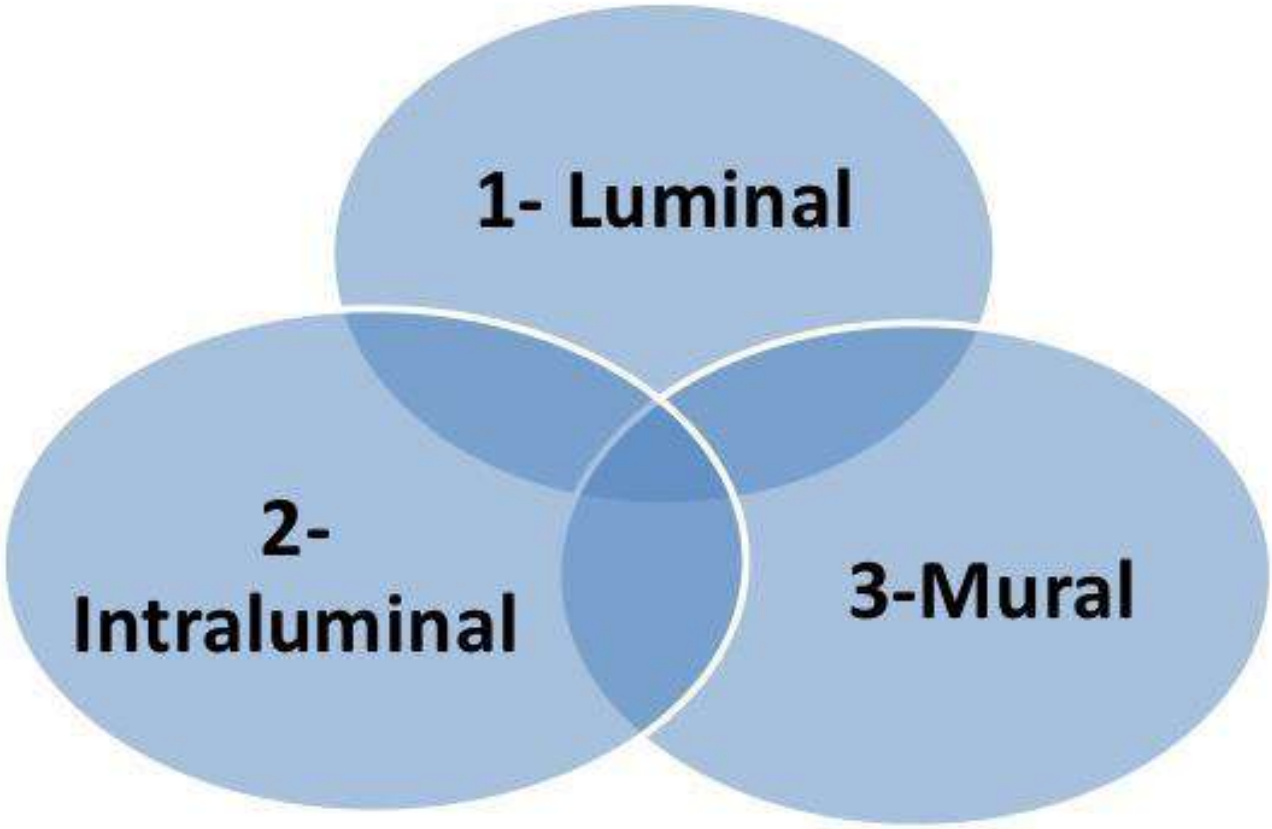
- **X-ray:**

Unilocular radiolucent.



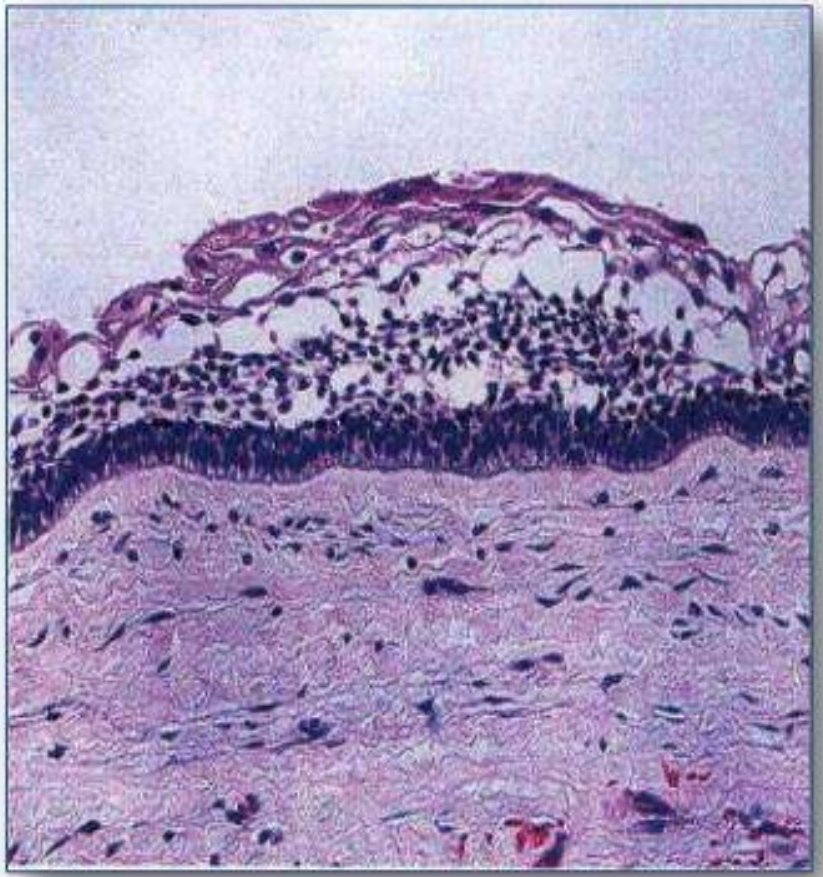


Histological Variants of Unicystic Ameloblastoma



Histological Variants of Unicystic Ameloblastoma

- **Luminal type:**
- The change is confined within the luminal surface of the cyst.
- Part of the epithelial lining changes into ameloblastic epithelium→ (columnar or cuboidal c)+hyperchromatic nuclei + reverse polarity.
- basilar cytoplasmic vacoulation.
- The overlying epithelial cells consist of loose stellate reticulum like cells.

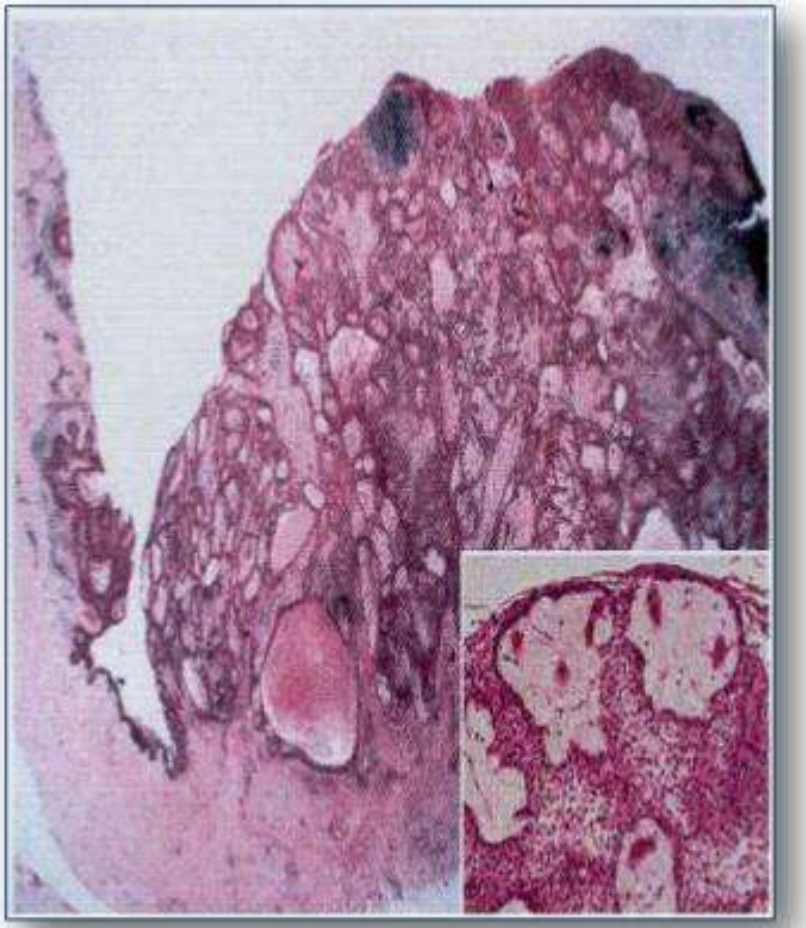


Histological Variants of Unicystic Ameloblastoma

- **Intraluminal type:**

- The nodules of ameloblastoma proliferate and project into the cyst lining.

- The lining often shows an arrangement similar to (plexiform ameloblastoma).

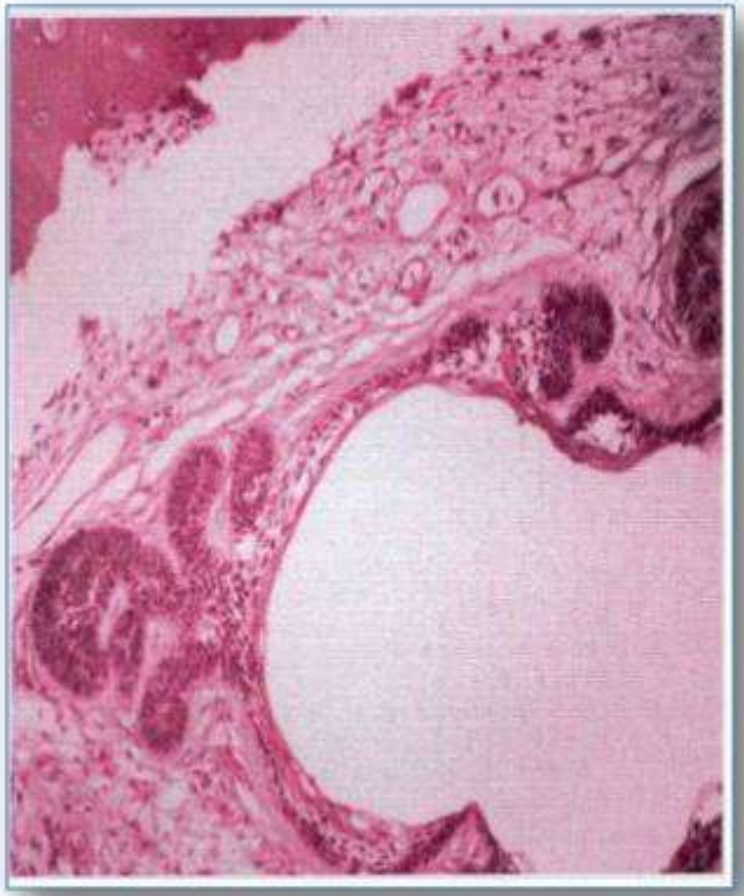


Histological Variants of Unicystic Ameloblastoma

- **Mural type:**

- The fibrous connective tissue wall of the cyst is infiltrated by ameloblastic masses.

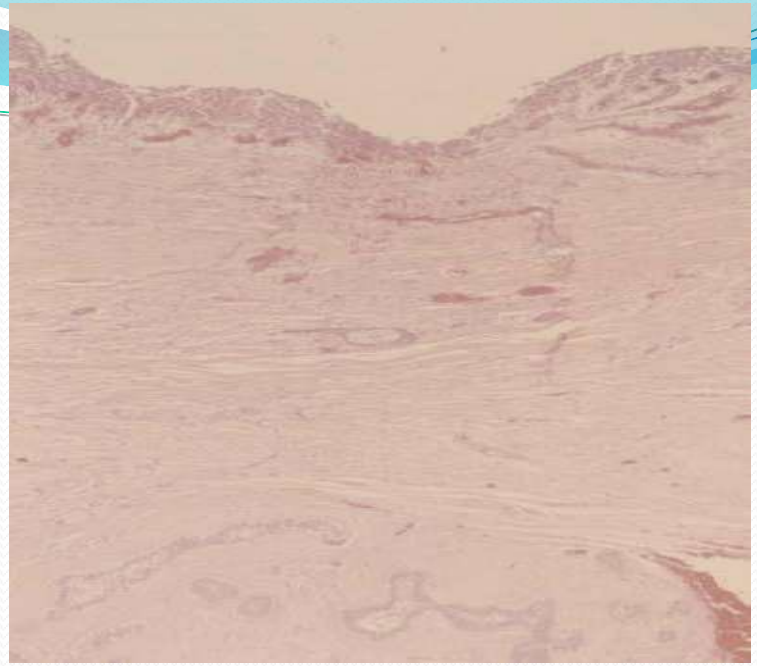
-The ameloblastic component showing (follicular or plexiform patterns).

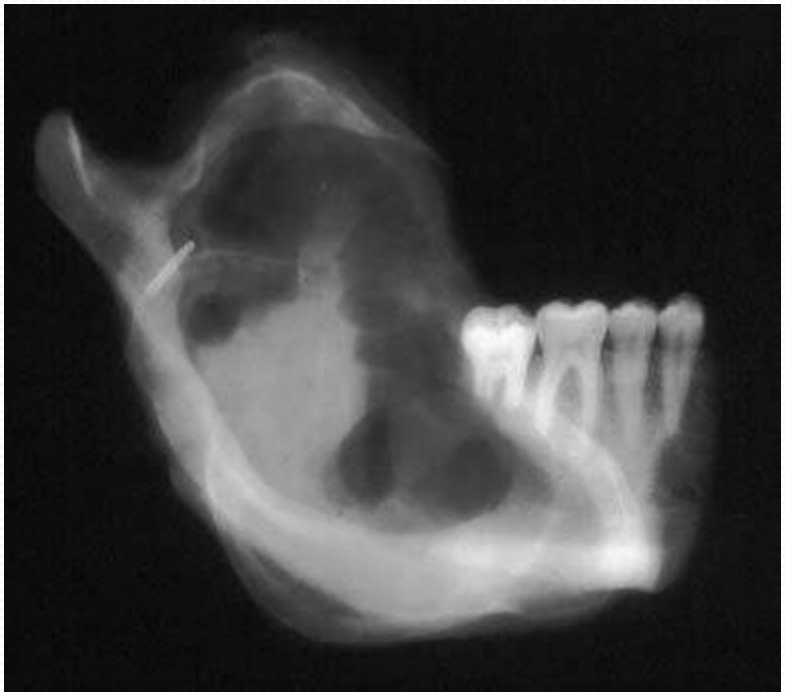


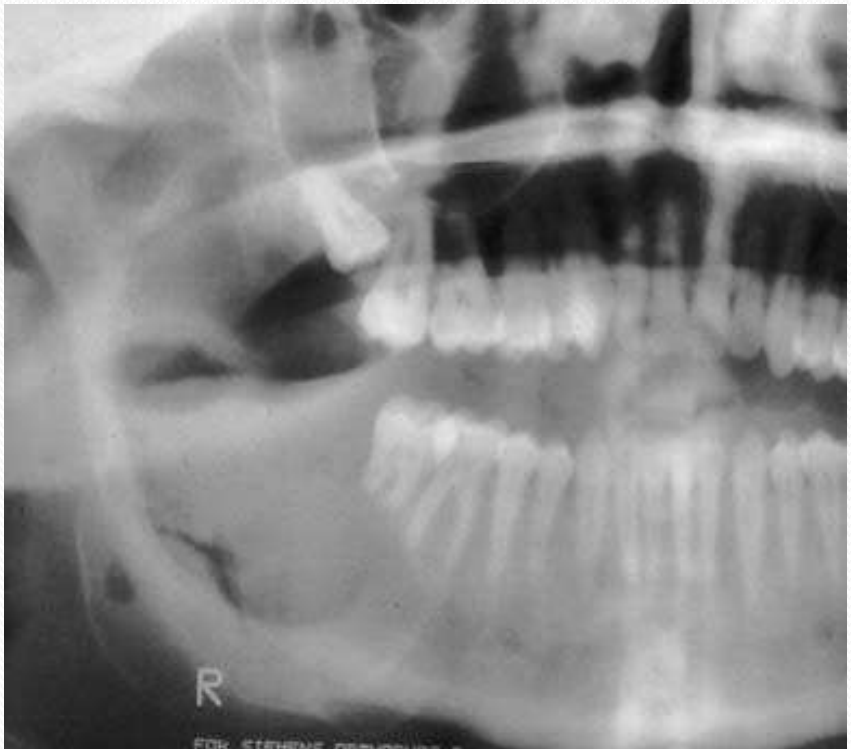
Treatment and Prognosis

- First take incisional biopsy

- The treatment of a luminal or intraluminal variant of the unicystic ameloblastoma is enucleation and curettage
- If Mural type: Resection



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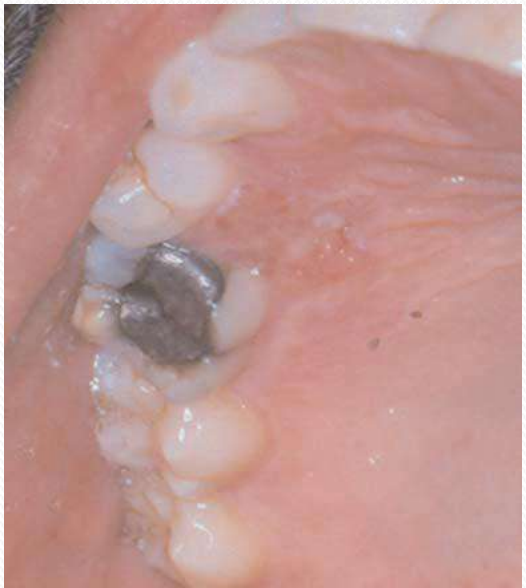
Peripheral Ameloblastoma

- The extraosseous ameloblastoma is the most rare variant
- Has same features of the intraosseous form of the tumor. Clinically, these tumors present as nonulcerated sessile or pedunculated gingival lesions

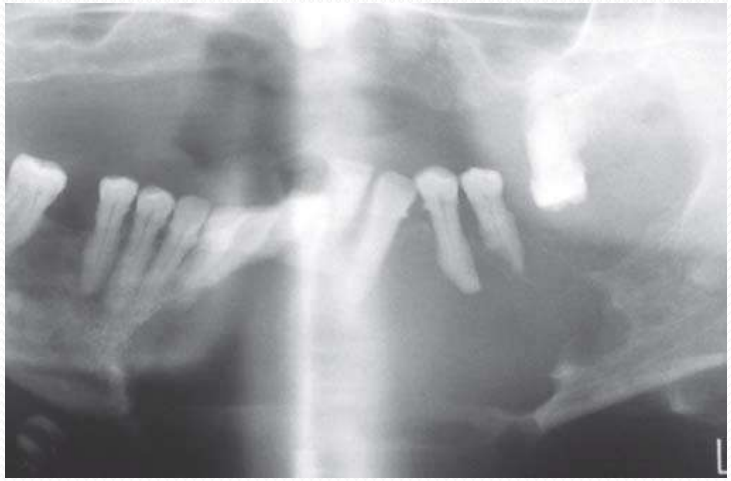


Malignant Ameloblastoma

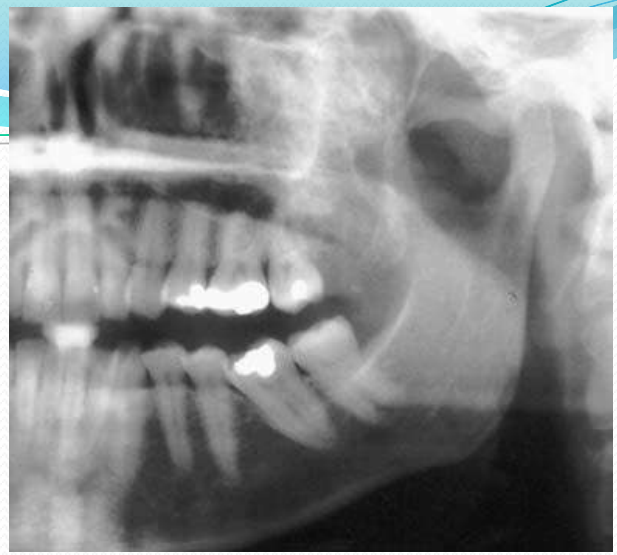
- neoplasms that have the histologic features of benign ameloblastoma as shown by the primary growth in the jaws and by any metastatic growth.
- Metastasis mostly to lung

Ameloblastic Carcinoma

- an ameloblastoma that has cytologic features of malignancy in the primary tumor, in a recurrence, or in any metastatic deposit.

