Dental Pain

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Dental pain is unpleasant sensation felt by the patient in relation to odontogenic cause.


PAIN
ODONTOGENIC
NON ODONTOGENIC

NON ODONTOGENIC PAIN

MUSCULO SKELETAL	MYOFASCIAL PAIN [MPDS] TMD [BRUXISM]
NEUROPATHIC	TRIGEMINAL NEURALGIA GLOSSOPHARYNGEAL NEURALGIA
NEUROVASCULAR	MIGRAINE CLUSTER HEADACHES
INFLAMMATORY	ALLERGIC SINUSITIS BACTERIAL SINUSITIS
SYSTEMIC DISORDERS	CARDIAC PAIN HERPES ZOSTER NEOPLASTIC DISEASE
PSYCHOGENIC	MUNCHAUSENS SYNDROME

Odontogenic causes

- **Dentine sensitivity**
  - caries
  - trauma
- **Pulpitis**
  - Reversible(hyperemia).
  - Irreversible(acute or chronic)

Odontogenic causes

- **Pain originating from periodontium.**
  - acute apical periodontitis
  - Chronic apical periodontitis (granuloma)
  - periapical abscess
  - Periodontal abscess
- **Pericoronitis.**
- **Dry socket.**

Odontogenic causes

- Cracked tooth syndrome.
- Orthodontic treatment.
- Acute necrotizing ulcerative gingivostomatitis.

Assessment of pain

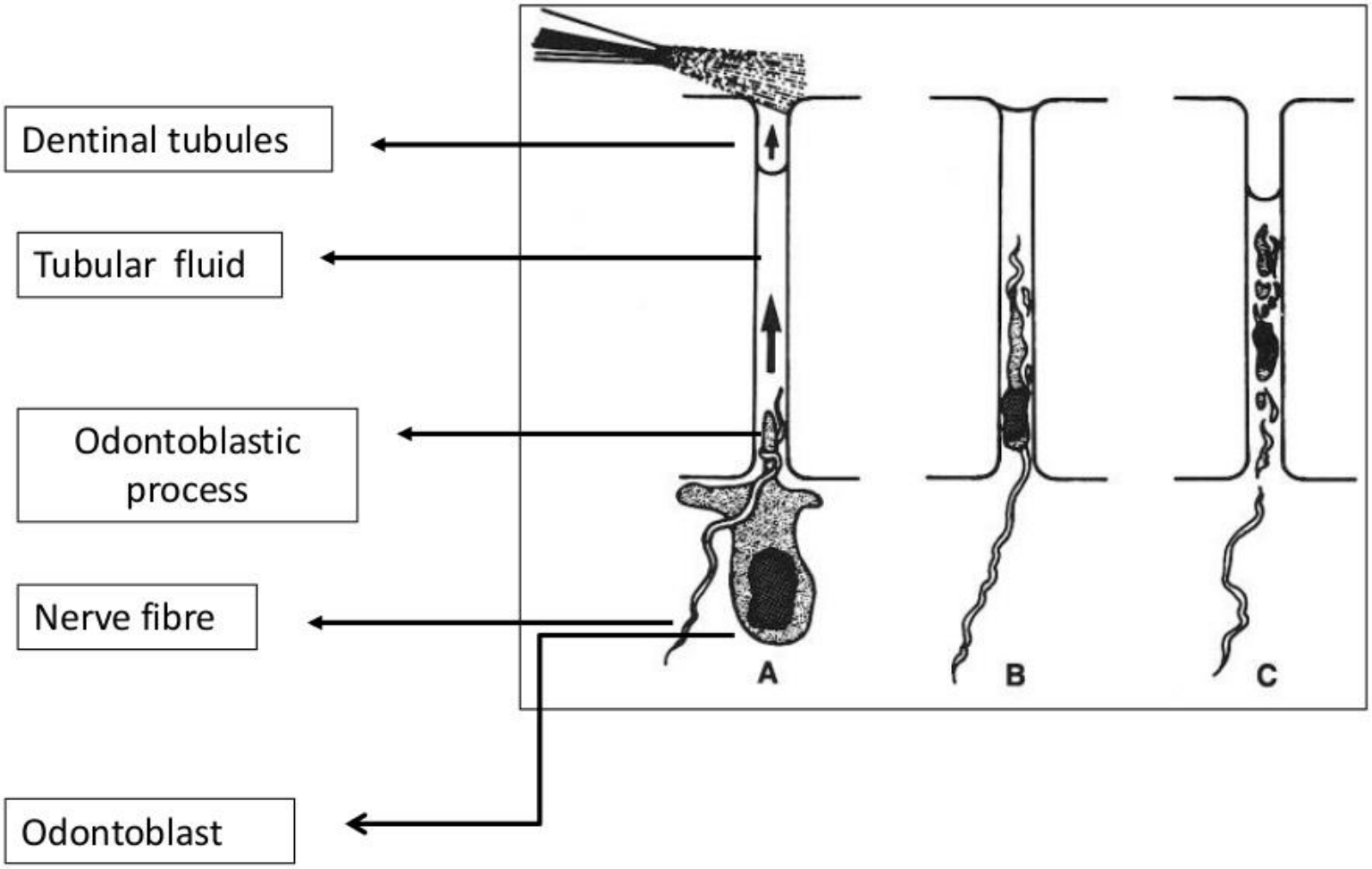
I. Site of pain
II. Radiation of pain
III. Character of pain
IV. Severity of pain
V. Duration of pain.
VI. Periodicity of pain
VII. Spontaneity of pain
VIII. Aggravating factors
IX. Reliving factors..
X. Associated signs &symptoms.

Objective Testing

- ***Visual Examination***
- ***Radiographs***
- ***Percussion***
- ***Palpation***
- ***Mobility***
- ***Vitality pulp testing***
- ***Periodontal probing***
- ***Selective anesthesia***
- ***Test cavity***
- ***Occlusion***

Dentine sensitivity:-

- S&S:
  The pain is best described as a sensation of sudden shock and is sharp in nature. It is never spontaneous in onset.
  Precipitating by a noxious stimulus, either hot or cold, sweet or sour, or touch.
- Localized &last for short time.
- Diagnosed by inspection or by x-ray…..etc.
- Management:-removal of caries & isolation of dentine.


Dentinal tubules
Tubular fluid
Odontoblastic process
Nerve fibre
Odontoblast
A
B
C

Pulpitis:-I-Reversible pulpitis

- Causes:

1. trauma
2. thermal shock
3. chemical
4. caries

- symptoms
- sharp pain with stimuli (esp cold)
- subside within seconds after its removal

Hyperemia :

Hyperemia → increased blood flow → increased pulpal pressure

Application of heat → increased pulpal pressure

↓

stimulate the nerve endings → Pain

- diagnosis
- 1-pain for short duration
- 2-visual checking for caries or fracture
- 3- percussion -ve
- 4-vitaltity test +ve
- Treatment:
- Conservative treatment

II-Irreversible pulpitis:-

- Cause:

1. trauma
2. Thermal shock
3. Chemical irritation

- Symptoms:

1. Pain with food impaction &cold & sweet food and may be spontaneous intermitted or continuous.

- The pain persist after removal of the stimuli and increase when the patient lies down or bend over and relived with cold
- The pain may referred to adjacent teeth or to ear or tmj area

- Diagnosis:

1. Visual deep cavity or secondary caries.
2. probing will cause pain.
3. Percussion +ve.
4. Vitality test hyperplasia respond of the pulp (esp. cold) which persist after its removal.
5. In late stage cold relief the pain.

- Treatment :
- Rct or extraction

Reversible pulpitis

- Characteristic short, sharp pain
- **Stimulated** by hot, cold or sweet stimuli
- **Few seconds** duration/**disappears** when stimulus removed
- Pulp sensibility tests may elicit an exaggerated response
- Tooth not tender to percussion (TTP)

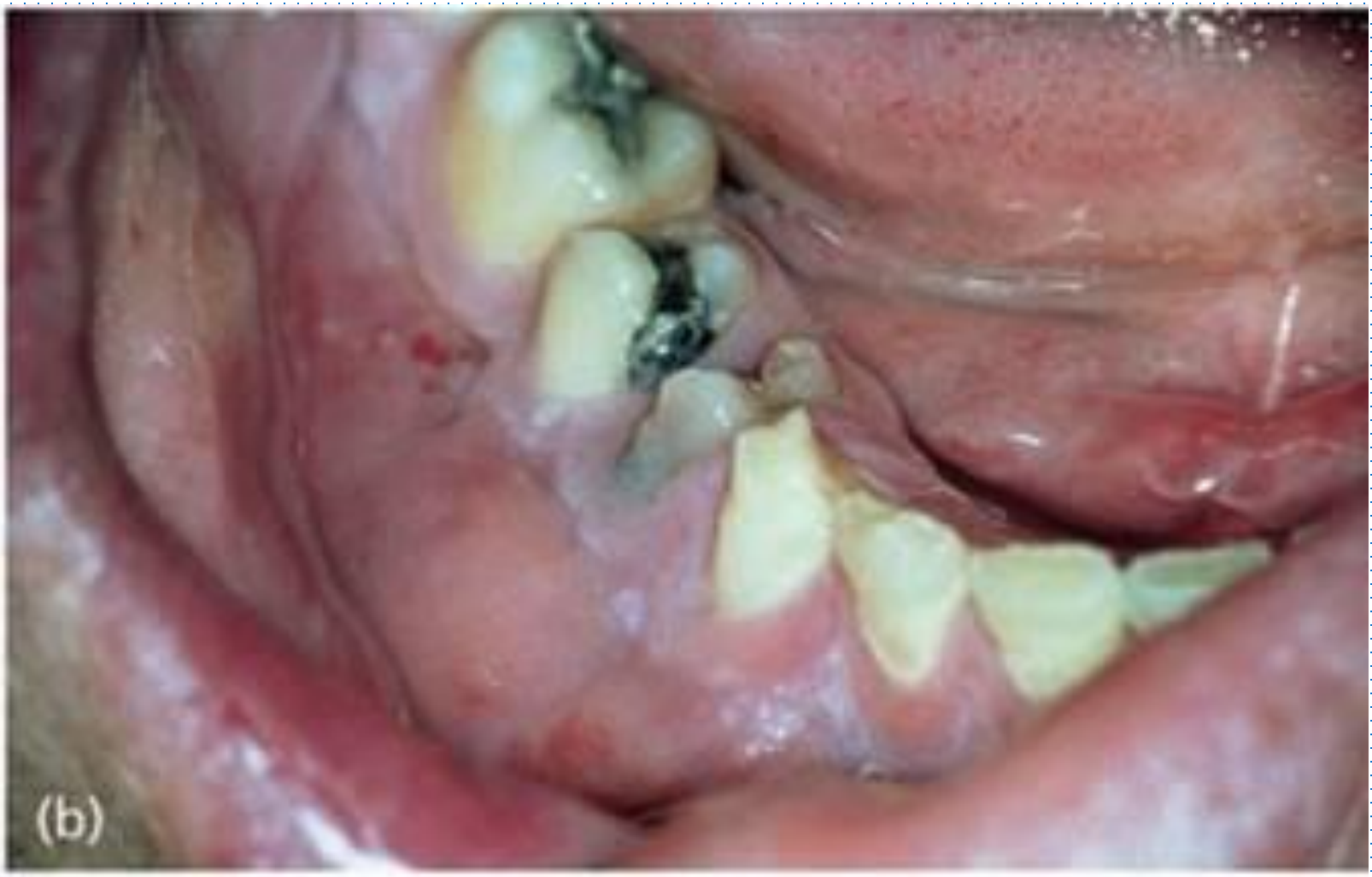
Irreversible pulpitis

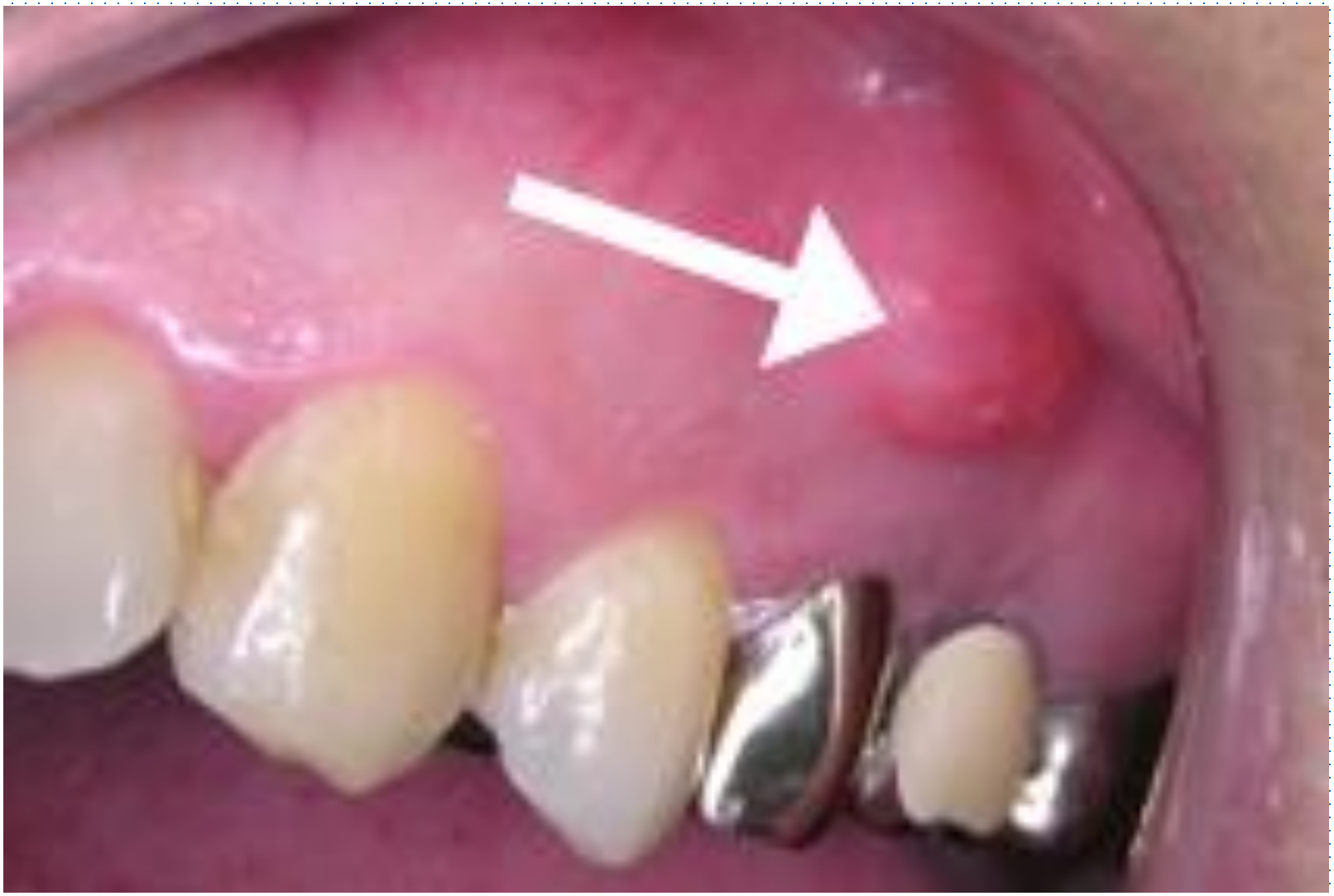
- Characteristic dull, throbbing pain (but may experience bouts of sharp pain)
- Onset usually unprovoked/**exacerbated** by hot, cold, sweet stimuli
- **Several minutes/hours** duration/**persists** when stimulus removed
- Pulp sensibility tests may elicit an exaggerated/negative response
- Tooth not TTP (unless late stage presentation)

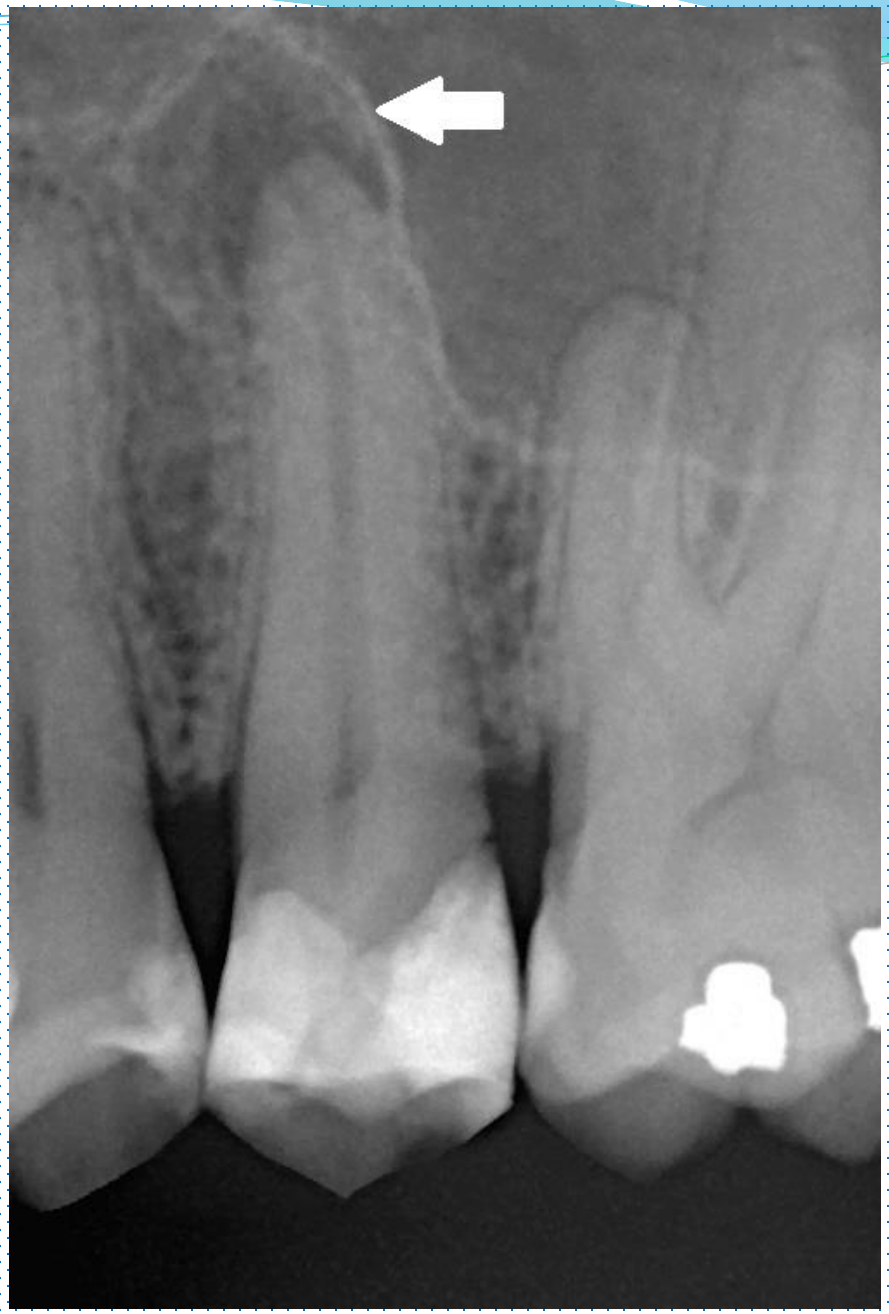
Abscess:-

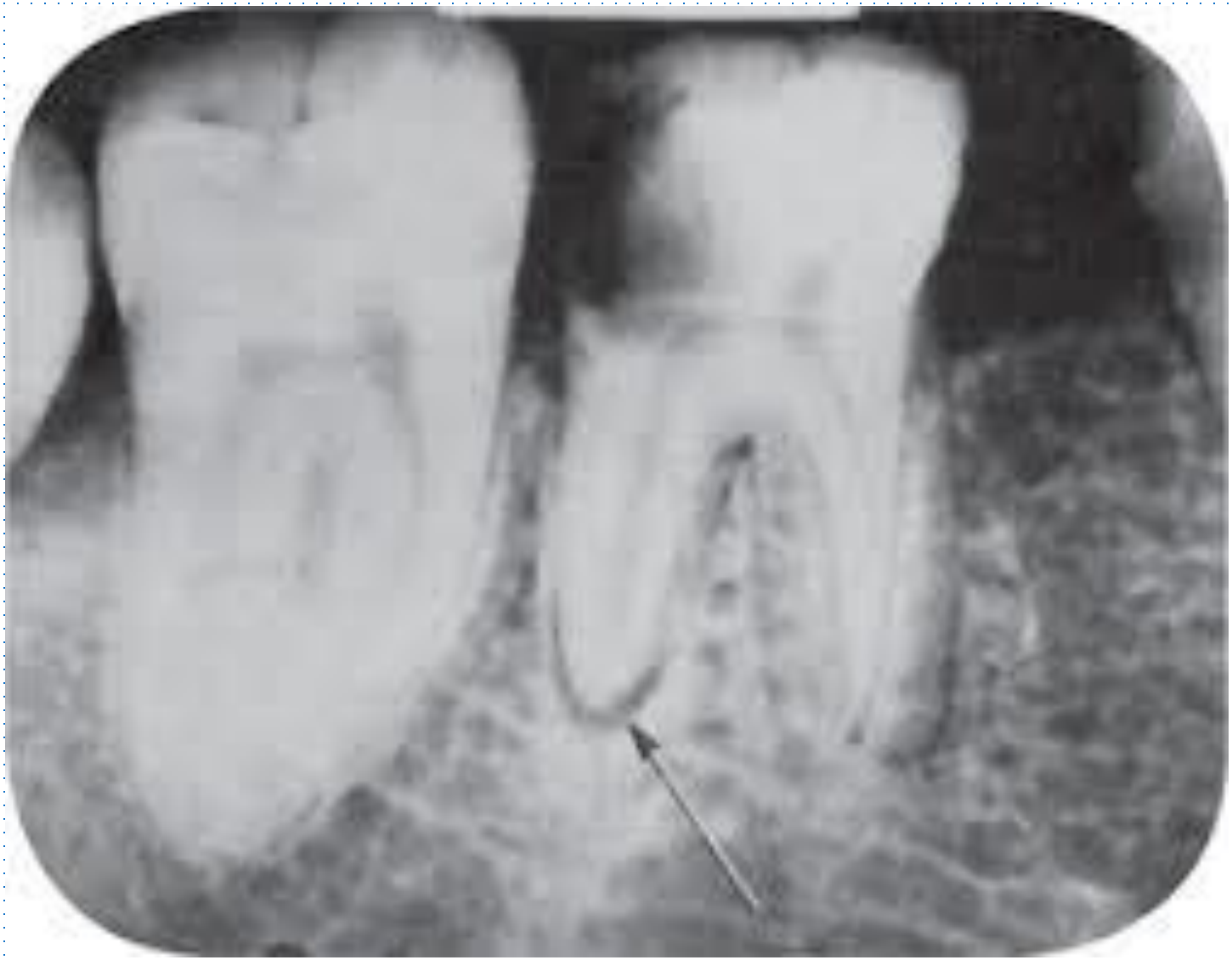
Point of comparison	Periapical abscess	Periodontal abscess
Periodontal pocket	no	Yes
Vitality test	- ve	+ve
Sensitivity to percussion	Yes	May or may not (lateral percussion)
radiographicaly	Angular bone loss and furcation rl	Changes in apical area
sweling	Localized to apex	Gingival tissue

Apical periodontitis

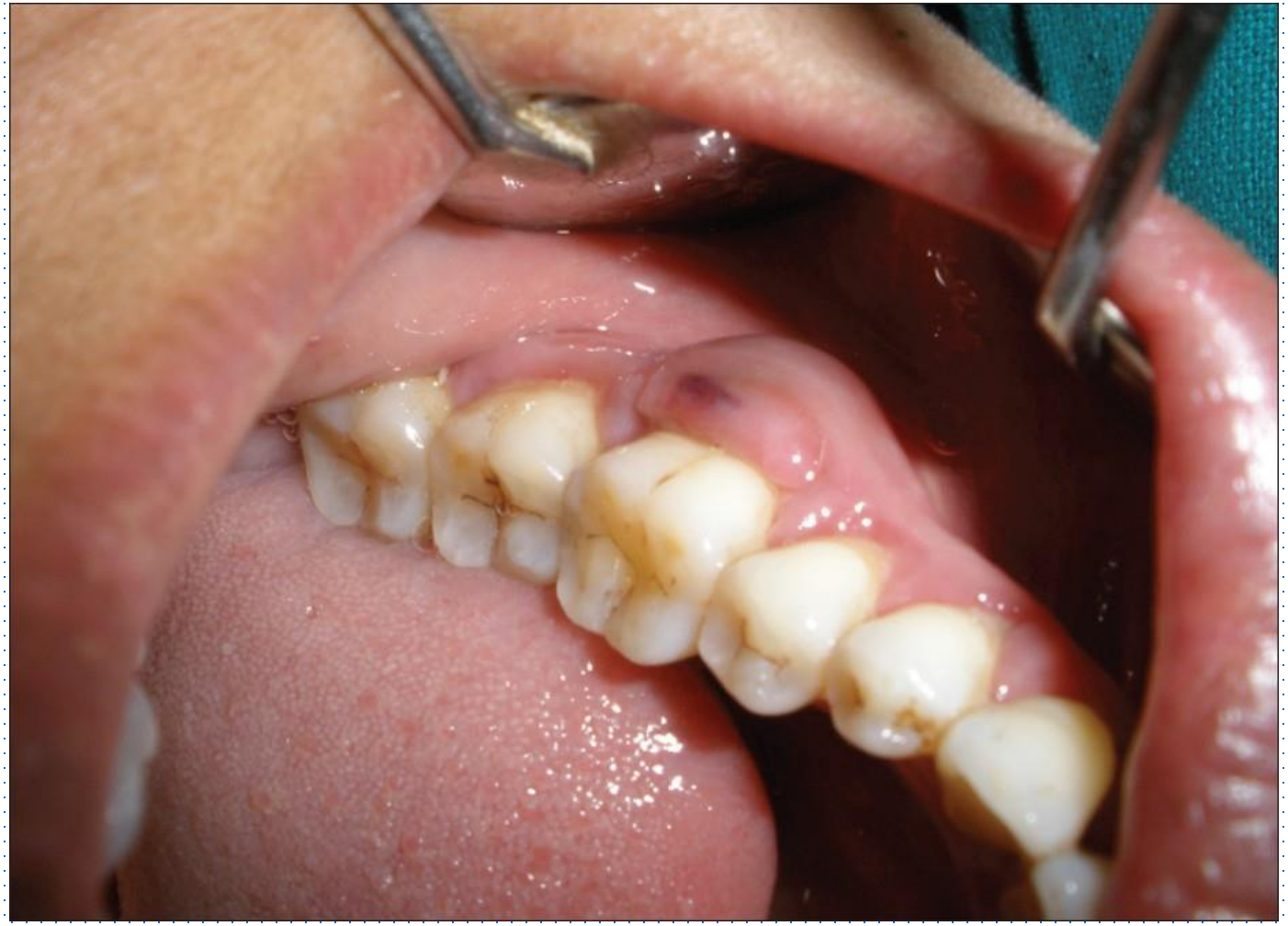


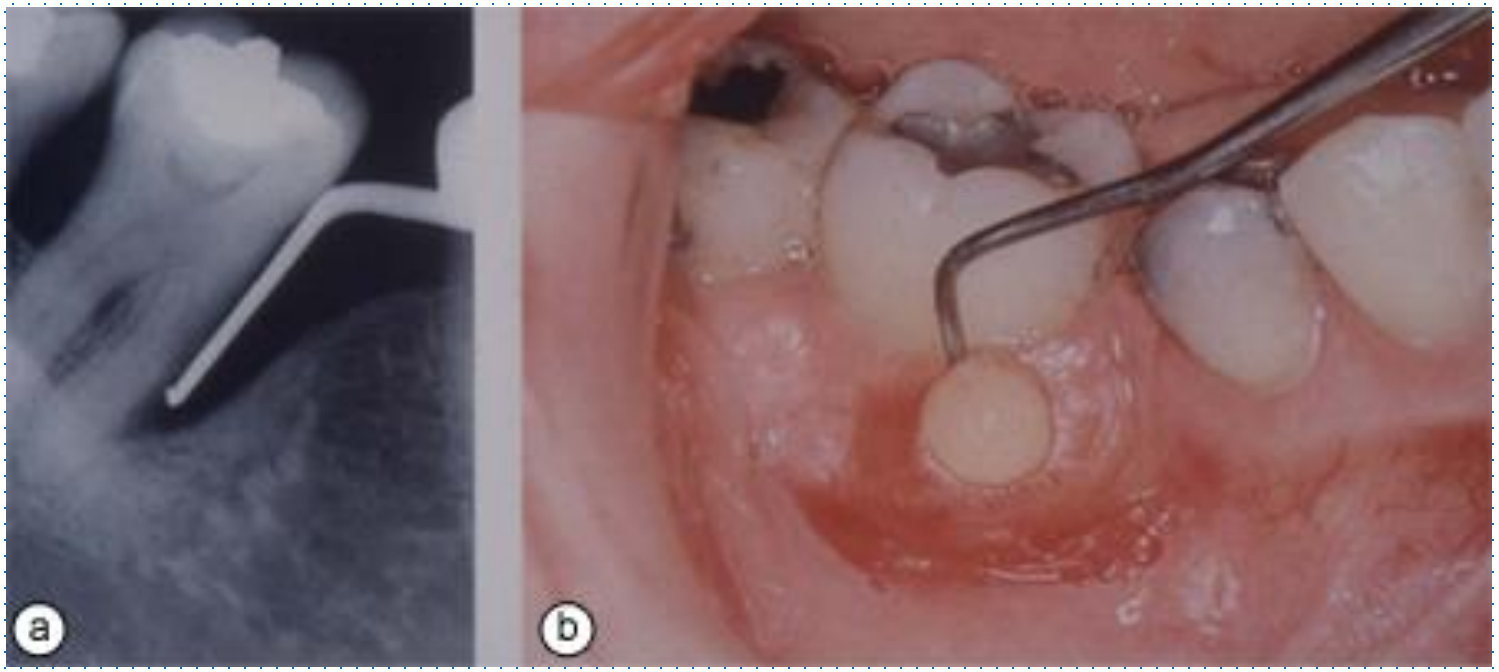




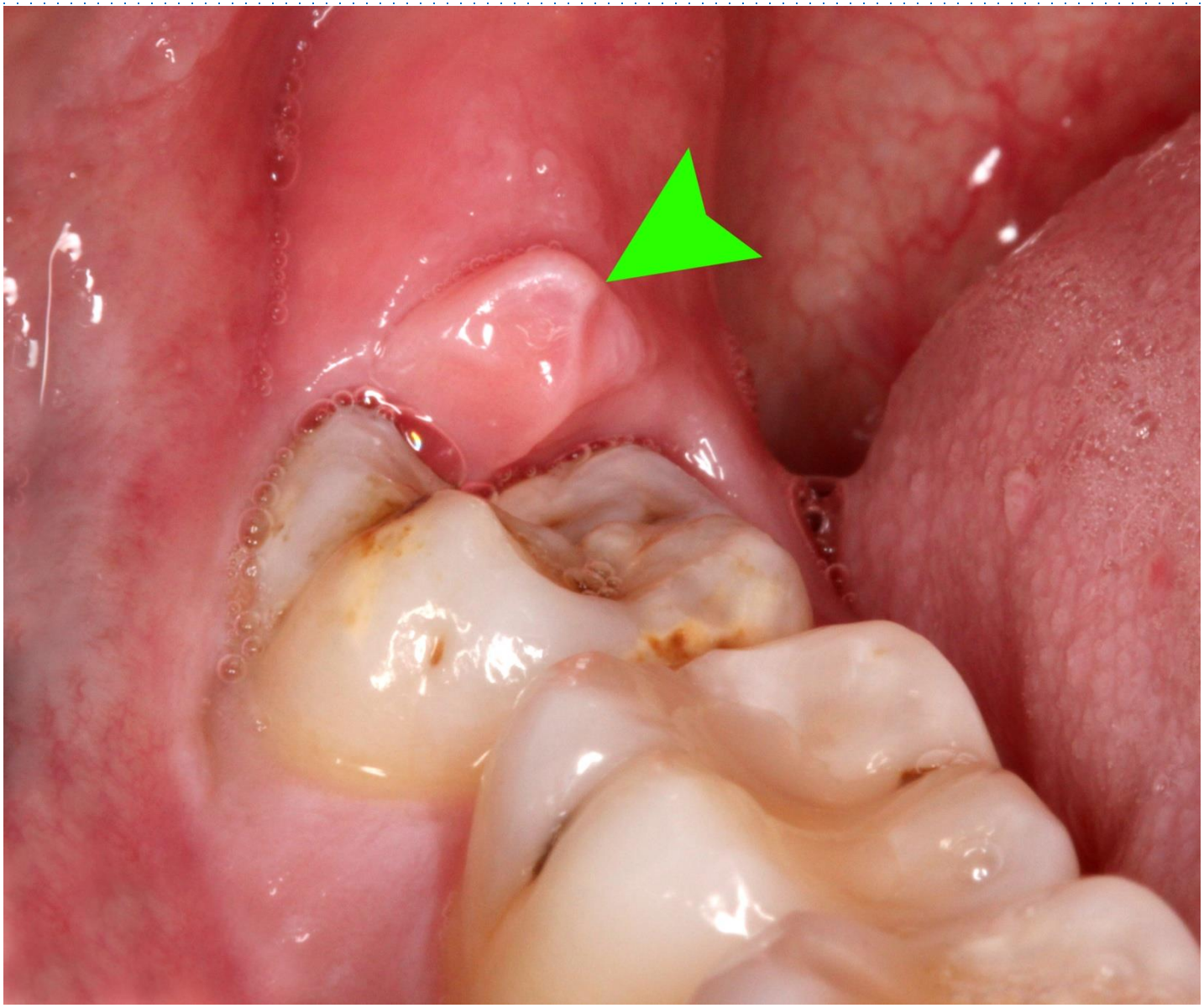


Periodontal abscess




a
b

Pericoronitis: an inflammation of the operculum around the crown of partially erupted a tooth (usually the third molar).



S&S:

- Radiating pain in back of the mouth and the inability to comfortably open or close the jaw. Not only is it painful to close against the inflamed operculum behind the erupting mandibular molar.
- sever pain & inflammation .
- Trismus
- Halitosis

Management:-

I. Conservative: irrigation ,anti-inflammatory &analgesic

II. Surgical:

- Operculectomy.
- Tooth extraction.

Dry socket:-failure of formation or maintenance of blood clot in extraction socket leaving bare bone.

S&S:-

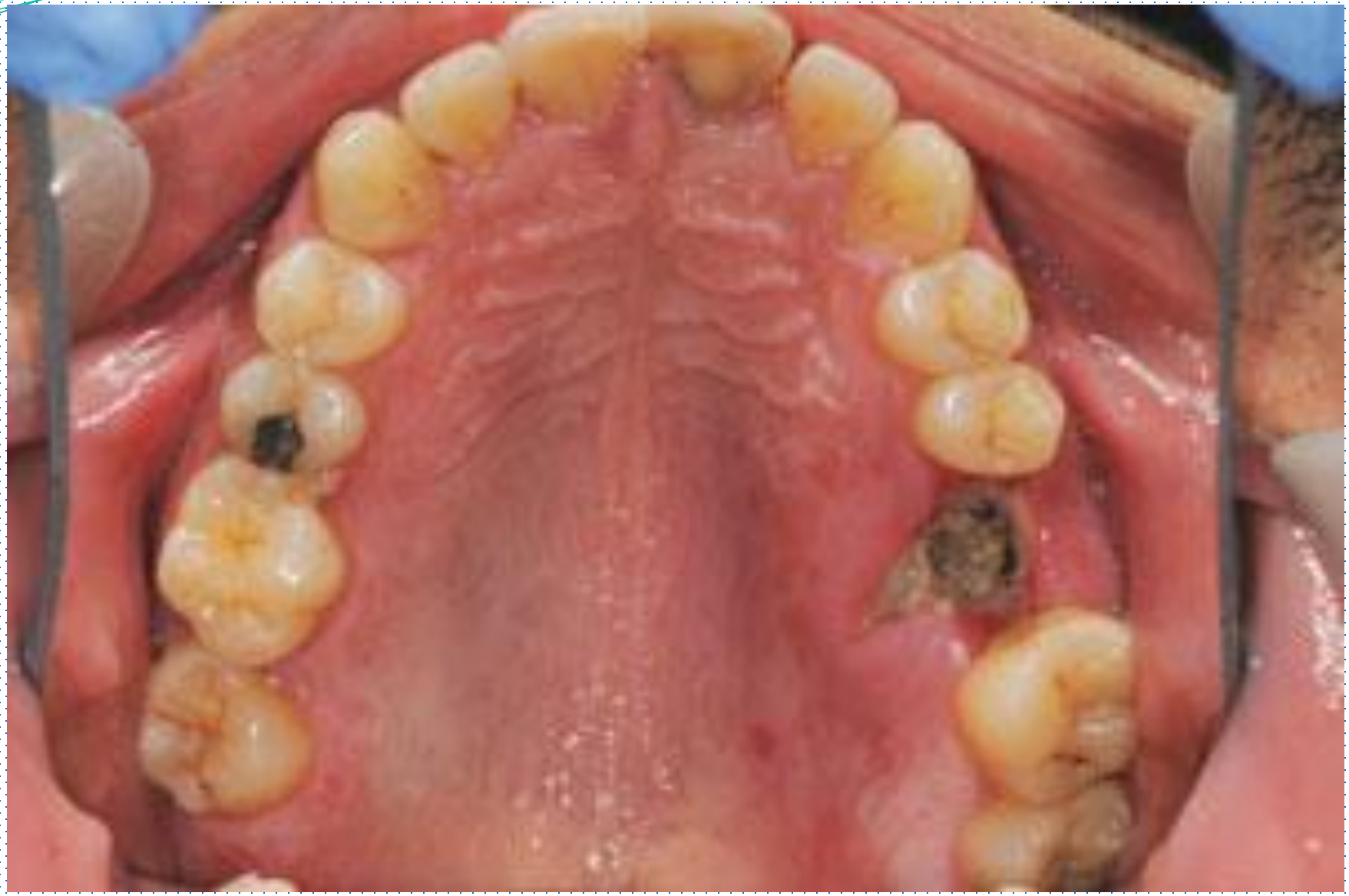
- sever pain.
- Halitosis..
- Bare bone.

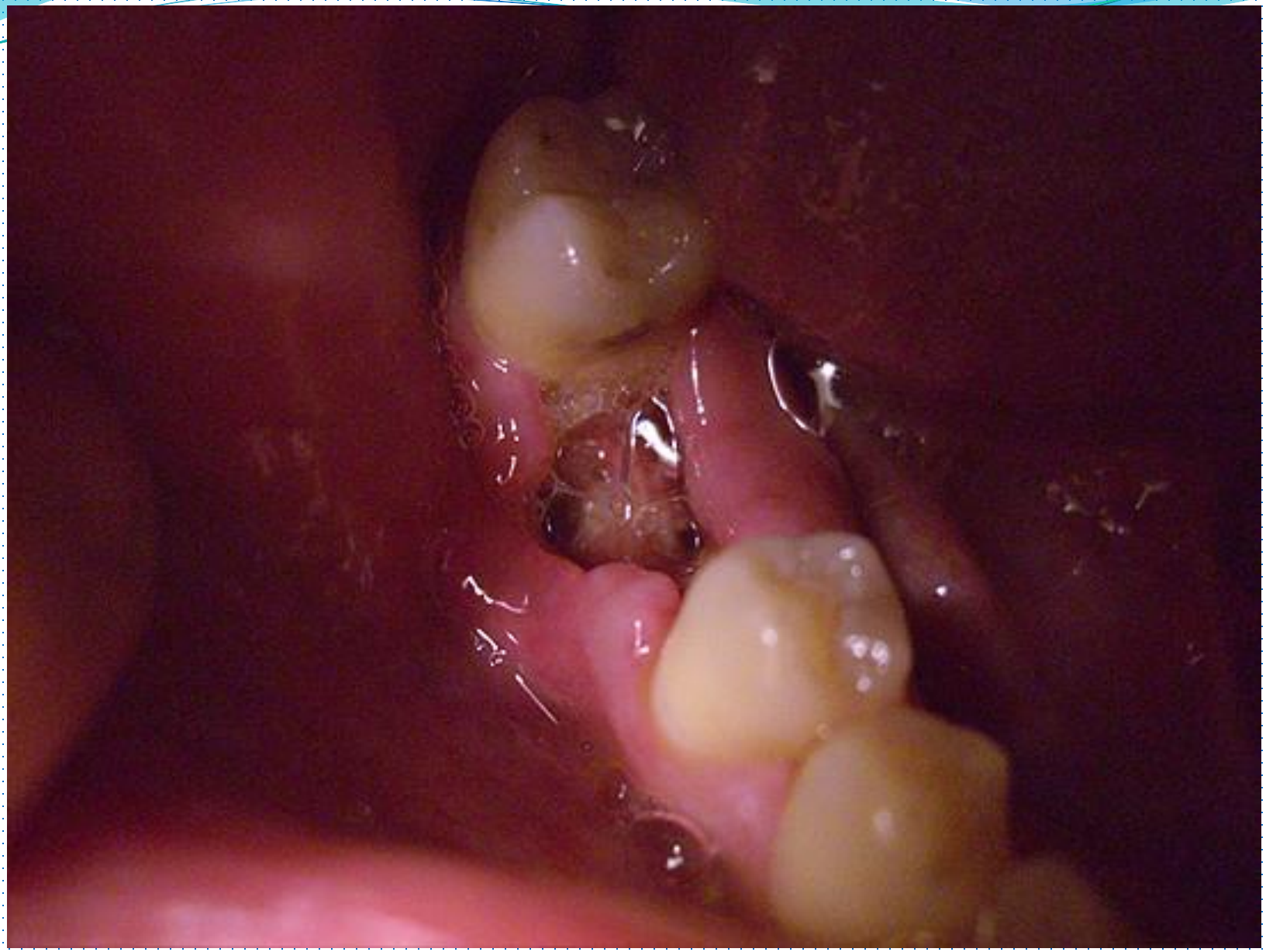
❖ **Management:**

I. Irrigation under L.A.

II. Alveogel or an abundant material is placed in the socket

III. Analgesic.





Atypical odontalgia

- The pt. localized the pain to a tooth or group of teeth are clinically and radiographically normal.
- The pt. may have already undergone dental treatment in an attempt to alleviate their symptoms, including the extraction of one or more tooth, with only temporary relief of symptoms.
- The dentist must resist extracting healthy tooth in such pt.


Thank You

Dental management of patients with cardiovascular diseases

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HYPERTENSION

Hypertension or high blood pressure, is a chronic medical condition in which the blood pressure in the arteries is elevated.

Blood pressure is summarized by two measurements, **systolic and diastolic**, which depend on whether the heart muscle is contracting (systole) or relaxed between beats (diastole).

High blood pressure is said to be present if it is often at or **above 140/90 mmHg**.

N.B. Only a physician can make the diagnosis of hypertension and decide upon its treatment. The dentist, however, should detect abnormal blood pressure measurements, which then become the basis for referral to or consultation with a physician, the dentist should not make a diagnosis of hypertension but rather should tell the patient that his or her blood pressure reading is elevated, and that a physician should evaluate the condition.

Causes of Hypertension

- About 90% of patients have no identifiable cause for their disease, which is referred to as **essential, primary, or Idiopathic hypertension.**

- The remaining 10% of patients, an underlying cause or condition may be identified; for these patients, the term **secondary hypertension** is applied.

Causes of 2nd hypertension

- Chronic kidney disease
- Coarctation of the aorta
- Cushing's syndrome and other glucocorticoid excess states, including chronic long- Term steroid therapy
- Drug-induced or drug-related (e.g., NSAIDs, oral contraceptives, decongestants)
- Primary aldosteronism and other mineralocorticoid excess states
- Thyroid or parathyroid disease

Signs and Symptoms of Hypertension

Early

- Elevated blood pressure readings
- Narrowing and sclerosis of retinal arterioles
- Headache
- Dizziness
- Tinnitus

Signs and Symptoms of Hypertension

ADVANCED

- Rupture and hemorrhage of retinal arterioles
- Papilledema : is optic disc swelling that is caused by increased intracranial pressure.
- Left ventricular hypertrophy
- Proteinuria
- Congestive heart failure
- Angina pectoris
- Renal failure
- Dementia Encephalopathy

Treatment of hypertension

1- non-drug therapy (life style modification)

- losing weight
- adopting a diet rich in vegetables, fruits, and low dairy products
- reducing cholesterol and saturated fats
- decreasing salt intake
- limiting alcohol intake
- Quitting smoking
- engaging in daily aerobic physical activity

Treatment of hypertension

2-drug therapy (medical treatment)

- Diuretics ,
- Beta blockers ,
- Calcium channel blockers ,
- ACE inhibitors ,
- Direct vasodilators,

THE DENTAL MANAGEMENT

The primary concern when one is providing dental treatment for a patient with hypertension is that during the course of treatment, the patient might experience an acute elevation in blood pressure that could lead to a serious outcome such as stroke or MI

This acute elevation in blood pressure could result from the release of endogenous catecholamines in response to stress and anxiety, from injection of exogenous catecholamines in the form of vasoconstrictors in the local anesthetic.

THE DENTAL MANAGEMENT

1\ ***The B.P. , pulse rate***, should be monitored preoperatively to be as a baseline record .

a-blood pressure **less than 120/80** : Any required dental treatment, both surgical and nonsurgical

b- blood pressure More than l20/80 but **Less than 140/90** (prehypertension): Any required dental treatment , both surgical and nonsurgical

THE DENTAL MANAGEMENT

c- blood pressure More than 140/90 but **Less than 160/100** (stage I): Any required dental treatment, both surgical and nonsurgical

d-blood pressure More than 160/100 but **Less than 180/110** (stage II) : Any required dental treatment, both surgical and nonsurgical

- it may be advisable to leave the blood pressure cuff on the patient's arm, and periodically check the pressure during treatment. If the blood pressure rises above 179/109, the appointment should be terminated .

THE DENTAL MANAGEMENT

e- blood pressure **more than 180/110** , in this case (avoid elective dental treatment) but if the patient needs urgent dental treatment (pain, infection, or bleeding) that may necessitate treatment.

- In this instance, the patient should be managed in hospital dental clinic,
- and we should consult the physician,
- and measures such as intraoperative blood pressure monitoring, electrocardiography monitoring,
- establishment of an intravenous line, and sedation may be used.

THE DENTAL MANAGEMENT

2\Once it has been determined that the hypertensive patient can be safely treated, a Management plan should be developed and the dentist should make every effort to ***reduce the stress and anxiety*** associated with dental treatment .and this obtained by:

- encourage the patient.
- morning appointment
- avoid long or stressful appointment
- use premedication such as valium (if needed) 2-5 mg the night before the dental treatment and /or 2-5 mg one hour before the dental procedure .

THE DENTAL MANAGEMENT

- avoid stimulating the gag reflex . because they have a nausea as a side effect of the antihypertensive drugs .
- if the patient is over stressed , then stop the dental treatment and terminate the appointment.
- Nitrous oxide plus oxygen inhalation sedation is an excellent intraoperative anxiolytic for use in patients with hypertensioHo **Care should be used to ensure adequate oxygenation at all times, especially at the termination of administration . Hypoxia is to be avoided because of the resultant elevation in blood pressure that may occur.**

THE DENTAL MANAGEMENT

3\ The ***local anaesthesia*** :

We can use xylocaine *2%* with adrenaline 1/ 100 000 but we have to use an aspirating syringe to avoid the intravascular injection , also we should not use more than two carpoules,because excessive use of the vasoconstrictors may cause significant elevation of blood pressure, with arrhythmia.

THE DENTAL MANAGEMENT

4\ Because some antihypertensive agents tend to produce orthostatic hypotension, sudden changes in chair position during dental treatment should be avoided .

so that at the end of dental treatment the ***dental chair should be returned slowly to an upright position***. After patients have had time to adjust to the change in posture, they should be physically supported while slowly getting out of the chair till they obtained good balance and stability. if they complain of dizziness or light headedness, they should sit back down until they recover equilibrium.


TAKE
A BREAK

ISCHEMIC HEART DISEASE

It is a condition in which there is insufficient blood supply to the heart muscles by the coronary artery.

Atherosclerosis (thickening of the intimal layer of the arterial wall caused by the accumulation of lipid plaques ,elevation in serum lipid levels is a major risk factor for atherosclerosis ,the atherosclerotic process results in a narrowed arterial lumen with diminished blood flow and oxygen supply), **is considered the most common cause of ischemic heart disease .**

ISCHEMIC HEART DISEASE

Atherosclerosis is lead to the narrowing of the lumen of the coronary artery leading to reduction of blood supply to a portion of the heart muscles resulting in a myocardial ischemia . this ischemia may be manifested as angina pectoris when there is a narrowing of the lumen of the coronary artery, but when there is an obliteration of the coronary artery it will lead to myocardial infarction .

ISCHEMIC HEART DISEASE

The predisposing factors involve:

- opacity ,
- smoking ,
- hypertension ,
- familial history,
- coronary artery spasm ,
- congenital abnormalities of the heart ,
- embolisim ,
- Patients with diabetes mellitus have a greater incidence of coronary atherosclerotic heart disease .

ISCHEMIC HEART DISEASE

Angina pectoris

- It is characterized by a brief pain , transient pain , **severe pain at the retrosternal area** radiating to the left shoulder and left arm .
- the patient become breathlessness.
- The pain is resulting from temporary ischemia of the myocardium,,
- the pain arises when the patient is exposed to a physical or emotional exertion .
- the attach is usually treated by rest, change the environment, and the use of nitroglycerine tablets sublingually . and recently the condition could be treated by coronary angioplasty and stent placement „ also treated surgically the coronary artery bypass graft (CABG) .

TYPES OF ANGINA

1\ STABLE ANGINA.

This pain typically is precipitated by physical effort such as walking or climbing stairs but may also occur with eating or stress. Pain is relieved by cessation of the precipitating activity, by rest, or by the use of nitroglycerin, and it has a good prognosis .

TYPES OF ANGINA

2\ UNSTABLE ANGINA

In this case the pain is more intense pain than stable angina , the pain is precipitated by less effort than stable angina, or pain that occurs at rest, the pain lasts longer than 15 minutes . This pain is not readily relieved by nitroglycerin. In this case the patient develop a progressive angina , it has a poor prognosis ,and may develop a myocardial infarction within a short time .

TYPES OF ANGINA

3\ A relatively uncommon form of angina, **Prinzmetal's variant angina**, occurs at rest and is Caused by focal spasm of a coronary artery, usually with varying amounts of atherosclerosis.

4\ The term **acute coronary syndrome** describes a myocardial ischemia that ranges from unstable angina at one end of the spectrum to non-ST segment Ml at the other.

ISCHEMIC HEART DISEASE

MYOCARDIAL INFARCTION

- If the degree of ischemia that results from coronary atherosclerosis is significant and prolonged, the area of myocardium supplied by that vessel may undergo necrosis.

- Reduced blood flow may result from thrombosis of the affected artery, a hypotensive episode, an increased demand for blood, or emotional stress. The infarction, or area of necrosis, may be **subendocardial or transmural**, the latter involving the entire thickness of the myocardium.

MYOCARDIAL INFARCTION

Complications of MI include:

- weakened heart muscle, resulting in acute congestive heart failure,
- postinfarction angina, infarct extension,
- cardiogenic shock,
- pericarditis,
- arrhythmias.
- Causes of death in patients who have had an acute MI (AMI) include ventricular fibrillation, cardiac standstill, congestive heart failure, embolism, and rupture of the heart wall or septum

MYOCARDIAL INFARCTION

Chest pain is the most important symptom of coronary atherosclerotic heart disease..

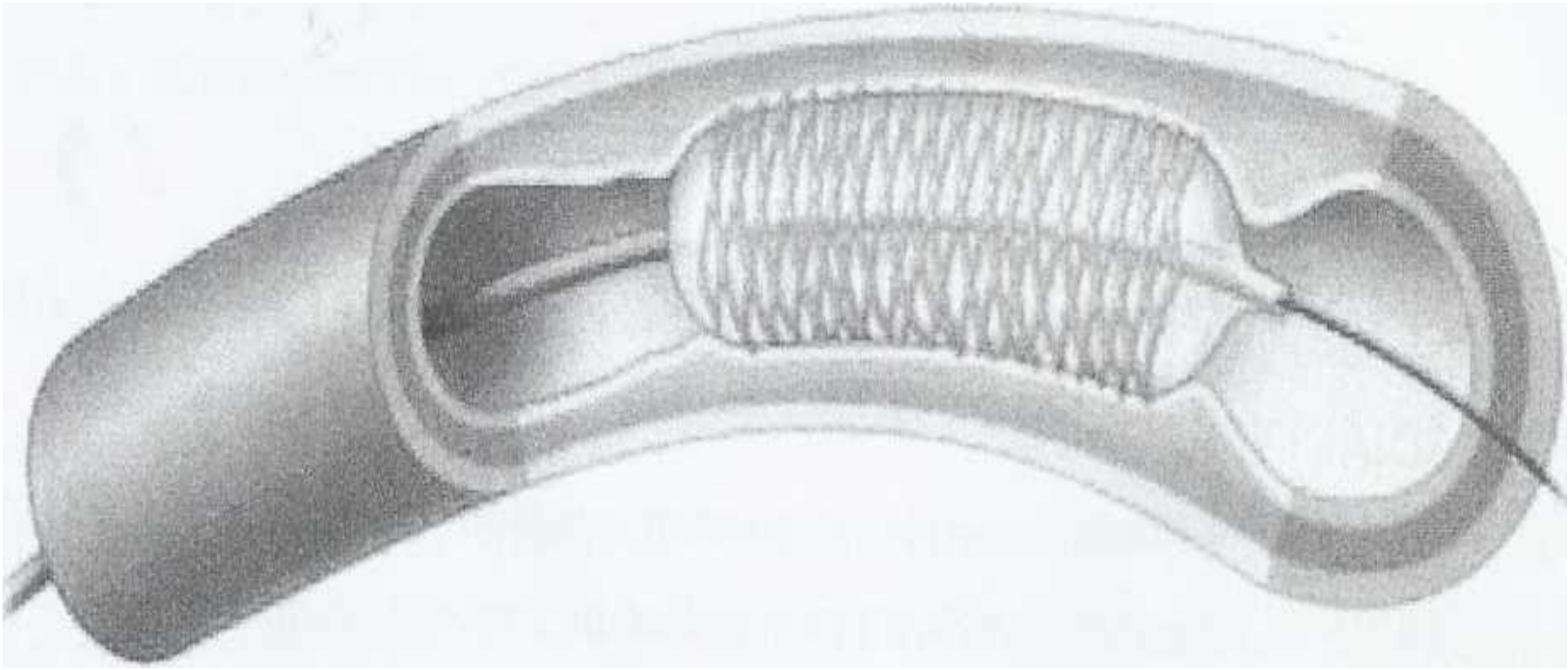
This pain usually is more severe than angina , Vasodilators or cessation of activity does not relieve the pain caused by infarction.

Patients who have experienced an MI should be hospitalized or should receive emergency treatment as soon as possible . The basic management goal is to minimize the size of the infarction and prevent death from lethal arrhythmias.

The medical treatment IHD

- Identification and treatment of associated diseases that can precipitate or worsen angina (anemia, obesity, hyperthyroidism)
- Reduction in coronary risk factors (hypertension, smoking, hyperlipidemia)
- Application of general and non - pharmacologic methods (lifestyle modifications)
- Nitrates (vasodilators)
- Beta blockers
- Calcium channel blockers
- Antiplatelet agents
- Percutaneous transluminal coronary angioplasty with stenting

  Coronary artery bypass graft



Expandable metallic stent that is left in place after deflation and withdrawal of the balloon catheter.

THE DENTAL MANAGEMENT

A- Patients with stable angina and a history of myocardial infarction (more than six months):

Those patients are considered as a intermediate risk, because the stress and anxiety during the dental procedure ,and excessive amount of vasoconstrictor in local anesthesia may precipitate the attack of angina pectoris ,MI, arrhythmia, or sudden death .

THE DENTAL MANAGEMENT

1- We have to identify the patient with a history of angina pectoris , and if the patient is not under medical control we have to **refer him to physician** for an evaluation and control.

2- If the patient is under good medical control, for those patients we can do any routine dental treatment, but we have to take in consideration the following points :

b- We have to check the **B .P . and pulse rate** to be as a base line record .

c- The patient should bring his own supply of **nitroglycerine tablets** to dental clinic and the patient Is given one tab . sublingually preoperatively (if it is needed)

d- **Reduce the stress and anxiety** for the patient by :

- Use valium 2-5 mg night before and/or2“5 mg one hour before the dental procedure .(if it is needed).
- Establish a honest and supportive relationship with the patient.
- We can use nitrous oxide Inhalation as a sedation , but we have to avoid hypoxia »
- the Complex and lengthy dental procedures should be spread over several appointments

THE DENTAL MANAGEMENT

3-The local anaesthesia:

a- Xylocaine 2% with adrenaline 1/100 000, but we have to use aspirating syringe to avoid intravascular injection , and we should not use more than two carpoules .

b- we can use use mepvlcaine 3% without vasoconstrictor.

C- Avoid the use of adrenaline in retraction cord .

THE DENTAL MANAGEMENT

4- the patient should be given **antibiotic prophylaxis** regimen :

- If the patient has a history of coronary artery angioplasty with stent placement during the first month after stent placement, but after one month the antibiotic prophylaxis regimen is not needed ,because the stent is covered by endothelium within two to four weeks.
- If the patient has a history of a coronary artery bypass graft (CABG), then he should be given antibiotic prophylaxis regimen during the first six months after the heart surgery .

5- if the patient developed chest pain during the dental procedure , We have to stop the dental procedure and give one tab . of **nitroglycerine sublingually** ====== then:

a- If the pain relieved after 5 “minutes , we can either continue the treatment or postpone it depends on the condition of the patient.

b- If the pain continues longer than 5-- minutes, we can give two additional nitroglycerine tabs . sublingually one tab . each 5 - minutes , if after three nitroglycerine tabs . (15 - minutes) the chest pain is persist then send the patient to the medical emergency care .

6- **The use of anticoagulants:**

a- Patients who take **aspirin or another platelet aggregation antagonist** such as clopidogrel can expect some Increase in bleeding. This effect generally is not clinically significant, and bleeding may be controlled through local measures. Discontinuation of these agents before dental treatment generally is unnecessary.

b- Patients who take **warfarin for anticoagulation** must have a current international normalized ratio (INR) determined before any invasive procedure can be performed.

c- The Local hemostatic measures that used to control bleeding include the use of hemostatic agents in the sockets (gel foam ,surgical), suturing, gauze pressure packs.

THE DENTAL MANAGEMENT

B- patients with unstable angina and recent MI (less than six months).

Those patients are considered as a major risk because during the first several weeks after an infarction, the conduction system of the heart may be unstable, and patients are prone to serious arrhythmias and re-infarction.

1- You have to **avoid any routine dental care.**

2- If treatment becomes necessary, it should be performed as conservatively as possible and directed primarily toward **pain relief, infection control**, and this dental treatment should be done in a hospital dental clinic and under the supervision of the physician taking in consideration the following points :

a- Check the **B . P . and pulse rate** to be as a base line record

b- Establishment of an **I.V. line i.e. use I.V. canula** to be ready for emergency care, continuously monitoring the **ECG and vital signs, using a pulse oximeter.**

c- Preoperatively you have to:

- Give the patient **nitroglycerine tab .** sublingually .
- Give **valium tabs. 5 - mg** the night before and one hour preoperatively .

d- We can use nitrous oxide inhalation as a sedation , but we have to avoid hypoxia , and check the oxygen saturation .

THE DENTAL MANAGEMENT

3-The Local anesthesia:

It Is better to use local anesthesia without vasoconstrictor such as mepvicaine 3% without vasoconstrictor ,But if it is necessary to use anesthesia with vasoconstrictor this should be discussed with the physician because of the possibilities of precipitating cardiac **tachycardias, arrhythmias, and increases in blood pressure**,.and we can use Xylocaine 2 % with adrenaline 1 / 100 000 but we have to use **aspirating syringe** to avoid intravascular injection , and we should **not use more than two** carpoules.

THE DENTAL MANAGEMENT

4- If the patient developed **chest pain** during the dental procedure, stop the dental procedure and give the patient nitroglycerine tab sublingually, if the chest pain is relieved after the first, second , third , tab . you have to terminate the appointment.

4- The use of anticoagulants:

a- Patients who take aspirin or another platelet aggregation antagonist such as clopidogrel can expect some increase in bleeding. This effect generally is not clinically significant, and bleeding may be controlled through local measures. Discontinuation of these agents before dental treatment generally is unnecessary.

b-Patients who take warfarin for anticoagulation must have a current international normalized ratio (INR) determined before any invasive procedure can be performed.

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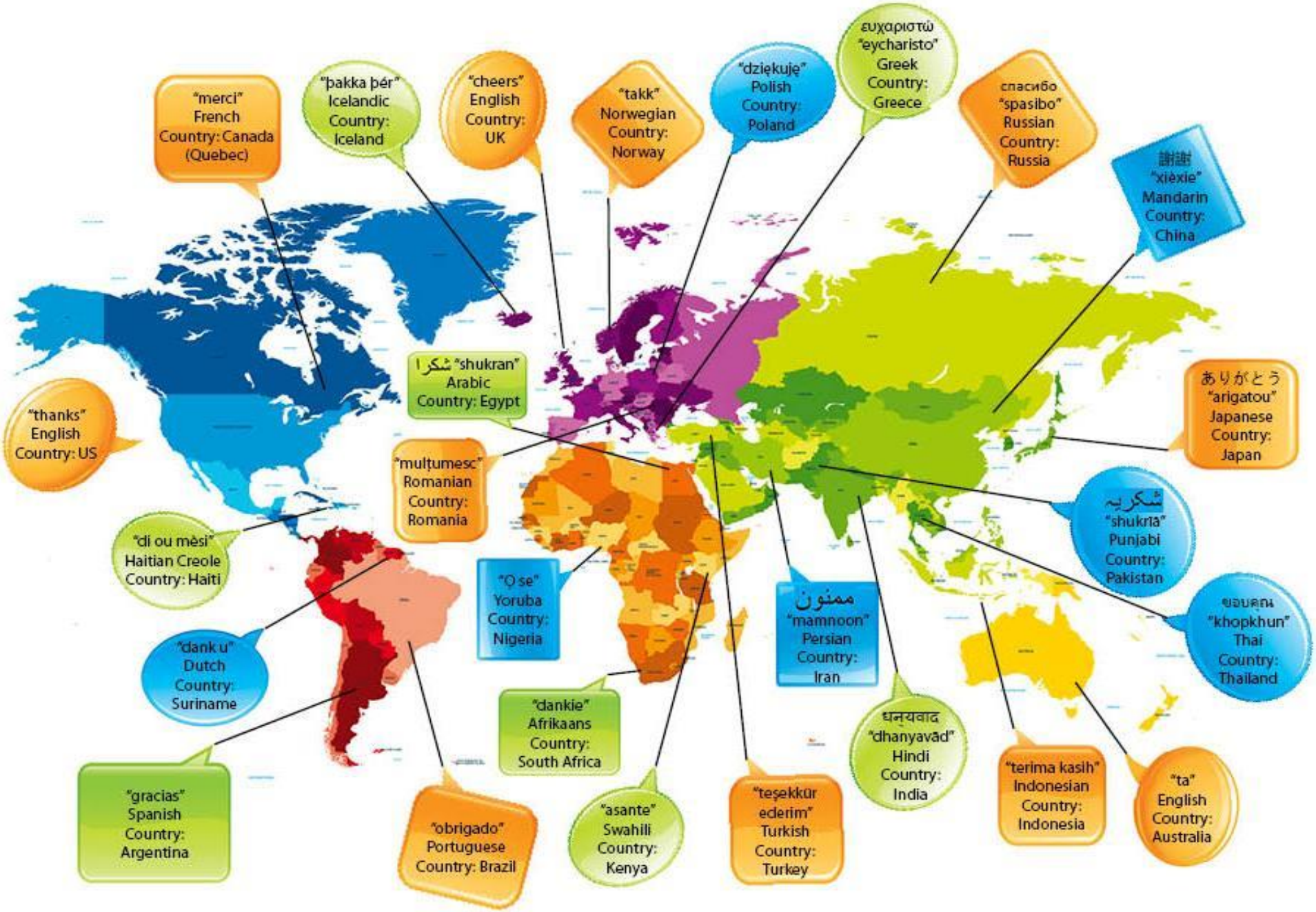
- the use of hemostatic agents in the sockets (gel foam ,surgicel),
- Suturing.
- gauze pressure packs.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Spanish
Country:
Argentina
"obrigado"
Portuguese
Country: Brazil
"asante"
Swahili
Country:
Kenya
"teşekkür
ederim"
Turkish
Country:
Turkey
"ta"
English
Country:
Australia

Dental management of patients with cardiovascular diseases

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THE INFECTIVE ENDOCARDITIS (IE)

It is a disease caused by a microbial infection of the heart valves or endocardium in the presence of a heart defect such as **rheumatic heart disease or congenital heart disease**.

A clinically and pathologically similar infection that may occur in the endothelial lining of an artery, usually adjacent to a vascular defect (e.g., coarctation of the aorta) or an arteriovenous [AV] shunt), is called **infective endarteritis**.

PATHOPHYSIOLOGY AND COMPLICATIONS

The **turbulence of blood flow** produced by certain types of congenital or acquired heart disease, such as flow from a high to a low pressure chamber or across a narrowed orifice, **traumatizes the endothelium** , this leads to the deposition of platelets and fibrin on the surface of endothelium which result into a **nonbacterial thrombotic endocarditis (NBTE)** Initially, these masses are sterile and do not contain microorganisms.

PATHOPHYSIOLOGY AND COMPLICATIONS

With the occurrence of a **transient bacteremia**, (in fact any injury to the soft tissue and bone can produce transient bacterimia) however, **bacteria can be adhere to the mass**. Additional platelets and fibrin are then deposited onto the surface of the mass, which serves to protect the bacteria that undergo rapid multiplication within the protection of the vegetative mass, Bacteria are slowly and continually released from the vegetations and shed into the bloodstream, resulting in a continuous bacteremia; **fragments of the friable vegetations break off and embolize**. A variety of host immune responses to bacteria may occur. This sequence of events results in the clinical manifestations of IE.

The causative microorganisms

- A total of 80% to 90% of cases of identified IE are due to **streptococci and staphylococci** ..
- Fungal infection could be a causative factor.

N.B. In past the mortality rate was 100% before the use of antibiotics .but nowadays the mortality rate is greatly reduced , but still about 10% of the patients die because of the disease .

SIGNS AND SYMPTOMES

- The **classic findings** of IE include fever, heart murmur, and positive blood culture,
- The **Peripheral manifestations** of IE due to emboli and/or immunologic responses These include petechiae of the palpebral conjunctiva, the buccal and palatal mucosa, and extremities , Osler's nodes (small, tender, subcutaneous nodules that develop in the pulp of the digits), splenomegaly and clubbing of the digits.
- The most common complication of IE, and **the leading cause of death, is heart failure** that results from severe valvular dysfunction. This most commonly occurs as a problem with aortic valve involvement followed by mitral and then tricuspid valve infection.

SIGNS AND SYMPTOMES

- Embolization of vegetation fragments leads to complications in up to 35% of cases of IE, with **stroke** being the most common.
- **Myocardial infarction** can occur as the result of embolism of the coronary arteries,
- Emboli also may involve other systemic organs, including the liver, spleen, kidney, and abdominal mesenteric vessels. **The incidence of embolic events is markedly reduced by the initiation of antibiotic therapy.**
- **Renal dysfunction** is also common and may be due to immune complex glomerulonephritis or infarction.

THE DENTAL MANAGEMENT

The aim of the dentist is to prevent the bacterial endocarditis from occurring in the susceptible patients . in fact any Injury to the soft tissue and bone can produce transient bacteremia , and In susceptible patients can results in endocarditis

Cardiac Conditions Associated With the Highest Risk of Endocarditis for Which Prophylaxis With Dental Procedures is Recommended

- Prosthetic cardiac valve
- Previous infective endocarditis
- Congenital heart disease (CHD)
- Unrepaired cyanotic CHD
- Completely repaired CHD with prosthetic material or device by surgery or Catheter intervention during the first 6 months after the procedure
- Repaired CHD with residual defects at the site or adjacent to the site of a Prosthetic patch or prosthetic device, which inhibits endothelialization
- Coronary artery bypass graft surgery, Angioplasty, coronary artery stents,
- Cardiac transplantation recipients who develop cardiac valvulopathy

1\ A.H.A. Recommended **Prophylactic Regimen For Dental Procedures**

I- patients Not Allergic To Penicillin (Standard Regimen)

- Amoxicillin 2 Gms Orally . One Hour Before The Dental Procedure.

Ii- patients Allergic To Penicillin (Alternate Regimen).

- Clindamycine 600 Mgs Orally. One Hour Before The Dental Procedure.
- Or cephalexin 2 Gms Orally One Hour Before The Dental Procedure.
- Or Azsthromycin Or Clarithromycin 500 Mg. Orally . One Hour Before The Dental Procedure.

2\ **patients given general anaesthesia for oral surgical or dental procedure or who are unable to use oral medication**:- (alternate regimen).

I- PATIENTS NOT ALLERGIC TO PENICILLIN

- AMPICILLIN 2 gms I.V. OR I.M. 30 MINUTES BEFORE DENTAL PROCEDURE.

II- PATIENTS ALLERGIC TO PENICILLIN:

- CLINDAMYCIN 600 mg. i.V. 30 MINUTES BEFORE DENTAL PROCEDURE.
- OR Cefatrixion 2 gm iv or im 30 min. before the dental procedure .

THE RECOMMENDED DOSE FOR THE CHILDREN IS AS FOLLOW

Amoxicillin	50 mg / kg
Clindamycine	20 mg / kg
Cephalexine	50 mg / kg
Ampcillin	50 mg / kg
Azithromycine & Clarithromycine	50 mg / kg

N.B. The children dose should not exceed the adult dose .

Special considerations when we prescribe the prophylactic regimen:

- Patients who are already taking penicillin or amoxicillin for eradication of an infection (e.g., sinus infection) or for long-term secondary prevention of rheumatic fever are likely to have viridans group streptococci that are relatively resistant to penicillin or amoxicillin,, Therefore, clindamycin, azithromycin, or clarithromycin should be selected for prophylaxis if treatment is **immediately necessary**- Because of cross resistance with cephalosporins, this class of antibiotics should be avoided- An alternative approach is to wait for at least 10 days after completion of antibiotic therapy before administering prophylactic antibiotics- In this case, the usual regimen can be used.

- If multiple coverage periods are needed then the antibiotic should be alternated i.e. first using amoxicillin and then use Clarithromycine and then Cephalexine and so on ,but 7-10 days should be elapsed between one coverage period and another. this done in order to prevent the development of resistant micro organisms.

- Coverage period may extend for 5—7 days in certain cases such as Surgical procedures with sutures or surgical areas which are slow to heal - and this obtained by giving the patient a prophylactic dose preoperatively and then continue with a treatment dose till the suture removal .


New graces ever gaining
From this our day of rest,

Rheumatic fever and Rh. heart disease

Rheumatic fever is an acute Inflammatory disease arises from the Infection of the throat & tonsils by **group A hemolytic streptococci** which has a high virulence.

It is an immune related disease**,** that the products of bacteria will sensitize the connective tissues leading to antigen formation (Ag) which stimulate the antibody formation {Ab.) leading to **Ag – *Ab.* reaction** leading to connective tissue-necrosis .

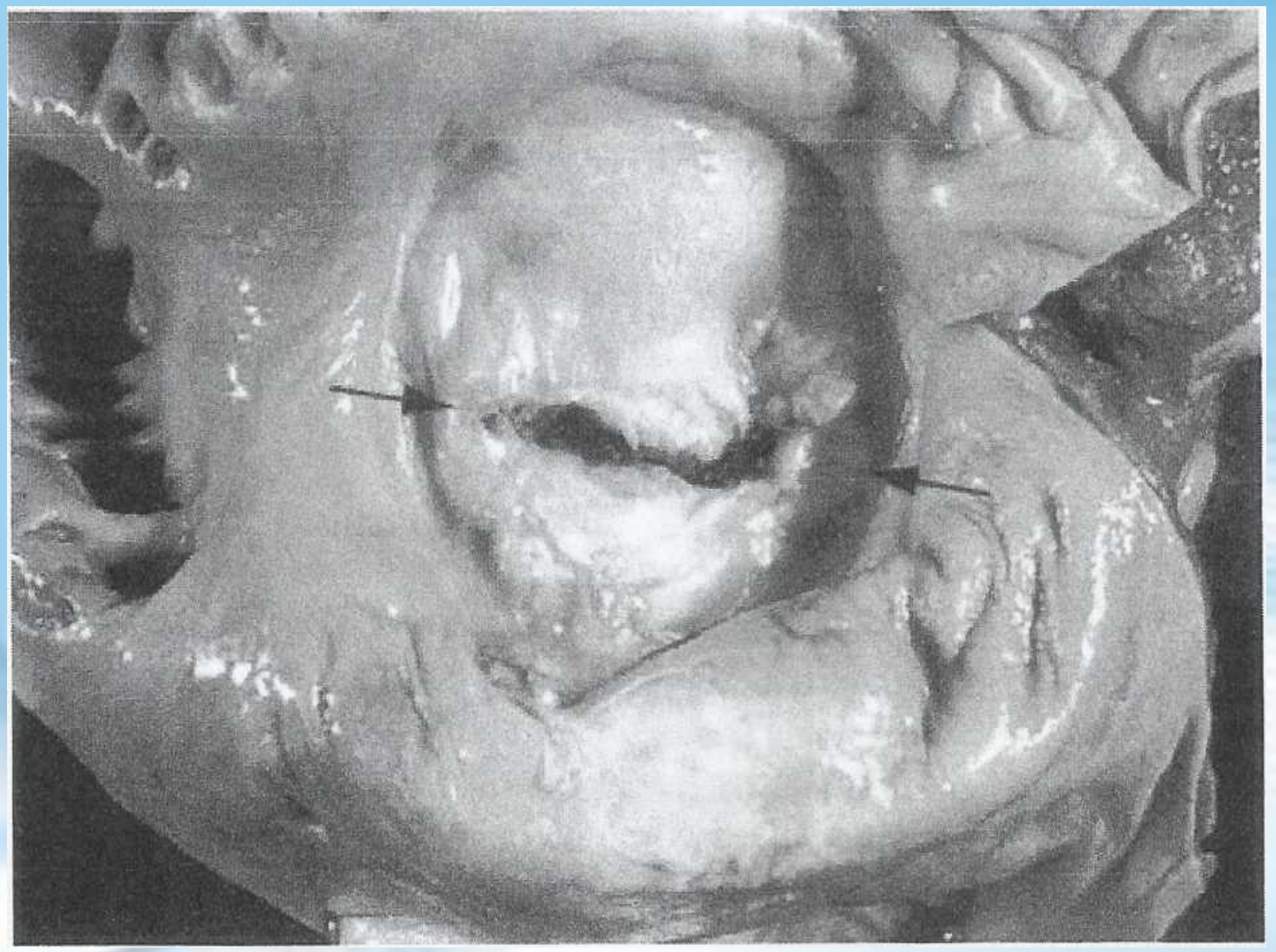
The most common affected connective tissue is that of the heart especially the heart valves (mitral and aortic valves).also the connective tissue of the lung, large joints, subcutaneous tissue .

when the rheumatic fever involve the heart It Is known as **RHEUMATIC HEART DISEASE .**

Chronic rheumatic heart disease (RHD) is characterized by repeated Inflammation with fibrinous resolution.

The cardinal anatomic changes of the valve Include leaflet thickening, commissural fusion, and shortening and thickening of the tendinous cords.

Monthly Injections of long acting penicillin (phenoxymethyi penicillin) must be given for a period of five years In patients having one attack of rheumatic fever. If there is evidence of carditis, the length of therapy may be up to 40 years .



Mitral stenosis with diffuse fibrous thickening and distortion of the valve leaflets in chronic rheumatic heart disease.

Signs and symptoms of RHD

Signs and symptoms

There are no standard symptoms for RHD and it may not show any sign in most cases. However, severe cases may have symptoms like:

- Chest pain
- Joint pain and inflammation
- Heart palpitations
- Shortness of breath
- Fainting (syncope)

Echocardiography is often considered the best method to detect valve disease of the heart. Also a **physical examination** that can detect a heart murmur, and a **chest x-ray** to check for enlargement of the heart.

THE DENTAL MANAGEMENT

1-Check the **BP; Pulse rate; regularity of the pulse;** to be as a Base line record.

2-The most important goal of dental therapy in patients with valvular heart disease is the need to prevent infective endocarditis, so those patients have to be identified and an **antibiotic regimen for prophylaxis of bacterial endocarditis** should be given preoperatively .

N.B. If the patient has a Rh. Fever, but have *NO* HEART INVOLVEMENT , he is treated as a normal patient and no need for antibiotic cover .

THE DENTAL MANAGEMENT

3- Patients with valvular disorders; frequently receive anticoagulant therapy consisting of coumadin derivatives, such as dicumarol and warfarin, then you have to **check the prothrombine time and INR** and adjust the dose of anticoagulants according to lab . findings ,

THE DENTAL MANAGEMENT

4-The local anesthesia :

a- We can use xylocaine *2%* with adrenaline 1/ 100 000 but we have to use an aspirating syringe to avoid the intravascular injection , also we should not use more than two carpoules .

b- also we can use mepvicaine 3% without vasoconstrictor.


Work
Relax
B

THE CONGENITAL HEART DISEASE

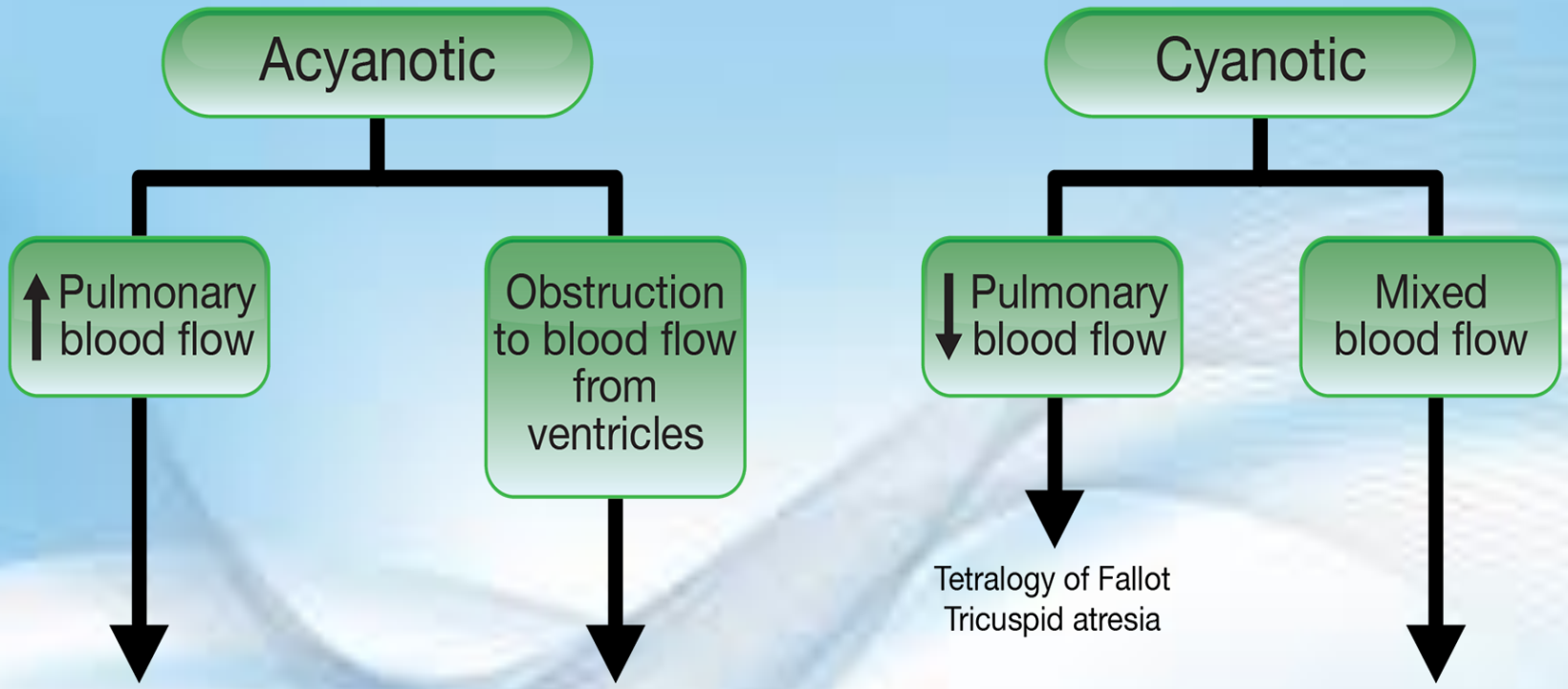
Congenital heart defects are problems with the heart's structure that are present at birth.

These defects can involve the **internal walls of the heart**, **valves inside the heart**, **or the arteries and veins** that carry blood to the heart or out to the body.

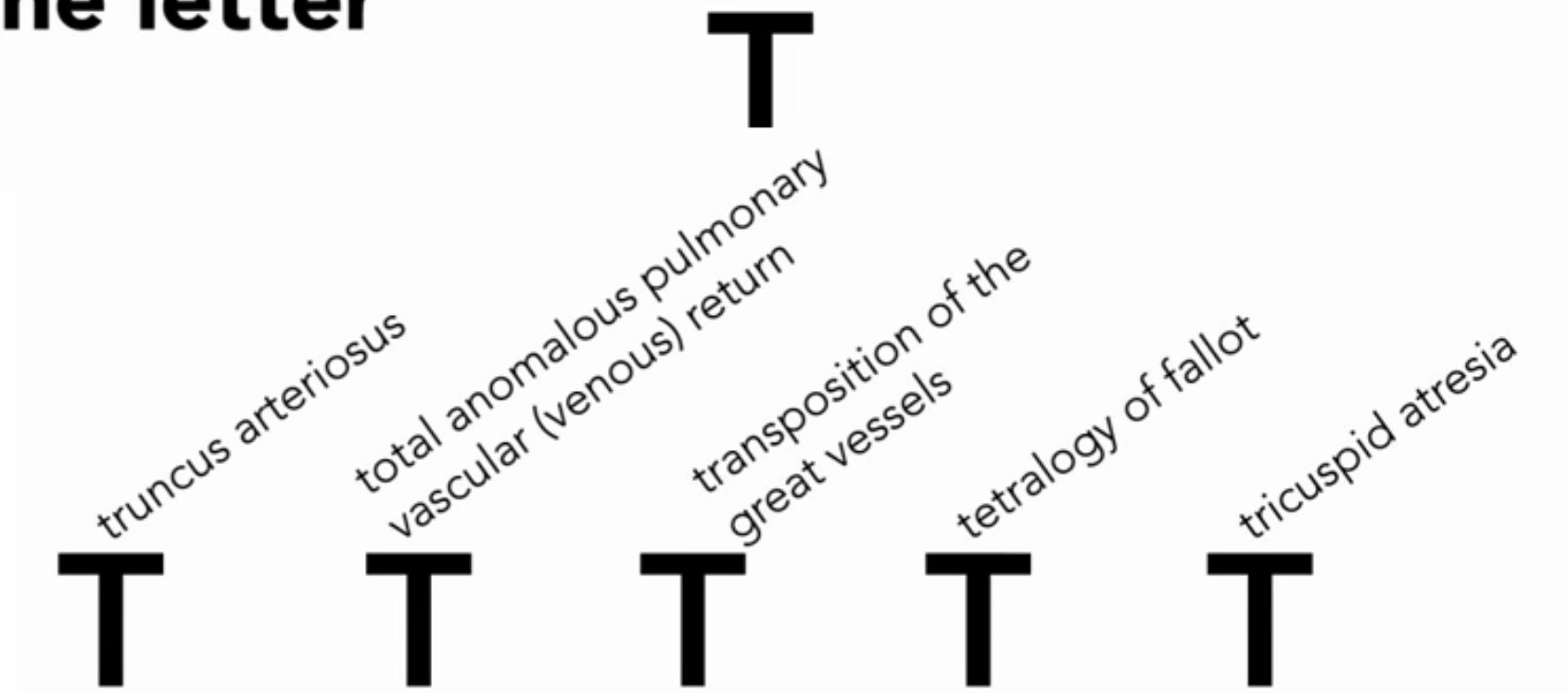
Congenital heart defects change the normal flow of blood through the heart There are many different types of congenital heart defects. They range from simple defects with no symptoms to complex defects with severe, life-threatening symptoms .