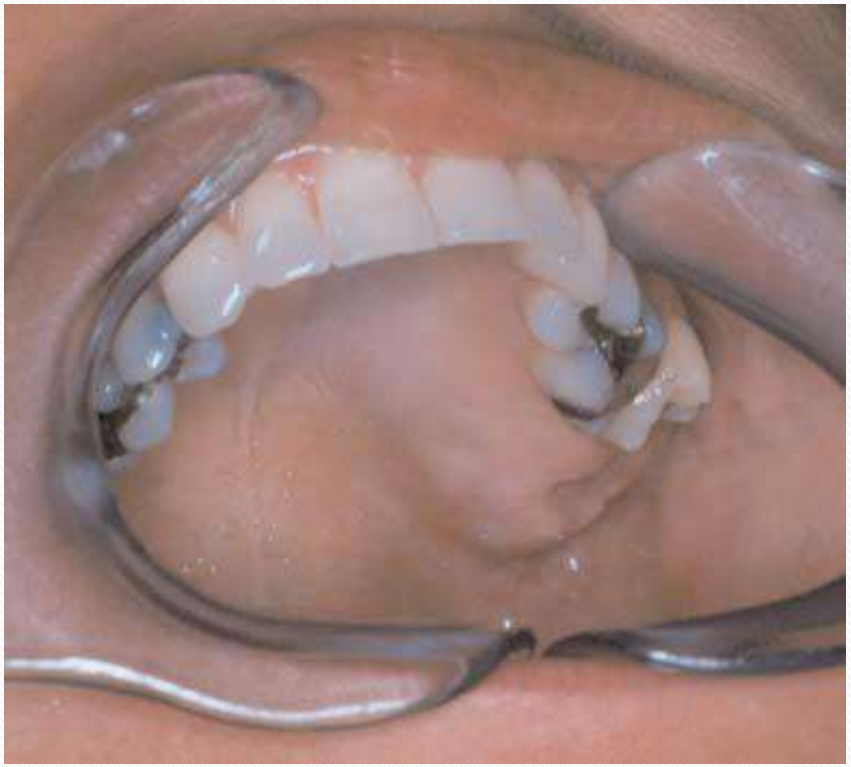


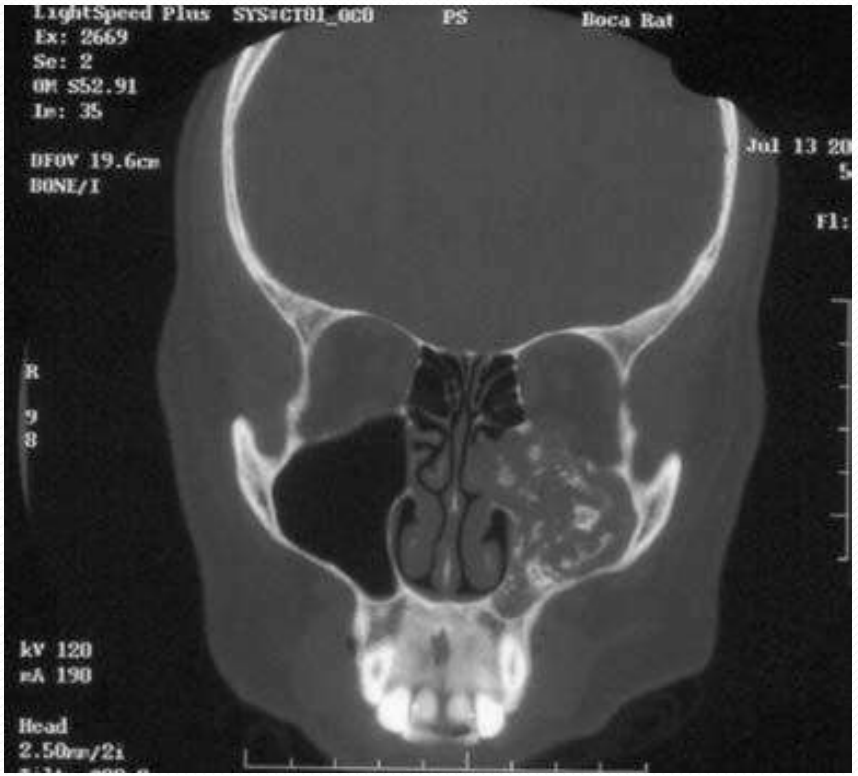
Calcifying Epithelial Odontogenic Tumor (Pindborg tumor)



- 30 and 50 years of age
- Predominantely in mandible
- Radiographically, the most common presentation is a mixed radiopaque/radiolucent lesion, frequently associated with an impacted tooth
- Liesegang rings
- Treated identically to the ameloblastoma and odontogenic myxoma, with 1.0 cm bony linear margins

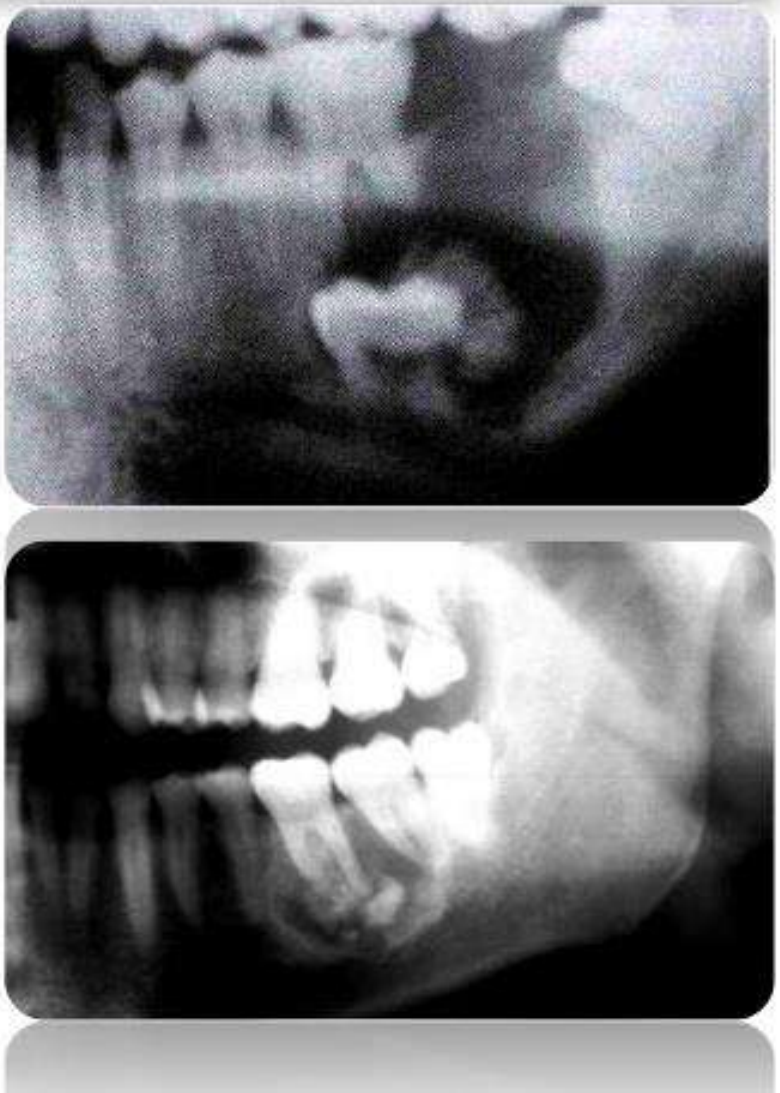
Pindporg tumor



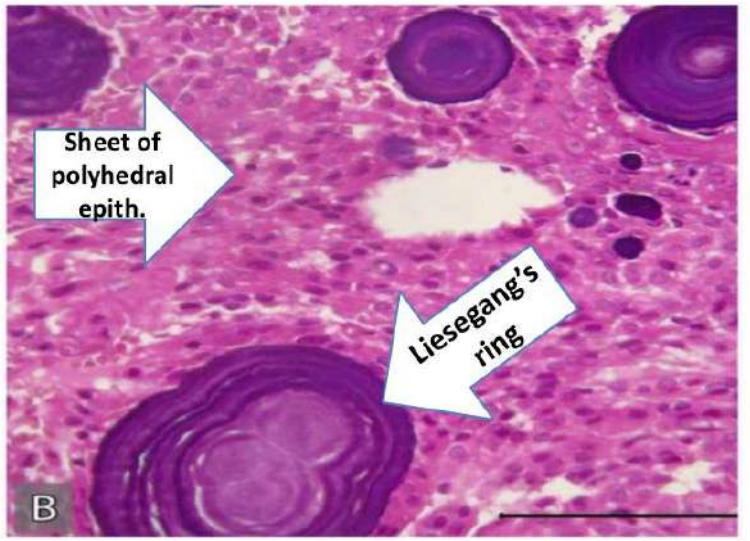


X-ray of (CEOT)(Pindborg's Tumor)

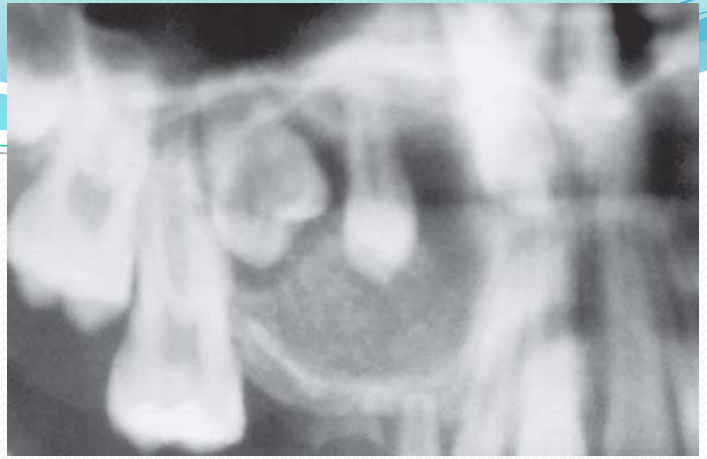
- Irregular **radiolucent** area.
- Containing **radioopaque** masses close to unerupted tooth



Features



- mixed radiopaque/radiolucent lesion, frequently associated with an impacted tooth.
- Histologically, Large areas of amorphous eosinophilic hyalinized (amyloid-like) material are also present.
- Calcifications, which are a distinctive feature of the tumor, develop within the amyloid- like material and form concentric rings, known as Liesegang rings



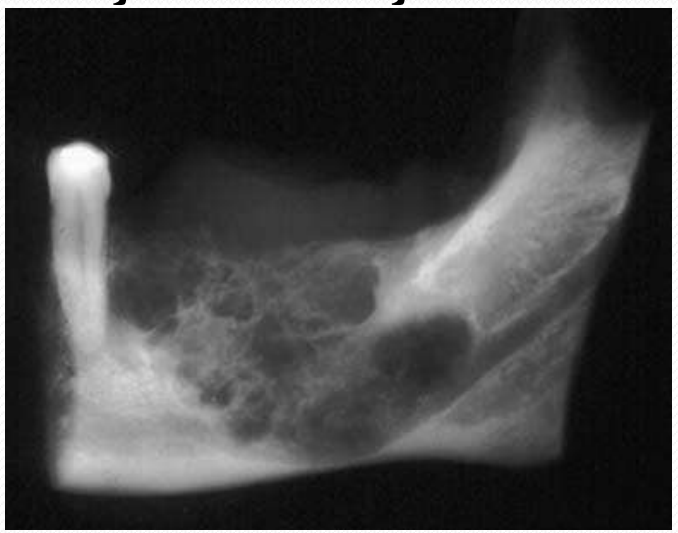
Adenomatoid Odontogenic Tumor

- Age: 2nd and 3rd decade
- predilection for the anterior region of the jaws and is found twice as often in the maxilla
- Females are affected about twice as often as males
- a well-circumscribed unilocular radiolucency that involves the crown of an erupted tooth, frequently a canine
- **Treatment:** enucleation and curettage surgery is curative

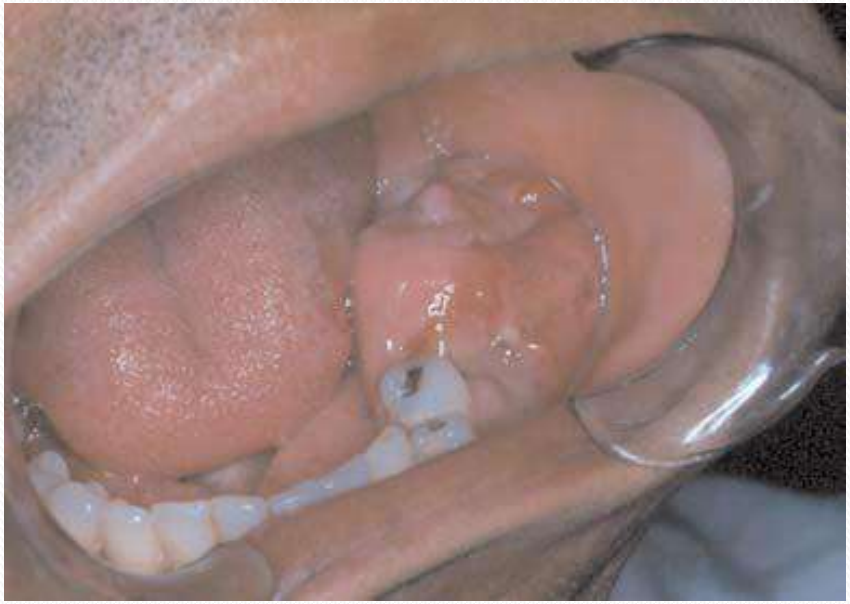
Tumors of ectomesenchyme origin

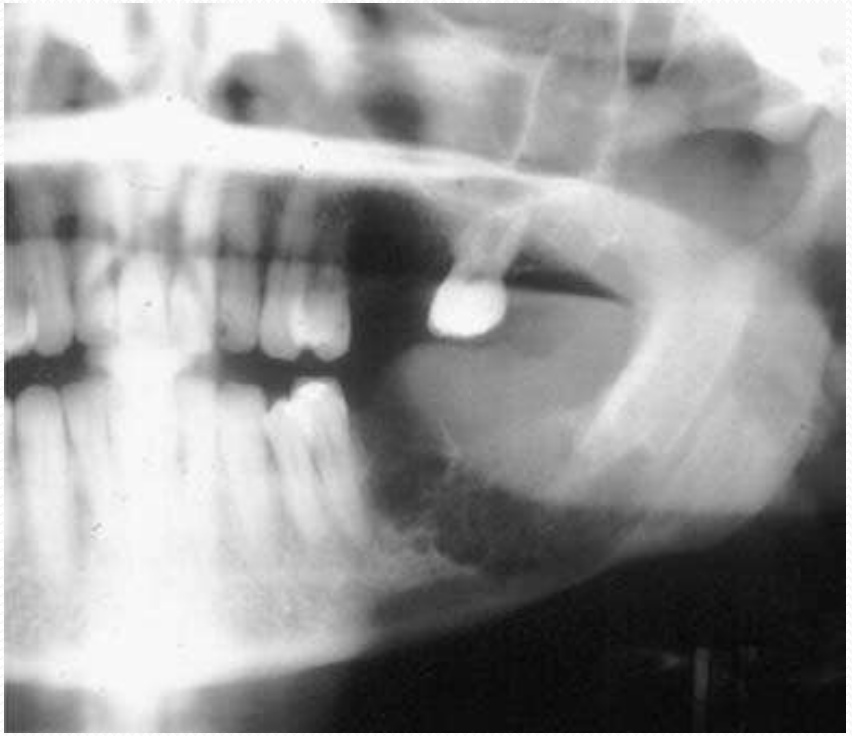
Odontogenic Myxoma

- Most commonly in the third decade of life.
- Posterior mandible
- slow growing with a potential for aggressive behavior and a high recurrence rate after subtherapeutic removal
- unilocular or multilocular radiolucency that may displace or cause root resorption
- “stepladder” pattern
- Resection with 1.0 cm bony linear margins



Myxoma





Cementoblastoma

- neoplasm of cementum
- mass of cementum-like tissue as an irregular or rounded mass attached to the root of a tooth, usually a mandibular first molar.
- Clinically, cementoblastomas mainly affect young adults, particularly males, typically below the age of 25
- Radiographically, radiopaque mass with thin radiolucent margin, attached to the roots of a tooth. Resorption of related roots is common, but the tooth remains vital.

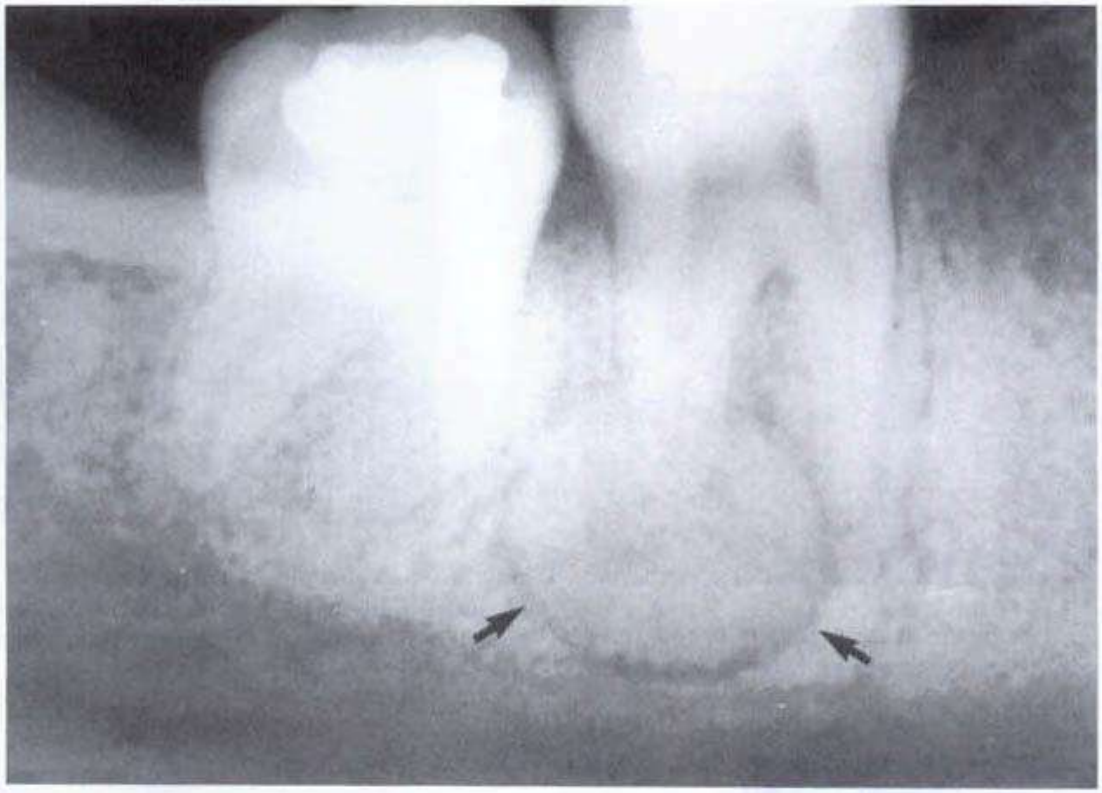


Fig. 8.30 Periapical radiograph showing the typical appearances of a cementoblastoma, a radiopaque mass with a radiolucent rim attached to the root apex. (By kind permission of Mr E Whaites.)

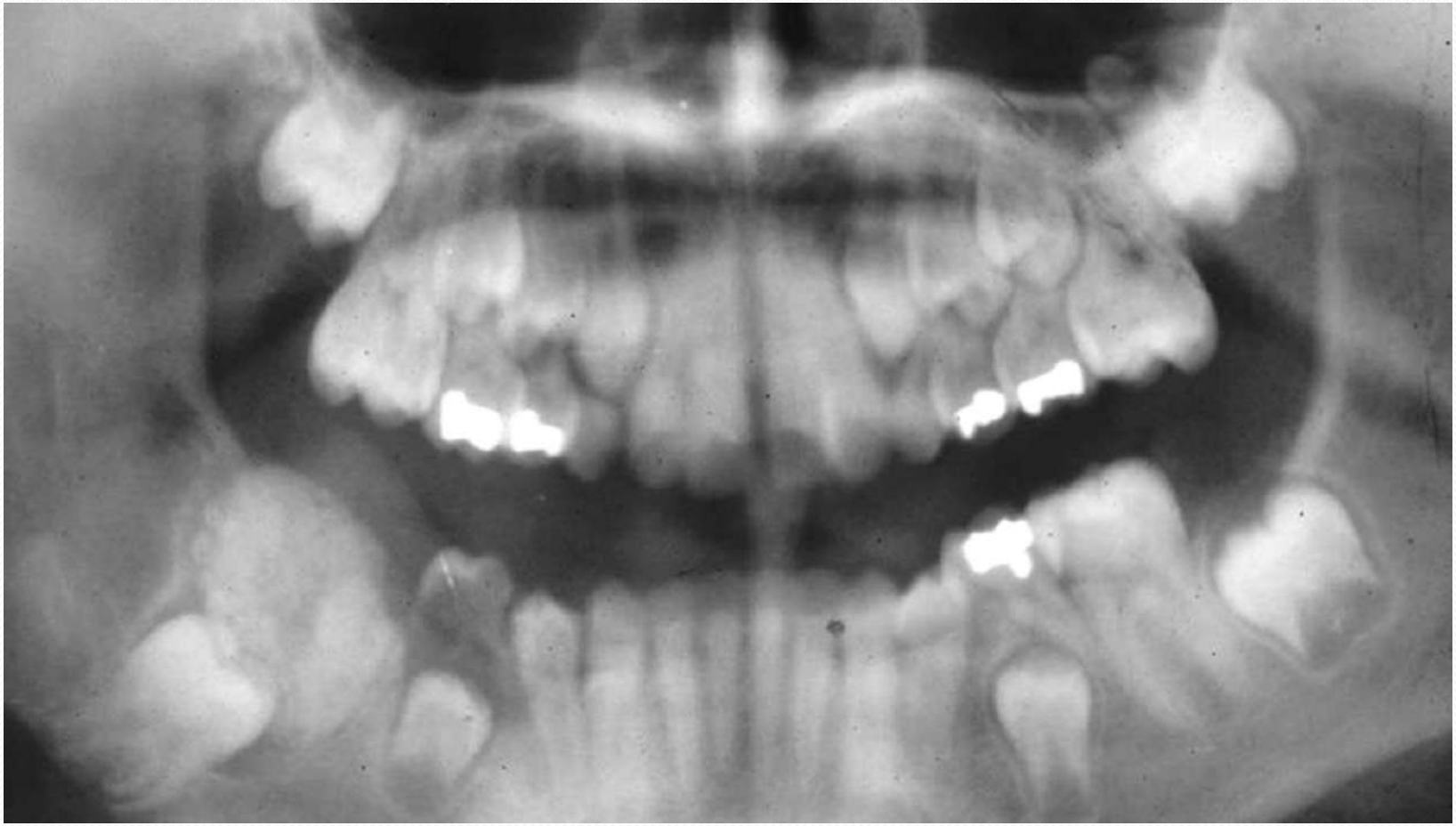
Mixed tumors

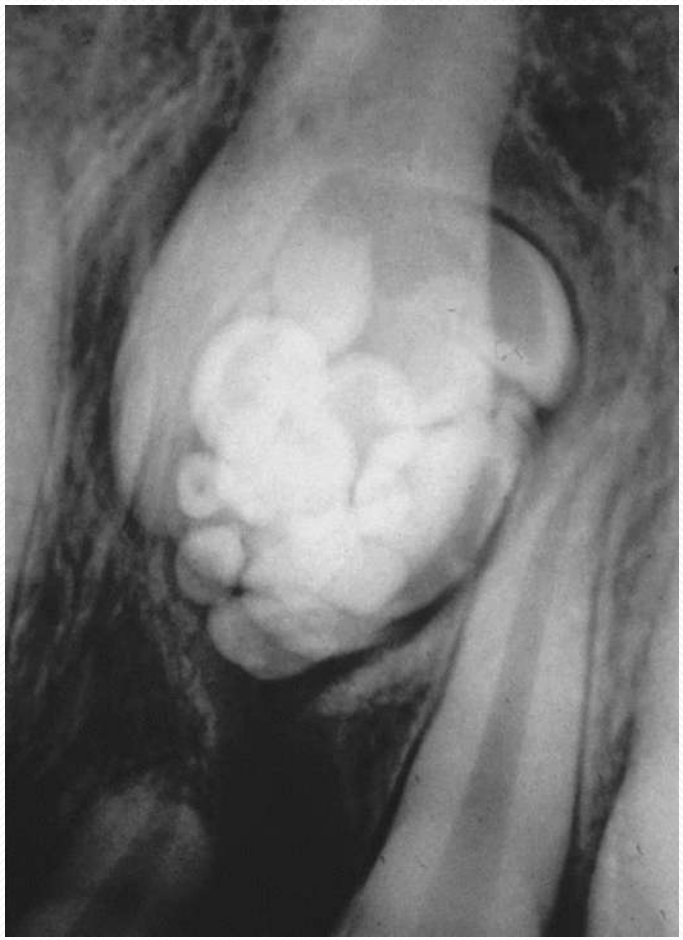
Odontoma

- are the most frequently occurring odontogenic tumors
- Odontomas present centrally within the jaws in one of two forms: *compound*, in which multiple small toothlike structures exist; and

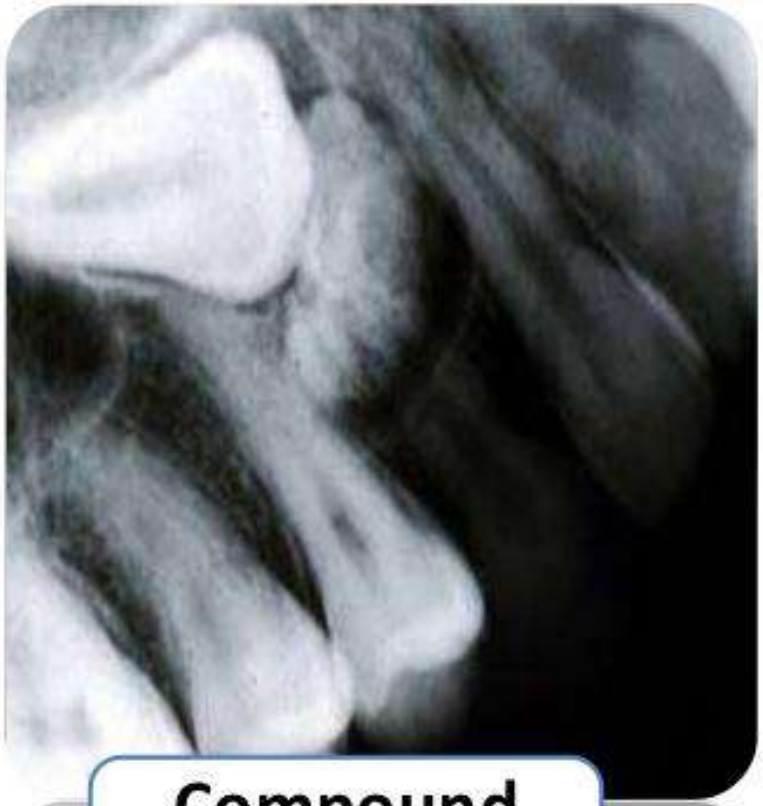
complex, in which irregular masses of dentin and enamel are present with no anatomic resemblance to a tooth.