Congenital heart defects are the most common type of birth defect, affecting 8 of every 1,000 newborns

Classification of Congenital Heart Disease

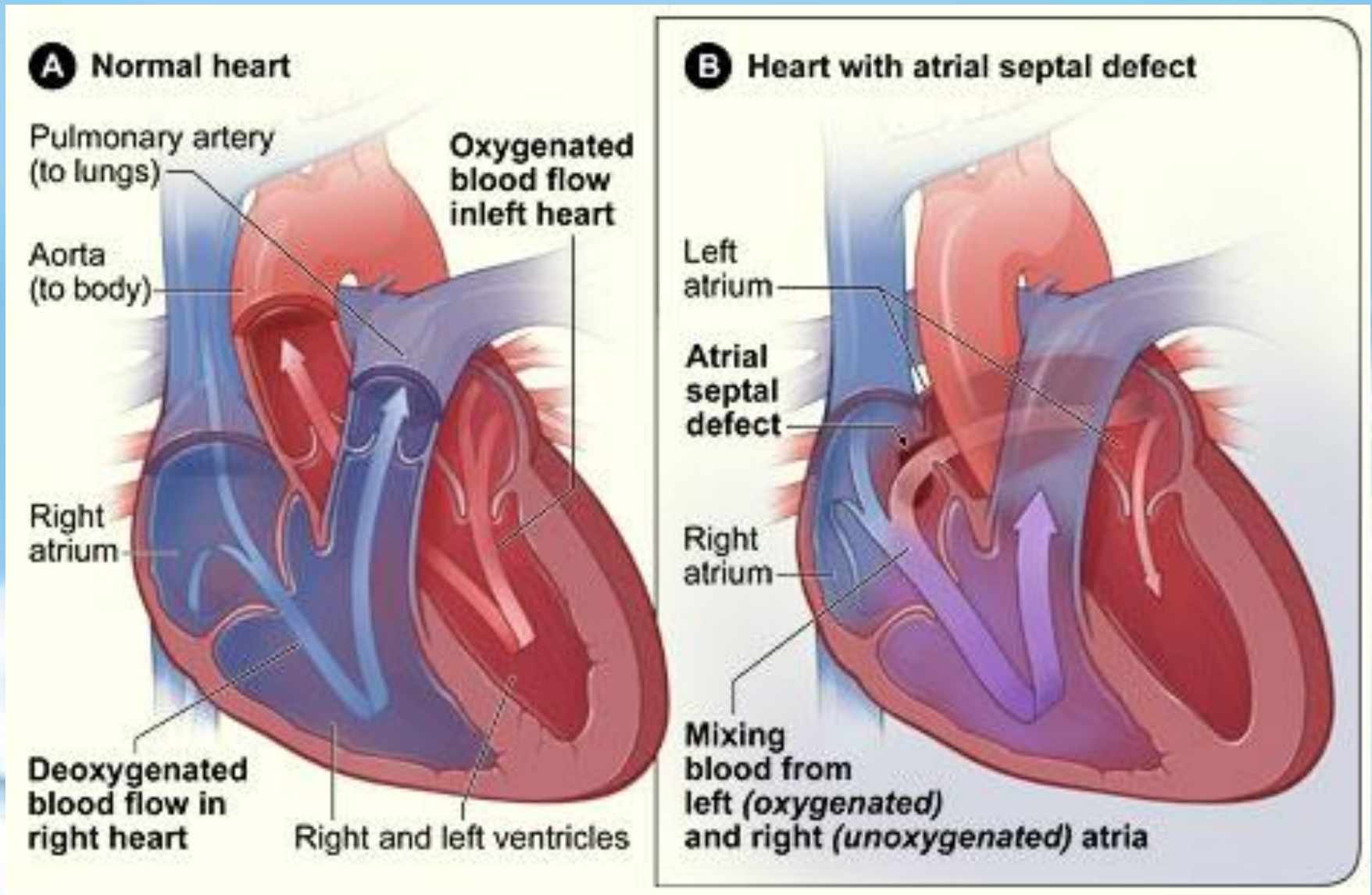


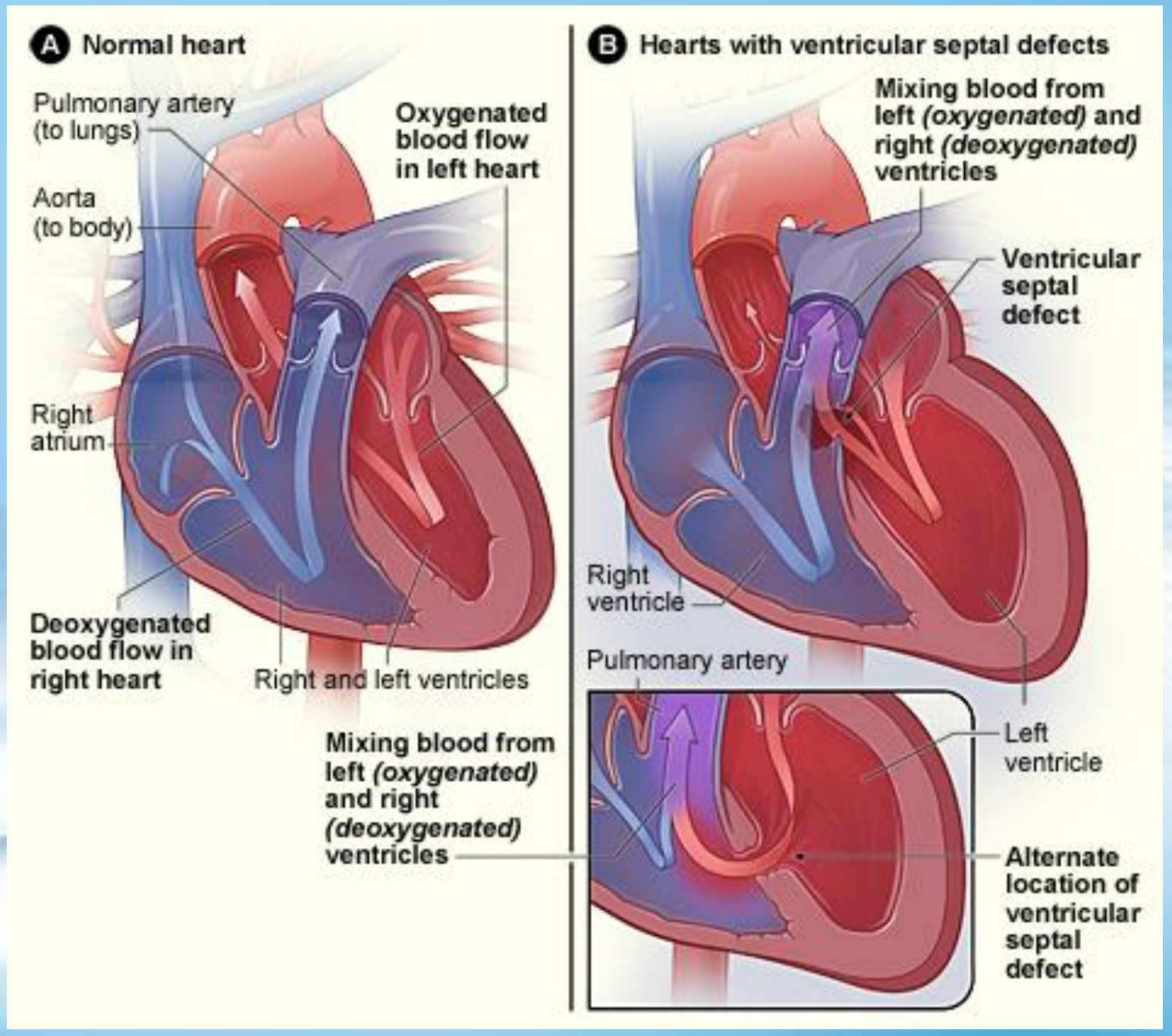
The first thing to remember about the cyanotic congenital heart diseases is that they all begin with the letter



An **aterial septal defect (ASD)** is a hole In the part of the septum that separates the atria - the upper chambers of the heart. This heart defect allows oxygen-rich blood from the left atrium to flow Into the right atrium Instead of flowing to the left ventricle as It should.

A ventricular septal defect (VSD) is a hole in the part of the septum that separates the ventricles . The hole allows oxygen rich blood to flow from the left ventricle into the right ventricle instead of flowing into the aorta and out to the body as it should.


A Normal heart
Pulmonary artery (to lungs)
Aorta (to body)
Right atrium
Oxygenated blood flow inleft heart
Deoxygenated blood flow in right heart
Right and left ventricles
B Heart with atrial septal defect
Left atrium
Atrial septal defect
Right atrium
Mixing blood from left (oxygenated) and right (unoxygenated) atria

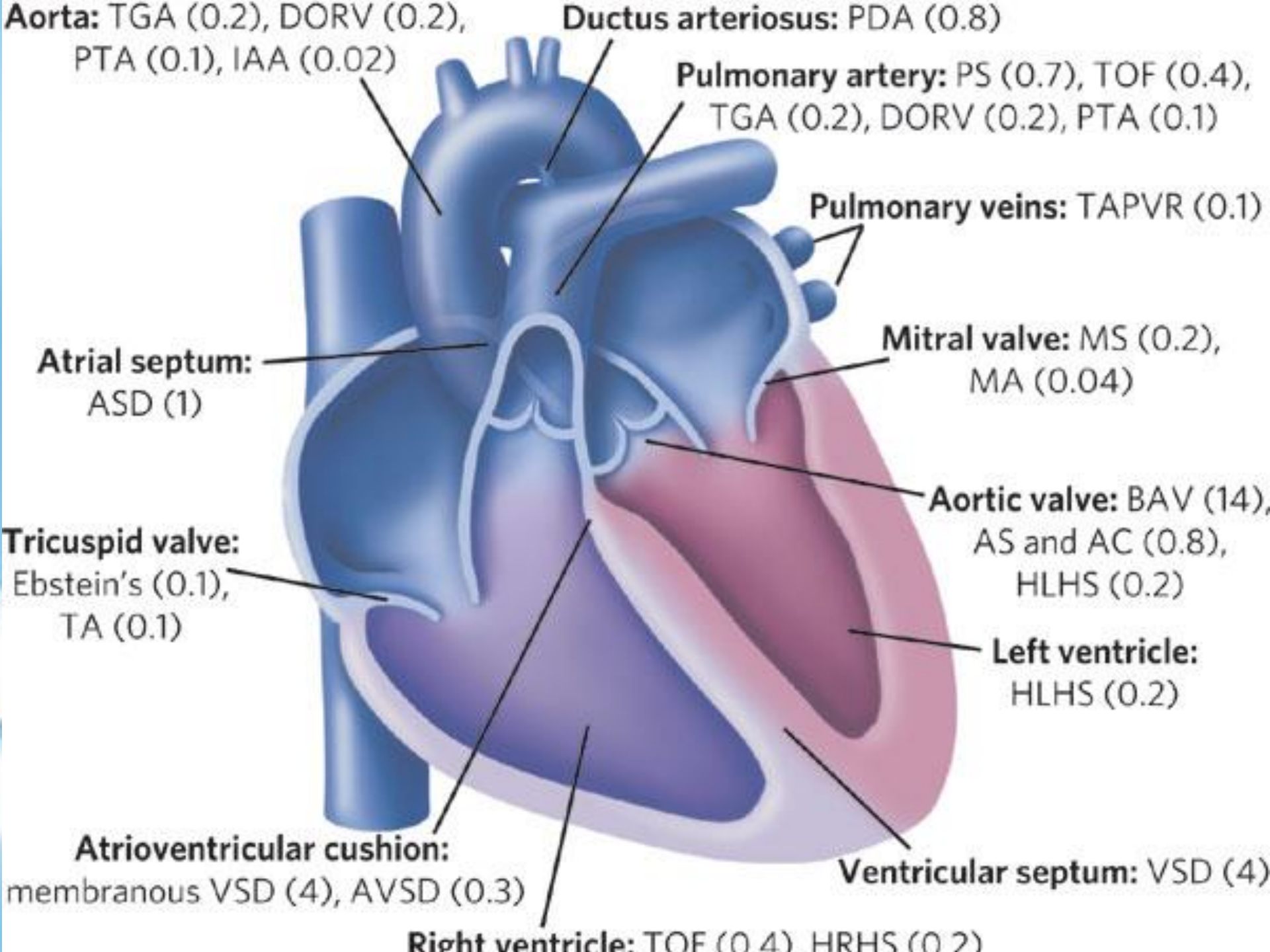

A Normal heart
Pulmonary artery (to lungs)
Aorta (to body)
Right atrium
Oxygenated blood flow in left heart
Deoxygenated blood flow in right heart
Right and left ventricles
Mixing blood from left (oxygenated) and right (deoxygenated) ventricles
B Hearts with ventricular septal defects
Mixing blood from left (oxygenated) and right (deoxygenated) ventricles
Ventricular septal defect
Right ventricle
Pulmonary artery
Left ventricle
Alternate location of ventricular septal defect

Patient ductus arteriosus **(PDA)** characterized by a connection between the aorta and the pulmonary artery

All babies are born with a ductus arteriosus. As the baby takes the first breath, the blood vessels in the lungs open up, and blood begins to flow the ductus arteriosus is not needed to bypass the lungs

Most babies have a closed ductus arteriosus by 72 hours after birth.

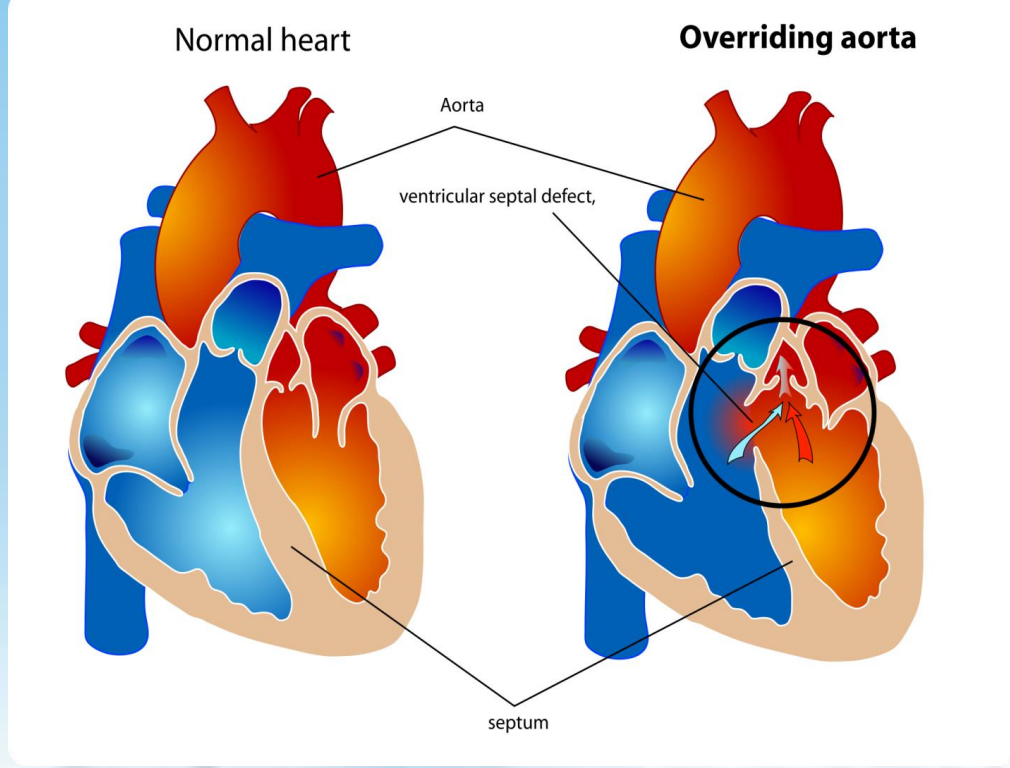
In some babies, however, the ductus arteriosus remains open (patient).

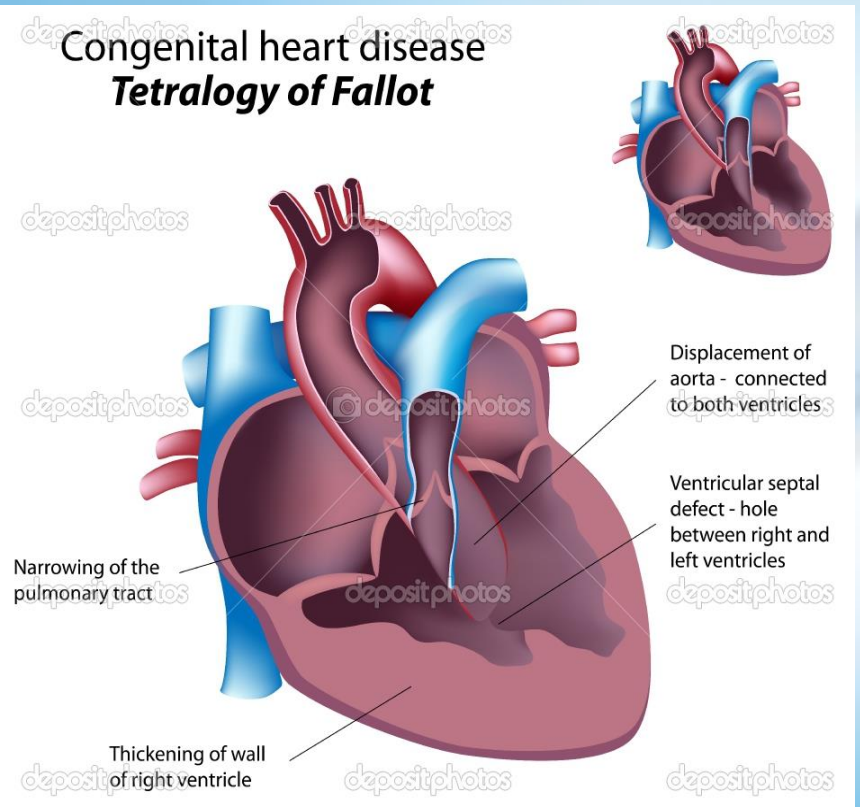

Aorta: TGA (0.2), DORV (0.2), PTA (0.1), IAA (0.02)
Ductus arteriosus: PDA (0.8)
Pulmonary artery: PS (0.7), TOF (0.4), TGA (0.2), DORV (0.2), PTA (0.1)
Pulmonary veins: TAPVR (0.1)
Atrial septum: ASD (1)
Mitral valve: MS (0.2), MA (0.04)
Aortic valve: BAV (14), AS and AC (0.8), HLHS (0.2)
Tricuspid valve: Ebstein's (0.1), TA (0.1)
Left ventricle: HLHS (0.2)
Atrioventricular cushion: membranous VSD (4), AVSD (0.3)
Ventricular septum: VSD (4)
Right ventricle: TOF (0.4), HRHS (0.2)

The most common complex heart defect is **tetralogy of Fallot**, a combination of four defects:

- **Pulmonary valve stenosis.**
- **A large VSD.**
- **An overriding aorta.** The aorta sits above both the left and right ventricles over the VSB, rather than just over the left ventricle. As a result, oxygen poor blood from the right ventricle can flow directly into the aorta instead of into the pulmonary artery to the lungs.
- **Right ventricular hypertrophy.** The muscle of the right ventricle is thicker than usual because of having to work harder than normal.

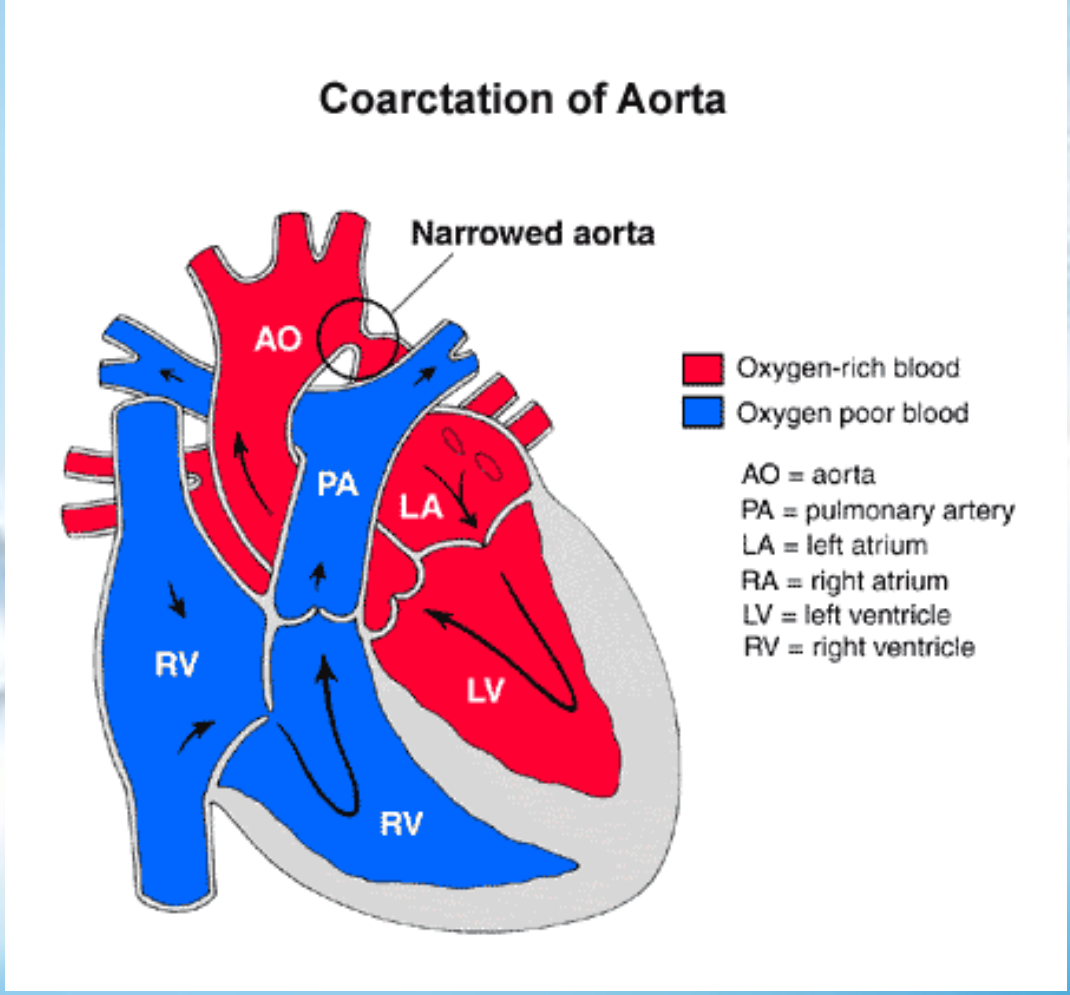
These defects prevent enough blood from flowing to the lungs to get oxygen, while oxygen-poor blood flows directly out to the body.


Normal heart
Overriding aorta
Aorta
ventricular septal defect,
septum


Congenital heart disease
Tetralogy of Fallot
Displacement of aorta - connected to both ventricles
Ventricular septal defect - hole between right and left ventricles
Narrowing of the pulmonary tract
Thickening of wall of right ventricle

Coarctation of aorta (Narrowing of the aorta }

This narrowing restricts the amount of blood to the lower part of the body

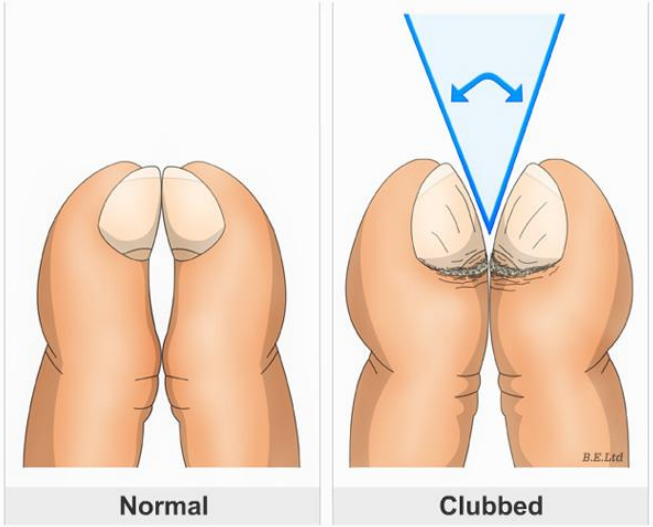


SIGNS AND SYMPTOMES

They depend on the number, type, and severity of the defect and these include :

- Polycythemia with hemorrhagic and thrombotic disorders.
- Cyanosis (a bluish tint to the skin, lips, and fingernails).
- fingers clubbing.
- Fatigue (tiredness)
- Respiratory infections
- Heart murmurs which is an unusual sounds heard during a heartbeat which can be detected by stethoscope.
- Congestive heart failure
- under-developing of limbs and muscles, poor feeding or growth.

Finger clubbing



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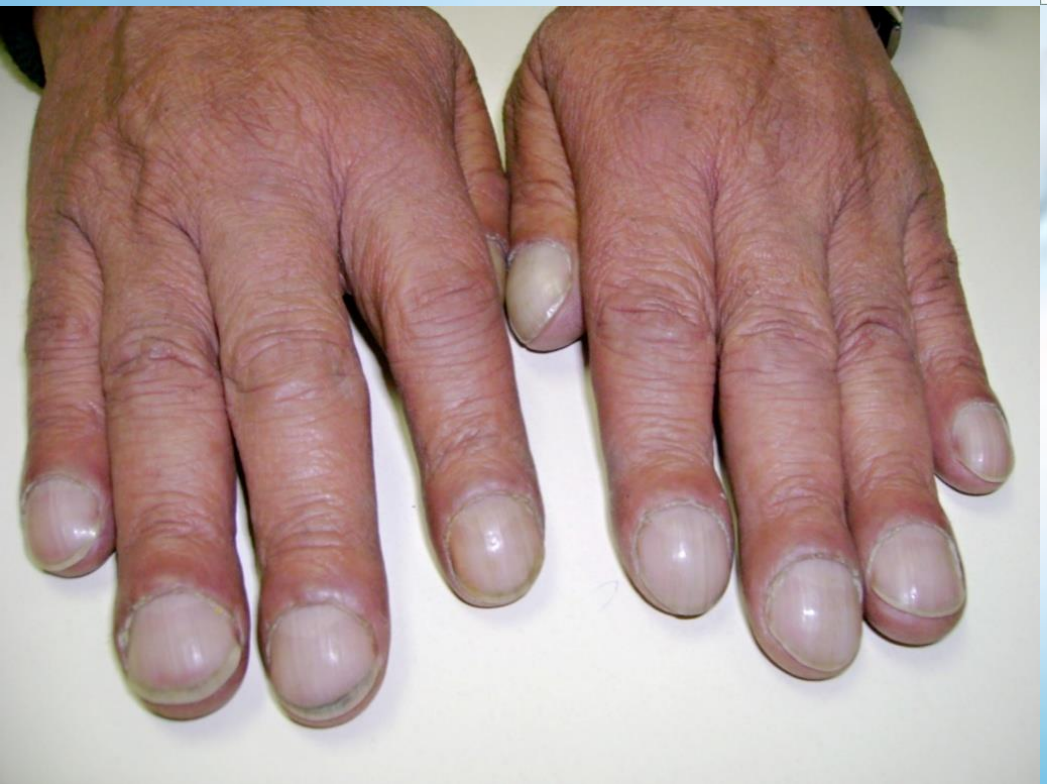
pascalis spyrou



THE DENTAL MANAGEMENT

1- Check the **BP; pulse rate; regularity of the pulse;** to be as a baseline record.

2- **Consult their physician** to know:

- The type of the defect they have
- The medications they use
- If the patient did an open heart surgery to correct the defect or not .
- We have to know if the patient has a residual defect or not after the open heart surgery.

3- **A prophylactic antibiotic** cover is given to those patients before the dental procedure :

a- If the patient has a congenital heart disease **not corrected surgically**, then he should given the antibiotic regimen for prophylaxis of bacterial endocarditis.

b- If the patient has an **open heart surgery** to correct the heart defect, then he should be given the regimen for prophylaxis of bacterial endocarditis **during the first six months** following the open heart surgery .

c- If the patient has **multiple defects ,and /or there is a residual defect the patient,** then he should be given antibiotic prophylaxis **even after six months.**

4- Patients with congenital heart disease ; frequently receive anticoagulant therapy consisting of coumadin derivatives, such as dicumarol and warfarin, then you have to check the **prothromnbine time and INR** and adjust the dose of anticoagulants according to lab . Findings.

N.B. those patients may have thrombocytopenia, Polycythemia so you have to send them for complete blood count.

SURGICALLY CORRECTED CARDIOVASCULAR LESSI0NS

1- Correction of A.S.D. or V.S.D.

2- Ligation or resection of patient ductus arteriosus

3- Surgical correction of coarctation of aorta .

4- Replacement of diseased heart valves (prosthetic heart valves)

5- Coronary artery bypass graft (CABG).

6- Implantation of trans venous pacemaker or defibrillator

7- Heart transplantation

THE DENTAL MANAGEMENT

1- Preparation of the patient for the open heart surgery before the O.H.S. the cardiologist refer the patient to the dentist for full mouth treatment, and the dentist should perform this **full mouth treatment** according to the following guidelines :

a- Patients with **advanced periodontal disease** with very poor oral hygiene, are better advised to extract their teeth and replaced by denture .

b- Patients with **good dental health**, are better advised to keep their teeth with oral hygiene instruction.

c- Patients with **active carious lesions** are advised to do restorative and endodontic treatment for their teeth together with oral hygiene instruction .

Note: the dental treatment should be done with antibiotic regimen Cover.

2-After the open heart surgery, those patients are at risk of:

- infective endocarditis
- infective endarteritis
- During the first six months after the open heart surgery those patients should be under a prophylactic antibiotic cover to prevent bacterial endocarditis .
- bleeding tendency secondary to the **anticoagulants**.

a- During the **first six months** after the open heart surgery those patients should be under a prophylactic antibiotic cover to prevent bacterial endocarditis .

b- **After six months** of open heart surgery, we have to consult the physician for the need of antibiotic cover or not. because some cases are need a prophylaxis antibiotic even after six months such as :

- prosthetic heart valves
- residual defect after correction of congenital heart disease .
- in-surgical correction of multiple heart defects such as tetralogy of fallot.

c- **Prothrombine time** should be monitored because the patient using anticoagulants and the PT should be at least 1.5 — 2 times of normal. Also **INR** should be checked to avoid post operative bleeding


Thank
You

Dental management of patients with cardiovascular diseases

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CONGESTIVE HEART FAILURE

Heart failure is the inability of the heart to function efficiently as a pump, which results in inadequate emptying of the ventricles during systole or incomplete filling of the ventricles during diastole. This in turn results in a decrease in cardiac output, with an inadequate volume of blood supplied to the tissues also the venous return to the heart is decreased , causing systemic congestion .

HF may involve one or both ventricles., heart failure represents the end stage of many of the cardiovascular diseases such as Ml, Angina

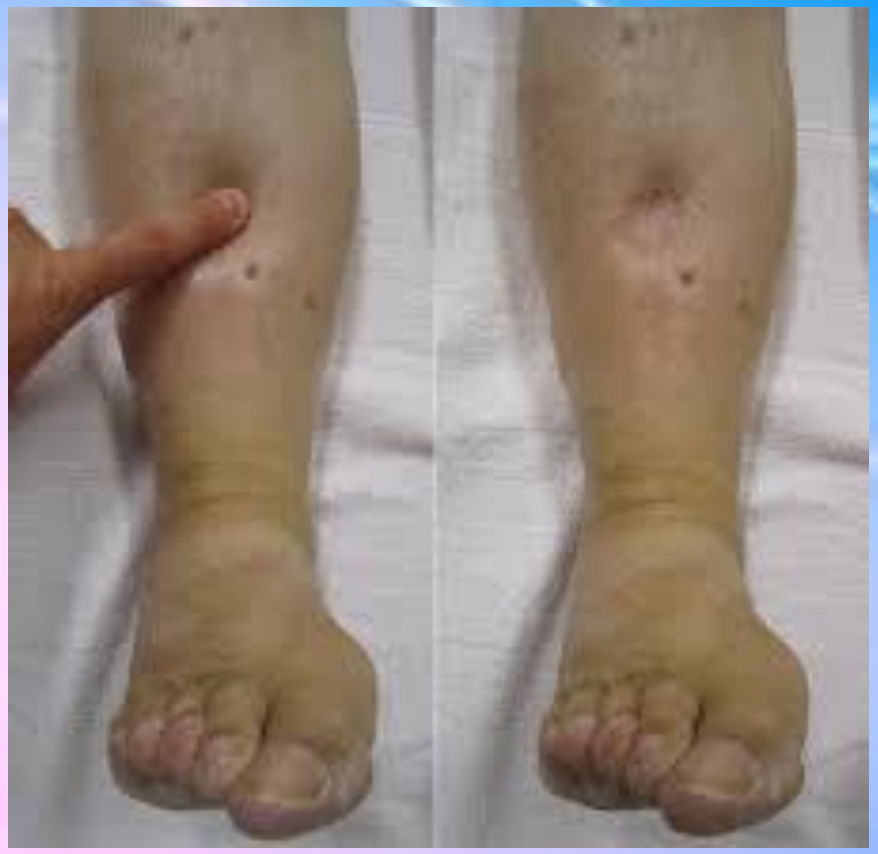
Common Causes of Heart Failure

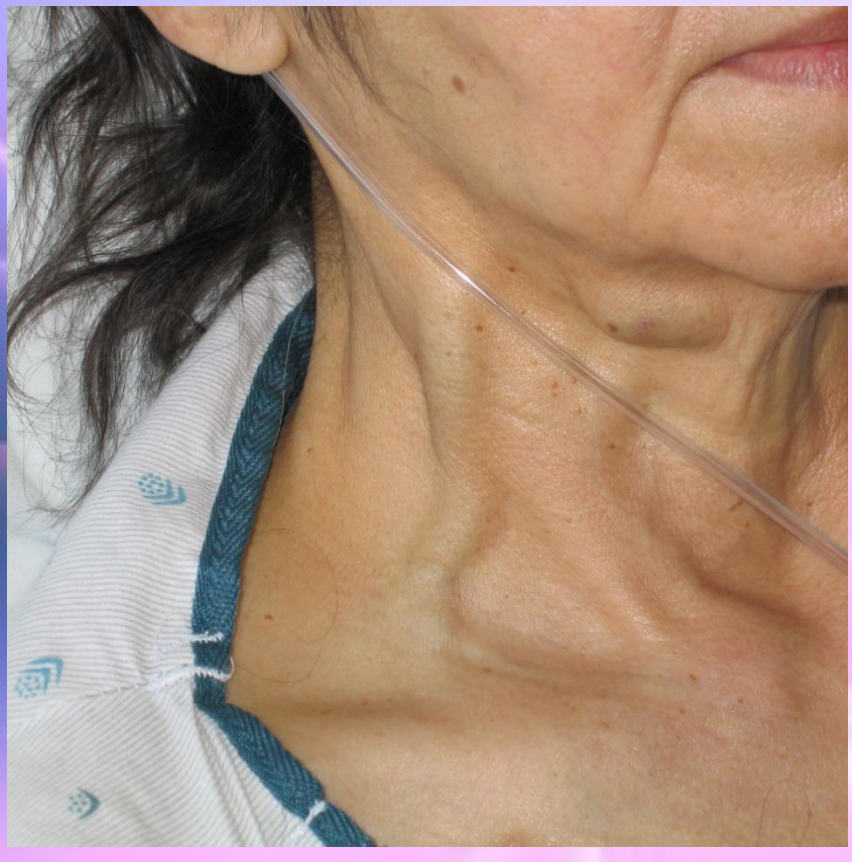
- Coronary heart disease
- Hypertension
- Cardiomyopathy
- Valvular heart disease
- Myocarditis
- Infective endocarditis
- Congenital heart disease
- Pulmonary hypertension
- Pulmonary embolism
- Endocrine disease

Signs & Symptoms

- Dyspnea (shortness of breath)
- Fatigue and weakness
- Orthopnea (dyspnea in recumbent position)
- Acute pulmonary edema (cough or progressive dyspnea)
- Exercise intolerance (inability to climb a flight of stairs)
- Dependent edema (swelling of feet and ankles after standing or walking)
- Hyperventilation followed by apnea during sleep
- Heart murmur
- Increased venous pressure
- Distended neck veins
- Cyanosis
- Clubbing of fingers

Pitting edema in a patient with heart failure. A depression (“pit”) remains in the edematous tissue for some minutes after firm fingertip pressure is applied.





Distended jugular vein

NYHA Classification of Heart Failure (New York Heart Association)

Class I: *No* limitation of physical activity. No dyspnea, fatigue, or palpitations with ordinary physical activity.

Class II: Slight limitation of physical activity. These patients have fatigue, palpitations, and dyspnea with ordinary physical activity but are comfortable at rest.

Class III: Marked limitation of activity. Less than ordinary physical activity results in symptoms, but patients are comfortable at rest.

Class IV: Symptoms are present at rest, and any physical exertion exacerbates The symptoms.

THE DENTAL MANAGEMENT

1- We have to keep in our mind that HF is a symptom complex that is the end result of an underlying disease such as coronary heart disease, hypertension, or cardiomyopathy; therefore, **the cause of HF must be identified and manage the patient according to the underlying cause .**

2- Asymptomatic heart failure **(class I, and II)** they are considered as intermediate risk for occurrence of a serious event (acute Ml, unstable angina, or sudden death) during treatment., so they **can receive any routine outpatient dental care.**

3- patients with heart failure **(class III, or IV)** they are considered as a major risk for the occurrence of a serious event (acute MI unstable angina, or sudden death) during treatment. So that they are not candidates for elective dental care.

But if the dental care becomes necessary for the **control of pain ,or infection** ,in this case it is advisable to be done **in a hospital** dental clinic under **supervision of physician** together with establishment of an **I.V. line, pulse oximeter** ,and **intraoperative blood pressure** checking.

4- Measuring and **evaluating vital signs** (i.e., pulse rate and rhythm, blood pressure, respiratory rate).

5- **The patient is placed in a semi supine or upright position and avoid the supine position because of pulmonary edema may lead to dyspnea .**

6-Drug considerations:

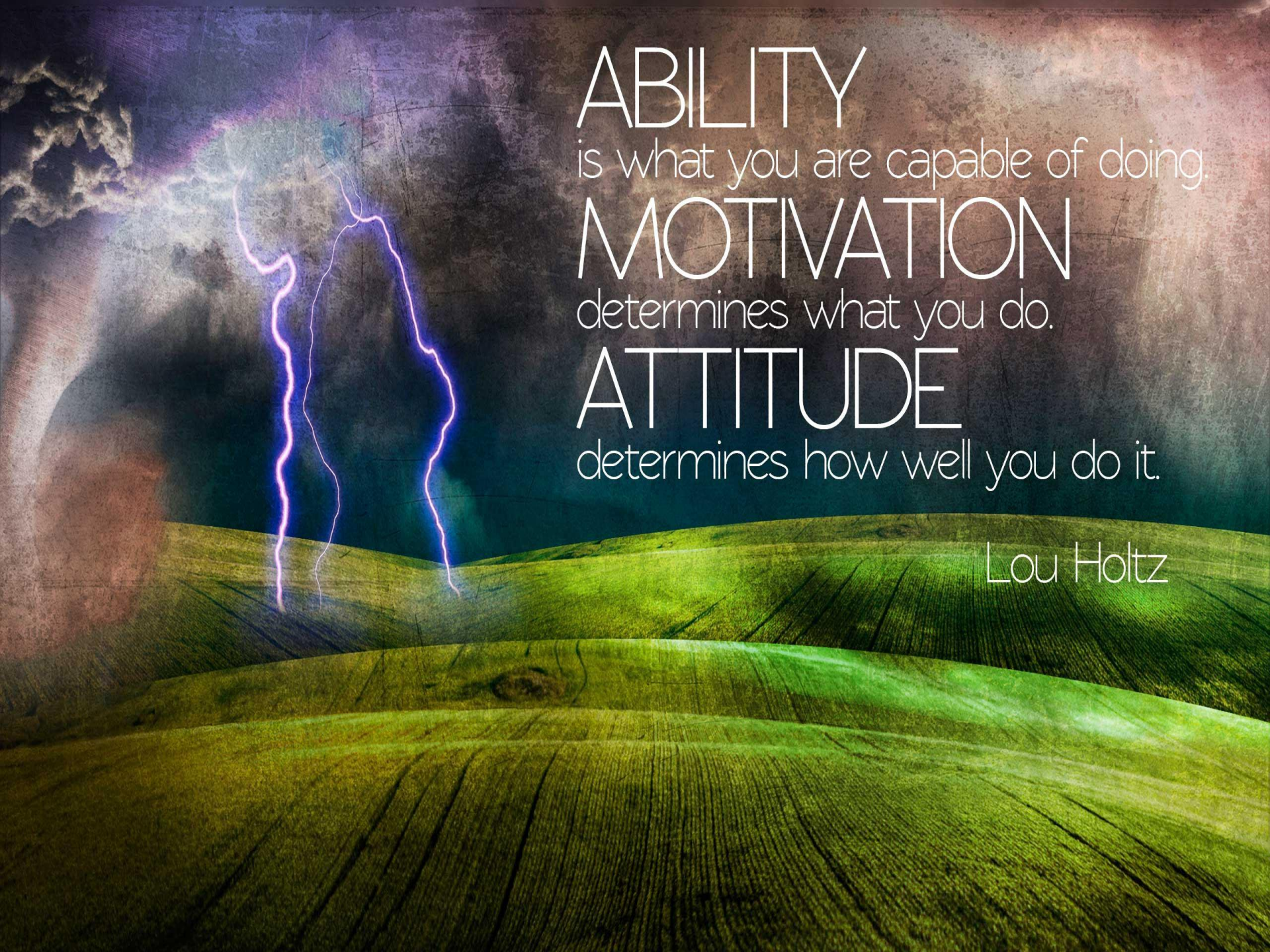
a- All medications that are being taken should be identified

b-For patients taking digoxin, in this case epinephrine in local anesthesia should be avoided, because the combination can potentially precipitate **arrhythmias**. If it is considered essential to use epinephrine, it should be used cautiously. A maximum of two cartridges of *2%* lidocaine with 1:100,000 epinephrine is recommended, with care taken to avoid intravascular injection.

C-For patients who are class III or IV, vasoconstrictors should be avoided; we can use **mepvicaine 3% without vasoconstrictor**, however, if their use is considered essential, it should be discussed with the physician.

7- If the patient use an anticoagulant, check the **PT, INR, BLEEDING TIME** .

8-Discus the need for a **prophylaxis antibiotic regimen** , to prevent the bacterial endocarditis, when the cause of congestive heart failure is Congenital heart disease. Or valvular heart disease.


ABILITY
is what you are capable of doing.
MOTIVATION
determines what you do.
ATTITUDE
determines how well you do it.
Lou Holtz

CARDIAC ARRHYTHMIAS

Cardiac arrhythmia, which refers to any **variation in the normal heartbeat,** includes disturbances of rhythm, rate, or the conduction pattern of the heart.

Cardiac arrhythmias are present in a significant percentage of the population, many of whom will seek dental treatment. Most arrhythmias are of little concern to the patient or the dentist; however, some can produce symptoms, and a few may be life threatening.

It has been shown that potentially fatal arrhythmias can be precipitated by strong emotion such as anxiety or anger, as well as by various drugs, both of which can be experienced in the dental setting,, Therefore, patients with significant arrhythmias must be identified before undergoing dental treatment.

ETIOLOGY

- Primary cardiovascular disease as MI, angina , congenital heart disease
- Chronic obstructive Pulmonary disorders
- Systemic diseases {thyroid disease)
- Drug related side effect ((digoxin toxicity)
- Electrolyte Imbalances

SIGNS & SYMPTOMS

- Slow heart rate (<60 beats/min) Bradycardia is defined as a heart rate less than 60 beats per minute
- Fast heart rate (>100 beats/min) Tachycardia in an adult is defined as a heart rate greater than 100 beats per minute
- Irregular rhythm
- Palpitations
- Fatigue
- Dizziness
- Syncope
- Angina
- Congestive heart failure which lead to : Shortness of breath, Orthopnea, Peripheral edema

Identifying Patients With Cardiac Arrhythmias

1-Patients with a known **history of arrhythmia** should be questioned as to the type of arrhythmia (if known), how it is being treated, medications being taken, presence of a pacemaker or defibrillator, effects on their activity, and stability of their disease.

2-Patients with a **history of other heart, thyroid, or chronic pulmonary disease** should be identified, as these may be a cause of or contributor to the arrhythmia,

3-Patient does not report an arrhythmia, but may be **taking one or more of the Anti arrhythmic Drugs**

4-Vital signs are suggestive of arrhythmia (rapid pulse rate, slow pulse rate, irregular pulse)

DENTAL MANAGEMENT

1-Identification of the patients susceptible to developing cardiac arrhythmia, by:

a-history , especially the patients with a history of significant heart disease , thyroid disease, chronic pulmonary disease, open heart surgery,

b-Clinical findings seen in patient with Vital signs that are suggestive of arrhythmia (rapid pulse rate, slow pulse rate, irregular pulse).

2-if the patient is **well controlled** (after consultation with the physician he can **receive any routine dental treatment** ,but Complex and lengthy dental procedures should be spread over several appointments to avoid overstressing the patient.

But if the patient is **not controlled** you have to avoid the routine dental treatment and if the patient needs **emergency treatment** for the control of pain , or infection ,in this case it should be performed as conservatively as possible, and it Is advisable to be done **in a hospital** dental clinic under supervision of physician together with establishment of an I.V. **line ,pulse oximeter** ,and **intraoperative blood pressure** checking .

3-Consultation with the physician for:

a-determine the cause and the severity of the arrhythmia .

b-the need for **prophylactic antibiotic** cover especially when the arrhythmia is due to congenital or valvular heart disease.

c-patients with atrial fibrillation **may use warfarin** so, we have to **check the P.T &INR**, and a controlled values should be obtained before any surgical procedure .

4-Reduce the stress & anxiety by;

a-establishment of good relation with the patient

b-use of valium preoperatively

c-short appointments in the morning,

d-comfortable chair position

e-we may use nitrous oxide Inhalation in case of apprehensive patient.

5- **Local anesthesia:**

a- For patients taking digoxin this case epinephrine should be avoided because the combination can potentially precipitate arrhythmias

b-in the well controlled cases you can use xylocaine with adrenaline 1/100 000 but no more than two carpoules with the use of aspirating syringe . (note : The use of vasoconstrictors in local anesthetics poses potential problems for patients with arrhythmias because of the possibility of precipitating cardiac tachycardia or another arrhythmia) .

6-Patients with **implanted pacemaker:** we should **avoid** the use of devices with electromagnetic field such as **ultrasonic scalers and electro surgery.**

7-If the patient developed cardiac arrhythmia during the dental procedure ,then you have to be ready for **cardiopulmonary resuscitation**, and call for an emergency medical care.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Dental management of patients with Diabetes mellitus

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Diabetes mellitus

is a chronic disease complex with **metabolic and vascular components**, the metabolic disorder of carbohydrate metabolism due to disturbance of the normal insulin mechanism (characterized by hyperglycemia). the vascular component affect both macro vascular & micro vascular causing serious diseases involved heart, kidney, eyes, lower limbs & neuropathies

Etiology

Diabetes mellitus may be the result of any of the following:

- **Genetic disorder**
- **Primary destruction of islet cells** through inflammation, cancer, or surgery
- **An endocrine condition** such as hyper pituitarism or Hyperthyroidism.
- **An iatrogenic disease** that occurs after steroids have been administered.

Pathophysiology

Insulin is a hormone secreted by the B-cells of the pancreatic islets of langerhans , it promotes the entry of glucose into the body's cells.

Glucose is the most important stimulus for insulin secretion.

Insulin remains in circulation for only several minutes (half-life 4 to 8 minutes); it then interacts with target tissues and binds with cell surface insulin receptors. Secondary intracellular messengers are activated and interact with cellular effector systems, including enzymes and glucose transport proteins. Lack of insulin or insulin action allows glucose to accumulate in the tissue fluids and blood.

Types of diabetes mellitus

Type 1 DM results from the body's failure to produce insulin, and presently requires the person to inject insulin or wear an insulin pump. This form was previously referred to as "**insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes**".

Type 2 DM results from insulin resistance, a condition in which cells fail to use insulin properly, sometimes combined with an absolute insulin deficiency. This form was previously referred to as **non insulin-dependent diabetes mellitus (NIDDM) or "adult-onset diabetes".**

Gestational Diabetes occurs when pregnant women without a previous diagnosis of diabetes develop a high blood glucose level. It may precede development of type 2 DM.

Signs and Symptoms

In patients with type 1 diabetes, the onset of symptoms is sudden and more acute, symptoms include ,

- **polydipsia , polyuria , polyphagia**,
- **loss of weight, loss of strength**,
- **marked irritability, drowsiness. and malaise.**
- Other signs and symptoms related to the complications of diabetes include skin lesions, cataracts, blindness, hypertension, chest pain, and anemia.

N.B. The rapid onset of myopia in an adult is highly suggestive of diabetes mellitus.

Investigations

- **Fasting venous blood glucose.**
- 126 mg/100 mL or greater on two or more occasions.
- **Two-hour postprandial glucose (random)** :
- 200 mg/100 mL or higher on two or more occasions are diagnostic of diabetes mellitus .
- **Random blood sugar and glycosilated hemoglobin (HbA1c) (**normal 6-6.5)

Medical management

- Diabetes mellitus is not a curable disease.

- Patients with **Type 1 diabetes are treated with insulin,** available human insulin& analogues include rapid-acting, short-acting, intermediate-acting, and long-acting preparations. Rapid acting and short-acting preparations are used as meal (or bolus) insulin and intermediate-acting and long-acting insulin serve as basal insulin.

- The management of **Type 2 diabetes** involves:
  - ✓ **lifestyle interventions(weight loss, diet control , and physical activity.)**
  - ✓ **drug therapy (anti-hypoglycemic agents),**
  - ✓ **control of risk factors for cardiovascular disease.** This includes control of blood glucose levels, blood pressure, lipid levels, and anti-platelet therapy as indicated.

Anti-hypoglycemic agents

Oral metformin (Glucophage),

- Its major action is to reduce hepatic insulin resistance, gluconeogenesis, and glucose release .
- It does not increase insulin levels and thus is not associated with significant risk of hypoglycemia .
- It is contraindicated in patients with symptomatic congestive heart failure, renal insufficiency or hepatic insufficiency.

Insulin Shock

Patients who are treated with insulin must closely adhere to their diet. If they **fail to eat** in a normal manner but continue to take their regular insulin injections, they may experience **a hypoglycemic reaction caused by an excess of insulin (insulin shock**).

A hypoglycemic reaction also may be due to an **overdose of insulin or an oral hypoglycemic agent**. Reaction or shock caused by excessive insulin usually occurs in three well-defined stages, each more severe and dangerous than the one preceding it.

Signs and-Symptoms of Insulin Reaction

MILD STAGE

Hunger, Weakness, Tachycardia , Pallor , Sweating , Paresthesia

MODERATE STAGE

Incoherence, Uncooperativeness, Lack of judgment, Poor orientation

SEVERE STAGE

Unconsciousness, Tonic or clonic movements, Hypotension Hypothermia, Rapid thready pulse.

Complications

Vascular complications result from microangiopathy and atherosclerosis.

- Ketoacidosis (type 2 diabetes)
- Diabetic retinopathy/blindness and Cataracts
- Diabetic nephropathy/renal failure.
- Accelerated atherosclerosis (coronary heart disease)
- Ulceration and gangrene of feet.
- Diabetic neuropathy (dysphagia, gastric distention, diarrhea, muscle weakness/cramps, numbness, tingling, deep burning pain).
- Early death.

Oral manifestations of D.M.

In poorly controlled diabetes , patient has:

xerostomia ,

- bacterial ,viral, frugal infections including candidiasis & more rare are mucormycosis ,
- poor wound healing ,
- high caries incidence,
- gingivitis & periodontal disease ,
- periapical abscesses
- burnning mouth .

Oral manifestations of D.M.

- lichen planus may also more in diabetic patient due to alter in immune system.
- Diabetic neuropathy may lead to Paresthesia & tingling, numbness or pain caused by pathologic changes involving nerves in oral region .

N.B. Early diagnosis & treatment may allow for regression of the symptoms but in long standing cases the changes will be irreversible .

Detection of the Patient With Diabetes (known D.M.)

Detection by history:

- Are you diabetic?
- What medications are you taking?
- Are you being treated by a physician?

Establishment of severity of disease and degree of control.

- When were you first diagnosed as diabetic?
- What was the level of the last measurement of your blood glucose?
- What is the usual level of blood glucose for you?
- How are you being treated for your diabetes?
- How often do you have insulin reactions?
- How much insulin do you take with each injection, and how often do you receive injections ?
- Do you test your urine for glucose?
- When did you last visit your physician?
- Do you have any symptoms of diabetes at the present time?

Detection of the Patient With Diabetes (undiagnosed patients)

- History of signs or symptoms of diabetes or its complications.
- High risk for developing diabetes:
  - ✓Parents who are diabetic .
  - ✓Gave birth to one or more large babies
  - ✓History of spontaneous abortions .
  - ✓obese .
  - ✓Over 40 years of age .
- Referral or screening test for diabetes.

Dental Management of Diabetic Patient

1- Non-insulin-dependent patient and Insulin-controlled patient : **If diabetes is well-controlled**, all dental procedures can be performed without special precautions.

- Morning appointments are usually best.
- Patient advised to take usual insulin dosage and normal meals on day of dental appointment, information confirmed when patient comes for appointment.
- advise patient to inform dentist or staff if symptoms of insulin reaction occur during dental visit.

- Glucose source (orange juice) should be available to the patient if symptoms of insulin reaction occur.

- If extensive surgery is needed:
  - ✓ Consult with patient's physician concerning dietary needs during postoperative period.
  - ✓ Antibiotic prophylaxis can be considered for patients with brittle diabetes and those taking high doses of insulin who also have chronic states of oral infection.

2- **If not well-controlled** (i.e., does not meet any of above criteria; fasting blood glucose <70 mg/dL or >200 mg /dL

- Provide appropriate emergency care .
- Request referral for medical evaluation, management, and risk factor modification
  - ✓If symptomatic, seek immediate referral.
  - ✓If asymptomatic, request routine referral.

3- **Patient with Diabetes and Acute Oral Infection;**

- Non-insulin-controlled patients; may require insulin, and consultation with physician .
- Insulin-controlled patients; usually require increased dosage of insulin; consultation with physician required.
- Patient with brittle diabetes or receiving high insulin dosage, should have culture taken from the infected area for antibiotic sensitivity testing,& Antibiotic therapy should be initiated . In cases of poor clinical responses to the first antibiotic , a more effective antibiotic is selected according to sensitivity test results.
- Infection should be treated with the use of standard methods
  - ✓ Warm intraoral rinses .
  - ✓ Incision and drainage ;
  - ✓ Pulpotomy , pulpectomy , extractions, etc.
  - ✓ Antibiotics .

Dental Management of Diabetic Patient

4- Local Anesthetics and Epinephrine

However epinephrine has a pharmacologic effect opposite that of insulin, so blood glucose could rise with the use of epinephrine .

In diabetic patients with hypertension, myocardial infarction, cardiac arrhythmia caution may be indicated with epinephrine. Guide- lines for these patients are similar to those for Patients with cardiovascular conditions and may be even more strict for those with diabetes and cardiovascular conditions, as along with determination of functional capacity.


Thank You!

Pregnancy and Breast Feeding

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A pregnant patient, although not considered medically compromised, poses a unique set of management considerations for the dentist. Dental care must be rendered to the mother without adversely affecting the developing fetus, and although routine dental care generally is safe for the pregnant patient, the delivery of such care involves some potentially harmful elements, including the use of ionizing radiation and certain drugs. Thus, **the prudent practitioner must balance the beneficial aspects of dentistry with potentially harmful procedures by minimizing or avoiding exposure of the patient (and the developing fetus).**

Additional considerations arise during the postpartum period if the mother elects to breast feed her infant. Although most drugs are only minimally transmitted from maternal serum to breast milk, and the infant's exposure is not significant, the dentist should avoid using any drug that is known to be harmful to the infant.

OVERVIEW OF PREGNANCY

Physiology and Complications

- **Endocrine changes** are the most significant basic alterations that occur with pregnancy. They result from the increased production of maternal and placental hormones and from modified activity of target end organs.
- **Fatigue** is a common physiologic finding during the **first trimester** that may have a psychologic impact. A tendency toward **syncope and postural hypotension** also has been noted.
- During the **second trimester**, patients typically have a sense of **well-being** and relatively few symptoms.
- During the **third trimester**, increasing **fatigue and discomfort and mild depression** may be reported.

- During late pregnancy, a phenomenon known as **supine hypotensive syndrome** may occur that manifests as an abrupt fall in blood pressure, bradycardia, sweating, nausea, weakness, and air hunger when the patient is in a supine position. Symptoms and signs are caused by **impaired venous return** to the heart resulting from compression of the inferior vena cava by the gravid uterus. This leads to decreased blood pressure, reduced cardiac output, and impairment or loss of consciousness. The remedy for the problem is for the patient to roll over onto her left side, which lifts the uterus off the vena cava. Blood pressure should rapidly return to normal.

- Blood changes in pregnancy include **anemia** and a decreased hematocrit value. Anemia occurs because blood volume increases more rapidly than red blood cell mass. As a result, a fall in hemoglobin and a marked need for additional folate and iron occur. A majority of pregnant women have insufficient iron stores—a problem that is exaggerated by significant blood loss. However, there is disagreement over whether or not to routinely provide iron supplementation.
- Although changes in platelets are usually clinically insignificant, most studies show a **mild decrease in platelets** during pregnancy. Several blood clotting factors, especially fibrinogen and factors VII, VIII, IX, and X, are increased. As a result of the increase in many of the coagulation factors, combined with venous stasis, pregnancy is associated with a **hypercoagulable state**. Interestingly, however, the prothrombin time, activated partial thromboplastin time, and thrombin time **all fall slightly** but remain within the limits of normal nonpregnant values.

- Several white blood cell (WBC) and immunologic changes occur.
- The **WBC count increases** progressively throughout pregnancy, primarily because of an increase in **neutrophils**, and is nearly doubled by term.The reason for the increase is unclear but may involve elevated estrogen and cortisol levels. **This increase in neutrophils may complicate the interpretation of the complete blood count during infection.**

- Changes in respiratory function during pregnancy include elevation of the diaphragm which decreases the volume of the lungs in the resting state, thereby reducing total lung capacity by 5% and the functional residual capacity (FRC), the volume of air in the lungs at the end of quiet exhalation, by 20%.
- These ventilatory changes produce an increased rate of respiration **(tachypnea) and dyspnea that is worsened by the supine position**. Thus, it is not surprising that sleep during pregnancy is impaired, especially during the third trimester.

- Pregnancy predisposes the expectant mother to an increased appetite and often a craving for unusual foods. As a result, the diet may be unbalanced, high in sugars, or nonnutritious. **This can adversely affect the mother's dentition and also contribute to significant weight gain. Taste alterations and an increased gag response are common as well**. The pH and production of saliva are probably unchanged. No evidence exists that pregnancy causes or accelerates the course of dental caries**.**
- **Nausea and vomiting, or "morning sickness**," may complicate up to 70% of pregnancies.

The general pattern of fetal development should be understood when dental management plans are being formulated. Normal pregnancy lasts approximately 40 weeks. During the first trimester, organs and systems are formed (organogenesis). Thus, the fetus is most susceptible to malformation during this period.

After the first trimester, the major aspects of formation are complete, and the remainder of fetal development is devoted primarily to growth and maturation. **Thus, the chances of malformation are markedly diminished after the first trimester**. A notable exception to this relative protection is the fetal dentition, which is susceptible to malformation from toxins or radiation, and to tooth **discoloration caused by administration of tetracycline**.

Complications of pregnancy are infrequent when appropriate prenatal care is provided and the mother is healthy.

Common complications include **infection, enhanced inflammatory response, glucose abnormalities, and hypertension**. Each of these entities increases the risks for preterm delivery, perinatal mortality, and congenital anomalies.

Insulin resistance is a contributing factor to the development of gestational diabetes mellitus (GDM), which occurs in 2% to 6% of pregnant women. GDM increases the risks for infection and large birth weight babies.

Hypertension is of particular interest because it can lead to end organ damage or **preeclampsia**, a clinical condition of pregnancy that manifests as hypertension, proteinuria, edema, and blurred vision. *Preeclampsia, defined as hypertension* with proteinuria, progresses to ***eclampsia if seizures or*** **coma develop**. The cause of eclampsia is unknown but appears to involve sympathetic overactivity associated with insulin resistance, the renin-angiotensin system, lipid peroxidation, and inflammatory mediators.

Another consideration related to fetal growth is spontaneous abortion (miscarriage). Spontaneous abortion is the natural termination of pregnancy before the 20^{th} week of gestation, and occurs in approximately 15% of all pregnancies. The most common causes of spontaneous abortion are morphologic or chromosomal abnormalities which prevent successful implantation. It is most unlikely that any dental procedure would be implicated in spontaneous abortion, provided fetal hypoxia and exposure of the fetus to teratogens are avoided. Febrile illness and sepsis also can precipitate a miscarriage; therefore, prompt treatment of odontogenic infection and periodontitis is advised.

DENTAL MANAGEMENT

Medical Considerations

The dentist should assess the general health of the patient through a thorough medical history. Information to ascertain includes current physician, medications taken, use of tobacco, alcohol, or illicit drugs, history of gestational diabetes, miscarriage, hypertension, and morning sickness. If the need arises, the patient's obstetrician should be consulted.

Establishing a good patient-dentist relationship that encourages openness, honesty, and trust is an integral part of successful management. This kind of relationship greatly reduces stress and anxiety for both patient and dentist.

measuring vital signs is important for identifying undiagnosed abnormalities (preeclampsia) and the need for corrective action. At a minimum, blood pressure and pulse should be measured.

Preventive Program

Oral hygiene instructions: This essentially consists of a plaque control program that minimizes the exaggerated inflammatory response of gingival tissues to local irritants that commonly accompany the hormonal changes of pregnancy.

Maternal plaque control, however, has implications for caries risk for the infant. Studies conducted over the past few deccades have shown that reduced oral streptococcal levels in the pregnant mother reduce the risk that the infant will become infected and develop caries.

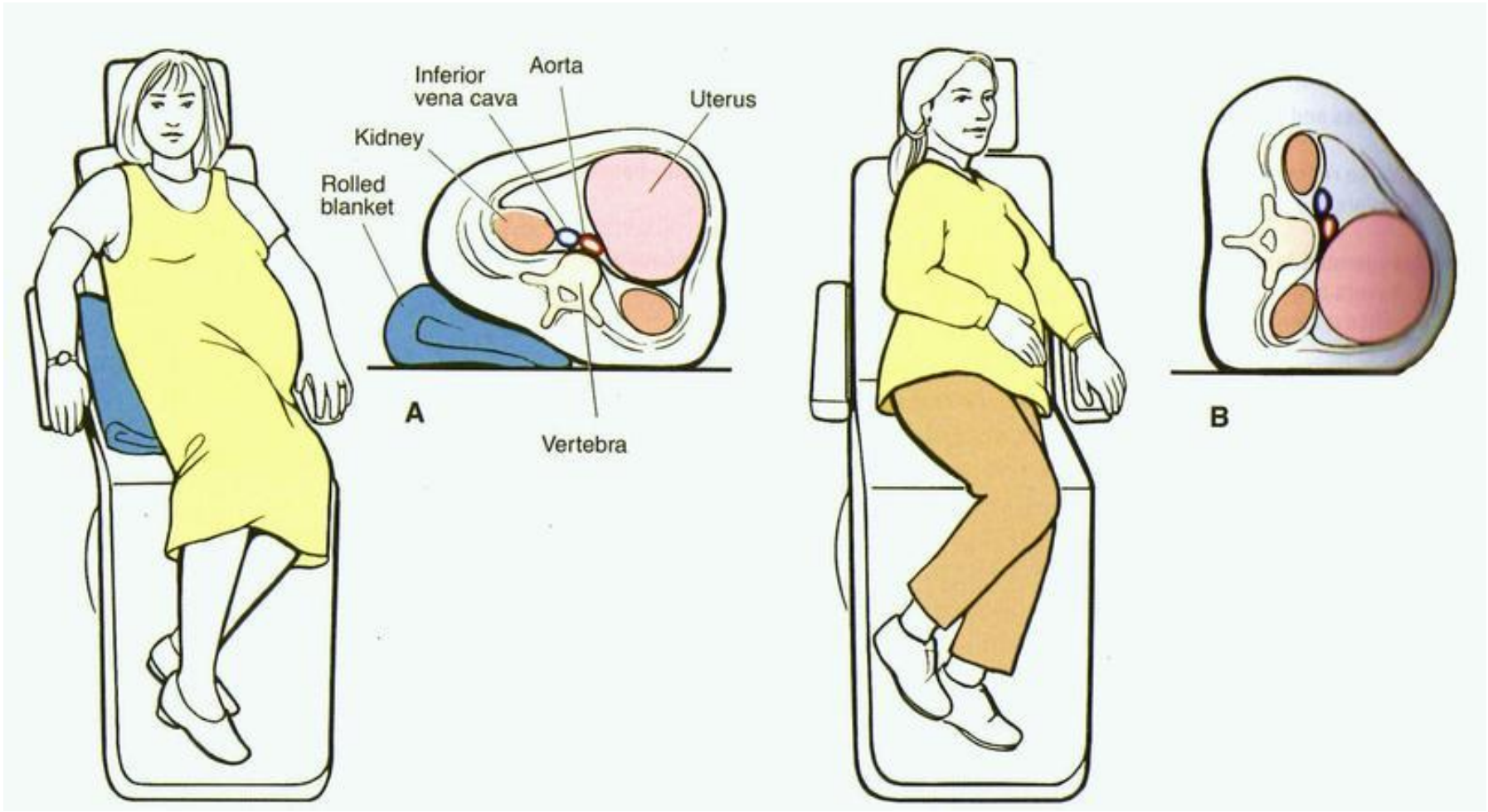
Treatment Timing

Other than as part of a good plaque control program, elective dental care is best avoided during the first trimester because of the potential vulnerability of the fetus.

The second trimester is the safest period during which to provide routine dental care. Emphasis should be placed on controlling active disease and eliminating potential problems that could occur later in pregnancy or during the immediate postpartum period, because providing dental care during these periods often is difficult.

The early part of the third trimester is still a good time to provide routine dental care. After the middle of the third trimester, however, elective dental care is best postponed. This is because of the increasing feeling of discomfort that many expectant mothers may experience.

Prolonged time in the dental chair should be avoided, to prevent the complication of supine hypotension. If supine hypotension develops, rolling the patient onto her left side affords return of circulation to the heart.


Inferior vena cava
Aorta
Uterus
Kidney
Rolled blanket
A
Vertebra
B

Dental Radiographs

In spite of the safety of dental radiography, ionizing radiation should be avoided, if possible, during pregnancy, especially during the first trimester, because the developing fetus is particularly susceptible to radiation Damage However, should dental treatment become necessary, radiographs may be required for accurate diagnosis and treatment.

Drug Administration

The principal concern is that a drug may cross the placenta, with the potential for toxic or teratogenic effects on the fetus.

Before prescribing or administering a drug to a pregnant patient, the dentist should be familiar with the FDA categorization of prescription drugs for pregnancy based on their potential risk of fetal injury

The current five pregnancy labeling categories are as follows:

***Category A**: Controlled studies in humans have failed* to demonstrate a risk to the fetus, and the possibility of fetal harm appears remote.

***Category B**: Animal studies have not indicated fetal* risk, and human studies have not been conducted, *or animal studies have shown a risk, but controlled* human studies have not.

***Category C**: Animal studies have shown a risk, but* controlled human studies have not been conducted, *or studies are not available in humans or* animals.

***Category D**: Positive evidence of human fetal risk* exists, but in certain situations, the drug may be used despite its risk.

***Category X**: Evidence of fetal abnormalities and fetal* risk exists based on human experience, and the risk outweighs any possible benefit of use during pregnancy.

Drugs in categories A and B are preferable for prescribing during pregnancy. However, many commonly prescribed drugs used in dentistry fall into category C, so the safety of their use often is uncertain.

Drugs in category C present the greatest difficulty for the dentist and the physician in terms of therapeutic and medicolegal decisions; therefore, consultation with the patient's physician may be needed.

Local Anesthetics. Local anesthetics administered

with epinephrine generally are considered safe for use during pregnancy and are assigned to pregnancy risk classification categories B and C. Although both the local anesthetic and the vasoconstrictor cross the placenta, subtoxic threshold doses have not been shown to cause fetal abnormalities. Because of adverse effects associated with high levels of local anesthetics, it is important not to exceed the manufacturer's recommended maximum dose.

Analgesics. The analgesic of choice during pregnancy

is acetaminophen (category B). Aspirin and nonsteroidal antiinflammatory drugs convey risks for constriction of the ductus arteriosus, as well as for postpartum hemorrhage and delayed labor

The risk of these adverse events increases when agents are administered during the third trimester. Risk also is more closely associated with prolonged administration, high dosage, and selectively potent antiinflammatory drugs, such as indomethacin. Codeine *(disambiguation).* and propoxyphene are associated with multiple congenital defects and should be used cautiously and only if needed

Antibiotics.

Penicillins (including amoxicillin), erythromycin (except in estolate form), cephalosporins, metronidazole, and clindamycin are generally considered to be safe for the expectant mother and the developing child.

The use of tetracycline, including doxycycline (FDA category D), is contraindicated during pregnancy. Tetracyclines bind to hydroxyapatite, causing brown discoloration of teeth, hypoplastic enamel, inhibition of bone growth, and other skeletal abnormalities

Anxiolytic

Drug	FDA Pregnancy Risk Category	Use During Pregnancy	Risk	Use During Breast Feeding
Sedative-Hypnotics				
Barbiturates	D	Avoid	Neonatal respiratory depression	Avoid
Benzodiazepines (diazepam, lorazepam)	D	Avoid	Possible risk for oral clefts with prolonged exposure	Avoid
Triazolam	X			
Nitrous oxide	Not assigned	Best used in second and third trimesters and for <30 minutes; consult physician		Yes
Sialagogues				
Cevimeline	C	No information		No information
Pilocarpine	C	Yes		Avoid

During Breast Feeding.

A potential problem arises when a nursing mother requires the administration of a drug in the course of dental treatment. The concern is that the administered drug may enter the breast milk and be transferred to the nursing infant, in whom exposure may result in adverse effects.

A significant fact is that the amount of drug excreted in the breast milk usually is not more than about 1% to 2% of the maternal dose. Therefore, most drugs are of little pharmacologic significance for the Infant

In addition to careful drug selection, nursing mothers may take the drug just after breast feeding and avoid nursing for 4 hours or longer if possible. This timing should result in even further reduced drug concentrations in the breast milk.

Oral Complications and Manifestations

The most common oral complication of pregnancy is pregnancy gingivitis . This condition results from an exaggerated inflammatory response to local irritants and less-than-meticulous oral hygiene during periods of increased secretion of estrogen , progesterone and altered fibrinolysis.

- In approximately 1% of gravid women, the hyperplastic response may exacerbate in a localized area, resulting in a **pyogenic granuloma or "pregnancy tumor**

- Hyperplastic gingival changes become apparent around the second month and persist until after parturition, at which time the gingival tissues usually regress and return to normal, provided that proper oral hygiene measures are implemented and any calculus present is removed. Surgical or laser excision occasionally is required as dictated by symptoms, bleeding, or interference with mastication.

Many women are convinced that pregnancy causes tooth loss (i.e., “a tooth for every pregnancy”), or that calcium is withdrawn from the maternal dentition to supply fetal requirements (i.e., “soft teeth”). **Calcium is present in the teeth in a stable crystalline form and hence is not available to the systemic circulation to supply a calcium demand. However, calcium is readily mobilized from bone to supply these demands**. Therefore, although calcium supplementation for the purpose of preventing tooth loss or soft teeth is unwarranted, the physician may prescribe calcium to fulfill the general nutritional requirements of mother and fetus.

Pregnant women often have a **hypersensitive gag reflex.** This, in combination with morning sickness, may contribute to episodes of regurgitation, potentially leading to halitosis and enamel erosion. The dentist should advise the patient to rinse after regurgitation with a solution that neutralizes the acid (e.g., baking soda, water).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Thyroid diseases

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Thyroid diseases

The thyroid gland is situated in the anterior portion of the neck just below and bilateral to the thyroid cartilage, ^consists of two lateral lobes connected by-an isthmus, in some individuals a superior portion of glandular tissue, or the pyramidal lobe, can be identified.

Embedded in the thyroid gland are the parathyroid glands.

It develops from the thyroglossal duct and portions of the ultimobranchial body, it originates from the posterior portion of the tongue and descends to its final location passing through the developing hyoid bone. Remnants of thyroid tissue may be found anywhere along the path of the thyroglossal duct and they may become cystic or neoplastic.

Function

The thyroid gland secretes 3 hormones:

T4 (Thyroxine), Tetraiodothyronine, it has a half life of 1 week and it is converted to

T3 , Triiodothyronine, which is the active form and has a half life of 1 day.

the hormones influence the growth and maturation of tissues, total energy expenditure and act on metabolism by regulating protein synthesis.

Calcitonin, secreted from the medullary C cells, it is involved, along with parathyroid hormone and vitamin D, in regulating serum calcium and phosphorus levels and skeletal remodeling.

Blood levels of T4 and T3, are controlled by a feedback mechanism mediated by hypo thalamic-pituitary-thyroid axis. The hypothalamus releases thyrotropin releasing hormone (TRH) in response to external stimuli (stress, illness, metabolic demands, low levels of T4 and T3), TRH stimulates the pituitary gland to secrete thyroid stimulating hormone (TSH) which causes the thyroid gland to secrete T4 and T3. high levels of T3 and T4 turn off the release of TSH and low levels turn it on. 95% of the thyroid hormones are bound to plasma proteins, the most important are being thyroxine binding globulin (TBG).

Laboratory tests and imaging techniques

Radioactive Iodine uptake (RAIU), using ^{131}I or ^{123}I, the latter exposes the patient to a lower radiation dose. The uptake varies inversely with plasma iodide concentration and directly with the functional state of the gland. The normal value is 10%-30% RAIU and above this indicates hyperfunction.

Measuring serum T4 and T3. Elevated levels indicate hyperthyroidism while low levels indicate hypothyroidism.

Measurement of the basal serum TSH concentration, high level indicates hypothyroidism and low level indicate hyperthyroidism.

Thyroid scan using ^{123}I injected and a scanner is used to localize areas of radioactive concentration. It is used to localized thyroid nodules and locate ectopic functional thyroid tissues.

Ultrasonography to detect the lesions, differentiate solid and cystic lesions and guide needle for aspiration of cyst or biopsy.

CT scan and MRI.

Thyroid diseases

Thyroid diseases are:

Hyperthyroidism.

Hypothyroidism.

Thyroiditis.

Thyroid neoplasm.

Hyperthyroidism (Thyrotoxicosis)

Excess of T4 and T3 in blood stream.

Causes:

- Thyroid diseases; like Graves' disease, ectopic thyroid tissue, multinodular goiter, thyroid adenoma and thyroiditis.
- Ingestion of thyroid hormone or food containing thyroid hormones.
- Pituitary diseases involving the anterior lobe.

Clinical presentation

Skin; is warm and moist, rosy complexion and the patient blushes easily, palmar erythema, profuse sweating, excessive melanin pigmentation of the skin, thin fine hair with areas of alopecia and soft nails. Dermopathy; result from increased concentration of hyaluronic acid and chondrotin sulfate in the dermis in focal areas of the skin causing compression of dermal lymphatics and non-pitting edema.

Cardiovascular; arrhythmia (palpitation, tachycardia), angina, myocardial infarction and congestive heart failure. These are caused by increased metabolic activity caused by excessive hormone secretion which leads to increased circulatory demand resulting in increased stroke volume and heart rate. Thyroid hormones have a profound effect on the sensitivity of tissues to catecholamines like epinephrine and these agents must not be administered to them.

Gastrointestinal; there is weight loss even with increased appetite, anorexia, nausea and vomiting are rare, many patients have Achlorhydria and about 3% 'develop pernicious anemia.

Central nervous system; nervousness, anxiety, sleep disturbance, emotional lability, impaired concentration, fatigue, weakness and tremor (hands, fingers and tongue).

Eyes; ophthalmopathy, it may produce the greatest long term disability, there is eyelid retraction, periorbital edema, chemosis and bilateral exophthalmos or proptosis due to enlargement of the extraocular muscles and fat within the orbit as a result of mucopolysaccharide infiltration. It may progress to visual loss due to exposure keratopathy or compressive optic neuropathy.

Skeletal; there is bone loss with increased excretion with calcium and phosphorus into the urine.

Others; there is glucose intolerance and rarely diabetes, decreased serum cholesterol, thrombocytopenia also develop, increase in RBCs total number to carry the additional oxygen needed for increased metabolic activity.

Thyrotoxic crisis (thyroid storm)

It is a rare complication that occur in 1% of the patients, most of them have a long history of thyrotoxicosis. Precipitating factors include; infection, trauma, surgical emergency and operation.

Signs and symptoms extreme restlessness, nausea, vomiting, abdominal pain, fever, profuse sweating, marked tachycardia, arrhythmia, pulmonary edema and congestive heart failure. Coma may follow severe hypotension and death.

Laboratory Endings

Classically there is decreased TSH and increased free T4 concentration, sometimes there is low TSH and normal free T4 and increased free T3, others may show increased TSH and increased T4 especially in pituitary adenoma secreting TSH or in thyroid hormone resistance syndrome.

Medical management of hyperthyroidism

Antithyroid agents; like Carbimazole and Propylthiouracil. These may have adverse effects like Agranulocytosis, Thrombocytopenia and Aplastic anemia.

Radioactive iodine; it is contraindicated in pregnancy and during breast feeding,, it may cause hypothyroidism.

Subtotal thyroidectomy; in large goiter or thyroid nodules, but the patient is given antithyroid drugs before the operation until he/she becomes euthyroid. Complications include hypoparathyroidism and recurrent laryngeal nerve injury.

Beta-adrenergic blockers like Propranolol to alleviate sympathetic overactivity.

Hypothyroidism

It can be congenital or acquired.. The acquired adult type (Myxedema) follows thyroid gland failure, pituitary gland failure, radiation of the thyroid gland (radioactive iodine), autoimmune diseases (Hashimoto's thyroiditis), surgical removal and excessive antithyroid drug therapy.

It is more common in women than in men and occurs between 30-60 years old individuals.

Most infants with congenital hypothyroidism (Cretinism) have thyroid dysgenesis that is ectopic, hypoplastic or thyroid agenesis.

Clinical presentation

Neonatal Cretinism characterized by:

Dwarfism, overweight, a broad flat nose, wide set eyes, thick lips, large protruding tongue, poor muscle tone, pale skin, retarded bone age, delayed eruption of the teeth, malocclusion and mental retardation. These features can be prevented by early detection and treatment.

Adult hypothyroidism (Myxedema)

Dull expression, puffy eyelids, alopecia of the outer third of the eyebrows, dry rough skin, dry brittle and coarse hair, increased size of the tongue, slowing of mental activity, slurred hoarse speech, anemia, constipation, weight gain, muscle weakness and deafness. Congestive heart failure may occur in patients with severe Myxedema.

Untreated patients with severe Myxedema may develop hypothyroid (Myxedema) coma, which can be fatal, it occurs in elderly patients during winter months, it is precipitated by stressful conditions like cold, operations, infections or trauma.

Medical management of hypothyroidism

Synthetic preparations containing sodium Levothyroxine (T4) or sodium Liothyronine (T3).

T4 potentiates the action of Warfarin and may cause further prolongation of prothrombin time.

Administration of T4 for diabetics may cause hyperglycemia.

Dental management

- **Obtaining thorough history.**

- The clinician should include the thyroid gland in **examination**
- The anterior neck region should be inspected for the presence of goiter or the presence of an old surgical scar indicating previous thyroid surgery, the gland should-be palpated, when the patient swallows the gland moves superiorly.

  The posterior dorsal region of the tongue should be inspected for the presence of any nodule that may indicate lingual thyroid,

Dental management

Hyperthyroidism (Thyrotoxicosis)

1-Beware of the clinical manifestations of thyrotoxicosis, so that undiagnosed or poorly treated patients are identified and referred for medical evaluation and treatment. Those patients may develop thyrotoxic crisis (thyroid storm) which can be precipitated by a surgical procedure or acute infections, so we avoid surgical procedures, acute infections in such patients.

Management of thyrotoxic crisis include:

- Recognize signs and symptoms.
- Seek medical assistance immediately.
- Cold wet packs and ice packs to cool the patient.
- 100-300 mg hydrocortisone injection.
- I.V. infusion of hypertonic glucose solution.
- Cardiopulmonary resuscitation and monitoring of the vital signs.
- Antithyroid drugs and potassium iodide.
- The use of vasoconstrictor agents in LA or retraction cord or to control bleeding should be avoided in such patients.

2- In well controlled patients, normal procedure and management is implemented, acute and chronic infections should be treated. Those patients can be given normal concentrations of vasoconstrictor agents.

3- In patients taking non-selective beta blockers, epinephrine may cause an increase in blood pressure through inhibition of the vasodilatory action of epinephrine attained through blocking beta2 receptors.

4- Propylthiouracil.(antithyroid drug) can cause agranulocytosis and leukopenia which may run the risk of infection, the dentist can consult the patient's physician or’order screening tests to rule out the presence of these complications before surgical procedures. This drug can also increase the anticoagulant effect of Warfarin. Aspirin and NSAIDs increase the amount of T4 and make control of thyroid diseases more difficult.

5- Consultation with the physician is recommended as part of the management program.

Hypothyroidism

1- Identification and recognition of the patients through history and clinical examination, referral of the untreated patients for diagnosis and treatment.

2- In patients who are diagnosed but untreated or poorly treated, we should avoid, surgical procedures, infections and CNS depressants and narcotics, those patients especially with severe symptoms of hypothyroidism may develop Myxedematous coma.

Management of Myxedematous coma:

- The dentist must be able to recognize the signs and symptoms.
- Seek immediate medical aid.
- Cover the patient to conserve heat.
- 100-300 mg Hydrocortisone.
- Cardiopulmonary resuscitation as needed.
- Administration of i.v. hypertonic saline and glucose.
- Parenteral Levothyroxine.

3- In well controlled patients we implement normal procedure and management and avoid infections, in addition to management of malocclusion and enlarged tongue.

Oral complications and manifestations

Thyrotoxicosis

- Osteoporosis of alveolar bone.
- Dental caries and periodontal diseases appear rapidly.
- Premature loss of deciduous teeth and early eruption of permanent teeth.
- Presence of lingual thyroid in few patients with thyrotoxicosis, such patients should have thyroid scan to make sure that this is not the only thyroid tissue present before surgical removal.

Hypothyroidism

(Infants with Cretinism)

- Thick lips.
- Enlarged tongue.
- Delayed eruption of teeth.
- Malocclusion.
- Skeletal retardation.

(Adults with Myxedema)

- Enlargement of the tongue.
- Generalized-swollen gingiva.


Thank You!

Dental management of patients with Pulmonary Disease

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Chronic obstructive pulmonary disease (bronchitis and emphysema) and asthma are common pulmonary diseases that cause obstruction in airflow.

Chronic bronchitis

is defined as a condition associated with excessive tracheobronchial mucus production (at the bronchial level) sufficient to cause a chronic cough with sputum production for at least 3 months in at least 2 consecutive years in a patient in whom other causes of productive chronic cough have been excluded.

Emphysema *is defined as the* presence of permanent enlargement of the air spaces distal to the terminal bronchioles accompanied by destruction of alveolar walls or septa (at the acinar level) without obvious fibrosis.

Etiology

Worldwide, the most important cause of COPD is tobacco smoking

In addition to cigarette smoking, long-term exposure to occupational and environmental pollutants and the absence or deficiency of α1-antitrypsin are other factors that contribute to COPD

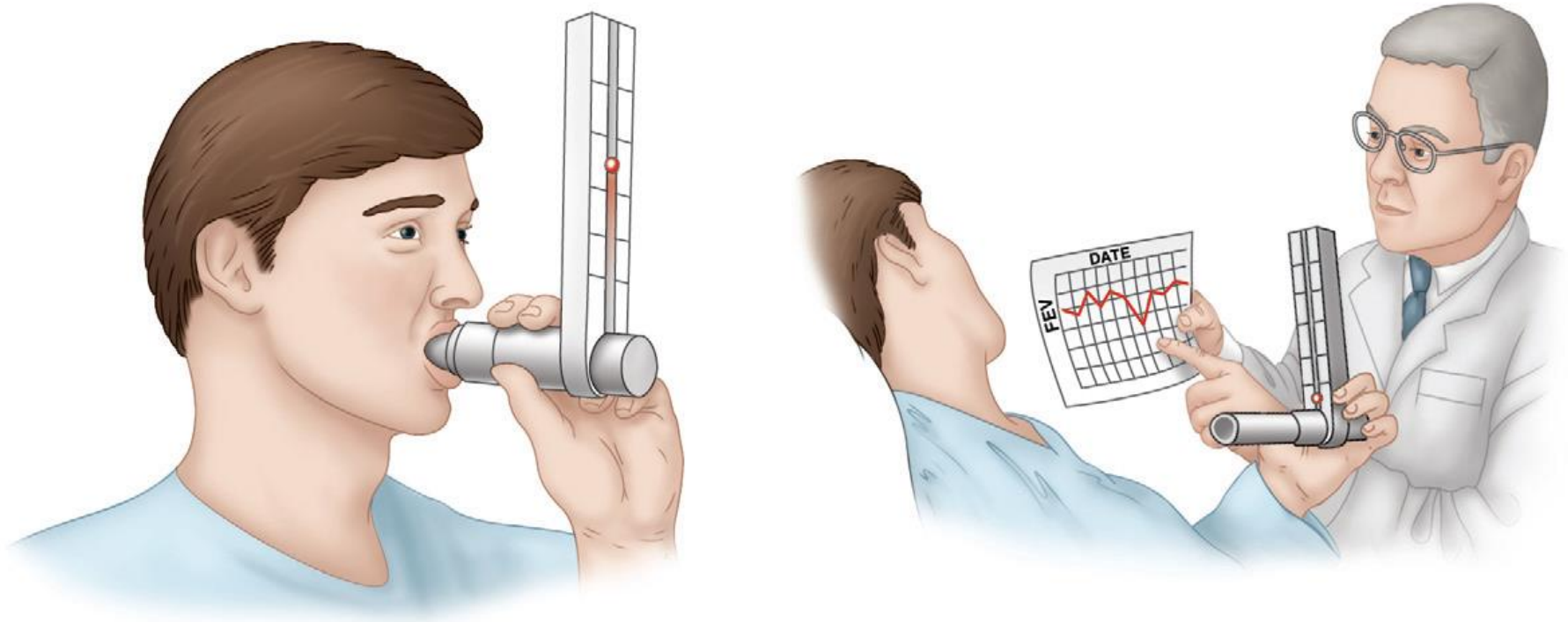
Lab. findings

Measures of expiratory airflow are the key diagnostic procedures performed. Forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1) are determined by spirometry

Arterial blood gas measurement and chest radiographs aid in the diagnosis

Patients with chronic bronchitis have an elevated partial pressure of carbon dioxide (PCO2) and decreased partial pressure of oxygen (PO2) (as measured by arterial blood gases), leading to secondary erythrocytosis, an elevated hematocrit value, and compensated respiratory acidosis.