- Mx: enucleation and curettage and are not known to recur

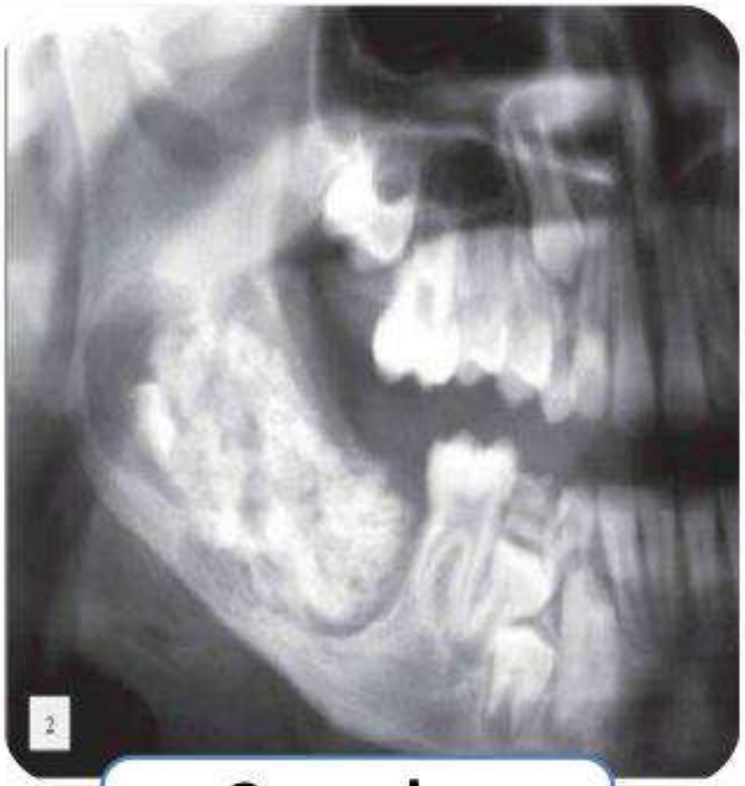




X-ray of Odontoma



Compound Odontoma



Complex Odontoma

Types of surgical procedures for the removal of jaw tumors

- Enucleation with or without curettage
- Resection (en block resection)

I. Marginal (segmental)
II. Partial resection
III. Total resection
IV. Composite

Marginal /partial resection

- Ameloblastoma
- Calcifying epithelial odontogenic tumor
- Myxoma
- Squamous odontogenic tumor

Composite resection

- Malignant ameloblastoma
- Carcinoma

ORTHOGNATHIC SURGERY

By

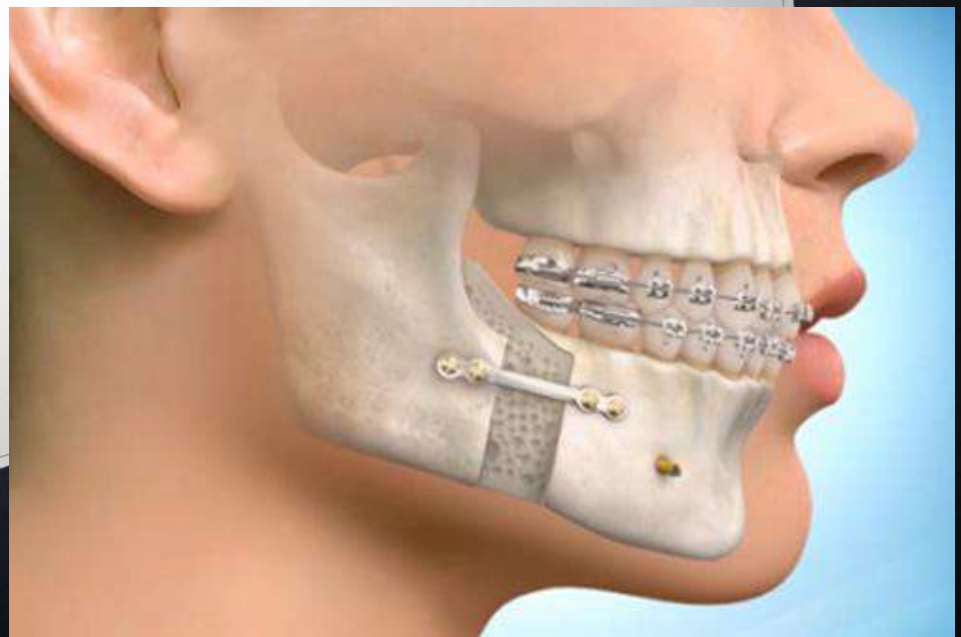
Dr. Sabah alheeti

CABMS. بورد عربي في جراحة الوجه والفكين

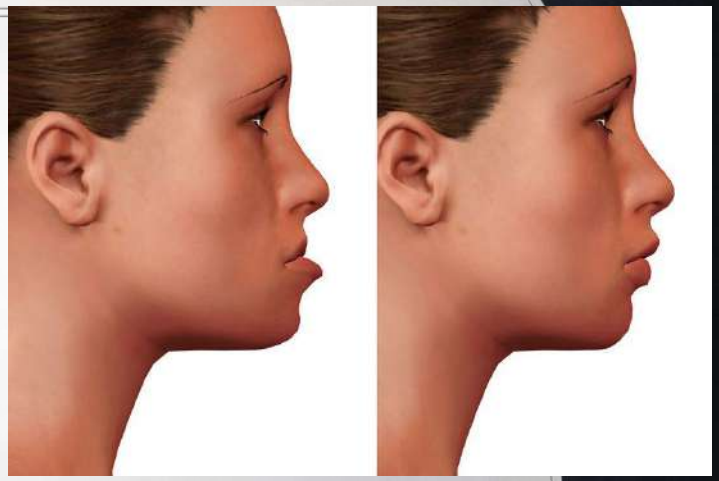
FIBMS. بورد عراقي في جراحة الوجه والفكين

ORTHOGNATHIC SURGERY

- Surgery to treat facial disproportion or surgery for correction of dentofacial deformities.
- Objectives are function, aesthetics, stability and TMJ dysfunction, sleep apnoea.



THE MANAGEMENT PROTOCOL



1) History
2) Clinical examination
3) Investigations
4) Initial diagnosis
5) Treatment planning
6) Presurgical orthodontics
7) Surgery
8) Post- surgical orthodontics
9) When appropriate, restorative dentistry, psychological support and speech therapy will be required

History (Cc, HPI, PDH,PMH)

Clinical examination

Frontal analysis

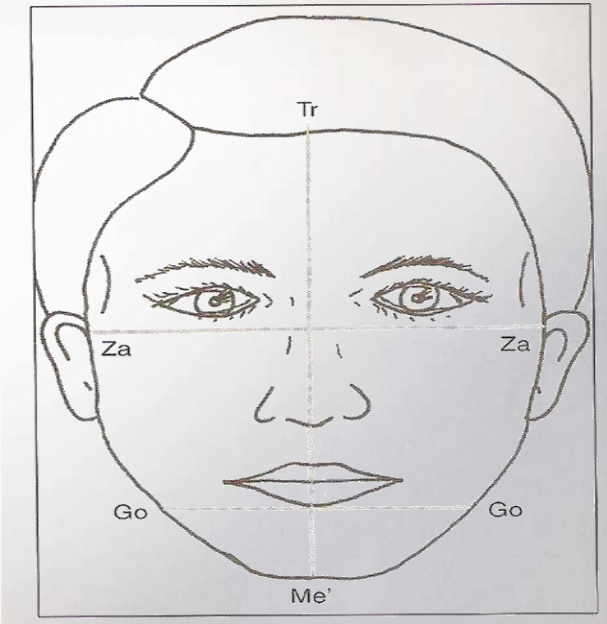
Lateral analysis

Investigations

FRONTAL ANALYSIS

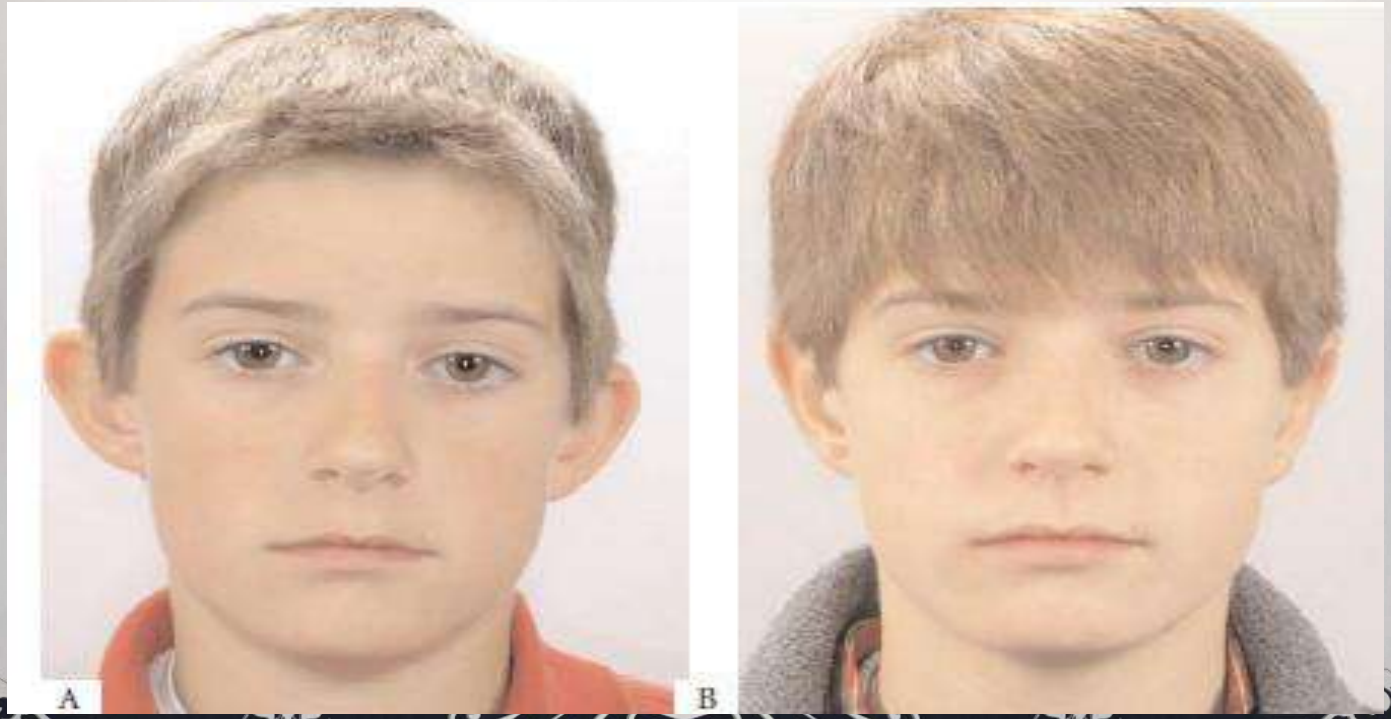
FACIAL FORM

- Facial height : Bizygomatic width : 1.35 : 1
- Bigonial width : 30% less than bizygomatic width



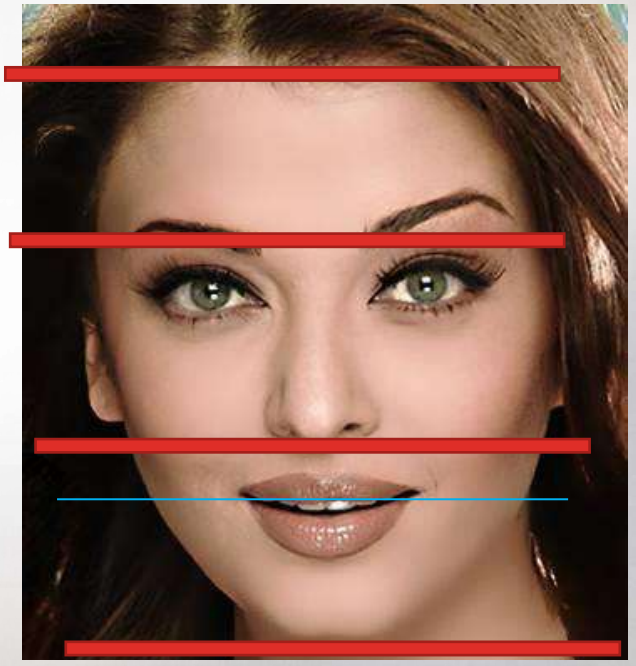
TRANSVERSE DIMENSIONS

The facial transverse fifths

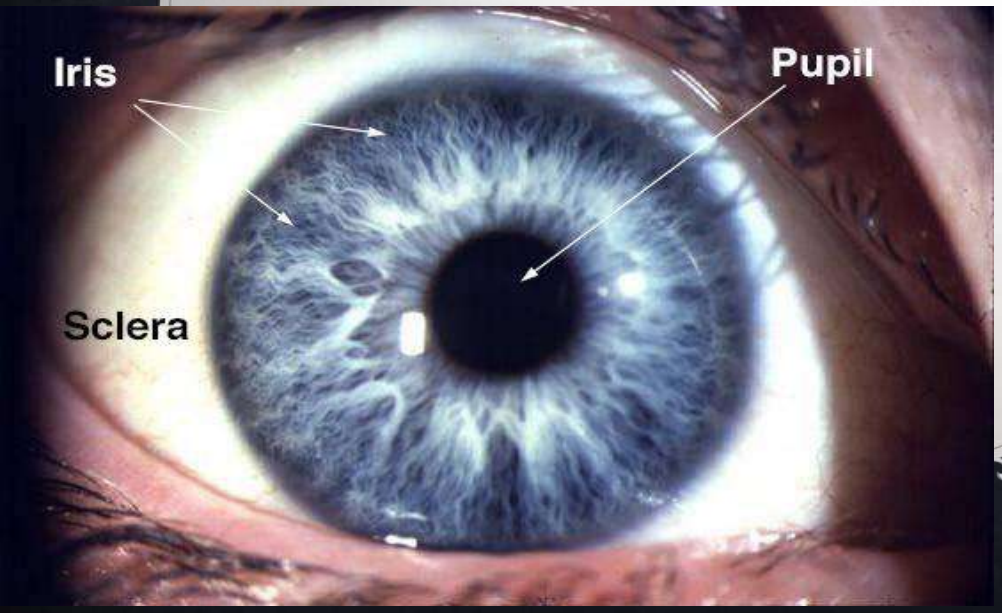


VERTICAL RELATIONSHIP

- Face divided into three equal parts:
- Trichion to Glabella
- Glabella to Subnasale
- Subnasale to Menton



❖In mid face deficiency, sclera seen below iris



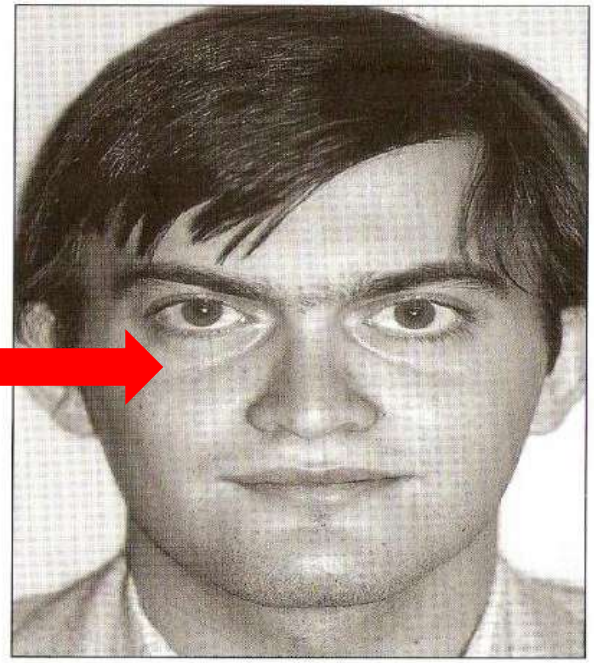
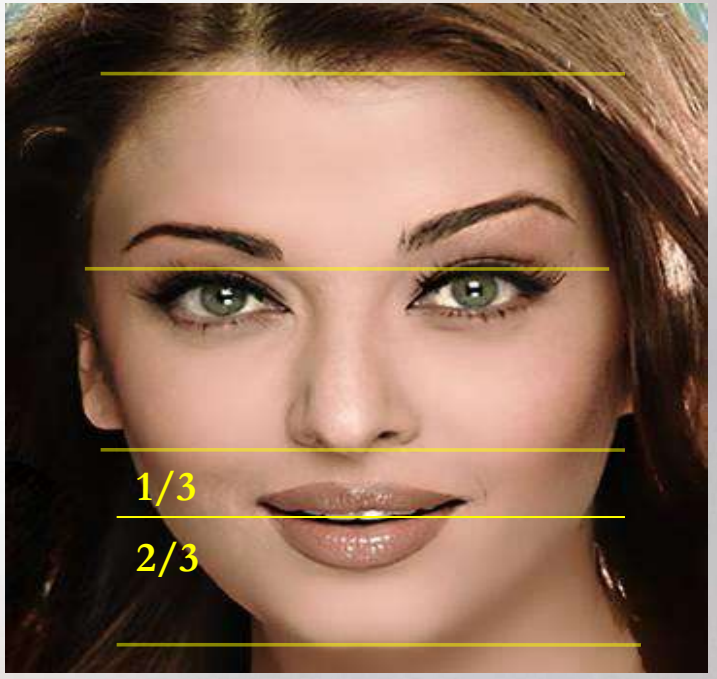


Fig 2-13 Sclera shows below the irides of the eyes in this individual with midface deficiency.

UPPER LIP LENGTH

- Upper lip length:
  - Males=22+/- 2 mm
  - Females=2o+/- 2 mm



Lip- incisor measurment

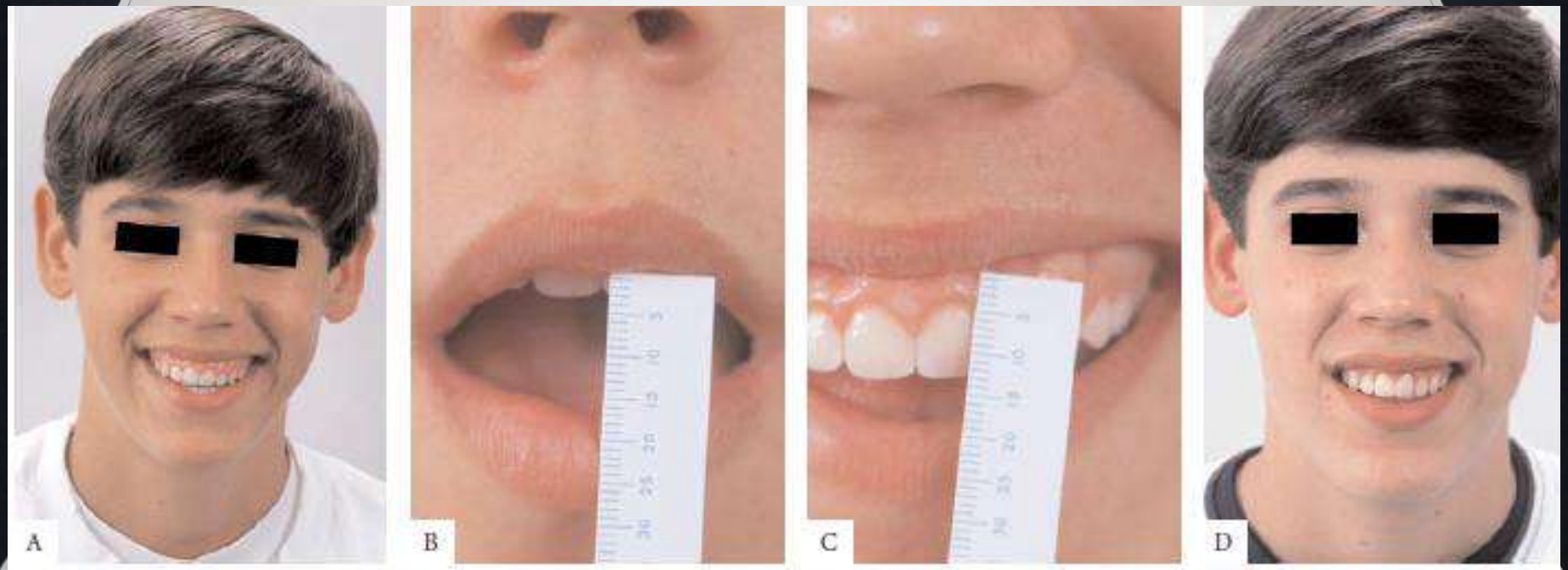


FIGURE 54-4 A–D, *Case illustration of direct measurement of lip-tooth-gingival relationships.*

FACIAL MIDLINE AND OCCLUSAL CANT

- Interpupil line should be parallel to floor.

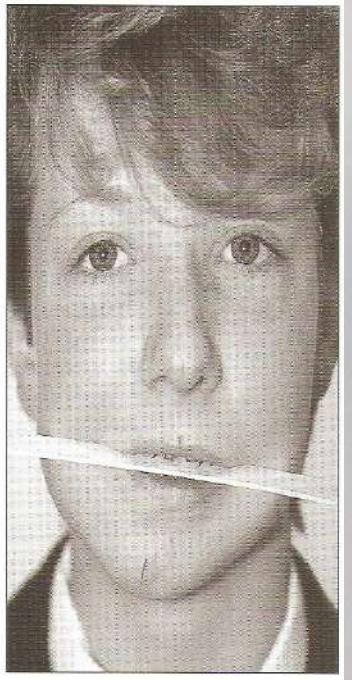
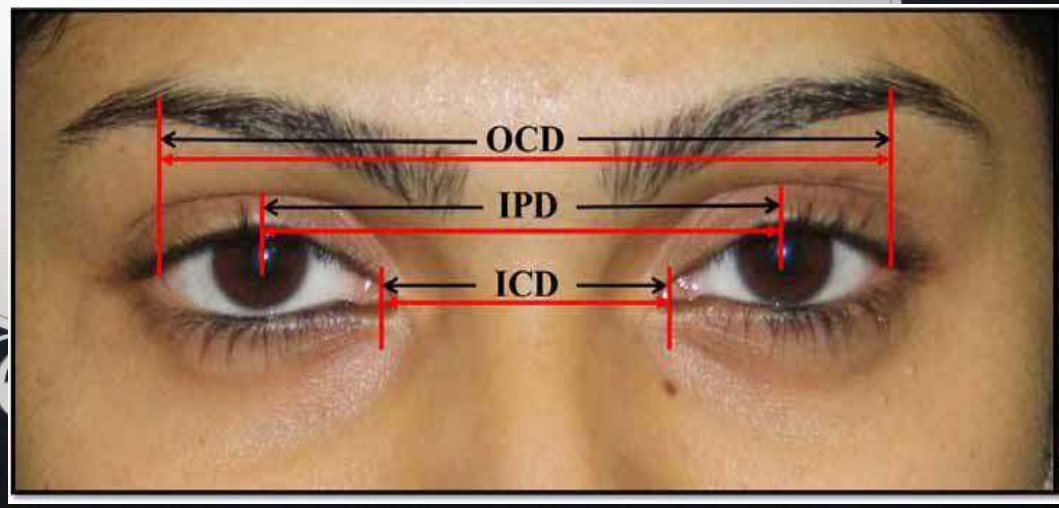


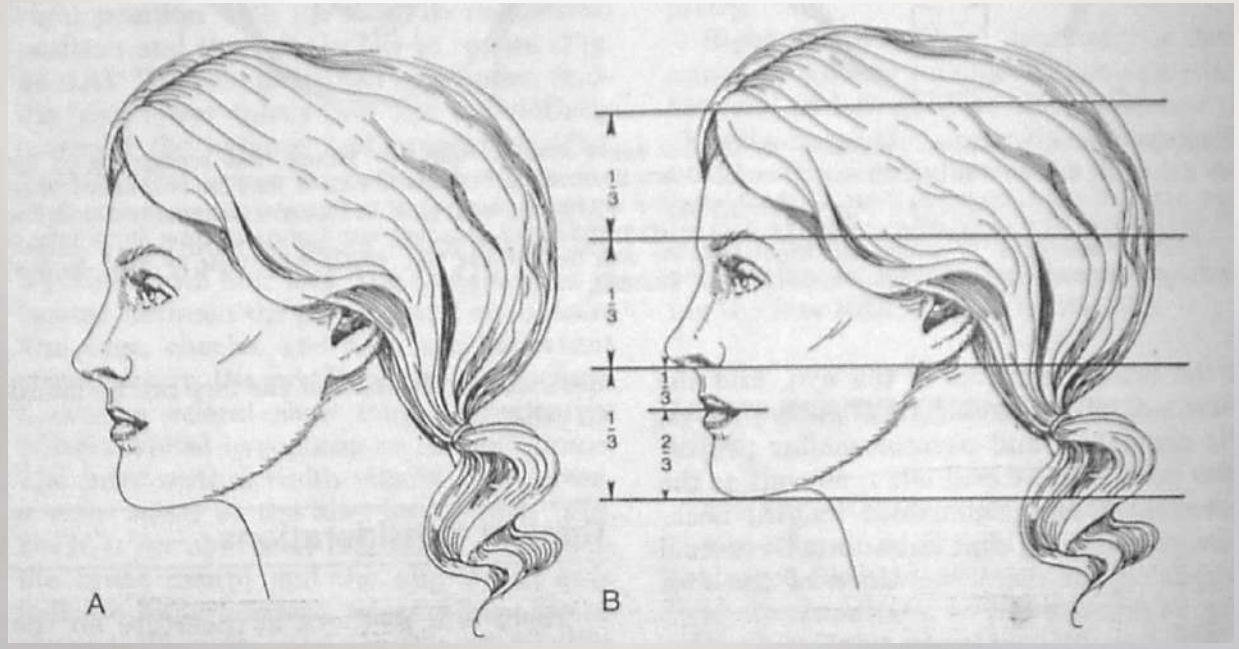
Fig 2-19 The cant in the occlusal plane can be evaluated in relation to the interpupillary line by asking the patient to bite on a wooden spatula. During this assessment, make sure that the interpupillary line is parallel to the floor.

FRONTAL ANALYSIS

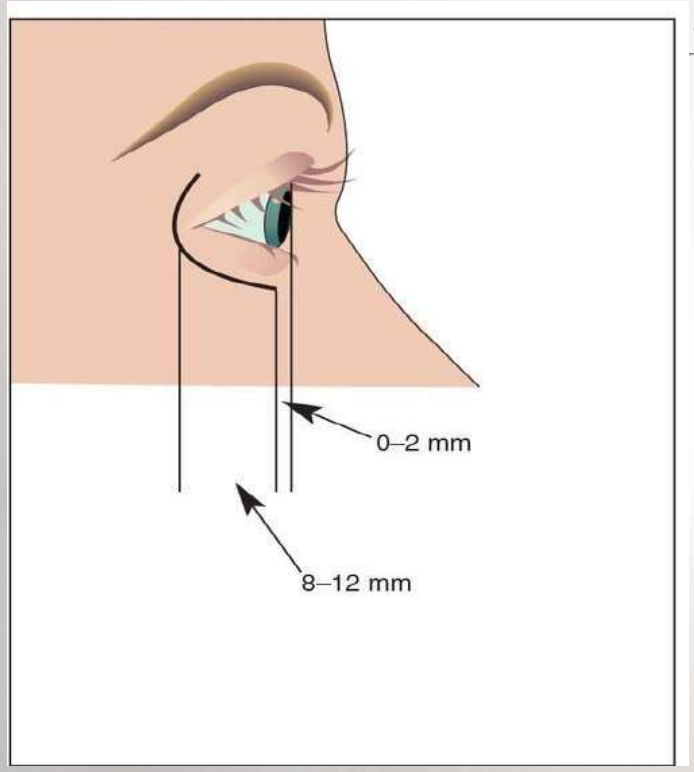
- Normal intercanthal distance 32 ± 3 mm for whites 35 ± 3 mm blacks
- Normal interpupillary distances 65 ± 3 mm.
- The inter canthal distance, alar base width and palpebral tissue width should all be equal.



LATERAL / PROFILE ANALYSIS



INFRAORBITAL RIM



- The lateral orbital rim lies 8 to 12 mm behind the globe, and the globe projects 0 to 2 mm ahead of the infraorbital rim.

LABIOMENTAL FOLD

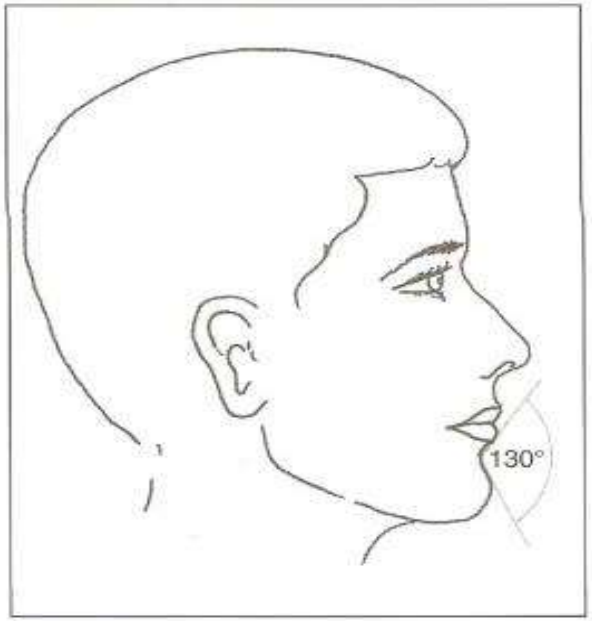


Fig 2-28 Labiomental fold.

NASOLABIAL ANGLE

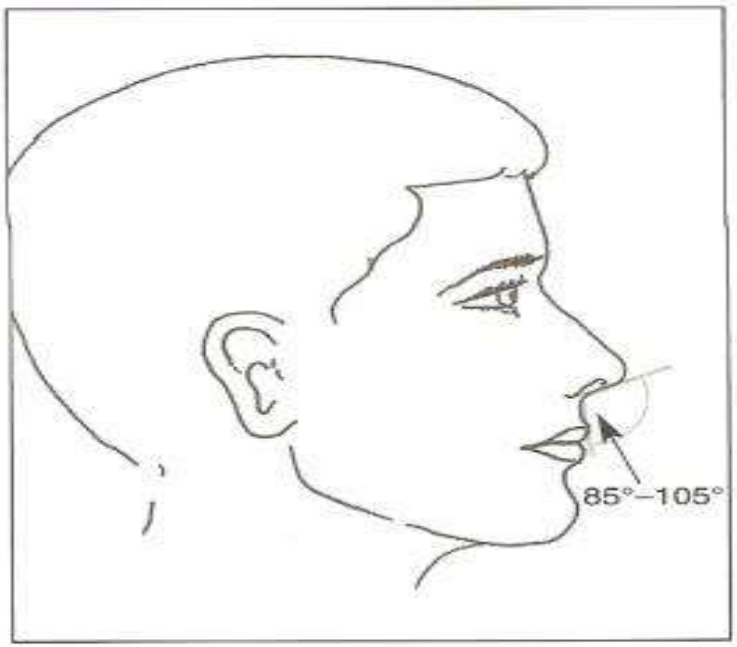


Fig 2-29 The nasolabial angle, measured between the columella of the nose and the upper lip (Sn–Ls), should be 85 to 105 degrees.

CHIN- THROAT ANGLE

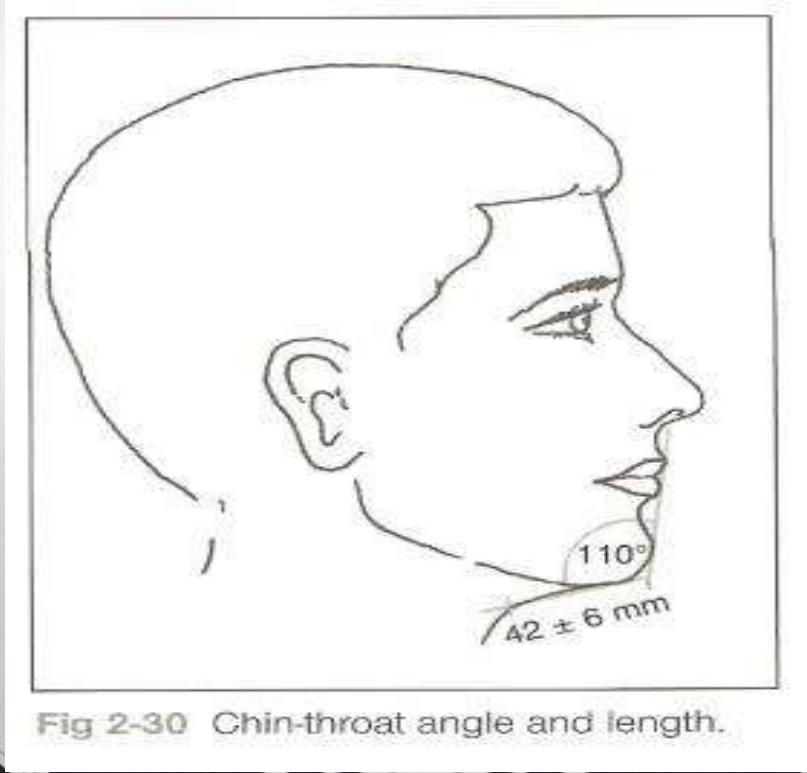


Fig 2-30 Chin-throat angle and length.

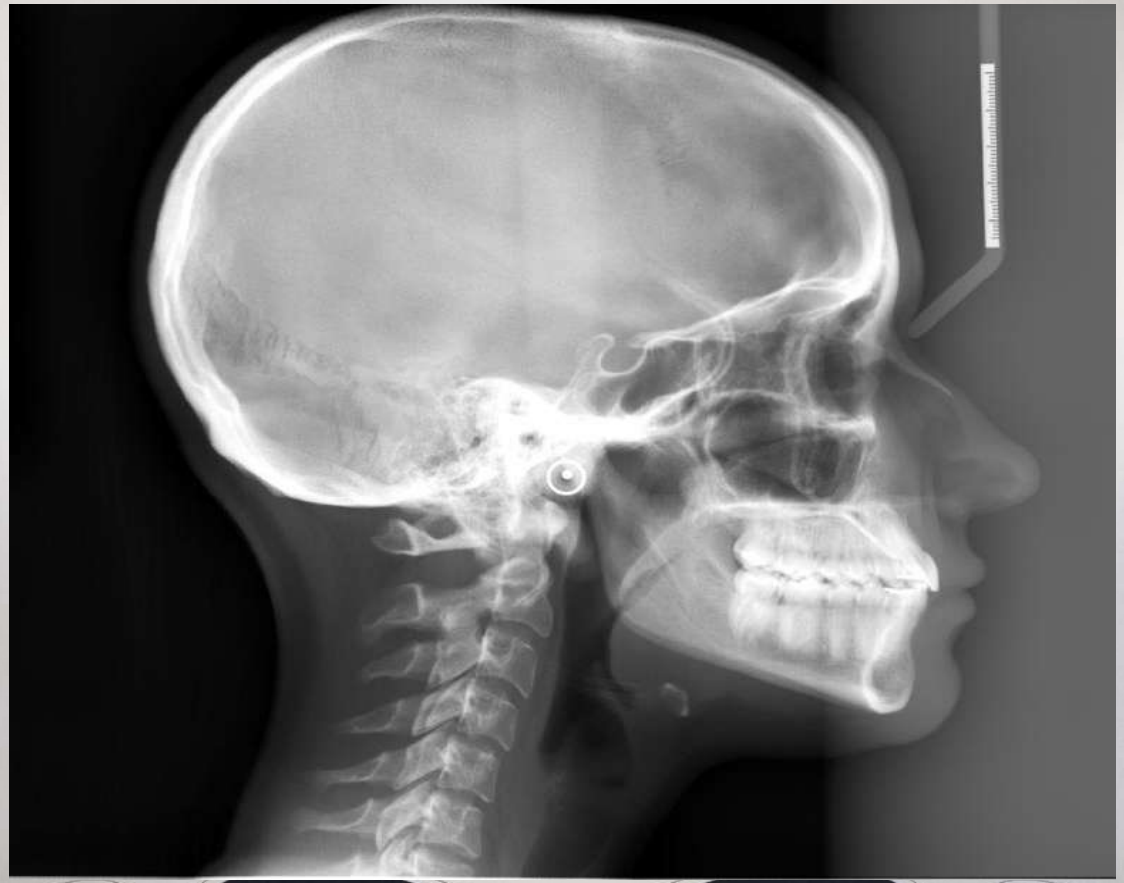
INVESTIGATIONS

- Photographs
- Study model analysis
- Radiographs
- Laboratory Ix

RADIOGRAPHS

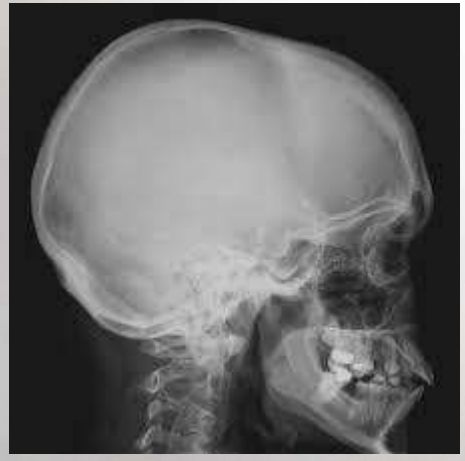
- Lateral cephalometric
- OPG
- Periapical

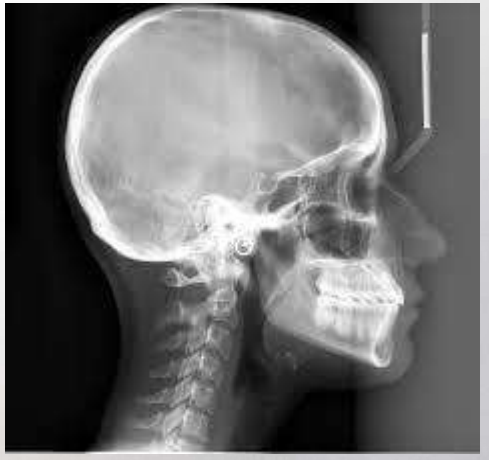
LATERAL CEPHALO.



QUESTION

- What is the difference between lateral cephalometric and lateral skull radiograph?