Patients with emphysema have a relatively normal (PCO2) and a decreased (PO2), which maintain normal hemoglobin saturation, thus avoiding erythrocytosis.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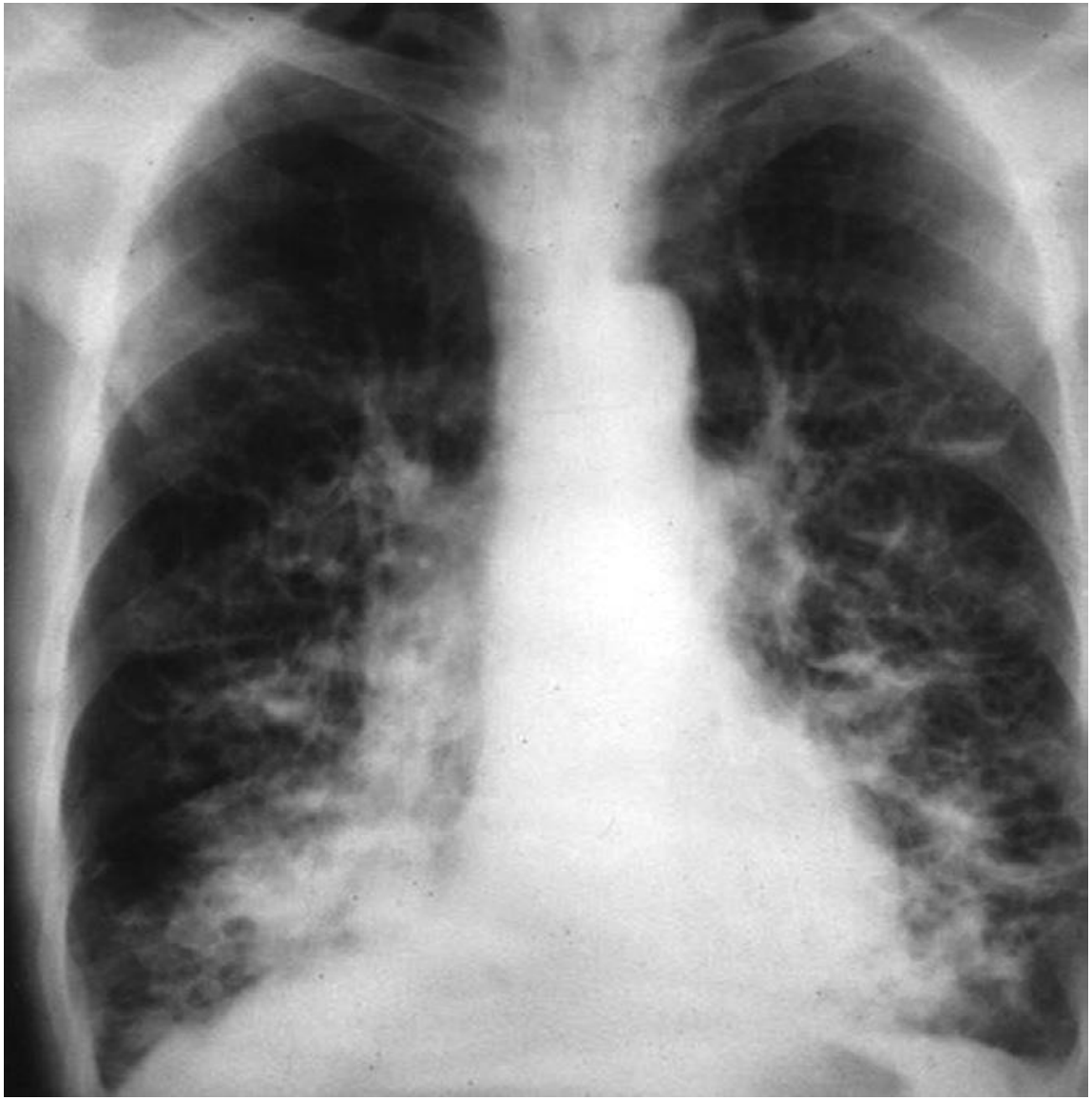

Chest radiograph of a patient with chronic obstructive pulmonary disease showing prominent vascular markings

MEDICAL MANAGEMENT

Management of COPD includes smoking cessation, influenza and pneumococcal vaccinations, and use of short- and long-acting bronchodilators. Other recommended measures include regular exercise, good nutrition, and adequate hydration.

Antibiotics are used for pulmonary infections, and low-flow supplemental O2 (2 L/minute) is recommended when the patient's PO2 is 88% or less

DENTAL MANAGEMENT

1- Before initiating dental care, clinicians should assess the severity of the patient's respiratory disease and the degree to which it has been controlled

2- A patient coming to the office for routine dental care who displays shortness of breath at rest, a productive cough, upper respiratory infection, or an oxygen saturation level less than 91% (as determined by pulse oximetry) is unstable. Accordingly, the appointment should be rescheduled and an appropriate referral for medical attention should be made.

3- If the patient is stable and the breathing is adequate, efforts should be directed toward the avoidance of anything that could further depress respiration .

- Patients should be placed in a semisupine or upright chair position for treatment, rather than in the supine position, to prevent orthopnea and a feeling of respiratory discomfort.
- Pulse oximetry monitoring is advised. Humidified low-flow O2—generally at a rate of 2 to 3 L/minute—may be provided and should be considered for use when the oxygen saturation level is less than 95%.
- No contraindication to the use of local anesthetic has been identified. However, the use of bilateral mandibular blocks or bilateral palatal blocks can cause an unpleasant airway constriction sensation in some patients. This concern may be more important in the management of a patient with severe COPD with a rubber dam, or when medications are administered that dry mucous secretions. Humidified low-flow O2 can be provided to alleviate the unpleasant airway feeling produced by nerve blocks, use of a rubber dam, and/or medications.

- If sedative medication is required, low-dose oral diazepam (Valium) may be used. Nitrous oxide–oxygen inhalation sedation should be used with caution in patients with mild to moderate chronic bronchitis. It should not be used in patients with severe COPD and emphysema, because the gas may accumulate in air spaces of the diseased lung.

- Narcotics and barbiturates should not be used, because of their respiratory depressant properties.
- Anticholinergics and antihistamines generally should be used with caution in patients with COPD, on account of their drying properties and the resultant increase in mucus tenacity; because patients with chronic bronchitis may already be taking these types of medications, concurrent administration could result in additive effects.

- Patients taking systemic corticosteroids may require supplementation for major surgical procedures because of adrenal suppression.
- Macrolide antibiotics (e.g., erythromycin, azithromycin) and ciprofloxacin hydrochloride should be avoided in patients taking theophylline because these antibiotics can retard the metabolism of theophylline, resulting in theophylline toxicity.
- The dentist should be aware of the manifestations of theophylline toxicity. Signs and symptoms include anorexia, nausea, nervousness, insomnia, agitation, thirst, vomiting, headache, cardiac arrhythmias, and convulsions.

Oral Complications and Manifestations

Patients with COPD who are chronic smokers have an increased likelihood of developing halitosis, extrinsic tooth stains, nicotine stomatitis, periodontal disease, premalignant mucosal lesions, and oral cancer. Anticholinergics are associated with dry mouth. In rare instances, theophylline has been associated with the development of Stevens-Johnson syndrome.

ASTHMA

Asthma is a chronic inflammatory disease of the airways characterized by reversible episodes of increased airway hyperresponsiveness resulting in recurrent episodes of dyspnea, coughing, and wheezing.

The bronchiolar lung tissue of patients with asthma is particularly sensitive to a variety of stimuli. Overt attacks may be provoked by allergens, upper respiratory tract infection, exercise, cold air, certain medications (salicylates, nonsteroidal antiinflammatory drugs, cholinergic drugs, and β-adrenergic blocking drugs), chemicals, smoke, and highly emotional states such as anxiety, stress, and nervousness.

complications

The most serious manifestation of asthma, may occur. Status asthmaticus is a particularly severe and prolonged asthmatic attack (one lasting longer than 24 hours) that is refractory to usual therapy. Signs include increased and progressive dyspnea, jugular venous pulsation, cyanosis, and pulsus paradoxus (a fall in systolic pressure with inspiration).

Status asthmaticus often is associated with a respiratory infection and can lead to exhaustion, severe dehydration, peripheral vascular collapse, and death.

Signs & symptoms

Typical symptoms and signs of asthma consist of reversible episodes of breathlessness (dyspnea), wheezing, cough that is worse at night, chest tightness, and flushing. Onset usually is sudden, with peak symptoms occurring within 10 to 15 minutes.

Respirations become difficult and are accompanied by expiratory wheezing. Tachypnea and prolonged expiration are characteristic. Termination of an attack commonly is accompanied by a productive cough with thick, stringy mucus.

Laboratory Findings

Commonly ordered tests include 6-minute walk test, spirometry before and after administration of a short-acting bronchodilator, chest radiographs (to detect hyperinflation), skin testing (for specific allergens), bronchial provocation (by histamine or methacholine chloride challenge) testing, sputum smear examination and cell counts (to detect neutrophilia or eosinophilia), arterial blood gas determination, and antibody-based enzyme-linked immunosorbent assay (ELISA) for measurement of environmental allergen exposure

Medical management

- prevention or elimination of precipitating factors (e.g., smoking cessation) and comorbid conditions (rhinosinusitis, obesity)
- Antiasthmatic drug selection is based on the type and severity of asthma and whether the drug is to be used for long-term control or quick relief
- Inhaled antiinflammatory agents as first-line drugs (the preferred inhalational agent is a corticosteroid preparation, with a leukotriene inhibitor as an alternative) for the long-term management and prophylaxis of persistent asthma .
- β-adrenergic agonists are recommended for intermittent asthma and are secondary agents that should be added (i.e., not to be used alone) for persistent asthma when antiinflammatory drugs are inadequate alone.

Medical management

- For relief of acute asthma attacks, inhaled shortacting β2-adrenergic agonists are the drugs of choice because of their fast and notable bronchodilatory and smooth muscle relaxation properties
- Theophylline is a mild to moderate bronchodilator to be used as an alternative.

DENTAL MANAGEMENT

Prevention of Potential Problems

Identify patients with asthma by history, followed by assessment to elucidate the surrounding details of the problem, along with prevention of precipitating factors.

Through a good history, the dentist should be able to determine the severity and stability of disease.

Questions should be asked that ascertain the type of asthma (e.g., allergic versus non-allergic), the precipitating substances, the frequency and severity of attacks, the times of day when attacks occur, whether this is a current or past problem, how attacks usually are managed, and whether the patient has received emergency treatment for an acute attack.

For severe and unstable asthma, consultation with the patient's physician is advised. Routine dental treatment should be postponed until better control is achieved.

Patients who have nocturnal asthma should be scheduled for late morning appointments, when attacks are less likely.

Use of operatory odorants (e.g., methyl methacrylate) should be reduced before the patient is treated.

Patients should be instructed to regularly use their medications, to bring their inhalers (bronchodilators) to each appointment, and to inform the dentist at the earliest sign or symptom of an asthma attack.

Prophylactic inhalation of a patient's bronchodilator at the beginning of the appointment is a valuable method of preventing an asthma attack.

Because stress is implicated as a precipitating factor in asthma attacks and dental treatment may result in decreased lung function, all dental staff members should make every effort to identify patients who are anxious and provide a stress-free environment through

- ✓ establishment of rapport and openness.
- ✓ Preoperative and intraoperative sedation may be desirable. If sedation is required, nitrous oxide–oxygen inhalation is best. Nitrous oxide is not a respiratory depressant, nor is it an irritant to the tracheobronchial tree.
- ✓ Oral premedication may be accomplished with small doses of a shortacting benzodiazepine.
- ✓ Reasonable alternatives with children are hydroxyzine (Vistaril), for its antihistamine and sedative properties,

Contained sulfites were a cause of allergic-type reactions in susceptible individuals. Sulfite preservatives are found in local anesthetic solutions that contain epinephrine or levonordefrin, although the amount of sulfite in a local anesthetic cartridge is less than the amount commonly found in an average serving of certain foods. Although rare, at least one case of an acute asthma attack precipitated by exposure to sulfites has been reported.

Thus, **the use of local anesthetic without epinephrine or levonordefrin may be advisable for patients with moderate to severe disease.**

Patients with asthma who are medicated over the long term with systemic corticosteroids may require supplementation for major surgical procedures if their health is poor . However, long-term use of inhaled corticosteroids rarely causes adrenal suppression unless the daily dosage exceeds 1.5 mg of beclomethasone dipropionate or its equivalent.

Administration of aspirin-containing medication or other nonsteroidal antiinflammatory drugs to patients with asthma is not advisable, because aspirin ingestion is associated with the precipitation of asthma attacks in a small percentage of patients.

Patients who are taking theophylline preparations should not be given macrolide antibiotics (i.e., erythromycin and azithromycin) or ciprofloxacin hydrochloride, because these agents interact with theophylline to produce a potentially toxic blood level of theophylline.

Antihistamines have beneficial properties but should be used cautiously because of their drying effects.

Management of Potential Problems: Asthma Attack

- The signs and symptoms should be recognized quickly and an inhaler provided rapidly. A short-acting β2-adrenergic agonist inhaler (Ventolin, Proventil) is the most effective and fastest-acting bronchodilator. It should be administered at the first sign of an attack.
- Long-lasting β2 agonist drugs like salmeterol (Serevent) and corticosteroids do not act quickly and are not given for an immediate response, but they may provide a delayed response.

Management of Potential Problems: Asthma Attack

- With a severe asthma attack, use of subcutaneous injections of epinephrine (0.3 to 0.5 mL, 1 : 1000) or inhalation of epinephrine (Primatene Mist) is the most potent and fastest-acting method for relieving the bronchial constriction.
- Supportive treatment includes providing positive-flow oxygenation, repeating bronchodilator doses as necessary every 20 minutes, monitoring vital signs (including oxygen saturation, if possible, which should reach 90% or higher), and activating the emergency medical system, if needed


Thank You
The DreamWitch

Dental management of patients with blood dyscrasia

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A\ Disorders of the RBCs

Anemia

is the reduction in the oxygen carrying capacity of the blood, it is associated with the decreased number of circulating RBCs or abnormalities in the Hemoglobin Hb. contained in the RBCs, which is the oxygen carrying molecule of the erythrocytes, it is also responsible for the transport of C02.

- In anemia Hb. level is below 12 g/dl in adult female and below 13 g/dl in adult male.

- Anemia is not a disease but rather a feature or symptom that results from many underlying causes.

TABLE 22-1 Types of Anemia

Classification by RBC Size and Shape	Cause
Microcytic (MCV ≤ 80 fL*)	
Iron deficiency anemia	Decreased production of RBCs
Thalassemias	Defective hemoglobin synthesis
Lead poisoning	Inhibition of hemoglobin synthesis
Normocytic (MCV 80-100 fL*)	
Hemolytic anemia Sickle cell anemia Glucose-6-phosphate dehydrogenase deficiency	Increased destruction of RBCs
Aplastic anemia	Decreased production of RBCs
Renal failure	Decreased production of RBCs
Anemia of chronic disease	Decreased production of RBCs
Macrocytic (MCV > 100 fL*)	
Pernicious anemia	Decreased production of RBCs
Folate deficiency	Decreased production of RBCs
Hypothyroidism	Decreased production of RBCs

*Also expressed in μm^3 units.
fL, Femtoliter; *MCV*, Mean corpuscular volume; *RBC*, Red blood cell.

Types of anemia

1- Deficiency anemia

- ***Iron deficiency anemia',*** it is caused by blood loss, poor iron intake, poor iron absorption or increased demands for iron. It is more common in women than in men due to blood loss during menstruation and pregnancy.

- ***Vitamin B12 (cobalamin) deficiency, Folate (Folic acid) deficiency and Pernicious anemia***; Vit. B12 and folic acid are needed for RBCs formation and growth within the bone marrow. Vit B12 is bound to gastric intrinsic factor secreted by the parietal cells and absorbed in the terminal ileum, deficiency of the intrinsic factor causes Pernicious anemia.

Types of anemia

2-Hemolytic anemias:

- Hemoglobinopathies', these are inherited abnormalities of the Hb formation like **Sickle cell anemia and Thalassemia.**
- Enzymopathies(glucose-6-phosphate dehydrogenase [**G-6-PD] deficiency**),
- Erythrocyte membrane defects as in **Spherocytosis.**
- Damage to erythrocytes; which could be autoimmune, drug induced or infective. World wide **Malaria** is the most common cause of hemolytic anemia.

3-Other anemias:

- ***Aplastic anemia*;** it is a pancytopenia with anon-functioning bone marrow, many cases are idiopathic but possible causes include: Chemical like Benzene, drugs, hepatitis virus, irradiation and graft versus host disease.
- ***Anemia caused by bone marrow infiltration by abnormal cells; like in Leukemia and Multiple Myloma.***
- *Anemia associated with systemic diseases*; like in chronic inflammation and connective tissue diseases such as Rheumatoid Arthritis, Liver disease, Hypothyroidism,Hypopituitarism,Hypoadrenocorticism, Uremia and HIV infections.

Clinical presentation

symptoms include; fatigue, palpitations, dyspnea, bone and abdominal pain and tingling of the fingers and toes.

Signs include; jaundice, pallor, brittle nails and Koilonchia (spoonshaped nails). There may be hepatosplenomegaly and LAP.

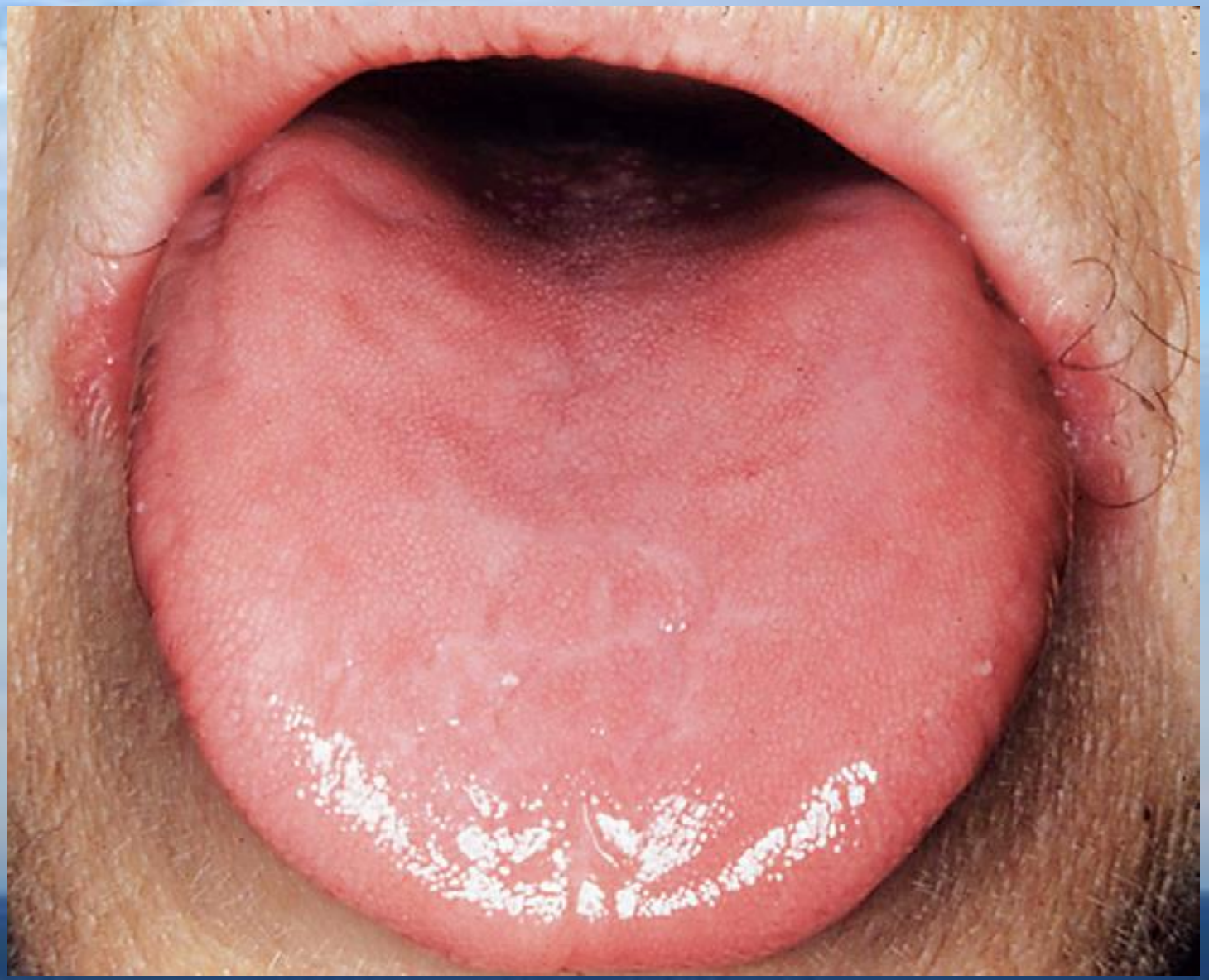
Oral manifestations

- Pale mucosa,
- oral ulcerations,
- angular cheilitis,
- glossitis and loss of papillae with atrophic changes in the oral mucosa,
- burning mouth symptoms.
- In patients with hemolytic anemia, there may be oral evidence of jaundice due to excessive red cells destruction,

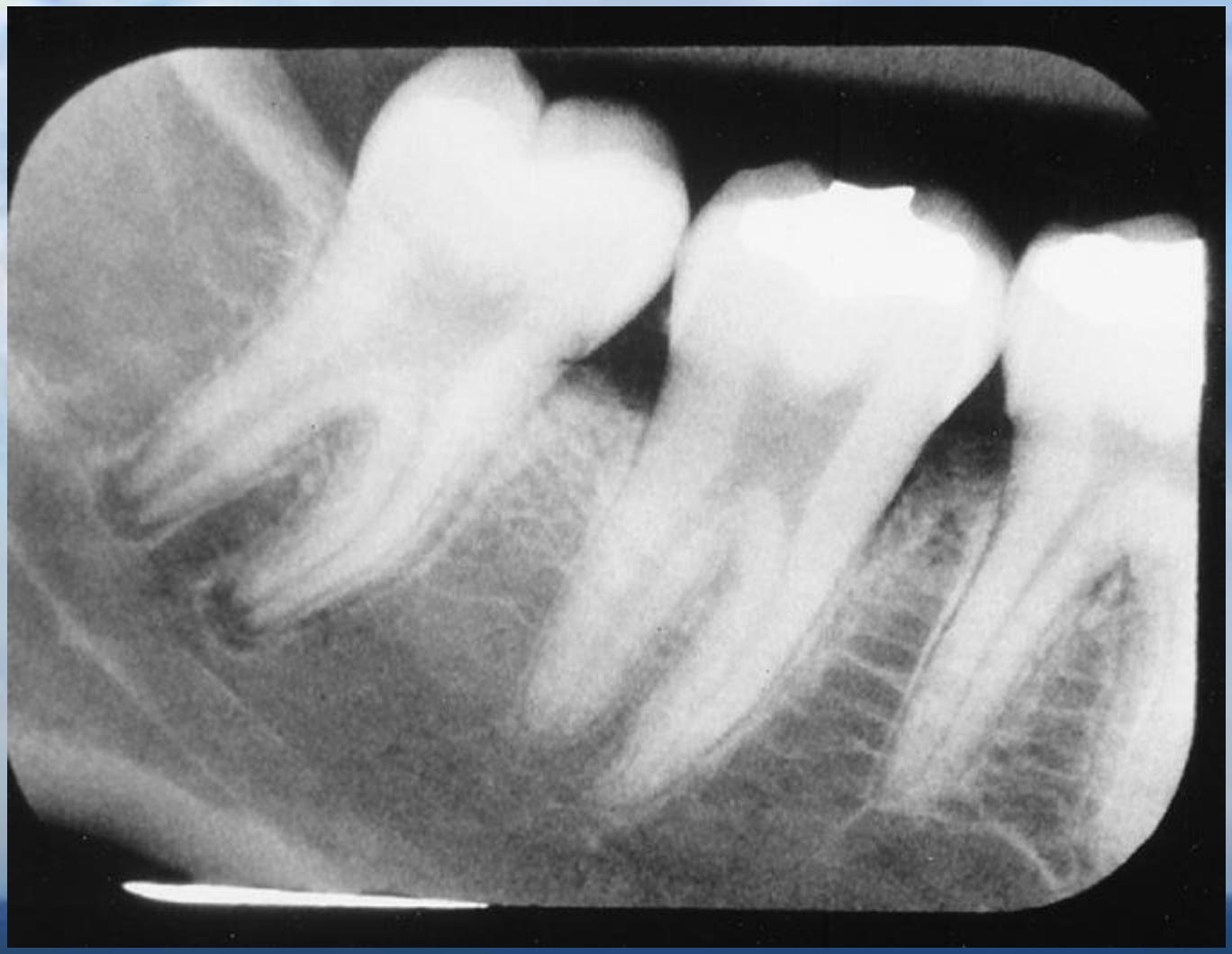
Oral manifestations

- the trabecular pattern of bone may be affected due to hyperplasia of marrow elements so radiographs show enlarged marrow spaces and osteoporosis, the trabeculae between the teeth appear horizontal (stepladder).
- Skull radiographs show **hair on end** appearance due to the new bone formation on the outer table of the skull.
- Vaso-occlusive events can lead to osteomyelitis, necrosis and peripheral neuropathy. Dental hypoplasia and delayed eruption of teeth often occur.

Atrophic glossitis



prominent horizontal trabeculations



Laboratory tests

These include; Hb, hematocrit and RBCs indices (MCV, MCH, MCHC), total WBCs count, platelet count.

Medical management

The goal is to eliminate the underlying causes.

- In deficiency anemias,; replacement of iron or folic acid,
- patients with Pernicious anemia can be managed by Cyanocobalamin injections.
- Blood transfusion may be needed when the Hb level falls below 7 g/dl and hematocrit is below 20%, it carries the risk of circulatory overload, infections and allergic reactions.
- Erythropoietin is used in treatment of anemia of chronic renal failure or cytotoxic drugs.
- Patients with Sickle cell anemia may require prophylactic Penicillin to prevent infections.

Dental management

- **Identification of the conditions associated with anemia through obtaining careful history,** the questions should include history of dietary intake, malnutrition, alcohol or drug use, history of blood loss especially for women during menstruation and pregnancy. The clinician should also identify signs and symptoms of anemia and can also order some screening tests, if the results of one of the tests or more are abnormal, the patient should be referred for medical evaluation and treatment.

- **the clinician should ensure that the patient's underlying condition is under therapeutic control** before proceeding with routine dental care. Patients with signs and symptoms of anemia and Hb level below 11 g/dl with abnormal heart rate or reduced oxygen saturation (below 91% in oximetry) are considered unstable and routine dental treatment should be deferred.

- **Local anesthesia (LA)** is satisfactory for pain control, conscious sedation can be given only if there is supplemental oxygen, elective operations under general anesthesia (GA) are not carried out when Hb level is below 10 g/dl.
- In **patients with G6PD deficiency**, certain drugs should be avoided since they can cause hemolysis, such as Sulfonamides (Sulfamethoxazole), Aspirin, Chloramphenicol and to a lesser extent Penicillin, Strepromycin and Isoniazide. Also dental infections should be avoided and if they occur they should be treated effectively.

- In **patients with Sickle cell anemia**, routine dental care can be provided for stable patients during non-crisis period, appointments should be short and the procedures should be not complicated, oral infections should be avoided, LA without vasoconstrictor for routine dental care is used while for surgical procedures LA with vasoconstrictor 1:100000 can be used. **Barbiturates and strong narcotics** should be avoided and **Diazepam** used when sedation is needed, prophylactic Antibiotics for surgical procedures are used, liberal use of **Salicylates** should be avoided and pain control can be achieved with acetaminophen **(Paracetamol)** and Codeine. In general infection, dehydration, hypoxia, acidosis and cold should be avoided in patients with Sickle cell anemia because the can precipitate acute crisis.

B\ WBCs Disorders

Leukemia

Is cancer of the WBCs that affects the bone marrow and circulating blood. It involves exponential proliferation of lymphoid or myloid cells. Leukemias is classified by the clinical course into: **acute** and **chronic**, and by the cell of origin into: **lymphoid** or **myloid (non-lymphoid).**

In acute leukemia there is a rapidly progressive disease that result from accumulation of immature, functionless WBCs in the bone marrow and blood, it is more common than chronic leukemia. While in the chronic leukemia there is slower onset and the cells are more mature.

There are 4 types of leukemia with many subtypes:

Acute Lymphoblastic Leukemia ALL, it is the most common type in children.

Acute Mylogenous Leukemia AML, the most common type in adults.

Chronic Lymphocytic Leukemia CLL, the second most common type in adults.

Chronic Myloid Leukemia CML.

Clinical presentation

Include; fatigue, **easy bruisibility, bone pain**, anemia, thrombocytopenia, malaise, pallor, anorexia, dyspnea on exertion, bleeding tendency, petechiae and ecchymoses of the skin and mucous membrane, recurrent infections, fever, weight loss, LAP, enlargement of the spleen and CNS disease.

Diagnosis is made by examination of the peripheral blood and bone marrow.

General management

It consists of chemotherapy to reduce the number of the malignant WBCs

The other line of treatment is bone marrow transplantation and peripheral blood stem cell transplantation.

Oral manifestations

Are more common in acute leukemia than in chronic leukemia, they include:

- Localized or generalized gingival enlargement, caused by infiltration of immature WBCs, it occurs in about 35% of acute leukemias and 10% of the chronic leukemias.
- The gingiva bleeds easily, sometimes spontaneously oral hygiene measures and chemotherapy may cause resolution.
- Oral ulcerations.

Oral manifestations

- Recurrent oral infections, due to the immature WBCs and as a complication of chemotherapy
- Localized mass of leukemic cells in the gingiva and other site of the oral cavity, it is termed Chloroma (Granulocytic Sarcoma).
- Pallor of oral mucosa.
- LAP.

B\ WBCs Disorders

Lymphoma

Lymphoma is a solid malignant tumor that originate in the lymph nodes or extranodal lymphoid tissues in any part of the body.

Lymphoma comprises Hodgkin's lymphoma or disease and non- Hodgkin's lymphoma NHL.

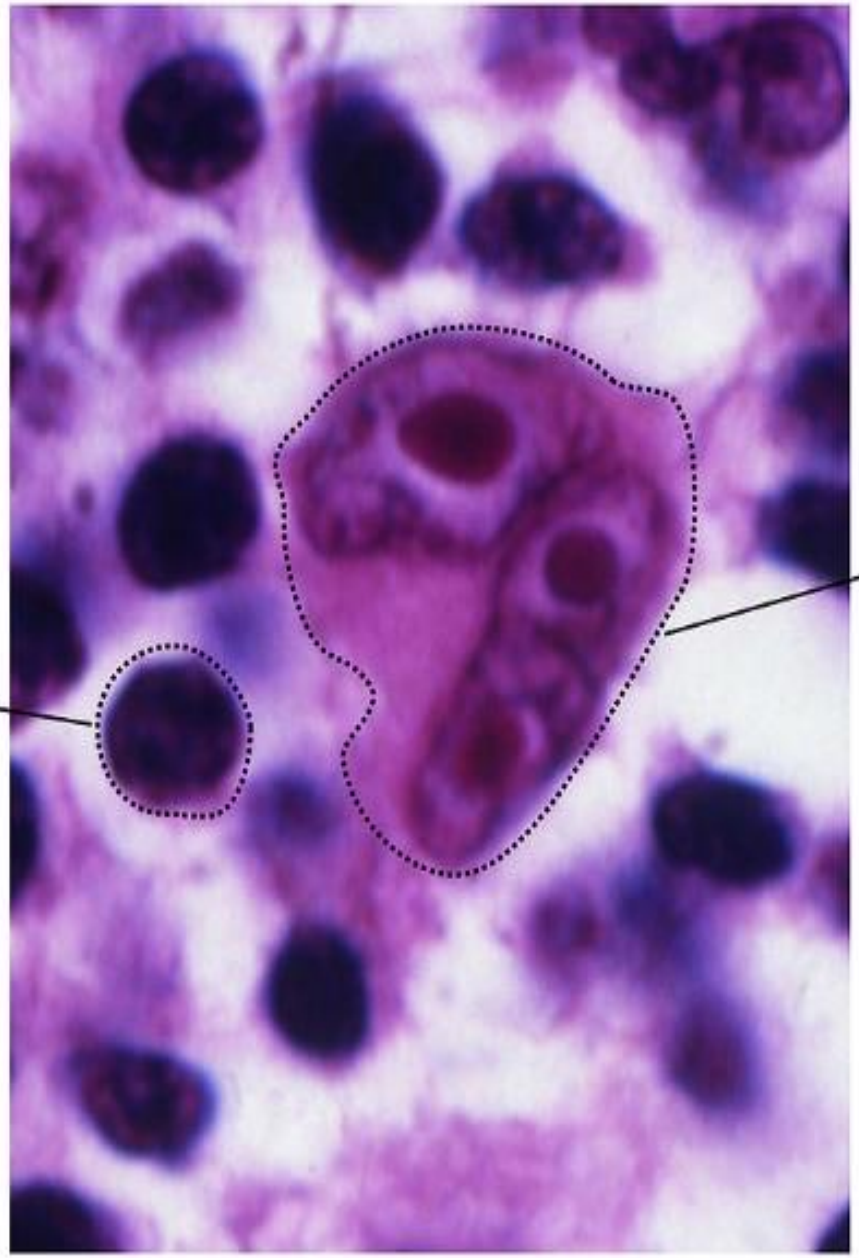
NHL is more common than the Hodgkin's type.

Hodgkin's disease; it is a neoplasm of B lymphocytes, it contains a characteristic tumor cell (Reed Sternberg cell). The cause is unknown but EBV may be implicated.

It presents as a painless enlargement of non-tender lymph nodes involving head and neck, axillary, mediastinal or groin lymph nodes.

Fever, night sweats, fatigue and weight loss may be experienced by the patient. The diagnosis is based on nodal biopsy and bone marrow aspirate.

Medical management requires staging on the basis of history, physical examination, lab. Findings and imaging.


Normal
lymphocyte
Reed-Sternberg
Cell

Non-Hodgkin's lymphoma; a large group of lymphoproliferative disorders of either B lymphocytes (more than 80% of the cases) or T lymphocytes origin. There are many types of NHL.

The cause is unknown but some genetic factors and chromosomal abnormalities in addition to other environmental factors such as infection with EBV. irradiation and drugs were implicated as possible causative factors.

The clinical presentation include; LAP, fever, weight loss, abdominal or chest pain and extranodal tumors. The diagnosis is based on biopsy of the lymph nodes or extranodal tumor. Proper staging is required which consists of blood investigation, imaging and bone marrow biopsy.

Medical management of lymphoma generally consist of chemotherapy and radiotherapy

Oral manifestations

- Cervical LAP.
- Intraoral tumors that may involve Waldeyer's ring, salivary glands, mandible, palate, gingiva or floor of the mouth.
- Oral ulcerations.
- Oral complications secondary to treatment include; burning mouth symptoms, xerostomia, infections, trismus, impaired craniomandibular growth and **osteoradionecrosis**.

Burkitt's Lymphoma

It is an aggressive type of B cell NHL. It is the most common lymphoma of children.

Recently described type associated with HIV infected individuals.

Oral manifestations

Include; tumors of the maxilla or mandible that cause bone destruction, mobility of the teeth, pain and paresthesia. On radiograph it appears as an osteolytic lesion with poorly demarcated margins.

Medical management is by combination chemotherapy.

Multiple Myloma

It is a lymphoproliferative disorder that results from overproduction of cloned malignant plasma cells resulting in bony lesions involving the skeletal system.

Clinical presentation

The malignant cell proliferation cause bone resorption that appear radiographically as a radiolucent **punched out lesions and replace bone marrow leading to anemia, leucopenia, thrombocytopenia.**

There is Amyloid deposition in various tissues that may lead to renal failure. Infection is common.

The most prominent symptom is persistent bone pain affecting the spine, ribs and sternum, weight loss, headache and Hypercalcemia.

Medical management

High dose chemotherapy and Prednisone which can be followed by bone marrow transplantation or autologous stem cell transplantation.

Treatment of anemia by **erythropoietin**. Antibiotics to prevent ant treat infections.

Bisphosphonates are used to reduce bone pain and maintain bone strength.

Oral manifestations

- Painful bony lesions, that appear as osteolytic punched out lesions which may be associated with cortical bone expansion.
- Extramedullary plasma cell tumor.
- Deposition of Amyloid in soft tissues like tongue.
- Osteonecrosis of the bone associated with Bisphosphonates treatment, it usually appears after surgery especially tooth extraction as a painful, non-healing socket. Treatment is directed to limiting the progression of necrosis through debridement, irrigation with antiseptics and antibiotics.

To minimize the likelihood of developing necrosis:

- Early treatment of any source of odontogenic infection preferably before starting treatment with Bisphosphonates.
- Non-surgical approaches are to be preferred.
- If extraction is required it should be as conservative as possible.
- The risk of necrosis should be discussed with the patient.

Dental management of WBCs Dyscrasias

- The clinician attempts to identify and recognize the presence of WBCs disorders through obtaining a **thorough history** about the signs and symptoms of these disorders, such as easy bruising or bleeding tendency, also family history of WBCs disorders.

Thorough extraoral and intraoral examination of the head and neck, oral cavity and oropharynx to identify any abnormalities that are suggestive of WBCs disorders. Screening blood investigations may be needed and if the results are abnormal, the patient is referred for further evaluation and routine dental care can be deferred.

- **Pretreatment assessment and preparation of the patient:**
  - ✓ Full knowledge of the patient's condition is required, the aim of this phase is to prevent oral infections, all potential sources of infection must be eliminated through restorative, periodontal and surgical treatment preferably 3 weeks prior to medical treatment.
  - ✓ Oral hygiene measures should be encouraged. When extraction is planned it should be as conservative as possible avoiding any hemostatic packing agents and attaining primary closure.
  - ✓ Prophylactic antibiotic are recommended before oral surgical procedure,2 g oral Penicillin 1 hour before the procedure, 500 mg 4 times daily for 1 week.
  - ✓ Patients with platelet count below 50.000/mm^3 should not undergo oral surgical procedures unless correction by transfusion is carried out.

- Oral health care **during medical treatment:** During treatment the patient is susceptible to many oral complications that require care:
  - ✓ **Mucositis;** appear 7-10 days after initiation of treatment and resolve after it. The non-keratinized mucosa is more severely affected.

    oral hygiene measures should be maintained to minimize infection, antiseptic and antimicrobial mouth washes e.g. Chlorhexidin are recommended, topical anesthetics and systemic analgesics can be given.

- ✓ **Neutropenia and Infection;** neutropenia leads to gingival inflammation, oral ulceration and infection which can be severe but with minimal clinical signs.

  Unusual bacterial infections, fungal and viral infections occur in patients with Leukemia, Lymphoma and Multiple Myloma on chemotherapy and require treatment.

  When oral infections develop, a specimen of the exudate should be sent for culture and antibiotic sensitivity tests.

- ✓ **Bleeding;** thrombocytopenia may case submucosal hemorrhage and sometimes spontaneous gingival bleeding, oral hygiene measures should be improved, when bleeding occurs local hemostatic measures should be used first like using pressure, gelatin sponge with thrombin or the use of oral antifibrinolytic agents. If these measures fail transfusion may be needed.

- ✓ **Graff versus host disease**: it occurs after bone marrow transplantation when immunologically active donor T cells react against host tissues, it can be acute (within 2-3 weeks) causing rash, mucosal ulcerations, increased liver enzymes and diarrhea. Or it could be chronic (3-12 months) producing features like Sjogren's syndrome, scleroderma, lichenoid changes, xerostomia, mucositis, dysphagia and damage to liver.

  It can be prevented by corticosteroids and immunosuppressive drugs.

  Adverse effects of drugs; such as gingival overgrowth with patients taking Cyclosporine.

- ✓ **Disturbance** of **growth and development;** due to treatment with chemotherapy and radiotherapy during childhood leading to micrognathia, malocclusion and teeth abnormalities.

➢ Post-treatment management:

- ✓ patients in remission state can have routine dental care while patients with poor prognosis should receive emergency care only.
- ✓ When invasive procedures are planned (e.g. oral surgery), platelet count and bleeding time should be investigated, the patient's physician should be consulted.
- ✓ In patients with surgically removed spleen, prophylactic antibiotic is needed, since they are at risk of bacterial infections, especially in the first 6 months after splenectomy.
- ✓ In patients with acute symptoms, routine dental care should be deferred.
- ✓ LA regional block should be avoided if possible in patients with bleeding tendency.Conscious sedation can be given and GA is allowed.


THANK
YOU

Dental management of patients with bleeding disorders

Dr. Usama Aldaghir
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Bleeding Disorders

These are conditions that alter ability of **blood vessels, platelet, and coagulation factors** to maintain homeostasis ,

Inherited bleeding disorder are genetically transmitted while acquired bleeding disorder occur as the result of diseases that affect vascular wall integrity, platelets, coagulation factors, such as drugs, radiation, or chemotherapy for cancer.

Classification of Bleeding Disorders

1- Non thrombocytopenic purpura

A- Vascular wall alterations

- Scurvy (vit. C deficiency)
- Infection
- Chemicals
- Allergy.

B- Disorders of platelet function

- Genetic defects (Bernard-Soulier disease)
- Drugs (a) Aspirin (b) NSAIDs (c) Alcohol (d) Beta-lactam antibiotics (e) Penicillin (f) Cephalothins
- Allergy
- Autoimmune disease
- von Willebrand's disease (secondary factor VIII deficiency)
- Uremia

Classification of Bleeding Disorders

2-Thrombocytopenic purpura

A-Primary—idiopathic

B- Secondary

- Chemicals
- Physical agents (radiation)
- Systemic disease (leukemia)
- Metastatic cancer to bone
- Splenomegaly
- Drugs (a) Thiazide diuretics (b) Estrogens (c) Gold salts
- Vasculitis
- Mechanical prosthetic heart valves
- Viral or bacterial infections

Classification of Bleeding Disorders

3-Disorders of coagulation

A- Inherited

- Hemophilia A (deficiency of factor VIII)
- Hemophilia B (deficiency of factor IX)
- Others

B- Acquired

- Liver disease
- Vitamin deficiency (a) Biliary tract obstruction
  (b) Malabsorption.
  (c) Excessive use of broad-spectrum antibiotics
- Anticoagulation drugs (a) Heparin (b) Coumarin (c) Aspirin and NSAIDs
- Disseminated intravascular coagulation (DIC)
- Primary fibrinogenolysis.

Pathophysiology

Three phases of homeostasis for controlling the bleeding .

1- primary phase:

Vascular phase

- Vasoconstriction in the area of injury, Begins immediately after injury.

Platelet phase

- Platelets and vessel wall become "sticky”
- seconds after injury, mechanical plug of platelets seals off openings of cut vessels

Pathophysiology

2- *Secondary phase* (Coagulation phase)

- Blood lost into surrounding area coagulates through extrinsic and common pathways
- Blood in vessels in area of injury coagulates through intrinsic and common pathways
- Takes place more slowly than other phases

3- *Tertiary phase (*Fibrinolytic phase)

- Release of antithrombotic agents
- Spleen and liver destroy antithrombotic agents

Vascular phase

Begins immediately after injury involves vasoconstriction of arteries & veins in injured tissue . In normal condition the endothelial cells synthesize & secrete three potent anti platelet agents prostacyclin, nitric oxide & certain adenine nucleotides. Exposure of vessel wall sub endothelial tissues, collagen & basement membrane through chemical or traumatic injury serves a tissue factor (tissue thromboplastin) & initiates coagulation via the extrinsic pathway. Injured endothelial cell release adenosine diphosphate (ADP) which include platelet adhesion. Also promote thrombus formation through exposure of subendothlial tissue to von willebrand factor(vWF) Endothelial cell also contribute to normal homeostasis & vascular integrity through synthesis of type IV collagen fibronectin & vWF

platelet phase

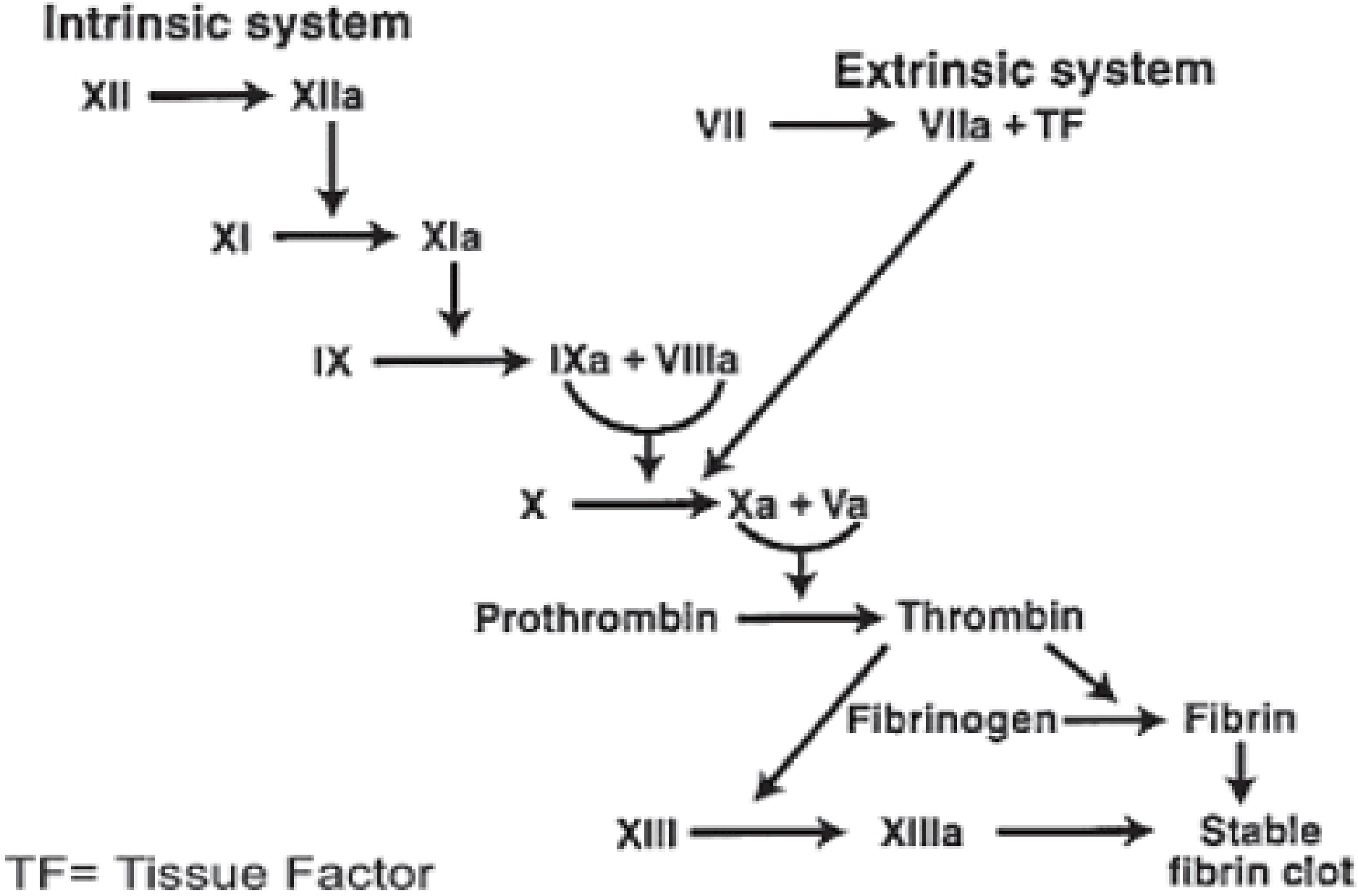
Platelet are cellular fragments they don't have nucleus stay 8-12 days in the circulation. Nonviable platelets are removed and destroyed by the spleen. Function of platelets include; maintenance of vascular integrity formation of platelet plug to aid in initial control of bleeding & stabilization of platelet plug through involvement in the coagulation process.

Coagulation phase

The process of coagulation is mean fibrin-forming system take 9-18 minutes from injury to a fibrin-stabilized clot.

Two systems involved in the coagulation of the extrinsic system is initiated through tissue factor after tissue injury. This process activate factor Vll (Vlla) which called in the past tissue thromboplastin.

The intrinsic system is initiated by surface contact to activate factor XII. Both systems (pathways) use common pathway to form the end product fibrin.



Fibrinolytic phase

Fibrin-lysing (fibrinolytic) system is needed to prevent coagulation of intra vascular blood away from the site of injury & to dissolve the clot once it has served its function in homeostasis. This system involve plasminogen, a proenzyme for the enzyme plasmin which produced in the liver. Endogenous plasminogen activator released by endothelial cells at the site of injury.

The effect of plasmin on fibrin & fibrinogen is to split off large piece that are broken up into smaller segments called fibrin degradation products (FPDs), those increase vascular permeability &interfere with thrombin induced fibrin formation thus causing bleeding problems.

A significant disorder that may occur in the vascular or platelet phase leads to an immediate clinical bleeding problem after injury or surgery. These phases are concerned with controlling blood loss immediately after an injury and, if defective, will lead to an early problem. However, if the vascular and platelet phases are normal, and the coagulation phase is abnormal, the bleeding problem will not be detected until several hours or longer after the injury or surgical procedure. In the case of small cuts, for example, little bleeding would occur until several hours after the injury, and then a slow trickle of bleeding would start. If the coagulation defect were severe, this slow loss of blood could continue for days.

Clinical presentation (Signs and Symptoms)

Signs associated with bleeding disorders **may appear in the skin or mucous membranes or after trauma or invasive procedures**.

Liver disease

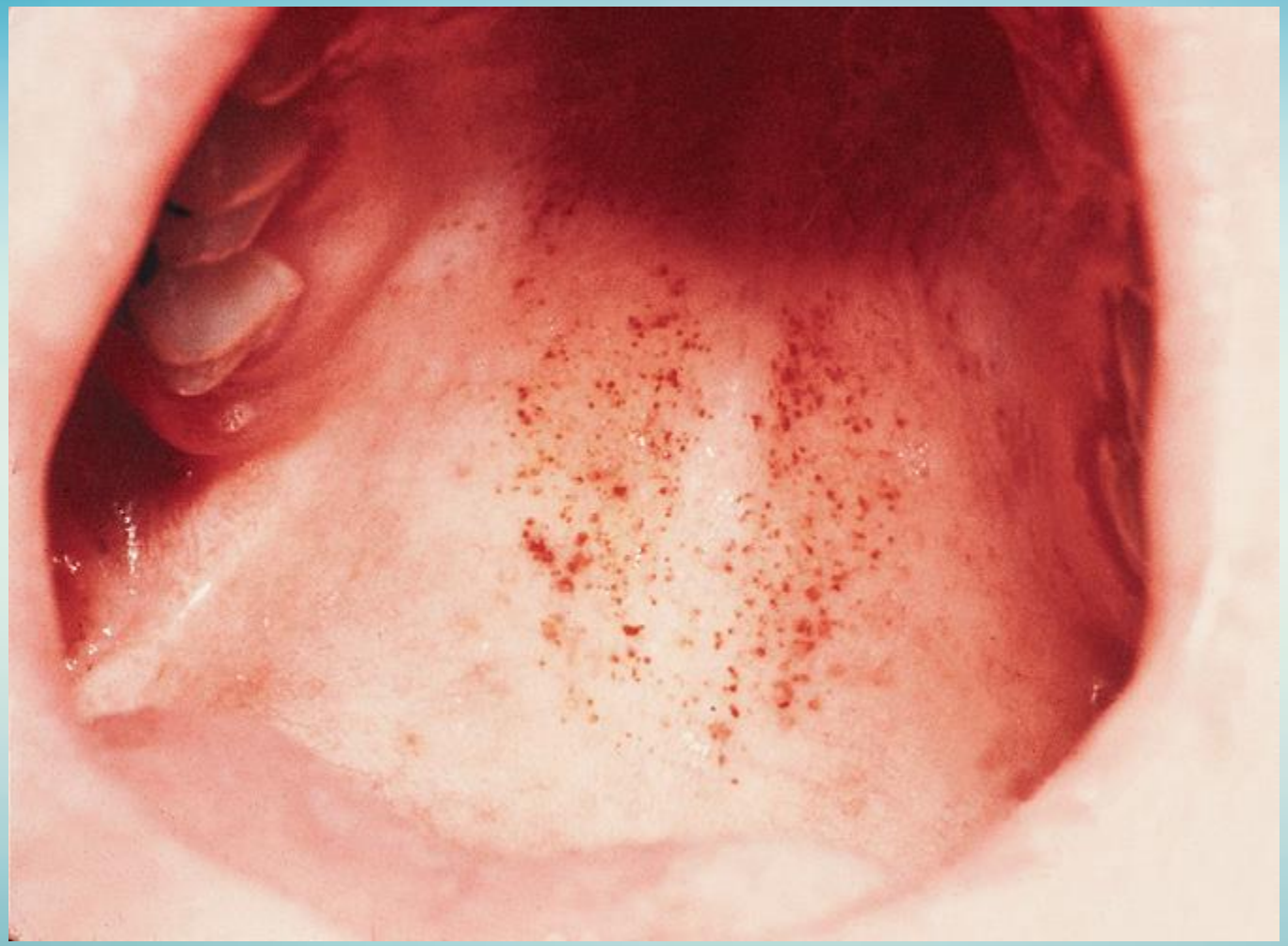
Jaundice, Spider angiomas, Ecchymosis, Petechiae on the skin or mucosa due to a reduction in platelets which occurs because of hypersplenism that results from the effects of portal hypertension .

Genetic coagulation disorders

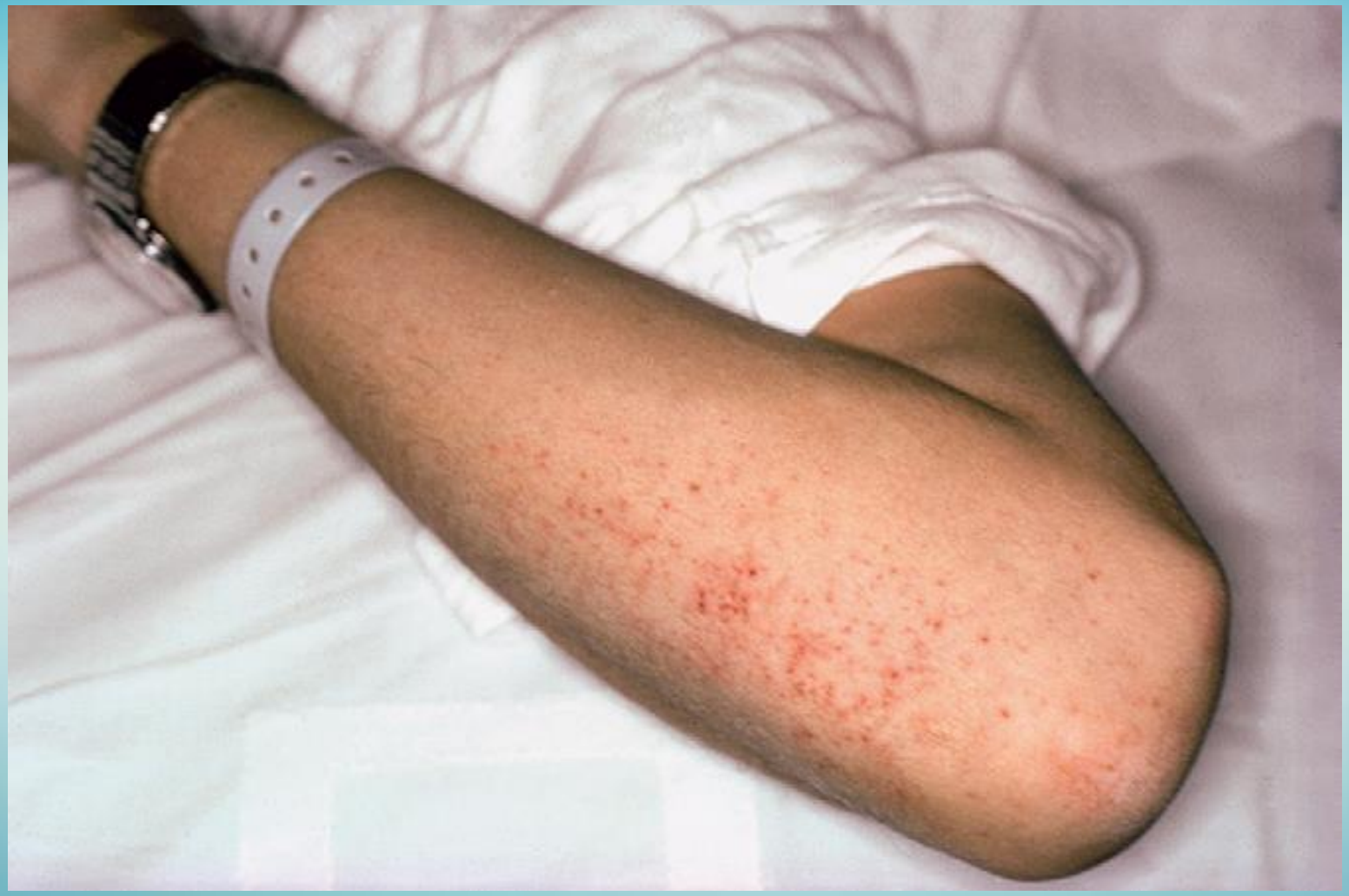
Ecchymosis, hemarthrosis & dissecting hematomas .

Chronic leukemia: ulceration of oral mucosa, hyperplasia of gingiva ,petechiae ,ecchymosis

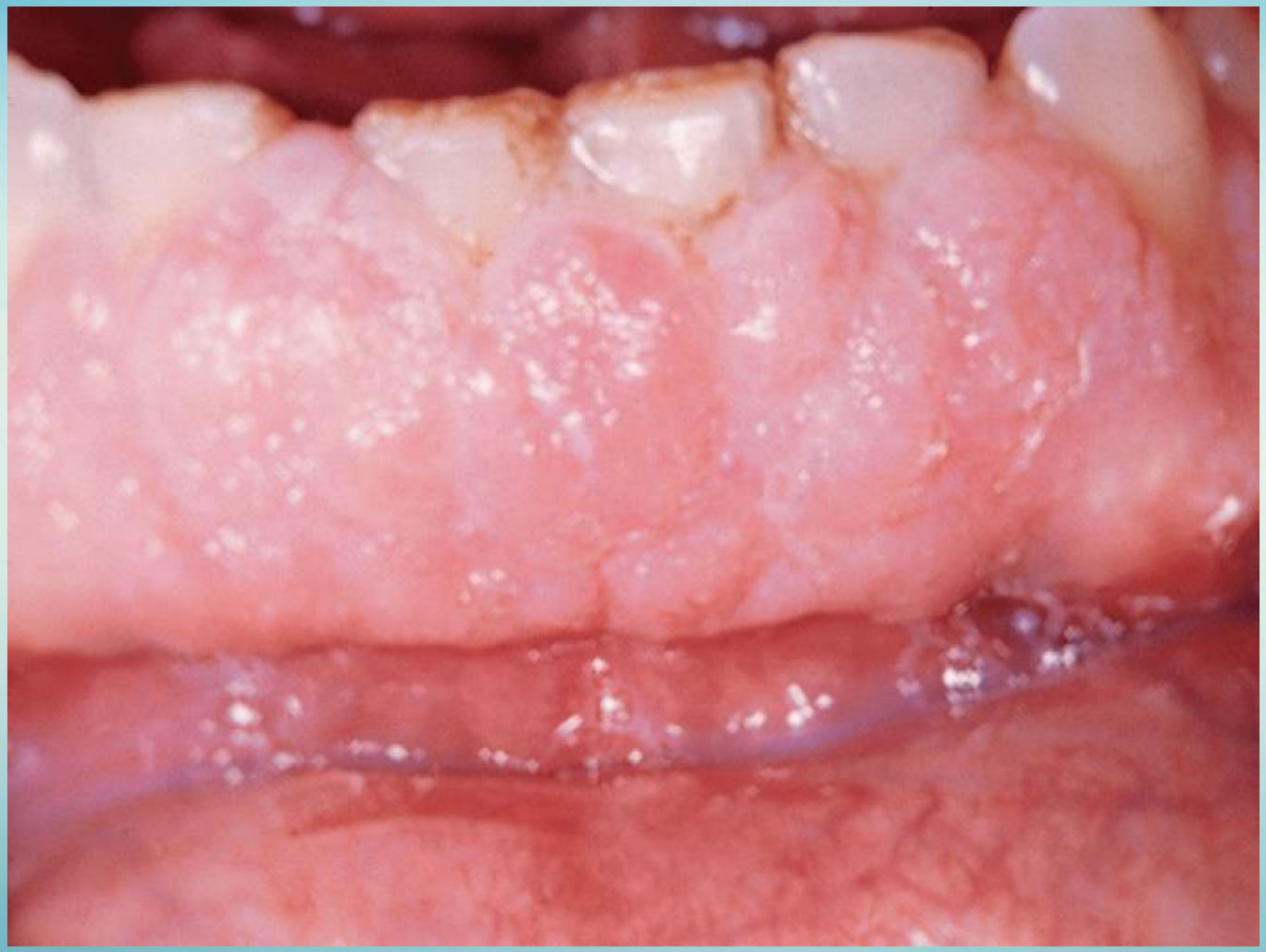
Petechiae of the palate



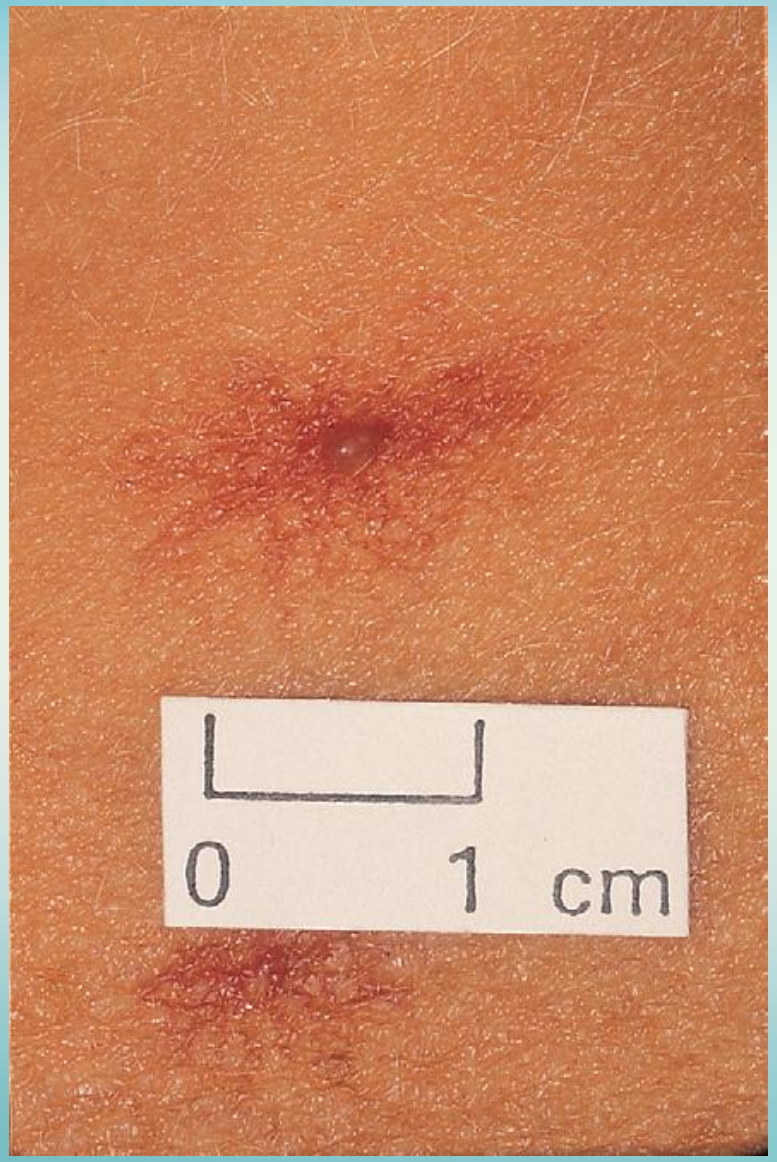
Petechiae of the skin



Hyperplasia of the ginginva



Spider angioma



Hemarthrosis

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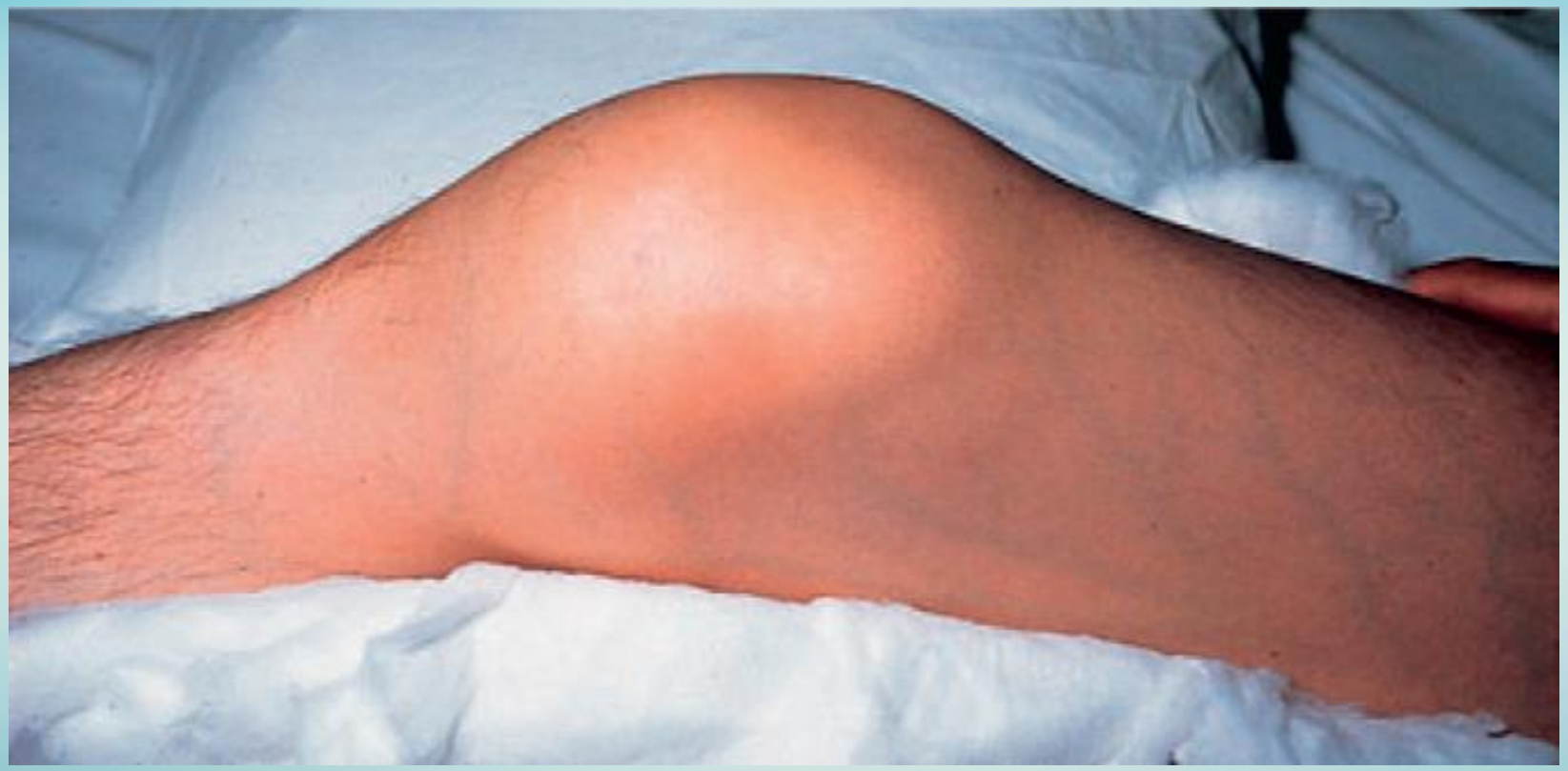

Laboratory tests

Partial thromboplastin time PTT: (25-35 seconds) this check the intrinsic system (factor VIII, IX, XI & XII) & common pathway factor (V&X) prothrombin & fibrinogen.

Prothrombin time PT : (11-15 seconds) used to check the extrinsic pathway (factor VII) & common pathway (V&X) prothrombin & fibrinogen)

- { VII & X and prothrombin) are vitamin k-dependent & are depressed by coumarin – like & should be checked by INR
- PT is prolonged when any factor is below 10% of its normal value.

Platelet count; 140 000-400 000 cubic mm normal.

- 50 000 -100 000 manifest excessive bleeding after sever trauma .
- Below 50 000 manifest bleeding after minor trauma skin & mucosal purpura .
- 20 000 /cubic mm spontaneous bleeding .

Bleeding time(BT); used to screening for disorder platelet dysfunction &thrombocytopenia.

Thrombin time(TT) : thrombin is added to the patient blood sample as activating agent ,it's convert fibrinogen into insoluble fibrin which makes up the essential clot .it bypasses the intrinsic ,extrinsic & common pathway .normal (9-13 sec). 16-18 sec.,consider prolonged. Which usually caused by excessive plasmin & or fibrin-split product.

Disorder of common pathway :-A prolonged a PTT & PT indicate a common pathway factor deficiency. Congenital deficiency of factor V&X, prothrombin & fibrinogen are rare , acquired deficiency like vit. K deficiency& liver diseases indicate Conditions that cause both testes (a PTT & PT) are prolonged.

MEDICAL MANAGEMENT

Vascular defects;

Aquired connective tissue disorder like scurvy vit C deficiency has capillary- fragility & delayed wound healing . long use of **steroid therapy** also lead to thinning of connective tissue may result in bleeding

Serum sickness can lead to purpura through **immune complex deposits** in the vessel walls .drugs like **penicillin , sulfonamide thiazide diuretics** & **hepatitis** associated with serum sickness .

Platelet disorders

Von Will Brand disease; most common disorder .autosomal dominant traits cause mild to moderate bleeding due to defect in platelet adhesion .vWB factor made from a group of glycoprotiens , this glycoprotien needed to carry factor VIII,(unbound factor VIII in circulation will be destroyed),& to allaw platelet to adhere to the tissue .the complex of factor VIII & vWB factor attaches to the surface of circulating platelets it's from this location that the vWB factor contribute to hemostasis .

Clinical finding ;

Patient with mild form of the disease may have -ve history .sever form may have +ve family history & sever bleeding after trauma or surgery it manifested as cutaneous & mucosal bleeding because of platelet adhesion lacking ,hemarthrosis ,epistaxis.

laboratory investigation ; PT& PTT are normal. aPTT prolonged , platelet count normal. another investigation needed to establish the diagnosis & type of vWB include immunoassay of vWF & specific assays for factor VIII .

TREATMENT ;by given cryoprecipitate .

MEDICAL MANAGEMENT

Platelet disorders

Disorders of platelet release ; in some cases platelet may fail to complete the release of **PF3 (platelet factor),** this due to defective thromboxane production due to anti inflammatory drugs, aspirin, NSAIDs (endomethacin ,ibuprofen ,sulfm pyrazone),beta-lacton anti biotic penicillin &cephalaxin .

MEDICAL MANAGEMENT

Coagulation disorders;

Hemophilia A;

Is an x-linked recessive trait. the abnormal homeostasis due to deficiency in factor VIII, this factor is bounded to vWF in circulation .

Clinical findings ; sever HE. cause sever bleeding . hemarthrosis , ecchymosis ,soft tissue hematomas, after trauma or surgery there will be sever bleeding which may threaten life. Spontaneous bleeding from mouth ,gingivae , tongue ,lips.

Hemophilic patient greatly affected by contamination from blood transfusion by HIV & hepatitis C virus.

Replacement of factor VIII include ;

- minor spontaneous bleeding 25-30 % replacement.
- minor dental surgery 50% replacement.
- Major surgery 80-100% replacement.

The choice of which type of factor concentrate should be used is based on specific findings from the patient management history & infection disease exposure .

MEDICAL MANAGEMENT

Coagulation disorders;

Hemophilia B;(christmas disease,factor IX deficiency)

X -linked recessive trait.

Clinical manifestation the same as hemophilia A . screening laboratory tests results are similar to both A&B .specific factor assays for factor IX establish the diagnosis .

purified factor IX product are recommended for treatment of minor & major bleeding recombinant factor IX is now available for clinical use .

MEDICAL MANAGEMENT

Coagulation disorders;

Disseminated intra vascular coagulation DIC

DIC is a condition that results when the clotting system is activated in all or a major part of vascular system. despite widespread fibrin production , the major clinical problems are bleeding not thrombosis , the syndrome is associated with infection , bum , snake bite ,shock .antigen/antibody complex , acidosis .obstetric complication (abruption placenta ,missed abortion ,amniotic fluid embolism).

Clinical finding of DIC ;

Sever bleeding from small wound ,purpura ,spontaneous bleeding from the nose ,gum ,GIT.UT.

Treatment; control of bleeding or thrombosis ,replacement of coagulation factors ,platelet & fibrinogen .patient given cryoprecipitate(fresh frozen plasma) . If thrombosis is a major problem heparin IV is given.

Anti coagulant drugs;

Heparin; heparin is used in high doses to treat thrombo embolism (IV bolus of 5000IU& iv infusion over 5-10 days period) & in low dose as prophylaxis for thromboembolism . Heparin should be given to the hospitalized patient ,it has plasma half life of 1-2 hours .it's required monitoring with a PTT.

Coumarin ; warfarin ; is most widely used as oral anti coagulant that inhibits the biosynthesis of VIT K-dependent coagulation proteins (factor VII,IX ,X & prothrpmbin). It metabolized by the liver & excreted by urine . PT is used to monitor warfarin therapy .the international normalized ratio is used to allows better comparison of PT values among different **laboratories INR for warfarin is 2.5 with rang 2.0-3.0 .**

anti platelet drugs ;

anti platelet treatment has been reported to reduce over all mortality from vascular complication by 30%. **Aspirin** has been widely used ,exert it's anti platelet action through anti thrombotic function & impairing aggregation . Non-steroidal anti-inflammatory drugs such as **ibuprofen** also inhibits the function of platelet .

Dental management of the patients with bleeding disorder

1-Patient identification

I. History

a. bleeding problems in relative .

b. bleeding problems after operation or teeth extraction .

c. bleeding after trauma or cutting wound .

d. medication causing bleeding problems; (aspirin ,anticoagulant drugs, long term anticoagulant therapy, certain herbal preparation)

e. presence of disease (leukemia ,liver disease ,hemophilia congenital heart disease &renal disease) .

f. spontaneous bleeding from nose & mouth .

II. **Clinical examination** ;

- jaundice .
- pallor.
- spider angioma .
- ecchymosis .
- patechiae .
- oral ulcers.
- hemarthrosis.

III- Screening laboratory test.

- PT—Activated by tissue thromboplastin
  - ✓Tests extrinsic and common pathways
  - ✓Normal (11-15 seconds, depending on laboratory)

- aPTT—Initiated by phospholipid platelet substitute and activated by addition of contact activator (kaolin)
  - ✓Tests intrinsic and common pathways
  - ✓Normal (25-35 seconds, depending on laboratory)

- TT—Activated by thrombin
  - ✓ Tests ability' to form initial clot from fibrinogen
  - ✓ Normal (9-13 seconds)

- PFA-100 (platelet function analyzer)
  - ✓ Tests platelet function
  - ✓ Normal if adequate number of platelets of good quality present
  - ✓ Normal (<175 seconds)

- Platelet count
  - ✓ Tests platelet phase for adequate number of platelets
  - ✓ Normal (140,000-400,000/mm 3)
  - ✓ Clinical bleeding problem can occur if less than 50,000/mm

IV- Surgical procedure ;

- excessive bleeding after surgery may be the first signs .
- The dentist should use the appropriate local procedures
- If these measures should fail, consultation with the patient's physician or hematologist is indicated
- Screening laboratory tests may be ordered to better identify the source of the problem before the consultation.

Dental management of the patients with bleeding disorder

2-Medical Considerations

No surgical procedures should be performed on a patient who is suspected of having a bleeding problem on the basis of history and physical examination findings. Such a patient should be screened by the dentist through appropriate clinical laboratory tests or should be referred to a hematologist for diagnosis, treatment, and management recommendations.

BOX 24-8 Selection of Screening Laboratory Tests for Clinical Recognition of the Patient with a Potential Bleeding Problem Based on History and Examination Findings

1. No clinical or historical clues to cause of bleeding problem: excessive bleeding occurs after surgery
2. History or clinical findings or both suggest possible bleeding problem but no clues to the cause: PT, aPTT, TT, platelet count
3. Aspirin therapy: PFA-100 if available
4. Warfarin (Coumadin) therapy: INR;
   low-molecular-weight heparin: aPPT
5. Possible liver disease: platelet count, PT
6. Chronic leukemia: platelet count
7. Malabsorption syndrome or long-term antibiotic therapy: PT
8. Renal dialysis (heparin): aPTT
9. Vascular wall alteration: BT (results often inconsistent)
10. Primary fibrinogenolysis (active plasmin in circulation), cancers (lung, prostate): TT

Dental management of patients who are receiving Antiplatelet Therapy

1- aspirin

- If the PFA-100 is moderately prolonged, the patient will not experience excessive bleeding with minor surgery unless some other bleeding disorder is present.

- If major surgery must be performed under emergency conditions, **desmopressin (DDAVP)(minrin)** can be used to reduce the risk of excessive bleeding. This should be done in consultation with the patient's physician or hematologist

Dental management of patients who are receiving Antiplatelet Therapy

2- NSAIDs

- Most invasive dental procedures can be performed without adjusting the dosage of the NSAID.

N.B. It should be remembered that the clinical risks of bleeding with aspirin or nonaspirin NSAIDs are enhanced by the use of alcohol or anticoagulants and by associated conditions such as advanced age, liver disease, and other coexisting coagulopathies

3- **clopidogrel and ticlopidine**

- In a recent study involving patients taking single or dual antiplatelet therapy who had invasive dental procedures (extractions, periodontal surgery, subgingival scaling, and root planning) it was found that no episodes of prolonged bleeding occurred.
- At this time it appears to be safe for patients taking single ticlopidine or clopidogrel therapy or dual therapy with aspirin to be maintained on their medication(s) for invasive dental procedures.
- For major oral surgical procedures that can't be delayed the **thienopyridines** may have to be discontinued until after the surgery. Consultation with the patient's physician is recommended.

Dental management of patients who are receiving Coumarin Therapy.

- The literature clearly supports the continuation of warfarin anticoagulation therapy for minor oral surgery and other similarly invasive dental procedures if the INR is 3.5 or less.
- Before performing surgical or invasive dental procedures, the dentist should obtain medical consultation for all patients who are taking warfarin.
- If acute infection is present, surgery should be avoided until the infection has been treated. When the patient is free of acute infection and the INR is 3.5 or less, minor surgery can be performed. The procedure should be done with as little trauma as possible.

- If excessive postoperative bleeding occurs after an extraction, **Gelfoam** with thrombin may be placed in the socket to control it. In addition, primary closure over the socket is desirable. **Oxycel, Surgicel, or microfibrillar** collagen may be used in place of Gelfoam

- tranexamic acid (Cyklokapron) or EACA (Amicar) mouthwash should be applied during the first 2 postoperative days to help control excessive bleeding.

- If excessive bleeding cannot be controlled by the local methods listed earlier, the dentist should consult the patient's physician. Available options include discontinuation of warfarin, which would take several days before an effect on bleeding would occur; administration of vitamin K; and administration of fresh frozen plasma or a prothrombin concentrate.

  Another option is to administer recombinant factor VIIa

- If the dosage of anticoagulant must be adjusted, the patient's physician should instruct the patient. It will take 3 to 5 days before the effect of the dose reduction is reflected in the lower INR. On the day of surgery, the INR should be checked again to determine whether the desired reduction has occurred. If no excessive bleeding occurs on the day after the dental procedure is performed, the patient's physician can direct the patient to return to his or her usual warfarin dosage.

- One approach is to have the patient's physician discontinue warfarin therapy 4 days before major oral surgery and to begin a **series of 30-mg subcutaneous enoxaparin**

- Through discontinuation of warfarin, the INR is allowed to normalize, and enoxaparin provides anticoagulation.

- Dentist must be aware that certain drugs will potentiate the anticoagulant action of warfarin include acetaminophen, metronidazole, salicylates, broad-spectrum antibiotics, erythromycin, and the new cyclooxygenase (COX)-2–specific inhibitors (celecoxib and rofecoxib).
- Other Drugs that the dentist may use that will antagonize the anticoagulant action of warfarin are barbiturates, steroids, and nafcillin. Other drugs that have the same effect are carbamazepine

So postoperative pain control can be attained with the use of minimal doses of acetaminophen with or without codeine.

Aspirin and NSAIDs must be avoided.

Dental management of patients with HEMOPHILIA ;

- **injection** ; block anesthesia ,lingual infiltration , or injection in the floor of the mouth ,&intra muscular injection should be avoided unless replacement of factors have been used in the patient with mild to sever factor VIII deficiency . infiltration anesthesia &intra-ligamentary injection usually given with out factor replacement.

- orthodontic treatment ,root canal with out over instrumentation , polishing can be done . periodontal surgery ,root planning extraction ,dento alveolar surgery &complex oral surgery need **factor replacement.**

- **preoperative consult the hematologist** to confirm the diagnosis severity of the disease ,mild to moderate form usually treated in dental clinic , sever cases treated in the hospital. replacement of factor VIII ,one hour before procedure .

- **dental treatment** ;good surgical technique , treat acute infection , pressure packs , use gelfoam with thrombin to control bleeding , splint for patient with multiple extraction .

- **postoperative** ; in dental clinic patient need second dose of factor VIII . hospitalized patient will need additional doses of factor replacement. also should check for signs of allergy . avoid uses of aspirin or NSAI Ds.

Patients with Von WELLBRAND'S DISEASE;

- Mild form ,surgical procedure can be done in the dental clinic without use of desmopressin & EACA or tranexamic acid . more sever require factor VIII construction .

- Dental management establish for good oral hygiene, palatal splint ,

- treatment of acute infection .

- post operative patient should examined 24-48 hours for bleeding if bleeding present give the patient tranexamic acid & EACA.

Dental management of patients with thrombocytopenia

- 30000 cubic mm(platelet count) infiltration & block anesthesia can given also most routine dental procedure can performed .
- 50000 cubic mm extraction or dento-alveolar surgery can be done.
- More advanced surgery need 80,000- 100,000/cubic mm or higher
- Patients with platelet counts below these levels will need platelet replacement before undergoing the planned procedures.
- The need for platelet transfusions can be reduced through the use of local measures , along with desmopressin and EACA or tranexamic acid to control bleeding. Also, topical platelet concentrates can be applied.

Oral Complications and Manifestations

patients with thrombocytopenia

- spontaneous gingival bleeding.
- Oral tissues (e.g., soft palate, tongue, buccal mucosa) may show petechiae, ecchymoses, jaundice, pallor, and ulcers.

patients with coagulation disorders

- Hemarthrosis of the temporomandibular joint (TMJ) is a rare finding in and is not found in patients with thrombocytopenia.


thanks

Dental management of patients with end – stage renal diseases (ESRD)

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ESRD is a bilateral, chronic, and progressive deterioration of kidneys that results in UREMIA and ultimately leads to death. The rate of this deterioration depends on the underlying causative factors, which in many cases remain unknown, however some of the more common known causes of ESRD are; accelarated malignent hypertension, diabetes mellitus, chronic glomerulonephritis, bilateral peylonephritis, polycystic kidney disease, obstructive uropathy, systemic lupus erythematosus ,

The early phase of ESRD is usually asymptomatic except for some mild laboratory abnormalities and is called renal insufficiency. More damage occurs progressively resulting in decreased ability of the kidneys to perform their excretory, endocrine, and metabolic functions beyond compensatory mechanisms, the disease then becomes frank renal failure, and the resulting syndrome is called UREMIA which is uniformly fatal if not treated. Usually normal kidney functions are maintained until 50% of the nephrons are destroyed, when a period of relative renal insufficiency ensues, during which the patients are asymptomatic but demonstrate laboratory abnormalities that reflect a diminished glomerular filtration rate. When damage goes on beyond this point compensatory mechanisms (nephron hypertrophy) are overwhelmed and signs and symptoms of uremia appear.

SEQUELAE AND COMPLICATIONS

- The failing kidneys are unable to handle the sudden large intake of sodium and water which contributes to the development of **fluid overload**. The cardiovascular system is affected by a tendency to develop congestive heart failure, pulmonary edema or both. Arterial hypertension, left ventricular hypertrophy also occurs which may compromise coronary circulation. There is also a tendency for accelerated atherosclerosis.

- Reduction or loss of the glomerular filtration function results in build up of nonprotien nitrogen products (mainly urea) in the blood, a condition known as **azotemia.**

- Tubular impairment results in acids accumulation and the development of **metabolic acidosis** , the major result of which, is ammonia retention . The patient may tend to hyperventilate to attempt a respiratory compensation for the metabolic acidosis. It is worth noting that in such patients compensatory mechanisms are already taxed beyond normal, and any increase in demand as in sepsis or febrile illnesses can lead to serious consequences.

- Because of tubular malfunction **sever electrolyte and fluid disturbances** occur. Sodium depletion due to sodium excretion along with excess amounts of dilute urine is commonly encountered. With progressive azotemia, hyperkalemia also may develop and becomes evident as urine output falls.

- Patients with ESRD suffer several haematologic abnormalities, these include:
  - **ANEMIA,** which is due to decreased erythropoietin production, shortened red cell survival, RBC haemolysis, bleeding, and inhibition of RBC production
  - **LEUKOCYTE PRODUCTION AND FUNCTION CHANGES**, caused by reduced bioavailability of interleukin 2, down regulation of phagocyte adhesion molecules, increased bioavailability of interleukins 1,6, and tumor necrosis factor, cell-mediated immune defects, and hypogammaglobulinemia that lead to diminished granulocyte chemotaxis, phagocytosis, and bactericidal activity.
  - **COAGULOPATHY,** characterized by a tendency to abnormal bleeding and bruising, which are attributed to abnormal platelet aggregation and adhesiveness, decreased platelet factor 3 production, impaired prothrombin consumption, and defective platelet production.