POINTS

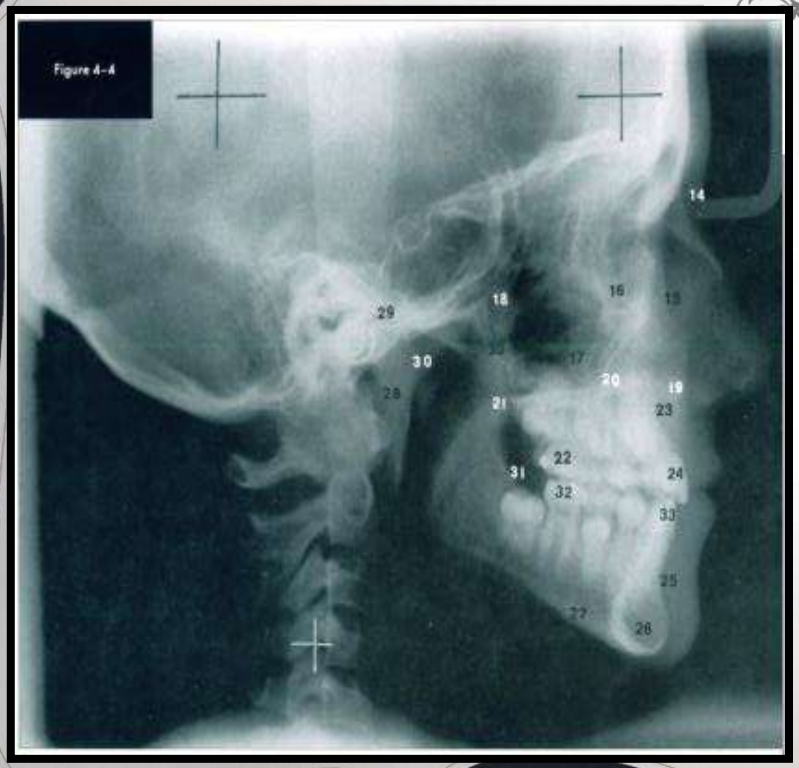


Figure 4-4
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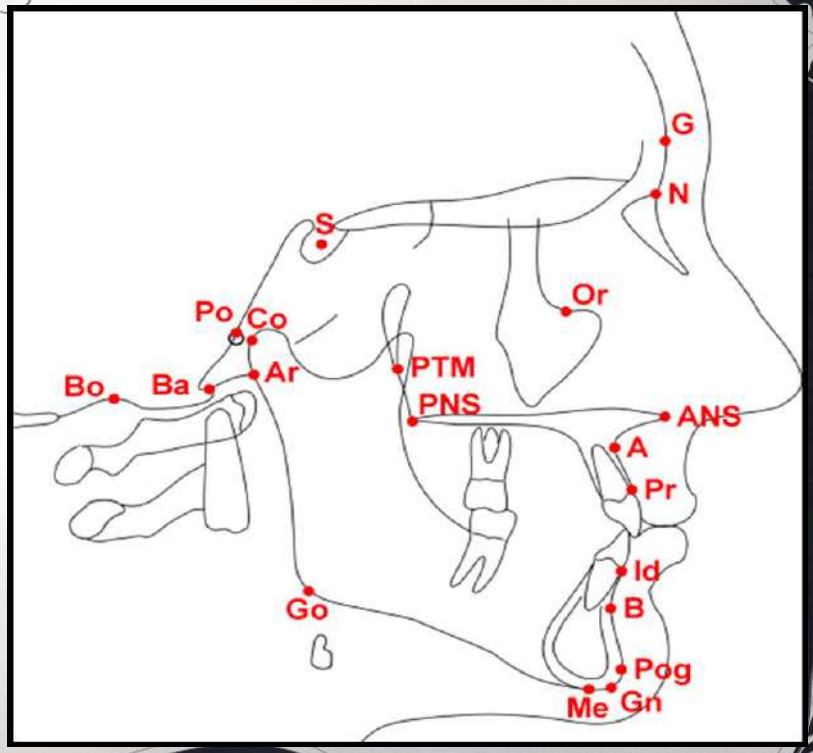
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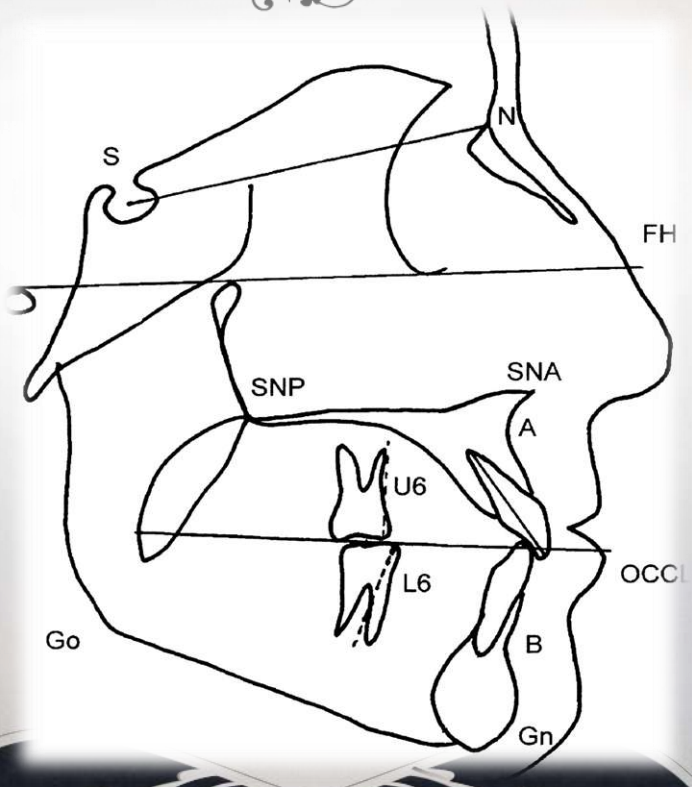
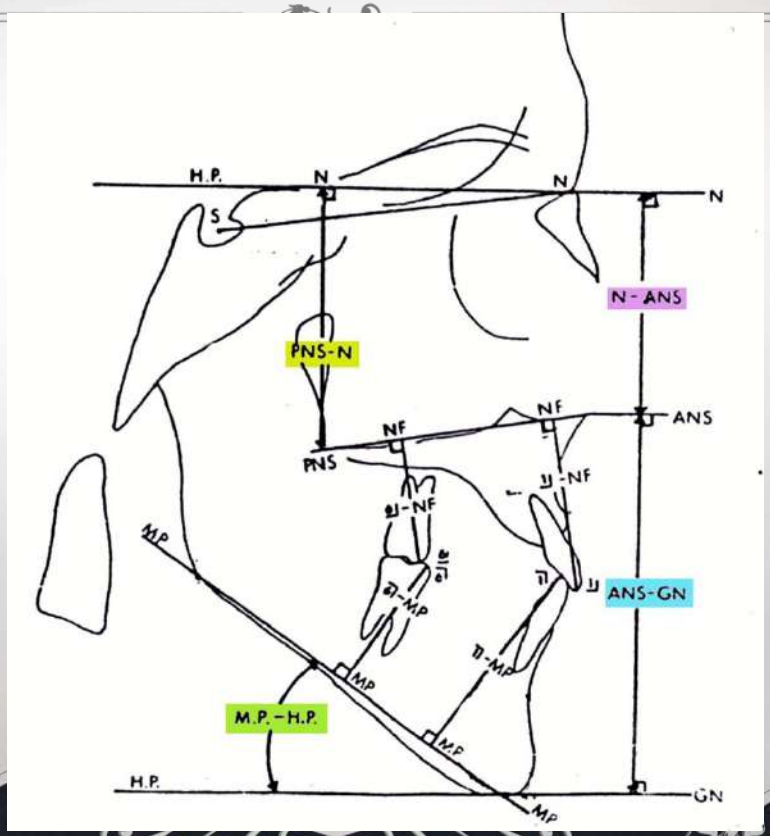
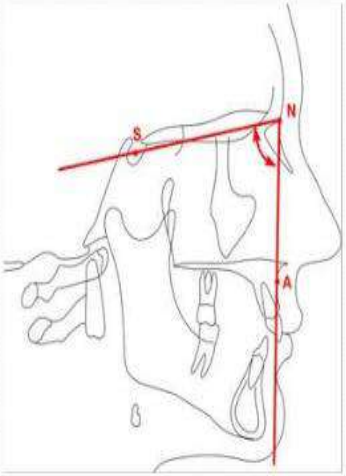
PLANES



VERTICAL MEASURES

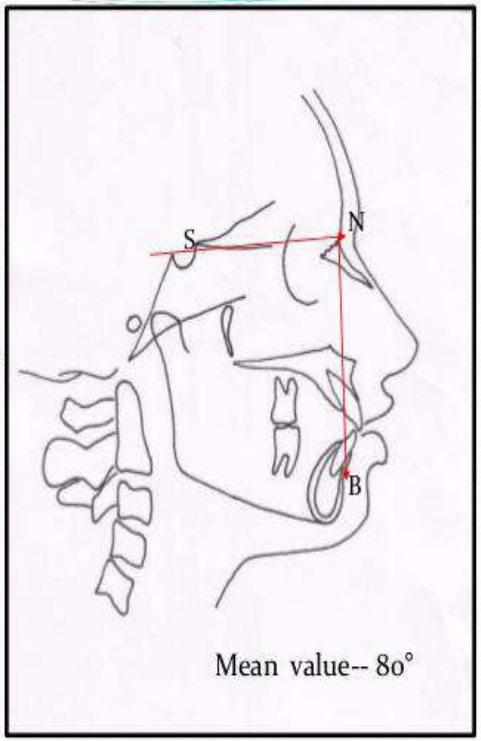


SNA angle(82 ± 2)

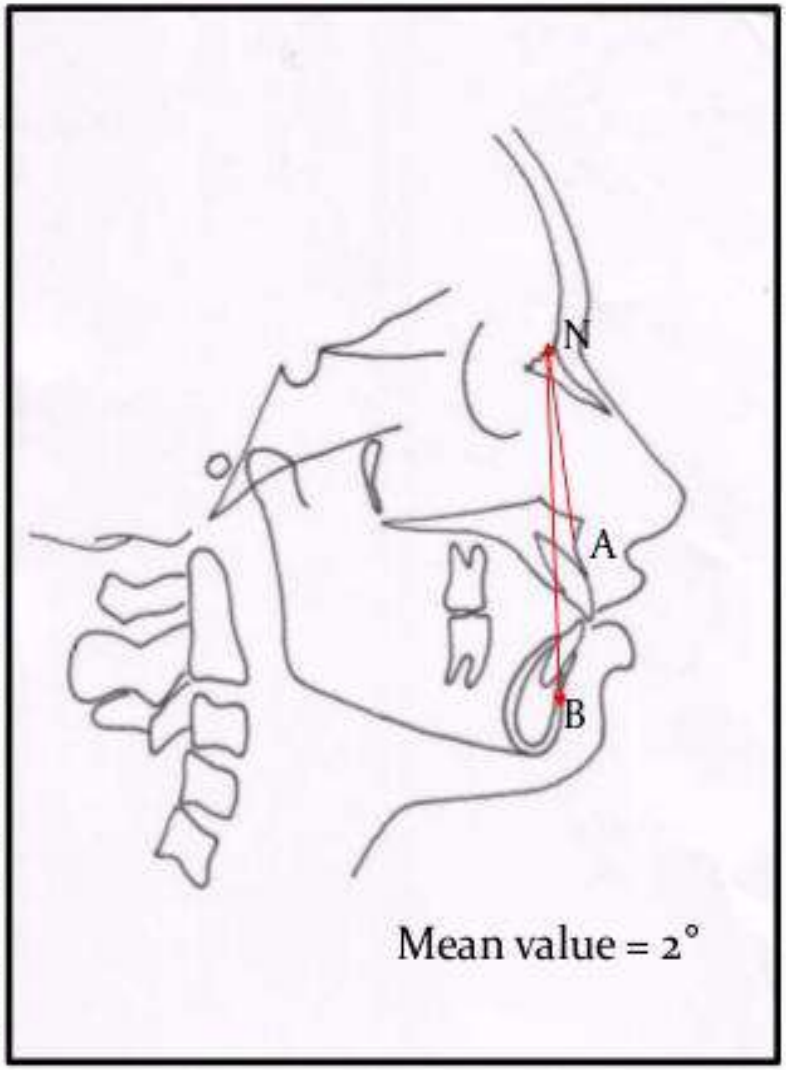


- **S.N.B angle**

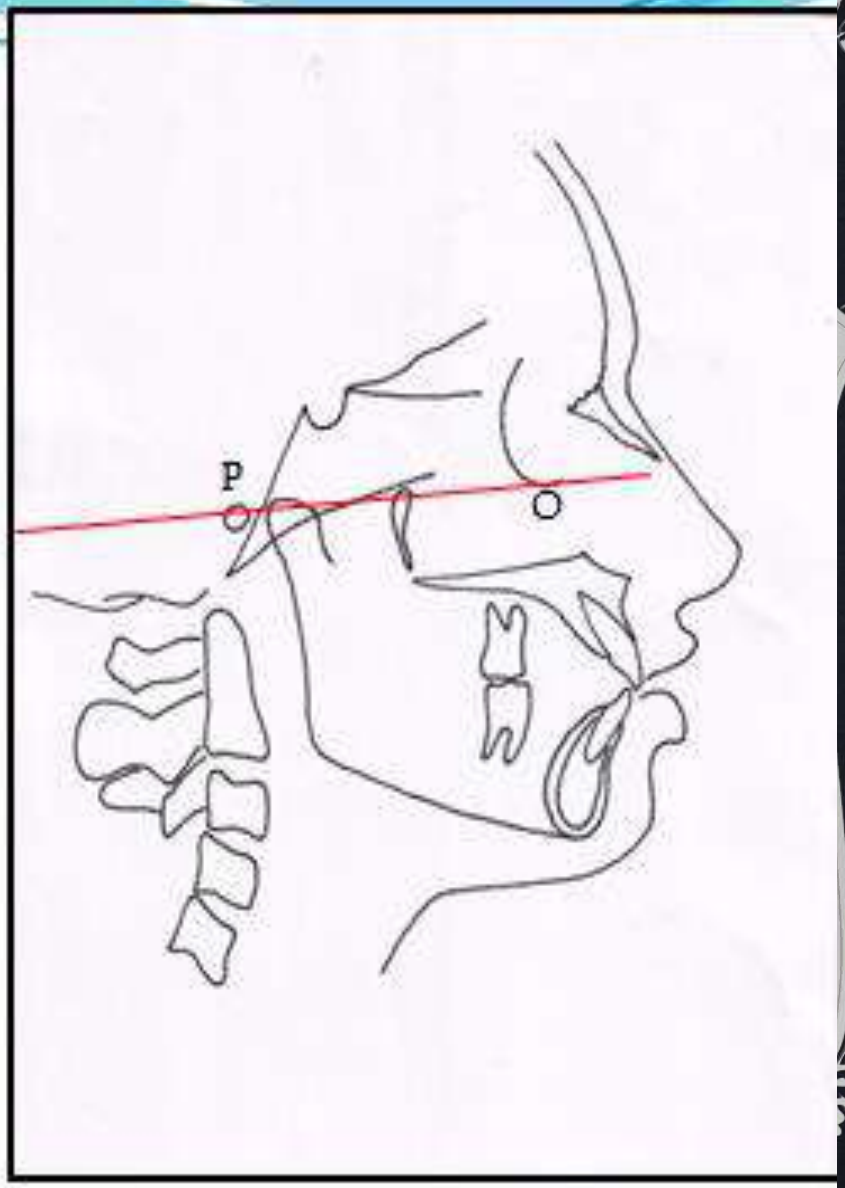
- ✓ Indicates antero-posterior positioning of the mandible in relation to cranial base
- ✓ > 80°-- prognathic mandible
- ✓ < 80°-- retrusive mandible



- **A.N.B angle**
  - ✓ Denotes relative position of maxilla & mandible to each other
  - ✓ > 2° -- class 2 skeletal tendency
  - ✓ < 2° -- skeletal class 3 tendency

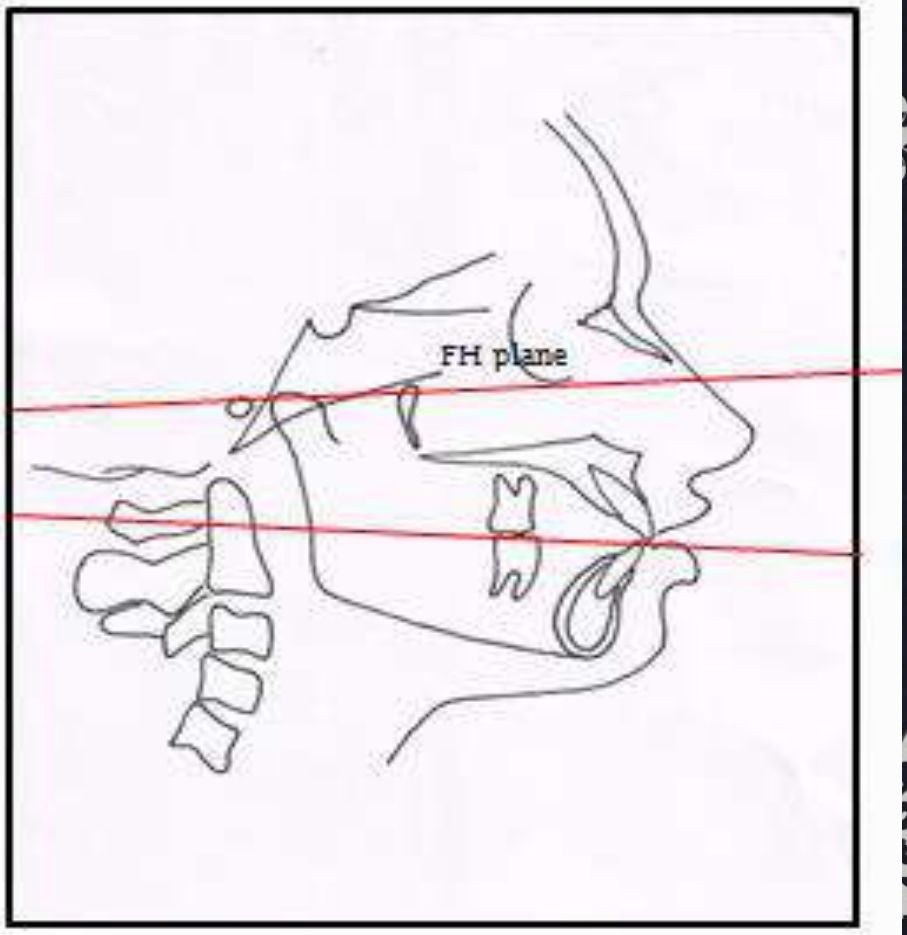


- **Horizontal planes**
  - *Frankfurt Horizontal plane*



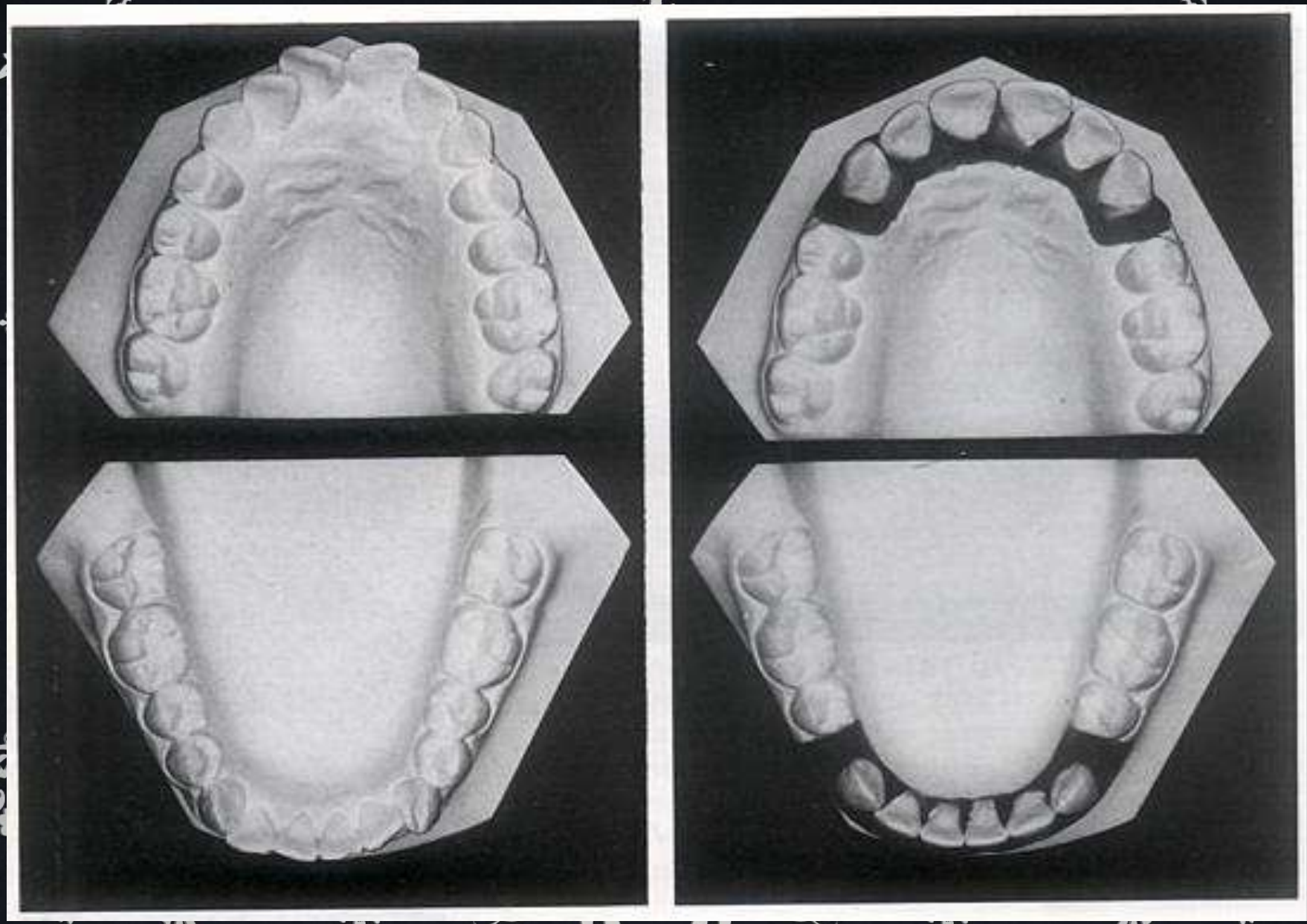
- *Dental parameters*
  - **Cant of occlusal plane**

- ✓ Mean value = 9.3°, Range = 1.5 to 14°
- ✓ Gives a measure of slope of occlusal plane relative to FH plane



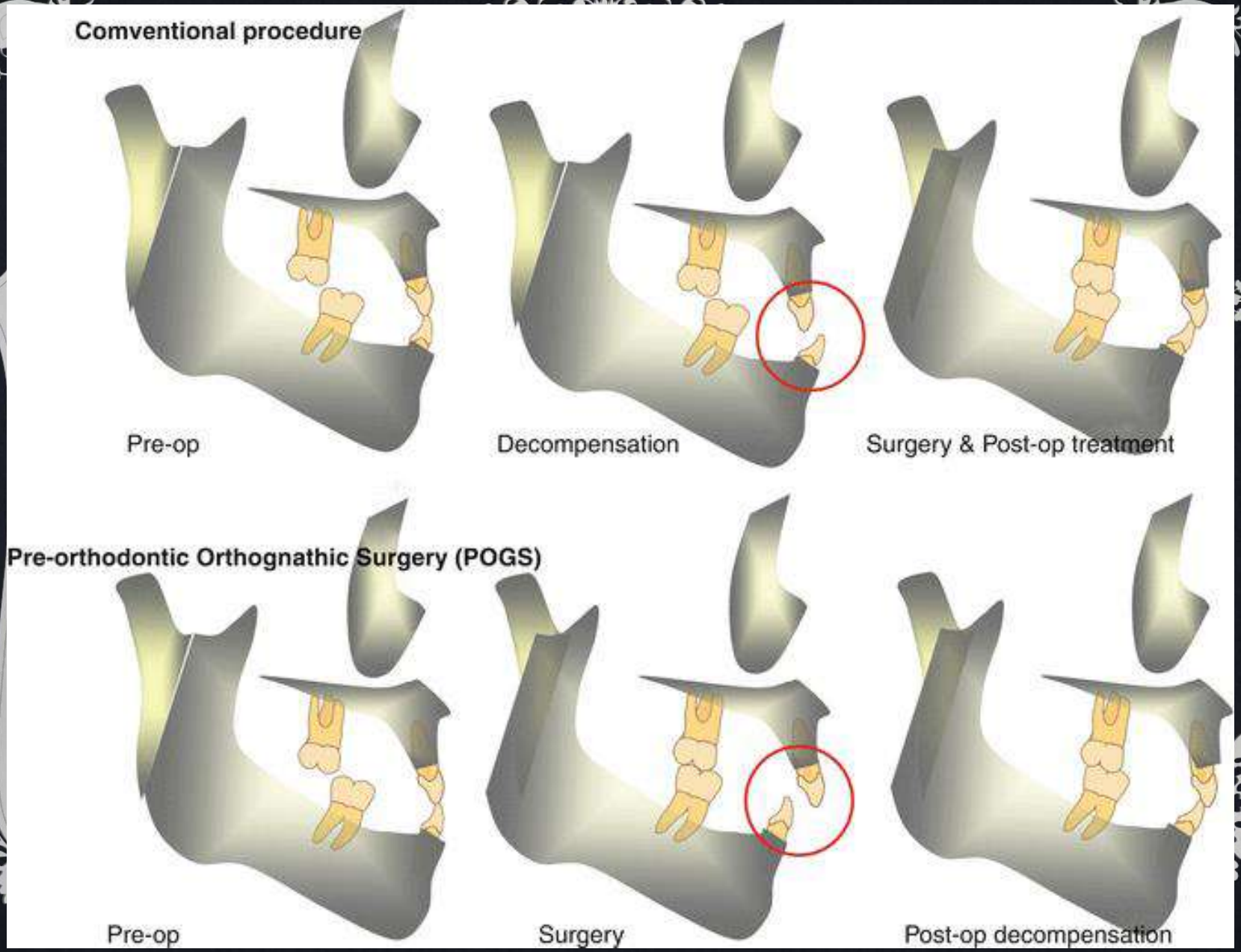
STUDY MODEL

- To get the definite idea about the extent of bone / arch advancement or reduction required in the surgery.
- To get a post-operative relationship of the jaws, dentition and occlusion.
- To decide about the post-surgical orthodontic treatment.
- As a vehicle for fabrication of splints for stabilization after surgery.