- Bone disorders , collectively known as **RENAL OSTEODYSTROPHY** are seen in ESRD, they include **Osteomalacia** (increased unmineralized bone matrix), **Osteitis fibrosa**(bone resorption and marrow fibrosis), and OSTEOSCLEROSIS (enhanced bone density). Renal osteodystrophy patients show a tendency for spontaneous fractures with slow healing, aseptic necrosis of the hip, extraosseous calcifications and myopathy. The underlying cause is a rise in serum phosphate ievel secondary to decreased glomerular filtration rate, a raised serum phosphate forces serum calcium to be deposited in bone resulting in low serum calcium which in turn stimulates parathyroid hormone production and secondary hperparathyroidism development, this is aided by the fact that the failing kidneys of ESRD are unable to produce 1,25-dihydroxycholecalciferol .

Clinical picture

- Uremic patients demonstrate mental depression and even psychosis in the late stages of disease along with muscular hyperactivity and convulsions. Lethargy, listlessness, and dizziness are also common. Gastrointestinal signs of anorexia, nausea, vomiting, diarrhea, gastroenteritis, and peptic ulcer disease are common.

- Stomatitis manifested by oral ulceration, and candidiasis can occur.
- Parotitis may occur in uremic patients .
- Pallor of skin and mucous membranes due to anemia,
- Hyperpigmentation of skin due to carotene like pigments retention,
- Pruritis, and uremic frost are also common.
- Ecchymosis , petechiae, purpuric spots, gingival bleeding, epistaxis, and occult gastrointestinal bleeding are all common presenting signs in uremia

Medical management

Conservative Care

This approach to treatment is the first step and may prove adequate for prolonged periods of time. Conservative care attempts to slow the progression of renal disease, preserve life quality, decrease the retention of nitrogenous waste products, control fluid and electrolytes imbalance, control or correct treatable associated conditions such as hypertension, congestive heart failure, obstructive urinary disease, volume depletion, infection, diabetes mellitus, secondary hypereparathyroidism, anemia. When kidney deterioration goes on and the number of functioning nephrons drops to a point that azotemia is unpreventable or uncorrectable by conservative measures then dialysis becomes inevitable.

Dialysis

Is a medical procedure that filters blood artificially, it can be accomplished by either peritoneal or haemodialysis.

A- Peritoneal Dialysis: this method is reserved for patients who are in acute renal failure or require only occasional dialysis. When compared to haemodialysis it is easier to perform, less costly, lacks the need for anticoagulation, and safer when blood borne infection transmission is considered.

B- Haemodialysis: is the method of choice when dialysis must be a chronic treatment .It requires the surgical creation of a permanent arteriovenous fistula, infection of the later is a main concern as it can result in septicemia, septic emboli, infective endarteritis or endocarditis, inaddition hepatitis B and C and HIV infection transmission is an other concern .

Abnormal bleeding tendency in E.S.R.D. patients made worse by the need for anticoagulation during haemodialysis, and by platelet destruction by the dialysis machine adds another concern when oral surgical procedures are to be carried out.

DENTAL MANAGEMENT

A- Patients under conservative care :

- Patient identification achieved via a detailed history taking .
- Consultation with patients physician. If this consultation reveals that:

  a- Patients condition is well controlled, then both urgent or even elective dental care can be safely provided to patients on outpatient basis.

  b- Patients condition is in advanced stages of renal failure or complicated by other systemic diseases common to renal failure like hypertension, congestive heart failure, pulmonary edema, then dental treatment must be provided in a hospital-like setting, and deferral of treatment may be required until adequate control of patients condition is reached.

- If it is decided to treat the patient on out-patient basis, patients **blood pressure** should be closely monitored both pre and intra operatively. Any abnormal preoperative readings should be reported to patients physician for possible control measures. During treatment the dentist should do his best to minimize the physical and emotional stress of dental treatment.

- The patient should receive pretreatment screening for **anemia and bleeding disorders** if a hemorrhagic procedure is intended (Hb%, Hematocrit level, Bleeding time, Platelet count).

- In general non hemorrhagic dental procedures can be safely done to patients with hematocrit levels higher than 25%, however the hematocrit level should be significantly raised to reach close to lower normal value if a hemorrhagic surgery is intended. Measures to raise the hematocrit level include:

  a- Erythropoietin use in a dose of 50=75 I.U./Kg tri weekly, **b-** RBCs or even whole blood transfusions, though possible, are less desirable options, because of fluid overload, and infection transmission risks

- Bleeding disorders in uremic patients are best controlled with measures like;

  a- Topical thrombin use **b-** Microfibrillar collagen use **c-** Sutures **d-** Systemic desmopressin in adose of 0.3 ug/Kg over 30 minutes **e-** Conjugated estrogens use **f-** Cryoprecipitate use or platelet transfusions are less desirable

- Attention to good surgical technique should be paid to decrease the risk of excessive bleeding and infection .

- Drug prescribing to patients with end stage renal disease presents a serious problem that should be discussed with the patients physician. In general the rules that need to considered are:
  - The drugs that are excreted primarily by the kidneys and are also nephrotoxic like **aminoglycosides**, **tetracyclines**, **acetyl salicylic acid**, and other non steroidal anti inflammatory drugs, acyclovir,....are to be avoided.
  - The drugs excreted by the kidneys may reach toxic levels when the glomerulr filtration rate drops seriously, thus the dosage and frequency of administration of these drugs may need to be altered,
  - The dosage and frequency of administration of other drugs may need to be adjusted in ESRD patients for reasons other than nephrotoxicity or renal metabolism and excretion.

- The local anesthetic lidocaine can be used safely in ESRD patients and it requires no dose adjustments.
- General anesthesia is reserved for major surgical procedures provided that the Hb% exceeds 10g/100ml.
- Orofacial infections must be treated vigorously by antibiotics and culture , sensitivity tests are quite useful in such patients.
- ESRD patients must be hospitalized in cases of sever infections or major surgical procedures.

B- Patients receiving haemodialysis

The ideal method of managing such patients consists of the same steps just mentioned for managing patients under conservative care in addition to the followings :

- Consult with the patients physician regarding the need for a preoperative prophylactic antibiotic cover.

  The American heart association cites poor evidence and an apparent low risk for not making a recommendation to provide prophylactic antibiotic cover.

- Drug prescribing is again a matter of concern since some of the drugs are removed by haemodialysis , thus dosage adjustments are necessary.

- Avoid applying blood pressure-measuring cuffs and intra-venous injections in the arteriovenous fistula-containing arm .

- Avoid dental treatment on the day of dialysis as patients usually grow tired after haemodialysis sessions and the anticoagulant effect of heparin may require 6-8 hours to clear off. If an urgent hemorrhagic procedure must be done immediately following dialysis session protamine sulfate can effectively block the effect of heparin
- Screen haemodialysis patients for HBsAg and HIV before treatment and treat the patients as potential carriers.

Note :The method of managing patients receiving peritoneal dialysis is identical to that of patients under conservative care.


THANK
YOU

DENTAL MANAGEMENT OF PATIENTS RENAL INSUFFICIENCY

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The adrenal glands are located bilaterally at the superior pole of each kidney. Each adrenal gland consists of an outer cortex and an inner medulla.

The adrenal medulla functions as a sympathetic ganglion and secretes catecholamines, primarily epinephrine, whereas the **adrenal cortex** secretes several steroid hormones with multiple actions

The adrenal cortex which makes about 90% of the bulk of the gland consists of **three zones** :

Zona Glumerulosa, which is the outer most zone, it predominantly secrets aldosterone, a mineralocorticoid essential for sodium, potassium levels regulation, and maintenance of extracellular fluid volume.

Aldosterone secretion is regulated by the renin-angiotensin system, ACTH, and plasma sodium and potassium levels. A fall in renal arteriolar pulse pressure, which results from decreased intra vascular volume or a sodium imbalance, causes renin release which in turn activates angiotensin and the later causes aldosterone release which stimulates sodium absorption along with osmotic ally equivalent amounts of water at the distal tubules and collecting ducts of kidneys.

- **Zona Fasciculate**, which is the middle zone, it secretes glucocorticoids, Cortisol the primary glucocorticoid, has several important physiologic actions on metabolism, cardiovascular function, the immune system, and for maintaining homeostasis during periods of physical or emotional stress.

Cortisol acts as an **insulin antagonist**, increasing blood levels and peripheral use of glucose by activating key enzymes involved in hepatic gluconeogenesis and inhibiting glucose uptake in peripheral tissues (i.e., skeletal muscles).

In adipose tissue, cortisol activates lipolysis, resulting in the release of free fatty acids into circulation.

Cortisol **increases blood pressure** by potentiating the vasoconstrictor action of catecholamines and angiotensin II on the kidney and vasculature.

Its **antiinflammatory action** is modulated by its inhibitory action on (1) lysosome release, (2) prostaglandin production, (3) eicosanoid and cytokine release, (4) endothelial cell expression of intracellular and extracellular adhesion molecules (ICAMs and ECAMs, respectively) that attract neutrophils, and (5) leukocyte function. Cortisol also **activates osteoclasts and inhibits osteoblasts**.

Cortisol secretion normally follows a diurnal rhythm with peak plasma levels at the time **of awakening in the morning and lowest levels at evenings**. This pattern is reversed in individuals who work at night and sleep the day.

Regulation of cortisol secretion occurs via the hypothalamic-pituitary-adrenal (HPA) axis. Central nervous system afferents mediating diurnal (circadian) rhythm, and responses to stress, stimulate the hypothalamus to release corticotropin-releasing hormone (CRH), which stimulates the production and release of adrenocorticotropic hormone (ACTH), which in turn stimulates the adrenal cortex to produce and secrete cortisol.

Normally 20 mg of cortisol are secreted daily.

During periods of stress the HPA axis is stimulated, resulting in increased secretion of cortisol. Various stressors such as trauma, burns, fever, illness, hypoglycemia, emotional upset can stimulate the HPA~axis.

Surgery is a potent activator of the HPA axis with a peak response noted in the immediate postoperative period, which can be reduced by morphine-like analgesics, benzodiazepines, local anesthetics, suggesting that pain response mechanisms increase the requirement for cortisol.

Zona Reticularis the inner zone secretes androgens, **d**ehydroepiandrosterone is the principal androgen secreted by the cortex, also estrogen precursors are secreted from this zone.

The effects of the adrenal androgens are the same as those of testicular androgens, masculinization and the promotion of protein anabolism and growth.

ADRENAL DISORDERS

HYPERADRENALISM

Hyperfunction of the adrenal glands results in overproduction of cortisol, aldosterone, androgens, or estrogens in isolation or in combination and can produce syndromes that are dependent on the adrenal product that is in excess.

- The most common form of hyperadrenalism is due to;

glucocorticoid excess either endogenous or exogenous, which leads to the development of **Cushing's Syndrome.**

Cushing's Syndrome _ classically produces weight gain, round or moon shaped facies, buffalo hump on the upper back, abdominal striae, hirsutism, acne, hypertension, heart failure, glucose intolerance, diabetes mellitus, osteoporosis and bone fractures, impaired healing, and psychiatric disorders which include mental depression, mania, anxiety disorders, cognitive dysfunction, and psychosis.

Long term use of steroids may also increase the risk for peptic ulcers, cataract formation, glaucoma, and growth suppression.

- **Mineralocorticoid Excess** primary aldosteronism, is associated with hypertension, hypokalemia, and dependent edema.
- **Androgen Excess-related disorders** are rare and primarily affect the reproductive organs.

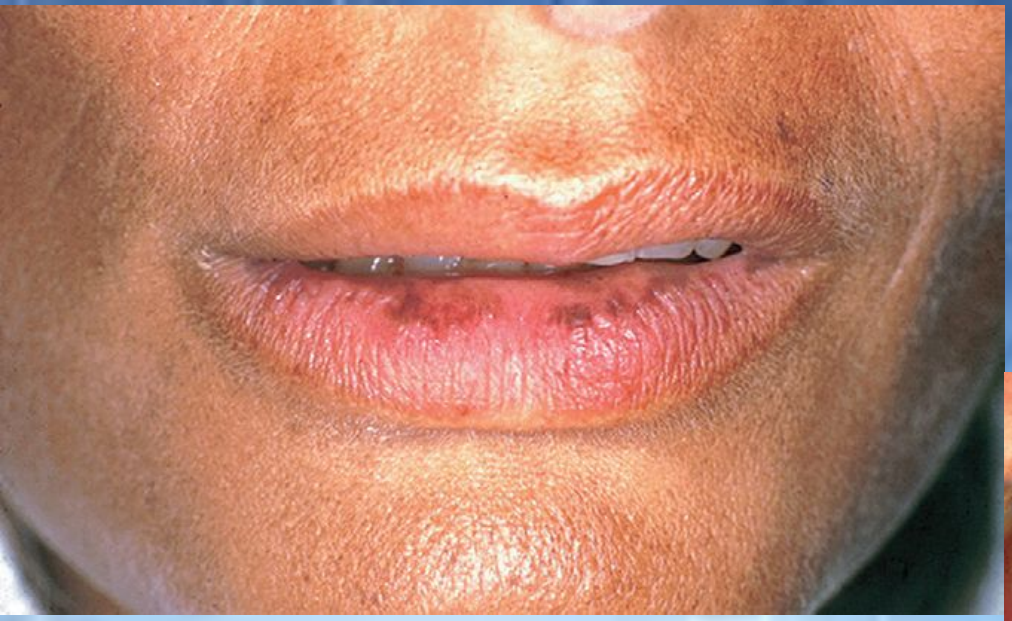
HYPOADRENALISM (Adrenal Insufficiency)

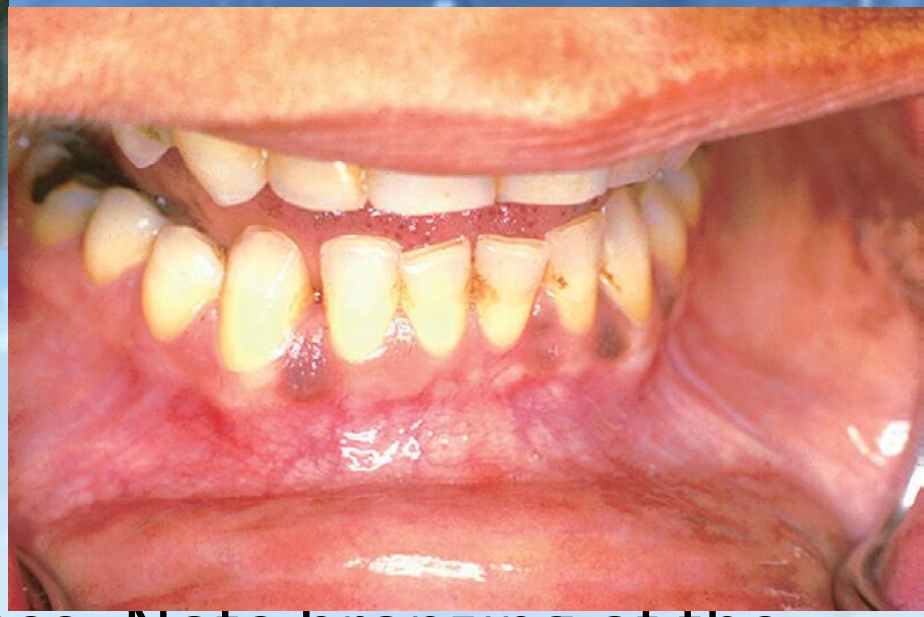
Insufficient adrenocortical function (insufficient glucocorticoid production) may occur primarily or secondarily.

- Primary Adrenocortical Insufficiency
- **Addison's disease**, is uncommon, it occurs at a rate of approximately 8 per million annually. Primary adrenal insufficiency follows progressive destruction of the adrenal cortex which is usually of an idiopathic nature, haemorrhage, sepsis, tuberculosis, HIV or cytomegalovirus infections, fungal infections, malignancy, adrenalectomy, and drugs are other possible causes.
- The signs and symptoms of the disease are the result of adrenocortical hormones deficiency. Usually clinical evidence of deficiency appears when 90% of the adrenal cortex has been destroyed.

The presenting signs and symptoms of primary adrenal insufficiency (Addison's disease) relate to cortisol and aldosterone deficiency, patients usually complain of weakness, fatigue, weight loss, abnormal skin and mucous membrane pigmentation, and if the patient is challenged with stress like infection or surgery, adrenal crisis may be precipitated. The patient will present with profuse sweating, weak pulse, hypotension, dyspnea, cyanosis, nausea, vomiting, headache, weakness, dehydration, fever, myalgia, arthralgia, and hyponatremia.

The condition constitutes a medical emergency and if not treated urgently, the patient may develop sever hypotension, hypothermia, hypoglycemia, and circulatory collapse, and may even die.





Patient with Addison's disease. Note bronzing of the skin with pigmentation of the lip and the oral mucosa

Secondary Adrenocortical Insufficiency.

this may result from destructive hypothalamic pituitary disorders or, more commonly, to long term corticosteroid use which may cause a partial insufficiency that is related only to glucocorticoids. The condition usually does not produce signs and symptoms unless the patient is significantly stressed and does not have adequate circulating cortisol, thus an adrenal crisis is a possible sequela, however, an adrenal crisis in a patient with secondary adrenocortical insufficiency is rare and tends to be milder than that seen in patients with primary adrenocortical insufficiency because aldosterone secretion is normal. Severe hypotension, dehydration and shock are seldom encountered in secondary insufficiency category.

LABORATORY FINDINGS

From dental management perspective, cortisol deficiency stands as a primary concern.

- Cortisol levels can be measured in urine, plasma, and saliva, and of the three fluids saliva appears to be the most sensitive, however, cortisol level values may be altered by a variety of factors, including circadian rhythm, diet, and stress.
- Early morning normal plasma cortisol levels range from 10-20ug/dl, late afternoon values range from 3-10 ug/dl.
- Late night salivary cortisol testing is recommended to minimize the effects of circadian rhythm.
- Positive cortisol screening tests should be followed by provocative tests of the HPA axis, these include :
- Synthetic ACTH (cosyntropin) stimulation test
- CRH test, differentiates ACTH dependent from ACTH independent disorders
- Dexamethasone suppression test, used for screening for adrenal hyperfunction
- The ACTH stimulation test is the most reliable test for adrenal hypofunction, it directly evaluates the adrenal reserve. A subnormal test response, though suggestive of adrenal insufficiency, has little correlation with the patient ability to respond to stress.

Medical management of Addison's disease patients is aimed at, managing the adrenal disease (elimination of the infectious agent or neoplasm), and hormonal replacement. Glucocorticoid replacement is accomplished at levels that correspond to the normal physiologic output of the adrenal cortex, about 20-30mg of hydrocortisone or its equivalent per day, the need for supplemental glucocorticoids during periods of stress, illness and trauma continues indefinitely. Mineralocorticoid replacement is accomplished by daily administration of O.o5- o. 1 mg fludrocortisone.

Secondary Adrenal Insufficiency

Secondary adrenal insufficiency resulting from destructive hypothalamic- pituitary disorders, requires correcting the ACTH dependent disorder and replacing the missing glucocorticoid. Secondary adrenal insufficiency that results from long term use of steroids in the management of nonendocrine disorders requires steroids replacement. The aim is to achieve resolution of the disease symptoms while minimizing adverse effects including secondary adrenal insufficiency. Depending on the condition, dosages are targeted to be equal to or less than the dai ^"replacement dose of the preparation used. Hydrocortisone is usually dispensed at about 20mgdaily, prednisone or prednisolone at 5mg daily, and dexamethasone at 0.5 mg daily

Such regimens given as single morning doses are usually less suppressive. Higher and divided doses are more suppressive and usually take at least three weeks to result in clinical manifestations of glucocorticoid deficiency.

Patients who are provided steroids for low adrenal reserve and to prevent adrenal crisis during and after surgery are a serious concern. The general.consensus has been that these, at-risk, patients should be provided supplemental steroids during periods of stress, trauma, and illness. Surgery is known to cause an increase in plasma cortisol level both during and after surgery, this increase ranges between two and ten folds above base line between 4 and 10 hours after surgery. The level of response depends on the magnitude of the surgery, the severity of postoperative pain, and whether general anesthesia is used.

Adults secrete 75-150mg of cortisol a day in response to major surgery and only 50mg a day following minor procedures. Cortisol secretion in the first 24 hours after surgery rarely exceeds 200mg.

Studies have shown that the vast majority of patients who take mean doses of 5-1 Omg prednisone daily for longtime maintain adrenal function and do not require supplementation for minor procedures, also a significant proportion of patients who took 5-50mg prednisone daily for between 6 days and 10 years and stopped therapy before surgery produced plasma cortisol ievels similar to those of normal subjects for up to 7days after minor or major surgery.

In general the risk of adrenal crisis following minor surgery appears to be low. If doubt exists as to the adrenal cortical status of a patient w'ho is to have surgery, a stimulation test of the HPA axis, ACTH stimulation test, is indicated as a preoperative screening evaluation of adrenal function. If the evaluation demonstrates inadequate HPA axis function, or inadequate adrenal reserve, or if preoperative testing is not performed, a perioperative glucocorticoid coverage should be provided according to the recommendations put forth by **salem et al :**

- For minor surgical stress the glucocorticoid target is about 25mg of hydrocortisone equivalent on the day of surgery. A patient who takes 5mg prednisone every other day should receive 5 mg prednisone on the day of surgery preoperatively.

- For moderate surgical stress, the glucocorticoid target is about 50-75mg of hydrocortisone equivalent per day for 1 to 2 days. A patient who takes 1 Omg prednisone daily should receive lOmg prednisone or (parenteral equivalent) preoperatively and 50 mg hydrocortisone I.V. intraoperatively. On the first postoperative day, patient should receive 20mg of hydrocortisone I.V. 8 hourly, the patient should be returned to the preoperative dose on the second postoperative day.
- For major surgical stress the glucocorticoid target is about 100-150mg of hydrocortisone equivalent per day for 2-3 days. A patient who takes 40mg prednisone a day for years should take 40mg prednisone or (the parenteral equivalent) preoperatively and 5Omg hydrocortisone I.V. 8 hourly following the initial dose for the first 48-72 hours postoperatively. A patient who takes 5mg prednisone and w'ho is to have a similar major surgery is recommended to receive 5mg prednisone or the (parenteral equivalent) as a preoperative dose, with 25mg of hydrocortisone given intraoperatively and 25mg administered every 8hours postoperatively for 48 hours.
- If the postoperative course is uneventful, the patient is returned to the usual glucocorticoid dosage uponxompFetion ofthe.regimen. Factors that may complicate the postoperative course and exacerbate^adrenal insufficiency include liver dysfunction, febrile illness, sepsis, nausea, and vomiting, and drugs (aminoglutethimide, etomidate, ketoconazole, phenytoin, barbiturates, rifampicin).

Adrenal Crisis

Adrenal crisis is an acute adrenal insufficiency episode with life threatening outcome, fortunately, the condition is rare. The condition requires immediate treatment with IV injection of glucocorticoid. A 100 mg hydrocortisone bolus dose is slowly injected followed by similar doses of hydrocortisone every 6-8 hours during the first 24 hours, blood pressure is also supported with fluids, electrolytes, and vasopressors, along with correction of hypoglycemia.

DENTAL MANAGEMENT Patients with Hyperadrenalism

Patients with hyperadrenalism or who take corticosteroids for prolonged periods have an increased likelihood of having **hypertension, diabetes, delayed wound healing, osteoporosis, and peptic ulcer disease**.

- To minimize the risk of an adverse outcome, blood pressure should be taken at baseline and monitored during dental appointments.
- Blood glucose levels should be determined and invasive procedures should be performed during periods of good glucose control.
- Follow-up appointments should be arranged to assess proper wound healing.
- Because osteoporosis has a relationship with periodontal bone loss, implant placement and bone fracture, periodic measures of periodontal bone loss are indicated, and measures should be instituted that promote bone mineralization and avoid extensive neck manipulation if osteoporosis is severe.
- Because of the risk of peptic ulceration, postoperative analgesics for long-term steroid users should not include aspirin and other nonsteroidal antiinflammatory drugs (NSAIDs).

Evidence indicate that the vast majority of patients with adrenal insufficiency may undergo routine dental treatment without the need for supplemental glucocorticoids. Individuals at risk for adrenal crisis are those who undergo stressful surgical procedures (extraction of bony impactions, osteotomies, bone resection, cancer surgery), and have no or low adrenal function because of primary or secondary adrenal insufficiency.

The two major factors influencing the recommendation for supplemental corticosteroids are the type of adrenal insufficiency and the level and type of stress. Currently, only patients with primary adrenal insufficiency are recommended to receive supplementation, and this recommendation applies only when surgery is being performed and/or in the management of a dental or systemic infection Patients with secondary adrenal insufficiency and those who take daily or alternate-day corticosteroids have enough exogenous and endogenous cortisol to handle routine dental procedures and surgery, if their usual steroid dose (or parenteral dose equivalent) is taken the morning of the procedure. Thus, the recommendation is for patients to take their usual daily dose of steroid within 2 hours of the surgical procedure, and that the surgeon, anesthetist, and nurses be advised of possible complications associated with the patient's adrenal state.

Routine dental procedures do not stimulate cortisol production at levels comparable with those that occur at the time of surgery and do not require supplementation, even in patients with controlled primary adrenal insufficiency.

Patients undergoing surgery should be closely monitored for blood and fluid loss and for hypotension during the postoperative period. If hypotension appears during monitoring, intravenous fluids are to be given and additional doses of corticosteroid considered if fluid replacement fails to rectify the blood pressure. Patients are returned to their usual glucocorticoid dosage as soon as their vital signs are stabilized.

Additional measures recommended to minimize the risk of adrenal crisis associated with surgical stress

- Surgery should be scheduled in the morning when cortisol levels are highest.
- Proper stress reduction should be provided because fear and anxiety increase cortisol demand. Nitrous oxide–oxygen inhalation and benzodiazepine sedation are helpful in minimizing stress and reducing cortisol demand.
- In contrast, reversal of and recovery from general anesthesia and extubation, and not the trauma of surgery itself, are major determinants of secretion of ACTH, cortisol, and epinephrine. Thus, general anesthesia increases glucocorticoid demand for these patients.
- Barbiturates also should be used cautiously because these drugs enhance the metabolism of cortisol and reduce blood levels of cortisol.
- In addition, inhibitors of corticosteroid production (e.g., ketoconazole metyrapone, aminoglutethimide) should be discontinued at least 24 hours before surgery, with the consent of the patient's physician.
- Surgeries that last longer than 1 hour are more stressful than shorter surgeries and should be considered major surgical procedures that can require the need for steroid supplementation.
- Blood and fluid volume loss exacerbate hypotension, thereby increasing the risk for development of adrenal insufficiency–like symptoms. Thus, methods of reducing blood loss are important in this setting.

- Likewise, a fasting state can contribute to hypoglycemia which can mimic features of an adrenal crisis, but does not require glucocorticoids for resolution.
- Patients who take anticoagulants are at increased risk for postsurgical bleeding and hypotension.
- In addition, inadequate pain control during the postoperative period increases the risk of adrenal crisis. Clinicians should provide good postoperative pain control by means of long-acting local anesthetics (e.g., bupivacaine) given at the end of the procedure.

- Monitoring of blood pressure throughout the procedure is critical for recognition of the development of an adrenal crisis. During surgery, blood pressure should be evaluated at 5-minute intervals and before the patient leaves the office. A systolic blood pressure below 100 mm Hg or a diastolic pressure at or below 60 mm Hg represents hypotension. A diagnosis of hypotension dictates that the clinician must take corrective action.
  - This would include proper patient positioning (i.e., head lower than feet),
  - fluid replacement,
  - administration of vasopressors,
  - and evaluation for signs of adrenal dysfunction versus hypoglycemia.

- Immediate treatment during an adrenal crisis requires the administration of 100 mg of hydrocortisone or 4 mg of dexamethasone IV and immediate transportation to a medical facility. In as much as significant cortisol increases generally are not seen before or during the operation but are increased in the postoperative period, approximately 1 to 5 hours after the procedure commensurate with the pain response, and the rise in cortisol levels is blunted by the use of analgesics and midazolam, good pain control with local anesthesia and analgesics is recommended for these patients.

Oral Complications and Manifestations

In primary adrenal insufficiency, diffuse or focal brown macular pigmentation of the oral mucous membranes is a common finding . Pigmentation of sun-exposed skin often follows the appearance of oral pigmentation and is accompanied by lethargy. Patients with secondary adrenal insufficiency may be prone to delayed healing and may have increased susceptibility to infection.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Neurologic Disorders

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1- Epilepsy

is not a specific diagnosis but rather a term that refers to a group of disorders characterized by chronic and recurrent, paroxysmal changes in neurologic function (seizures), altered consciousness, or involuntary movements caused by abnormal and spontaneous electrical activity in the brain. Seizures may be convulsive (i.e., accompanied by motor manifestations) or may occur with other changes in neurologic function (i.e., sensory, cognitive and emotional)

Seizure classification

Partial seizures *are limited in* scope (to a part of the cerebral hemisphere) and clinical manifestations involve motor, sensory, autonomic, or psychic abnormalities. Partial seizures are subdivided into *simple, in which consciousness is preserved, and complex, in which consciousness is impaired.*

Generalized **seizures** are more global in scope and manifestations. They begin diffusely, involve both cerebral hemispheres, are associated with alteration in consciousness, and frequently produce abnormal motor activity

Etiology

- Epileptic seizures are **idiopathic** in more than half of all affected patients.
- Vascular (cerebrovascular disease) and developmental abnormalities (cavernous malformation), intracranial neoplasms (gliomas), and head trauma are causative in about 35% of adult cases.
- Other common causes include hypoglycemia, drug withdrawal, infection, and febrile illness (e.g., meningitis, encephalitis).
- Seizures occur with genetic conditions such as Down syndrome,

Seizures sometimes can be evoked by specific stimuli. Approximately 1 of 15 patients reports that seizures occurred after exposure to **flickering lights, monotonous sounds, music, or a loud noise**. Syncope and diminished oxygen supply to the brain also are known to trigger seizures. It is valuable for the dentist to know what factors have the potential to exacerbate a seizure in a particular patient, so that certain stimuli can be avoided.

Complications

Complications of seizures include trauma (as a result of falls) to the head, neck, and mouth and aspiration pneumonia. Also, frequent and severe seizures are associated with altered mental function, dullness, confusion, argumentativeness, and increased risk of sudden death.

Status Epilepticus. A serious acute complication of epilepsy (especially the tonic-clonic type) is the occurrence of repeated seizures over a short time without a recovery period, called status epilepticus

This condition most frequently is caused by abrupt withdrawal of anticonvulsant medication or an abused substance but may be triggered by infection, neoplasm, or trauma. Status epilepticus constitutes a medical emergency. Patients may become seriously hypoxic and acidotic during this event and suffer permanent brain damage or death. Patients with epilepsy also are at increased risk for sudden death and death due to accident

Signs and Symptoms

The clinical manifestations of generalized tonic-clonic convulsions (grand mal seizure) are classic and include :

1- An aura which is a momentary sensory alteration that produces an unusual smell or visual disturbance usually precedes the convulsion..

2- **The tonic phase** which consists of generalized muscle rigidity, pupil dilation, eyes rolling upward or to the side, and loss of consciousness. Breathing may stop because of spasm of respiratory muscles.

3- **The clonic activity** that consists of uncoordinated movements of the limbs and head, forcible jaw closing, and head rocking. Urinary incontinence is common,. The seizure usually does not last longer than 90 seconds .

4- Then, the movement ceases, muscles relax, and a gradual return to consciousness occurs, which is accompanied by headache, confusion, and mental dulling.

Laboratory Findings

The diagnosis of epilepsy generally is based on the history of seizures and presence of abnormalities on the electroencephalogram (EEG). Seizures produce characteristic **spike and sharp** wave patterns on the EEG tracing.

Other diagnostic procedures that are useful for ruling out other causes of seizures include computed tomography (CT), magnetic resonance imaging (MRI), single-photon emission computed tomography (SPECT), lumbar puncture, serum chemistry profiles, and toxicology screening.

MEDICAL MANAGEMENT

The medical management of epilepsy usually is based on long-term drug therapy.

Phenytoin (Dilantin), carbamazepine (Tegretol), and valproic acid (Depakene) are considered first-line treatments.

DENTAL MANAGEMENT

1- **History** : we have to take the history of the disease from the patient or family members and that involve :

Type of seizure

Cause of seizures

Medications

Frequency of physician visits

Frequency of seizures

Date of last seizure

Known precipitating factors

2- If poorly controlled seizures, For these patients, a consultation with the physician is advised before dental treatment. A patient with poorly controlled disease may require additional anticonvulsant or sedative medication, as directed by the physician.

3- if the patient is well controlled then he can receive any routine dental care Taking in consideration the following points :

I- Drug considerations

a- Patients who take anticonvulsants may suffer from Drowsiness, Slow mentation, Dizziness , Gastrointestinal upset, Allergic signs (rash, erythema multiforme)

b- Valproic acid (Depakene) can decrease platelet aggregation , so that Aspirin and non steroidal anti-inflammatory drugs (NSAIBs) should not be administered to patients who are taking valproic acid because they can further decrease platelet aggregation, leading to hemorrhagic episodes .

c- erythromycin should not be administered to patients who are taking carbamazepine .because, of interference with metabolism of carbamazepine, which could lead to toxic levels of the anticonvulsant drug.

d- Because gingival overgrowth is associated with phenytoin administration, every effort should be made to maintain a patient at an optimal level of oral hygiene. This may require frequent visits for oral hygiene measures . If significant gingival overgrowth exists, surgical reduction will be necessary.

II- The dental treatment should be done within a few hours of taking the anticonvulsant medication .

III- using a mouth prop preoperatively . Trying to insert a mouth prop is not advised during the seizure, because doing so may damage the patient's teeth or oral soft tissue and may be nearly an impossible task .

IV- removing dentures .

V- discussing with the patient the urgency of mentioning an aura as soon as it is sensed. The clinician also should be aware that irritability is often a symptom of impending seizure.

VI- If the patient has a seizure while in the dental chair, in this case :

a- you have to protect the patient and try to prevent injury. You should not move the patient to the floor ,also No attempt should be made to restrain the patient, although Passive restraint should be used only to prevent injury that may result when the patient hits nearby objects or falls out of the chair,

b- the chair should be placed in a **supported supine position** ,

c- you have to remove the instruments and instrument tray from the area . and

d- The patient's airway should be maintained patent. And if possible you have to Turn the patient to the side (to avoid aspiration).

e- Seizures generally last after a few minutes. After that, the patient may fall into a deep sleep from which he or she cannot be aroused. So that Oxygen (100%), and a maintenance of a patent airway, and mouth suction should be provided during this phase. Alternatively, the patient can be turned to the side to control the airway and to minimize aspiration of secretions.

f- after seizure examination for sustained injuries (e.g., lacerations, fractures) should be performed. If there is an avulsed or fractured teeth or a fractured appliance, we have to locate the tooth or fragments to exclude aspiration. If we don't found the lost piece then A chest radiograph may be required to locate a missing fragment or tooth.

Oral Complications and Manifestations

a- The most significant oral complication seen in epileptic patients is gingival overgrowth, which is associated with phenytoin (and rarely with valproic acid ,it is mainly occurs in youngsters than in adults. The anterior labial surfaces of the maxillary and mandibular gingivae are most commonly and severely affected.

b- Traumatic injuries such as broken teeth, tongue lacerations, and lip scars also are common in patients who experience generalized tonic-clonic seizures.

C- Stomatitis, erythema multiforme, These complications are more common during the first 8 weeks of treatment.

2- STROKE (CEREBROVASCULAR ACCIDENT)

It is a serious and often fatal neurologic event caused by sudden interruption of oxygenated blood to the brain, leading to a focal necrosis of brain tissue and possibly death. Even if a stroke is not fatal, the survivor often is to some degree debilitated in motor function, speech, or mentation

Etiology

Stroke is caused by the interruption of blood supply and oxygen to the brain as a result of **ischemia or hemorrhage**.

The most common type is **ischemic stroke** induced by thrombosis (in 60% to 80% of cases) of a cerebral vessel. Ischemic stroke also can result from occlusion of a cerebral blood vessel by distant emboli.

Hemorrhage causes about 15% of all strokes and carries a 1-year mortality rate greater than 60%

Cerebrovascular disease is the primary factor associated with stroke. Atherosclerosis and cardiac pathosis (myocardial infarction, atrial fibrillation) increase the risk of thrombolic and embolic strokes, whereas hypertensions the most important risk factor for intracerebral hemorrhagic stroke

complications

The most serious outcome of stroke is death, which occurs in 8% of those who experience ischemic strokes and 38% to 47% of those with hemorrhagic strokes within a month of the event.

If the victim survives, it is highly likely that a neurologic deficit or disability of varying degree and duration will remain.

Signs and sympotoms

A TIA is a “mini” stroke that is caused by a temporary disturbance in blood supply to a localized area of the brain. A TIA often is associated with numbness of the face, arm, or leg on one side of the body (hemiplegia); weakness, tingling, numbness, or speech disturbances that usually last less than 10 minutes most commonly a major stroke is preceded by one or two TIAs within several days of the first attack

Signs of stroke include hemiplegia, temporary loss of speech or trouble in speaking or understanding speech, temporary dimness or loss of vision, particularly in one eye (may be confused with migraine), unexplained dizziness, unsteadiness, or a sudden fall.

Laboratory Findings

Patients suspected of having had a stroke usually undergo a variety of laboratory tests and diagnostic imaging procedures to rule out conditions that can produce neurologic alterations, such as diabetes mellitus, uremia, abscess, tumor, acute alcoholism, drug poisoning, and extradural hemorrhage.

Such investigations often include urinalysis, blood sugar level, complete blood count, erythrocyte sedimentation rate, serologic tests for syphilis, blood cholesterol and lipid levels, chest radiographs, and electrocardiogram (ECG).

lumbar puncture also may be ordered by the physician to check for blood or protein in the cerebrospinal fluid (CSF) and for altered CSF pressure, which would be suggestive of subarachnoid hemorrhage.

EEG, cerebral angiography, CT , and MRI of the brain, are important for determining the extent and location of arterial injury.

Stroke Treatment

a- The immediate task is to save the life during the period immediately after the stroke. This is done by means of life support measures and transport to a hospital.

b- After the initial period, efforts to stabilize the patient and this obtained by the use of anticoagulant medications such as heparin, coumarin, aspirin, and dipyridamole in cases of thrombosis or embolism.

c-Rehabilitatjon generally is accomplished by intense physical, occupational, and speech therapy (if indicated). Although marked improvement is common, many patients are left with some degree of permanent deficit.

N.B. Intake of fruits and vegetables and moderate levels of exercise have a protective effect against stroke.

DENTAL MANAGEMENT

1. Identify the patient who are susceptible to have a stroke, and those involve :

Hypertension

Congestive heart failure

Diabetes mellitus

TIA or previous stroke

Increasing age >75 years

Elevated blood cholesterol or lipid levels

Coronary atherosclerosis

Cigarette smoking

2- if the patient has **a history of** stroke ,you have to Obtain thorough history of stroke including date of event, current status, medical therapy, and any residual disabilities.and manage him as follow :

a- Avoid elective care in patients who have had recent stroke or transient ischemic attack (TIA) or reversible ischemic neurologic deficit (RIND). And you can Provide only urgent dental care during the first 6 months . because those patients are unstable and In fact, up to one third of strokes recur within 1-6 months of the initial event, These individuals therefore should be approached with a degree of caution, and postpone of treatment is advised for 6 months,

b- usually those patients taking anticoagulant drugs, such as:

- Aspirin , dipyridamole, clopidogrel (Plavix), in this case the platelet aggregation is monitored by the platelet function analyzer (PFA)-I00. Abnormal results should be discussed with the physician, also the bleeding time test is useful in this case .
- Coumarin" in this case you have to obtain Pretreatment **INR** . An **INR** level of **3.5** or less is acceptable for performance of most invasive and noninvasive Dental procedures. If the **INR is** greater than **3.5** and oral surgery **is** planned, significant bleeding may occur, and the physician should be consulted for the reduction of the dosage of the anticoagulant.

4-Use measures that minimize hemorrhage (atraumatic surgery, pressure, gelfoam, suturing), as needed.

5-Schedule short, stress-free, midmorning appointments. Provide N20-02 inhalation as needed

6-Monitor blood pressure

7-Use minimum amount of anesthetic containing vasoconstrictor.

8-Avoid epinephrine in retraction cord.

9. patient with stroke may require assistance for patient transfer to the chair, effective oral evacuation (sucker) and airway management.

10 -Oral hygiene measures should be taken in consideration ,and Hygiene is often facilitated by an electric tooth brush, a large-handled toothbrush, or a water irrigation device. Flossing aids should be prescribed, and personal care providers should be instructed on how and when these services should be provided, also Frequent professional prophylaxis and the provision of topical fluoride and chlorhexidine are advisable.

Liver diseases

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The liver plays a vital role in metabolic function:

- Metabolism, breakdown and excretion of drugs and endogenous products (ammonia, bilirubin and sex steroids), most drugs including alcohol are metabolized in the liver.
- Ammonia is converted to urea and excreted by the kidney into the urine.

- Production of important substances such as; albumin, blood clotting factors, transporter proteins, complement, cholesterol and bile.
- Storage of glycogen, fat-soluble vitamins (A, D, B, K and B12), folate and minerals such as; copper and iron.
- Helps maintain homeostasis and blood levels of cholesterol and glucose.

Liver diseases lead to impairment in liver function. Liver diseases are congenital, parenchymal or extrahepatic. The most common liver diseases are:

- Viral hepatitis.
- Alcoholic liver disease.

Viral hepatitis

Most of these infections are caused by hepatitis viruses but other types of viruses can be implicated including; cytomegalovirus (CMV), Epstein-Barr virus, Herpes simplex virus and Coxsackie B virus.

It is the most common form of infectious hepatitis, there are distinctive types of hepatitis viruses; A, B, C, D, E and G, they all target the liver.

Pathophysiology

Hepatitis A and E usually resolve without complications, hepatitis B,

C and D may persist and can replicate in the liver when the virus is not completely eradicated.

The consequences are; recoveiy, persistent infection (carrier state), dual infection, chronic active hepatitis, fulminant hepatitis, cirrhosis, hepatocellular carcinoma and death.

Clinical presentation

Acute viral hepatitis It runs in 3 phases:

- **Prodromal preicteric) phase**; 1-2 weeks before the onset of jaundice, consists of; abdominal pain, anorexia, nausea, vomiting, fatigue, myalgia , malaise and fever.
- **Icteric phase**; it lasts for 2-8 weeks. It is characterized by jaundice, yellow brown cast of the eyes, skin, oral mucosa and urine, gastrointestinal symptoms like anorexia, nausea, vomiting, right upper quadrant pain and hepatosplenomegaly (HSM).
- **Convalescent, recovery or posticteric phase**; the symptoms disappear but HSM and abnormal liver function values may persist. It lasts for weeks or months and the recovery time for hepatitis B and C are longer.

Chronic hepatitis

Carrier state, it is the persistence of low levels of virus in the liver and serum viral antigens for longer than 6 months without signs and symptoms.

It persists for decades and it may progress to chronic active hepatitis.

Chronic active hepatitis

Characterized by active virus replication in the liver, signs and symptoms include; persistent hepatic cellular necrosis, elevated liver enzymes, for longer than 6 months, chronic liver destruction and ᶻ resultant fibrosis leads to cirrhosis. It is more common in patients infected with HCV.

Fulminant hepatitis A serious complication of acute viral hepatitis, there is massive hepatocellulr destruction with 80% mortality rate, it occurs more often in elderly and those with chronic liver disease.

Cirrhosis

Liver cell necrosis and inflammation followed by fibrosis, regeneration and vascular derangement. Liver function deteriorates and blood flow through the organ is obstructed.

It is a common sequel to hepatitis C and alcohol related damage.

Laboratory findings

- **Serum Transaminases** including; Alanine aminotransferase (ALT) and Aspartate aminotransferase (AST), these enzymes are released from **damaged hepatocytes**. ALT is more sensitive indicator of liver damage because it is found only in liver cells. AST is also found in heart, skeletal muscle, pancreas, kidney and RBCs. Normal values 30-40 U/L. -
- **Plasma bilirubin level**; normal value is less than 1 mg/ 100ml. jaundice is evident when bilirubin level approaches 2.5mg/ 100ml.
- **Serum alkaline phosphatase**; it is non-specific test may be normal or slightly elevated. Normal value is 20-140 IU/L.
- WBC count is increased.
- Prothrombin time may be elevated.
- Diagnosis is through serological tests.

Hepatitis A

Hepatitis A which is also called "infectious Hepatitis" is caused by the hepatitis A virus (HAV), which is RNA virus and is **rarely a serious disease.** Hepatitis A is endemic throughout the world, is seen particularly where socioeconomic and living conditions are poor. Spread of hepatitis A is largely feco-oral by consumption of contaminated water or food.

The **incubation** period is 2-6 weeks

No specific treatment is required.

Hepatitis B (Serum Hepatitis)

Hepatitis B is a serious disease caused by the hepatitis B virus (HBV) which is DNA viruse.

This virus is a robust one which can survive for about a week in dried blood on surfaces. It can cause lifelong infection, cirrhosis (scarring) of the liver, liver cancer, liver failure and occasionally fulminant hepatitis and death. Hepatitis B infection is endemic throughout the world with a prevalence of 2-8%.

Spread of hepatitis B is mainly parenteral (via unscreened blood or blood products, particularly by intravenous drug abuse, and by tattooing and body-piercing.

Although pure parotid saliva does not contain **HBsAg**, saliva collected from the oral cavity may contain HBV (presumably derived from serum via gingival exudate) and may be a source for non-parenteral transmission. However, the risk of transmission by this route appears to be low except where there is very close contact, as in families or children's nurseries or sexual contact. HBV can also be transmitted by human bites.

About 25% of needle stick injuries can transmit the infection from infected persons. This is a high risk for oral surgeons who are exposed to such injuries and who can transmit the infection to their patients as well. Postexposure prophylaxis with hepatitis B immunoglobulin (HBIG) and/or hepatitis B vaccine series should be considered for occupational exposures after evaluation of the HBsAg status of the source and the vaccination and vaccine-response status of the exposed person within 24 hours of contact.

HBV infection confers natural immunity.

Clinical features

The incubation period for HBV is 2-6 months, and the acute mortality rate is less than 2%. However, rarely, where there has been co- infection with HDV, the death rate has been as high as 30%.

About 30% of patients have no signs or symptoms. The effects of HBV infection range from subclinical infections without jaundice (anicteric hepatitis) to fulminating hepatitis, acute hepatic failure and death.

The prodromal period of 1-2 weeks is characterized by anorexia, malaise and nausea.

Diagnosis

- **Serum enzyme estimations**: aspartate transaminase (AST) and alanine transaminase (ALT) are raised in proportion to the severity of acute hepatitis. Alkaline phosphatase, alpha-fetoprotein and serum bilirubin levels are also raised.
- **Serological tests**:
  - **Hepatitis B surface antigen (HBsAgJ** also called Australia antigen, hepatitis-associated antigen, and hepatitis B antigen; is a protein found transiently in those with acute hepatitis B, and persists in the serum in carriers and in some who are non- infectious. In a typical case, HBsAg develops 20-100 days after exposure, is detectable in the serum for 1-120 days and then disappears. The serum becomes negative for HBsAg about 6 weeks after the onset of clinical jaundice. Persistence of HBsAg beyond 13 weeks of the clinical illness often implies that a carrier state is developing. **Hepatitis B surface antibody (anti-HBs)** develops after infection or vaccination and, in the absence of HBsAg, implies immunity.

- **Hepatitis B core antigen (HBcAg)** is a sensitive marker of viral replication indicating either current or recent infection.
- **Anti-HBc associated with anti-HBs appears to indicate recovery and immunity to hepatitis**. However, if anti-HBs is absent, **anti-HBc suggests the carrier state or chronic hepatitis.**

- **Hepatitis B envelope antigen (HBeAg);** its presence means ongoing viral activity and **high infectivity**, whereas the presence of **anti-HBe** signifies a more inactive state of the virus and less risk of transmission. If HBeAg persists beyond about 4 weeks of the onset of symptoms, the patient will probably remain infectious and develop chronic liver disease. Absence of HBeAg has usually indicated low infectivity.

In some cases the HBV undergoes a particular structural change, called a **pre-core mutation,** which results in an inability of the HBV to produce HBeAg, even though it is actively reproducing. This means that, even though no HBeAg is detected in the blood, HBV is still active and highly infective. Anti-HBe usually indicates complete recovery and loss of infectivity, provided HBeAg is lost.

Hepatitis C

Formerly known as post-transfusion non-A non-B hepatitis is caused by RNA virus. It is mainly transmitted by transfusion of infected blood and blood products and by intravenous drugs abusers. The virus can also be transmitted among health care workers and patients. At least six distinct genotypes, as well as >50 subtypes within genotypes, of HCV have been identified by nucleotide sequencing but genotype 1 accounts for most of the cases.

Clinical features

Hepatitis C has a similar incubation period to hepatitis B but most HCV-infected persons develop no signs or symptoms. Clinical hepatitis C is also usually a less severe and shorter illness than hepatitis B, but a greater proportion (25-80%) of patients have persisting abnormal liver function tests, up to 85% develop chronic liver disease and some develop liver cancer, or die (< 3%). About 15% of patients infected with HCV are co-infected with hepatitis G virus (HGV). Co-infection with HBV is also common.

Diagnosis

Diagnosis is from the detection of HCV RNA and anti-HCV antibody in the blood, but **anti-HCV IgG** is usually not detectable until 1-3 months after the acute infection.

Medical management

- Prevention through active immunization by vaccines that are inactivated virus vaccines, currently two vaccines are available for HAV and HBV, and they are given in 3 doses over 6 months period. The duration of immunity and the need for booster doses remain controversial. After vaccination, anti-HBs develops and confers 90% protection for many years and possibly for life but may not protect against pre-core variants. Vaccination also protects indirectly against hepatitis D. Hepatitis B vaccine rarely causes any significant adverse effects. Concerns that giving hepatitis B vaccine to infants might contribute to sudden infant death syndrome **(SIDS),** or that the vaccine may trigger autoimmune diseases, especially multiple sclerosis (MS) or lichen planus, proved unfounded.

- Prevention through passive immunization by immunoglobulins collected from human plasma that is free of HBsAg, HCV and HIV. This sterile solution contains antibodies against both HAV and HBV.
- There is yet no vaccine against hepatitis C.
- As any viral infection therapy is palliative and supportive, bed rest, fluids, nutritious high calorie diet is advised. Alcohol and drugs metabolized in liver are avoided.
- Interferon and antiviral drugs are used in HBV and HCV infections.
- Corticosteroids are reserved for fulminant hepatitis.
- Liver transplantation is the last resort.

Dental management

- Identification of potential or actual carriers of HBV, HCV and HDV is very difficult through history.
- All patients with viral hepatitis must be managed as though they are potentially infectious, so standard precautions for infection control should be implemented.
- It is recommended that all dental health care workers should receive vaccination against HBV.

- Patients with active hepatitis:
  - Should be referred for medical treatment, only urgent dental treatment should be provided with strict adherence to standard precautions of infection control and preferably in isolated operatory.
  - Aerosols should be minimized.
  - Drugs that are metabolized in liver should be avoided.
  - If surgery is necessary prothrombin time (PT) and bleeding time should be obtained.

- Patients with history of hepatitis:
  - Since most patients are unaware that they have had hepatitis, identification of carriers is very difficult, and requesting **screening tests** for every patient is not practical.
  - The only method for providing protection is to adopt a strict program of clinical asepsis for all patients.
  - If knowing the type of hepatitis virus through history is not possible, screening test can be ordered for the presence of HBsAg or Anti-HCV.

- Patients at high risk of HBV or HCV infection:
  - Many patients are at high risk of hepatitis B or hepatitis C, like recipients of certain blood products, people with occupational risk, hemodialysis patients, people from areas where HBV infection is endemic, drug addicts and sexually active homosexuals or heterosexuals with multiple partners.
  - Screening is recommended for **HBsAg and Anti-HCV.**
  - Patients who are carriers might have chronic active hepatitis leading to bleeding problems or metabolism problems that require treatment modification.
  - If accidental needle stick occurs, knowing if the patient is HBsAg positive or HCV positive is very important to determine the need for immunoglobulin, vaccine and follow up medical care.

- Patients who are hepatitis carriers:
  - Standard-precautions of infection control should be followed.
  - Such patients may have chronic active hepatitis with compromise liver function. Liver function tests can be ordered and consultation with physician may be needed.

- Patients with signs and symptoms of hepatitis
  - Only emergency dental care should be provided in isolated operatory with adherence to the standard precautions of infection control.
  - Routine dental care should be postponed and the patient referred to the physician.

- Dentists who are hepatitis virus carriers:
  - If the dentist is found to be positive for blood transmissible virus, exposure-prone procedures should not be performed, or strict adherence to aseptic technique should be followed to prevent transmission.
  - Periodic re-testing is necessary.

➢ Exposure to blood:

- In case of percutaneous or permucosal exposure through needle stick or puncture wound contaminated with blood from an individual who is HBsAg, the risk of infection may approach 30%.
- If the exposed is vaccinated; a test to evaluate Anti-HBsAg should be done:
  - ✓If inadequate levels→ HB immunoglobulin + vaccine booster should be administered.
  - ✓If adequate levels→ nothing further is required.
- If the exposed is not vaccinated→ HB immunoglobulin + initiation of vaccination is recommended.
- For HCV; no postexposure protocol or vaccine is available.

- Drug administration:
  - Patients who are completely recovered, no special considerations are needed.
  - In patients with chronic active hepatitis with impaired liver function; drugs that are metabolized in the liver should be avoided if possible or reduced doses used (Aspirin, Acetaminophen, Lidocain, Ibuprofen, Ampicillin, Tetracycline, Metronidazol). A quantity of 3 cartridges of 2% Lidocain (120mg) is considered limited.

Oral manifestations and complications

- Abnormal bleeding; results from abnormal synthesis of clotting factors, inadequate fibrin stabilization, excessive fibrinolysis or thrombocytopenia. Before surgery platelet count, PT and INR should be evaluated, if it is more than 3.5, severe postoperative bleeding may be expected.

- Hepatocellular carcinoma rarely metastasize to the jaws.

- Hepatitis C virus may be associated with oral features sicca syndrome, non-Hodgkin lymphoma and, in some populations with lichen planus.

Alcoholic liver disease

Alcoholism is a chronic addiction to ethanol in which a person craves and uncontrollably consumes ethanol and becomes tolerant to its intoxicating effect. It is the most common drug of abuse.

Alcohol is CNS depressant and it impairs the capacity to reason, it eventually interferes with cerebellar function causing ataxia, motor incoordination and unconsciousness.

Alcohol is hepatotoxicis and its metabolite acetylaldehyde is fibrinogenic, 10-15% of heavy alcohol users develop cirrhosis.

It is an increasing cause of hepatitis in developed countries. It injures the liver by blocking the metabolism of protein, fats and carbohydrates, and causes fatty change (alcoholic steatosis); in response there is inflammation (alcoholic hepatitis), which is probably initially reversible. The amount of alcohol that can injure the liver varies greatly from person to person.

Clinical presentation

The pathological effects of alcohol on the liver are expressed by 3 disease entities:

Fatty infiltrate; in which hepatocytes are engorged with fatty lobules and distended, it is considered reversible. No visible manifestations are present except for enlargement.

Alcoholic hepatitis; diffuse inflammation of liver with destructive cellular change, it is reversible. Clinically; nausea, vomiting, anorexia, malaise, weight loss, fever, HSM, jaundice, ascites, ankle edema and spider angioma.

Cirrhosis; the clinical manifestations are apparent onty after sufficient destruction of liver parenchyma.

Alcoholic liver disease is classified into mild, moderate and severe based on prognostic formula, the parameters used are; the presence of ascites, encephalopathy, serum bilirubin value, serum albumin value and increased PT in seconds.

Laboratory findings

- Increased bilirubin, AST, ALT, gamma-glutamyl transpeptidase, amylase, uric acid, triglycerides and cholesterol.
- Leukopenia (or Leukocytosis) and anemia.
- Thrombocytopenia (due to splenomegaly).
- Increased prothrombin time (PT), partial thromboplastin time (PTT) and thrombin time.

Medical management

- Identification through physical examination to evaluate impaired organ systems.
- Gradual withdrawal from alcohol, abrupt withdrawal leads to loss of appetite, tachycardia, anxiety, insomnia, hallucinations, disorientation, impaired memory and attention and agitation.
- High protein, high calorie and low sodium diet and vitamin supplementation.
- Management of CNS depression caused by withdrawal.
- Education of the patient about alcoholism.
- Management of complications.
- End stage cirrhosis requires liver transplantation.

Dental management

- Detection of such patients by:
  - History; the patient should be asked about the type, quantity, frequency, pattern and consequences of alcohol use, also family history of alcoholism.
  - Clinical examination for signs and symptoms of alcoholic liver disease.
  - Alcohol odor on breath.
  - Information from family members or friends.
- Guiding the patient for assessment or treatment and supportive care.
- minimizing relapse by avoiding psychoactive drugs, narcotics, sedatives and alcohol containing medications in patients recovering from alcoholism.

- In patients with history of alcohol liver disease or alcohol abuse, a physician should be consulted to verify the patient's current status, medications, laboratory values (if present) and contraindications for medications and surgery.
- If signs and symptoms of alcoholic liver disease are present the dentist can request some screening tests before surgical procedures; complete blood count (CBC) with differential, AST, ALT, platelet count,-thrombin time, PT and INR. Abnormal results should be discussed with the physician.
- Treatment considerations; 3 major dental treatment considerations apply for patients with alcoholic liver disease:
- Bleeding tendencies; can be managed with the assistance of physician, this may entail the use of local hemostatic agents like fresh frozen plasma, vitamin K, platelets and antifibrinolytic agents.

- The unpredictable metabolism of drugs; in mild to moderate liver disease, enzymatic induction is likely to have occurred leading to increased tolerance to LA, sedatives, hypnotics and GA, thus larger doses may be needed to attain the desirable effects of these drugs.

Avoiding drugs that are metabolized in the liver or using half dose may be considered in patients with cirrhosis or alcoholic hepatitis particularly if aminotransferases level (AST and ALT) is 4 times more than normal, serum bilirubin is more than 2mg/dl, serum albumin lower than 35g/L, with signs of ascites and encephalopathy or malnutrition.

- The risk of infection or spread of infection; because these patients have reduced reticuloendothelial capacity and altered cell mediated immune function, but antibiotic prophylaxis is not needed unless there is an ongoing infection. Consultation with the physician regarding the use of antibiotic may be considered especially in patients with moderate or severe liver disease.

Oral complications and manifestations

- Poor oral hygiene and caries which is due to neglect.
- Impaired gustatory function.
- Nutritional deficiency can result in anemia causing glossitis, loss of papillae, angular and labial cheilitis.
- Bleeding tendencies cause spontaneous gingival bleeding, mucosal ecchymoses and petechiae.
- Alcohol breath odor.
- Jaundiced mucosal tissues.
- Sialoadenosis; bilateral painless swelling of the parotid glands, it is attributed to demyelinating polyneuropathy that results in abnormal sympathetic signaling, abnormal acinar protein secretion and acinar cytoplasmic swelling.
- Oral squamous cell carcinoma, alcohol abuse and smoking are major predisposing factors.
- Bruxism and dental attrition.
- Xerostomia.

Liver Cancer

Cancer of the hepatocytes is termed hepatocellular carcinoma or malignant hepatoma. It is common in areas of high endemicity for HBV and HCV, but it also arises as metastatic cancer to the liver from the-colon, lungs, breasts, uterus or other parts of the body.

Risk factors for hepatocellular carcinoma include male gender, old age, a positive family history, chronic HCV or HBV, oral contraceptive use and cirrhosis.

Liver cancer in the early stages is asymptomatic but later, manifestations may include wasting, jaundice, pain or swelling in the abdomen, anorexia and fever.

Treatment usually consists of surgical resection (partial hepatectomy) but the prognosis is invariably poor. Liver transplantation may be required.

Dental management is similar to that applied in severe liver disease

Liver Transplantation

It is provided for treatment of end-stage liver disease such as from biliary atresia, metabolic disease, cirrhosis or malignancy.

The 1-year survival of liver transplantation is around 80% and, of survivors, 90% survive 5 years and 85% for 10 years. Graft-versus-host disease can follow liver transplantation.

All liver transplant recipients require lifelong immunosuppression to prevent a T-cell, alloimmune rejection response.

Liver transplant recipients may be susceptible to recurrence of their original diseases.

Dental management

The main concerns are bleeding tendencies and impaired drug metabolism.

Oral Manifestations

- Children needing liver transplants may have retarded tooth eruption and discolored and hypoplastic teeth.
- Gingival swelling may be seen in patients on ciclosporin or some other drugs.


Thank
you!

Dental Management of Patients with Allergy

Allergy is a disease condition, which results from an immunologic reaction to a noninfectious foreign substance known as an antigen or an allergen. Under certain circumstances, the repeated contact with the antigen may cause an inappropriate response (hypersensitivity) that can be harmful or destructive to the host's tissues.

Allergies are actually a series of repeat reactions to the foreign substance. The reactions involve different types of immunologic hypersensitivity, and involve elements of the nonspecific and the specific immune systems

Elements of The Immune System

1- <u>Nonspecific,</u> these include :

a- Mechanical reflexes, like coughing and sneezing, action of cilia, sphincters control.

b- Secretion of bactericidal substances, like stomach acid, ear wax, enzymes in tears and saliva.

c- Phagocytic cells, like neutrophils, monocytes, macrophages, d- Circulating chemicals, like complement, interferon.

2- Specific

a- Humoral immune system

Stem cells originating in the bone marrow differentiate to form two main lymphocyte populations. The first population, the cells of which undergo processing by the lymphoid tissue of the gut before becoming the immunologically competent B lymphocytes. B lymphocytes are the key cells in the humoral immune system, they recognize specific foreign chemical configurations via receptors on their cell membrane. Each clone (family) of B lymphocytes is primed to recognize it's own specific chemical configuration. For the antigen to be recognized by the specific B lymphocytes, it must be first processed by T helper lymphocytes land macrophages.

Once recognition has taken place, the B lymphocytes differentiate and multiply, forming the short lived plasma cells and long lived B memory lymphocytes. The memory B lymphocytes remain inactive until contact with the specific antigen occurs, this contact will transform the memory cells into plasma cells, which produce the antibodies (immunoglobulins) specific for the antigen involved.

Five classes of immunoglobulins are produced by plasama cells, these are IgG,

IgM , IgA, IgE, IgD, they differ in their chemical structure as well as their biological properties.

It is believed that the population of antibody-producing cells arise by random mutation, and that a single cell will then react to produce a clone of cells with specific function, this random mutation of lymphocytes allows the genesis of cells that are capable of responding to different antigens, although a single cell can react to only a single antigen.

Type I, type II, and type III hypersensitivity reactions are mediated with elements of the humoral immune system.

b- Cellular immune system

The cellular or the delayed immune system has the second lymphocyte population (T lymphocytes) playing the central role.

The primary function of this system is to recognize and eradicate antigens that are fixed in tissues or within cells, thus this system is involved in the protection against viruses, tuberculosis, brucellosis, syphilis.

T lymphocytes are dependent on a factor produced by the epithelial cells of the thymus for becoming immunologically competent from their non competent predecessors originating in the bone marrow.

T lymphocytes have no detectable antibody on their surface but have antibody-like receptors for antigens, the exact nature of which is still not clear. T lymphocytes

sensitised to a specific antigen will undergo blast transformation and proliferation on contact with the antigen, this interaction of the antigen with the T lymphocytes will also lead to the release of non specific factors, cytokines, which bring about a number of tissue changes associated with cell mediated hypersensitivity reactions.

It is likely that proliferation stimulation in some T lymphocytes involves processing the antigen by macrophages, dendritic and langerhan cells, possibly leading to presenting the antigen determinants at the surface of the macrophage

Cytokines produced by effector T lymphocytes include :

- Macrophage inhibition factor
- Macrophage activating factor
- B lymphocytes growth factor
- B lymphocytes differentiation factor
- Interferons
- interleukin-2

N.B. Type IV hypersensitivity reactions are mediated by elements of the cellular immune response.

Types of Immune Reactions

Type I - anaphylactic f immediate hypersensitivity) reactions

Type I hypersensitivity reactions are related to the humoral immune system. Previous usage of the term anaphylaxis emphasized the clinically severe form, anaphylactic shock, but current usage refers to the underlying mechanism and not clinical severity.

In man only IgE antibodies are able to produce anaphylactic reactions. As IgE antibodies adhere strongly to tissues, particularly to mast cells in tissues, anaphylactic reactions are caused by antigen-antibody reactions on the surfaces of mast cells, activating a series of enzymes leading to the release of vasodilators from the mast cells. These are histamine, serotonin and plasma kinins, bradykinin and kallidin. They cause vasodilatation, increased capillary permeability, polymorphonuclear leucocytes migration, bronchospasm.

Anaphylactic reactions usually occur soon after the second contact with an antigen,however, individuals may have repeated contacts with the antigen before finally becoming allergic to it. Anaphylactic reactions depend on whether the port of entry of the antigen is local, systemic or via the intestine. Relatively mild symptoms occur when antigen-antibody reactions take place on an exposed mucosal surface, the symptoms may be limited to rhinorrhoea and conjunctivitis, however, intense bronchospasm may be produced as a result of inhalation of the antigen by asthmatics whose disease condition is mediated via this type of hypersensitivity

reactions.

Systemic anaphylaxis on the other hand consists of a group of much more severe reactions, which may occur rapidly if the antigen is injected parenterally, as in the case of injecting drugs or foreign sera. The clinical features are bronchospasm, laryngeal edema resulting in extreme dyspnoea and cyanosis, marked fall in blood pressure (anaphylactic shock), there may also be nausea, vomiting, and diarrhoea.

Atopy is an example of this type of hypersensitivity reactions. Atopy is a hypersensitivity state influenced by hereditary factors.

Examples of atopic conditions are :

- **Asthma**
- **Urticaria**, an anaphylactic condition, which can develop as a result of absorption of antigen through the intestinal tract. It results in the formation of weal and flare lesions in the superficial skin. Common antigens are present in food (like strawberries, eggs, shell fish), drugs. Urticaria often occurs alone, but may He associated with other signs of anaphylaxis. Urticaria can result from non-allef^c reactions. Angioneurotic edema, which is a lesion occurring in the deeper layers of ^ki in other tissues like tongue or larynx.
- **Hay fever**

Type II- cytotoxic (hypersentivity) reactions

These are IgG or IgM- mediated. Here antigens on cell's surface combines with antibody and complement to destroy the cell. The clearest example of type II cytotoxic reaction is transfusion reaction resulting from mismatched blood.

Type III- immune complex *(* hypersensitivity) reactions

These are induced by deposition of antigen and antibody in tissues causing activation of complement, this results in a polymorphonuclear inflammatory response and also damage to cell membrane of adjacent tissues. Hydrolytic enzymes released from the granules of leucocytes also contribute to the vascular damage which is the hallmark of this type of immune reactions. Clinical examples include streptococcal glomerulonephritis, rheumatoid arthritis, polyarteritis, lepromatous leprosy.

Type IV- cell mediated (hypersensitivity reactions)

These reactions involve the cellular immune system, free antibodies play no part in them. They are usually delayed and will appear 48-72 hours after contact with the antigen. They include contact dermatitis, transplant rejection, infections, and graft versus host disease.

Contact dermatitis occurs when a substance of low molecular weight, that is not allergic by itself (a hapten), comes in contact with a tissue component, primarily a protein, and forms an antigenic complex which will cause sensitization of T lymphocytes.

Homograft rejection usually occurs unless the donor and recipient are genetically identical or the immune response is suppressed. Tuberculin skin testing is an example of infectious-type IV hypersensitivity reaction.

Persons previously exposed to Mycobacterium tuberculosis will, with a second exposure in the form of subcutaneous injection of altered bacteria, develop a delayed response in the form of induration ,erythema, swelling and even ulceration at injection site, take place 48-72 hours afterwards.

ALLERGY TO LOCAL ANESTHETICS

Fortunately true allergic reactions to local anesthetics are rare, and many of reactions misdiagnosed as allergic are either psychomotor or toxic. The signs and symptoms of a **psychomotor response to a local anesthetic are :**

- Hyperventilation
- Vasovagal syncope
- Bradycardia
- Pallor
- Sweating
- Hypotension
- Sympathetic stimulation
  - ✔ Anxiety
  - ✔ Tremor
  - ✔ Tachycardia
  - ✔ Hypertension

The most common adverse reaction to local anesthetics is a toxic reaction, resulting from the inadvertent I.V. injection of the anesthetic solution. The presenting signs and symptoms of **a toxic reaction to a local anesthetic are** :

- Talkativeness
- Slurred speech
- Dizziness
- Nausea
- Depression
- Euphoria
- Excitement
- Convulsions

Excessive amounts of an anesthetic can also cause a toxic reaction or a **reaction to the vasoconstrictor**, the signs and symptoms of later include :

- Tachycardia
- Apprehension
- Hyperactivity
- Sweating

Procaine is the local anesthetic with the highest incidence of allergic reactions. It's antigenic component appears to be paraaminobenzoic acid, one of the breakdown products of procaine.

Cross reactivity between paraaminobenzoic acid esters-local anesthetics are common, whereas paraaminobenzoic acid amides-local anesthetics do not cross react with each other. Amides and esters of paraaminobenzoic acid usually do not cross react,however, cross reactions between procaine and lidocaine-containing local anesthetic solutions have been reported, later they were linked to methylparaben, a ' germicide that has a structure similar to that of paraaminobenzoic acid, added to the local anesthetic solution. Methylparaben-free local anesthetic solutions are available now.

Sulfites (sodium metabisulfite, acetone sodium bisulfite), antioxidants added to local anesthetic solutions to protect the vasoconstrictor can cause serious allergic reaction particularly in asthmatic patients.

Dental management of patients with history of allergy to local anesthetics

- Establish a detailed history of the previous reaction that followed the use of the local anesthetic solution.
- Try to determine the nature of the previous reaction (psychomotor, toxic, vasoconstrictor, or allergic) and the type of the local anesthetic used.
- Common examples of reactions mislabeled as “allergy” are syncope after injection of a local anesthetic and nausea or vomiting after ingestion of codeine.

- If the reaction was consistent with an allergic reaction, meaning, the presenting signs and symptoms of the previous reaction consisted of one or more of the followings :
  - Skin rash
  - Urticaria
  - Swelling
  - Chest tightness
  - Dyspnea
  - Rhinorrhea
  - Conjunctivitis

a- Select an anesthetic from a different chemical group Paraaminobenzoic acid esters (procaine, tetracaine)

Paraaminobenzoic acid amides (lidocaine, prilocaine, mepivacaine, bupivacaine, articaine).

b- Aspirate, inject a drop of the alternative anesthetic, and wait for 5 minutes. If no reaction occurs, inject after aspirating the rest of the anesthetic needed. Be prepared to deal with an allergic reaction, if one should occur.

c- In cases of allergic reaction to different local anesthetic agents or if the anesthetic agent previously used cannot be identified, a 1% diphenhydramine with 1 : 100000 adrenaline solution may be used. This solution induces anesthesia of average duration of 30 minutes following infiltration or nerve block injections. 50 mg, is the maximum dose of diphenhydramine that can safely be given in as ingle appointmemt.

d- A second option if history of multiple allergies is present or if the type of local anesthetic that caused the previous reaction cannot be identified, refer the patient to an allergist for evaluation and provocative dose testing.

The use of diphenhydramine often is the more practical option. A 1% solution of diphenhydramine that contains 1 : 100,000 epinephrine can be easily compounded by a pharmacist, but it must be confirmed that methylparaben is not used as a preservative. This solution induces anesthesia of about 30 minutes average duration and can be used for infiltration or block injection. When it is used for a mandibular block, 1 to 4 mL of solution is needed. Some patients have reported a burning sensation, swelling, or erythema after a mandibular block with 1% diphenhydramine, but these effects were not serious and cleared within 1 or 2 days. No more than 50 mg of diphenhydramine should be given during a single appointment. Diphenhydramine also can be used in the patient who reports a previous allergic reaction to either an ester or amide local anesthetic

Management of patients with history of allergy to penicillin

Allergic reactions occur in 5%-10% of patients treated with penicillin or related drugs. About 0.04%-o.2% of patients treated with penicillin develop anaphylactic reactions, and about 10% of those die.

Hypersensitivity reactions to penicillin in order of decreasing frequency are :

- Maculopapular rash (type IV)
- Urticarial rash (type I)
- Fever (type III)
- Bronchospasm (type I)
- Vasculitis (type III)
- Serum sickness (type III)
- Exfoliative dermatitis (type IV)
- Anaphylaxis (type I)
- Interstitial nephritis (type II)

The route of administration of penicillin may affect the degree of severity of the allergic reaction. Oral administration of the drug produces less sever reactions than those which follow parenteral administration. Cross reactivity between penicillins is common.

- Patients with a history of penicillin allergy should be given erythromycin, or clindamycin for the treatment of oral infections or prophylaxis against infective endocarditis.

- Skin testing for allergy to penicillin is much more reliable than that for allergy to local anesthetics, however the procedure is not without risk. To be costeffective, the test should be conducted only on patients with a history of penicillin allergy who need penicillin for a serious infection. When testing for penicillin allergy is performed, both metabolic breakdown products of penicillin, the major derivative (penicilloyl polylysine), and the minor derivative mixture (MDM) must be testpd. If skin tests for both breakdown products are negative, the patient is considered not allergic to penicillin.

Cephalosporins will cross react in 5%-10% of penicillin sensitive patients. The risk is greatest with 1st or 2nd generation cephalosporins. Cephalosporins can be used in patients with a history of distant nonserious reaction to penicillin. Skin testing for penicillin sensitivity for these patients is recommended, and if it is negative, either penicillin or a cephalosporin may be used. If the skin test is positive, a skin test for the cephalosporin chosen is indicated.

Allergy to analgesics

Individuals, estimated at 0.2% are allergic to salicylates Allergic reactions to aspirin can be serious, and deaths have been reported. Aspirin will provoke a severe reaction in some of the patients who have asthma, who may also react in the same way to other nonsteroidal anti-inflammatory drugs.

Allergy to rubber products

Dentists should be aware that latex allergy can manifest as anaphylaxis during dental work when the patient or the dentist has been sensitized to latex. Anaphylaxis may occur in the sensitized person after contact has been made with rubber gloves, rubber dam material, bloodpressure cuffs, or any other product containing latex. Studies have shown that latex-allergic persons have IgE antibodies for specific latex proteins. Latex skin tests are a satisfactory means of identifying individuals who may be sensitized to latex. Nitrile gloves should be considered for use to minimize these adverse reactions to latex proteins

Management of severe type I hypersensitivity reactions

Type I hypersensitivity reactions usually occur soon after (minutes) the injection, ingestion, or the application of a topical anesthetic, medication, drug, or local anesthetic. Most of these reactions are mild and do not constitute an emergency, however, some are severe and may be life threatening (anaphylactic).

An anaphylactic reaction usually takes place within minutes. The signs and symptoms associated with anaphylactic reactions include itching of the soft palate, , nausea, vomiting, substernal pressure, shortness of breath, hypotension, pruritis, urticaria, laryngeal edema, bronchospasm, cardiac arrythmias. Both respiratory and circulatory depression occur early in the course of the reaction which could be fatal.

Control measures consist of the following steps :

- Have someone in the office call for medical aid.
- Place the patient in supine position.
- Make certain the airway is patent.
- Administer oxygen.
- Check the carotid or femoral pulse and respiration; if no pulse is present and the respiration is depressed:

  a- Inject 0.5 ml of 1:1000 adrenaline I.M. (into the tongue) or S.C.

  b- Support circulation by closed chest cardiac massage.

  c- Support respiration by mouth to mouth breathing or bag and mask,

  d- Repeat the injection of adrenaline if no response occurs.

N.B. Note: Intramuscular injection of epinephrine into the thigh has been reported to provide higher plasma concentrations than those administered into the arm.


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AIDS, HIV Infection, and Related Conditions

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AIDS is an infectious disease that is transmitted predominantly through intimate sexual contact and by parenteral means. In view of the nature of this bloodborne pathogen, HIV infection and AIDS have important implications for dental practitioners. Although HIV has rarely been transmitted from patients to health care workers, this may occur, and the patient with HIV infection or AIDS may be medically compromised and may need special dental management considerations.

Pathophysiology and Complications

- Transmission of HIV is by exchange of infected bodily fluids from sexual contact and through blood and blood products.

- The virus is found in blood, seminal fluid, vaginal secretions, tears, breast milk, cerebrospinal fluid, amniotic fluid, and urine. Blood, semen, breast milk, and vaginal secretions are the main fluids that have been shown to be associated with transmission of the virus.

- Vertical transmission to infants born of infected mothers can occur at birth or transplacentally.
- HIV has been found in saliva, and transmission by transfer of saliva possibly contaminated with blood has been reported from providing premasticated food from HIV infected parents to infants.
- Casual contact has not been demonstrated as a means of transmission. Inflammation and breaks in the skin or mucosa (e.g., presence of other sexually transmitted diseases) and high concentrations of HIV in bodily fluids increase the risk of transmission.

Once HIV has gained access to the bloodstream, the virus selectively seeks out T lymphocytes (specifically T4 or T helper lymphocytes) Upon infection, reverse transcriptase catalyzes the synthesis of a haploid, double-stranded DNA provirus, which becomes incorporated into the chromosomal DNA of the host cell. Thus integrated, the provirus genetic material may remain latent in an unexpressed form until events occur that activate it, at which time DNA transcription rapidly occurs and new virions are produced.

The virus is lymphotropic; hence, the cells it selects for replication are soon destroyed. Once the virus takes hold, it causes a reduction in the total number of T helper cells, and a marked shift in the ratio of CD4+ to CD8+ lymphocytes occurs. The normal ratio of T helper to T suppressor lymphocytes is about 2 : 1 (60% T helper, 30% T suppressor). In AIDS, the T4/T8 ratio is reversed.

CLINICAL PRESENTATION

Signs and Symptoms

During the first 2 to 6 weeks after initial infection with HIV, more than 50% of patients develop an acute flulike syndrome marked by viremia that may last 10 to 14 days. Others may not manifest this symptom complex. Symtomatic persons often develop lymphadenopathy, fever, pharyngitis and a skin rash but generally do not display circulation antibodies until the 6^{th} week to 6^{th} month.

stage 1 generally begins immediately after HIV exposure and may last for years. Affected persons are HIV antibody–positive but are asymptomatic and show no other laboratory abnormalities.

Stage 2 is characterized by progressive immunosuppression and symptomatic disease. Patients who demonstrate various laboratory changes (i.e., lymphopenia: T helper/T suppressor ratio usually less than 1) in addition to HIV antibody positivity also may show clinical signs or symptoms, such as enlarged lymph nodes, night sweats, weight loss, oral candidiasis, fever, malaise, and diarrhea.

Persons in **stage 3** have AIDS and can demonstrate a variety of immunesuppression-related diseases. Opportunistic infections predominate as the CD4+ T count approximates 200 cells/µL; then malignancies, wasting syndrome, and a progressive form of dementia can develop. Patients may become confused and disoriented or may experience short-term memory deficits. Others develop severe depression or paranoia and show suicidal tendencies.

Laboratory Findings

Most patients exposed to the virus, with or without clinical evidence of disease, show antibodies to the virus by the 6th month of infection. Patients with advanced HIV infection or AIDS have an altered ratio of CD4+/CD8+ lymphocytes, a decrease in total number of lymphocytes, thrombocytopenia, anemia, a slight alteration in the humoral antibody system, and a decreased ability to show delayed allergic reactions to skin testing (cutaneous anergy). CD4+ and CD8+ cell counts should be performed at the time of HIV diagnosis and then every 3 to 4 months.

The enzyme-linked immunosorbent assay (ELISA) is the screening test for identification of antibodies to HIV. It is 90% sensitive but has a high rate of false-positive results. Current practice is to screen first with ELISA. If the results are positive, a second ELISA is performed. All positive results are then confirmed with Western blot analysis.

This combination of tests is accurate more than 99% of the time. Positive ELISA and Western blot test results indicate only that the individual has been exposed to the AIDS virus. If results of the Western blot are indeterminate, HIV infection is rarely, if ever, present.

Nucleic acid amplification using polymerase chain reaction (PCR)-based assays of the viral RNA is performed to determine the viral load in the blood (i.e., degree of viremia) and monitor response to therapy.

MEDICAL MANAGEMENT

Antiretroviral therapy (ART) should be used in a manner that will achieve viral suppression and immune reconstitution while at the same time preventing emergence of resistance and limiting drug toxicity

Both ART and HAART involve use of combinations of antiretroviral drugs; however strictly speaking, HAART is defined as the use of at least three active antiretroviral medications.

Chemoprophylaxis:

Chemoprophylaxis regimens are recommended when CD4+ lymphocyte counts drop to specific levels to prevent initial episode of a disease or to suppress a developing opportunistic infection. These regimens exist for the prevention of *Pneumocystis pneumonia,* tuberculosis, toxoplasmosis, and other opportunistic diseases.

DENTAL MANAGEMENT

- History and clinical findings may indicate that the patient has HIV infection/AIDS. Of note, however, patients who know they are sero-positive and those at high risk for these conditions may not answer questions honestly, on account of the stigma or concern for privacy. Accordingly, the patient history should be obtained whenever possible with this understanding, verbal communication in a quiet, private location, and the sharing of knowledge and facts in an atmosphere of honesty and openness.

- Patients who, on the basis of history or clinical findings, are found to be at high risk for AIDS or related conditions should be referred for HIV testing, and medical evaluation.

Treatment Planning Considerations

- Patients who have been exposed to the AIDS virus and are HIV sero-positive but asymptomatic may receive all indicated dental treatment. Generally, this is true for patients with a CD4+ cell count of more than 350/µL. Patients who are symptomatic for the early stages of AIDS (i.e., CD4+ cell count lower than 200) have increased susceptibility to opportunistic infection and may be medicated with prophylactic drugs

- The patient with AIDS can receive almost any dental care needed and desired once the possibility of significant immunosuppression, neutropenia, or thrombocytopenia has been ruled out.
- White blood cell and differential counts, as well as a platelet count, should be ordered before any surgical procedure is undertaken. Patients with severe thrombocytopenia may require special measures (platelet replacement) before surgical procedures (including scaling and curettage) are performed. Medical consultation should precede any dental treatment for patients with these abnormalities.
- Patients may be medicated with drugs that are prophylactic for *Pneumocystis pneumonia, candidiasis,* herpes simplex virus (HSV) or CMV infection, or other opportunistic disease, and these medications must be carefully considered in dental treatment planning

- Most often, consultation with the patient's physician is beneficial. For example, **acetaminophen** should be used with caution in patients treated with zidovudine (Retrovir) because studies have suggested that granulocytopenia and anemia, associated with zidovudine, may be intensified, also, **aspirin** should not be given to patients with thrombocytopenia. **Propoxyphene** levels may be increased by ritonavir which may potentially lead to toxic effects such as drowsiness, slurred speech, or incoordination. **Antacids, phenytoin, cimetidine, and rifampin** should not be given to patients who are being treated with ketoconazole, because of the possibility of altered absorption and metabolism. Also, **midazolam and triazolam** should be avoided in patients taking select protease inhibitors, because benzodiazepine metabolism may be inhibited, leading to excessive sedation and/or respiratory depression.

- Any source of oral or dental infection should be eliminated in HIV-infected patients, who often require more frequent recall appointments for maintenance of periodontal health. Daily use of chlorhexidine mouth rinse may be helpful.

Occupational Exposure to HIV

The risk of HIV transmission from infected patients to health care workers is very low, reportedly about 3 of every 1000 cases (0.3%) In comparison, the risk of infection from a needlestick is 3% for hepatitis C and is 30% for hepatitis B.

After a needle stick, the rate of transmission of HIV can be reduced by post exposure prophylaxis (PEP). The CDC recommends PEP as soon as possible after exposure to HIV-infected blood. The number of PEP drugs recommended is based on the severity of the exposure as well as the HIV status of the source patient. A *less severe exposure (solid needle or superficial* injury) from a source-patient who is asymptomatic or has a low viral load (<1500 viral copies/mL) has a two-drug PEP. Use of at least a three-drug PEP regimen is recommended for *more severe exposure (large-bore* hollow needle, deep puncture, visible blood on device or needle used in patient's artery or vein) or when the patient is symptomatic, has AIDS, or a high viral load. The recommended *basic regimen for HIV PEP is* ***tenofovir*** plus ***emtricitabine*** or ***zidovudine*** plus ***lamivudine***.

Tests for seroconversion should be performed at 3, 6, and 12 months. To date, there have been six reports of occupational HIV sero-conversion despite combination PEP. If the exposed dental health care worker is pregnant, risk of infection versus unknown yet possible risks of PEP to the fetus, should be discussed.

Transmission of the virus appeared to occur through contaminated dental instruments. The risk of transmission in the dental setting is minimized by adherence to standard infection control procedures.

TABLE 18-4 Head, Neck, and Oral Lesions Commonly Associated with HIV Infection and AIDS

Oral Condition	Comment	Treatment
Persistent generalized lymphadenopathy	An early sign of HIV infection, found in about 70% of infected patients during the latent stage of infection. Must be present longer than 3 months and in two or more extrainguinal locations. Anterior and posterior cervical, submandibular, occipital, and axillary nodes are most frequently involved.	Usually not treated directly, may need biopsy to rule out lymphoma or other conditions.
Oral candidiasis Pseudomembranous Erythematous Hyperplastic Angular cheilitis	Most common intraoral manifestation of HIV infection. First found during the early symptomatic stage of infection. This indicates that AIDS will develop within 2 years in untreated patients. About 90% of patients with AIDS will develop oral candidiasis at some time during their disease course.	Nystatin often is ineffective. Topical clotrimazole is effective but has high rate of recurrence. Systemic fluconazole and itraconazole are effective but have a number of drug interactions and may result in drug-resistant candidiasis. If azoles fail, then intravenous amphotericin B can be administered.

HIV-associated periodontal disease		
Linear gingival erythema (LGE)	LGE does not respond to improved plaque control procedures. Condition is associated with candidiasis.	LGE usually responds to plaque removal, improved oral hygiene, and chlorhexidine rinses. Persistent cases usually respond to local measures plus systemic antifungal medications. Therapy for NUG, NUP, and NS involves debridement (removal of necrotic tissue and povidone-iodine irrigation), chlorhexidine rinses, metronidazole, follow-up care, and long-term maintenance.
Necrotizing ulcerative gingivitis (NUG)[16]	NUG relates to ulceration and necrosis of one or more interdental papillae with no loss of periodontal attachment.	
Necrotizing ulcerative periodontitis (NUP)	NUP consists of gingival ulceration and necrosis with attachment loss and does not respond to conventional periodontal therapy.	
Necrotizing stomatitis (NS)	May be seen as an extension of NUP or may involve oral mucosa separate from the gingiva.	

Oral Condition	Comment	Treatment
Herpes simplex virus (HSV) infection	Immunocompetent persons and HIV-infected patients experience about the same rate of recurrent HSV infection (10-15%), but in HIV-infected patients, the lesions are more widespread, occur in an atypical pattern, and may persist for months.	Systemic acyclovir, valacyclovir, or famciclovir for at least 5 days can be effective. Higher doses may be needed during severe immunosuppression. An elixir or syrup of diphenhydramine (Benadryl) of 12.5 mg/5 mL can be used for pain control.
Varicella-zoster virus (VZV) infection	Recurrent VZV infection is common in HIV-infected patients, but the course is more severe. Intraoral lesions are often severe and can lead to bone involvement with loss of teeth.	Valacyclovir 1 g PO tid; famciclovir 500 mg PO tid; acyclovir 800 mg PO 5 times per day. Intravenous acyclovir may be needed for severe herpes zoster in patients with immunosuppression.
Oral hairy leukoplakia (OHL)	White lesion most often found on the lateral border of the tongue. OHL on rare occasions has been found on the buccal mucosa, soft palate, and pharynx. Associated with Epstein-Barr virus infection. In an untreated patient with HIV symptomatic infection, the finding of OHL indicates that AIDS will develop in the near future.	Treatment often is not needed. Acyclovir or desiclovir can result in rapid resolution, but recurrence is likely. Retinoids or podophyllum resin therapy can lead to temporary remission. HIV therapy with ART can result in significant regression.
Kaposi sarcoma (KS)	Human herpes virus type 8 (HHV-8) involved in KS development. About 50% of patients with KS have oral lesions, and the oral cavity is the initial site of involvement in 20% to 25% of cases. The most common sites are the hard palate, gingival, and tongue. KS that occurs in an HIV-infected patient is diagnostic of AIDS.	Often regresses with HAART. Treatment involves irradiation, local and systemic chemotherapy. Focal symptomatic lesions can be excised, or injected with vinblastine or a sclerosing agent (sodium tetradecyl sulfate). Other options for dealing with these types of lesions are cryotherapy, laser ablation, and electrosurgery, but care must be taken to protect operating personnel from aerosolization of viral particles when the laser or electrosurgery unit is used.

TABLE 18-5 Less Common Oral Conditions Associated with HIV Infection

Oral Condition	Comment	Treatment
Aphthous stomatitis Minor Major Herpetiform	About 66% of lesions are of the more uncommon forms—major and herpetiform. With more severe reduction of CD4+ cell count, major lesions become more prevalent. Lesions that are chronic or atypical, or that do not respond to treatment, should be biopsied.	Treatment of major lesions that persist involves potent topical or intralesional corticosteroids. Systemic steroids generally are avoided, to prevent further immunosuppression. Thalidomide treatment has yielded good response but should be used for only a short time, because the drug can enhance HIV replication. Granulocyte colony-stimulating factor has produced significant improvement in a limited number of patients.
Human papillomavirus (HPV) Verruca vulgaris (wart) Oral squamous papilloma	The usual HPV types are found in oral lesions, but some uncommon variants such as HPV-7 and HPV-32 also are found. Lesions usually are multiple and may be found on any oral mucosal site.	Treatment of choice is surgical removal of the lesion(s). Other treatment modalities include topical podophyllin, interferon, and cryosurgery. Laser ablation and electrocoagulation have been used, but care must be taken because the plume may contain infectious HPV.

Histoplasmosis	Histoplasmosis is the most common endemic respiratory fungal infection in the United States and usually is subclinical and self-limiting. Dissemination of infection occurs in about 5% of patients with AIDS who live in areas in the United States where the fungus is endemic.	The treatment of choice for disseminated histoplasmosis is intravenous amphotericin B. Oral itraconazole also has been found to be effective and has fewer adverse effects, with better patient compliance.
Molluscum contagiosum	Molluscum contagiosum is caused by a poxvirus. The lesions are small papules with a central depressed crater. In immunocompetent persons, the lesions are self-limiting and are found on the genitals and trunk. In patients with AIDS, multiple lesions (hundreds) are found that do not regress (5% to 10% of patients with lesions have lesions of the facial skin).	Curettage, cryosurgery, and cautery have been used to treat these lesions, but they are painful and recurrences are common. Resolution of multiple lesions has been reported with HAART.
Thrombocytopenia	Thrombocytopenia is found in about 10% of HIV-infected patients. It may occur during any stage of the disease. Skin manifestations are most common, but petechiae, ecchymosis, and spontaneous gingival bleeding can occur in the oral cavity.	Platelet counts below 50,000/mm^3 may result in significant bleeding with minor surgical procedures. Platelet replacement may be indicated for these patients.
HIV-associated salivary gland disease	Found in 5% of HIV-infected patients and can occur any time during the infection. Bilateral swelling of the parotid is most common. In some patients, CD8+ lymphocytes infiltrate the gland and are associated with lymphadenopathy. Xerostomia may occur. Patients are at increased risk for B cell lymphoma.	Risk is increased for cysts of the parotid and lymphoma. Treatment involves antiretroviral therapy ± immune modulators. Associated xerostomia can be managed with sialogogues and saliva substitutes.

Hyperpigmentation	Melanin pigmentation has been reported to occur in HIV-infected patients. Several of the medications (ketoconazole, clofazimine, and zidovudine) used to treat these patients may cause melanin pigmentation. Addison-like pigmentation also may occur because of destruction of the adrenal gland. HIV infection itself may cause melanin pigmentation.	Usually, no treatment is indicated. Single lesions may have to be biopsied so that melanoma can be ruled out. Patients with Addison disease may require corticosteroids.
Lymphoma	Found in about 3% of patients with AIDS. Most are found in extranodal locations. Most lesions are non-Hodgkin B cell lymphoma and are related to the EBV. The CNS is the most common site, but oral lesions occur in the palate and gingiva and in other locations.	Treatment usually involves a combination of chemotherapy and radiation and is used for local control of disease. Prognosis is very poor, with death occurring within months of the diagnosis. HAART has reduced the prevalence of opportunistic infections and Kaposi sarcoma in HIV-infected patients but has not affected the prevalence of lymphoma.
Oral squamous cell carcinoma	Can be found in the oral cavity, pharynx, and larynx in HIV-infected persons. The same risk factors apply as for the general population, but the cancer occurs at a younger age (it appears that HIV infection accelerates onset of carcinoma).	Treatment of oral squamous cell carcinoma is the same as for non–HIV-infected patients: surgery, irradiation, chemotherapy, or combination therapy.


Thanks

Management of the Patient Undergoing Radiotherapy &/or Chemotherapy

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Radiotherapy

- Radiotherapy is a common therapeutic modality for malignancies of the head and neck. It destroys neoplastic (and normal) cells by interfering with nuclear material necessary for reproduction, cell maintenance, or both. The faster the cellular turnover, the more susceptible the tissue is to the damaging effects of radiation.

- In practice, normal tissues with rapid turnover rates are also affected to some degree. Therefore, hematopoietic cells, epithelial cells, and endothelial cells are affected soon after treatments with radiotherapy begin.

Radiation effects on oral mucosa

1. Mucositis (1 or 2 weeks).
2. Pain and dysphagia.
3. Loss of taste.
4. Delayed healing (late effect).
5. Submucosal fibrosis (late effect).

Radiation effects on salivary glands

- The salivary glands show considerable damage with resultant atrophy, fibrosis, and degeneration because of the destruction of the fine vasculature by the radiation. This manifests clinically as ***xerostomia*** (the decreased production of saliva) and gives the patient a dry mouth.

- Xerostomia complications are:
  - ✔ Rampant " radiation caries
  - ✔ Periodontitis is accelerated.
  - ✔ Dysgeusia, dysphonia and dysphagia.

Radiation effects on bone

- The bone within the radiation beam becomes virtually nonvital from an endarteritis that results in elimination of the fine vasculature within the bone.
- The turnover rate of any remaining viable bone is slowed to the point of being ineffective in self-repair.
- The continual process of remodeling normally found in bone does not occur.

Chemotherapy

- Like radiotherapy, the antitumor effect of cancer chemotherapeutic agents is based on their ability to destroy or retard the division of rapidly proliferating cells, such as tumor cells, nonspecifically.

- Unfortunately, normal host cells that have a high mitotic index are also adversely affected. Normal cells most affected are the epithelium of the Gastrointenstinal tract (including oral cavity) and the cells of the bone marrow.

Effects of chemotherapy

- On oral mucosa……Mucositis.
- On hematopoietic system……Myelosuppression. (leucopenia, thrombocytopenia and anemia)
- on oral microbiology…….. profound changes in the oral flora (overgrowth of indigenous microbes, superinfection with gram-negative bacilli, opportunistic infections)

General dental management

- In general, the principles of dental management for the patient who has had or will have radiotherapy apply equally well to the patient who has had or will have chemotherapy.

- The effects of the chemotherapy are almost always only temporary, and, with the passage of time, systemic health improves to optimal levels, which allows almost routine dental management.

Pretreatment evaluation and preparation

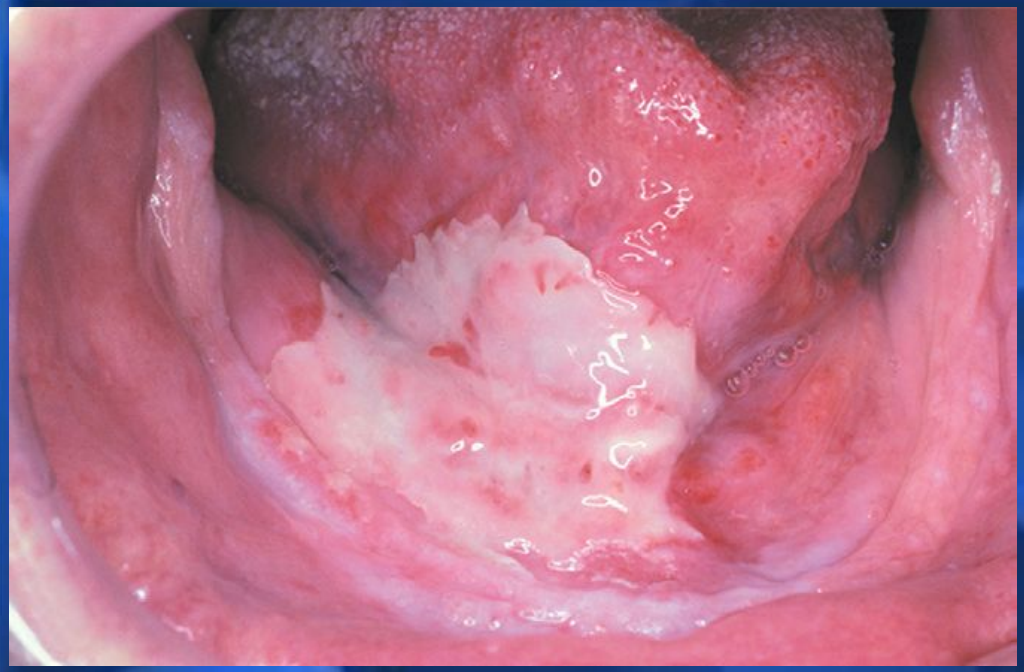
- Oral hygiene instructions and encouragement of non cariogenic diet.
- Elimination of all sources of irritation and infection.
- removable appliances should be removed during treatment (to prevent ulceration of the fragile mucosa).
- Because tobacco use and alcohol consumption irritate the mucosa, the patient should be encouraged to stop these before commencement of therapy.

Oral health care during therapy

Maintenance of oral health during cancer therapy is very important to outcome, because oral complications develop in a significant proportion of patients who undergo cancer irradiation and chemotherapy. Patients whose treatment will include head and neck irradiation or inpatient chemotherapy should have oral infections and potential problems eliminated before initiation of cancer therapy, with routine dental care delayed until after cancer therapy is complete. Outpatient chemotherapy requires dental treatment to be provided at appropriate times between cycles.

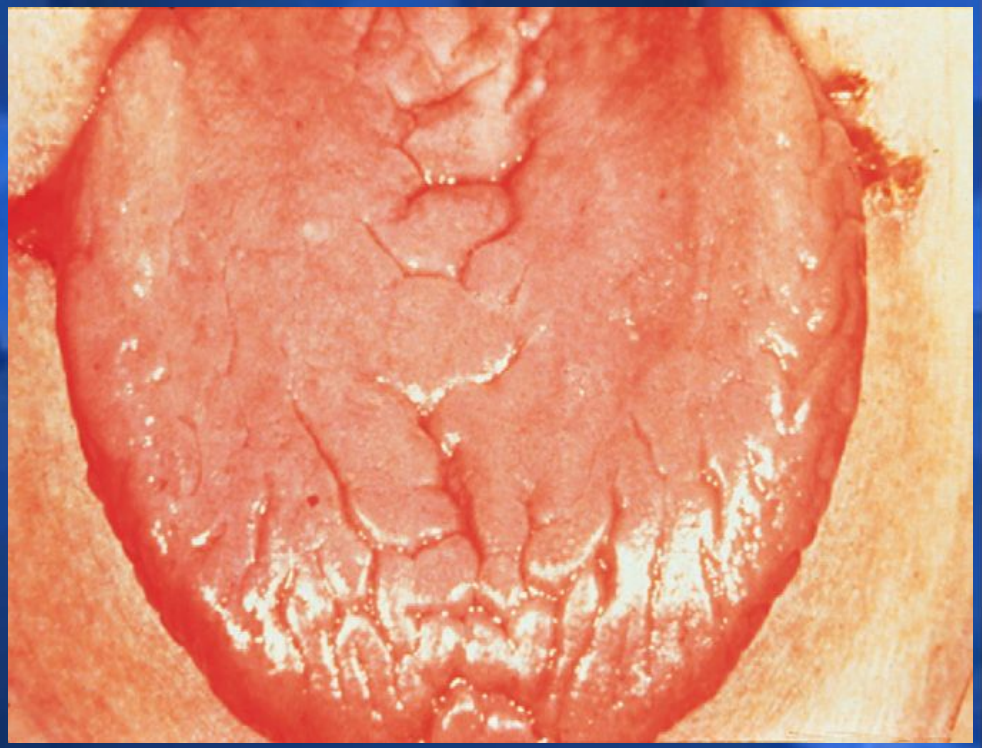
Management of Complications of Radiation Therapy and Chemotherapy

- ***Mucositis***: It is managed by antiseptic mouth rinses, topical anesthetics, anti-inflammatory agents like steroids, the patient should be kept well hydrated and nourished with avoidance of alcohol, tobacco and irritating foods.
- ***Xerostomia:*** (chronic dry mouth after radiotherapy)It is managed by frequent drinking of water, using salivary substitutes, sugarless chewing gum, lozenges and sialogogue drugs like Pilocarpine HCL (parasympathomimetic drug) to stimulate salivary flow.

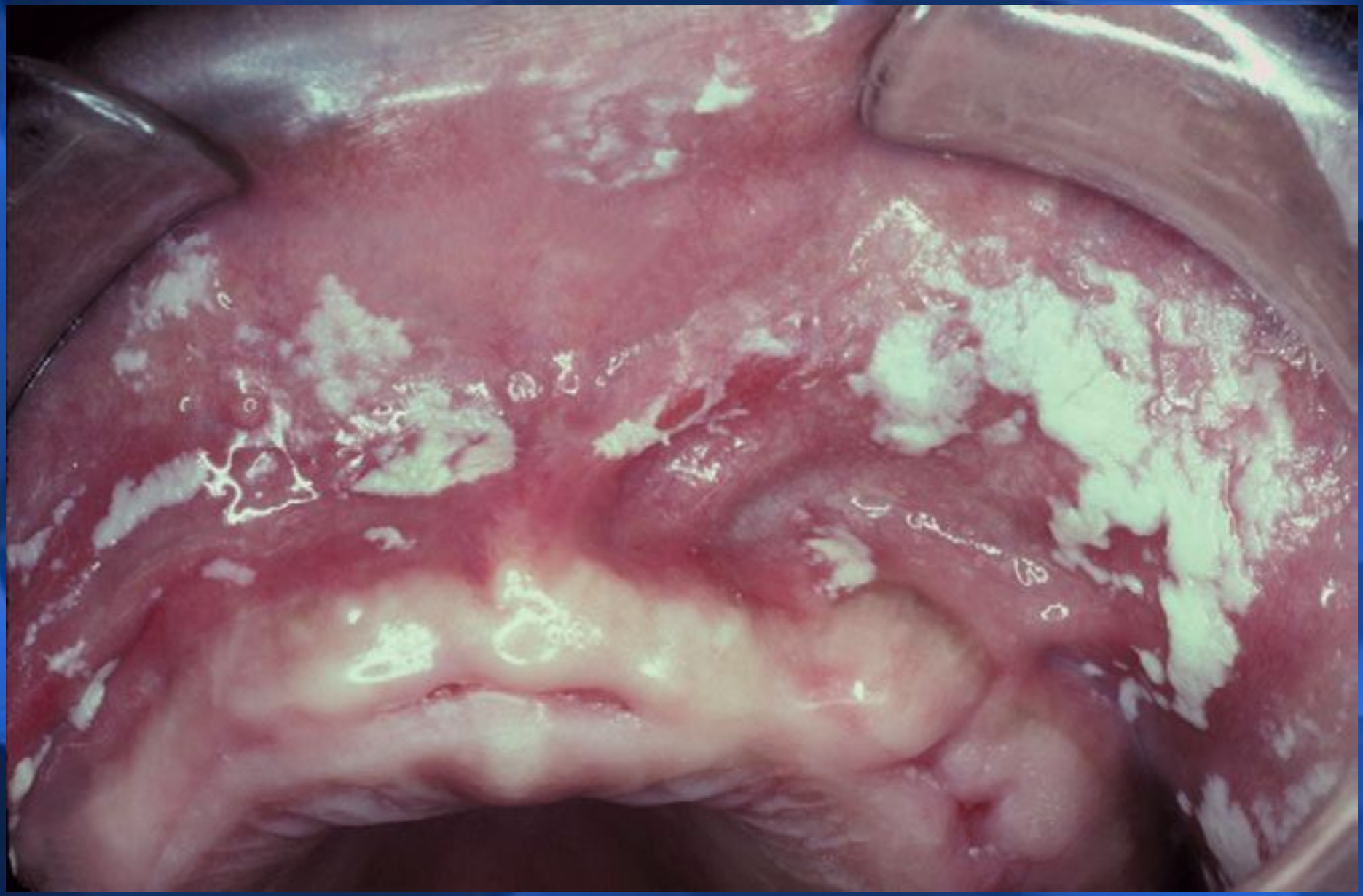


mucositis

xerostomia



- **Radiation caries:** Its management requires patient education, oral hygiene instructions, fluoride application, frequent dental visits and early restoration of teeth.
- **Secondary infections:** occurs as a result of immunosuppression and reduced salivary flow, when the WBC count falls below 2000 cell/ml the immune system is less able to manage these infections.
- **Sensitivity of the teeth:** it can have an acute or delayed onset, due to hyposalivation; it is managed by topical fluoride application.



Oral candidiasis (pseudomembranous form)

- **Bleeding:** Due to thrombocytopenia, when platelet count drops below 50.000/mm3, gingival bleeding and submucosal hemorrhage may occur; such patients are encouraged not to use tooth picks or dental floss. Management of bleeding is by local measures as applying pressure, using gelfoam and antifibrinolytic agents. Transfusion is recommended if local measures fail.

- **Diminished sense of taste:** as a result of damage to the microvilli of the taste cells, patients complain of a bitter or unpleasant taste, most of the patients will restore taste within 3-4 months after therapy. Zinc supplementation is reported to improve taste sensation.

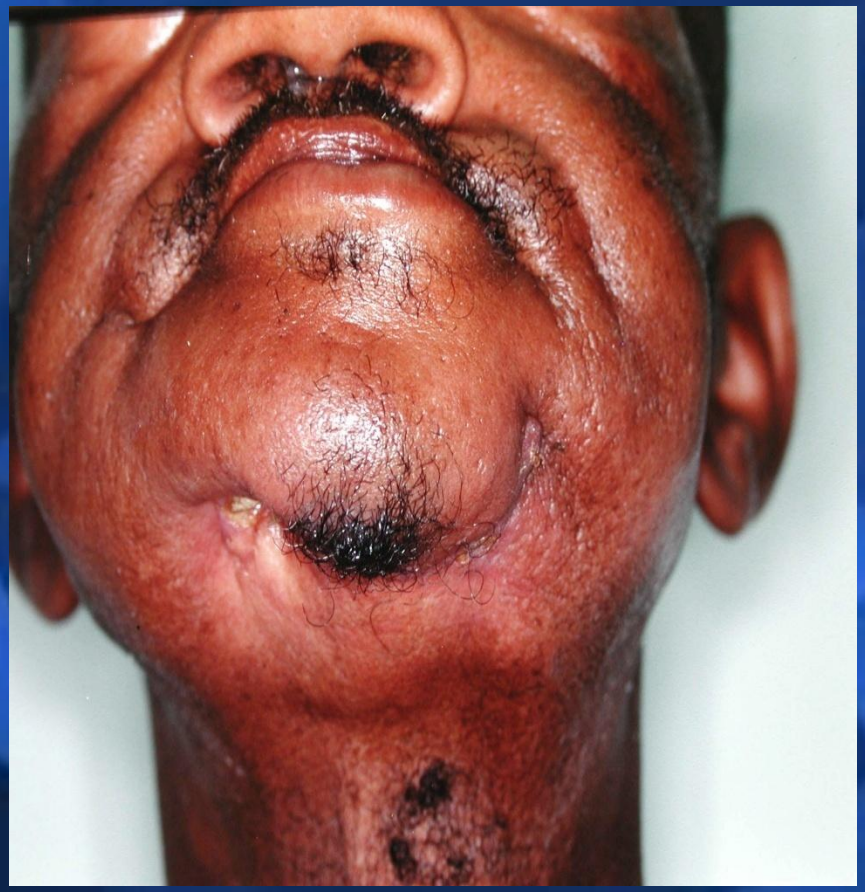
In most patients, the ability to taste returns in 3 to 4 months after completion of radiotherapy. In cases of chronic loss of taste, zinc supplementation has been reported to improve taste perceptions. Silverman recommends 220 mg of zinc two times a day for patients with severe chronic loss of taste. However, currently, no effective treatment is available for complete restoration of damaged taste.

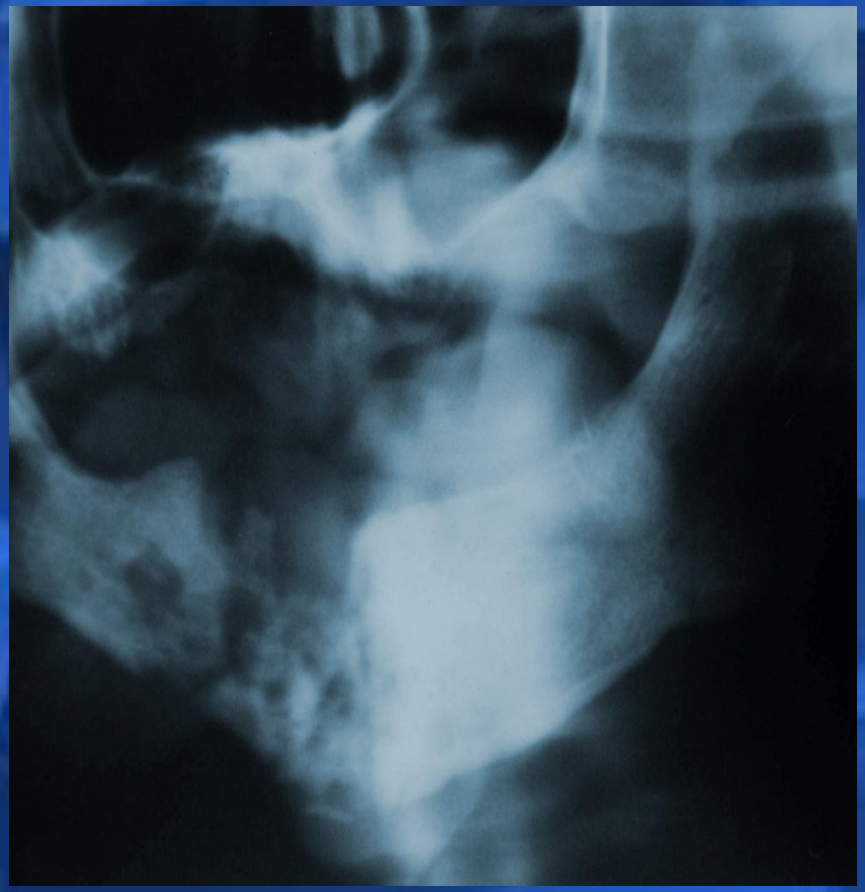
- **Muscular dysfunction:** it is a late complication of radiotherapy; it results from fibrosis and vascular damage of the muscles of mastication. Mouth opening should be maintained through physiotherapy.

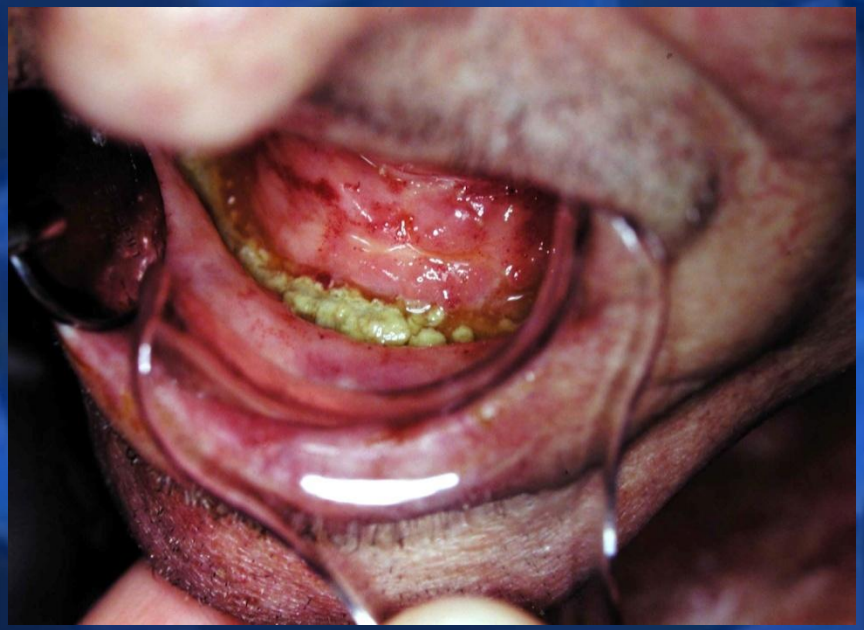
- **Carotid Atheroma:** It develops in patients who have received irradiation more than 45 Gy. It represents a risk factor for stroke; it requires evaluation by a specialist.

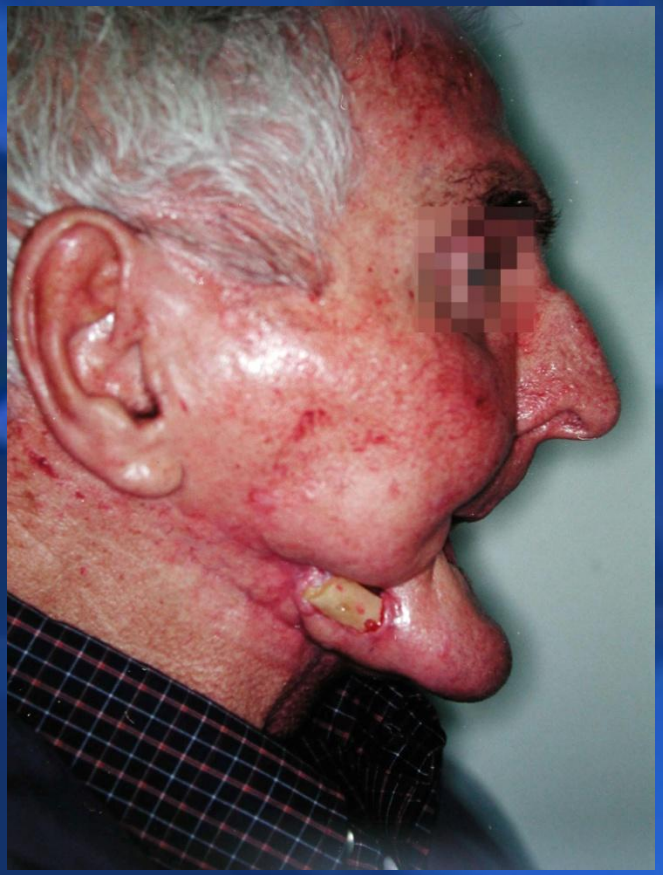
Osteoradionecrosis (ORN)

It is a late complication that arises after radiotherapy, it is a state of hypocellularity, hypovascularity and hypoxia of the irradiated jaw bones, characterized by exposed bone that fails to heal.









Factors that may increase the incidence of ORN:

- Dentate patient.
- Mandible, especially posterior region.
- Dose of radiation, the risk of ORN is greater when the dose exceeds 6500 cGy.
- Patients who continue smoking.
- Trauma (like extraction).
- Infection.

Prevention of ORN

- Maintain good oral hygiene.
- Eliminate smoking.
- Extraction of teeth should be done at least 2 weeks before radiotherapy.
- Avoid extraction during or after radiotherapy if possible.

- Minimize infection, use prophylactic antibiotics during extraction.
- Minimize or avoid vasoconstrictors in LA, consider hyperbaric oxygen.
- Minimize trauma through atraumatic procedure, avoid periosteal elevation as possible, eliminate bony edges or spicules, irrigation and obtaining primary closure

Management of ORN

- Daily irrigation of the exposed bone by normal saline, antiseptic or antibiotic solutions.
- Conservative local surgical debridement (trimming of the sharp edges of the sequestra) to avoid further trauma.
- Broad spectrum antibiotics (in acute secondary infections)

- Severe cases may require hyperbaric oxygen.

- For non healing wounds or extensive areas of ORN, surgical intervention may be indicated. In this instance, resection of the exposed bone and a margin of unexposed bone followed by primary soft tissue closure can be attempted.

Bisphosphonate-Associated Osteonecrosis

Bisphosphonates are synthetic analogues of inorganic pyrophosphate that have a high affinity for calcium. Bisphophonates also are potent inhibitors of osteoclastic activity. All bisphosphonate compounds accumulate over extended periods of time in mineralized bone matrix. Depending on the duration of the treatment and the specific bisphosphonate prescribed, the drug may remain in the body for years

Bisphosphonates are used to treat osteoporosis, Paget's disease of bone, multiple myeloma, and hypercalcemia of malignancy. In patients with osteoporosis, it is expected that bisphosphonates will arrest bone loss and increase bone density, decreasing the risk of pathologic fracture resulting from progressive bone loss. Bisphosphonates are given to patients with cancer to help control bone loss resulting from metastatic skeletal lesions.

Use of these agents has been shown to reduce skeletal-related events associated with multiple myeloma (such as fractures) and metastatic solid tumors, (such as breast, lung, and prostate cancers) in the bones.

- orally administered bisphosphonates often are used in patients with osteoporosis, while the injectable bisphosphonates are used in patients with cancer who develop primary lesions of bone or skeletal metastasis. BON can occur with the oral administration of bisphosphonates but is rare contrast BON is a much more common complication of injected bisphosphonates

Risk factors

- previous use of intravenous bisphosphonates (i.e., etidronate [Didronel], pamidronate [Aredia], zoledronic acid [Zometa]),
- diabetes mellitus,
- overall cancer stage and tumor burden,
- overall systemic and immune health, immunosuppressive drug use,
- any periodontal or other oral infection,
- and history of radiation to the jaws.
- Also, posterior sites are at higher risk than anterior sites, and the mandible is more often affected than the maxilla

According to the most recent recommendations from the American Association of Oral and Maxillofacial Surgeons (AAOMS), the working definition of BON (or BRONJ [bisphosphonate-related osteonecrosis of the jaw]) is based on the following criteria:

1. Current or previous treatment with a bisphosphonate

2. Exposed bone in the maxillofacial region that has persisted for more than 8 weeks

3. No history of radiation therapy to the jaws

The AAOMS staging (four stages) for BON (BRONJ)

- In the early stage (stage 0) of BON, no clinical or radiographic manifestations are evident. Patients usually are asymptomatic but may complain of nonspecific pain.

- Exposed bone becomes apparenta in stage 1, during which the patient is asymptomatic but may develop severe pain secondary to development of infection of the necrotic bone after exposure to the oral environment.

- In stage 2, the osteonecrosis often progresses, as evidenced by pain and erythema.
- Stage 3 is characterized by extension of exposed and necrotic bone beyond the region of alveolar bone, resulting in pathologic fracture, extraoral fistula formation, and establishment of oral antral–oral nasal communication.

- Patients with stage 2 or 3 BON may complain of severe pain and lack of sensory sensation (paresthesia). As noted previously, such changes may be an indication of peripheral nerve compression

Protocol for prevention of complications from cancer chemo- or radiation therapy:

a. Comprehensive examination
b. Establishment of excellent periodontal health (through eradication of any infection or inflammation)
c. Immediate extraction of all nonrestorable or questionable teeth
d. Elimination of dental caries
e. Maintenance of excellent oral hygiene and oral health

BOX 26-15 Treatment Strategies for BON (BRONJ)

At risk: Patients who are at risk for the development of BRONJ subsequent to exposure to a bisphosphonate do not require any immediate treatment. However, these patients should be informed of the associated risk, as well as the signs and symptoms of this disease process.

Stage 0: Provide symptomatic treatment, with conservative management for other local factors, such as caries and periodontal disease. Systemic management can include the use of medication for chronic pain and the control of infection with antibiotics, when indicated.

Stage 1: These patients benefit from the use of oral antimicrobial rinses, such as chlorhexidine 0.12%. No surgical treatment is indicated.

Stage 2: These patients benefit from the use of oral antimicrobial rinses combined with antibiotic therapy. It has been hypothesized that the pathogenesis of BRONJ might be related to factors adversely influencing bone remodeling. Additionally, the etiology of BRONJ does not include a primary infectious process. Most of the isolated microbes have been sensitive to the penicillin group of antibiotics. Quinolones, metronidazole, clindamycin, doxycycline, and erythromycin have been used with success in those patients allergic to penicillin. Microbial cultures also should be analyzed for the presence of *Actinomyces* bacteria. If this microbe is isolated, the antibiotic regimen should be adjusted accordingly. In some refractory cases, patients may require combination antibiotic therapy, long-term antibiotic maintenance, or a course of intravenous antibiotic therapy.

Stage 3: These patients benefit from débridement, including resection, combined with antibiotic therapy, which may offer long-term palliation, with resolution of acute infection and pain.

Regardless of disease stage, mobile segments of bony sequestrum should be removed without exposing uninvolved bone. The extraction of symptomatic teeth within exposed, necrotic bone also should be considered, because it is unlikely that the extraction will exacerbate the established necrotic process.


THINK
DO
BE
POSITIVE

Principles of Incisions and Flaps in Oral Surgery

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Oral & maxillofacial surgeon
B.D.S. – C.A.B.M.S

Oral surgery skills can be learned through the knowledge of the basic principles of surgery, knowledge of the anatomy of the region and good practical training. Whenever surgical intervention is considered, the operator must decide if the procedure is necessary weighing its benefits and its risks, and the patient must be made aware of the other possible non-surgical methods of treatment for the given problem. Also ail the short-term and long-term complications of the surgical procedure must be explained to the patient in relation to the known risks.

The main prerequisite for an operator performing surgical procedures is to ensure Asepsis and Antisepsis, to prevent pathogenic microbes from entering the body as well as spread of certain infectious diseases from one patient to another. This is accomplished through:

- Sterilization of instruments involving dry heat, moist heat (autoclave) and chemical means of sterilization.
- Preparation of the patient by seating the patient on the dental chair, disinfecting the skin around the mouth and the oral mucosa and covering the patient with sterile drapes.
- Preparation of the operator by disinfecting the hands and wearing the appropriate sterile gown and surgical gloves.

Flap is simply defined as a section of soft tissue that is outlined by surgical incisions, carries its own blood supply, allows surgical access to underlying tissues, can be replaced as required on its original position, maintained with sutures and is expected to heal. Most of the oral surgical procedures require the reflection of a full mucoperiostial flap incorporating mucosa, submucosa and periosteum to gain access to the area that is the object of surgery.

Incision

It is simply defined as a cut or wound made by cutting with a sharp instrument. The basic principles of incisions in oral surgery include:

- ✓ A blade number 15 is suitable for most oral surgical procedures. Sometimes a blade number 12 is used.
- ✓ A new and sterile blade should be used for each patient and it should be replaced with a new one intraoperatively if its cutting edge becomes blunted when necessary.
- ✓ The scalpel blade is mounted on the scalpel handle with the help of a needle holder, or hemostat, with which it slides into the slotted receiver with the beveled end parallel to that of the handle.


15


SWANN MORTON
ENGLAND B.S.
12

- ✓ The scalpel is grasped in a pen grasp for maximum control and tactile sensitivity.

- ✓ The incision should be made at right angle to the underlying bone to ensure good healing when the tissues are re-apposed.

- ✓ The scalpel should move at uniform speed and with sufficient firmness to cut through not only the mucosal surface but also the periosteum overlying the bone. It should be made, ideally, with a single movement, repeated strokes at the same place should be avoided as they may impair healing.


STAINLESS

Flap design

The essential points that should be considered include:

- ✓ 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, lingual nerve, submandibular duct, facial artery and vein. So thorough knowledge of the anatomy of the orofacial region is essential.

- ✓ The base of the flap should be wider than its apex (free gingival margin) to ensure adequate blood supply for better healing.

- ✓ The flap should be of adequate width for good visualization and accessibility of the operative field without subjecting the flap to tension and trauma during manipulation.

- ✓ When planning the flap the care should be given to the fact that the flap should be wider than the anticipated bony defect after completion of the procedure so that the flap margins, when sutured, should rest on intact and healthy bone to prevent wound dehiscence and poor healing.

- ✓ Delicate handling of the flap during the surgical procedure without excessive tension of crushing in order not to compromise the blood supply which leads to delayed healing.

- ✓ Vertical releasing incisions should start at the buccal vestibule and end at the interdental papilla which should either be excluded or included in the flap, the incision should always pass to the interdental papilla and not end at the labial or buccal surface of the tooth to ensure the integrity of the gingiva, but it should not pass through the papilla for accurate replacement of the flap. Vertical releasing incisions are contraindicated in certain sites in the oral cavity:

- ✓ Transverse incisions in the palate: to avoid injury to the greater palatine artery.
- ✓ Lingual surface of the mandible: to avoid injury to the lingual nerve, Canine eminence.
- ✓ In the area of mental foramen, between mandibular first and second premolars: to avoid injury to the mental nerve.

Types of Mucoperiosteal Flaps

Envelope Flaps

This type of flaps is made by a horizontal incision through gingival sulcus for the teeth or through the alveolar mucosa of the edentulous area with no vertical releasing incisions. The envelope flap is used for surgery of incisors, premolars and molars, on the labial or buccal and palatal or lingual surfaces. The main indications of this type of flaps include: surgical extraction of impacted mandibular third molars, palatal approach to impacted maxillary canines or removal of mandibular tori.

The main advantages of this flap are; easy re-approximation to original position, good blood supply and it can easily modified to two-sided or three-sided flap by adding vertical releasing incisions to either ends of the flap when necessary.

Disadvantages of this flap are the limited accessibility and visualization, difficulty in reflection with greater tension that can result in tearing at the ends of the flap, in addition to defect in attached gingival and the possibility of injury to the greater palatine artery during reflection of palatal flap.

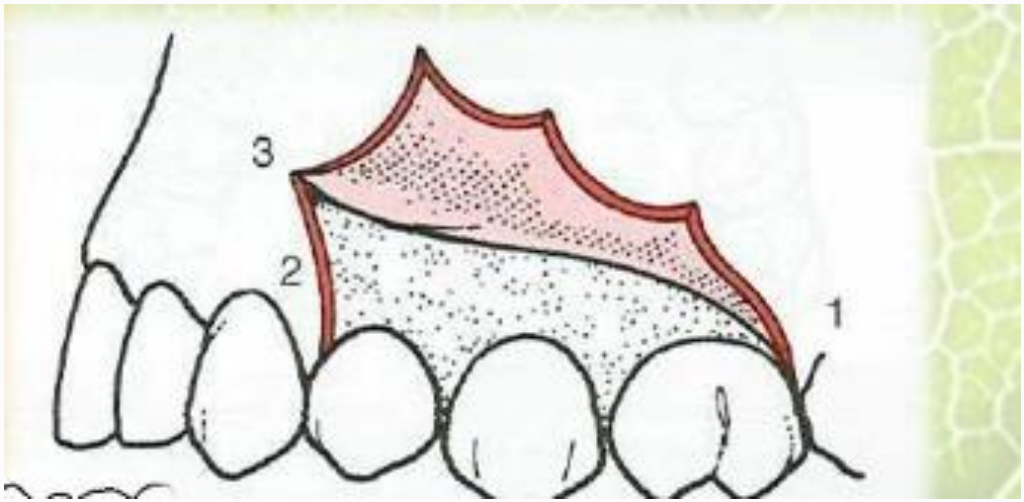
Envelope Flap

Two-sided Flap (Triangular Flap)

This flap is the made with a horizontal incision along the gingival sulcus or alveolar ridge mucosa and a vertical releasing incision. The vertical incision begins approximately at the vestibular fold and extends to the interdental papilla of the gingiva. This flap is performed labially or buccally on both jaws and is indicated in the surgical removal of root tips, impacted teeth, small cysts, and apicectomies.

Advantages are; it ensures an adequate blood supply, satisfactory visualization and accessibility, good re-approximation; it can be easily modified to a three-sided flap, or even lengthening of the horizontal incision.

Disadvantages are; limited access, tension when flap is retracted and it may result in defect of attached gingiva.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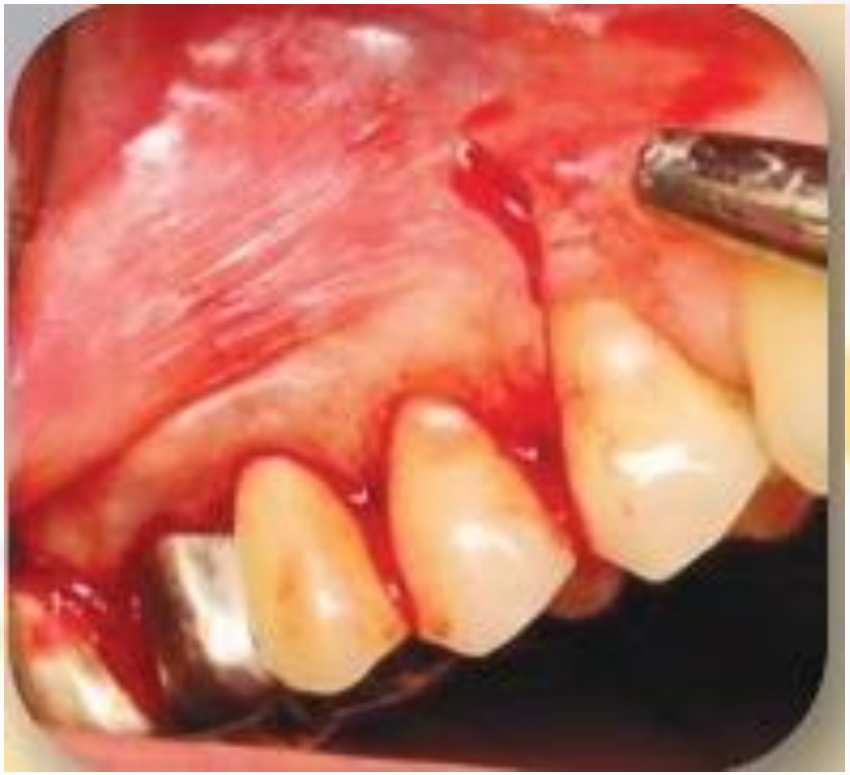
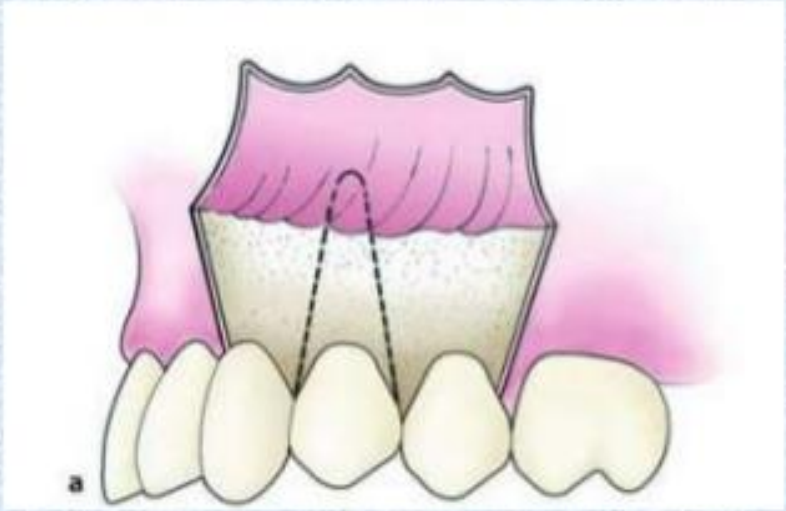


Three-sided Flap (Trapezoidal Flap)

This flap consists of a horizontal incision along the gingival or alveolar ridge mucosa and 2 vertical releasing incisions, this flap is indicated when an extensive surgical field exposure is required especially when two-sided flap is inadequate.

The main **advantages** include; very good accessibility and visualization of the surgical field with minimal tension on the tissue, and good re-approximation of tissue to the original position.

The **disadvantages** are the possibility of producing an attached gingival defect. This flap cannot be lengthened or modified once reflected.



Trapezoidal flap. a Diagrammatic illustration

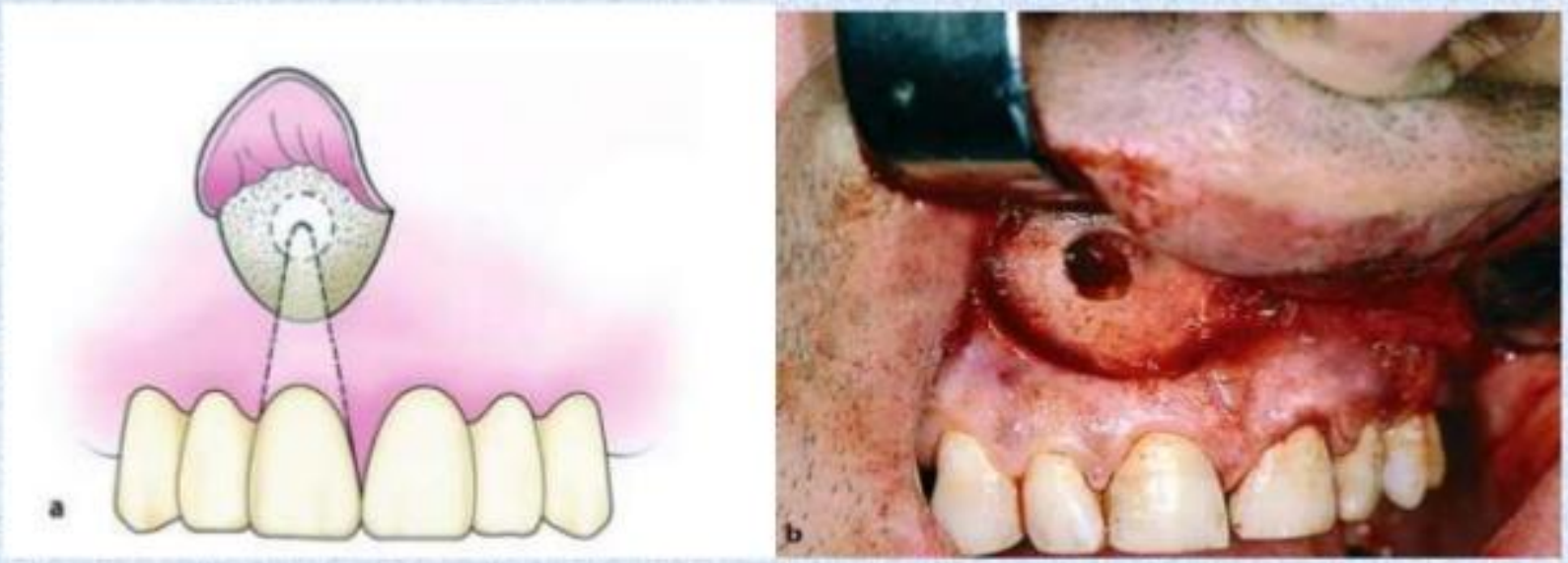
b. Clinical photograph

Semilunar Flap

This flap is the result of a curved incision, which begins just beneath the vestibular fold and has a bow shaped course with the convex part towards the attached gingiva. The lowest point of the incision must be at least 0.5 cm from the gingival margin, 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 of this flap are small incision, easy reflection, no attached gingival defect especially around prosthetic appliances (crowns and bridges) and easy oral hygiene.

Disadvantages of this flap are limited accessibility and visualization of the surgical field, re-approximation may be difficult due to the absence of reference points, tendency to tear due to excessive tension on reflection and the possibility that the flap may made over defective bone as a result of inadequate planning or underestimation of the size of the bony defect so that the margins of the flap will not rest on intact bone leading to collapse of the flap and wound dehiscence.

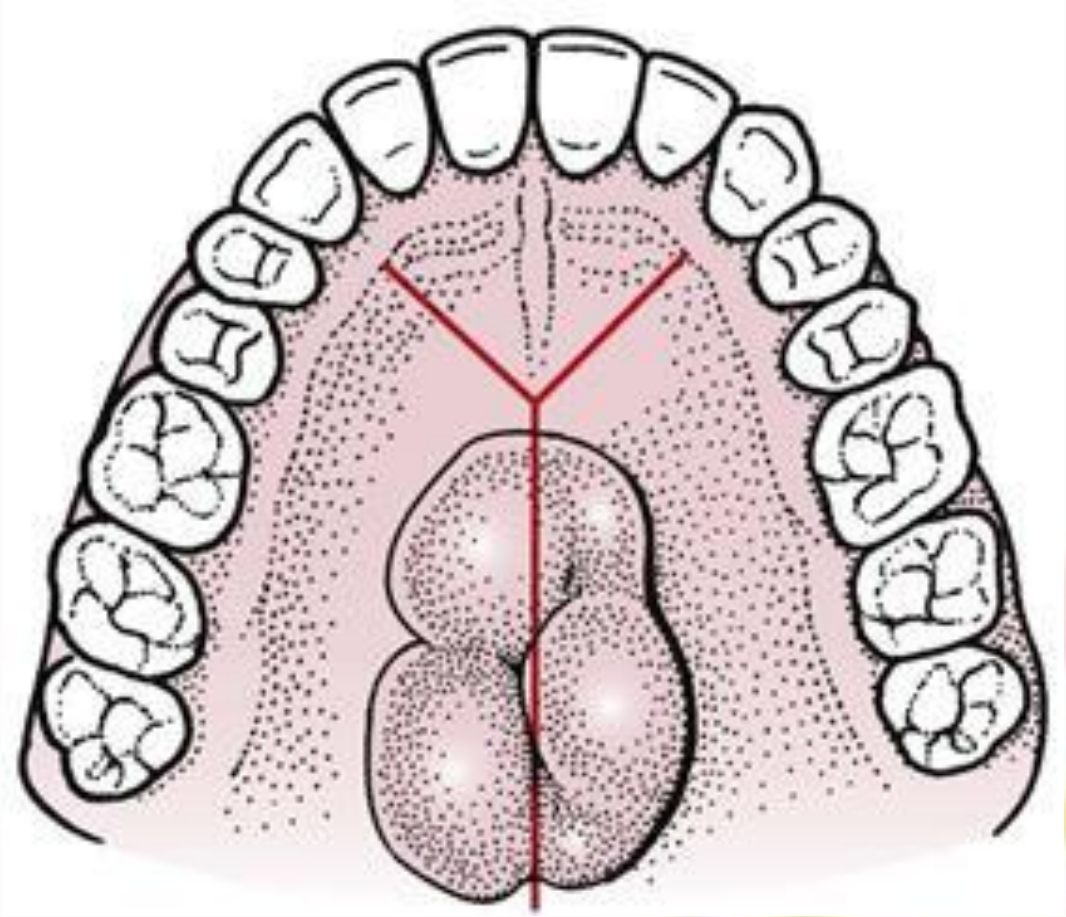


Semilunar flap. a Diagrammatic illustration. b Clinical photograph

Other types of flaps

These include flaps are the result of a Y-shaped and a Y shaped incision. These flaps are used in surgical procedures of the palate, mainly for the removal of exostoses (torus palatinus). The first flap consists of an incision along the midline of the palate with 2 anterolateral incisions made anterior to the canines, while in the second flap 2 additional posterolateral incisions are added to improve accessibility and its is indicated in large tori, but care should be taken not to sever the greater palatine vessels. The major disadvantage of these flaps is that they can easily tear as the mucosa overlying palatine tori can be fairly thin.

Flaps that are used for closure of oroantral fistula or communication include; buccal advancement flap which is in essence a three-sided flap that after reflection the periosteum is transversely incised so that the flap remains pedicled only by the mucosa allowing it to be advanced and sutured to the palatal tissues. The other flap is the palatal transpositional flap that incorporates the greater palatine vessel, it is rotated and sutured to the buccal tissues.



Flap reflection

The mucoperiosteal flap is reflected from the underlying bone using periosteal elevators. There are many any types of mucoperiosteal elevators like Howarth, Ash, the no.9 Molt, Seldin, or Freer types. The elevators should be firmly pushed at approximately 30-45° to the surface of the bone such that the periosteum is stripped from it. It is important to try to raise both mucosa and periosteum in one layer and this requires a considerable force to be applied. Reflection of the flap begins at the papilla, the periosteal elevator is pushed underneath the papilla in the area of the incision and is turned laterally to pry the papilla away from the underlying bone. This technique is used along the entire extent of the free gingival incision. If it is difficult to elevate the tissue at any one spot, the incision is probably incomplete, and that area should be re-incised.

periosteal elevator (Howarth)

A dry, sterile swab can be interposed between the periosteal, elevator and the bone. The elevator may also be used for holding the flap after reflecting, facilitating manipulations during the surgical procedure.

Oftentimes two elevators can be used to advantage, one working and the other aiding retraction in the subperiosteal plane. Adequate undermining of the wound margins is required in order to mobilize the flap. Generous reflection is the key to adequate vision, and wide exposure reduces traction trauma to the wound edges.

Suturing

After completion of the surgical procedure, thorough irrigation of the surgical field using sterile normal saline follows. Then the flap is repositioned to its original position and held in place using sutures to protect the underlying tissues from infection and irritating factors and prevent postoperative hemorrhage. Sutures are also used to repair soft tissue lacerations, ligation of vessels and control of bleeding, immobilization of flaps in their new position, and stabilization of drains in place. Suture diameters vary from 0.02 to 0.8 mm. This corresponds to 10/0 to 5 on the British Pharmacopoeia (BP) system. The finest suture that will hold the wound secure, without it breaking should be chosen. The amount of suture material used should be kept to a minimum, particularly when braided, to reduce bacterial colonization. Suture material can be a nidus for infection, and knots can be the focus of a persistent and chronic inflammatory reaction (suture knot sinus).

Suture Materials

Suture materials are classified as either absorbable or most-absorbable material depending on whether the body tissues will degrade the suture material and absorb it over time. Absorption takes place either by Hydrolysis or by proteolytic enzymatic degradation depending on the material used. They can also be classified as monofilament or multifilament.

- **Absorbable Sutures**

  They are used in suturing of deep layers of wounds when multilayered suturing is required, they are also used in children, mentally handicapped patients and in patients who cannot return to the clinic to have their sutures removed. They can cause inflammatory tissue reaction that can impede tissue healing. Some of the popular absorbable sutures include:

Plain Catguts it is made from collagen derived from healthy sheep or cattle intestine, its tensile strength is lost within 7-10 days, its absorption is through phagocytosis and enzymatic degradation which occurs within 7-10 days producing high tissue reaction. It is used for suturing subcutaneous tissues that do not require prolonged support. It is not suitable for suturing in oral surgery.

Chromic Catgut: it is made from collagen derived from healthy sheep or cattle intestine tanned with Chromium salts to facilitate handling and resist tissue degradation. It tensile strength is lost within 18- 21 days, its absorption is like that of the plain Catgut but it takes longer time and with moderate tissue reaction. It has the same indication as for the plain Catgat and it is not suitable in oral surgery.

Polyglactin (Vicryl): this synthetic suture material is made of copolymer of lactide and glycolide coated with polyglactin and calcium stearate. It is braided multifilament suture, 60% of its tensile strength remains for 2 weeks, and about 30% for 3 weeks. Its absorption is through hydrolysis with complete absorption taking place within 60-90 days, it induces mild tissue reaction. This suture is widely used in surgical practice but it is not advised for use where prolonged approximation under tension is required.

Polydioxanone (PDS): supplied as monofilament dyed or undyed, it is made of polyester polymer, 70% of its tensile strength remains at 2 weeks, 50% at 4 weeks and 14% at 8 weeks. Absorption occurs through hydrolysis which is complete in about 180 days, it is used when slight longer wound support is required.

- **Non-absorbable sutures**

  These sutures remain in the tissues and are not absorbed, but have to be cut and removed about 7 days after their placement. Commonly used sutures include:

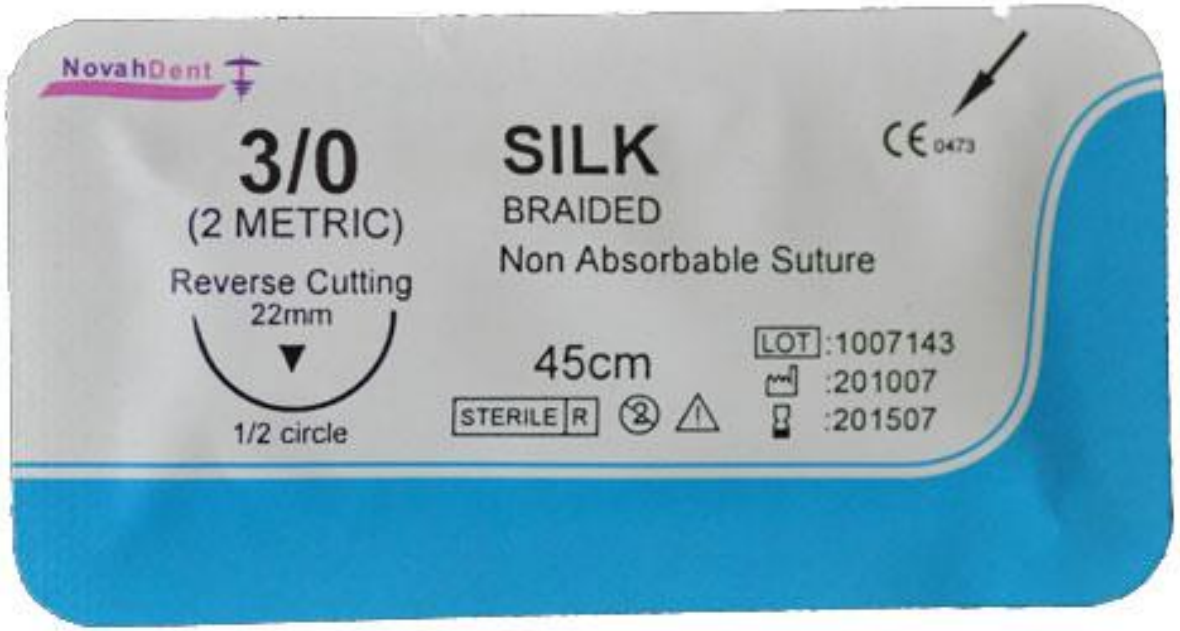
Silk: it is made of raw silk from silkworms, and it is supplied as braided or twisted, dyed or undyed, coated with wak or silicon or uncoated. 80%-100% of its tensile strength is lost within 6 months. Fibrous encapsulation occurs in the body within 2-3 weeks, it causes moderate to high tissue reaction. It is used in ligation and suturing when long term tissue support is needed.

Silk sutures are the easiest to use and the most economical, and have a satisfactory ability to make a secure knot.

Nylon: it is made of polyamide polymer and it is supplied as monofilament or braided multifilament dyed or undyed. It loses 15%-20% of its tensile strength per year. It causes mild tissue reaction and it is used mainly for skin, in plastic surgery, neurosurgery, and ophthalmic surgery.

Poly propylene (Prolene): it is made of polymer of propylene and it is supplied as a monofilament dyed or undyed. It produces low tissue reaction and remains encapsulated in the tissue. It is mainly used in skin.

One of the most commonly used suture for the oral cavity is 3/0 black silk. The size 3/0 has the appropriate amount of strength; the multifilament nature of the silk makes it easy to tie and well tolerated by the patient's soft tissues. The color makes the suture easy to be seen when the patient returns for suture removal. However, because of the multiple filaments, they tend to "wick" oral fluids along the suture to the underlying tissues. This wicking action may carry bacteria along with the saliva. Sutures that are holding mucosa together usually stay no longer than 5 to 7 days, so the wicking action is of little clinical significance.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Needles

Needles are usually made of stainless steel which is strong and flexible material. The choice of surgical needle is as important as the choice of the suture material. There are different shapes, sizes and cross sections of needles. Needles of 18-26 mm in length are suitable for use in oral surgery.

There are two basic needle types: Those that have the hole at the suture side of the needle and that need to be threaded with suture are “eyed.” Conversely, those that have the suture attached to the needle are “eyeless” or “swaged.” The advantages of the swaged needles include:

- The eyeless needle is composed of a single use needle and suture. This avoids the loss of sharpness that occurs with reusable needles.
- There is only a single strand of suture that is pulled through the tissues, and the gap that is created by the needle is fully plugged by the suture. This reduces potential leakage through the suture line.
- There is no re-threading of an eyeless needle, and its use is more time efficient.

As compared to a regular circle, needles are either; 1\4 circle, 1\2 circle, *3\4* circle, 3/8 circle, or 5/8 circle or they can have different shapes like straight needles, J needles, or compound curve needles.

According to the cross section of the needles, there are:

- Needles with round or oval cross section which are considered atraumatic and are mainly used for suturing thin mucosa. Their disadvantage is that great pressure is required when passing through the tissues, which may make suturing the wound harder. They are used in oral surgery especially in areas of thin mucosa they are also used in suturing of peritoneum, bowel, muscles and fat.
- Needles with triangular cross section; these are either cutting or reverse cutting needles. These designs allow minimal soft tissue trauma during needle insertion as they cut a path through the soft tissues and do not therefore require excessive force on the part of the operator.
- The passage of a needle through tissue should follow its curvature. This minimizes tissue damage and the appropriate size and shape of cutting, or round-bodied atraumatic needle, needs to be chosen for the least traumatic passage through tissue.

Needle Holder

These instruments come in a variety of sizes and design and operators tend to choose one that suits them. In general, they have a locking handles allowing the needle to be locked into the beaks of the instrument. They resemble Hemostats but with few differences; the beaks of the hemostat is longer and thinner than that of the needle holder, also the internal surface of the short beaks of the needle holder is grooved and crosshatched, permitting a firm and stable grasp of the needle, while the short beaks of the hemostat have parallel grooves which are perpendicular to the long axis of the instrument.

Tissue Forceps

Sometimes known as dissecting forceps, the important requirement is that they hold the soft tissues atraumatically so avoiding crushing and with little chance of slippage. This is achieved by a toothed design in the form of a wedge-shaped projection or tooth on one side, and a receptor on the other, which fit into each other when the handles are locked, although possibly causing tiny puncture points, is ideal for the purposes of suturing and holding soft tissues generally. The use of non-toothed forceps will result in crushing of the tissues as, to prevent tissue slippage from grasp, the instrument must be held too tightly.

Break

Principles of Incisions and Flaps in Oral Surgery

Principles of suturing

Suturing should be undertaken using a no-touch technique to reduce the risk of a needle-stick injury and the fewer the number of sutures used to produce the desired result, the better. Insertion of too many sutures tears the tissue unnecessarily, and the resulting tangle of suture thread tends to accumulate plaque and promote inflammation.

When re-approximating the flap, the suture is passed first through the mobile (usually facial) tissue, the needle is re-grasped with the needle holder and is passed through the attached tissue of the lingual papilla. But if the two margins of the wound are close together, the surgeon may be able to insert the needle through both sides of the wound in a single pass. However, for better precision it is better to use two passes in most situations.

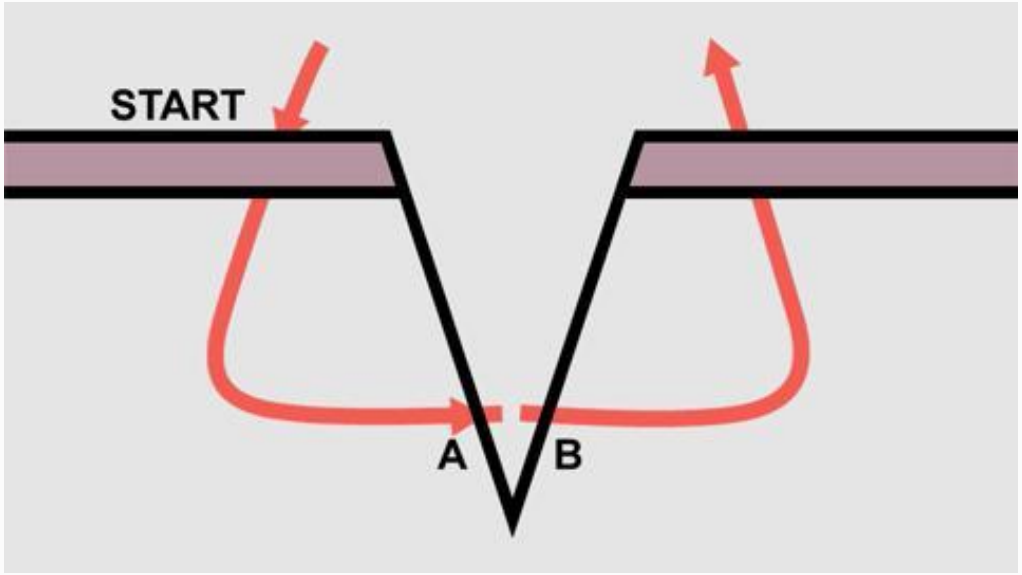
- The tissue of the flap should be held firmly by the tissue forceps and the needle passed through the mucoperiosteum about 3 mm from the margin, more if the flap is friable because of chronic infection. The needle is then pushed through the corresponding tissue on the other side of the incision, again about 3-5 mm from the margin. The needle should enter the surface of mucosa at right angle, and the passage of the needle should follow it curvature to prevent tearing of the flap.

- After the needle passes through both wound edges, the suture is pulled, so that the needle-bearing end is longer. Afterwards, the long end of the suture is wrapped around the handle of the needle holder twice. The short end of the suture is grasped by the needle holder and pulled through the loops. The suture is then tightened by way of its two ends, thus creating the first double-wrapped knot. Then a single-wrap knot is created, in the counterclockwise direction, which is named a safety knot.

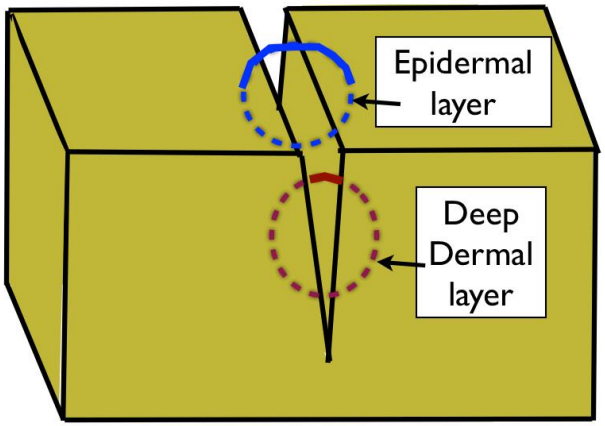
- Where possible, the knots should be drawn to lie to one or other side of the line of incision. Over-tightening of the suture, manifested by blanching of tissue, must also be avoided, it runs the risk of tissue necrosis and wound dehiscence. Overlapping of wound edges when positioning the knot should also be avoided.
- Before the sutures are inserted the non-flap side of the incision should be undermined to facilitate the insertion of the needle.
- Sutures placed intraorally are normally removed 5-7 days postoperatively. In the removal of sutures, normal dental tweezers should grasp the free ends of the thread and the suture should be cut by sharp scissors. The suture should then be pulled though in its entirety. The suture is better cut just as it enters the tissue to avoid pulling a contaminated suture through the tissue.

Suturing Techniques

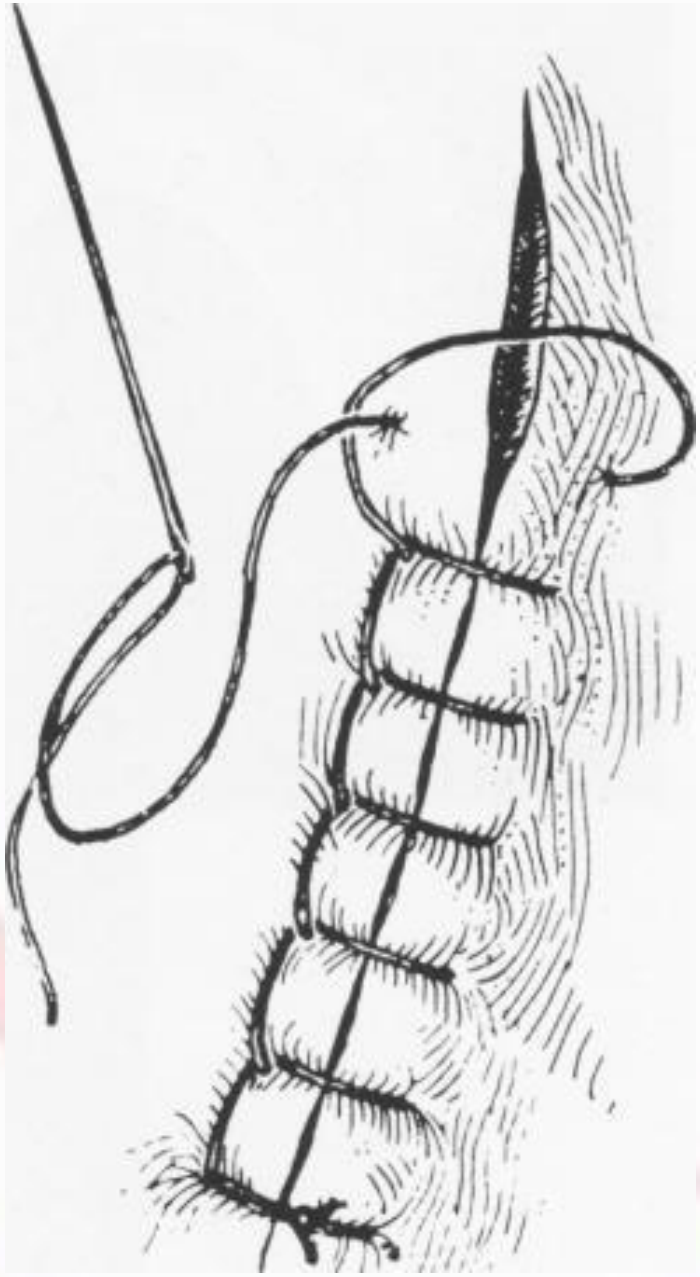
- **Simple Interrupted Sutures** This is the simplest and most frequently used type, and may be used in all surgical procedures of the mouth. The needle enters from the margin of the flap (mobile tissue) and exits at the same distance on the opposite side. The two ends of the suture are then tied in a knot. The advantage of the interrupted suture is that it is simple to execute and when sutures are placed in a row, inadvertent loosening of one or even losing one will not influence the rest.

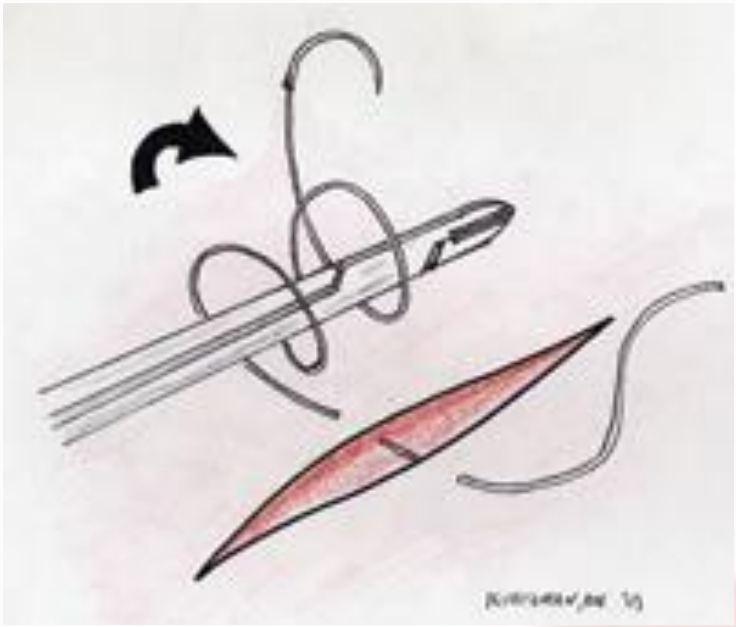


Layered Repair



- **Continuous Sutures** This is usually used for the suturing of wounds that are long, e.g., for re-contouring of the alveolar ridge in the maxilla and mandible. This technique for the continuous simple (or nonlocking) suture is as follows: after passing the needle through both flap margins, an initial knot is made just as in the interrupted suture but only the free end of the suture is cut off. The needle-bearing suture is then used to create successive continuous sutures at the wound margins. The last suture is not tightened, but the loop created actually serves as the free end of the suture that is used to tie the knot.



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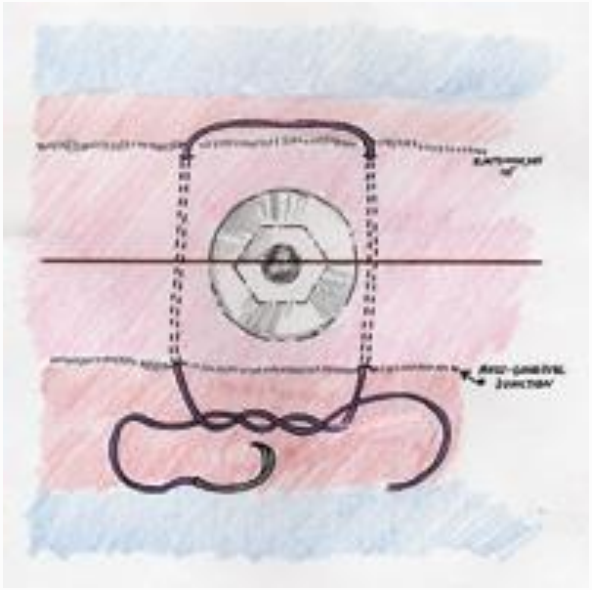
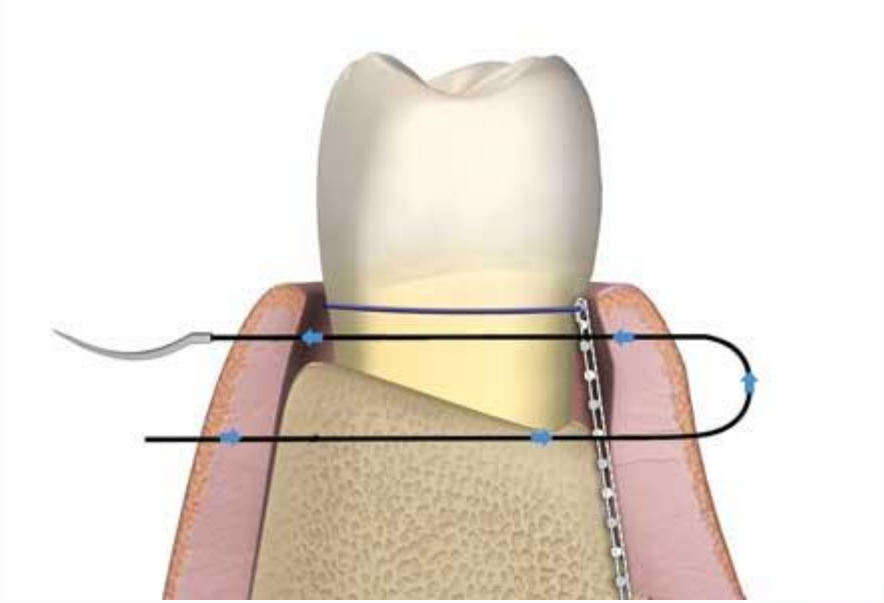
The continuous locking suture is a variation of the continuous simple suture. This type of suture is created exactly as described above, except that the needle passes through every loop before passing through the tissues, which secures the suture after tightening. Suturing continues with the creation of such loops, which make up parts of a chain along the incision. These loops are positioned on the buccal side of the wound, after being tightened.

The advantage of the continuous suture is that it is quicker and requires fewer knots, so that the wound margins are not tightened too much, thus avoiding the risk of ischemia of the area. Its disadvantage is that if the suture is inadvertently cut or loosened, the entire suture becomes loose.

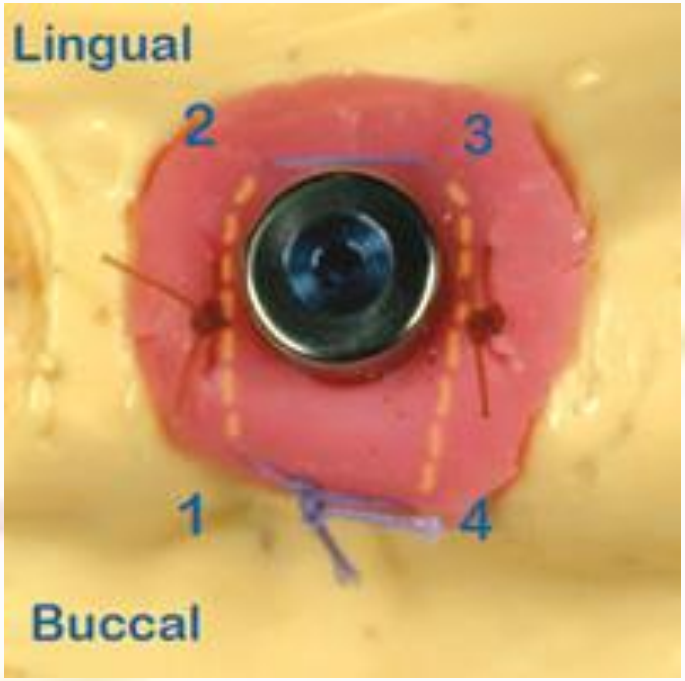
- **Mattress Suture:** This is a special type of suture and is described as horizontal and vertical. It is indicated in cases where strong and secure re-approximation of wound margins is required. The main indication for use of vertical mattress sutures is to evert the skin edges, the technique permits greater closure strength and better distribution of wound tension. The horizontal suture also allows eversion of the wound edges and is used in cases which require limiting or closure of soft tissues over osseous cavities, e.g., post-extraction tooth sockets. In the mattress suture the needle passes through the wound margins at a right angle, and the needle always enters and exits the tissues on the same side.

- **Figure of Eight suturing**: occasionally placed over top of socket to aid in hemostasis, it is usually performed to help in maintaining a piece of oxidized cellulose in tooth socket after tooth extraction.





Mattress suturing



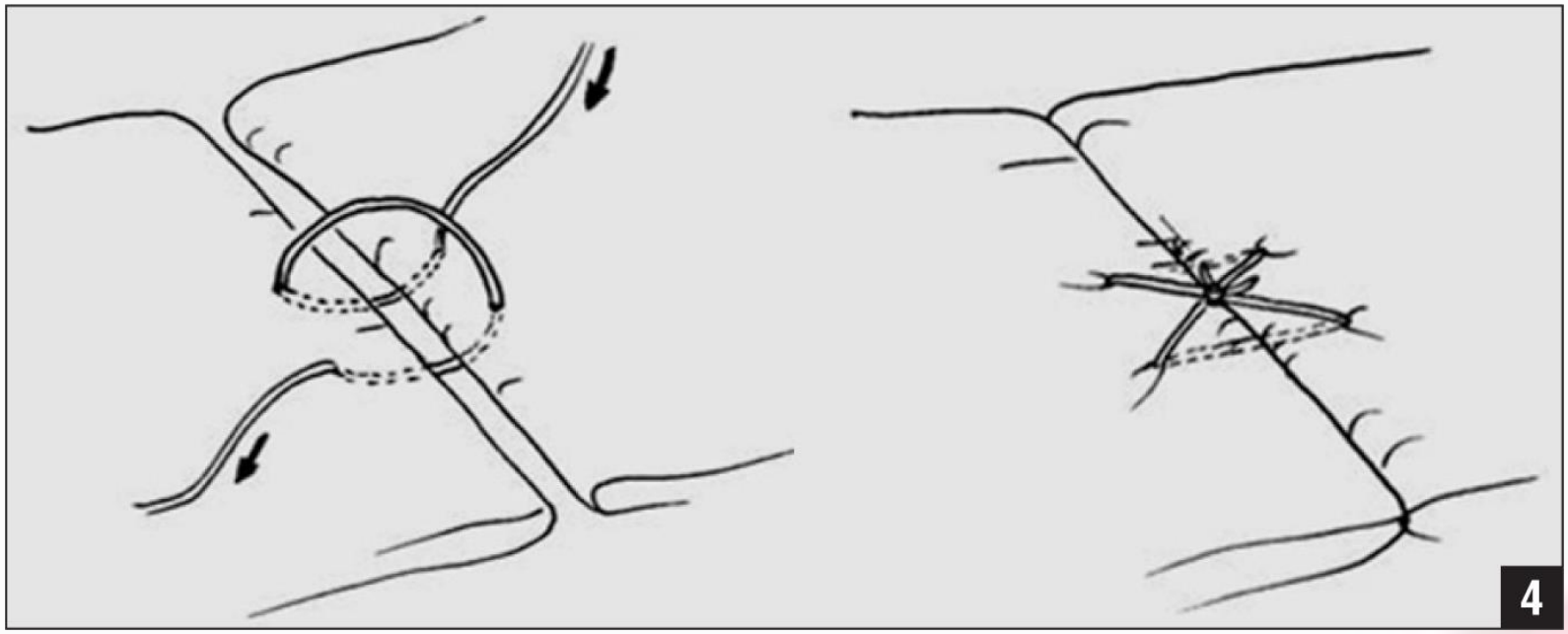
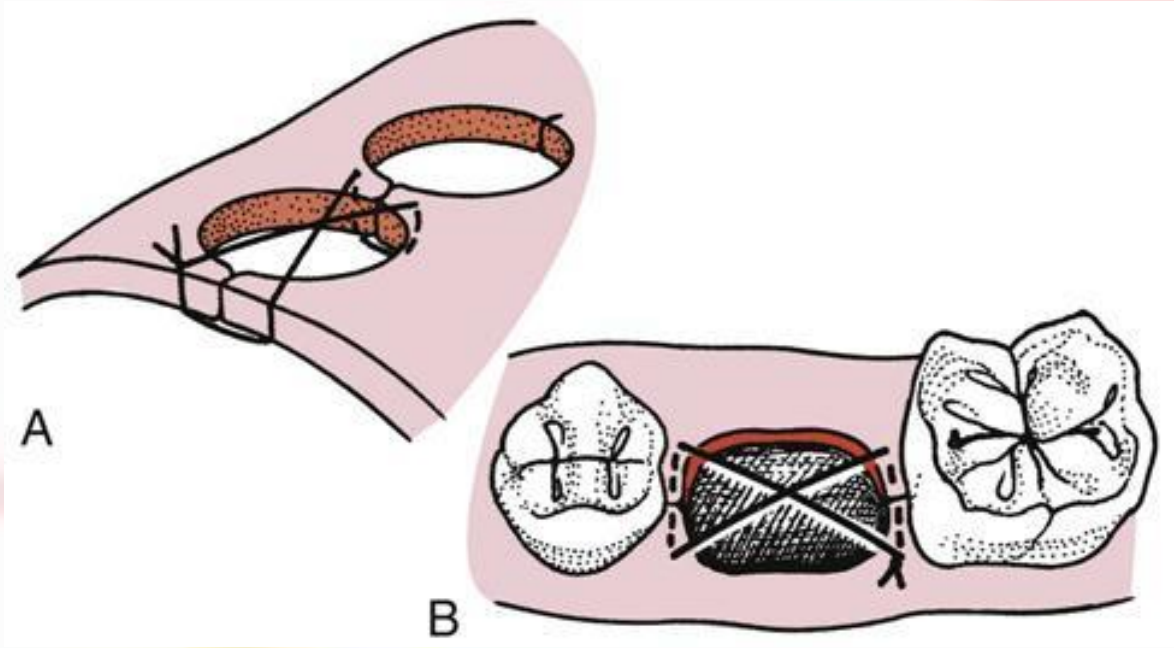


Figure of 8 suturing



Management of difficult extraction

Difficulties may be encountered during extraction of teeth for a variety of reasons and any oral surgeon should never ignore any warning of the possibility that such difficulties may be encountered during any proposed tooth extraction especially when the patient indicates that such difficulties have been experienced in previous occasions. These difficulties can be diagnosed through clinical examination and a good radiograph that shows the root morphology of the tooth and the surrounding and supporting structures which should be taken before extraction whenever possible.