PRESURGICAL ORTHODONTICS

1. Alignment of dental arch (relieve of crowding, rotation and occlusal interefences)
2. Levelling of occlusal plane
3. Decompensation of incisors and buccal segment
4. Arch coordination: correct transverse arch width discrepancies.
5. Means of fixation to facilitate surgical correction


Comventional procedure
Pre-op
Decompensation
Surgery & Post-op treatment
Pre-orthodontic Orthognathic Surgery (POGS)
Pre-op
Surgery
Post-op decompensation

SURGICAL PROCEDURES

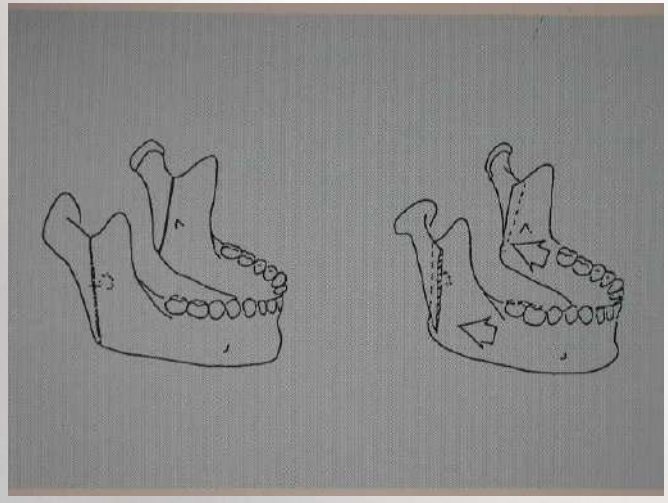
- Mandibular procedures
- Maxillary procedures
- combination

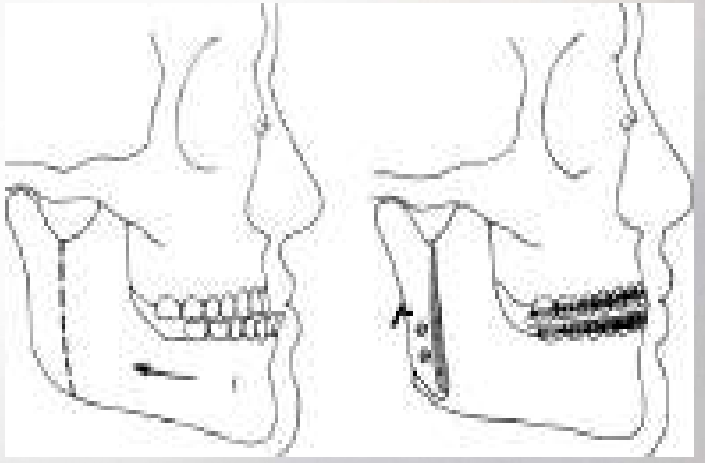
MANDIBULAR PROCEDURES

- Mandibular ramus surgery → sagittal split
  vertical oblique
  inverted L
- Mandibular body surgery
- Mandibular subapical procedures
- Genioplasty

VERTICAL SUBSIGMOID OSTEOTOMY

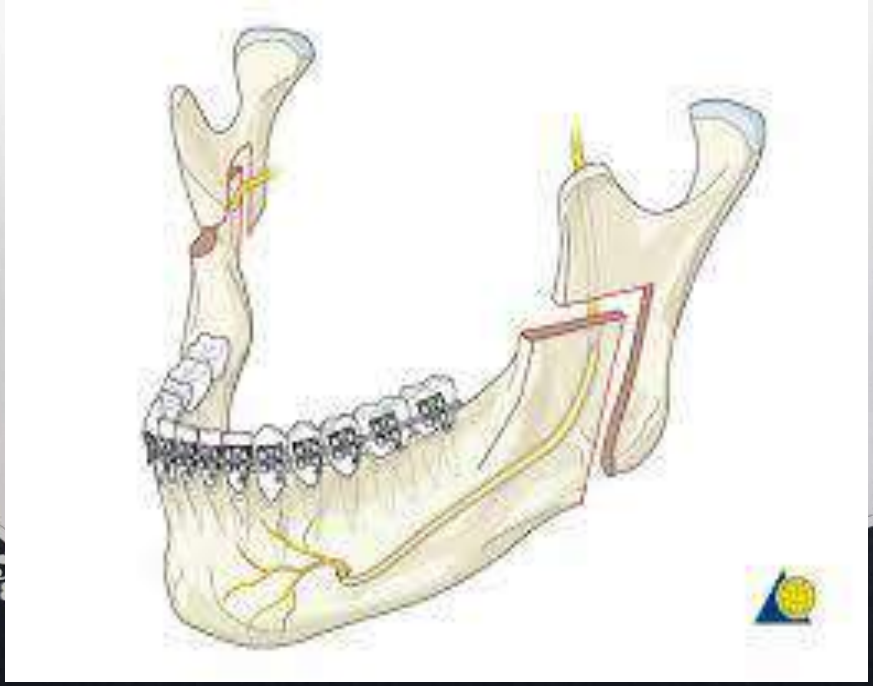
–**Used for mand. Prognathism**.





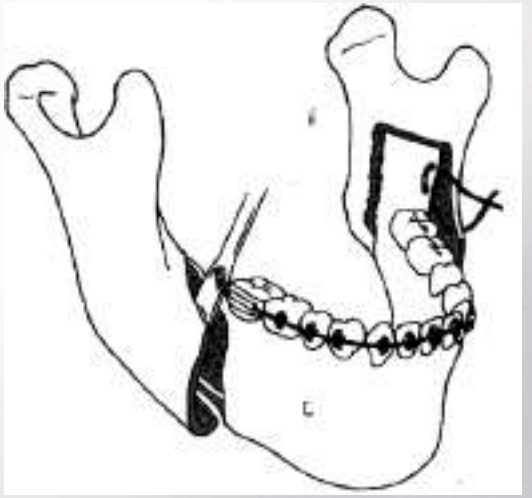
INVERTED L-OSTEOTOMY

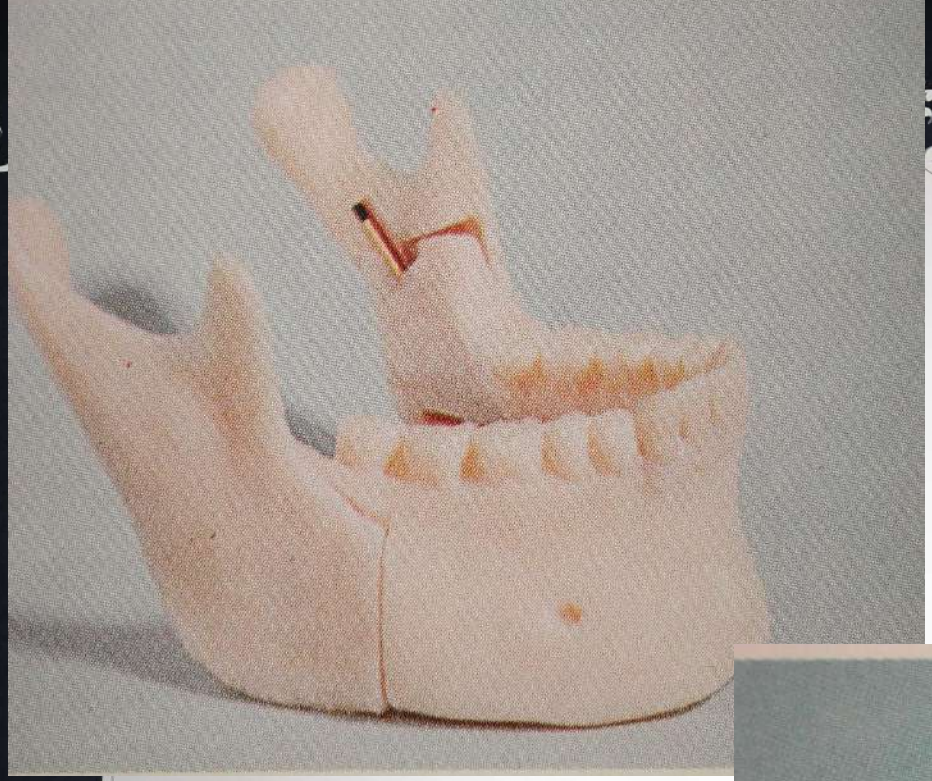
- Basically used for mandibular prognathism correction

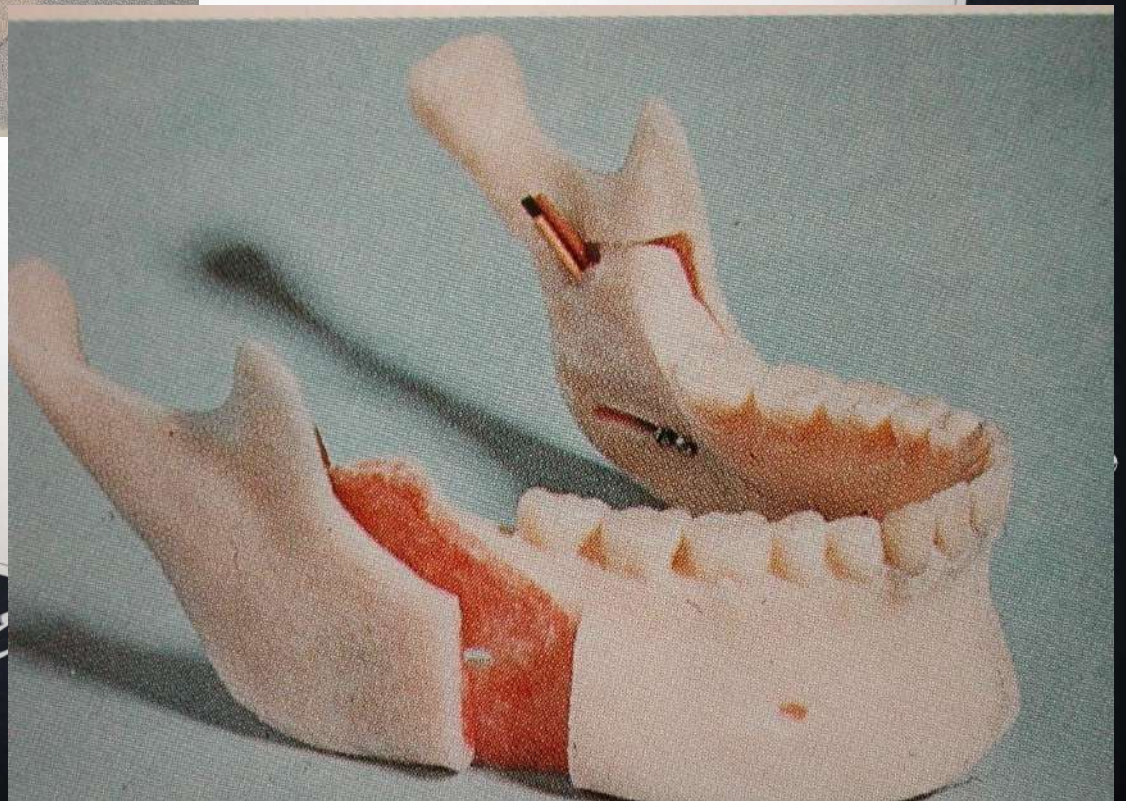


SAGITTAL SPLIT OSTEOTOMY

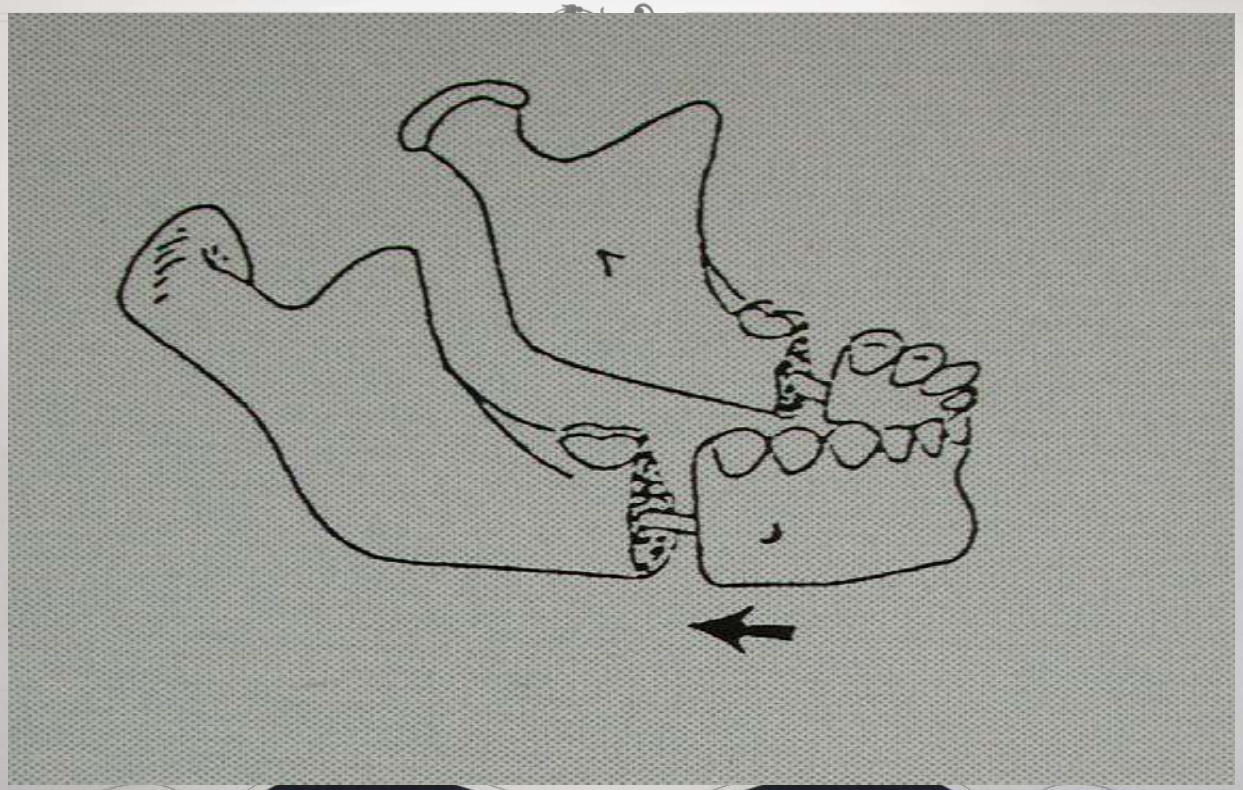
- Used to advance OR push back the mandible OR to correct asymmetry.



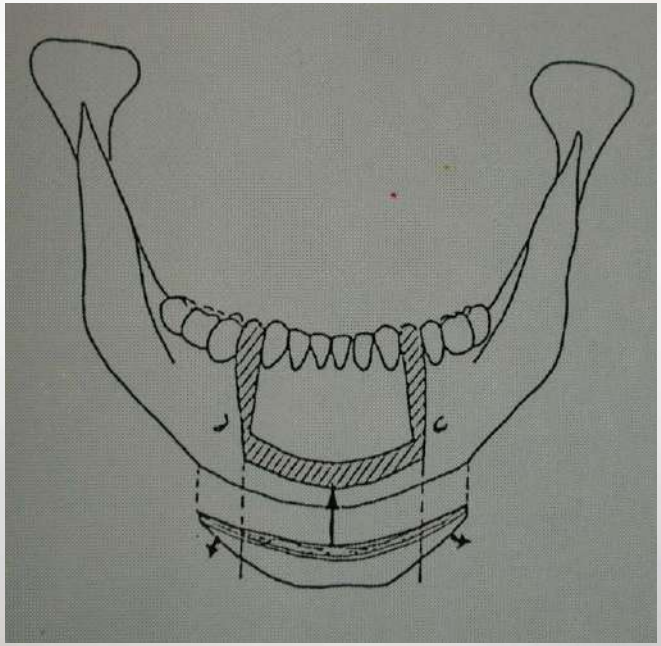




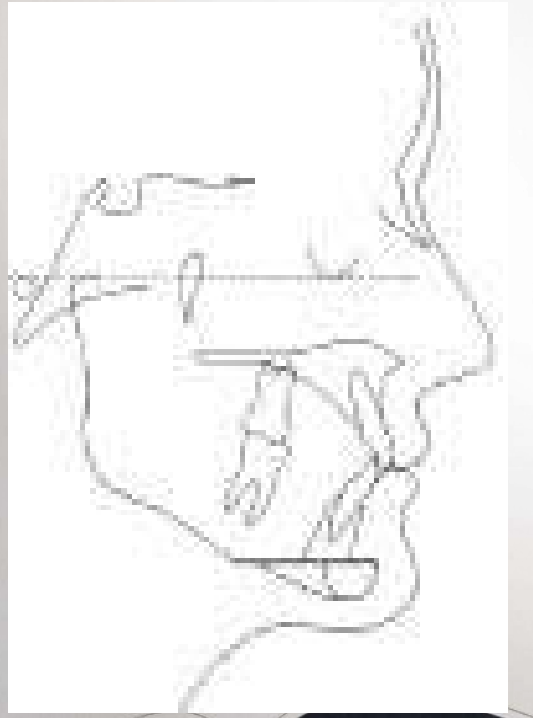
BODY OSTEOTOMY

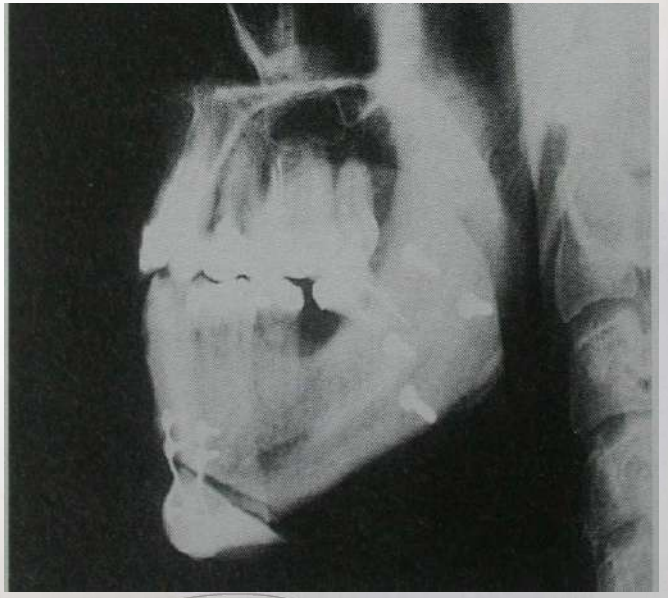


SUBAPICAL OSTEOTOMY



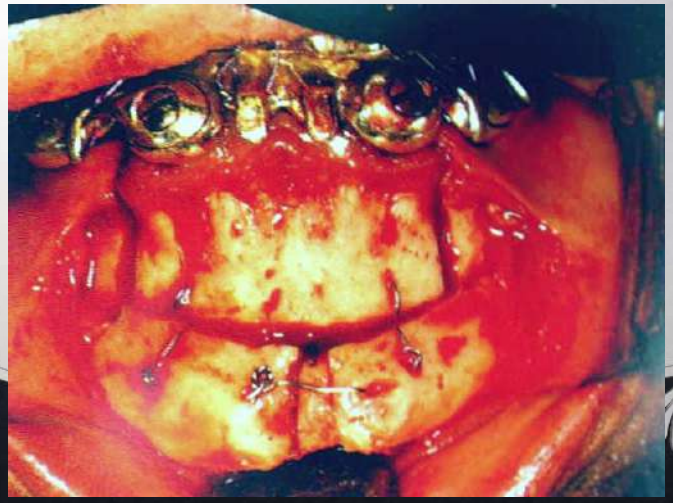
GENIOPLASTY





FIXATION

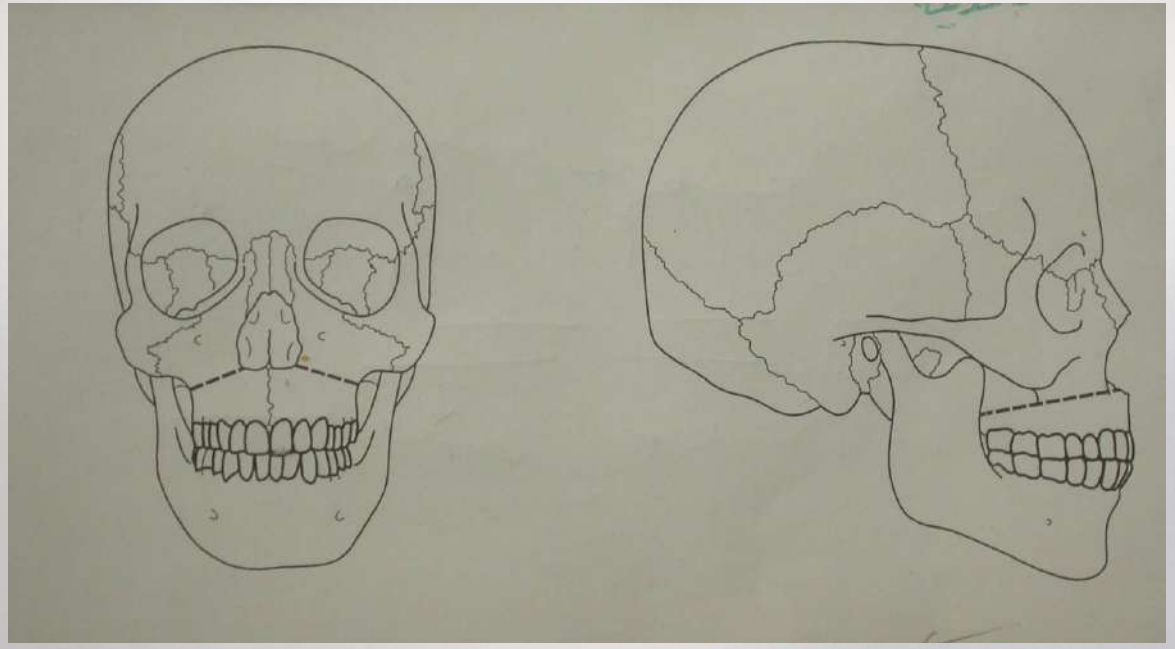
- Wires, plates and screws are USED TO FIX BONE POSITIONS.