The main indications for surgical extraction of teeth are:

- ✓ Retained roots and root tips.
- ✓ Teeth with root morphology that is unfavorable for simple tooth extraction, such as teeth with large bulbous roots due to hypercementosis or those with dilacerated roots or root tips.
- ✓ Teeth that have crowns with extensive caries, especially root caries, or that have large amalgam restorations.
- ✓ Teeth with ankylosed roots.
- ✓ Teeth that are fused with adjacent teeth or roots.
- ✓ Maxillary posterior teeth that are closely associated with maxillary sinus due to pneumatization of the sinus into the alveolar process or when there is an increased risk of fracture of maxillary tuberosity during simple extraction.

- ✓ Fully or partially impacted teeth.
- ✓ Teeth or retained roots with periapical lesions whose removal in entirety is not possible through curettage alone after simple extraction.
- ✓ Retained roots and root tips that are deeply buried in the alveolus and are asymptomatic are not indicated for surgical extraction especially in older individuals with poor general health, or when there is a risk of serious local complications or damage to vital structures like the inferior alveolar nerve, lingual nerve or maxillary sinus.

Steps of surgical extraction

- Surgical extraction of teeth is preceded by proper preparation and disinfection of the patient, and administration of local anesthesia then the surgical procedure proceeds as follows:
- Creation of a full mucoperiosteal flap, this could be envelope, two-sided or three-sided flap based on the anticipated difficulty and the need for better accessibility.
- For single rooted teeth and after reflecting the flap the operator may attempt to re-seat the forceps under direct visualization for better mechanical, advantage. The other options include; grasping a bit of buccal bone under the buccal beak of the forceps so that a small piece of buccal bone is removed with the tooth, application of an elevator, or removal of sufficient amount of bone to facilitate the application of the forceps or the elevator. In some cases a purchase point is made in the root where the elevator is applied and the root extracted.
- Bone removal using surgical hand piece and round burs to expose an adequate part of the tooth or root. Whenever possible the oral surgeon should be conservative by removing bone to allow the creation of a point of application for the elevator for luxation or sometimes removing only a small window of bone overlying the broken apex of roots to allow their retrieval through the socket.

- In multi-rooted teeth sectioning of the crown of the tooth and/or sectioning the roots so that they can be extracted as single rooted teeth. Care should be taken when extracting a retained root of upper maxillary molars using elevators, since applying excessive pressure in apical direction may result in displacement of the root into the maxillary sinus.
- After the tooth and all the root fragments have been removed, the flap is repositioned and the surgical area is palpated for sharp bony edges. If any sharp edges are present, they are smoothed with a bone file or a handpiece and bur.
- The wound is thoroughly irrigated and debrided of loose fragments of tooth, bone, calculus, and other debris.
- The flap is repositioned again and sutured in the usual fashion.

Indications for leaving root fragments

When a root tip has fractured and approaches of removal have been unsuccessful, and when the open surgical approach may be excessively traumatic, the surgeon may consider leaving the root in place. As with any surgical approach, the surgeon must balance the benefits against the risks of surgery. In some situations the risks of removing a small root tip may outweigh the benefits.

The conditions that must exist for a tooth root to be left in the alveolar process are:

- The root fragment must be small, usually no more than 4 to 5 mm in length.
- The root must be deeply embedded in bone and not superficial, to prevent subsequent bone resorption from exposing the tooth root and interfering with any prosthesis that will be constructed over the edentulous area.
- The tooth involved must not be infected, and there must be no radiolucency around the root apex. This lessens the likelihood that subsequent infections will result from leaving the root in position.
- The root should not be mobile.
- If the surgeon elects to leave a root tip in place the patient must be informed that, in the surgeon's judgment, leaving the root in its position will do less harm than surgery. In addition, radiographic documentation of the presence and position of the root tip must be obtained and retained in the patient's record, the patient should be recalled for follow-ups to track the fate of this root.
- The patient should be instructed to contact the surgeon immediately should any problems develop in the area of the retained root.

Multiple Extractions

If multiple adjacent teeth are to be extracted at a single session the surgeon should determine if there is need for interim partial immediate dentures, any type of soft tissue surgery, such as tuberosity reduction or the removal of undercuts or tori in critical areas. If dental implants are to be placed at a later time, it may also be desirable to limit bone trimming and socket compression. In some situations, dental implants may be placed at the same time as the teeth are removed, which would require the preparation of a surgical guide stent to assist in aligning the implants appropriately.

Extraction sequencing

- **Maxillary teeth should usually be removed first for several reasons:**
  - Infiltration anesthetic has a more rapid onset and also disappears more rapidly. This means that the surgeon can begin the surgical procedure sooner after the injections have been given.
  - Surgery should not be delayed because profound anesthesia is lost more quickly in the maxilla.
  - During the extraction process, debris such as portions of amalgams, fractured crowns, and bone chips may fall into the empty sockets of the lower teeth if the lower surgery is*performed first.
  - Maxillary teeth are removed with a major component of buccal force. Little or no vertical traction force is used in removal of these teeth, as is commonly required with mandibular teeth.

A single minor disadvantage for extracting maxillary teeth first is that if hemorrhage is not controlled in the maxilla before mandibular teeth are extracted, the hemorrhage may interfere with visualization during mandibular surgery but this is usually not a major problem because hemostasis should be achieved in one area before the surgeon turns attention to another area of surgery, and the surgical assistant should be able to keep the surgical field free from blood with adequate suction.

- **Posterior teeth are extracted first, this allows for the more effective use of dental elevators and forceps to extract the teeth.**

After extraction the buccolingual plates are pressed with firm pressure and the soft tissues are repositioned, sharp spicules of bone should be removed and smoothed with bone nibbler (Rongeur) and bone file, the area should be thoroughly irrigated with normal saline and the papillae in position.


ENDING SLIDE
THANK YOU FOR YOUR ATTENTION!

IMPACTED TEETH \ 2

Dr. Usama Aldaghir
Oral & maxillofacial surgeon
B.D.S. – C.A.B.M.S

THE IMPACTED MAXILLARY THIRD MOLAR

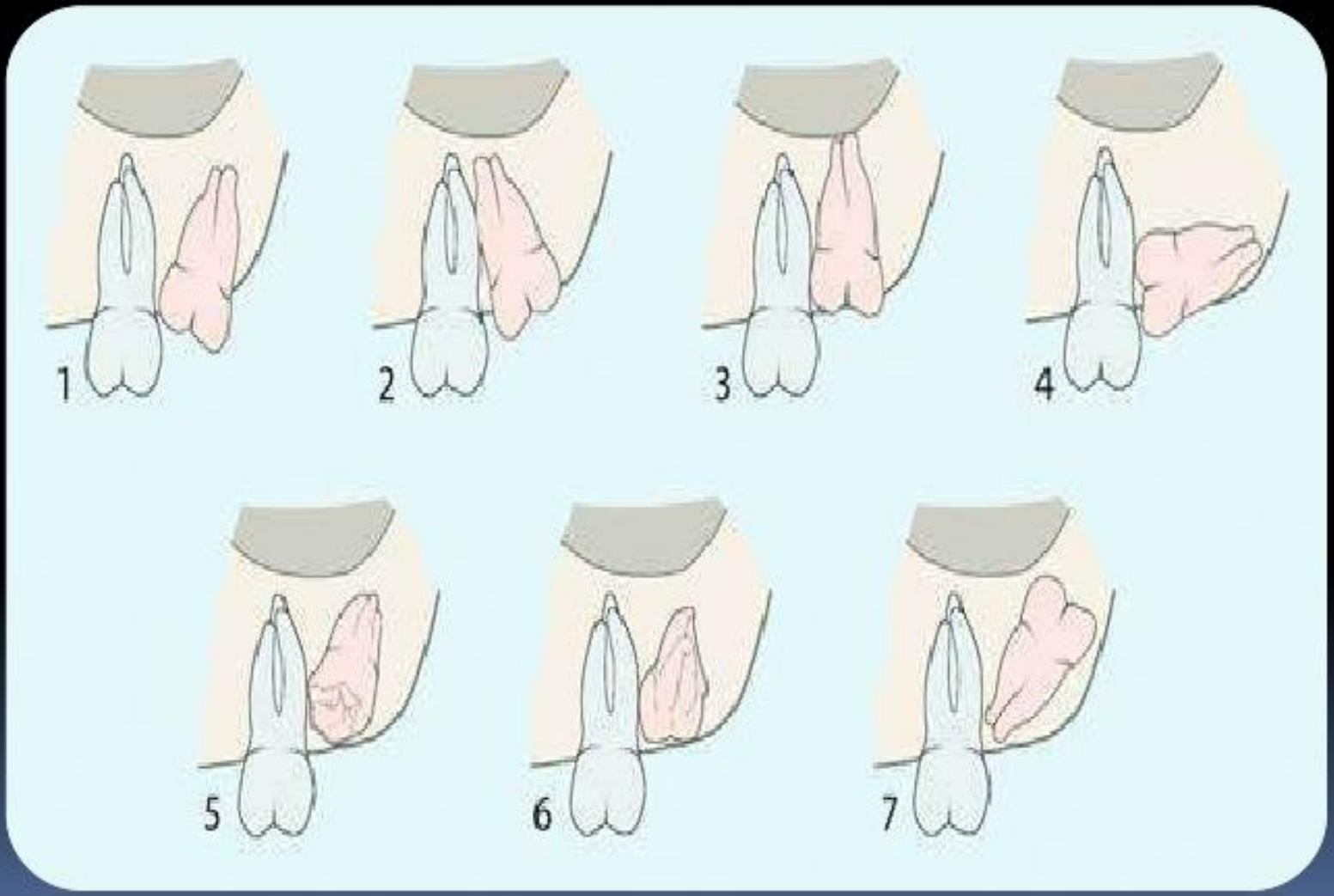
THE INDICATIONS FOR SURGICAL REMOVAL

- ✓When the tooth associated with a cyst formation .
- ✓Prosthetic & orthodontic demands
- ✓Pericoronitis , although it is rarely occnred in maxillary third molar .

THE CLASSIFICATION

1-ANGULATION (according to Archer 1975)

- Mesioangular
- Distoangular
- Horizontal
- Vertical
- Transverse (bucco- lingual)


1
2
3
4
5
6
7

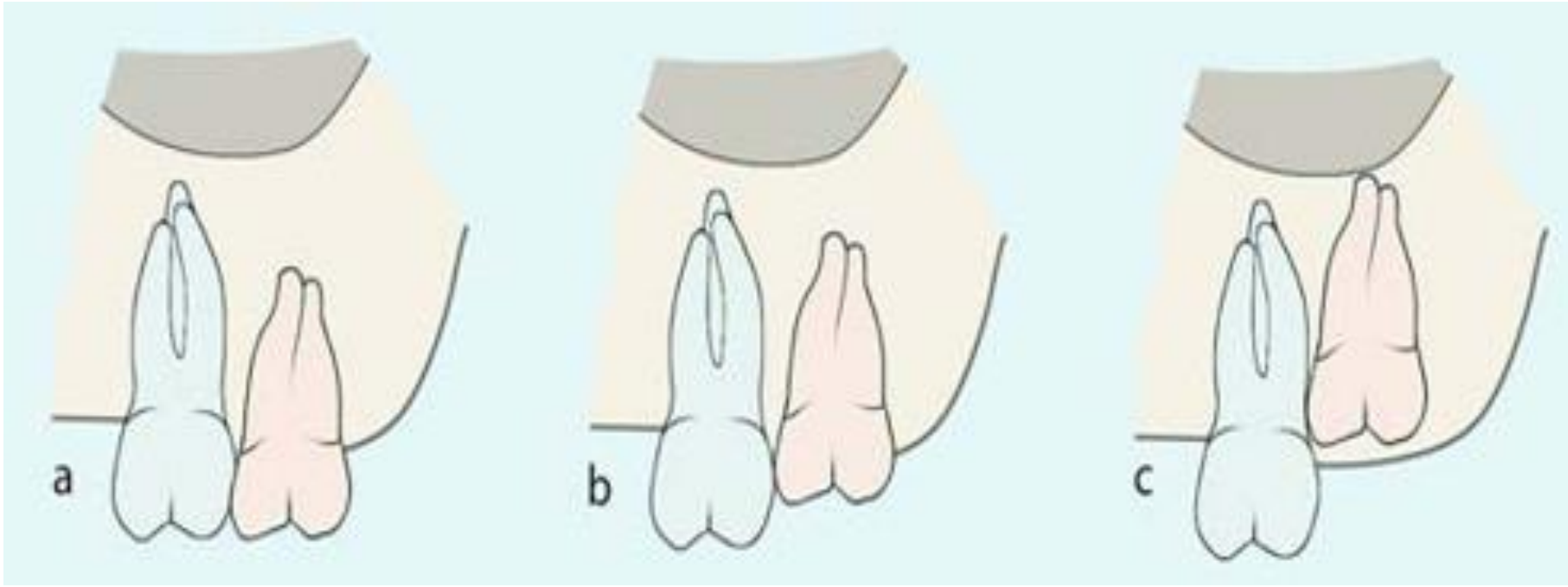
2- DEPTH OF IMPACTION (According to pell & gregory)

- **Class A :** The occlussal surface of the third molar at the same level with that of the second molar .

- **Class B :** The occlussal surface of the third molar is located between the occlussal plane and cervical line of the second molar .

- **Class C :** The occlussal plane of the third molar located deep to crvical line of the second molar .

N.B. Impacted teeth belonging to this category (Class C) are very difficult cases, because their extraction entails the removal of large amounts of bone, limited access, and there is a risk of displacing the impacted tooth into the maxillary sinus


a
b
c

THE RADIOGRAPHIC EXAMINATION

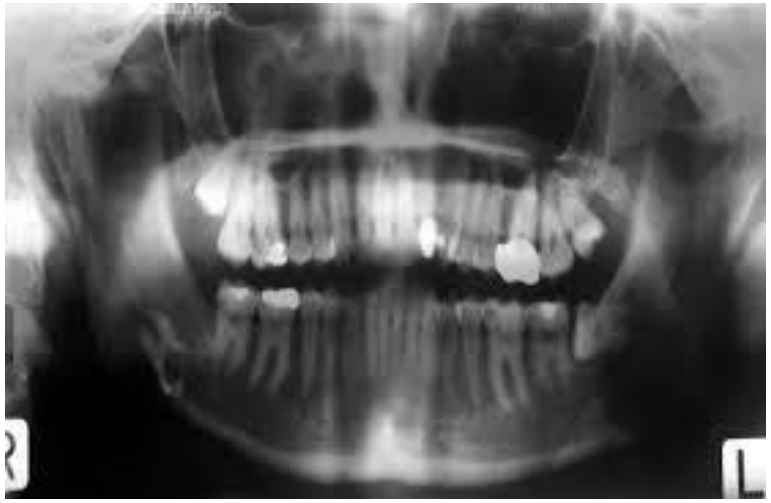
- Periapical film
- O . P . G . View
- CT. scan

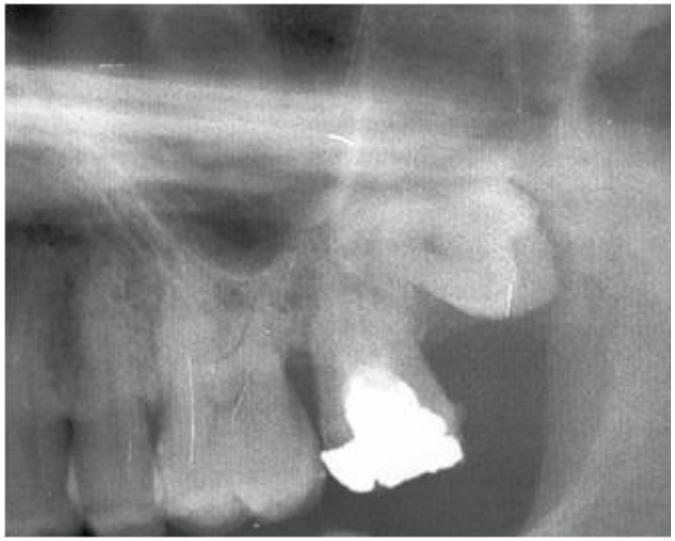
- In radiograph we notice the following :

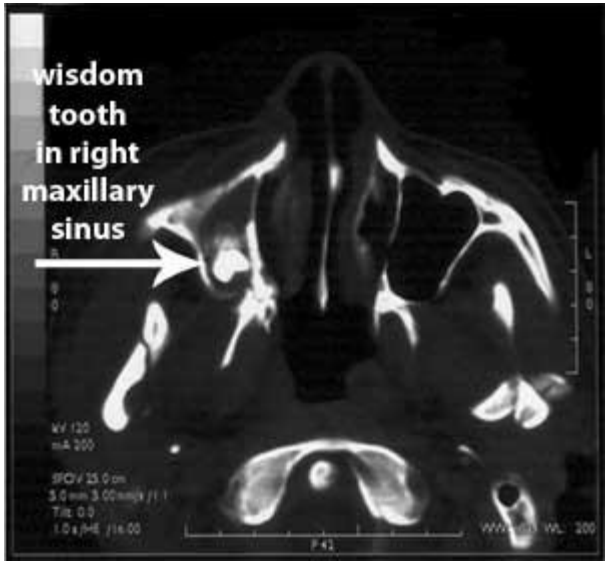
  a- the angulation

  b- the depth of impaction

  c- the relation of the impacted tooth with the maxillary sinus . d- presence of any pathological lession such as cyst or tumour e-shape and length of the roots.



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tooth
in right
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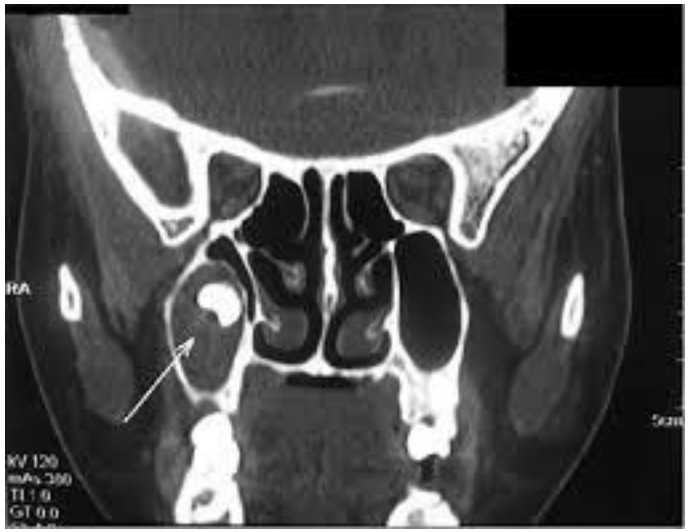


THE CLINICAL EXAMINATION :

WE CHECK THE FOLLOWING :

A- THE MOUTH OPENING
B- THE ORAL HYEGEN
C- PRESENCE OF PERICORONITIS (ALTHOUGH IT IS RARELY OCCURED IN MAXILLARY 3RD MOLAR) .

THE SURGICAL TECHNIQUE

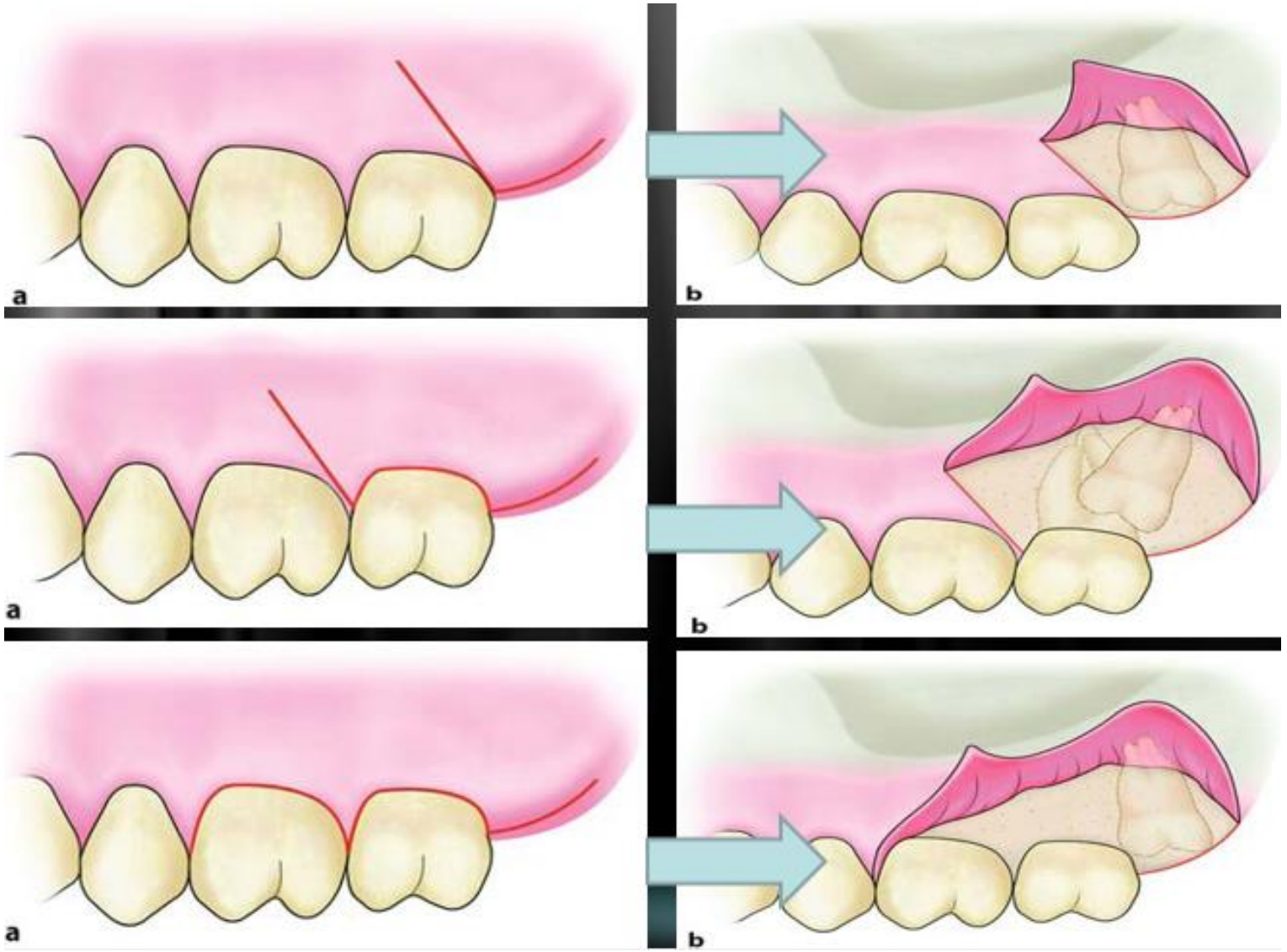
1- The flap design:

A- The envelope flap

this type is mostly used in maxillary 3rd molar . it extends posteriorly from the distobucc line angle of the second molar and anteriorly to the mesial side of the first molar

B- The triangular flap (tow sided flap)

this type is used when a greater access is needed usually the vertical incision is done at the distal side of the second molar, but in a deeply impacted tooth . the releasing incision is done from the mesial side of the second molar or even at the distal side of the first molar


a
b
a
b
a
b

2-The reflection and retraction of the flap

3-The bone removal

Often, after reflection of the flap, part of the crown of the impacted tooth is visible or there is bone protuberance over the crown. Because the bone in this case is thin and spongy, it may be removed from the buccal surface using a sharp instrument. If the buccal bone is dense and thick, then its removal is achieved using a surgical bur until the entire crown of the impacted tooth and part of its roots are exposed. Because extraction of the tooth in segments is not indicated, sufficient space must be created around its crown to be able to luxate the tooth.

4- Tooth sectioning

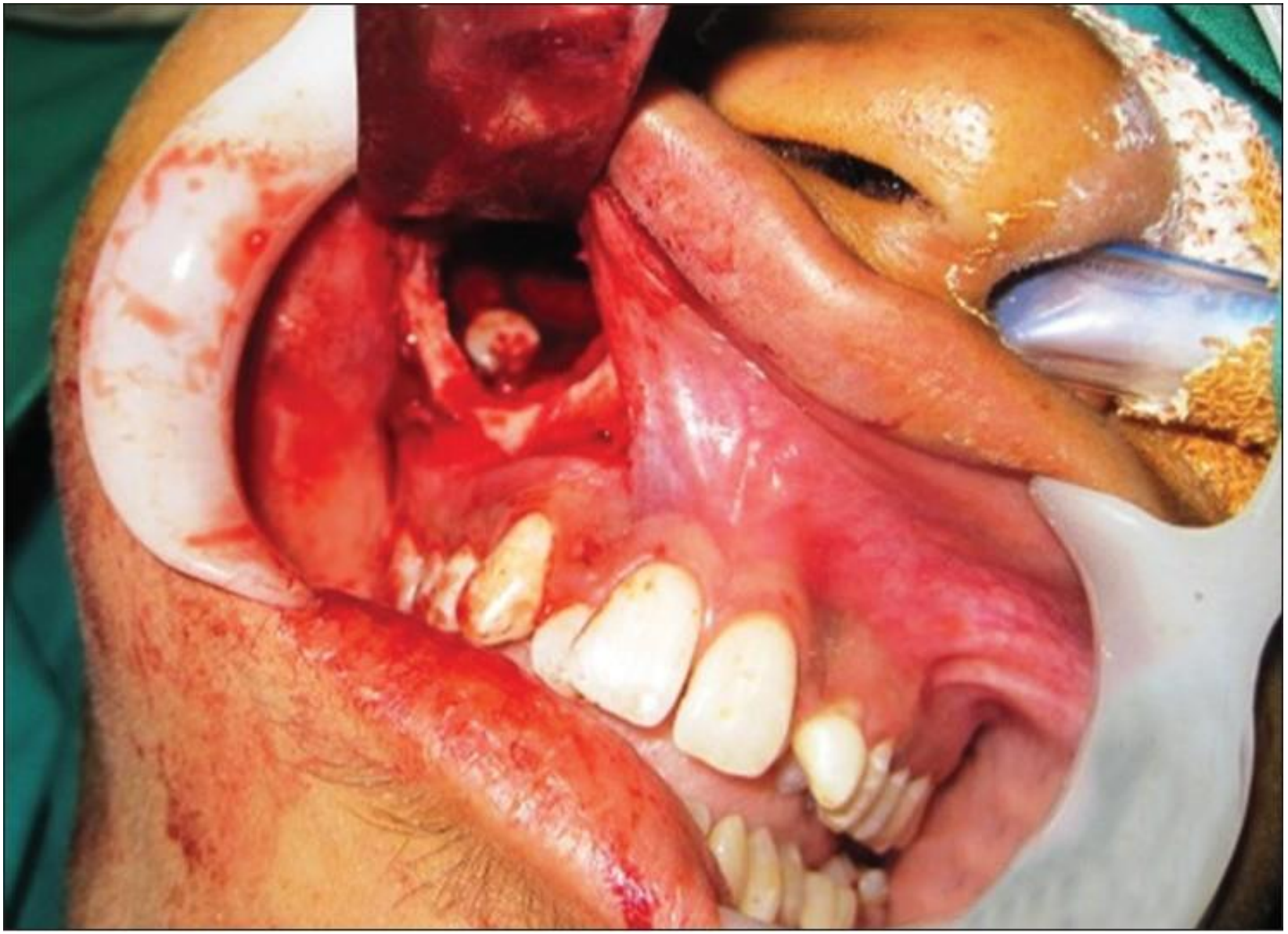
maxillary third molars are rarely need sectioning , because the overlying bone is relatively elastic.

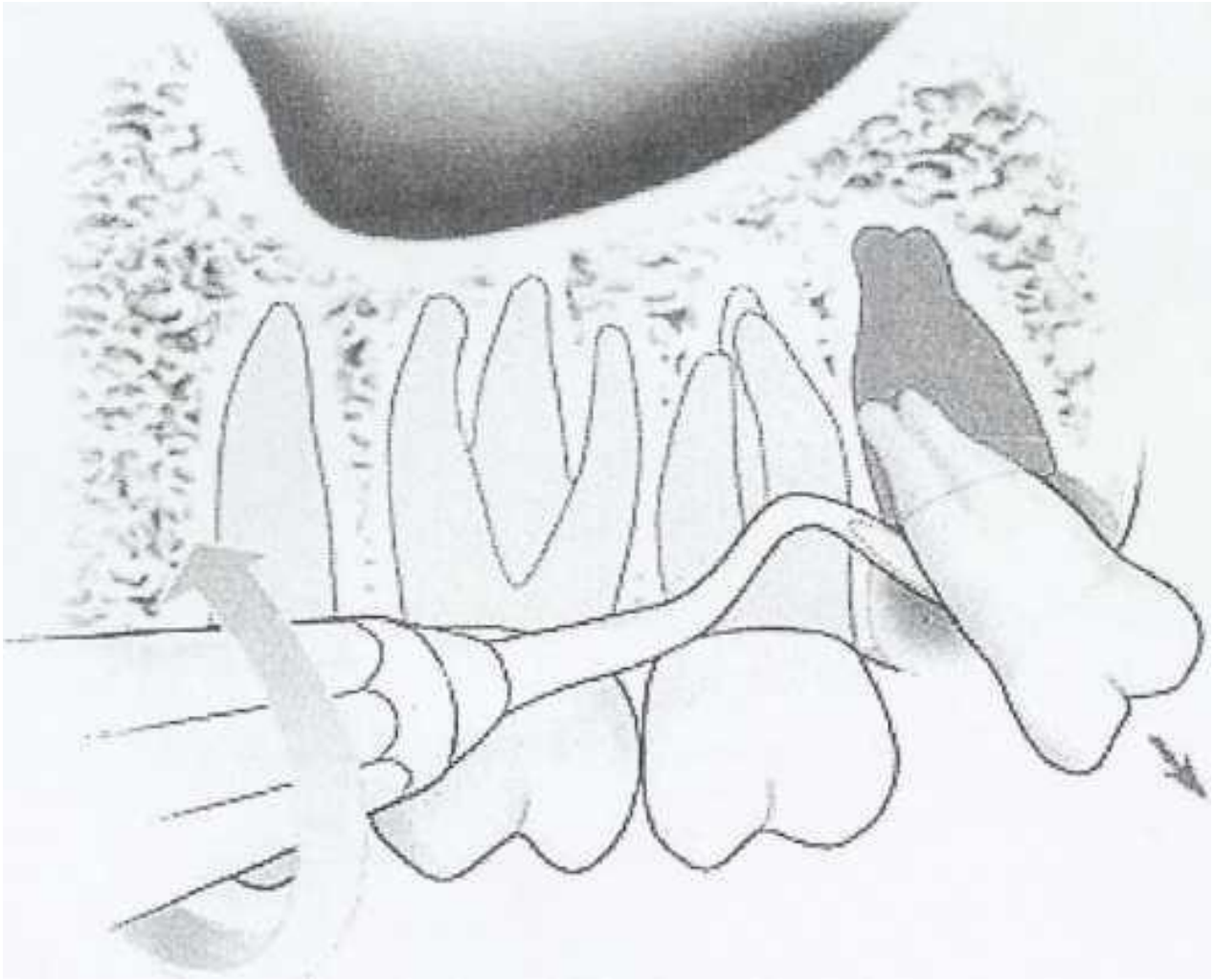
5-Tooth removal

the tooth is delivered by using a straight or double-angled elevator on the mesial aspect of the tooth, always buccally , the tooth is luxated carefully, posteriorly, outwards and downwards .

6- Toilet of the surgical area to remove any tooth or bone fragment by irrigation with normal saline.

7 - Suturing of the surgical incision to obtain a healing of primary intention .






Take a
Break
6 2 11 7 8 7 12 4 9 10 3 5

THE IMPACTED MXILLARY CANINE

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.

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 impacted canine presents in five basic positions as follows:

- Palatal localization
- Palatal localization of crown and labial localization of root
- Labial localization of crown and palatal localization of root
- Labial localization
- Ectopic positions

THE LOCALIZATION OF THE IMPACTED CANINE

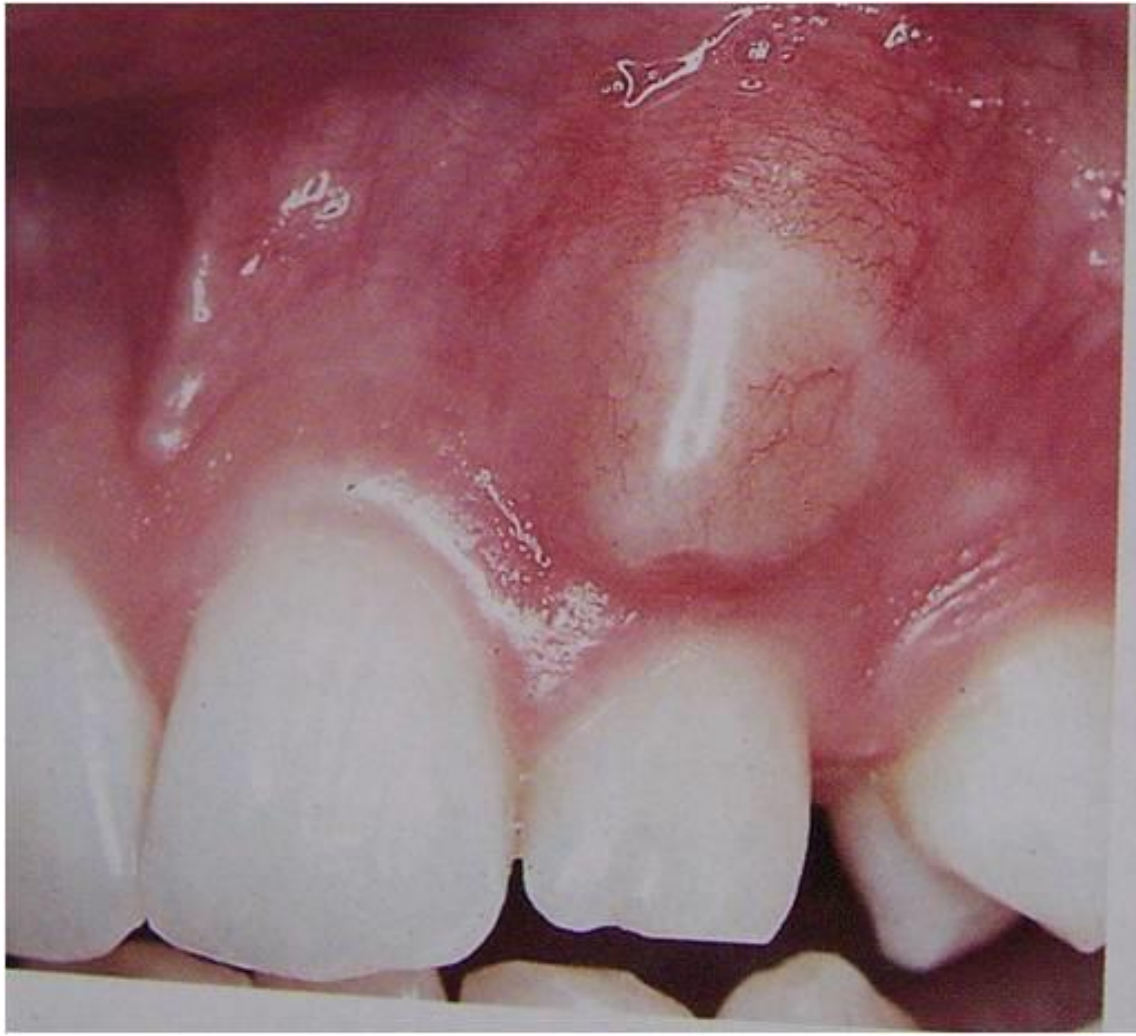
CLINICALLY

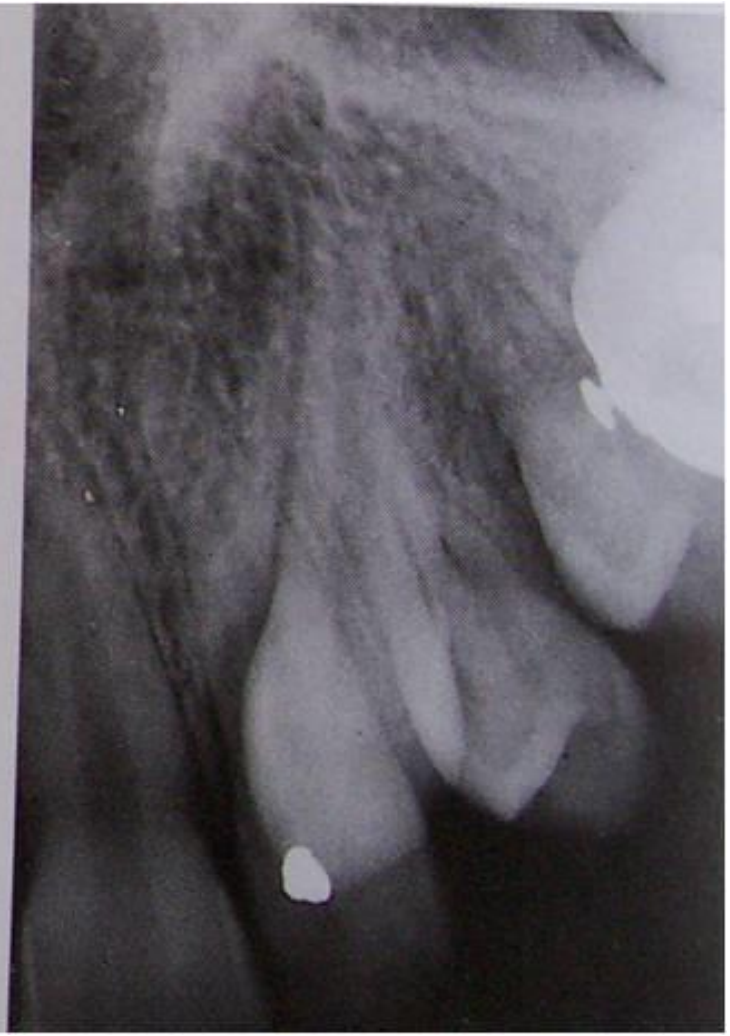
By inspection and palpation of the alveolar bone buccally and palatally For the presence of a bulges which indicates the presence of impacted canine.

RADIOGRAPHICALLY

The radiographs used involve :

- Parallex technique
- 0 .P .G . View
- Occlusal vertex view
- CT scan which gives a full details about the direction nd depth of the impacted tooth .





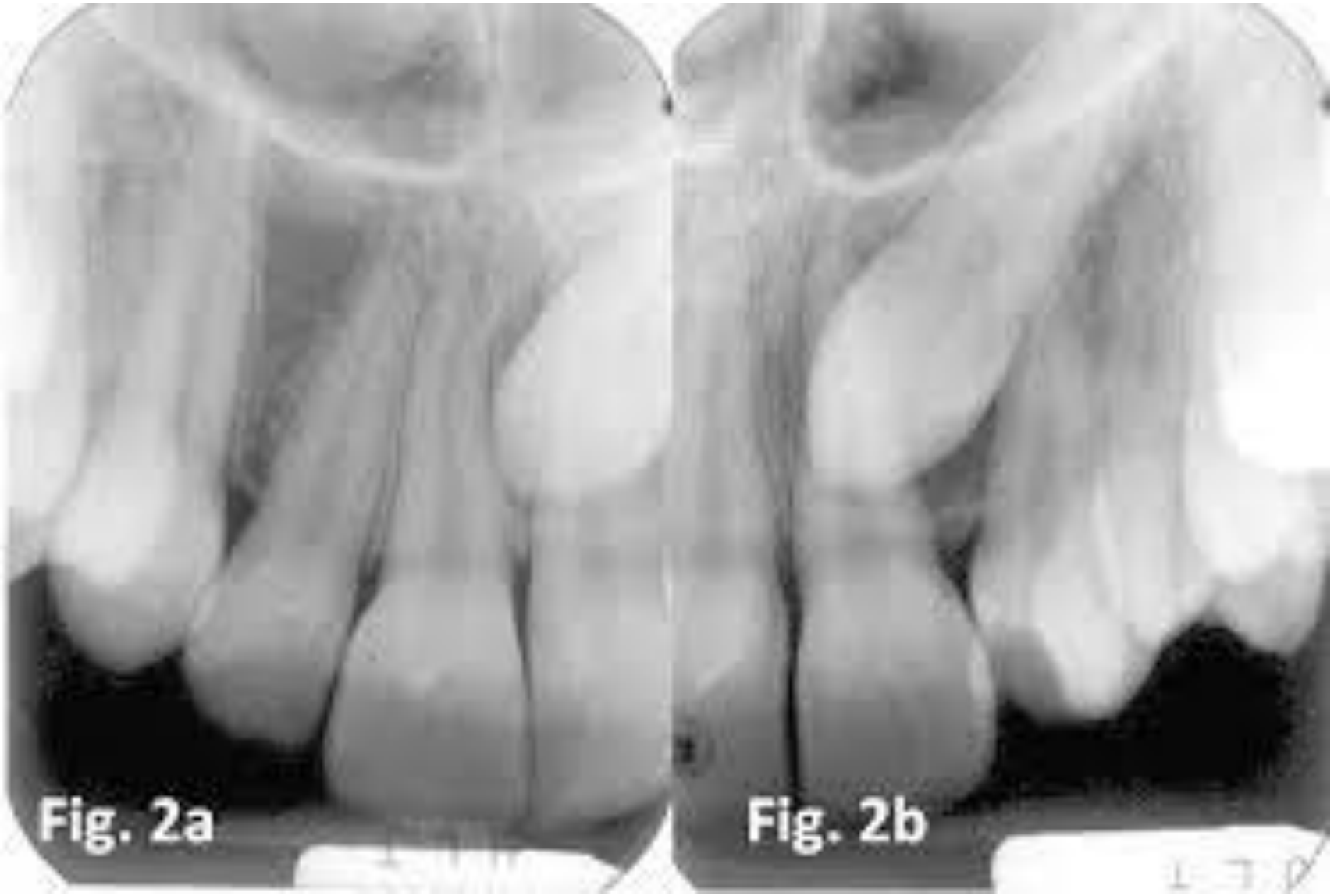
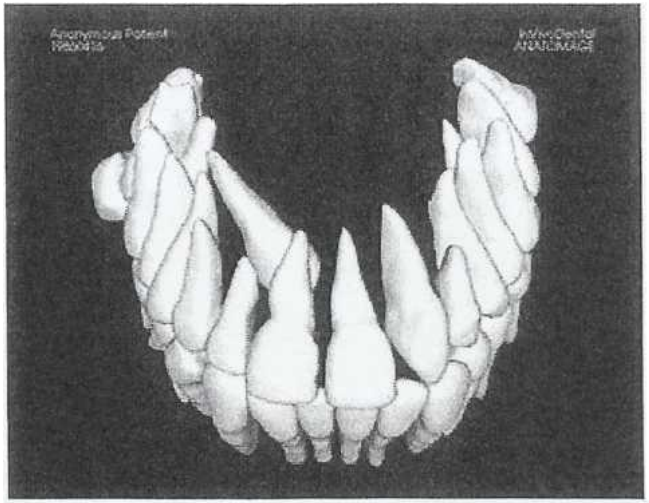


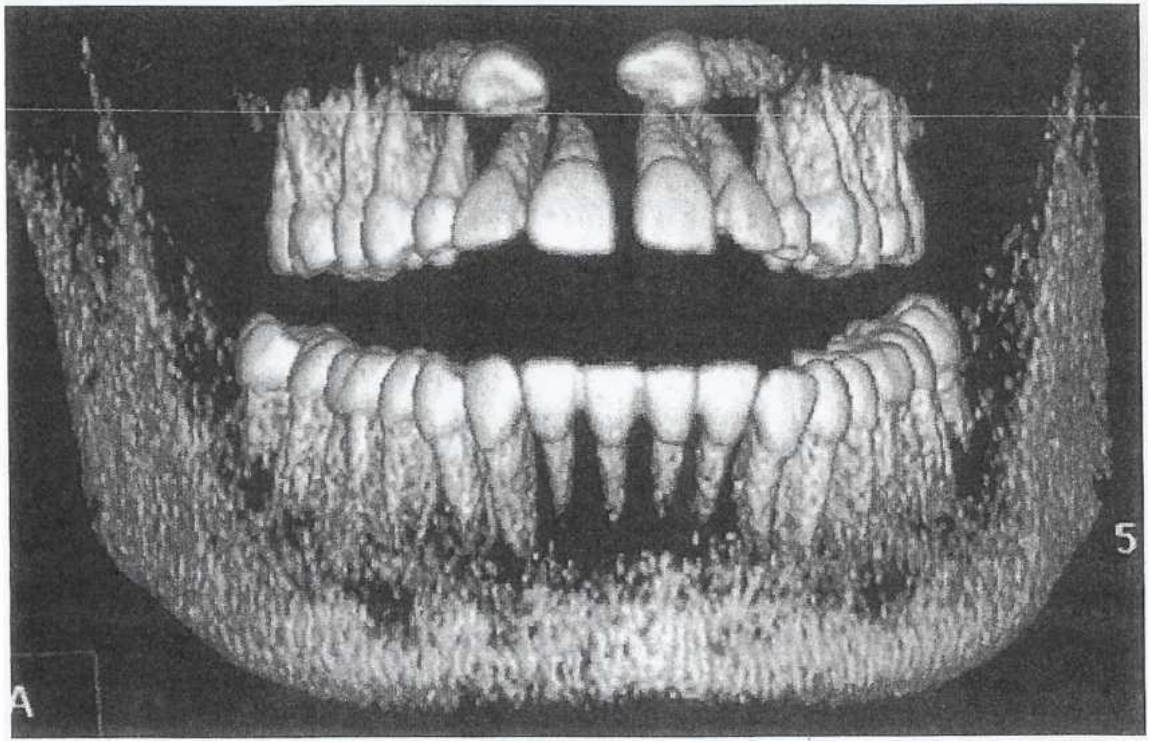
Fig. 2a
Fig. 2b

NOTE : in order to localize the impacted canine correctly we have to take two radiographic views in two planes , one perpendicular to the other, and the most widely used views are :

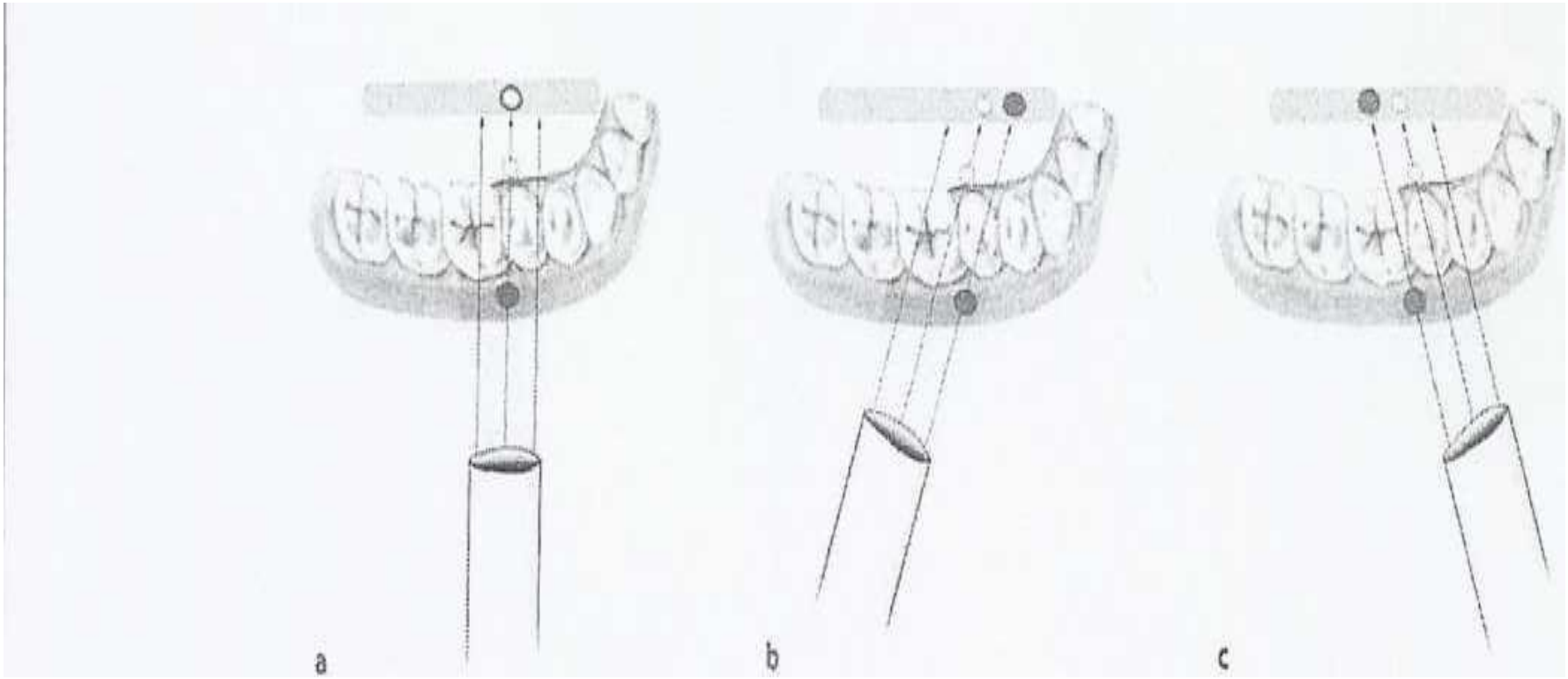
❖**O.P.G. or Periapical view** which shows the mesio- distal position and the depth of the impacted canine .

❖**Occlussal vertex view** , which shows the bucco- palatal position of the tooth


Anonymous Patient
InVivoDental
ANATOMAGE


5
A

Diagrammatic illustration of the radiographic method of localizing the buccal or lingual position of impacted teeth. Tooth movement depends on the proximal or distal shifting of the x-ray beam with regard to the initial position of the radiograph . the impacted tooth will seem to move in the same direction as the tube head when it is found palatally or lingually, and in the opposite direction compared to the tube head when it is found buccally .



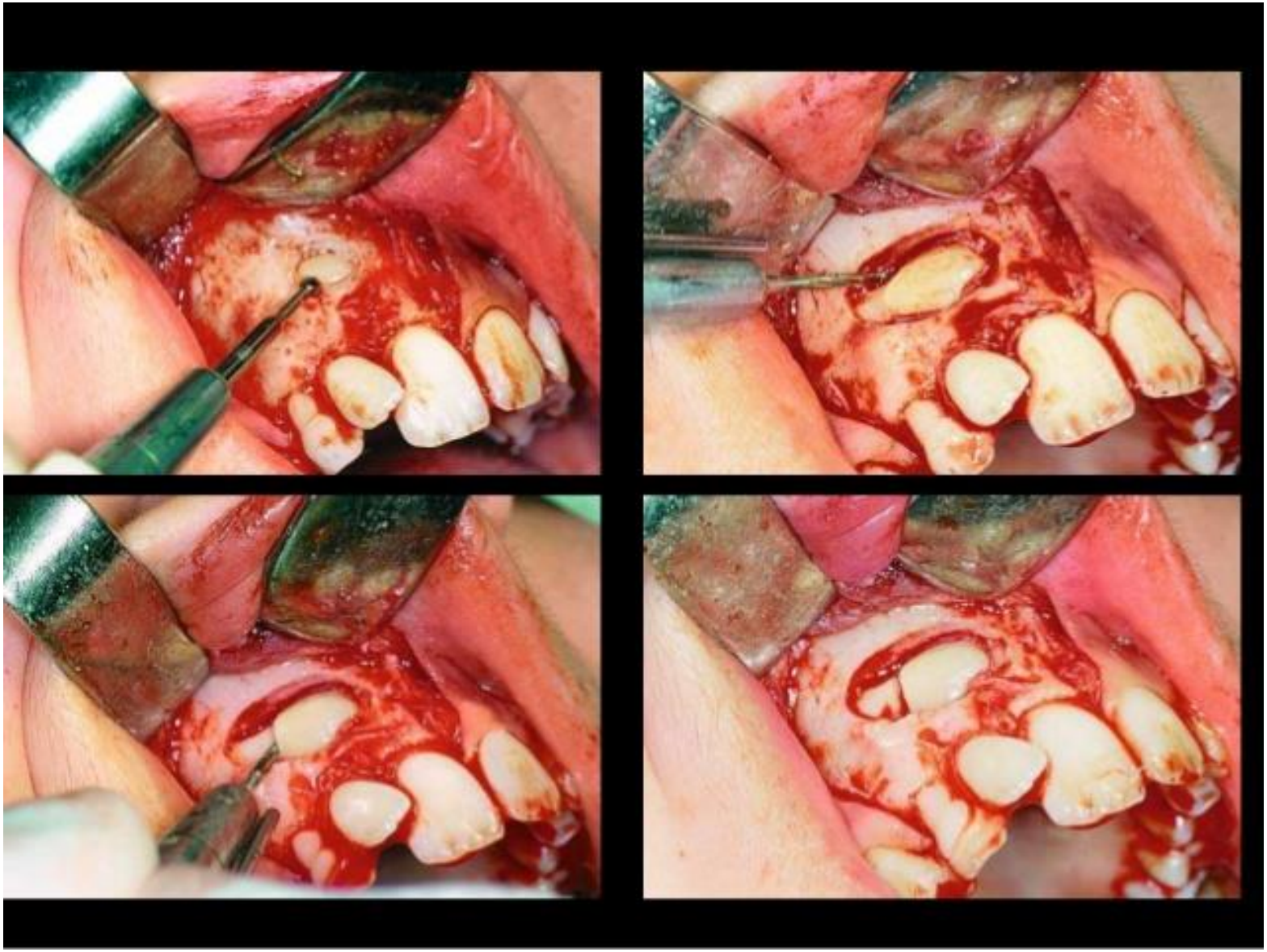
THE LINES OF TREATEMENT OF THE IMPACTED CANINE

- ✓ Surgical exposure for orthodontic treatment
- ✓ Surgical extraction
- ✓ Transplantation of the tooth into its normal position when there is sufficient Space for the tooth.
- ✓ Leave the tooth in situ when its removal causing trauma to the adjacent Vital structures

THE SURGICAL EXTRACTION

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 re-Fleeted , the bone covering the tooth is removed using a round bug 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

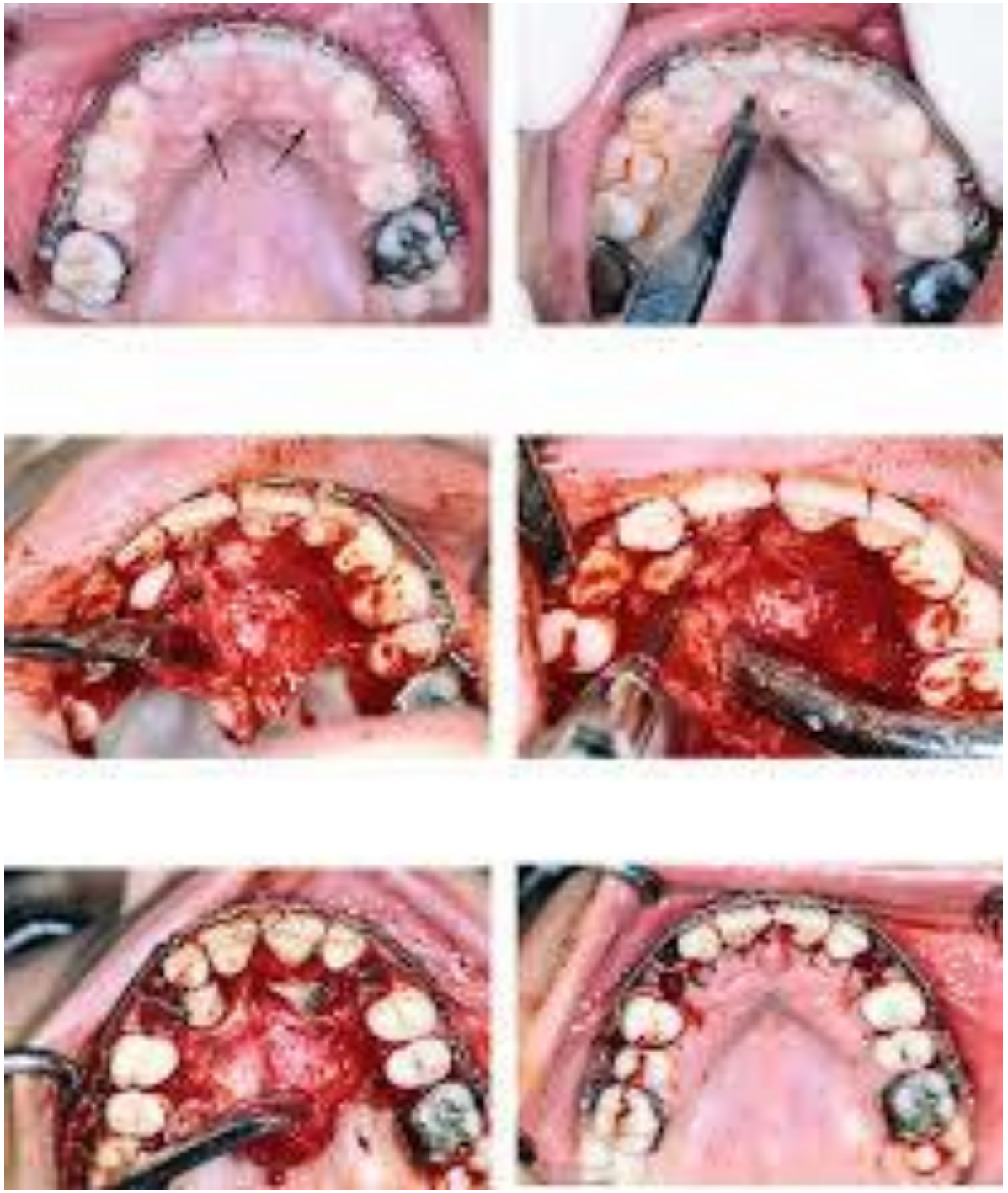
When the impacted tooth is positioned Palatally , the approach is achieved using a bilateral palatal flap. The incision for creation of the flap begins at the first or second ipsilateral premolar and, after continuing along the cervical lines of the teeth, ends at the first pro molar on the contralateral 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 .

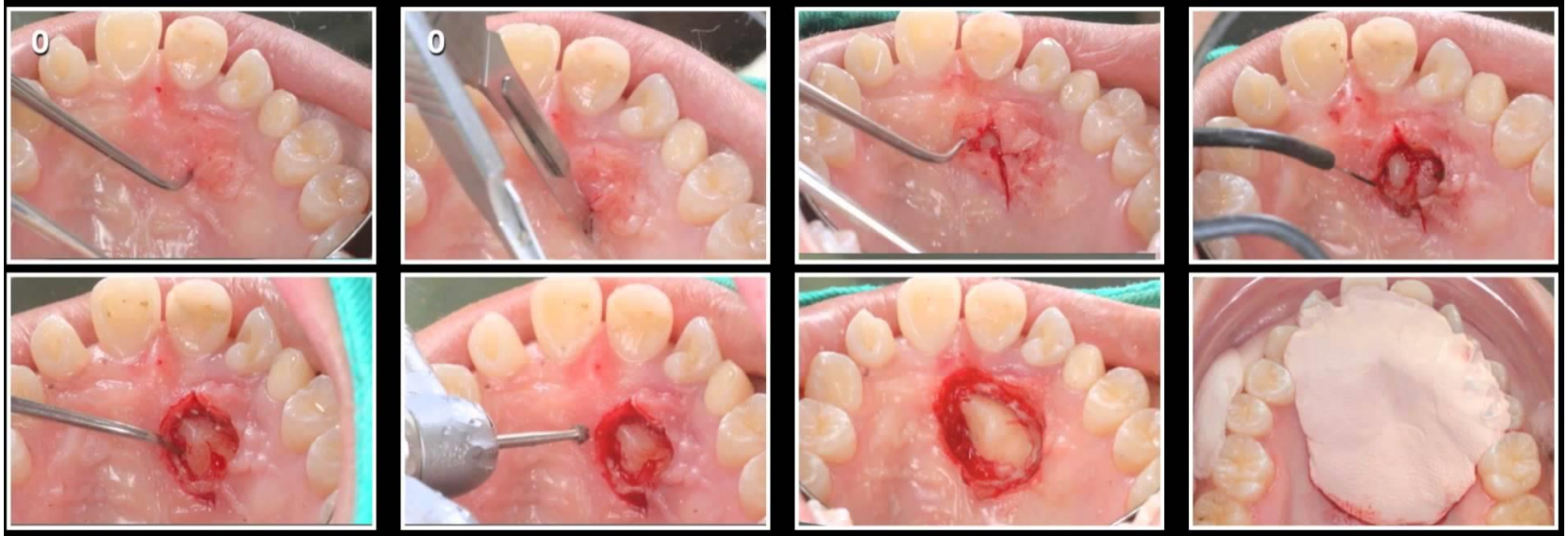
The crown is then removed, and, after using the round bur to create a purchase point on the root for placement of the angled elevators tip , the root is elevated from its bed .

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.



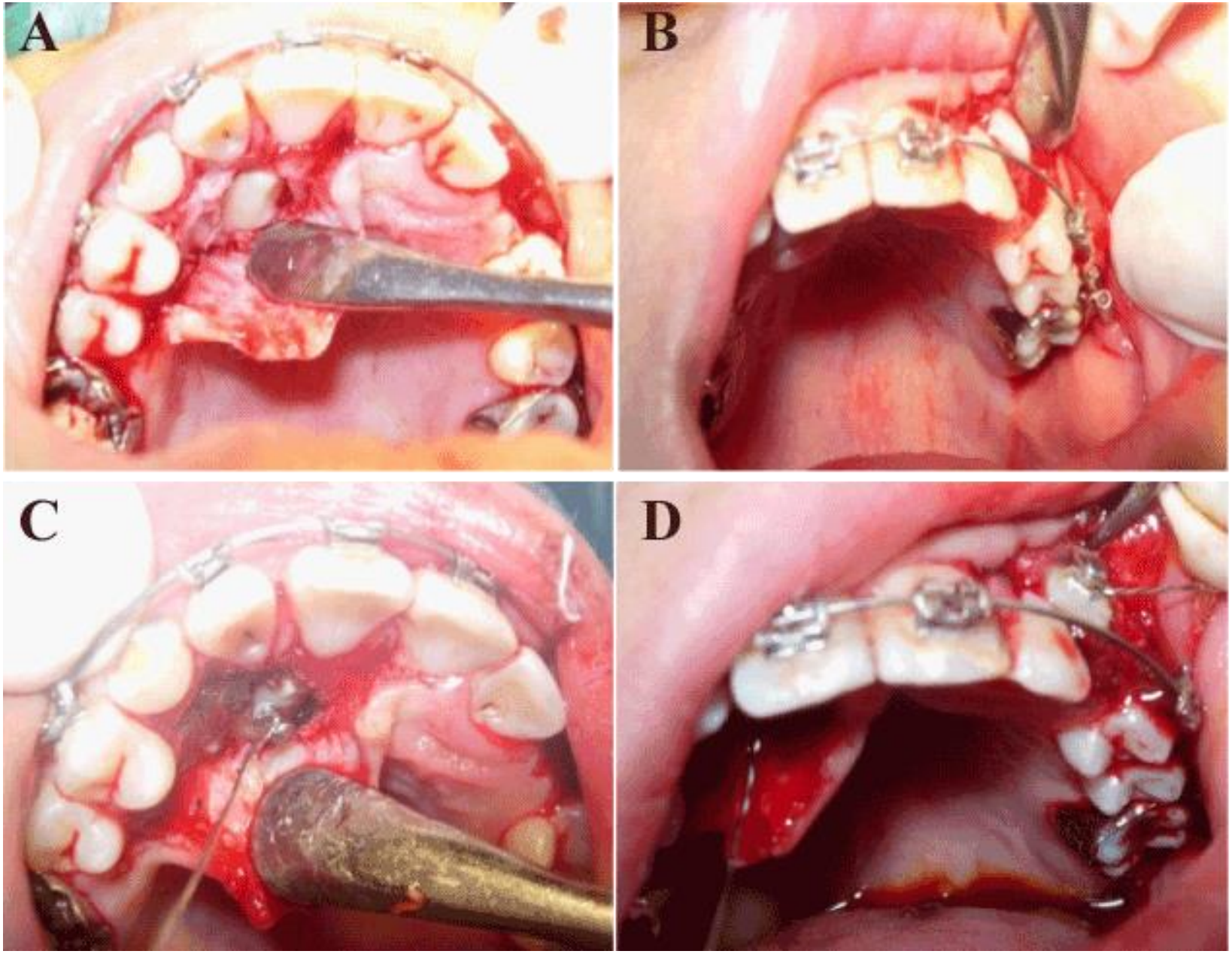
the surgical exposure for Orthodontic Treatment

In order to facilitate eruption of the impacted canine, a combination of surgical and orthodontic techniques is considered necessary. The procedure usually involves exposing the tooth, which is achieved by creating a flap and removing the bone over the tooth. If there are still deciduous teeth, supernumerary teeth, or odontomas , these are removed, and, after exposing a large part of the crown, orthodontic brackets are bonded to the crown, and the tooth is gradually aligned in its correct position. Sometimes the crowns of impacted teeth are covered only by soft tissue, in cases such as these, the soft tissue is removed using a scalpel or electrosurgical blade, thus creating a "window" at the crown of the tooth, which will help it to erupt, either on its own or with orthodontic treatment.



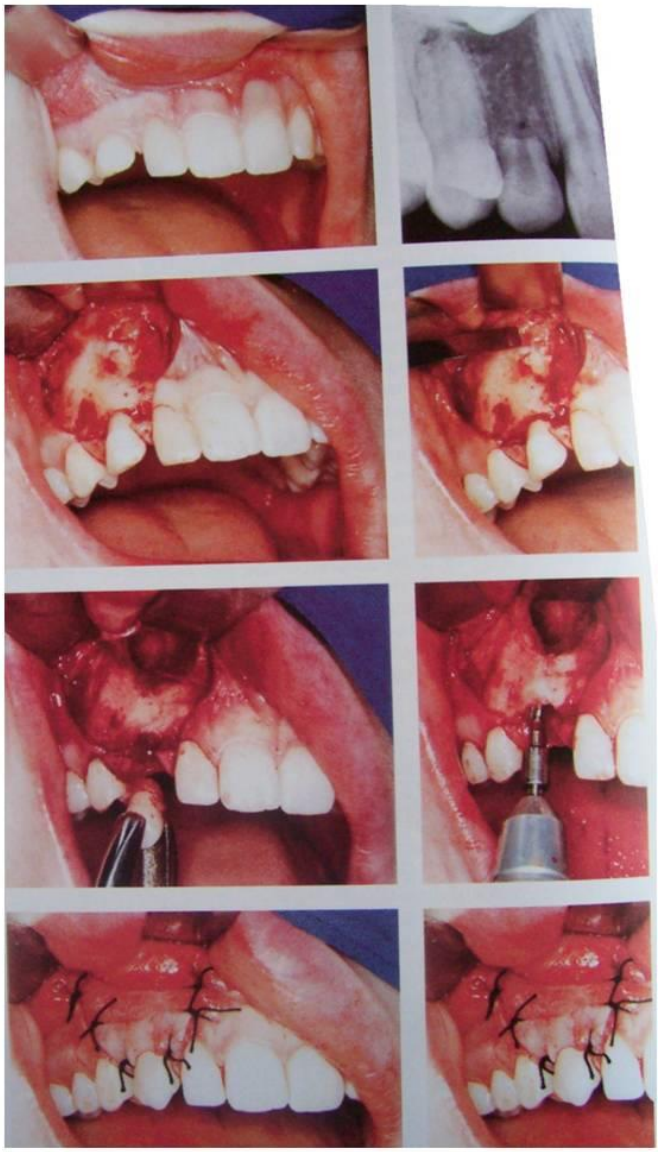
Impacted Canine with Palatal Position

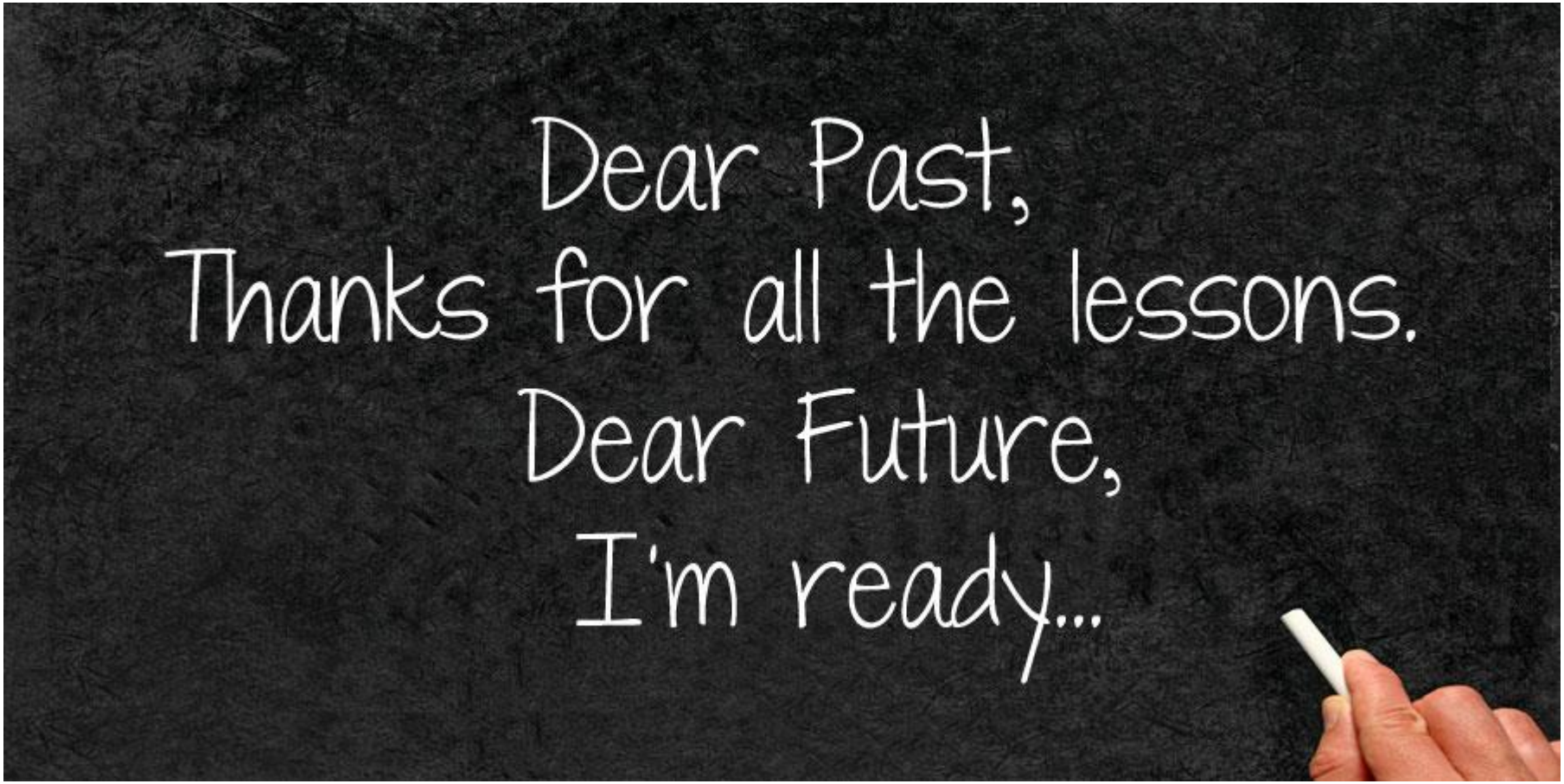
After removal of the deciduous teeth, a palatal flap is created, underneath which part of the bone covering the teeth is exposed. A round bur is then used to remove the bone covering the crowns and orthodontic brackets are placed for traction of the teeth into their normal position in the dental arch. The area is then irrigated with saline solution and the flap is closed with interrupted sutures


A
B
C
D

Transplantation of Labially Positioned Canine

- Transplantation of maxillary canine with labial position.
- Exposure of the crown
- Preparation of a new socket
- Splinting




Dear Past,
Thanks for all the lessons.
Dear Future,
I'm ready...

IMPACTED TEETH

Dr. Usama Aldaghir

Oral & maxillofacial surgeon

B.D.S. – C.A.B.M.S

The impacted tooth is that one which fail to erupt into the dental arch Within the expected time the impaction is due to:-

1- **dense overlying bone**

2- thick overlying soft tissue

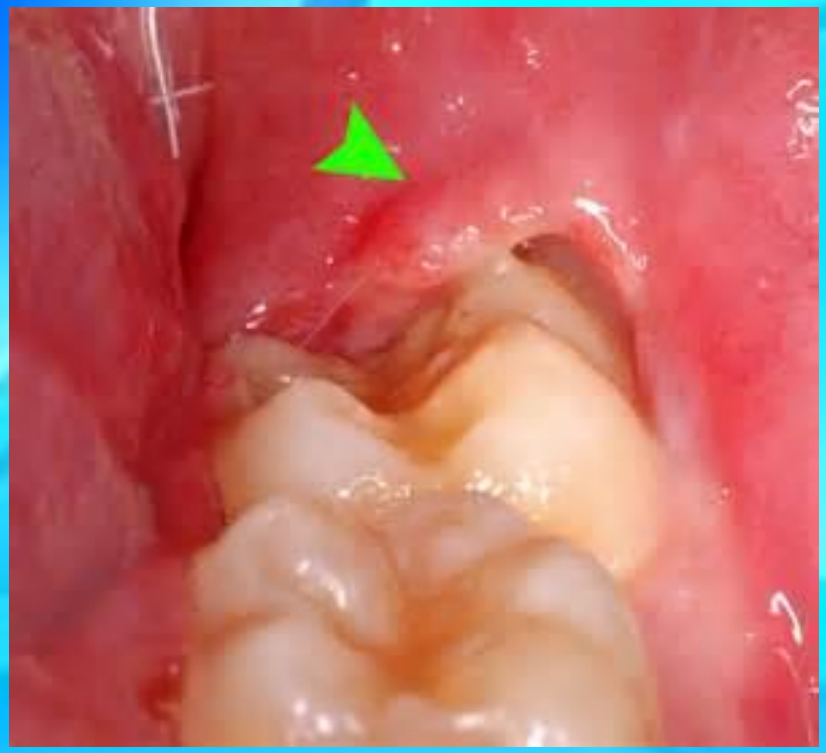
3- the tooth **angulation** is not favorable

4- the **adjacent tooth** might interfere with the eruption pathway.

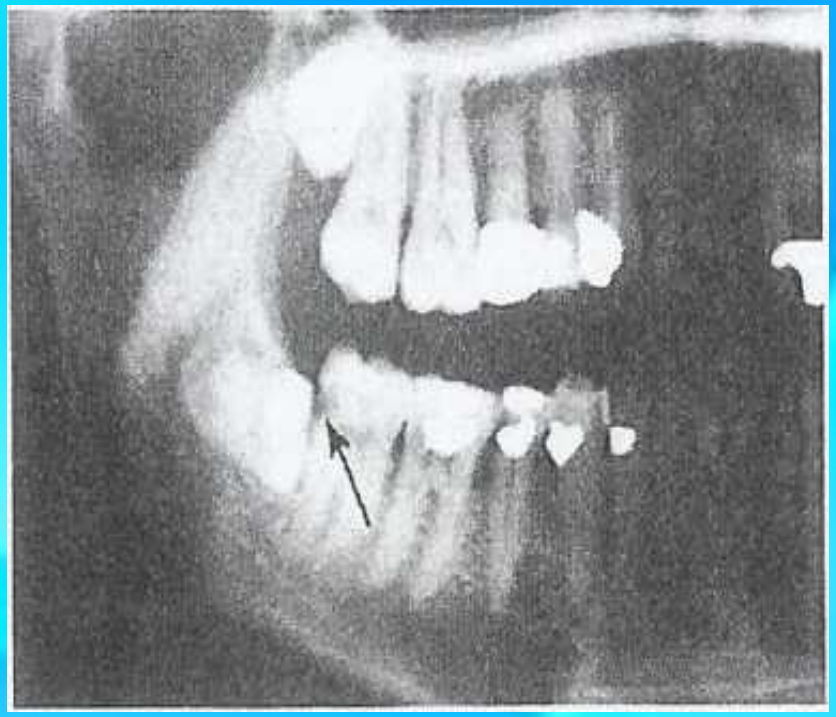
IMPACTED MANDIBULAR THIRD MOLAR

THE indications for removal of impacted mandibular third molar:-

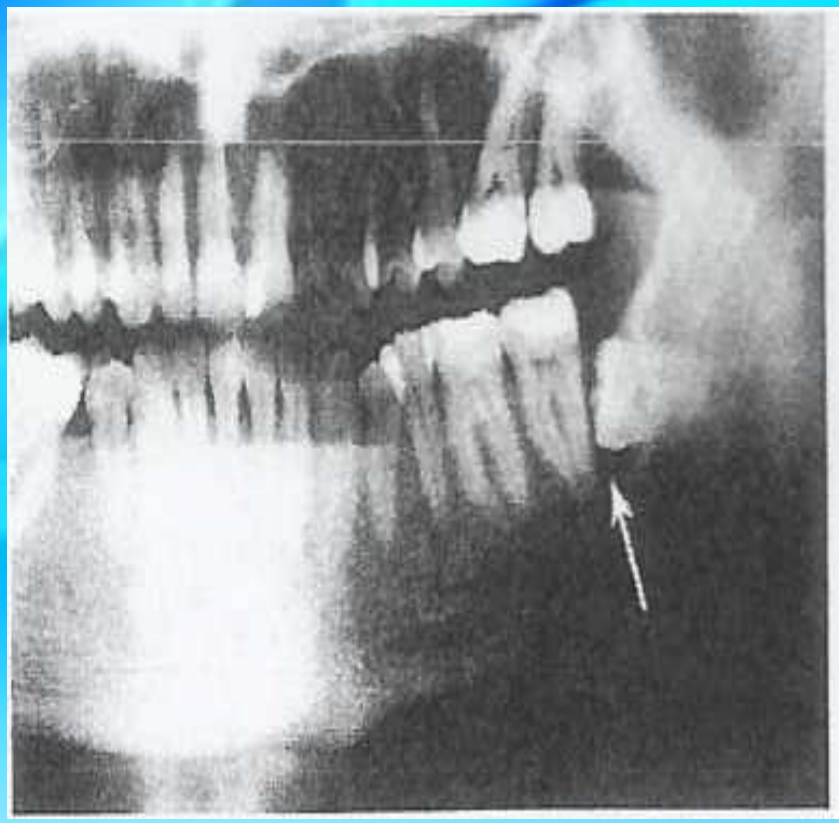
1) **Lack of space** in the jaw is probably the most common indication,
2) Impacted teeth with recurrent **pericoronitis.**
3) If Impacted tooth causes caries ; periodontal pocket; root resorption of **the Adjacent second molar** .
4) **Facilitation** of orthodontic treatment
5) Impacted tooth associated with **odontogenic tumor** or cyst.
6) For **prosthetic** purposes.
7) **Pain of unknown etiology.**
8) **Extraction before radiotherapy**.
9) Tooth in the **fracture line** of the mandible



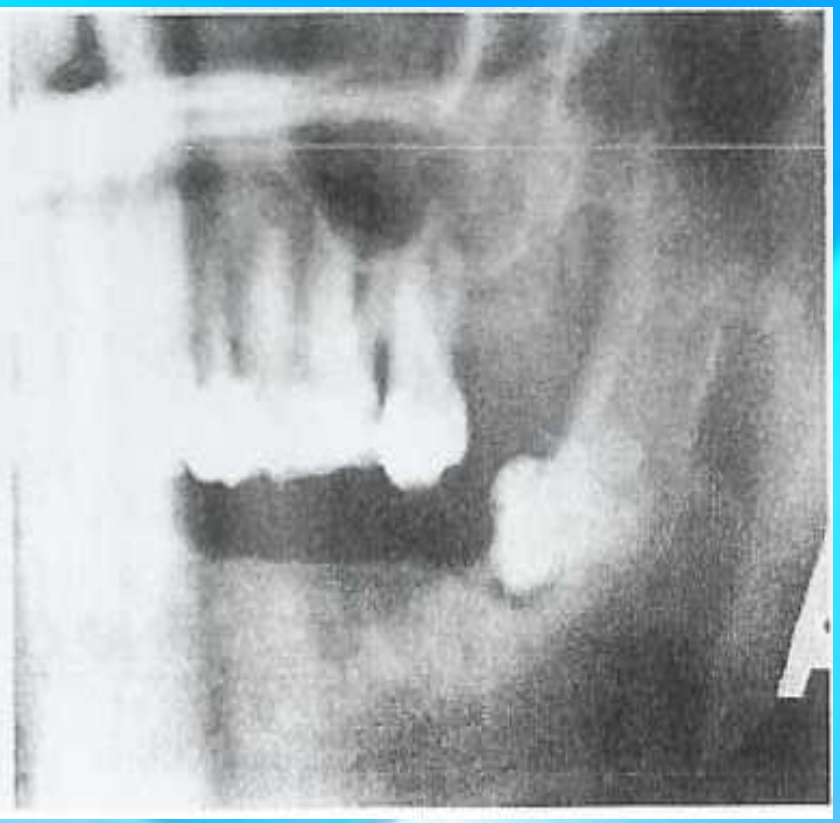
Clinical photograph. Characteristic swelling of the operculum due to constant biting from the antagonist



Caries on the distal surface of the second molar, caused by a semi-impacted mandibular third molar



Bone resorption at the distal surface of the root of a mandibular second molar, resulting in a periodontal pocket



Impacted mandibular third molar in edentulous, which erupted alter placement of a partial denture

Contraindications for Removal

1) **Sufficient space** for normal eruption
2) **Compromised health** status of the patient
3) Third molars **needed as abutment**.
4) **Asymptomatic impactions**, without pathology in patients over 30-35 years of age—where potential surgical trauma outweighs benefits from removal
5) **Patient declines surgery**
6) Probable excessive damage to adjacent structures ; such as inferior dental nerve, blood vessels, adjacent teeth , or previous constructed bridge.

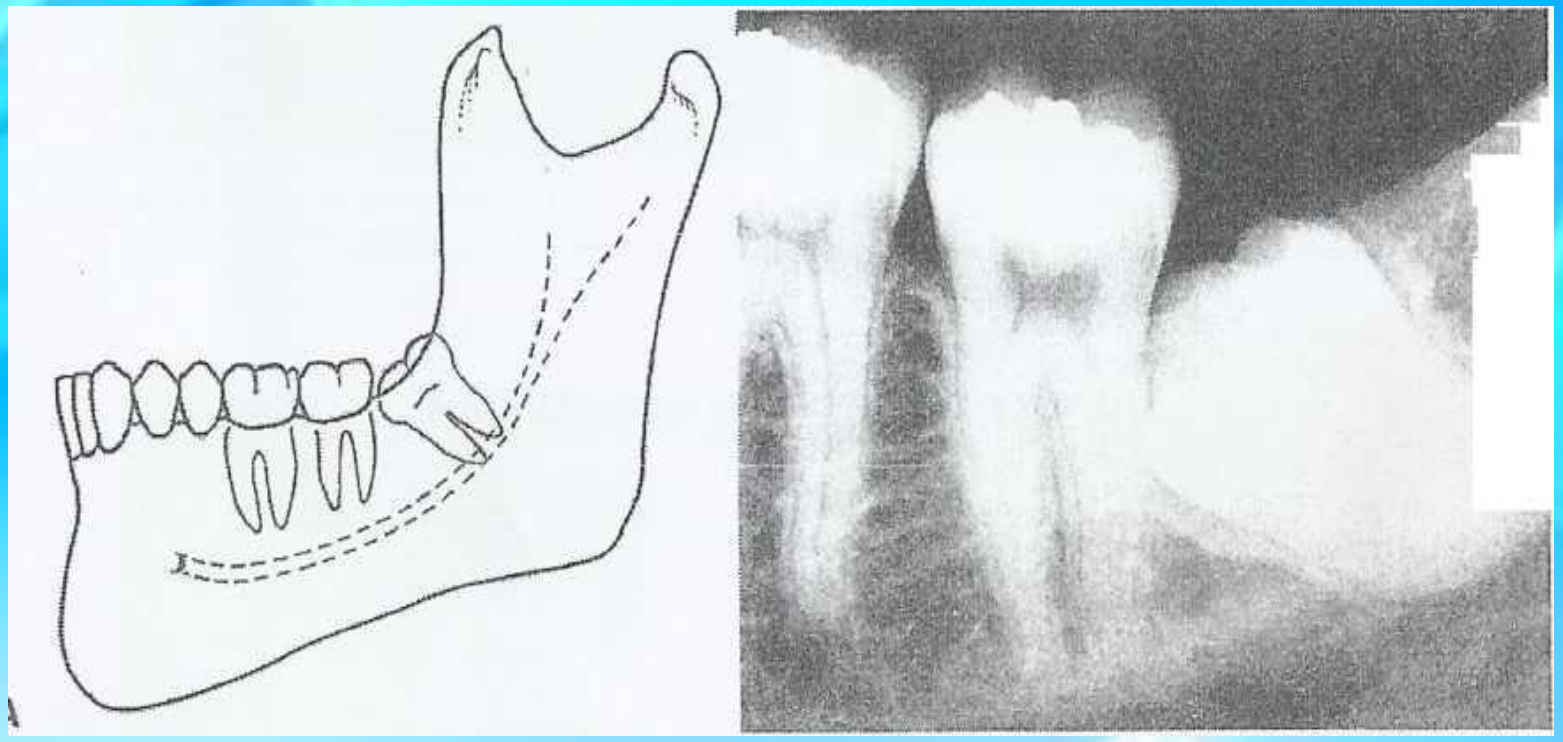
THE CLASSIFICATION SYSTEMS OF IMPACTED MANDIBULAR THIRD MOLAR.

They are classified according to:-

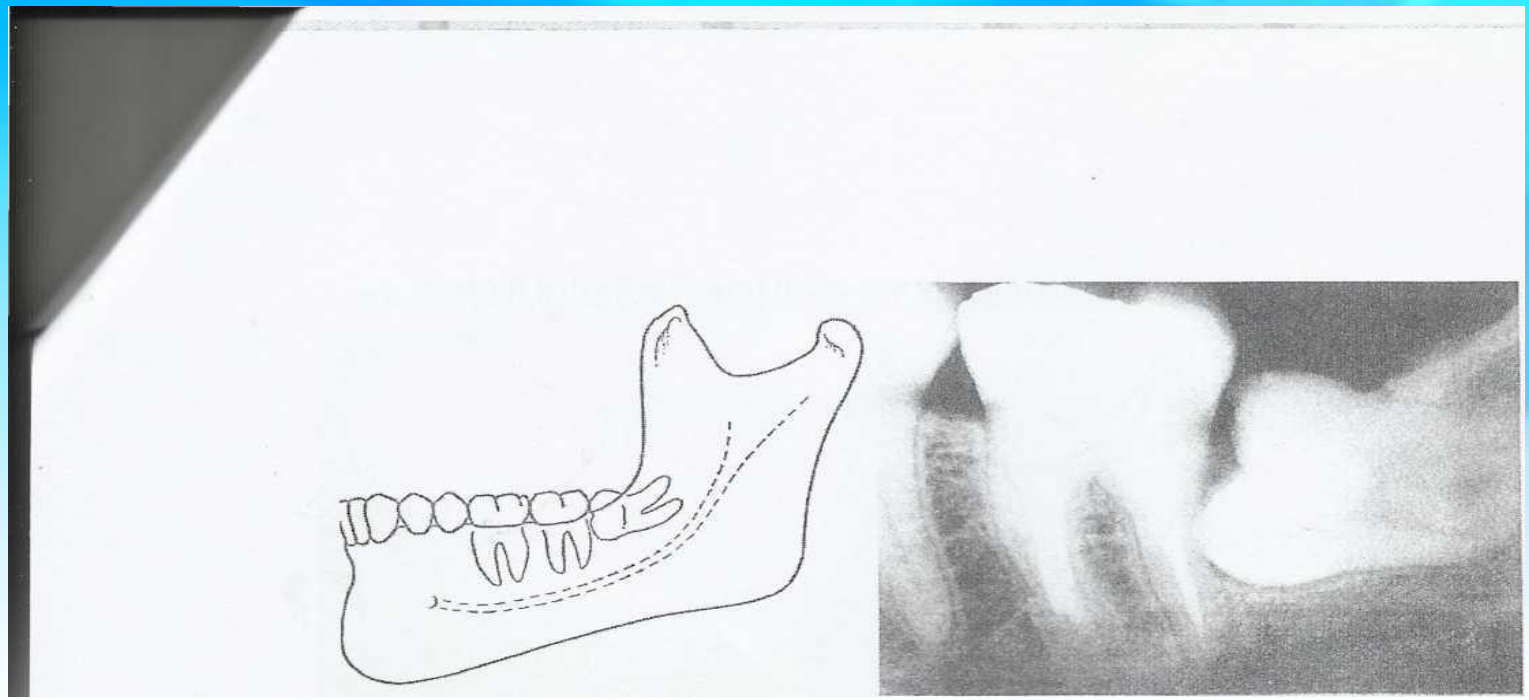
ANGULATION: (According to Winter)

This classification determines the angulation of **the long axis of the impacted third molar with respect to the long axis of the adjacent second molar**. This classification system provides an initial useful evaluation of the difficulty of extractions but is **not sufficient by itself** to define difficulty of molar removal fully.

- **Mesioangular** The mesio-angular impacted tooth is **tilted toward the second molar** in a mesial direction. This type of impaction is the **most commonly** seen, making up approximately **43%** of all impacted teeth.



- **Horizontal :** When **the long axis of the third molar** is **perpendicular to the second molar**, the impacted tooth is considered horizontal. This type of impaction is usually considered more difficult to remove than the mesioangular impaction. Horizontal impactions occur less frequently, being seen in approximately **3%** of all mandibular impactions



- **Vertical :**In the vertical impaction the long axis of the impacted tooth runs **parallel** to the long axis of the second molar. This impaction occurs with the second greatest frequency, accounting for approximately **38%** of all impactions .

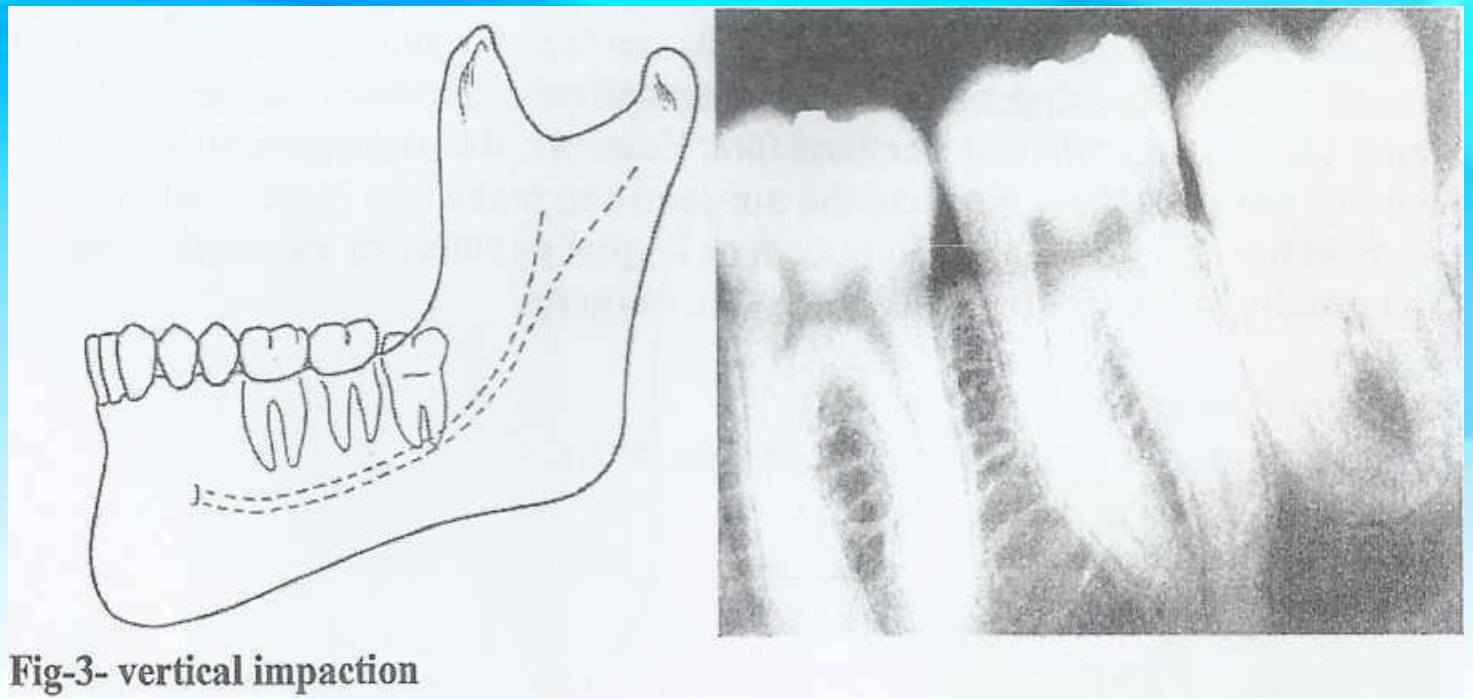


Fig-3- vertical impaction

Distoangular : the distoangular Impaction Is the tooth with the **most difficult** angulations for removal . In the distoangular impaction the long axis of the third molar is distally or posteriorly angled **away from the second molar**. This Impaction Is the most difficult to remove because the tooth has a withdrawal pathway that runs into the mandibular ramus , and Its removal requires significant surgical intervention. Distoangular impactions occur uncommonly and account for only approximately **6%** of all impacted third molars.

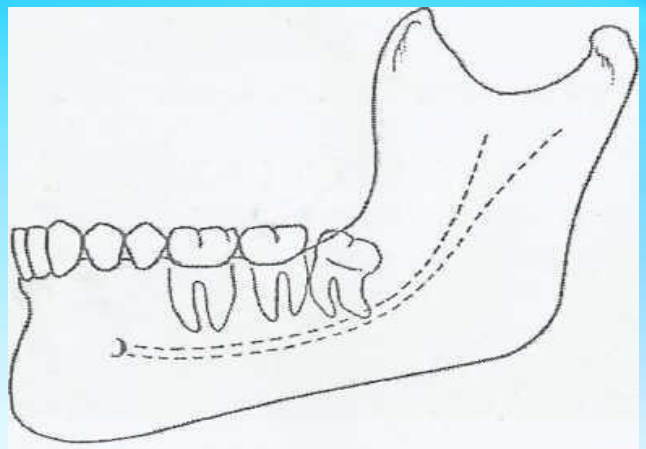
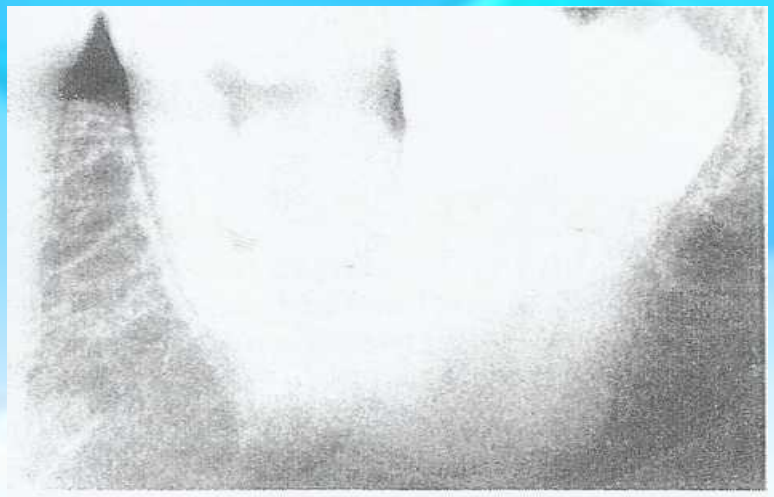
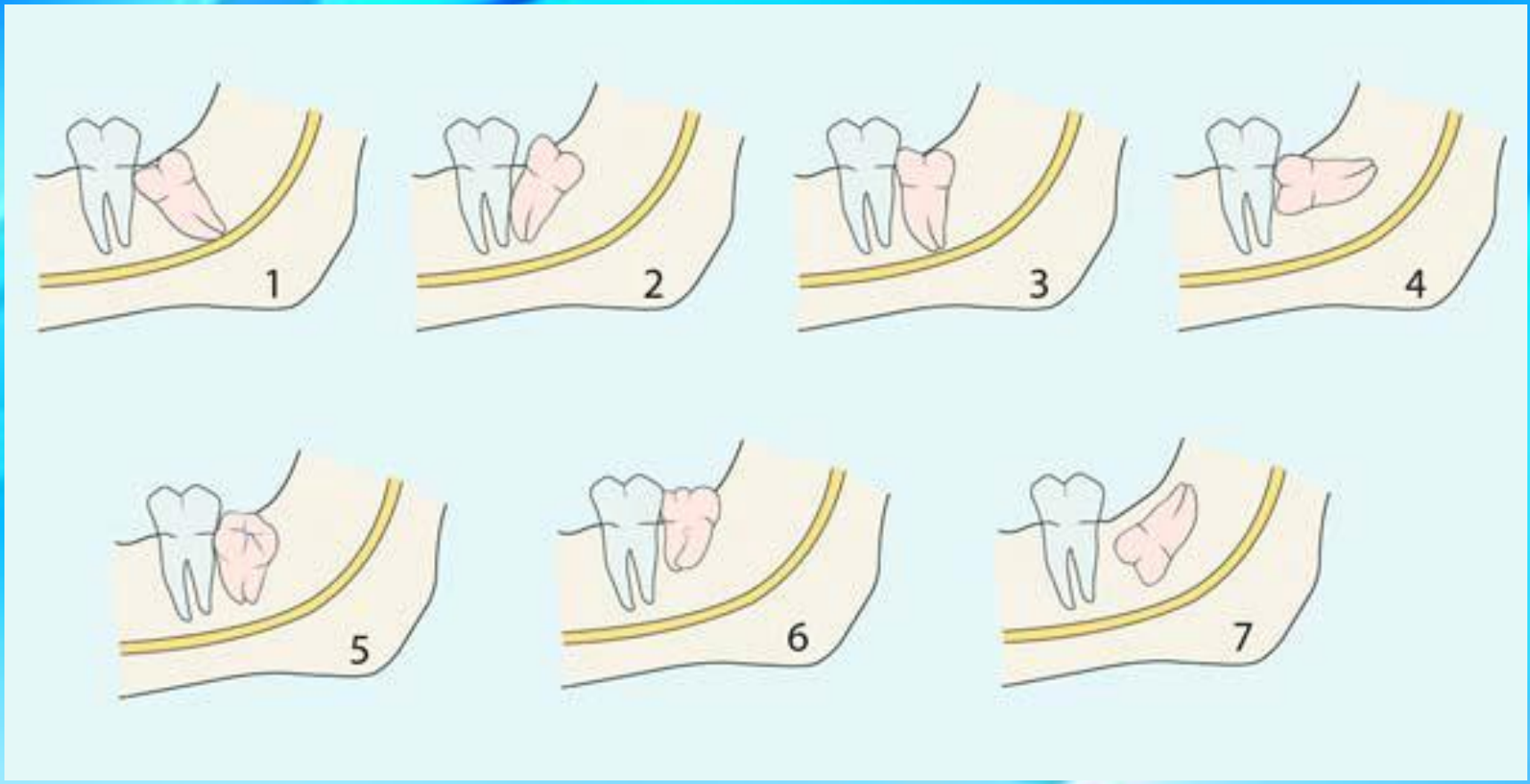


Fig-4- distoangular impaction



- **Transverse: i.e the tooth lies in a bucco-lingual direction,** the teeth can also be angled in buccal, lingual, directions . the most important point regarding this type during the surgical procedure for its removal is the possible presence of **a high-riding lingual nerve** still makes a buccal approach appropriate even when the tooth is inclined toward the lingual. The occlusal surface of the tooth can face the buccal or lingual direction. To determine buccal or lingua! version accurately, the dentist must take a perpendicular occlusal film. However, this determination is usually not necessary because the surgeon can make this identification early in the operation, and the buccal or lingual position of the tooth does **not greatly influence** the approach to the surgery.

- **Inverted impaction:** In this case the tooth is **present upside down**.


1
2
3
4
5
6
7

Relationship to the anterior border of ramus :

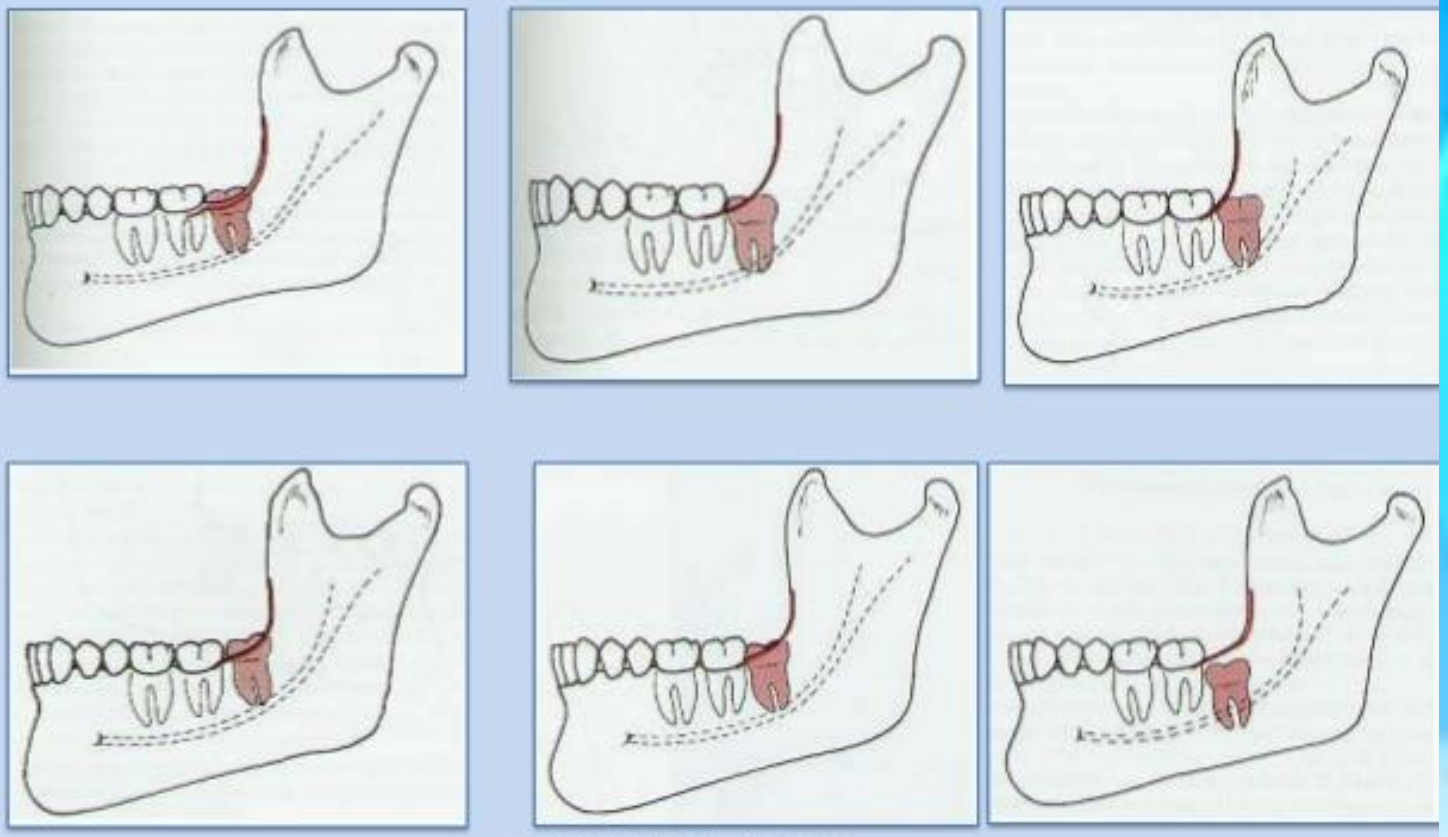
(Pell & Gregory classification 1,2 & 3)

Class 1 impaction: the tooth is **completely anterior** to the anterior border of ramus

Class 2 impaction: The tooth is positioned posteriorly so that approximately one half of the tooth is covered by the ramus.

Class 3 impaction: The tooth is located **completely within** the mandibular ramus.

PELL & GREGROY'S CLASSIFICATION



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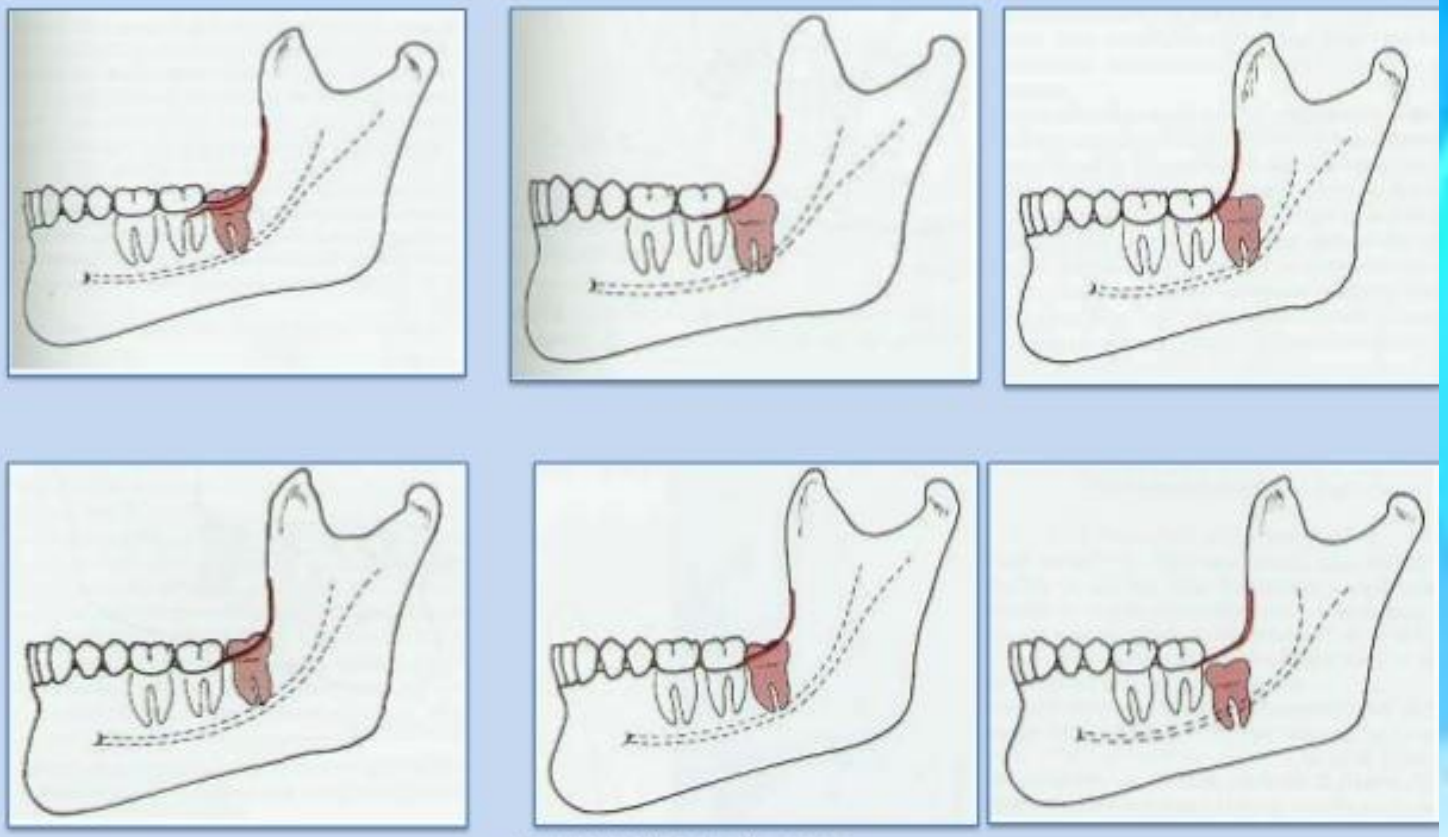
DEPTH OF IMPACTION: (Pell & Gregory classification A, B & C)

- **Class A impaction:** The occlusal surface of the impacted tooth is **level or nearly** level with the occlusal plane of the second molar .

- **Class B impaction:** The occlusal surface of the impacted tooth located between the **occlusal plane and the cervical line** of the second molar.

- **Class C impaction:** The occlusal surface of the impacted tooth is **below the cervical line** of the second molar .

PELL & GREGROY'S CLASSIFICATION



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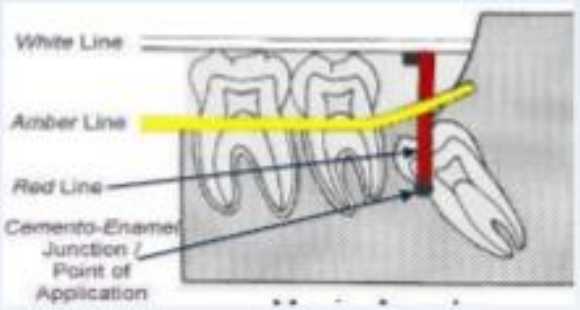
DIFFICULTY INDICES FOR REMOVAL OF IMPACTED MANDIBULAR THIRD MOLARS

WINTERS LINES / WAR LINES

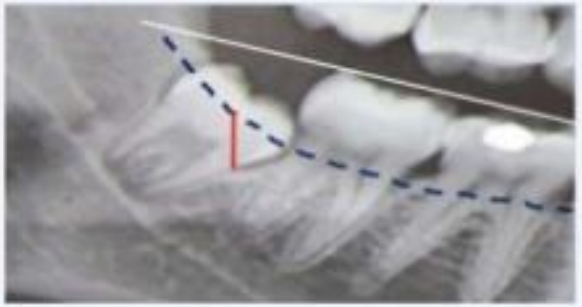
WHITE LINE

- Corresponds to occlusal plane of molar teeth.
- Indicates the **difference in occlusal level** of second and third molar



AMBER LINE

- Represents the bone level.
- Denotes the **alveolar bone covering the impacted tooth** and the portion of tooth not covered by the bone



RED LINE

- The red line is an imaginary line drawn perpendicular from the amber line to an imaginary point of application of an elevator
- Represents **depth of the tooth in bone** and the difficulty encountered in removing the tooth. Indicates the amount of bone that has to be removed before elevation
- If the length of red line is **more than 5 mm then extraction is difficult.**
- **For Every additional 1mm difficulty increases three times(3X).**

THE PREOPERATIVE ASSESSMENT

1- HISTORY: A Full history is taken regarding these points:

a) history of previous infection associated with the impacted tooth (**pericoronitis**)

b) a detailed **medical** history is taken

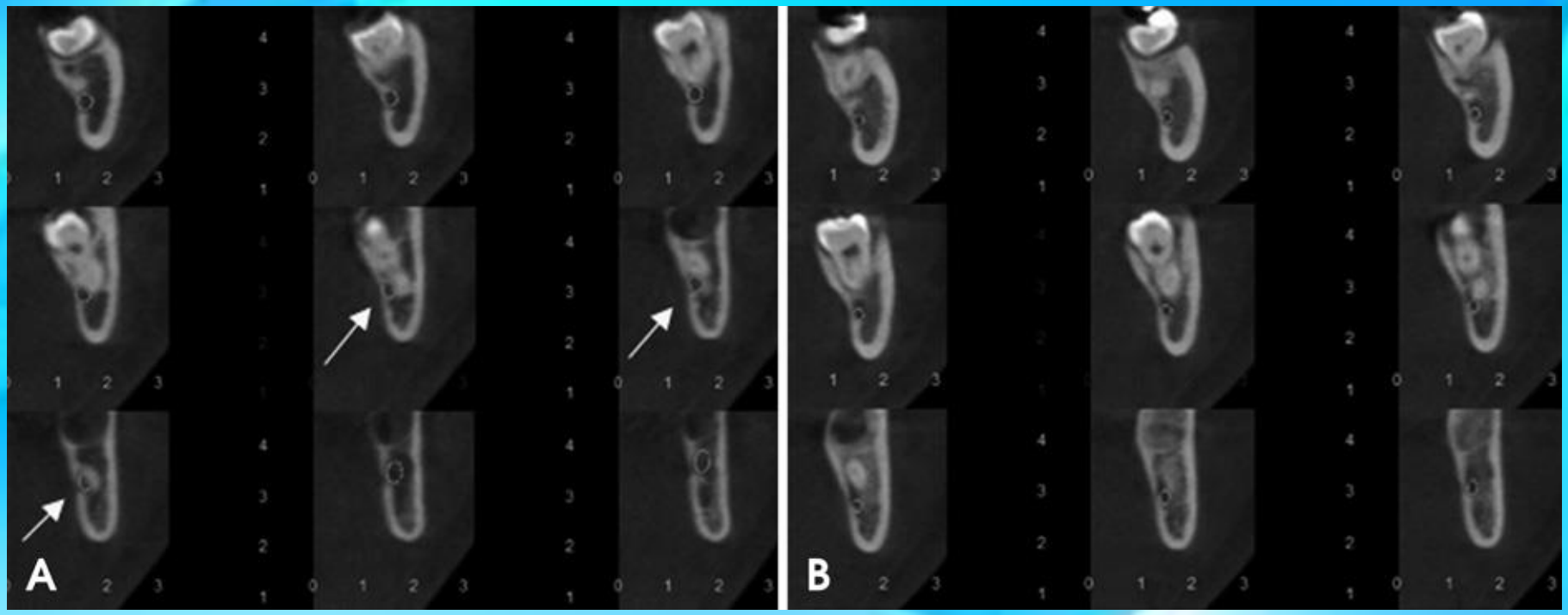
2-RADIOGRAPHICAL EXAMINATION: The radiographs used involve the following

a) **Periapical film**

b) O . P . G . View

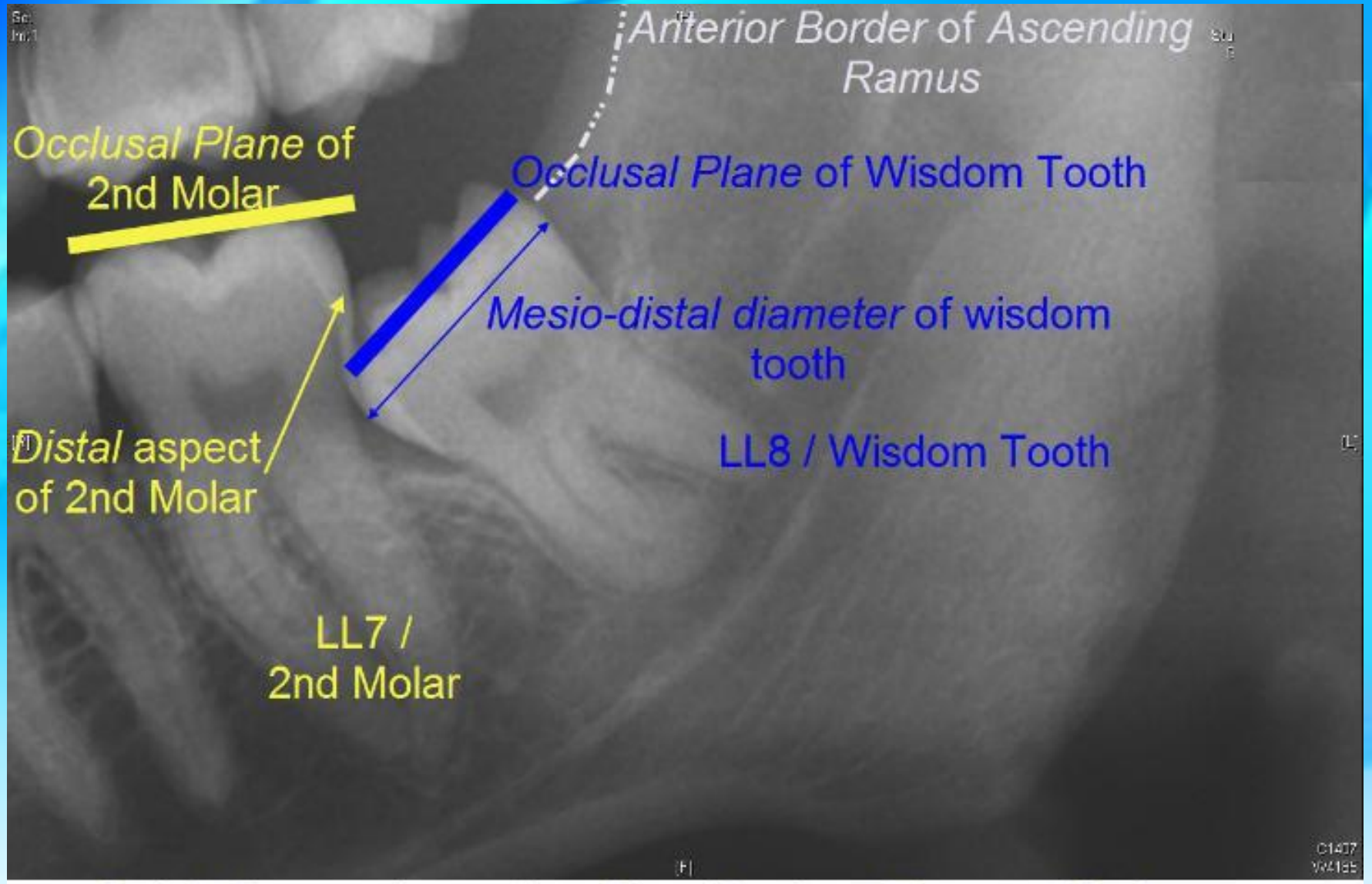
c) Lateral oblique view of-mandible

d) CT. Scan is important to give a full details about the impacted tooth.

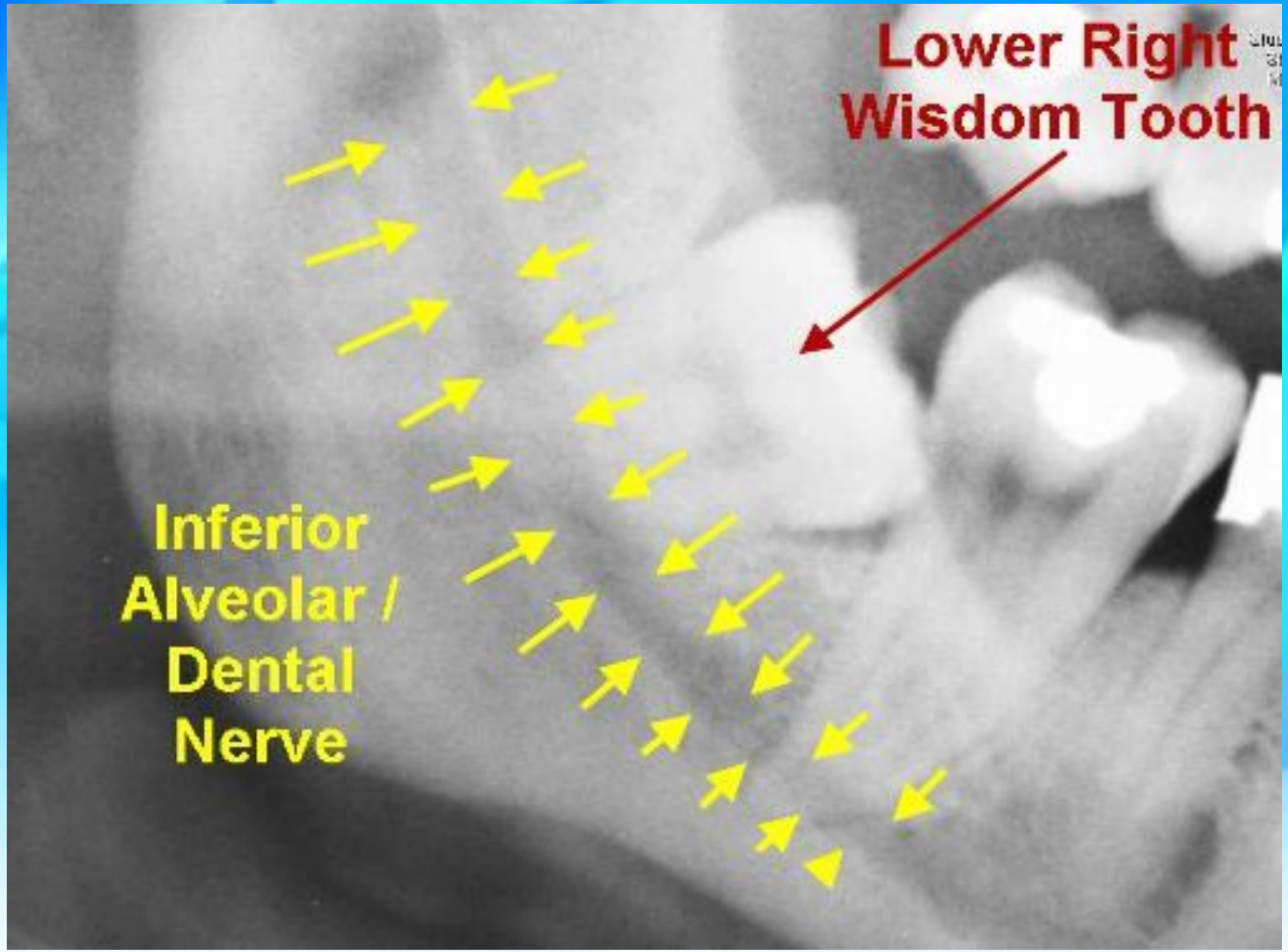

A
B

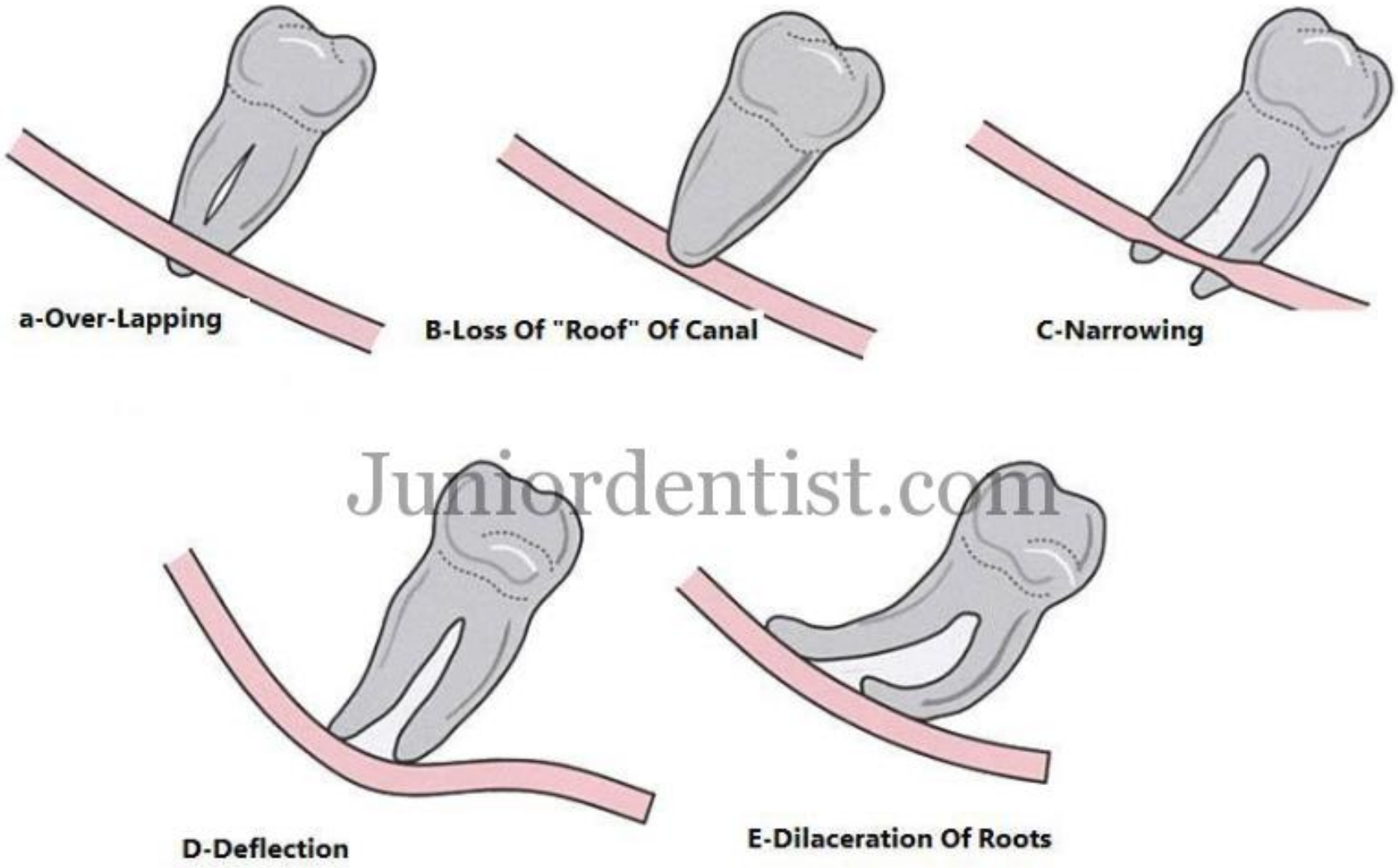
In radiograph we examine the following details:

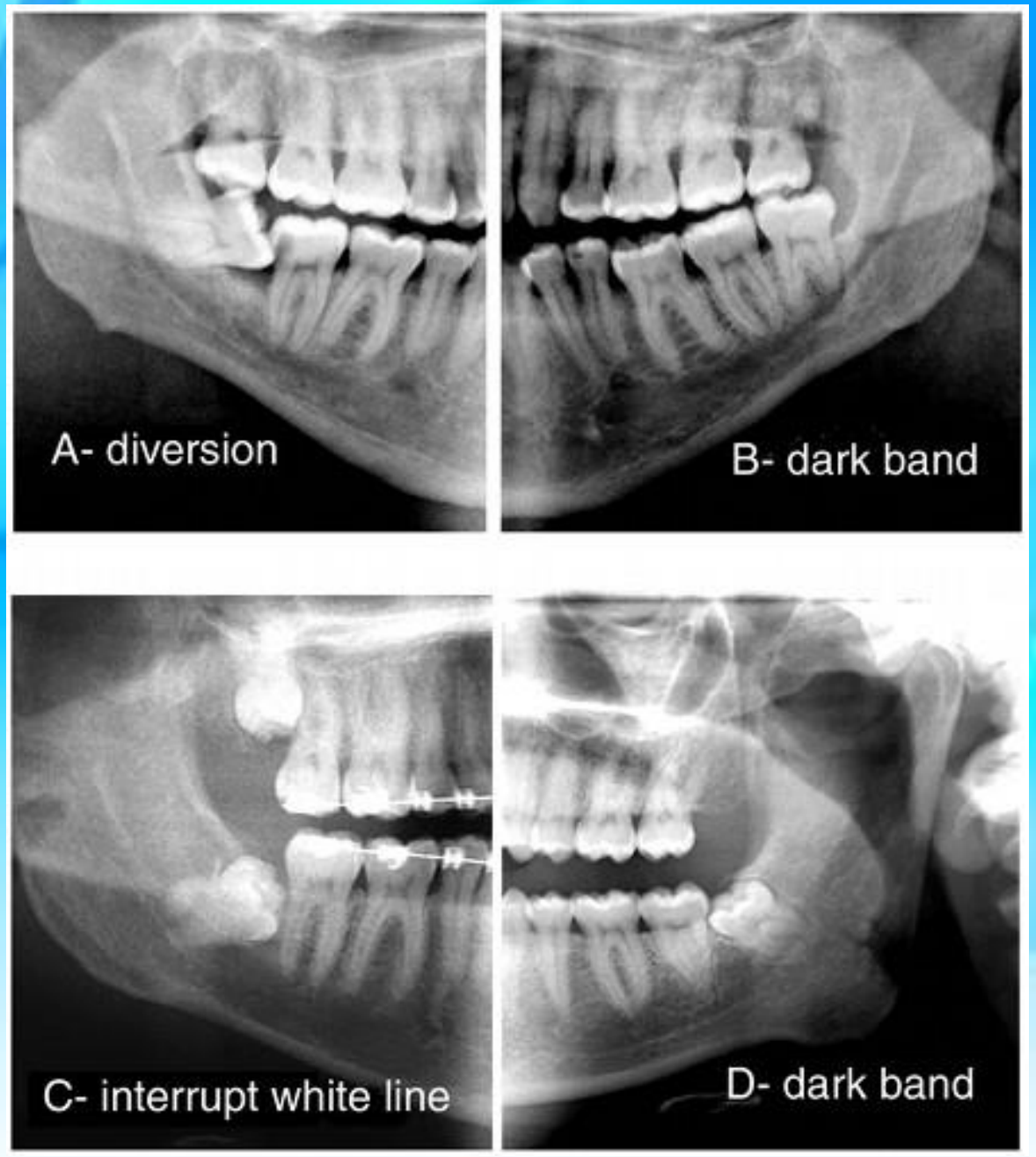
- ✓ **The direction of impaction**
- ✓ **Depth of the impaction**
- ✓ **Relation of the tooth to the inferior dental nerve:** impacted mandibular third molars frequently have roots that are superimposed on the inferior alveolar canal on radiographs. Therefore, one of the potential sequelae of impacted third molar removal is damage to the inferior alveolar nerve. This commonly results in some altered sensation (**Paresthesia or anesthesia**) of the lower lip and chin on the injured side. Although this altered sensation is usually brief (**lasting only a few days**), it may extend for weeks or months; on rare occasions it can be **permanent**. The duration depends on the extent of nerve damage. If the root ends of the tooth appear to be close to the inferior alveolar canal on a radiograph, the surgeon must take special care to avoid injuring the nerve ,The increasing availability of cone-beam computerized tomographic scans will make preoperative assessment of the root and canal relationship easier to view.



Pell & Gregory Impaction Classification - Terms of Reference


Lower Right
Wisdom Tooth
Inferior
Alveolar /
Dental
Nerve


a-Over-Lapping
B-Loss Of "Roof" Of Canal
C-Narrowing
Juniordentist.com
D-Deflection
E-Dilaceration Of Roots


A- diversion
B- dark band
C- interrupt white line
D- dark band

- ✓ **Shape & length of the roots**
- ✓ **Presence of a pathological lesion**
- ✓ **Density of the surrounding bone and the nature of the overlying tissue:** According to the overlying tissue there are three types of impactions are:

(1) soft tissue, An impaction is defined as a soft tissue impaction when the height of the contour of the tooth is above the level of the alveolar bone, and the superficial portion of the tooth is covered only by soft tissue.

Soft tissue impaction can be removed without bone removal

(2) partial bony: The partial bony impaction occurs when the superficial portion of the tooth is covered by soft tissue but at least a portion of the height of the contour of the tooth is below the level of the surrounding alveolar bone

(3) full bony: The complete bony impaction is an **impacted tooth that is completely encased in bone** so that, when the surgeon reflects the soft tissue flap, no tooth is visible .


Gum layer
Bony
Vertical
Impaction

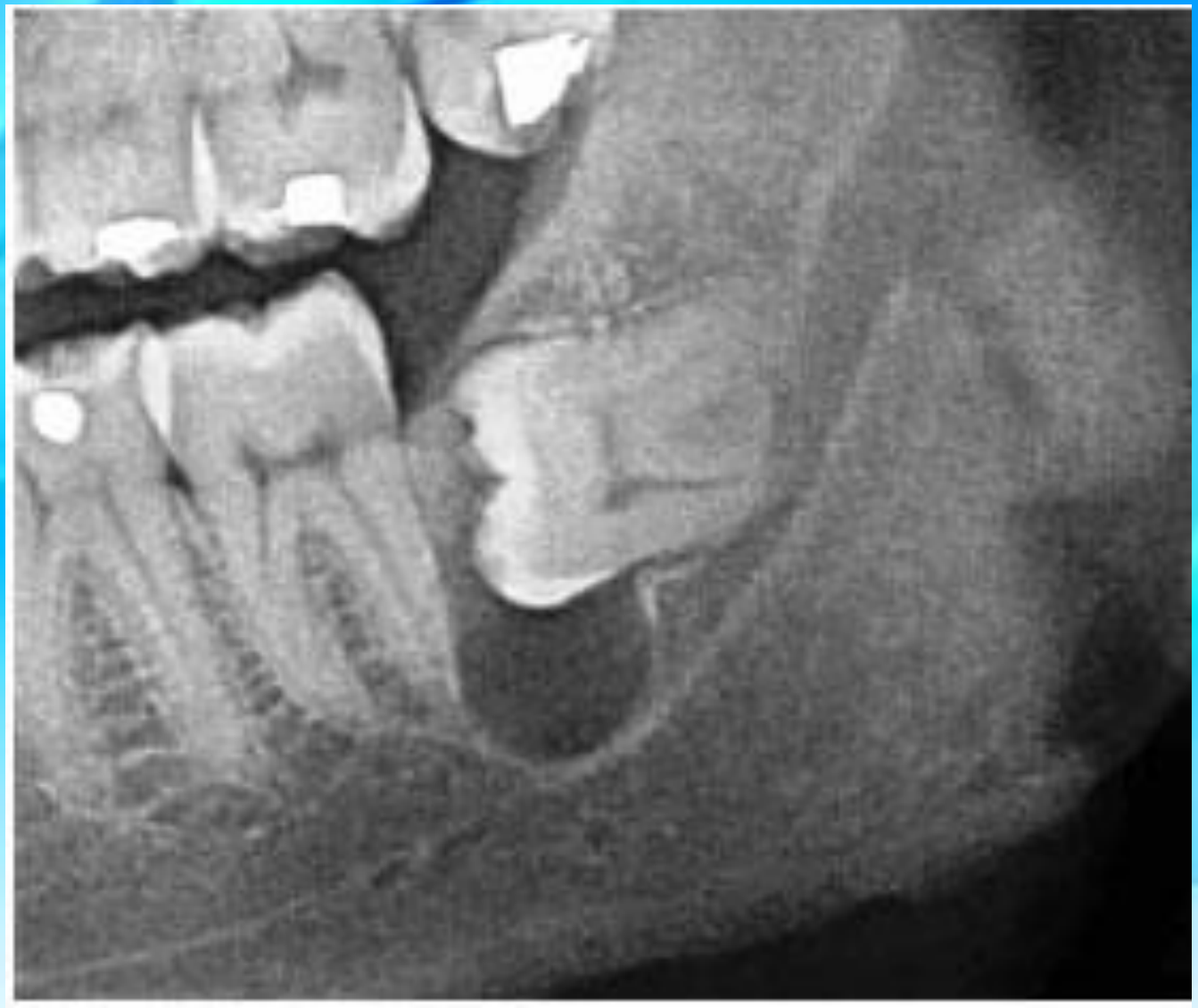

Gum layer
Soft Tissue
Vertical
Impaction

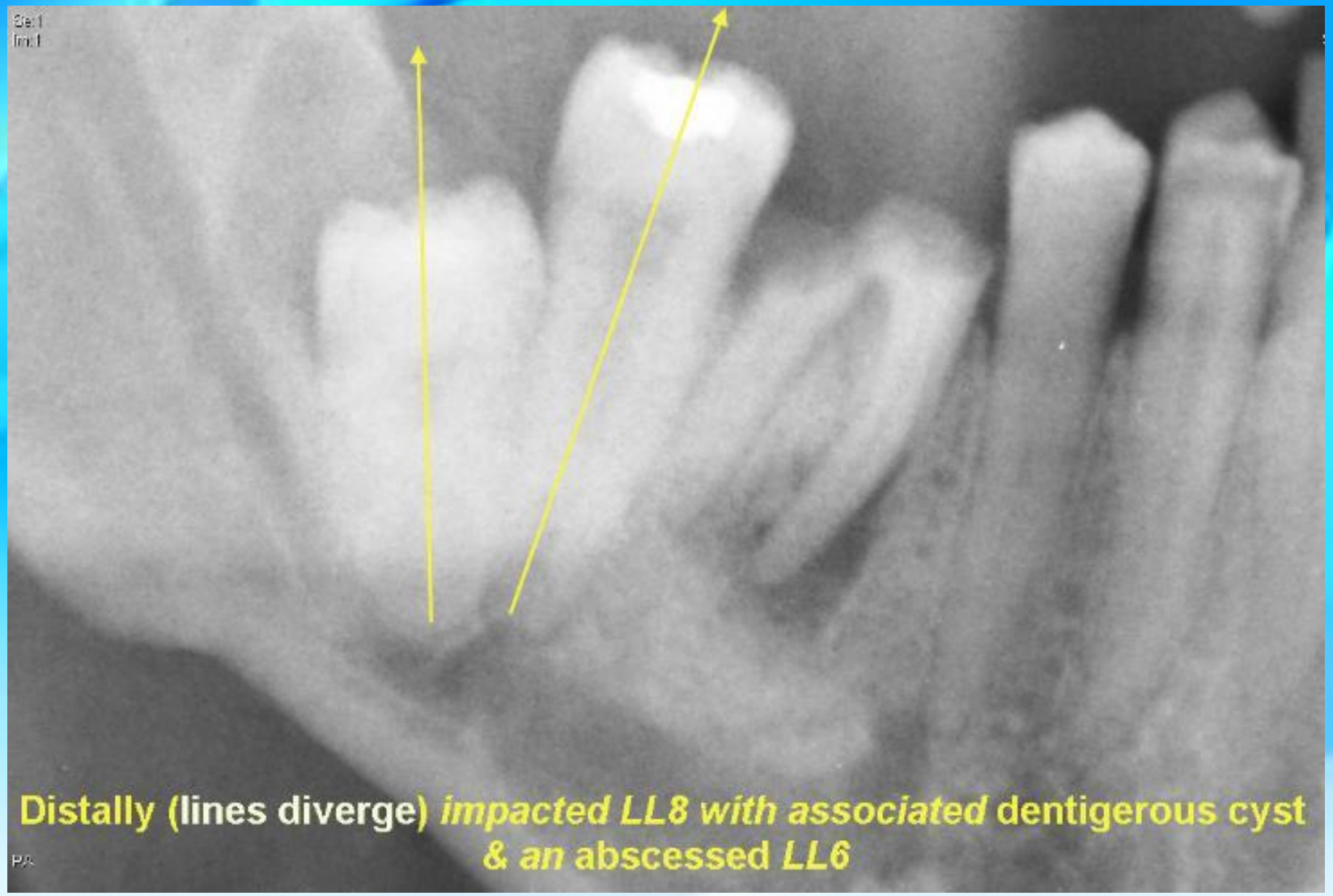
- ✓ **Size of follicular sac**

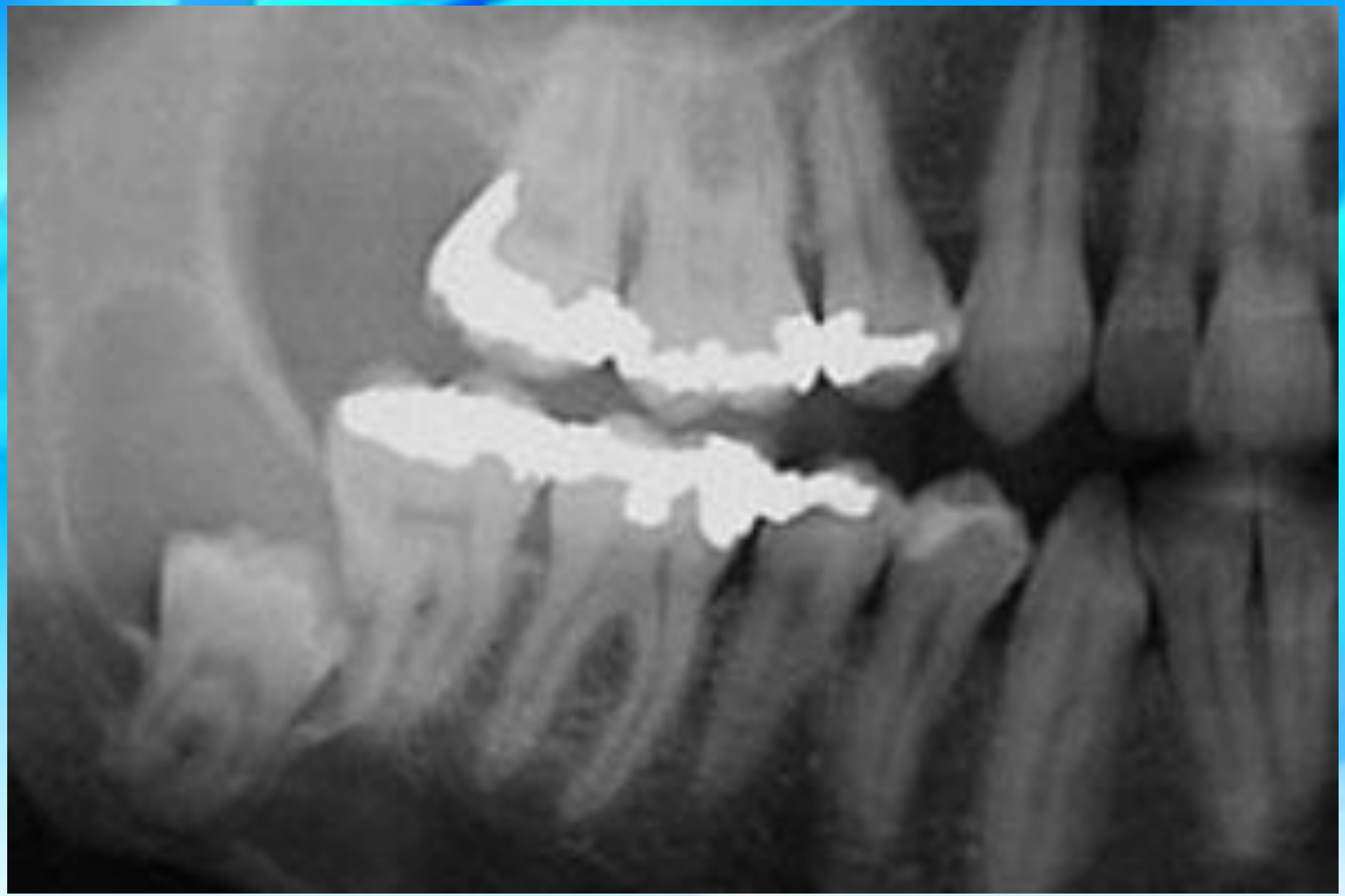
The size of the follicle around the impacted tooth can help determine the difficulty of the extraction. If the follicular sac **is wide** (almost cystic in size), much **less bone must be removed**, which makes the tooth more straightforward to extract . (Young patients are more likely to have large follicles, which is another factor that makes **extractions less complex** in younger patients.) However, if the follicular space around the crown of the tooth is narrow or nonexistent, the surgeon must create space around the crown, **increasing the difficulty** of the procedure and usually the time required to remove the tooth.

- ✓ **Contact with Mandibular Second Molar**

If space exists between the second molar and the impacted third molar, the extraction will **be easier** to approach because damage to the second molar is less likely. However, if the tooth is **a distoangular or horizontal impaction**, it is frequently in direct contact with the adjacent second molar. To remove the third molar safely without injuring the second molar, the surgeon must be cautious with pressure from elevators or with the bur when removing bone. If the second molar has caries or a large restoration or has been endodontically treated, the surgeon must take special care not to fracture the restoration or a portion of the carious crown. The patient should still **be forewarned** of this possibility.




Distally (lines diverge) impacted LL8 with associated dentigerous cyst
& an abscessed LL6



The surgical technique

Anaesthesia

The surgical procedure could be done either under local anesthesia for removal of a unilateral impaction or under general anesthesia for removal of a bilateral impaction.

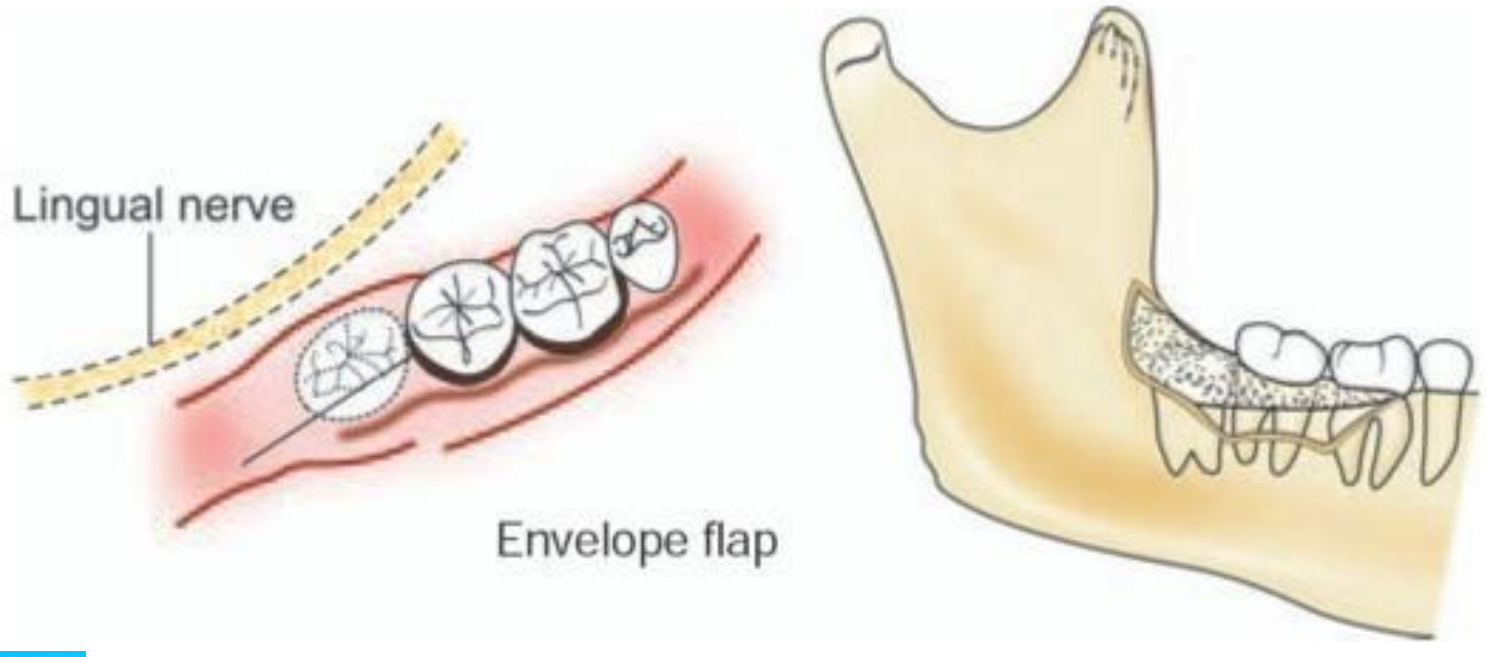
The flap design :There are two types of flaps mainly used :

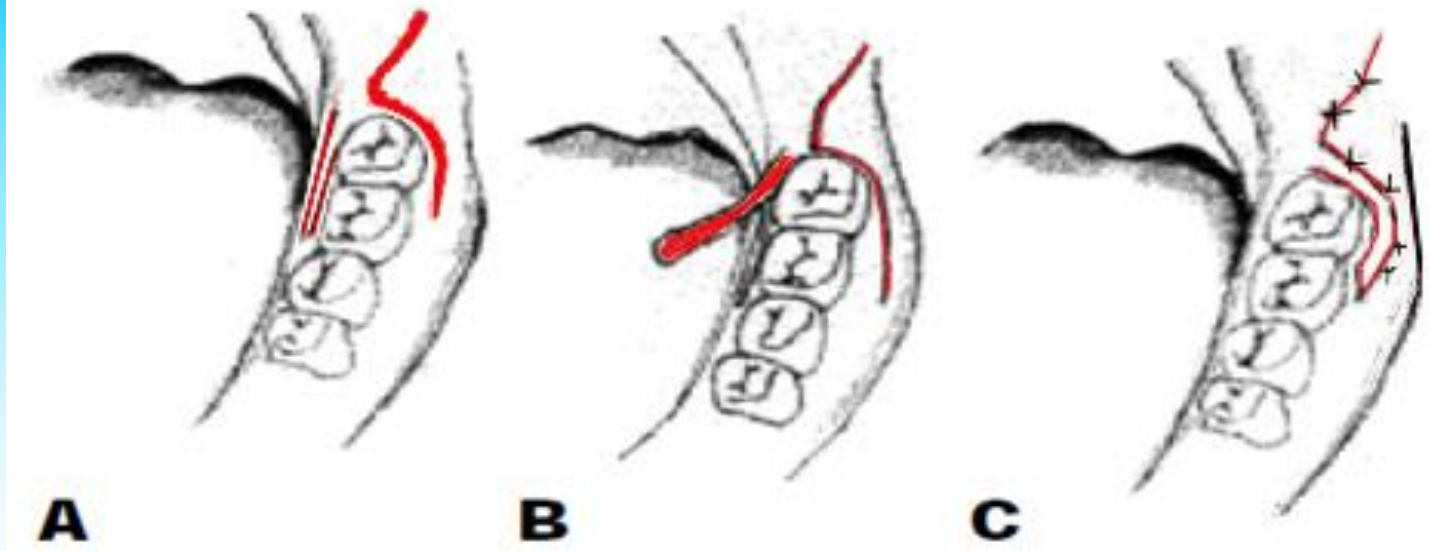
1-Envelope flap

The preferred incision for the removal of an impacted mandibular third molar is an envelope incision **that extends from the mesial papilla** of the mandibular first molar, around the necks of the teeth to the distobuccal line angle of the second molar, and then posteriorly to and laterally up the **anterior border of the mandibular ramus**

The incision must not continue posteriorly in a straight line because the **mandible diverges laterally**. An incision that extends straight posteriorly falls off the bone and into the sublingual space and may damage the **lingual nerve,** which is in proximity to the mandible in the area of the third molar. If this nerve is traumatized, the patient will probably have lingual nerve anesthesia, which is extremely disturbing to patients. The incision must always be kept over bone; therefore the surgeon should carefully palpate the retromolar area before beginning the incision.

The flap is reflected laterally to expose the external oblique ridge with a periosteal elevator


Lingual nerve
Envelope flap

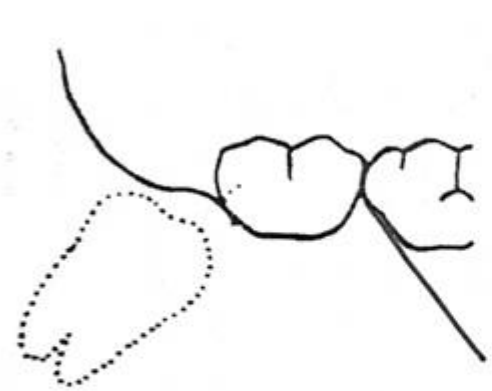

A
B
C

2-Triangular flap (Two - sided flap) three-cornered flap (envelope flap with a releasing incision) .this type of flap is used when the surgeon requires greater access to the **more apical areas** of the tooth, which might stretch and tear the envelope flap, the surgeon should consider using a three cornered flap.

The soft tissues are stretched up over the anterior border of the ascending ramus . The incision is Done in two steps . First start incision from the midpoint of distal surface of the second molar diagonally Across the impacted third molar toward the external oblique ridge . Then an incision is done across the gingival margin of the second molar forward to the mesial side of the second molar or distal side of the first molar (the interdental papilla between the first and second molar either left or involved with the flap) this depend on the depth and direction of impaction . then a releasing incision is done extended downward and forward to avoid trauma to the Facial artery which makes a loop at the inferior borderer of mandible at this area.

In case of the envelope flap the incision extend to the mesial side of the first molar without a releasing incision

NOTE : when we do the incision at the distal surface of the second molar , we should avoid trauma to the lingual nerve which lies in close proximity to the third molar at this area



BUCCAL VIEW

OCCLUSAL VIEW

THE FLAP REFLECTION

The flap is reflected with a Howarth's mucoperiosteal elevator starting the reflection from the junction of the releasing and gingival margin incision to avoid the tearing of the flap . Then the flap is retracted with a flap retractor and the lingual tissue is reflected to expose the crown of the impacted tooth and the bone distal to it.

THE BONE REMOVAL: Bone has to be removed to provide a space into which the tooth can be tilted. Bone removal is performed either by :

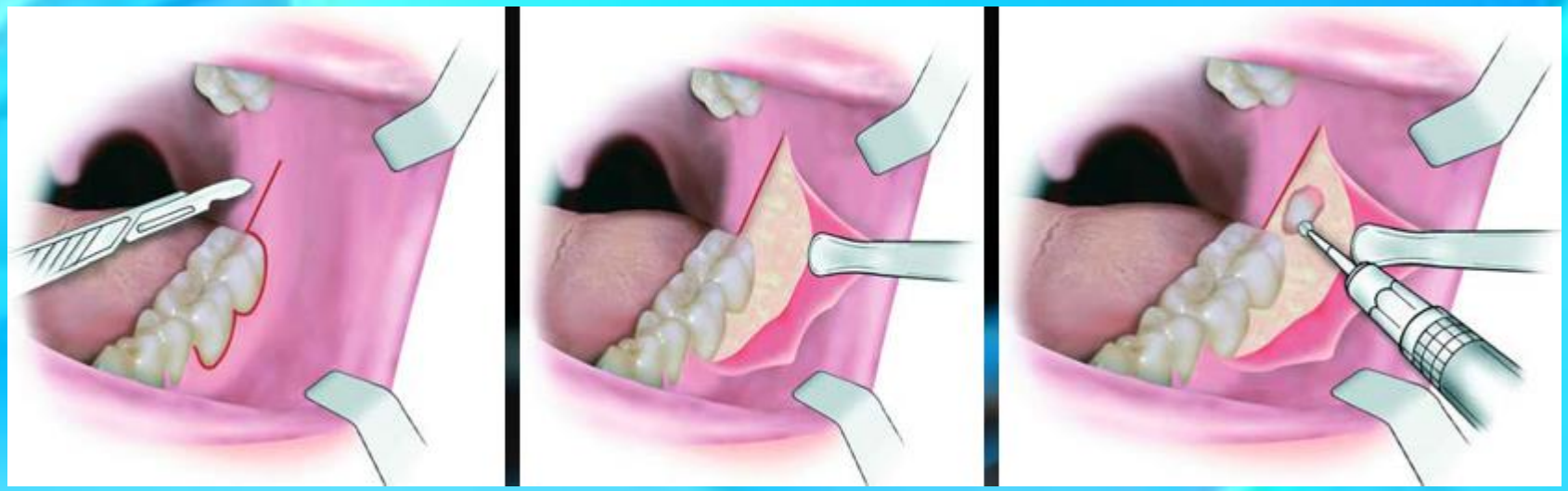
a-straight headpiece with a surgical burs

b- chisel and hummer

Burs rather than chisels should be used **when the patient is receiving a local anesthesia** with or without sedation or in **Older patients with brittle bone** . and at any age when the **external oblique ridge lies forward** so that the investing bone Is thick .

The bone on the occlusal surface of the tooth is removed **first** to expose the crown of the tooth using either Fissure bur or round bur, then the cortical bone on the buccal aspect of the tooth is removed down to the cervical line , Then bone is removed from the distal side also to the cervical line . bone should **not be** removed from the lingual surface To avoid trauma to the lingual nerve . Bone removal is continued on the buccal side into a semicircular trough and deepening down to the bifurcation area To provide a point of application for an elevator.

NOTE : Bone removal should be accompanied with continuous **irrigation w**ith normal saline to avoid the over heating of bone .



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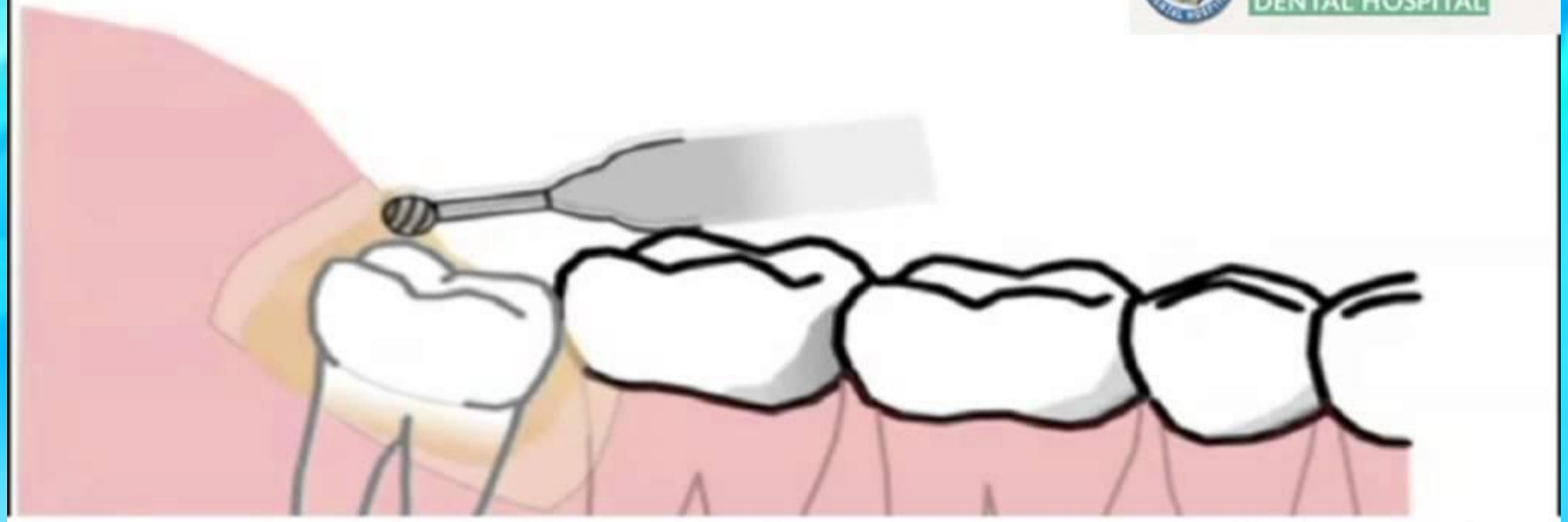

Wisdom tooth removal (full-bony)

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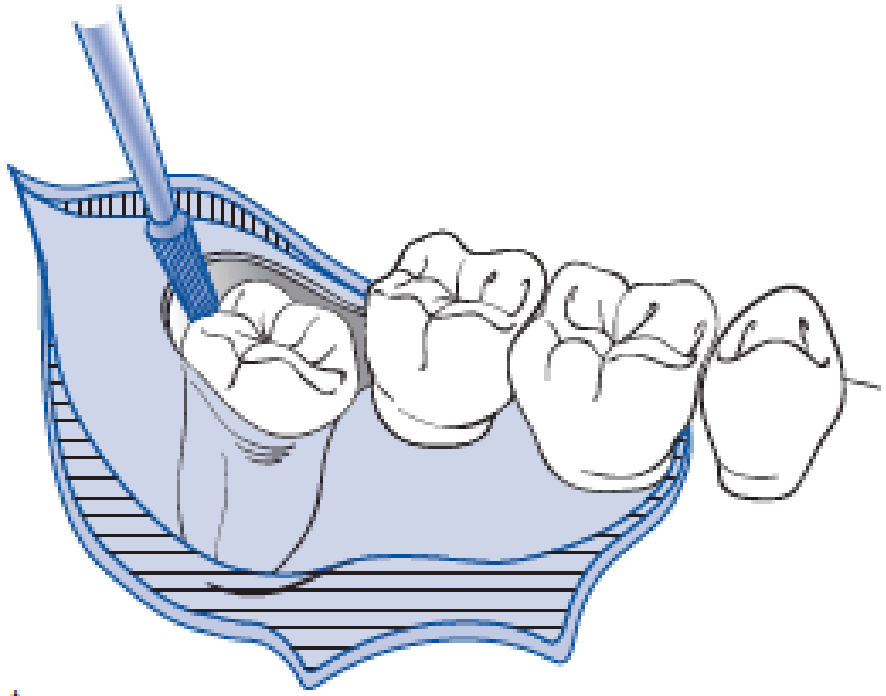
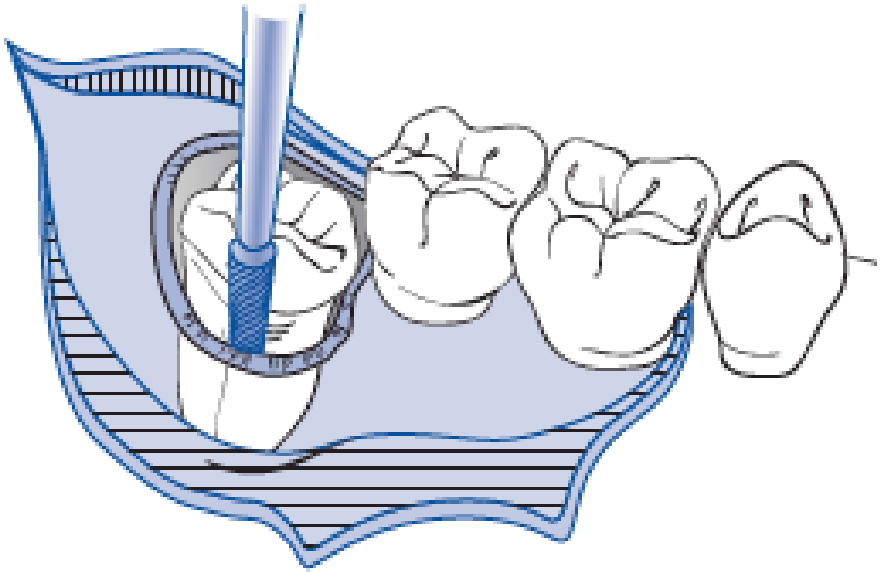


A



B

THE TOOTH SECTIONING

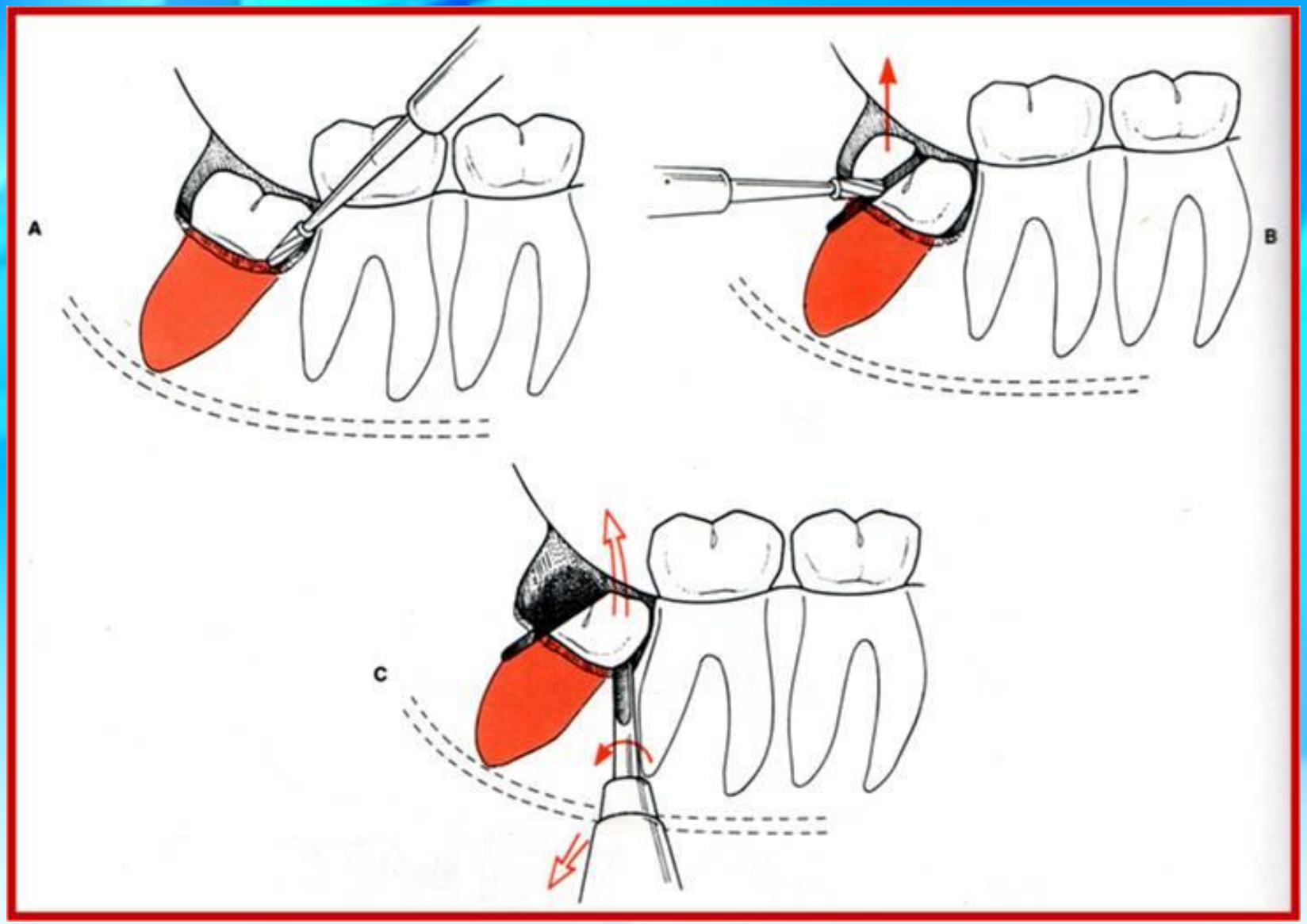
Tooth sectioning is important **to minimize the amount of bone** removal. the direction in which the impacted tooth Should be divided depends primarily on **the angulation** of the impacted tooth . tooth sectioning is performed by Straight hand piece and bur, and the tooth is sectioned three fourths of the way **toward the lingual** aspect.

The bur should not be used to section the tooth completely through in the lingual direction because this is more likely to injure the lingual nerve. **A straight elevator is inserted into the slot** made by the bur and rotated to split the tooth.

A) Mesioangular impaction :

The distal portion of the crown **is sectioned** OR the entire tooth can be **sectioned into two portions**

Then after removal of **the distal portion** , a straight elevator is inserted into a **purchase point** on the mesial aspect of third molar and the tooth delivered with rotational and lever motion of the elevator.


A
B
C

B) Horizontal impaction :

The tooth is sectioned by dividing the crown of the tooth from the **roots at the cervical line** . the crown Of the tooth **is removed** , and the roots are **displaced with a cryer elevator** into the space previously Occupied by the crown . if the roots are divergent, they may require sectioning into two separated Portions to be delivered individually

3. Divide **tooth** into sections and delivered with elevators

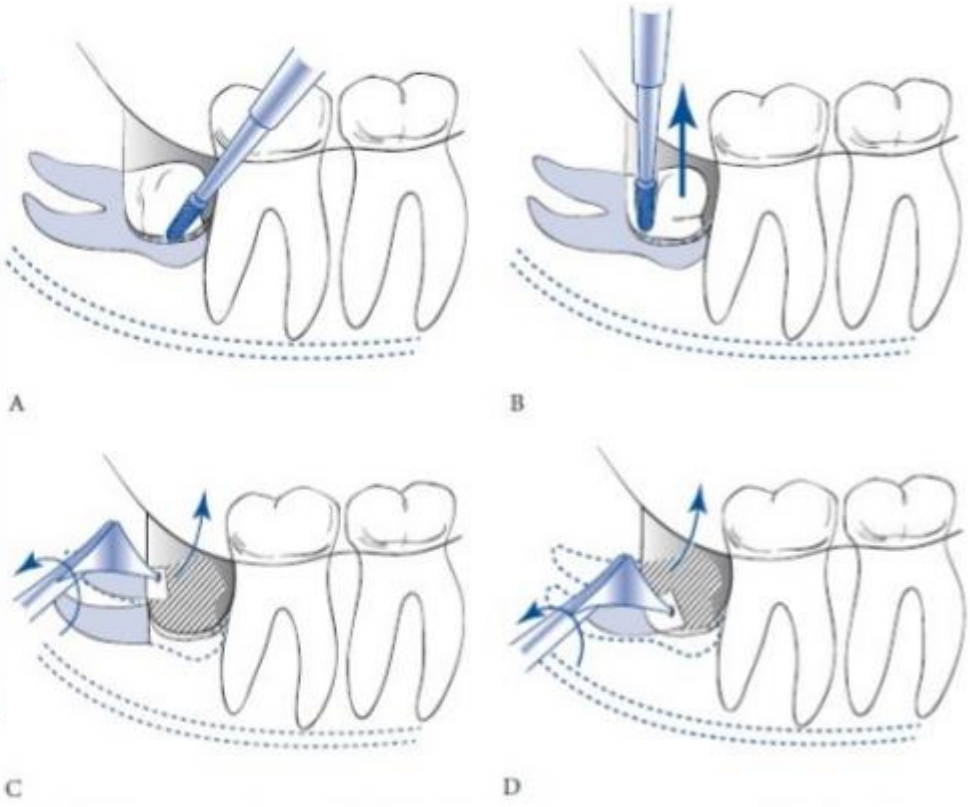
Horizontal impaction

B. B and D bone are removed

C. Crown is sectioned from the roots.

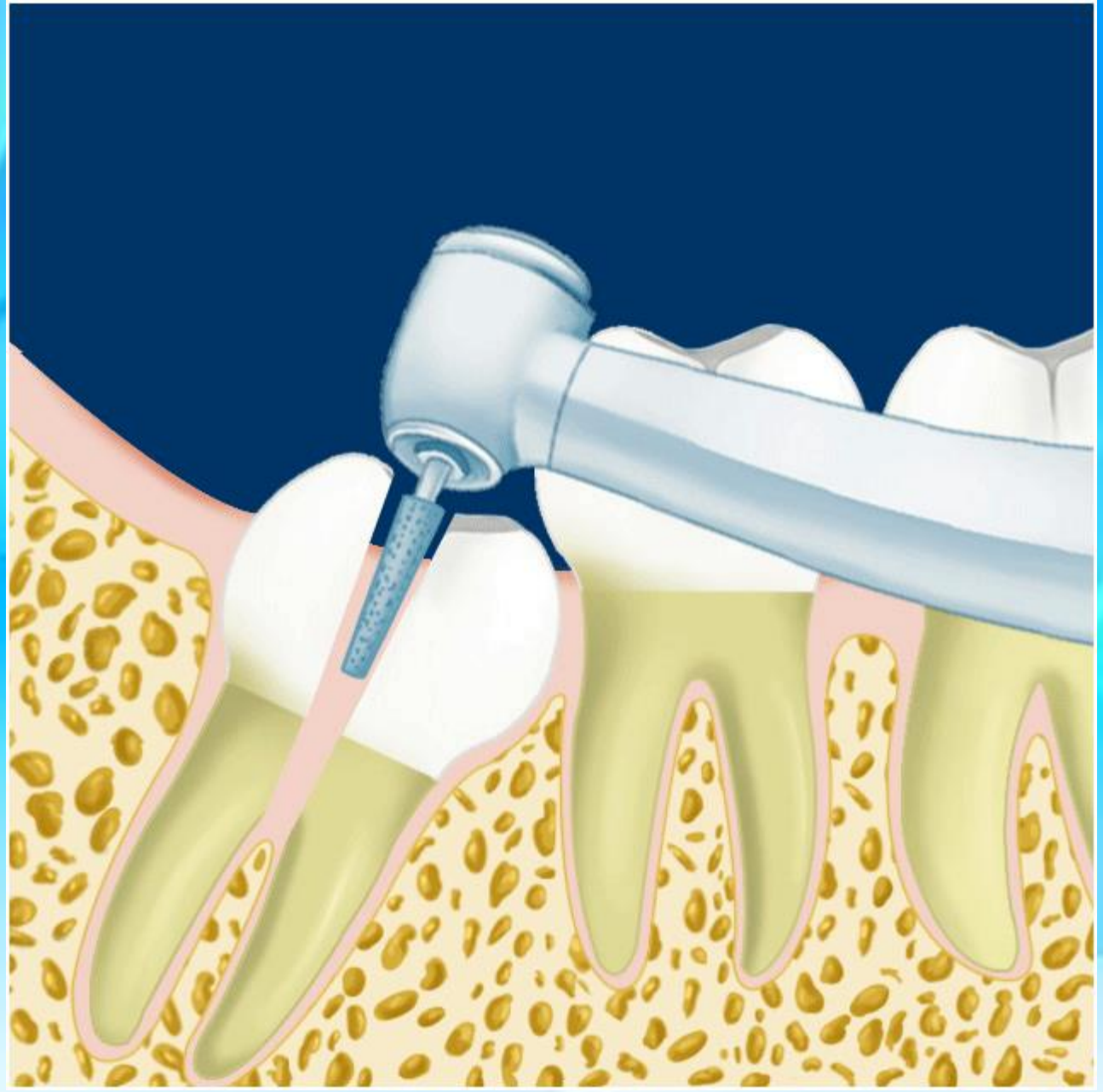
D. Roots are delivered together or independently with a Cryer.