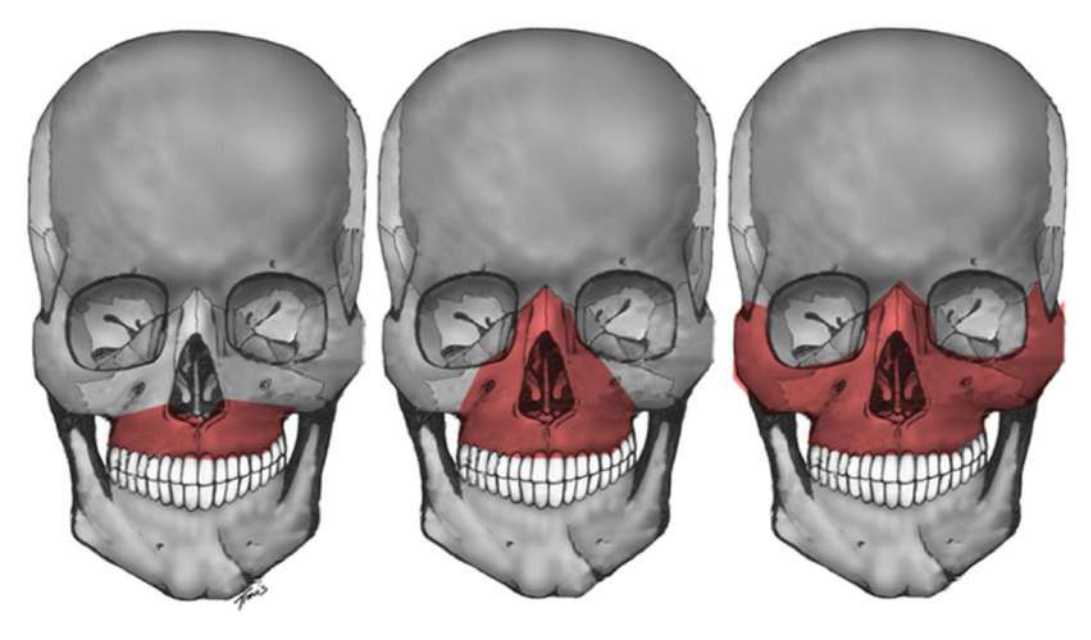
MAXILLARY PROCEDURES

- Lefort 1 (most widely used technique)
- Segmental maxillary procedures
- Lefort II
- Lefort III

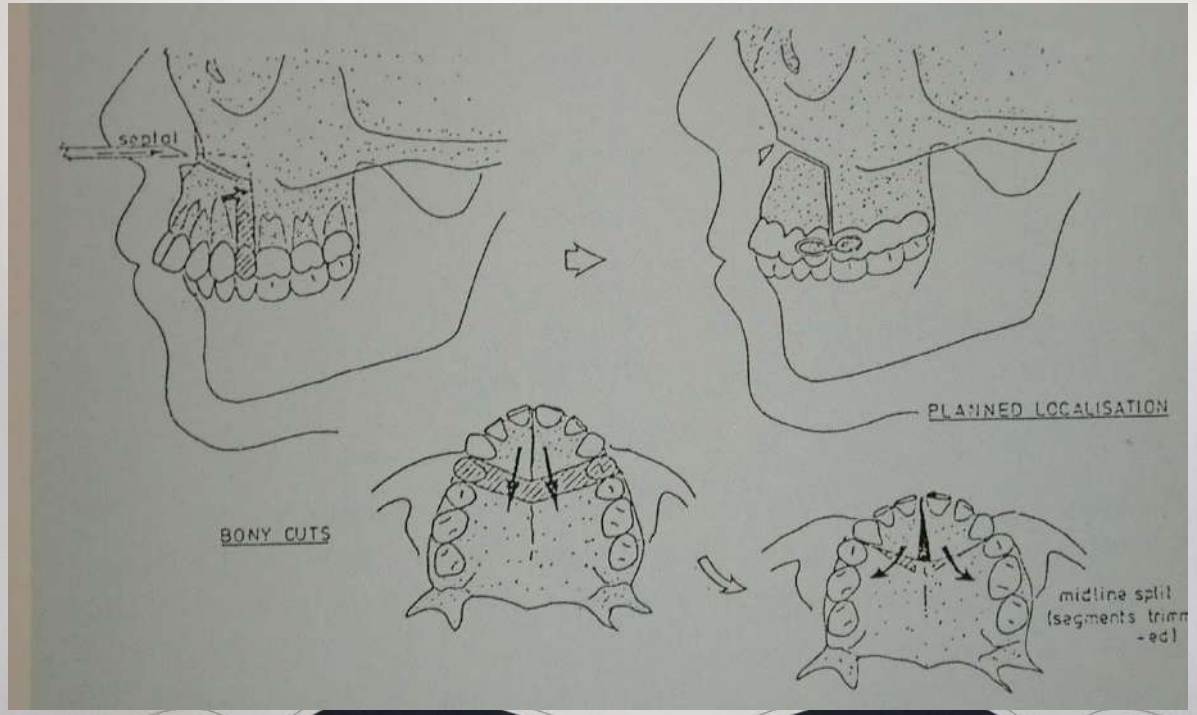
LE FORT I



LE FORT I, II, III



WASSMUND TECHNIQUE

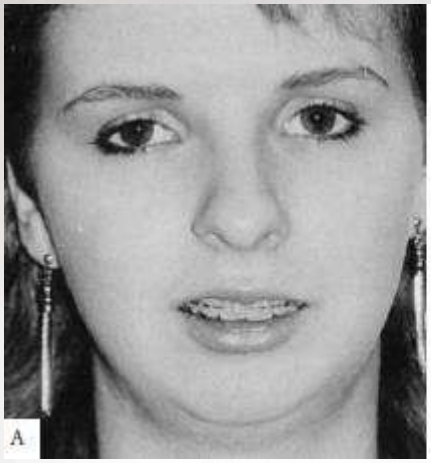


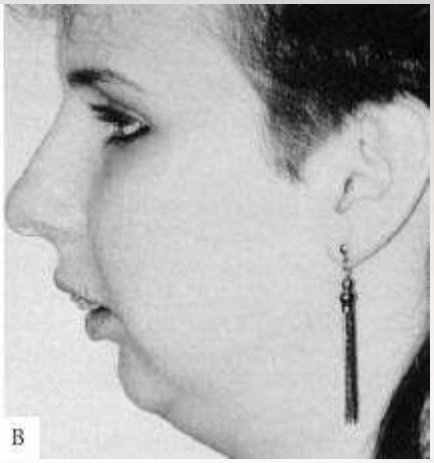
TREATMENT OF DENTOFACIAL ABNORMALITIES

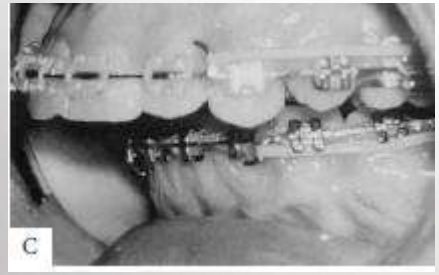
- Mandibular deficiency
- Mandibular excess
- Maxillary deficiency
- Maxillary excess

MANDIBULAR DEFICIENCY

- BSSO to advance the mandible forward ± Le Fort I impaction of maxilla if there is no satisfactory lip-incisor relation.


A


B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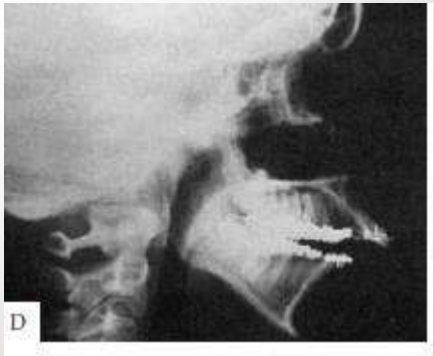
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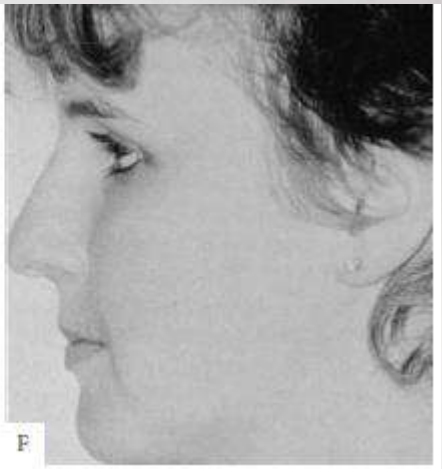
E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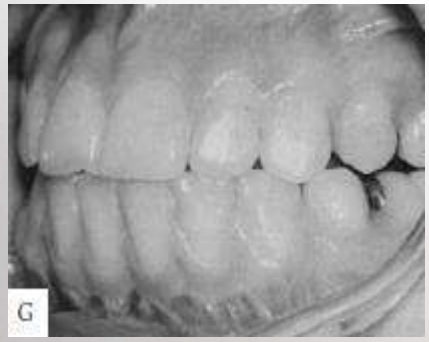
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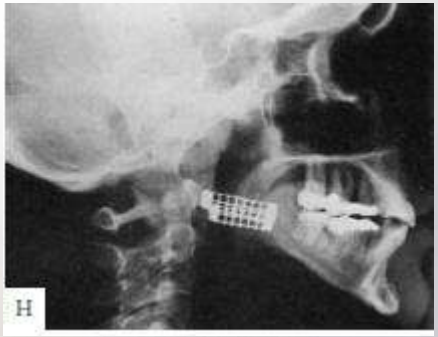
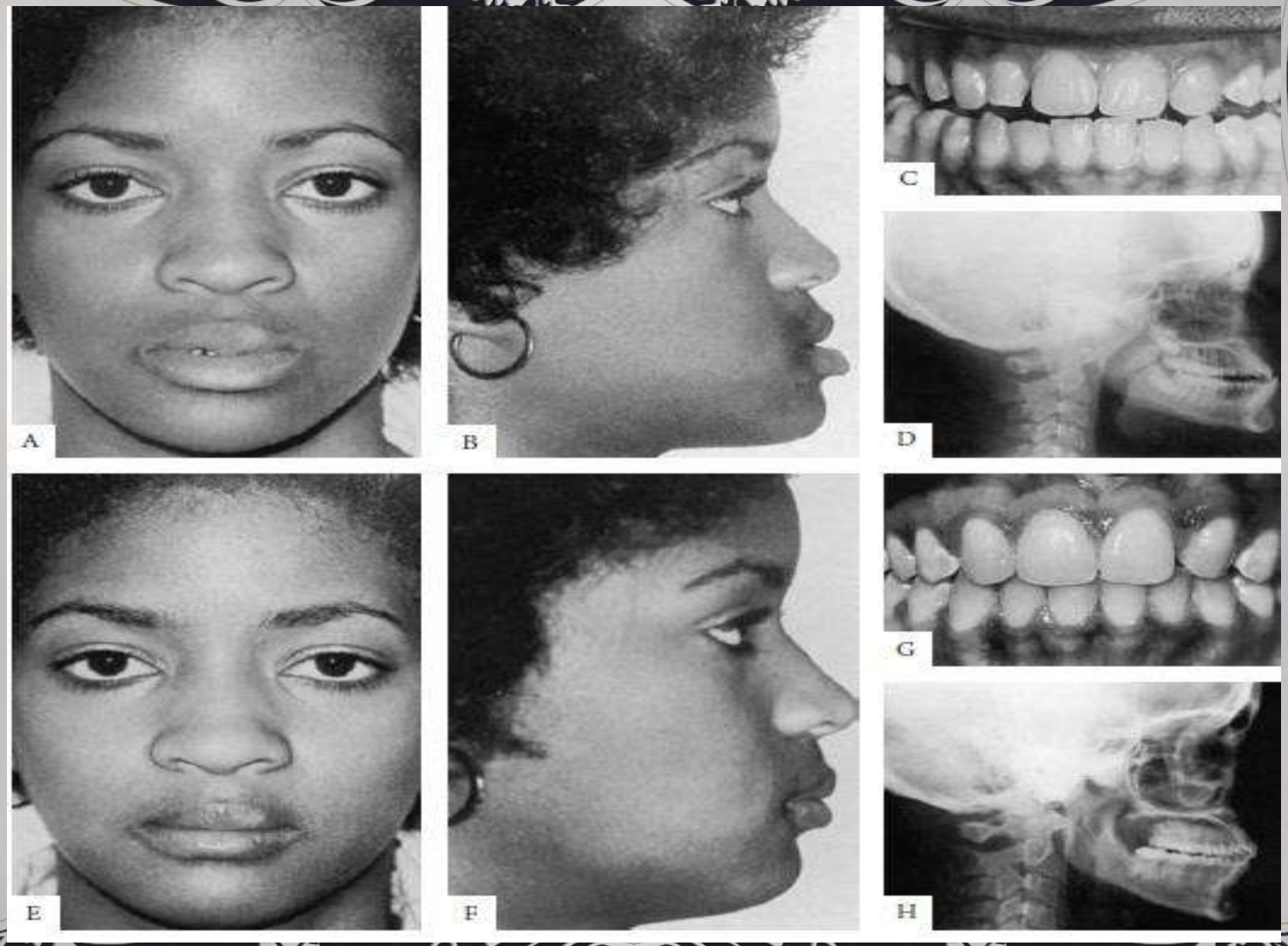

H

MANDIBULAR EXCESS

- Mild case (overjet < 8 mm): Bilateral sagittal split osteotomy (BSSO) or Vertical oblique osteotomy to retrude the mandible backward.
- Severe case : BSSO to retrude the mandible + Lefort I to advance the maxilla


A
B
C
D
E
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G
H

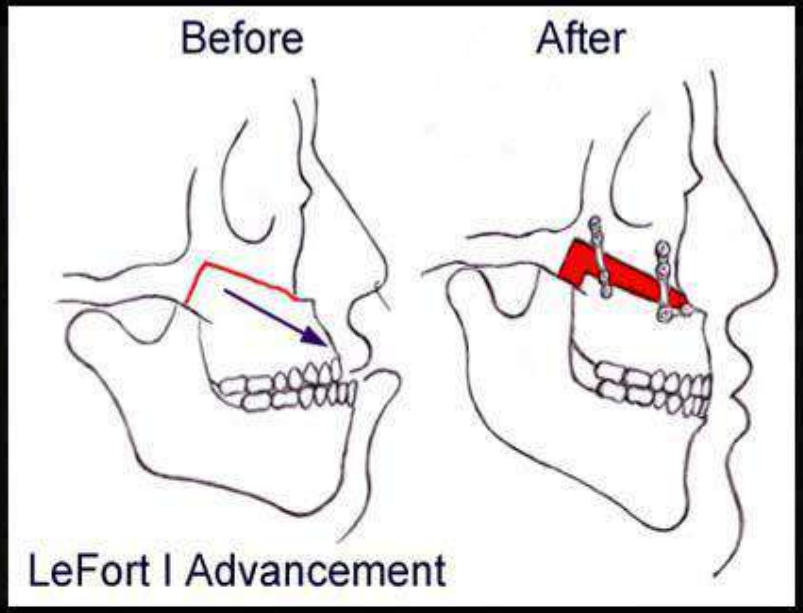
MAXILLARY DEFICIENCY

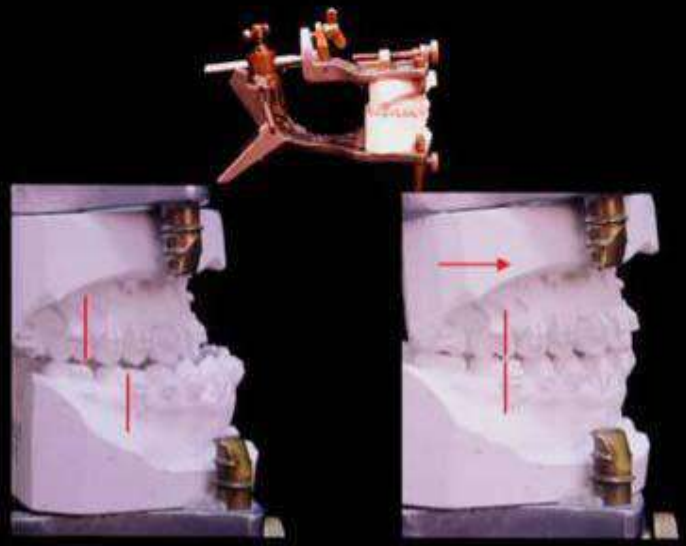
- In the case of A-P deficiency: Le Fort I ADVANCEMENT

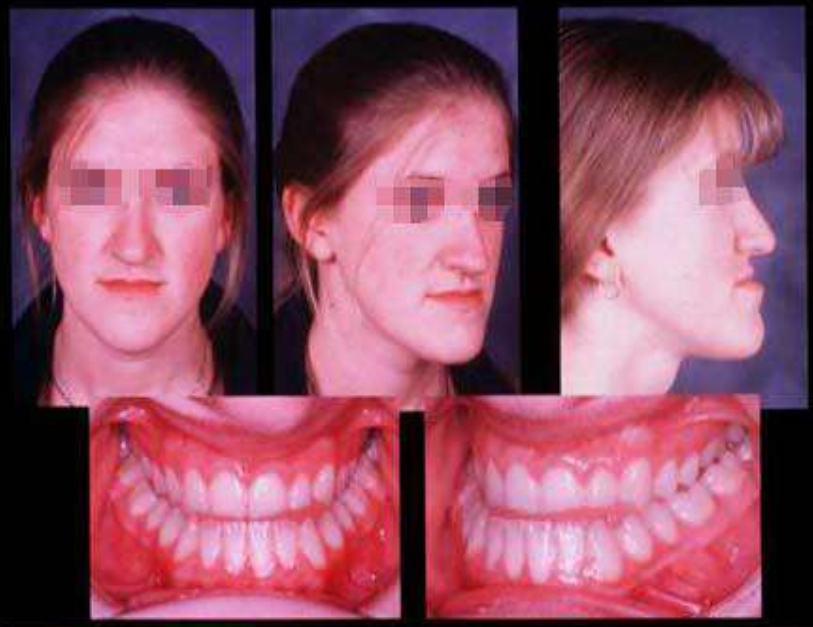
If O.J. > 9 mm bimaxillary surgery is required (Le Fort I + BSSO)

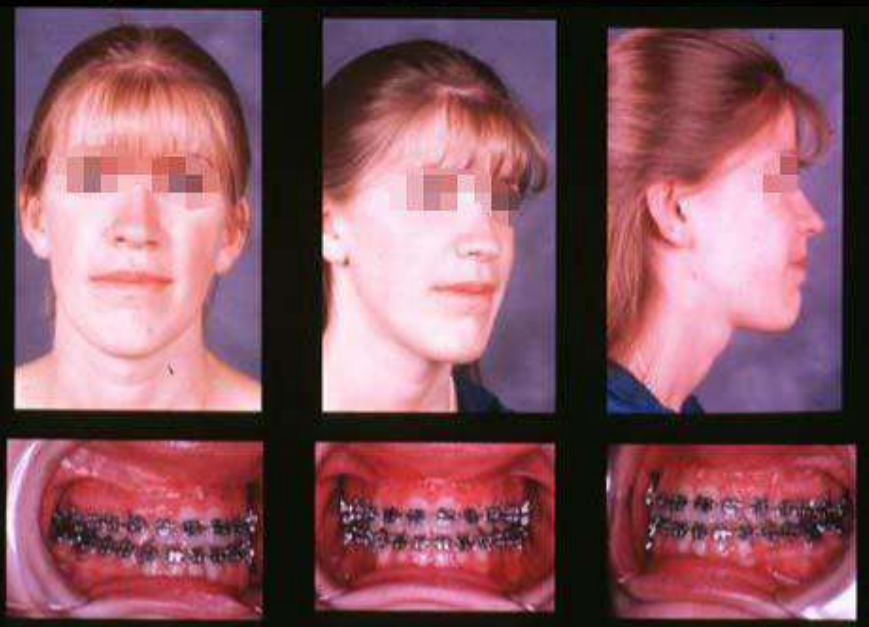
- In the case of vertical deficiency, Le Fort I with reposition of maxilla inferiorly
- In case of midface deficiency (infraorbital rim and malar hypoplasia), Le Fort III or modified Le Fort II osteotomy is necessary.

Midfacial Deficiency









MAXILLARY EXCESS

- growing child : require orthodontic therapy (headgear and multibanded fixed appliance).
- In non-growing patient, treatment is either by anterior maxillary segmental osteotomy (Wassmund) to setback canine-incisor segment after extraction of upper 1st premolars or by Le Fort I setback of entire maxilla .

QUESTION

- What is the most appropriate midline landmarks with which to align the dentition when correcting asymmetric maxillary midline with lefort I ??
- Glabella
- Anterior nasal spine
- Philtrum
- Dental midline

Pigmented Lesions of the Oral Mucosa

Oral and perioral pigmentation may be physiologic or pathologic in origin. Physiologic pigmentation is typically brown in appearance. However, in the course of disease, the oral mucosa and perioral tissues can assume a variety of discolorations, including brown, blue, gray, and black. Such color changes are often a tributed to the deposition, production, or increased accumulation of various endogenous or exogenous pigmented substances. However, although an area may appear pigmented, the discoloration may not be related to actual pigment but rather to the deposition or accumulation of organic or inorganic substances, including various metals and drug metabolites.

Endogenous Pigmentation

Melanin is found universally in nature. Melanin is the pigment derivative of tyrosine and is synthesized by melanocytes, which typically reside in the basal cell layer of the epithelium. Investigations into normal melanocyte homeostasis have yielded the discovery that keratinocytes actually control melanocytic growth. Yet the mechanisms by which melanocytes are stimulated to undergo cell division remain poorly understood. Their presence in the skin is thought to protect against the damaging effects of actinic irradiation, as well as to act as scavengers in protecting against various cytotoxic intermediates. The role of melanocytes in oral epithelium is not clear.

Overproduction of melanin may be caused by a variety of mechanisms, the most common of which is related to increased sun exposure. However, intraorally, hyperpigmentation is more commonly a consequence of physiologic or idiopathic sources, neoplasia, medication or oral contraceptive use, high serum concentrations of pituitary adrenocorticotropic hormone (ACTH), postinflammatory changes, and genetic or autoimmune disease. Thus, the presence or absence of other systemic signs and symptoms, including cutaneous hyperpigmentation, is of great importance from the standpoint of diagnosing the cause of oral pigmentation. However, if the etiology of the pigment cannot be ascertained, a tissue biopsy is warranted for definitive diagnosis and is especially critical for the diagnosis of focal pigmentation since malignant melanoma can present in a variety of different configurations.

In addition to biopsy and histologic study, various laboratory and clinical tests, including diascopy, radiography, and blood tests, may be necessary for definitive diagnosis of oral pigmentation. Dermascopy, also known as epiluminescence microscopy, is another increasingly employed clinical test that can be useful in the diagnosis of melanocytic lesions.

Focal Melanocytic Pigmentation:

Freckle/Ephelis

The cutaneous freckle, or ephelis, is a commonly occurring, asymptomatic, small (1–3 mm), well-circumscribed, tan- or brown-colored macule that is often seen on the sun-exposed regions of the facial and perioral skin. Ephelides are most commonly observed in light-skinned individuals and are quite prevalent in red- or light blond–haired individuals. Although the pigmentation itself is focal in nature, most patients have multiple freckles. Freckles are thought to be developmental in origin. Ephelides are usually more abundant in number and darker in intensity during childhood and adolescence. Freckles tend to become darker during periods of prolonged sun exposure (spring, summer) and less intense during the autumn and winter months. Yet the increase in pigmentation is solely related to an increase in melanin production without a concomitant increase in the number of melanocytes. With increasing age, the number of ephelides and color intensity tends to diminish. In general, no therapeutic intervention is required.

Oral/Labial Melanotic Macule

The melanotic macule is a unique, benign, pigmented lesion that has no known dermal counterpart. Melanotic macules are the most common oral lesions of melanocytic origin. Although the etiology remains elusive, trauma has been postulated to play a role. Sun exposure is not a precipitating factor. Melanotic macules develop more frequently in females, usually in the lower lip (labial melanotic macule) and gingiva. However, any mucosal site may be affected. Although the lesion may develop at any age, it generally tends to present in adulthood.

Congenital melanotic macules have also been described occurring primarily in the tongue. Overall, melanotic macules tend to be small (<1 cm), well circumscribed, oval or irregular in outline and often uniformly pigmented. Once the lesion reaches a certain size, it does not tend to enlarge further. Unlike an ephelis, a melanotic macule does not become darker with continued sun exposure. Overall, the oral melanotic macule is a relatively innocuous lesion, does not represent a melanocytic proliferation, and does not generally recur following surgical removal.

Oral Melanoacanthoma

Oral melanoacanthoma is another unusual, benign, melanocytic lesion that is unique to the mucosal tissues. Oral melanoacanthoma is an innocuous melanocytic lesion that may spontaneously resolve, with or without surgical intervention. Most patients report a rapid onset; and acute trauma or a history of chronic irritation usually precedes the development of the lesion. A biopsy is always warranted to confirm the diagnosis, but, once established, no further treatment is required.

Oral melanoacanthoma usually presents as a rapidly enlarging, ill-defined, darkly pigmented macular or plaque-like lesion, and most develop in black females.20,21 Although lesions may present over a wide age range, the majority occur between the third and fourth decades of life. In rare instances, multiple lesions may present simultaneously.

Oral melanoacanthomas are typically asymptomatic, although pain has been reported. Although any mucosal surface may be involved, the buccal mucosa is the most common site of occurrence. The size of the lesion is variable, ranging from small and localized to large, diffuse areas of involvement, measuring several centimeters in diameter. The borders are typically irregular in appearance, and the pigmentation may or may not be uniform.

Cutaneous melanoacanthoma represents a pigmented variant of seborrheic keratosis and typically occurs in older Caucasian patients. Dermatosis papulosa nigra is a relatively common facial condition that typically manifests in older black patients, often female, and represents multiple pigmented seborrheic keratosis. These small papules are often identified in the malar and preauricular regions of the face.

Diagnosis

The clinical presentation, in association with the history, may be disconcerting and should lead the clinician to consider malignant melanoma in the differential diagnosis.

Melanocytic Nevus

Melanocytic nevi include a diverse group of clinically and/or microscopically distinct lesions. Unlike ephelides and melanotic macules, which result from an increase in melanin pigment synthesis, nevi arise as a consequence of melanocytic growth and proliferation. In the oral cavity, the intramucosal nevus is most frequently observed, followed by the common blue nevus. In general, both genetic and environmental factors are thought to play a role in nevogenesis. The effect of sun exposure on the development of cutaneous nevi is well recognized. However, there are also age- and location-dependent differences in the presentation, number, and distribution of nevi. Although most melanocytic nevi are acquired, some may present as congenital lesions (including in the oral cavity).

Familial atypical multiple mole and melanoma syndrome is characterized by the formation of histologically atypical nevi, epithelioid blue nevus may be associated with the Carney complex, markedly increased numbers of common nevi are characteristic in patients with Turner's syndrome.

Cutaneous nevi are a common occurrence. The average Caucasian adult patient may have several nevi; some individuals may have dozens. The total number of nevi tends to be higher in males than females. In contrast, oral melanocytic nevi are rare, typically present as solitary lesions, and may be more common in females.

Oral melanocytic nevi have no distinguishing clinical characteristics. Lesions are usually asymptomatic and often present as a small (<1 cm), solitary, brown or blue, well circumscribed nodule or macule. Up to 15% of oral nevi may not exhibit any evidence of clinical pigmentation. Once the lesion reaches a given size, its growth tends to cease and may remain static indefinitely.

Oral nevi may develop at any age; however, most are identified in patients over the age of 30. The hard palate represents the most common site, followed by the buccal and labial mucosae and gingiva.

The nevus cells initially maintain their localization to the basal layer, residing at the junction of the epithelium and the basement membrane and underlying connective tissue .These junctional nevi are usually small (<5 mm), macular or nonpalp ble, and tan to brown in appearance. Over time, the clustered melanocytes are thought to proliferate down into the connective tissue, often in the form of variably sized nests of relatively small, rounded cells. Nonetheless, some nevus cells are still seen at the mucosalsubmucosal junction. Such nevi often assume a dome-shaped appearance and are referred to as compound nevi. As the lesion further matures, the nevus cells completely lose their association with the epithelial layer and become confined to the submucosal tissue, often with an associated decrease in the amount of pigmentation. At this point, the lesion is given the designation of intramucosal nevus and, clinically, may appear brown or tan or even resemble the color of the surrounding mucosa.

The 'common' blue nevus, which is the most frequent histologic variant seen in the oral cavity, is characterized by an intramucosal proliferation of pigment-laden, spindle-shaped melanocytes. The blue nevus is described as such because the melanocytes may reside deep in the connective tissue and the overlying blood vessels often dampen the brown coloration of melanin, which may yield a blue tint. Biopsy is necessary for diagnostic confirmation of an oral melanocytic nevus since the clinical diagnosis includes a variety of other focally pigmented lesions, including malignant melanoma.

Malignant Melanoma

Malignant melanoma is the least common but most deadly of all primary skin cancers. Similar to other malignancies, extrinsic and intrinsic factors play a role in the

pathogenesis of melanoma. A history of multiple episodes of acute sun exposure, especially at a young age; immunosuppression; the presence of multiple cutaneous nevi; and a family history of melanoma are all known risk factors for the development of cutaneous melanoma.

Cutaneous melanoma is most common among white populations that live in the sun belt regions of the world. However, mortality rates are higher in blacks and Hispanics. The incidence is increasing in patients, especially males, over the age of 45. The incidence is decreasing in patients under the age of 40. Overall, there is a male predilection, but melanoma is one of the most commonly occurring cancers in women of childbearing age.

The clinical characteristics of cutaneous melanoma are best described by the ABCDE criteria: asymmetry, irregular borders, and color variegation, diameter greater than 6 mm, and evolution or surface elevation. These criteria are very useful (although not absolute) in differentiating cutaneous melanoma from other focally, pigmented melanocytic lesions.

Primary mucosal melanomas comprise less than 1% of all melanomas. The majority develop in the head and neck, most in the sinonasal tract and oral cavity. The prevalence of oral melanoma appears to be higher among black-skinned and Japanese people than among other populations. The tumor presents more frequently in males than females.

Oral melanoma may develop at any age, but most present over the age of 50. Any mucosal site may be affected; however, the palate represents the single most common site of involvement. The maxillary gingiva is the second most frequent site. Oral melanomas have no distinctive clinical appearance. They may be macular, plaque-like or mass forming, well-circumscribed or irregular and exhibit focal or diffuse areas of brown, blue, or black pigmentation. Up to one-third of oral melanomas may exhibit little or no clinical evidence of pigmentation (amelanosis).In some cases, oral melanomas may present with what appear to be multifocal areas of pigmentation. This phenomenon is often explained by the fact that some tumors may exhibit both melanotic and amelanotic areas.

Additional signs and symptoms that may be associated with oral melanoma are nonspecific and similar to those observed with other malignancies. Ulceration, pain, tooth mobility or spontaneous exfoliation, root resorption, bone loss, and paresthesia/anesthesia may be evident. However, in some patients, the tumors may be completely asymptomatic. Thus, the clinical differential diagnosis may be quite extensive. It is for this reason that a biopsy of any persistent solitary pigmented lesion is always warranted. Oral mucosal malignant melanoma is associated with a very poor prognosis. Studies have demonstrated 5-year survival rates of 15 40%.Regional lymphatic metastases are frequently identified and contribute to the

poor survival rates. Less than 10% of patients with distant metastases survive after 5 years.

Multifocal/Diffuse Pigmentation

Physiologic Pigmentation

Physiologic pigmentation is the most common source of multifocal or diffuse oral mucosal pigmentation. Dark-complexioned individuals, including blacks, Asians, and South-Americans, frequently show patchy to generalized hyperpigmentation of the oral mucosal tissues. Although in many patients, the pigment is restricted to the gingiva, melanosis of other mucosal surfaces is not uncommon .The pigment is often observed in childhood and usually does not develop de novo in the adult. If there is a sudden or gradual onset of diffuse mucosal pigmentation in adulthood, even in darker-skinned patients, other sources for the melanosis should be given consideration.

Drug-Induced Melanosis

Medications may induce a variety of different forms of mucocutaneous pigmentation, including melanosis. The chief drugs implicated in drug-induced melanosis are the antimalarials, including chloroquine, hydroxychloroquine, quinacrine, and others. Other common classes of medications that induce melanosis include the phenothiazines, such as chlorpromazine , oral contraceptives, and cytotoxic medications such as cyclophosphamide and busulfan .

Intraorally, the pigment can be diffuse yet localized to one mucosal surface, often the hard palate, or it can be multifocal and involve multiple surfaces. Some drugs may even be associated with a specific pattern of pigmentation. Much like other forms of diffuse pigmentation, the lesions are flat and without any evidence of nodularity or swelling. Sun exposure may exacerbate cutaneous drug-induced pigmentation.

Smoker's Melanosis

Diffuse melanosis of the anterior facial maxillary and mandibular gingivae, buccal mucosa, lateral tongue, palate, and floor of the mouth is occasionally seen among cigarette smokers. Most smokers (including heavy smokers) usually fail to show such changes. However, it is probable that in certain individuals, melanin synthesis is stimulated by tobacco smoke products. Indeed, among dark-skinned individuals who normally exhibit physiologic pigmentation, smoking stimulates a further increase in oral pigmentation. The pigmented areas are brown, flat, and irregular; some are even geographic or map-like in configuration. Alcohol has also been associated with increased oral pigmentation. In alcoholics, the posterior regions of

the mouth, including the soft palate, tend to be more frequently pigmented than other areas. It has been suggested that alcoholic melanosis may be associated with a higher risk of cancers of the upper aerodigestive tract.
Diffuse or patchy melanotic pigmentation is also characteristically associated with oral submucous fibrosis. Unlike smoker's melanosis, oral submucous fibrosis is a preneoplastic condition caused by habitual chewing of areca (betel) nut.

Post inflammatory (Inflammatory) Hyperpigmentation

Postinflammatory hyperpigmentation is a well-recognized phenomenon that tends to develop more commonly in darkcomplexioned individuals. Most cases present as either focal or diffuse pigmentation in areas that were subjected to previous injury or inflammation. The acne-prone face is a relatively common site for this phenomenon. Although unusual, postinflammatory pigmentation may also develop in the oral cavity. In rare cases, the mucosa overlying a nonmelanocytic malignancy may become pigmented. Oral pigmentation has also been described in patients with lichen planus (lichen planus pigmentosus).

Melasma (Chloasma)

Melasma is a relatively common, acquired symmetric melanosis that typically develops on sun-exposed areas of the skin and frequently on the face. The forehead, cheeks, upper lips, and chin are the most commonly affected areas. There is a distinct female predilection, and most cases arise in darker-skinned individuals. Unlike other forms of diffuse melanosis, melasma tends to evolve rather rapidly over a period of a few weeks. Sun exposure tends to be an exacerbating, if not precipitating, event. The term melasma is most appropriately used to describe the pigmentary changes associated with pregnancy or ingestion of contraceptive hormones. Both pregnancy and use of oral contraceptives have also been associated with oral mucosal melanosis. Rare cases of idiopathic melasma have also been described in females and, much less commonly, males. Melasma may spontaneously resolve after parturition, cessation of the exogenous hormones, or regulation of endogenous sex-hormone levels.

Melanosis Associated with systemic or Genetic Disease:

Hypoadrenocorticism (Adrenal Insufficiency,Addison's Disease)

Hypoadrenocorticism is a potentially life-threatening disease, as much for its systemic complications as it's under diagnosis. A variety of etiologies may precipitate adrenal insufficiency. In adults, autoimmune disease represents one of the most

common causes. However, infectious agents, neoplasia, trauma, certain medications, and iatrogenic causes may lead to adrenal destruction or an impairment of endogenous steroid production. In rare cases, adrenal insufficiency may also be a consequence of genetic disease. Regardless of etiology, the end result is essentially the same, that is, a decrease in endogenous corticosteroid levels. As steroid levels decrease, there is a compensatory activation of ACTH secretion from the anterior pituitary gland. ACTH then acts on the adrenal cortex to stimulate steroid production and ACTH secretion stops. If low steroid levels persist, there is a loss of feedback inhibition, resulting in persistent secretion of ACTH into the serum. Concurrently, the serum levels of a-melanocyte-stimulating hormone (a-MSH) also increase.

Weakness, poorly defined fatigue, and depression are some of the typical presenting signs of the illness. However, in some patients, the first sign of disease may be mucocutaneous hyperpigmentation. Generalized bronzing of the skin and diffuse but patchy melanosis of the oral mucosa are hallmarks of hypoadrenocorticism. Any oral surface may be affected. In some patients, oral melanosis may be the first manifestation of their adrenal disease. Diffuse hyperpigmentation is more commonly associated with chronic rather than acute-onset disease.

The diagnosis of oral addisonian pigmentation requires a clinicopathologic correlation. Endocrinopathic disease should be suspected whenever oral melanosis is accompanied by cutaneous bronzing. Treatment consists of exogenous steroid replacement therapy. With appropriate therapy, the pigmentation will eventually resolve.

Cushing's Syndrome/Cushing's Disease

Cushing's syndrome develops as a consequence of prolonged exposure to relatively high concentrations of endogenous or exogenous corticosteroids. Most cases are iatrogenic in origin and associated with poorly controlled or unmonitored use of topical or systemic steroids. Cushing's syndrome may also arise as a result of various endogenous etiologies, including an activating pituitary tumor (Cushing's disease) and a primary, activating, adrenal pathology (hyperadrenocorticism), as well as ectopic secretion of corticosteroids, ACTH, or corticotropin-releasing hormone by various neoplasms.

Overall, Cushing's syndrome is more prevalent in female patients. However, prepubertal onset is more commonly seen in boys. Apart from the wide array of systemic complications, including weight gain and the characteristic "moon facies," diffuse mucocutaneous pigmentation may be seen in a subset of patients, specifically those whose pathology is associated with increased ACTH secretion. Thus, in most cases, the affected patients have a primary pituitary neoplasm. The

pattern of oral pigmentation is essentially identical to that seen in patients with adrenal insufficiency.
Serum steroid and ACTH determinations will aid in the diagnosis, and the pigment often resolves following appropriate surgical, radiation, or medicinal therapy for the specific source of the endocrinopathy.

Hyperthyroidism (Graves'Disease)

Melanosis is a common consequence of hyperthyroidism (Graves' disease), especially in dark-skinned individuals. The pigmentation tends to resolve following treatment of the thyroid abnormality. The mechanism by which excessive thyroid activity stimulates melanin synthesis remains unclear.

Peutz-Jeghers Syndrome

Peutz-Jeghers syndrome is an autosomal dominant disease. Clinical manifestations include intestinal polyposis, cancer susceptibility, and multiple, small, pigmented macules of the lips, perioral skin, hands, and feet. The macules may resemble ephelides, usually measuring <0.5 cm in diameter. However, the intensity of the macular pigment is not influenced by sun exposure. Although uncommon, similar-appearing lesions may also develop on the anterior tongue and buccal and labial mucosae. The lip and perioral pigmentation is highly distinctive, although not pathognomonic for this disease.

Café au Lait Pigmentation

Solitary, idiopathic café au lait ("coffee with milk") spots are occasionally observed in the general population, but multiple café au lait spots are often indicative of an underlying genetic disorder. Café au lait pigmentation may be identified in a number of different genetic diseases, including neurofibromatosis type I, McCune-Albright syndrome, and Noonan's syndrome. café au lait spots typically present as tan- or brown-colored, irregularly shaped macules of variable size. They may occur anywhere on the skin. Although unusual, examples of similar-appearing oral macular pigmentation have been described in some patients.

DEPIGMENTATION

Vitiligo

Common, acquired, autoimmune disease that is associated with hypomelanosis. Although the etiology and mechanisms remain unknown, the end result is a destruction of the melanocytes. In most cases, vitiligo is characterized by bilateral, symmetric areas of relatively generalized hypomelanosis. The vitiligenous lesions often present as well-circumscribed, round, oval or elongated, pale or white-colored macules that may coalesce into larger areas of diffuse depigmentation. As

the disease progresses, additional areas of involvement may become apparent. Topical corticosteroids and topical or, more commonly, systemic photochemotherapies (psoralen and ultraviolet A exposure) have proven to be effective nonsurgical therapies.

Hemoglobin and Iron- Associated Pigmentation

Ecchymosis

Traumatic ecchymosis is common on the lips and face yet is uncommon in the oral mucosa, except in cases related to blunt-force trauma and oral intubation. Immediately following the traumatic event, erythrocyte extravasation into the submucosa will appear as a bright red macule or as a swelling if a hematoma forms. The lesion will assume a brown coloration within a few days, after the hemoglobin is degraded to hemosiderin. patients taking anticoagulant drugs may present with oral ecchymosis, particularly on the buccal mucosa or tongue, either of which can be traumatized while chewing. Ecchymoses of the oral mucosa may also be encountered in patients with liver cirrhosis, leukemia, and end-stage renal disease undergoing dialysis treatment.

Purpura/Petechiae

Capillary hemorrhages will appear red initially and turn brown in a few days once the extravasated red cells have lysed and have been degraded to hemosiderin. The distinction between purpura and petechiae is essentially semantic and based solely on the size of the focal hemorrhages. Petechiae are typically characterized as being pinpoint or slightly larger than pinpoint and purpura as multiple, small 2 to 4 mm collections of extravasated blood. The same precipitating events can elicit either clinical presentation. Oral purpura/petechiae may develop as a consequence of trauma or viral or systemic disease . Petechiae secondary to platelet deficiencies or aggregation disorders are usually not limited to the oral mucosa but may occur concomitantly on the skin. Viral disease is more commonly associated with oral rather than cutaneous petechiae. In most cases, the petechiae are identified on the soft palate, although any mucosal site may be affected. When trauma is suspected, the patient should be instructed to cease whatever activity may be contributing to the presence of the lesions.

Hemochromatosis

Hemochromatosis is a chronic, progressive disease that is characterized by excessive iron deposition (usually in the form of hemosiderin) in the liver and other organs and tissues. Idiopathic, neonatal, blood transfusion, and heritable forms of this disease are recognized. Complications of hemochromatosis may include liver cirrhosis, diabetes, anemia, heart failure, hypertension, and bronzing of the skin.

Exogenous Pigmentation

Amalgam Tattoo

The single most common source of solitary or focal pigmentation in the oral mucosa is the amalgam tattoo. By definition, these are iatrogenic in origin and typically a consequence of the inadvertent deposition of amalgam restorative material into the submucosal tissue. The lesions are typically small, asymptomatic, macular, and bluish gray or even black in appearance. They may be found on any mucosal surface. However, the gingiva, alveolar mucosa, buccal mucosa, and floor of the mouth represent the most common sites. The lesions are often found in the vicinity of teeth with large amalgam restorations or crowned teeth that probably had amalgams, around the apical region of endodontically treated teeth with retrograde restorations or obturated with silver points, and in areas in and around healed extraction sites. If there is no radiographic evidence of amalgam, the lesion is not in proximity to any restored tooth, or the lesion suddenly appears, a biopsy is necessary. A typical differential diagnosis often includes melanotic macule, nevus, and melanoma.

Graphite Tattoos

Graphite tattoos are an unusual source of focal exogenous pigmentation. They are most commonly seen on the palate and represent traumatic implantation of graphite particles from a pencil. The lesions may be indistinguishable from amalgam tattoos, often presenting as a solitary gray or black macule. Since the traumatic event often occurs in childhood, many patients may not report a history of injury. Thus, a biopsy is often warranted.

Medicinal Metal-Induced Pigmentation

A variety of metallic compounds have been used medicinally for the treatment of various systemic diseases. With the exception of gold therapy (for rheumatoid arthritis), such medicaments are rarely or no longer in use. Gold and colloidal silver have both been associated with diffuse cutaneous pigmentation. Silver may cause a generalized blue-gray discoloration (argyria), whereas gold-induced pigment may appear blue-gray or purple (chrysiasis). In contrast to the systemic therapies, metal salts remain a component of some topical medications and other substances that are used in clinical practice. Examples include silver nitrate and zinc oxide .

Generalized black pigmentation of the tongue has been attributed to the chewing of bismuth subsalicylate tablets, a commonly used antacid. This phenomenon is unlike black hairy tongue , which is associated with elongation of the filiform papillae, hyperkeratosis, and superficial colonization of the tongue by bacteria.

Heavy-Metal Pigmentation

Diffuse oral pigmentation may be associated with ingestion of heavy metals. It remains an occupational and health hazard for some individuals who work in certain industrial plants and for those who live in the environment in and around these types of facilities. Other relatively common environmental sources include paints, old plumbing, and seafood. Lead, mercury, bismuth, and arsenic have all been shown to be deposited in oral tissue if ingested in sufficient quantities or over an extended period of time. These ingested metal salts tend to extravasate from vessels in areas of chronic inflammation. Thus, in the oral cavity, the pigmentation is usually found along the free marginal gingiva, where it often dramatically outlines the gingival cuff. This metallic line usually has a gray to black appearance. In some patients, the oral pigmentation may be the first sign of heavy-metal toxicity.

Additional systemic signs and symptoms of heavy metal poisoning may include behavioral changes, neurologic disorders, intestinal pain, and sialorrhea. Diffuse mucocutaneous melanosis may also be observed in some affected individuals.

Drug-Induced Pigmentation

Minocycline, which is a tetracycline derivative and frequently used in the treatment of acne, is a relatively common cause of drug-induced non–melanin-associated oral pigmentation.

Similar to tetracycline, minocycline can cause pigmentation of developing teeth. However, most patients are prescribed minocycline in early adulthood. When taken chronically, minocycline metabolites may become incorporated into the normal bone. Thus, whereas the teeth may be normal in appearance, the surrounding bone may appear green, blue, or even black. As a result, the palatal and alveolar mucosae may appear similarly and diffusely discolored. There is no treatment necessary for minocycline-induced pigmentation. The discoloration often subsides within months after discontinuation of the medication. However, the bone pigment may persist for longer periods of time, if not indefinitely.