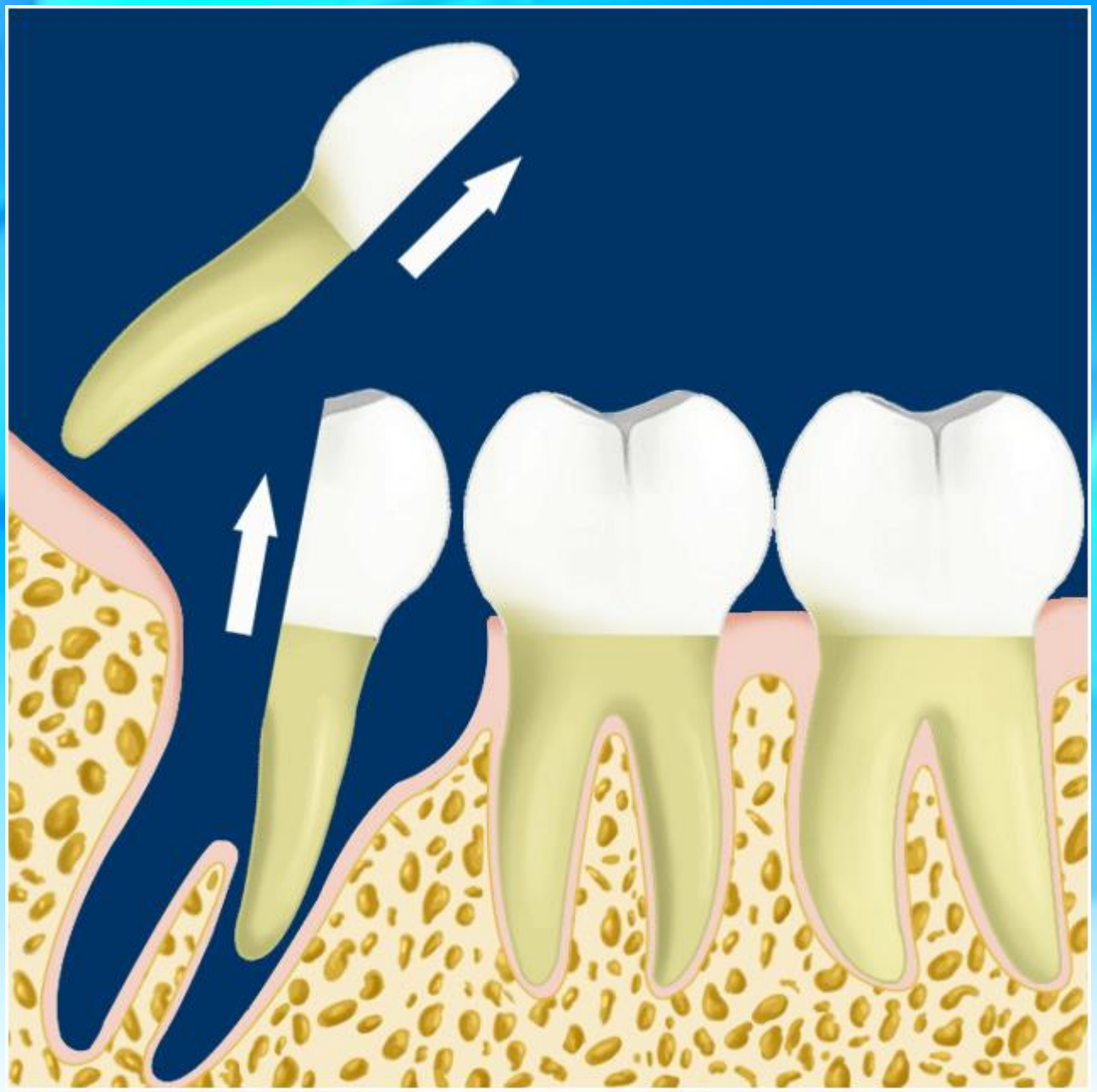
E. M root is elevated in similar fashion

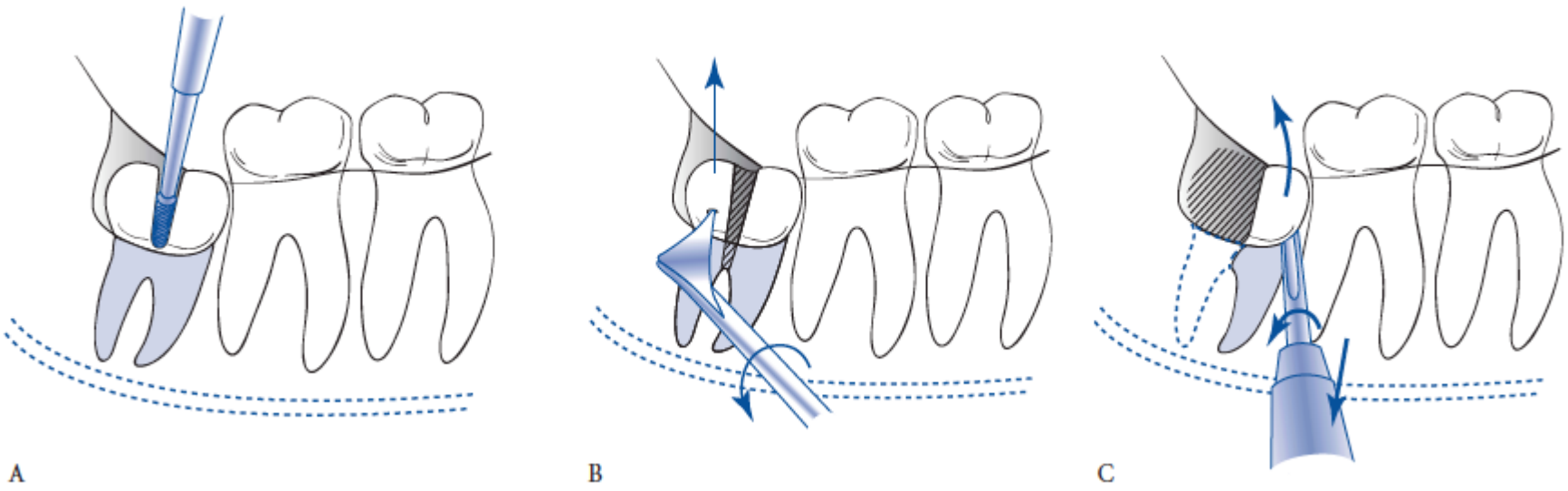


C) vertical impaction

The tooth is sectioned into mesial and distal sections . then the posterior portion of the tooth is elevated First with cryer elevator inserted into a **small purchase point in distal** portion of the tooth . Then a Small straight elevator is used to elevate the mesial aspect of the tooth by rotary and lever type of Motion . But in cases of a superficial impaction the tooth is not **need sectioning**



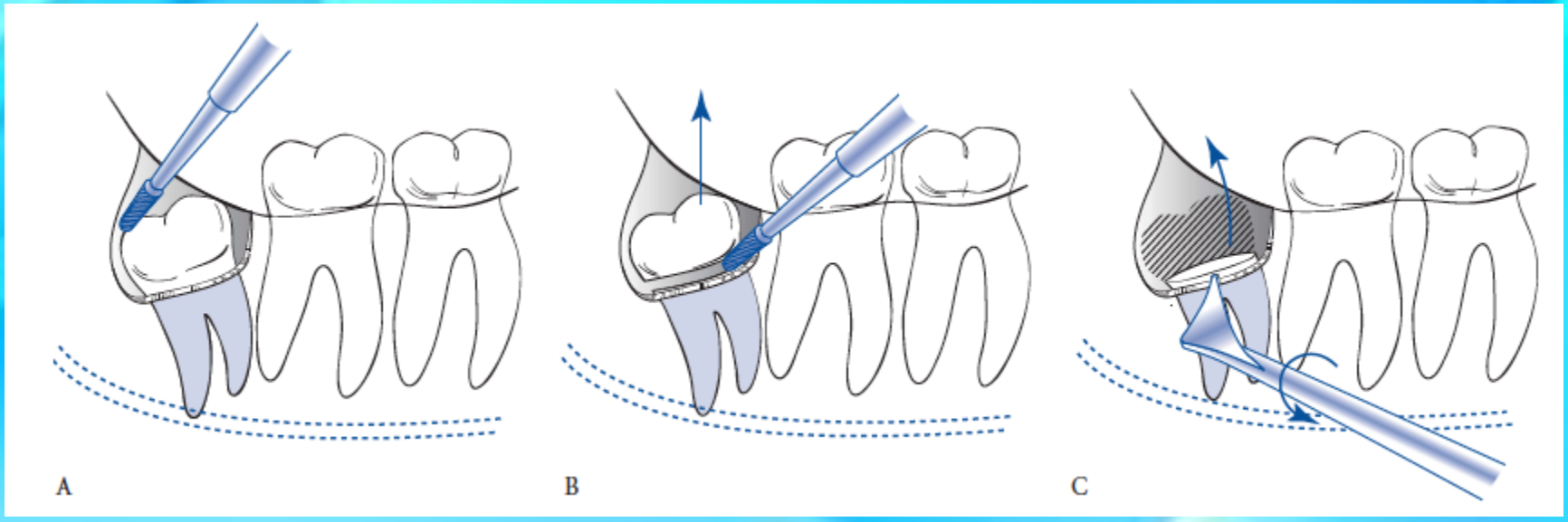



A
B
C

D) Distoangular impaction:

The crown is sectioned from the **roots at the cervical line** . The **entire crown is removed** . Then if the roots are fused , a cryer or straight elevator can be used to elevate the tooth into the space previously occupied by the crown . But if the roots are divergent , they are usually sectioned into two **pieces and individually** delivered .

In some cases we can do sectioning for the distal portion of the crown, then by straight elevator we remove **that distal portion** , after that the mesial portion is removed with an elevator.


A
B
C

TOILET OF THE SOCKET

The socket is irrigated with sterile normal saline, taking particular care to aspirate away all bone and tooth debris from beneath the periosteum under the buccal flap .

If bone or tooth debris is left in the crevice between the periostium and the buccal bone an **abscess will be formed there 3-4** weeks later.

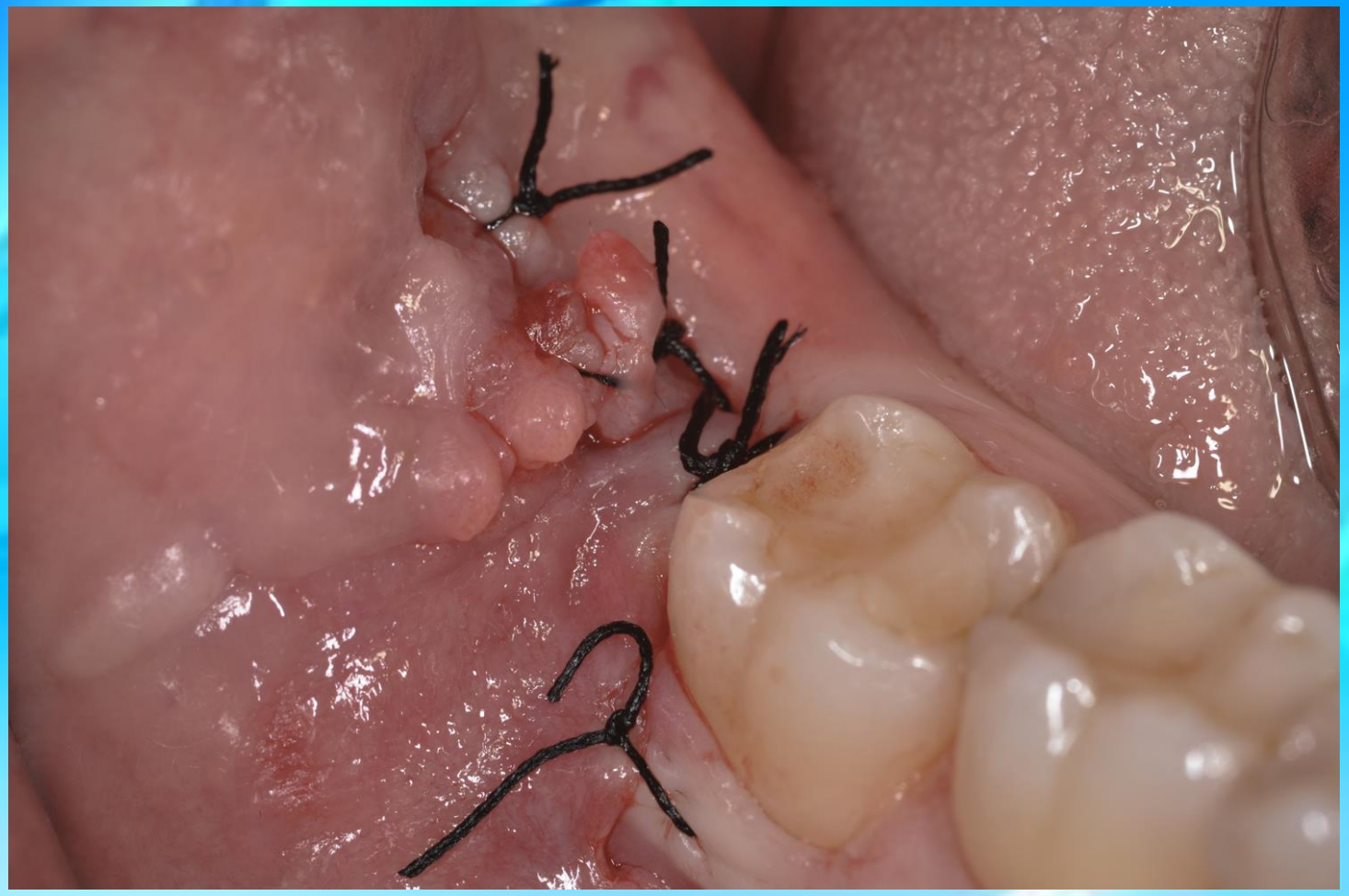
Any sharp margins should be smoothed with bone file, if they left, they may penetrate the flap and irritate the patient's tongue until sequestrated .

SUTURING OF THE FLAP

We should check adequate hemostasis before suturing the initial suture should be placed at the distal side of the second molar , additional suture is placed posterior to it, and a suture is placed through the papilla at the mesial side of the second molar .

THE POSTOPERATIVE CARE

The patient is instructed to have a soft diet, hot salty mouth wash which continues several times daily until the edematous swelling subsided, antibiotic and analgesic is prescribed to the patient, the patient should come back after seven days for suture removal




THANKS

- **Inoculation** : entry of pathogenic microbes into the body without disease occurring.

- **Infection** : entery and proliferation of pathogenic microbes in to the body resulting in triggering of the defense mechanism, a process manifesting as inflammation.

- **Inflammation** is the localized reaction of vascular and connective tissue of the body to an irritant, resulting in the development of an exudate rich in proteins and cells , Inflammation may be caused by,microbes, physical and chemical factors,(heat, irradiation).

Body response to infection

- Hyperemia
- Precipitation of fibrin network to wall off the infected region
- Phagocytosis of bacteria and dead cells
- Disposal of necrotic debris by macrophage

Signs and Symptoms of infection

These include systemic signs and symptoms like; fever, malaise and anorexia. There are also local signs and symptoms, which are present to some degree in all patients:

Redness, it results from vasodilatation.

Swelling, in infection it results from accumulation of tissue fluid or pus.

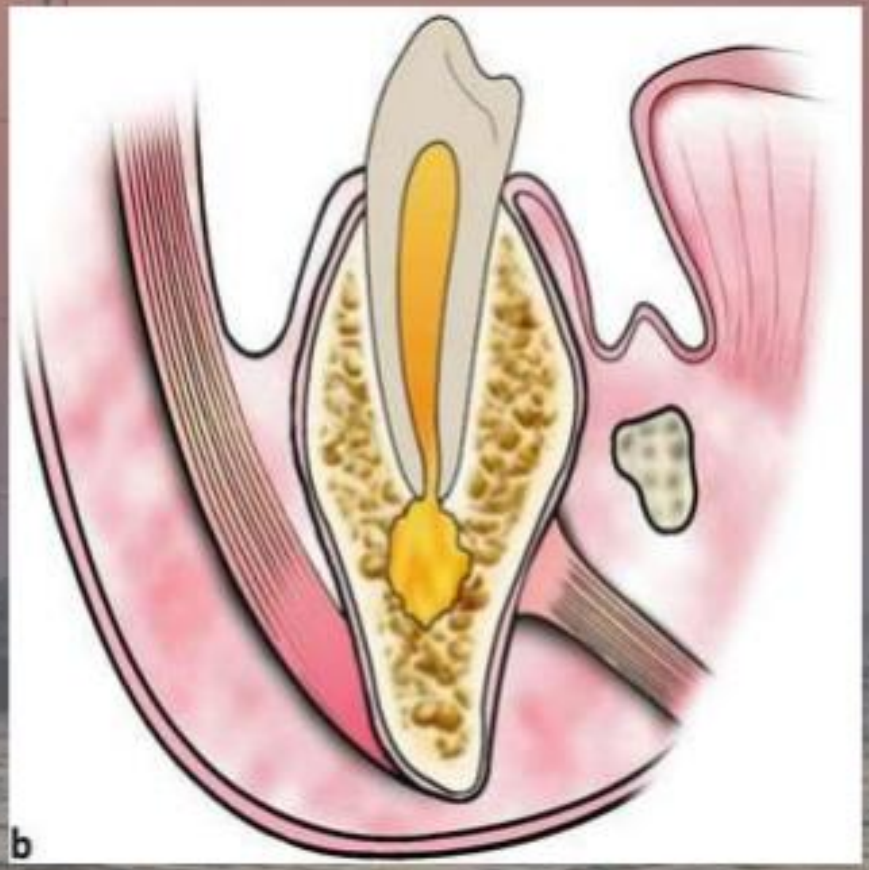
Heat, results from the inflow of warm blood from deeper tissues, increased velocity of blood and increased metabolic rate.

Pain, results from pressure on the nerve endings, also from the action of liberated or activated factors such as Kinins, Histamine and Bradykinin on nerve endings.

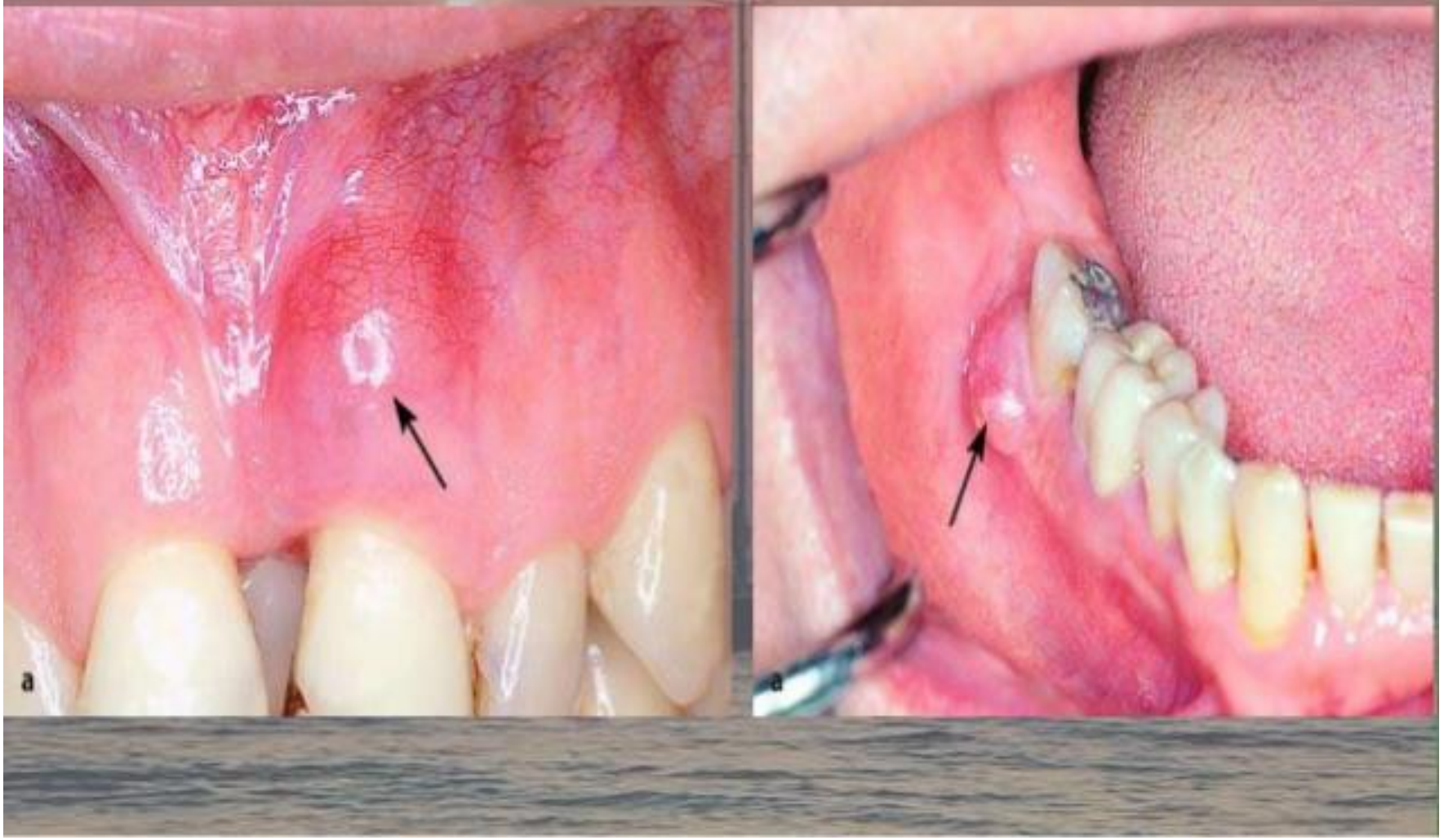
Loss of function, caused by mechanical factors and reflex inhibition of muscle movement associated with pain.

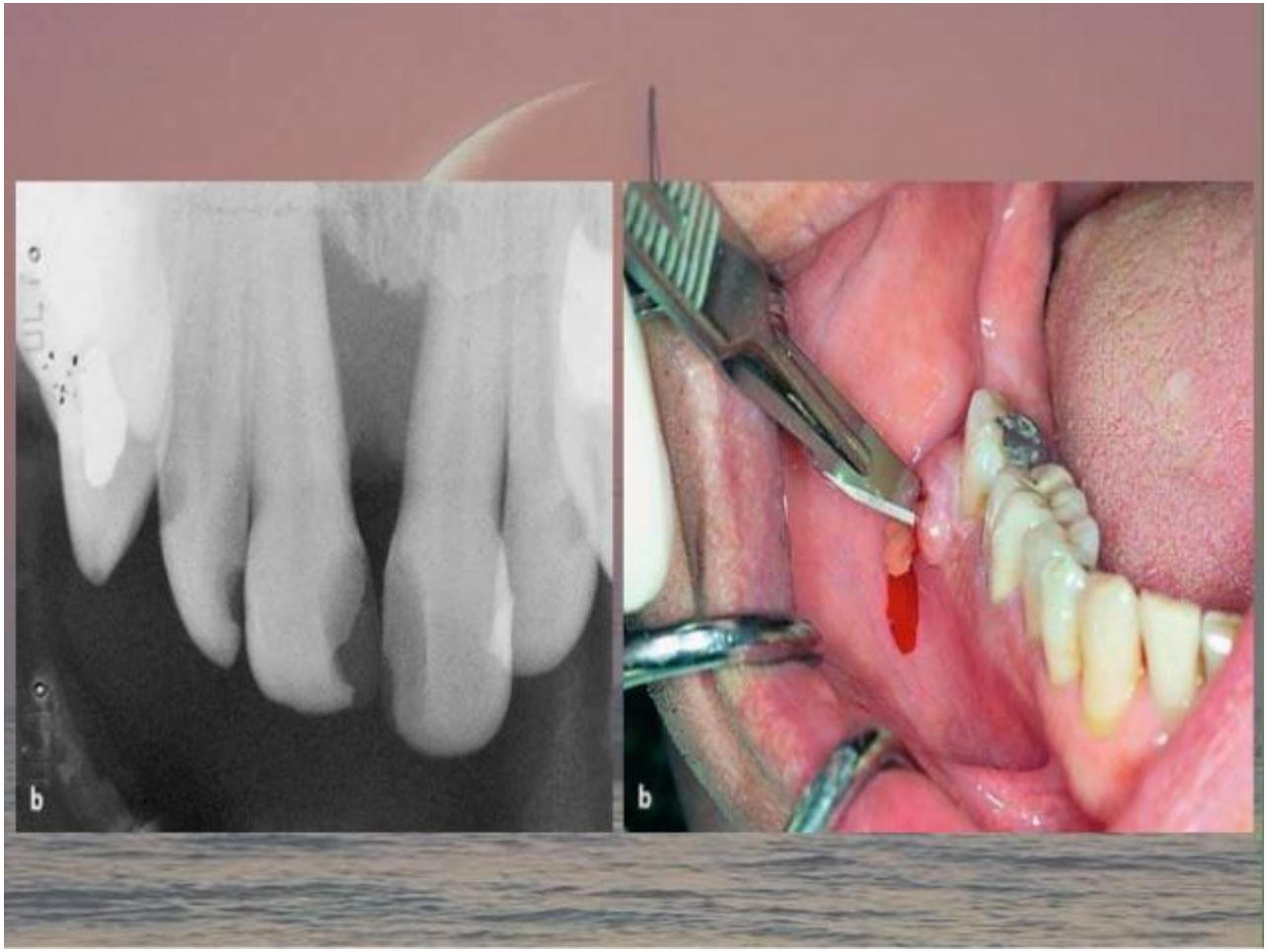
Odontogenic infections

- ***Periapical infections***



Periodontal infections

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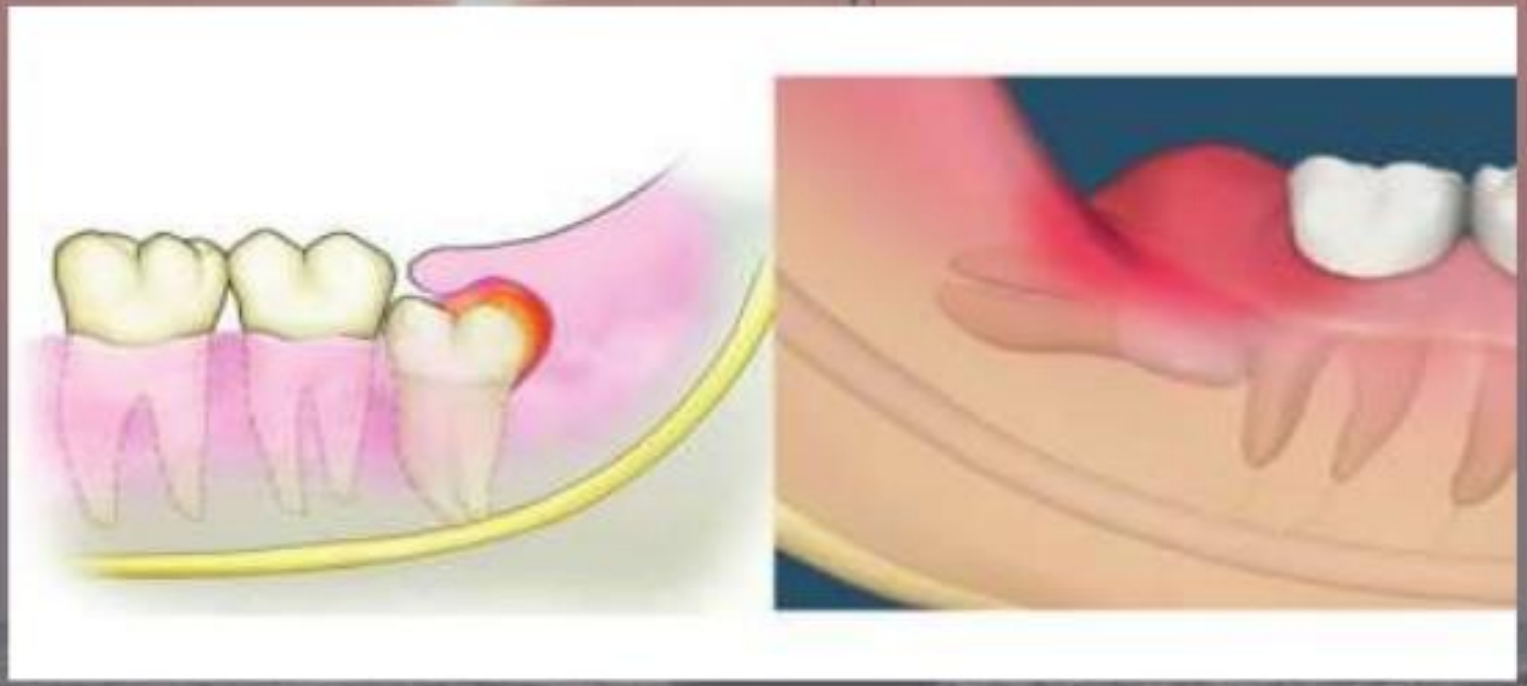
Pericoronal infections



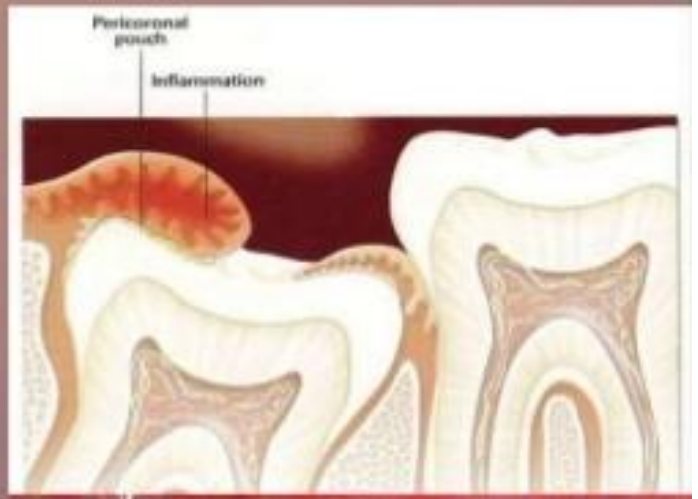
Pericoronal infections

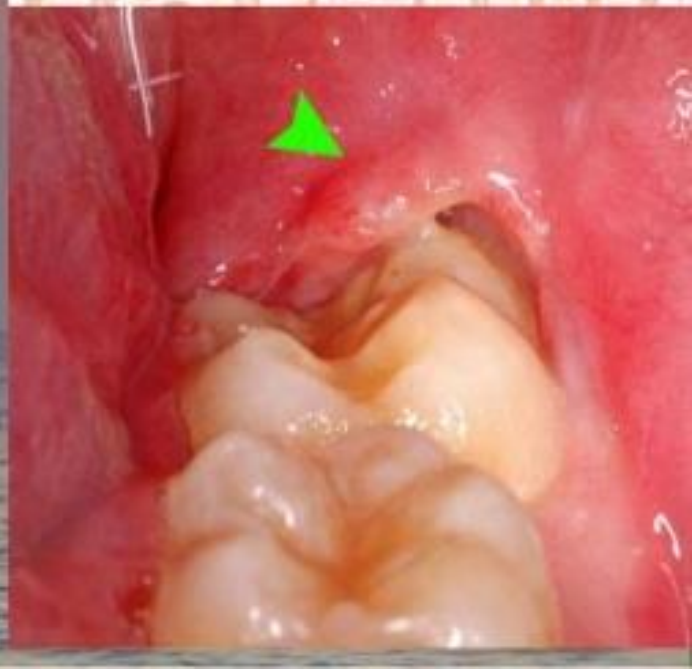
Etiology :

- Food impaction
- Trauma from opposing tooth
- Virulent micro-organism
- Lowering of the host resistance

Treatment

- Drainage of pus if present
- If no pus ,gentle irrigation of the pericoronal space with normal saline
- Extraction of opposing upper tooth or selective grinding
- Antibiotic
- Extraction of impacted tooth






Bacteriology
• Mostly mixed infections (aerobic and anaerobic)

Spread of odontogenic infections

- ***Cellulitis :spread of infection into the loss ct***
- ***Suppurative infections : charectarized by abscess formation***

Spread of odontogenic infections

Odontogenic infections may spread beyond the confines of the dentoalveolar bone into the soft tissue, they may be presented as:

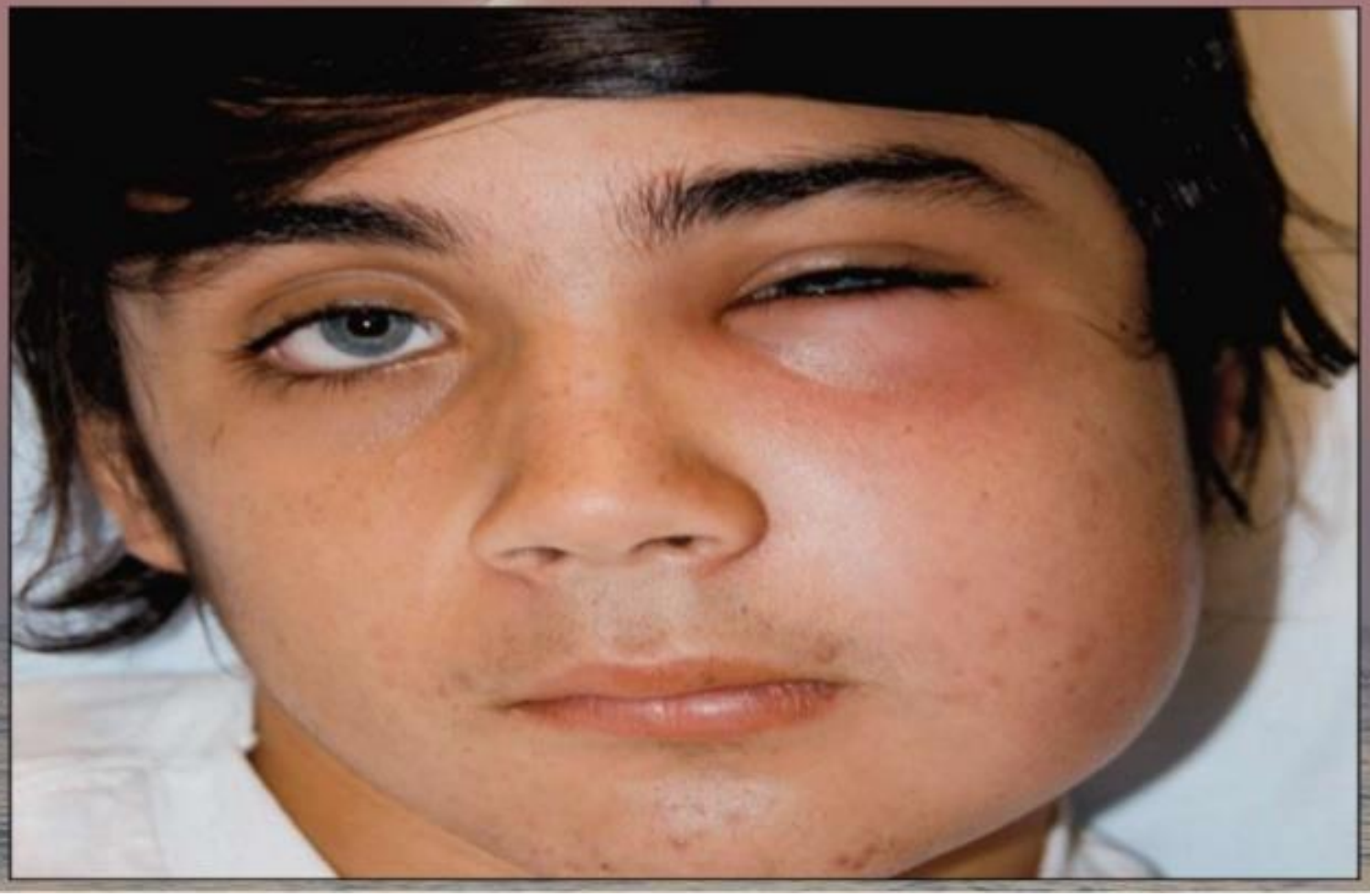
Cellulitis; which results from spreading of infection into the loose connective tissues (C.T.),it presents as a warm, diffuse, erythematous, indurated and painful mucosa! or cutaneous swelling. It does not result in the formation of large amount of pus, Streptococci are more often associated with cellulitis, and these organisms produce enzymes such as Streptokinase and Hyaluronidase that break down fibrin and C.T. ground substance, facilitating the rapid spread of Infection.

Antibiotics and removal of the cause of infection are usually sufficient Incision and drainage are indicated if there is no improvement or if evidence of purulent collection is identified.

Suppurative infections; characterized by abscess formation, which can be defined as a thick walled pocket of tissue containing pus. Pus consists of necrotic tissue, dead and living bacteria and dead white cells, it is often associated with Staphylococci and anaerobes such as Bacteroids. S. aureus produce the enzyme Coagulase that coats the bacteria with fibrin and reduces the ability of the host cells to phagocytize it.

The area of infection may or may not be fluctuant, treatment is by incision and drainage with antibiotics, followed by the treatment of the cause of infection.

Cellulitis



Suppurative infection

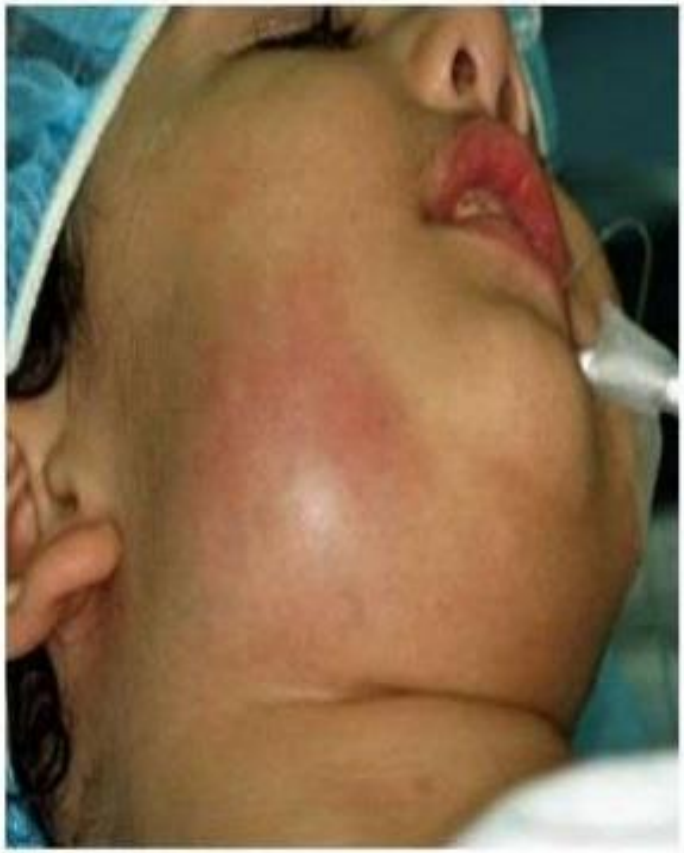


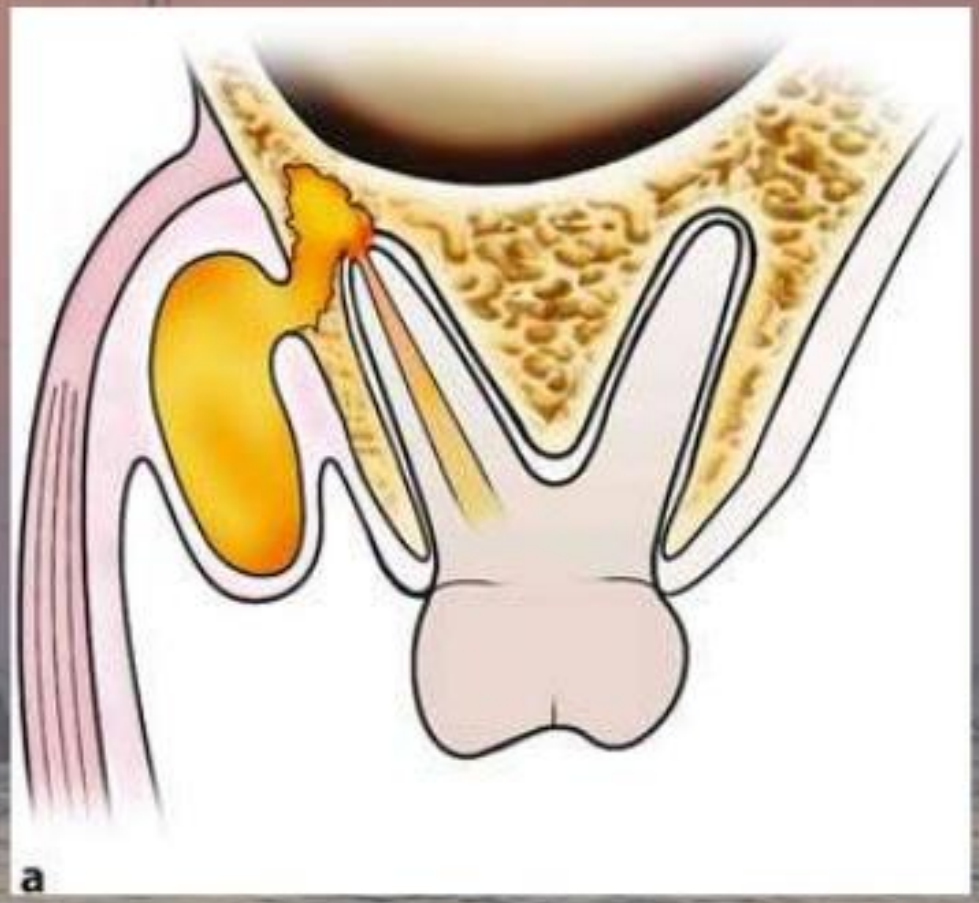
TABLE 15-3

General Differences Between Cellulitis and Abscess

Characteristic	Cellulitis	Abscess
Duration	Acute	Chronic
Pain	Severe and generalized	Localized
Size	Large	Small
Localization	Diffuse borders	Well circumscribed
Palpation	Doughy to indurated	Fluctuant
Presence of pus	No	Yes
Degree of *seriousness*	Greater	Less
Bacteria	Aerobic	Anaerobic

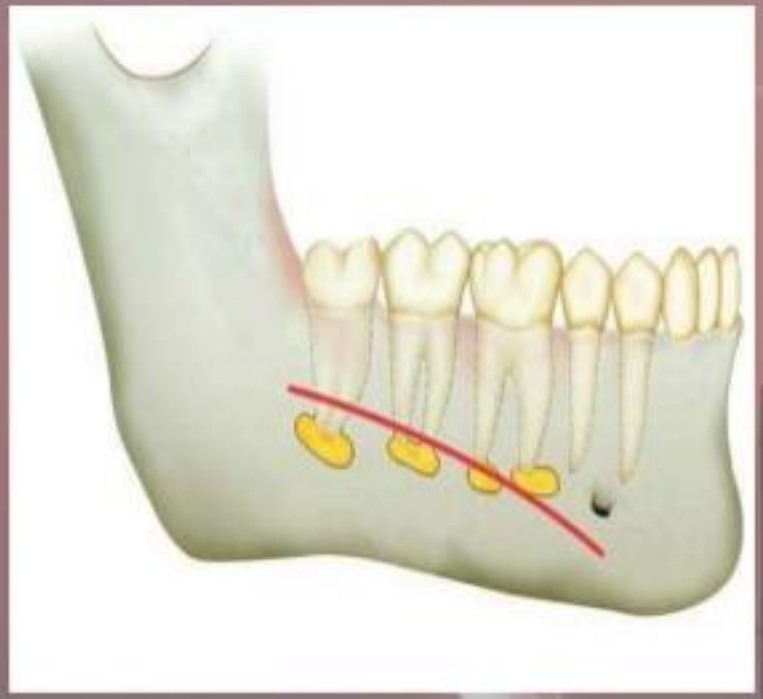
Routes of infection spreading

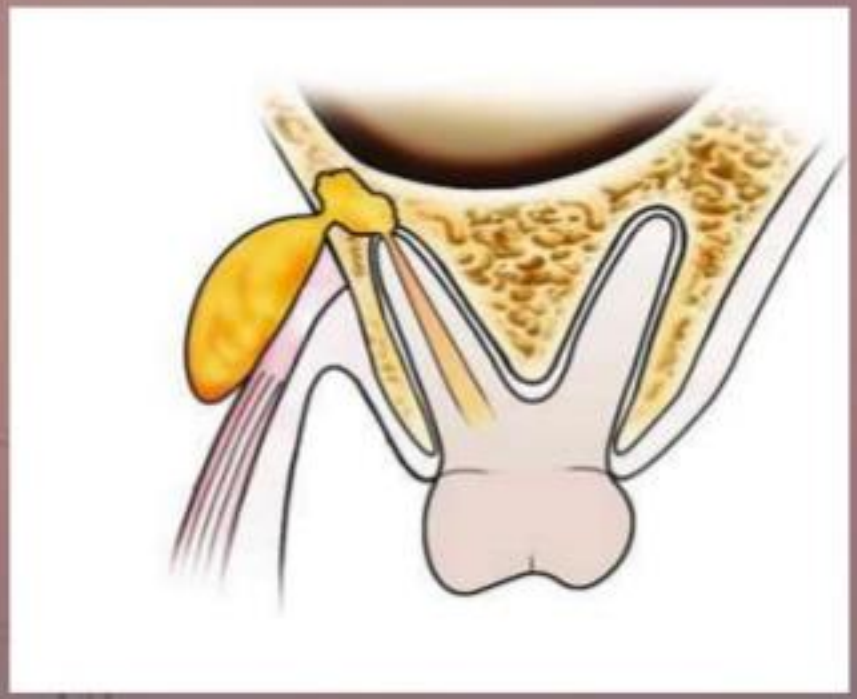
- Direct continuity
- Lymphatic
- Blood stream

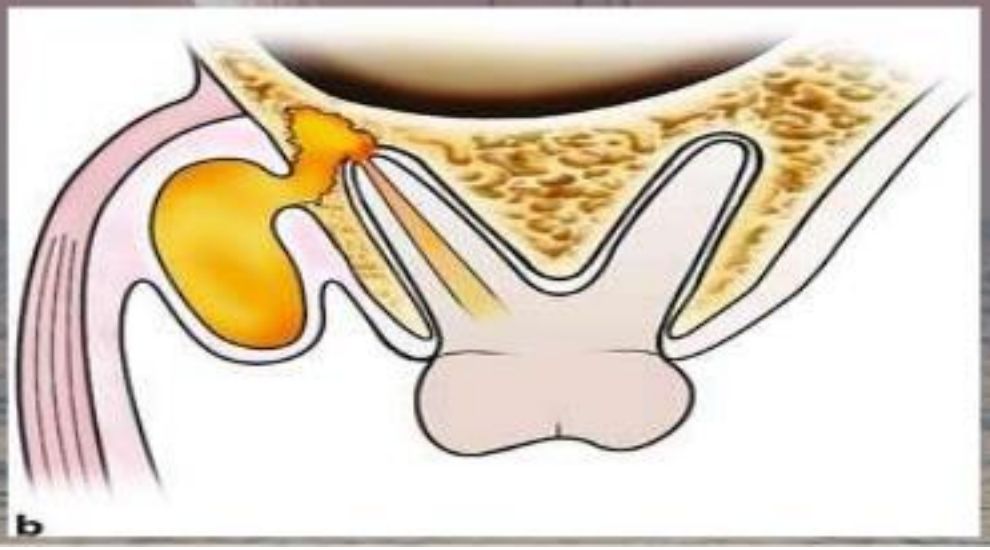


Factors that influence the spread of odontogenic infections

- Virulence of micro-organism
- Patient immune system
- Anatomical factors that influence the direction of infection spread :
    - A. site of source of infection
    - B.point at which pus escape
- Natural barriers (fascia ,muscle,bone)



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Physical examination

Include inspection, palpation and percussion. It aims to identify the signs and symptoms of infection like the presence of swelling, redness or draining fistula.

Palpation is used to examine the size, note the tenderness, assess local temperature and to determine the presence of fluctuation. Trismus should be noted with measurement of the interincisal distance.

Intraorally, the teeth and gingiva should be examined for the presence of caries, restorations or localized swelling. Percussion determines the areas of tenderness. Pulp testing may be needed to assess the vitality of the teeth. The intraoral examination should include the ducts of the parotid and submandibular salivary glands, tongue, soft palate, tonsillar fossa and oropharynx.

Manifestations of serious odontogenic infections may include; airway compromise, septicemia, fever, lethargy, fatigue, dehydration, rapid progression, dysphagia, odynophagia and drooling.

Other investigations are; radiographs to identify the cause of infection, periapical, occlusal and OPG radiographic views are used. Ultrasound, CT scan and MRI can also be used in deep infections.

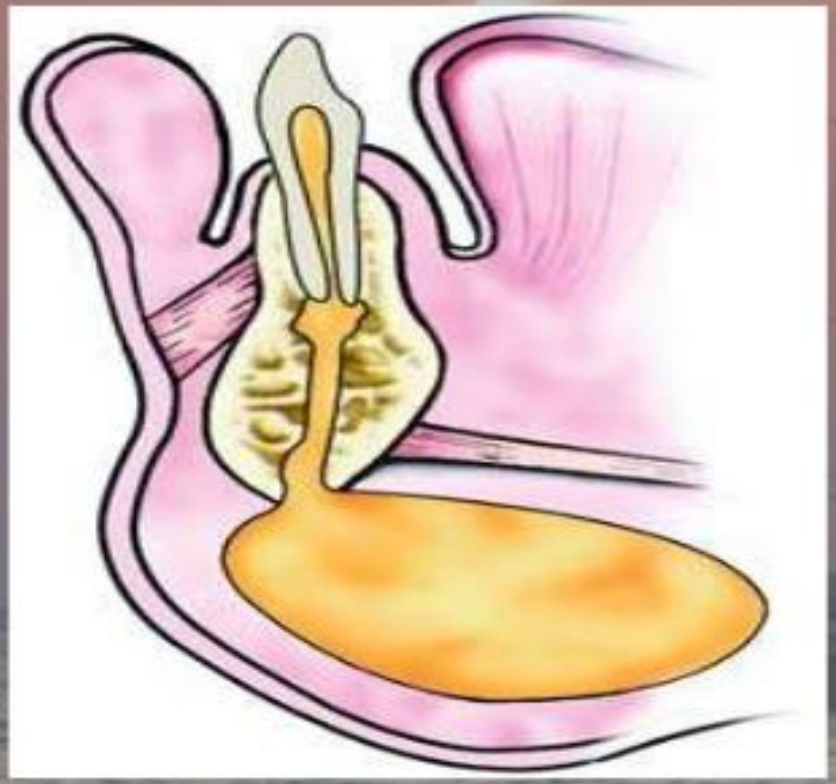
Laboratory studies are also indicated to evaluate the immune system, white blood cells (WBC) and differential WBC count.

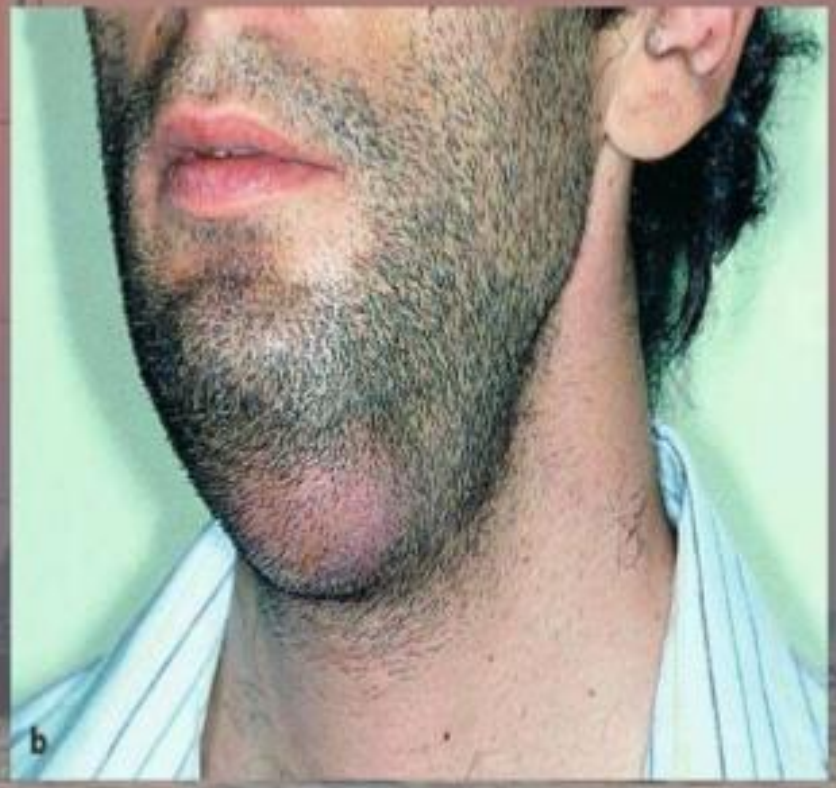

Infection of facial spaces

Fascial spaces are potential spaces, they are surrounded by muscles, loose C.T. and bone, they contain different anatomical structures and they are separated by collection of pus, blood or by surgeon's finger. They are contiguous and infection spreads readily from one space to another. A thorough knowledge of the anatomy of the face and neck is necessary to predict the pathways of spread of infections.

Fascial spaces are sometimes classified as **primary;** which are directly adjacent to the origin of the odontogenic infections, and **secondary;** that become involved following the spread of infection to the primary spaces.

Submental space infections





1. Submental space infection

Anatomic boundaries this space lies between the Mylohyoidmuscle above, skin, subcutaneous tissue, Platysma muscle and deep cervical fascia below, laterally by lower border of the mandible and anterior bellies of Digastric muscle. It contains submental lymph nodes embedded in adipose tissues.

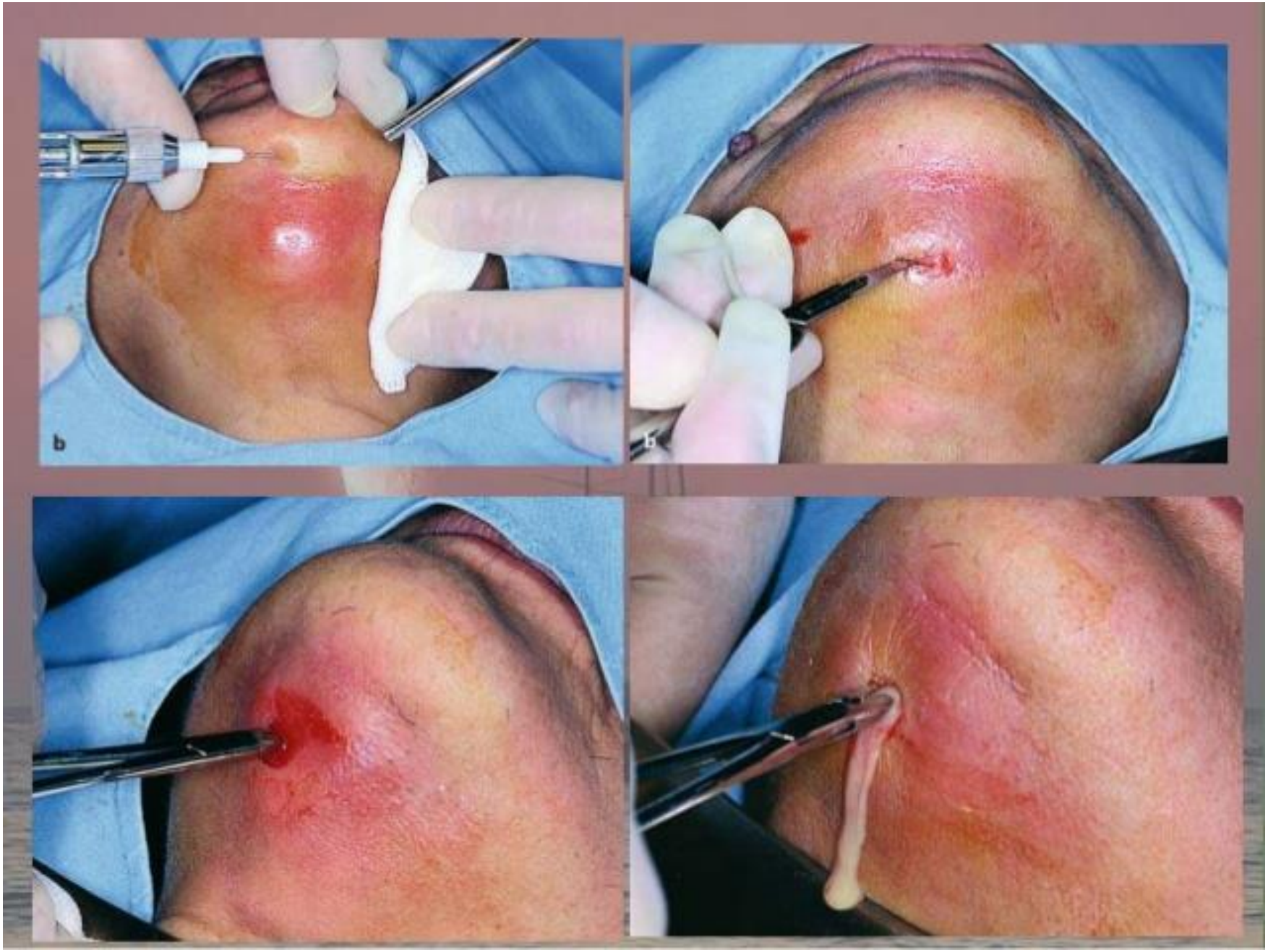
Source of infection a direct source is from infected lower incisors and canines, lower lip, skin overlyingthe chin or from the tip of the tongue and the anterior part of the floor of the mouth.

An indirect source of infection is from submandibular spaces.

The site of the swelling is mostly extraoral including the chin and submental areas which are firmlyswollen.

The site of incision and drainage is extraoral horizontal incision through the skin posterior to the crease behind the chin, providing dependent drainage and most esthetically acceptable scar.

It may be drained intraorally through the Mentalis muscle via the labial vestibule, but the dependent drainage can not be established.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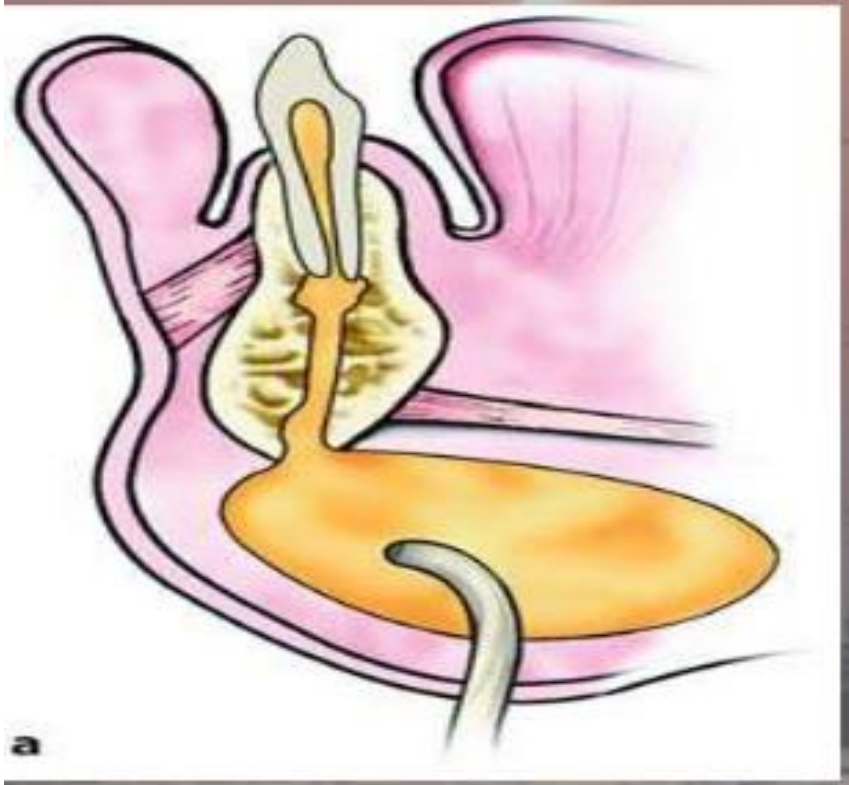
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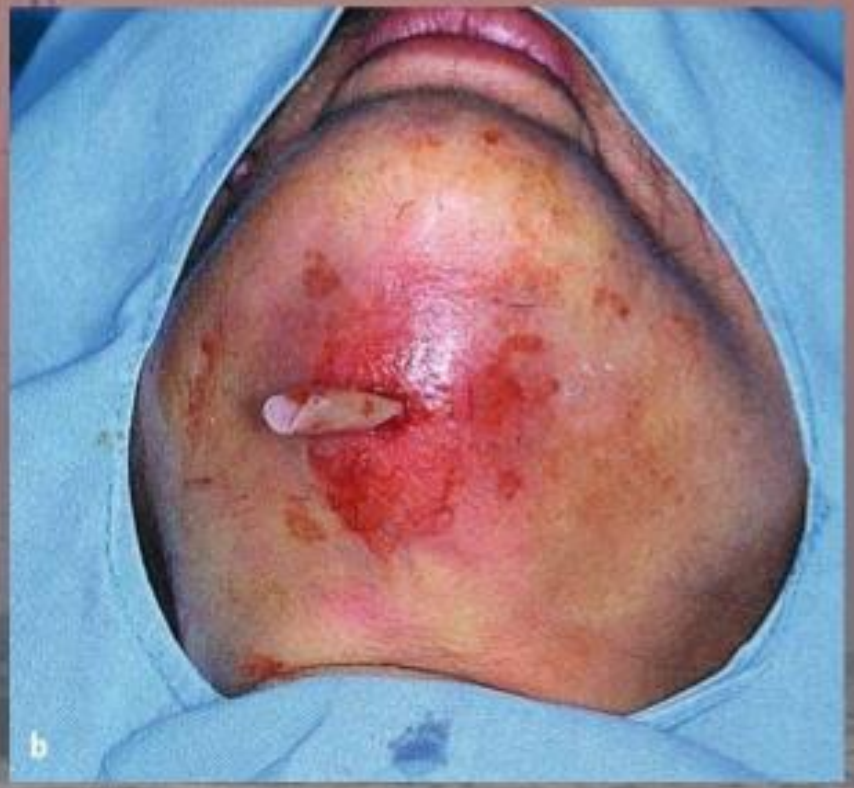
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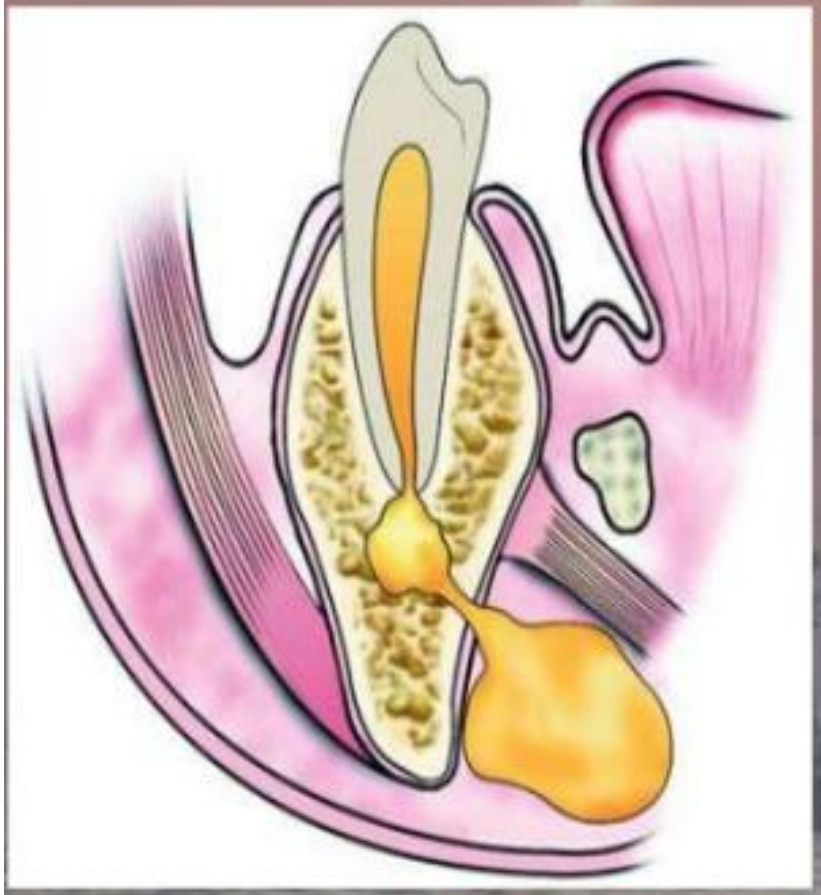
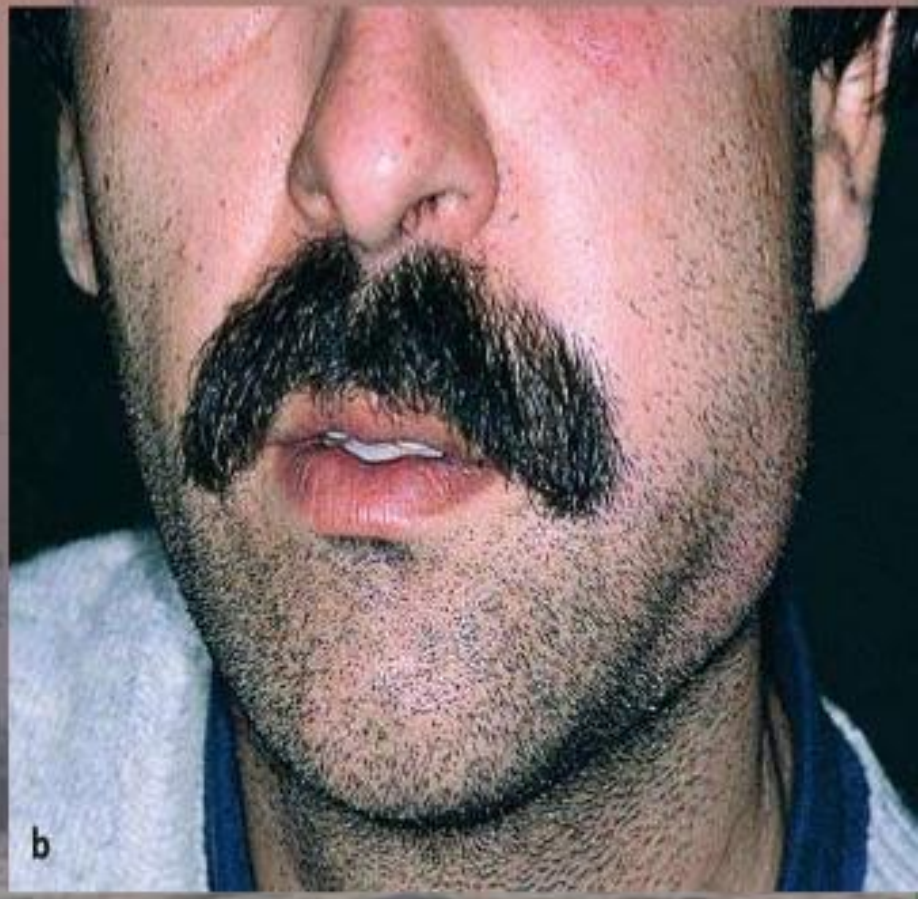
Submandibular space infections





Submandibular space infection

Anatomic boundaries it is bounded by Mylohyoid muscle superiorly, anterior and posterior bellies of Digastric muscle inferiorly, Mylohyoid, Hyoglossus and Styloglossus muscles medially, laterally the space is bounded by the skin, superficial fascia, Platysma, deep fascia and the lower border of the mandible. This space contains the submandibular salivary gland and lymph nodes in addition to facial artery and vein, lingual and Hypoglossal nerve as they course deep to the submandibular salivary gland.

Source of infection is from the lower molar teeth especially second and third molars, as the infection perforates the lingual cortex of the mandible below the Mylohyoid muscle attachment.

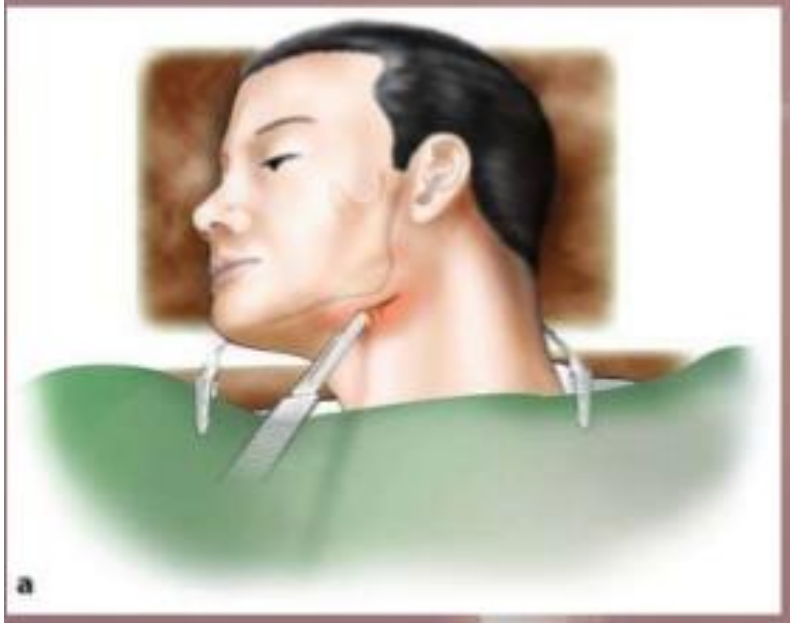
Infection can also spread from the tongue, posterior part of the floor of the mouth, upper posterior teeth, cheek, palate, the maxillary sinus and the submandibular salivary gland.

Indirectly the infection may spread from infected sublingual and submental spaces.

Submandibular space infections can spread posteriorly to the pharyngeal space.

Submandibular space infection presents as a firm or fluctuant erythematous swelling of the submandibular region, the swelling bulges over and obliterates the inferior border of the mandible, there may be trismus, other signs and symptoms of infection may or may not be present.

Site of incision and drainage it is extraoral incision made parallel and about 2 cm. below the inferior border of the mandible to avoid injury to the marginal mandibular branch of the facial nerve, the inciston extends through the skin and subcutaneous tissue only, While the space is entered bluntly to avoid structures within the space.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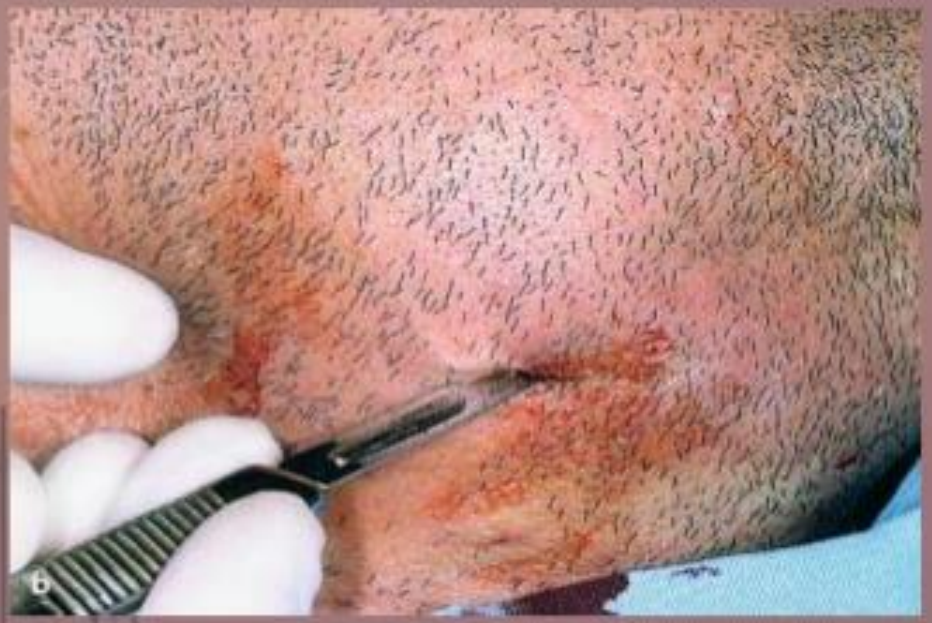
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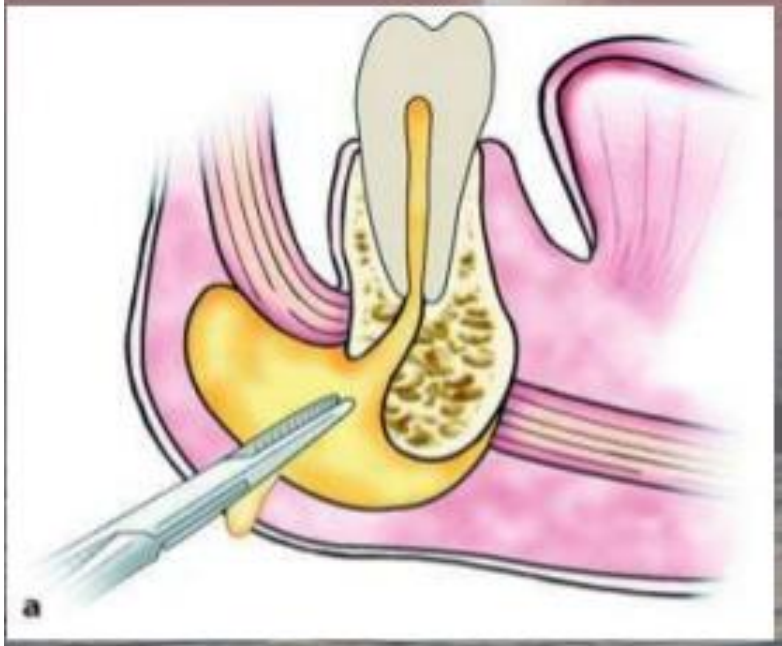
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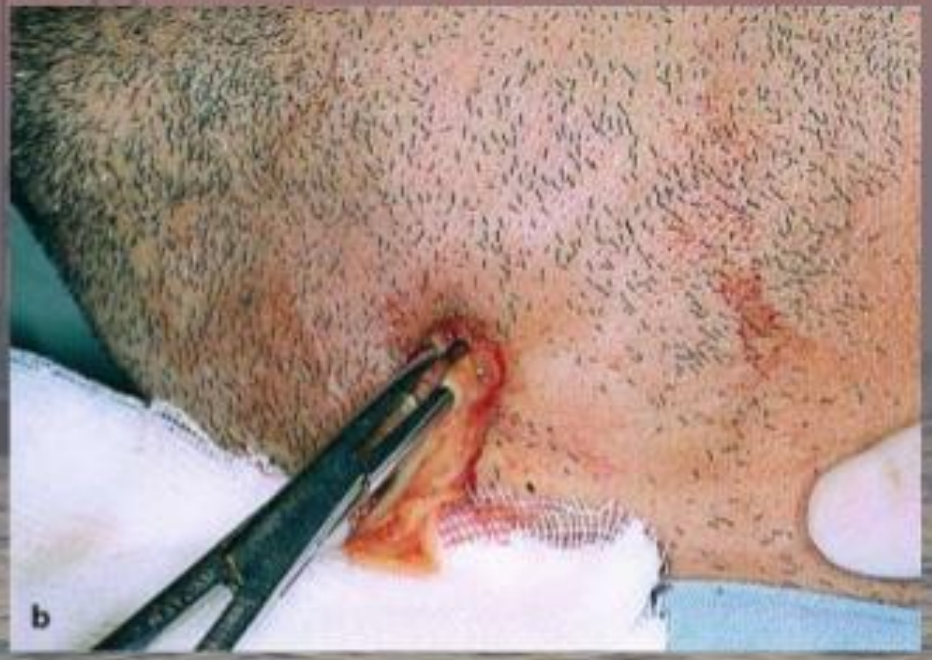
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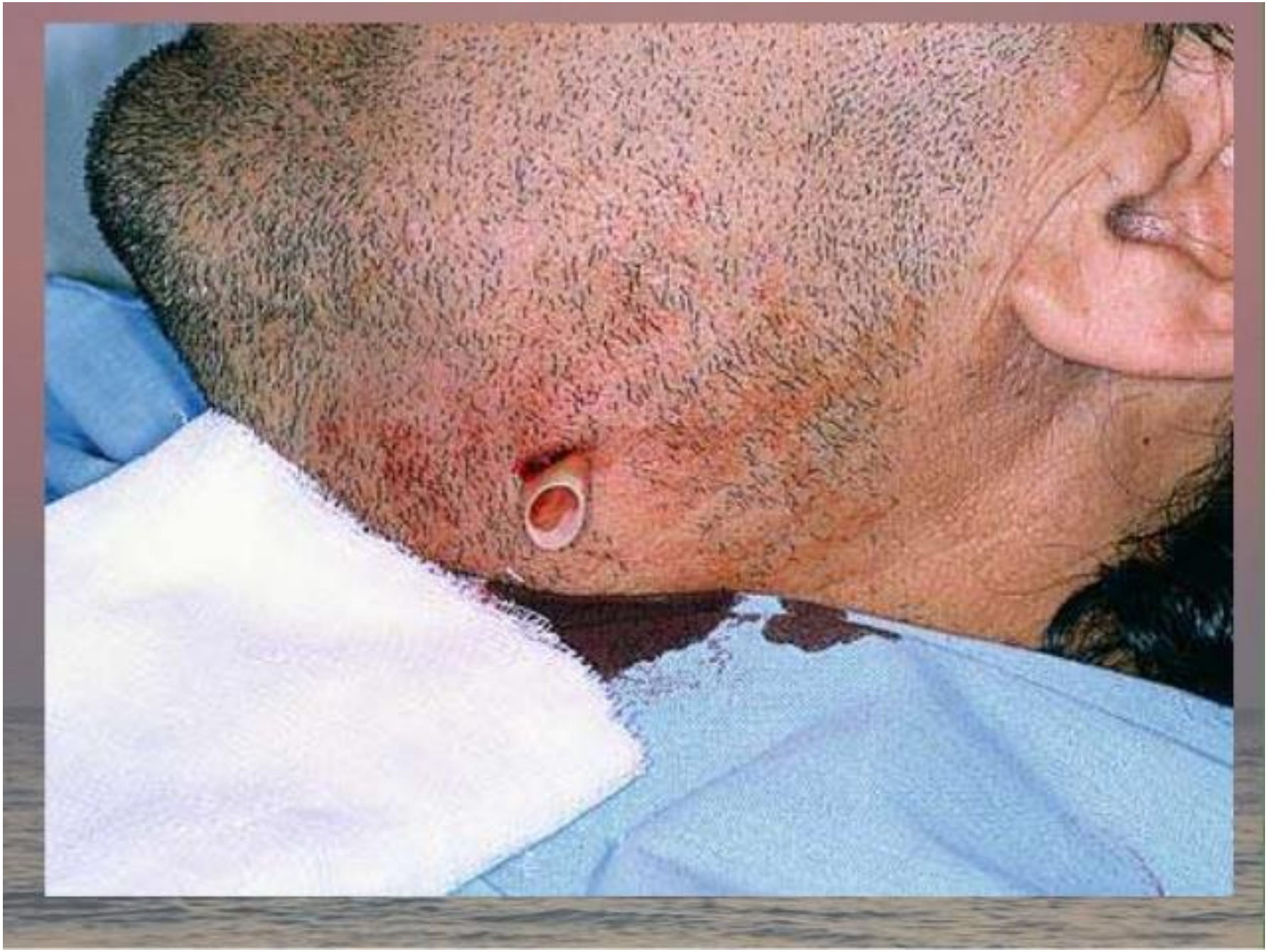
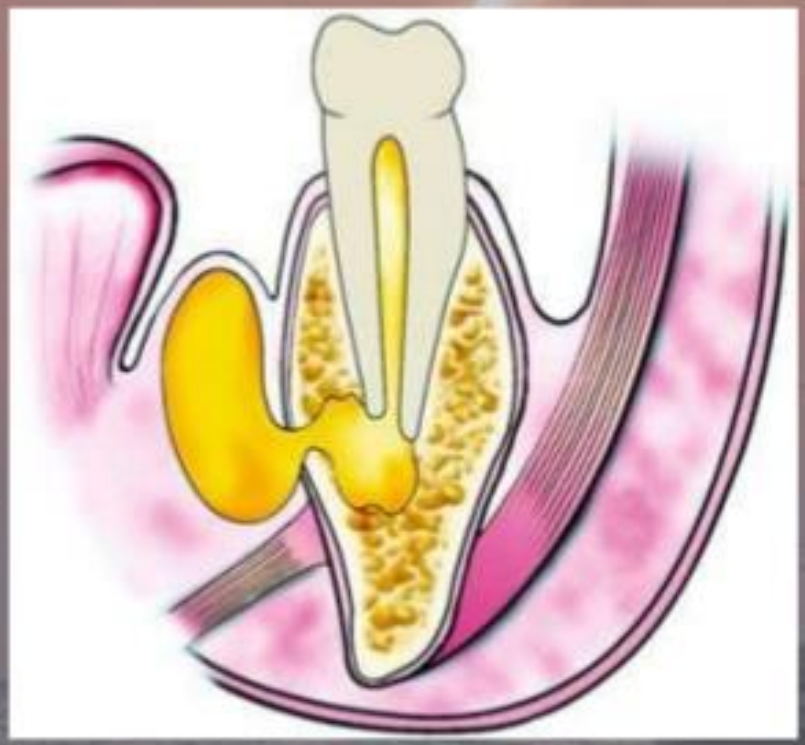
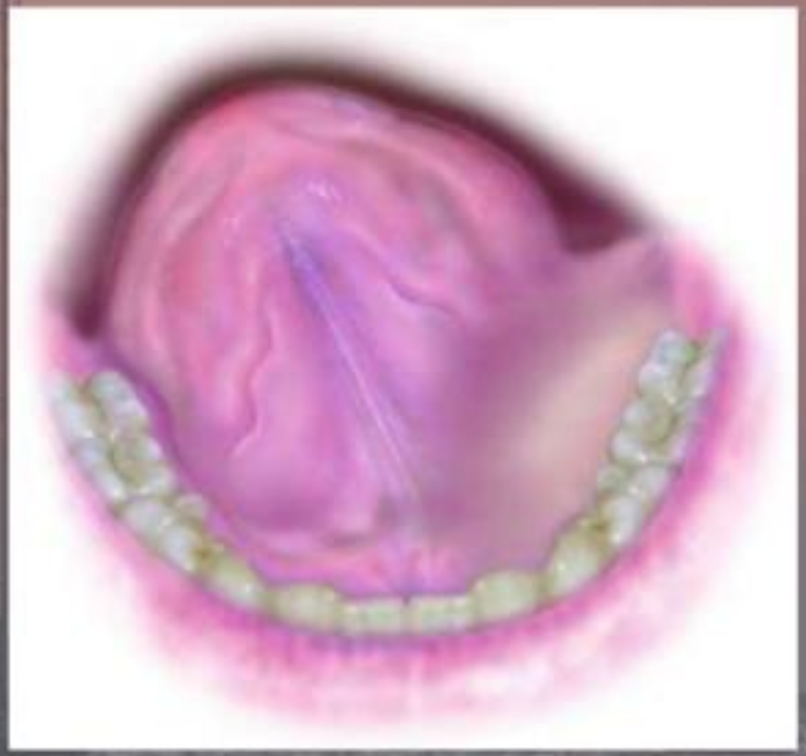


Sublingual space infections





Sublingual space

Anatomic boundaries this is a V- shaped space , it is bounded anteriorly and laterally by the mandible, superiorly by sublingual mucosa, inferiorly by the Mylohyoid muscle and medially by Genioglossus, Geniohyoid and Styloglossus muscles.

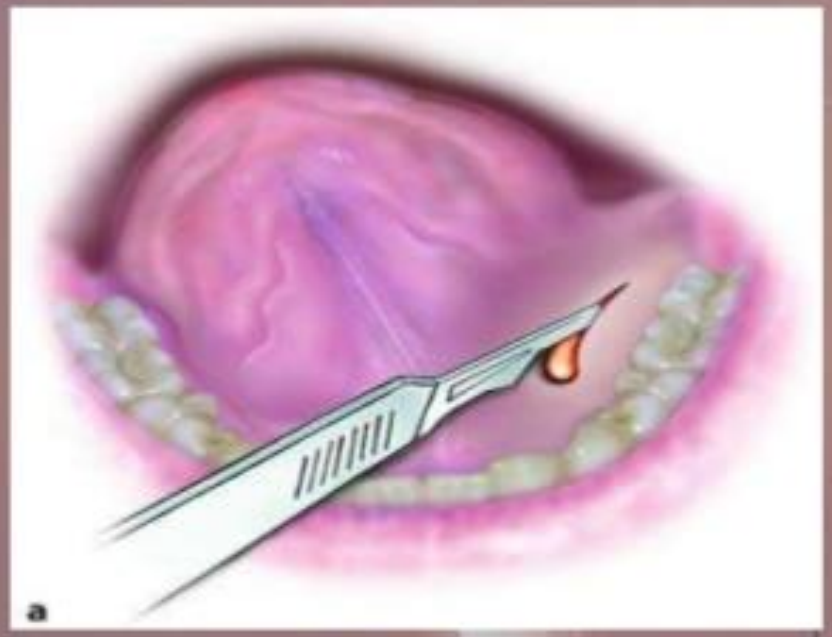
Source of infection it is usually from the premolar and less commonly from molar teeth when the infection perforates the lingual cortex of the mandible above the attachment of the Mylohyoid muscle.

Indirectly the infection may spread from submental andsubmandibular spaces. Infection from sublingual space may invade the submandibular and pharyngeal spaces.

Clinically there is erythematous swelling of the floor of the mouth that may extend through the midline since the barrier between the two sublingual spaces is weak, usually there is elevation of the tongue.

Site of incision and drainage intraorally by an incision through the mucosa only parallel to Wharton's duct and lingual cortex in anteroposterior direction and away from the sublingual fold.

This space may be drained extraorally through submandibular and submental incisions through the Mylohyoid muscle if the infection of these latter spaces is also evident.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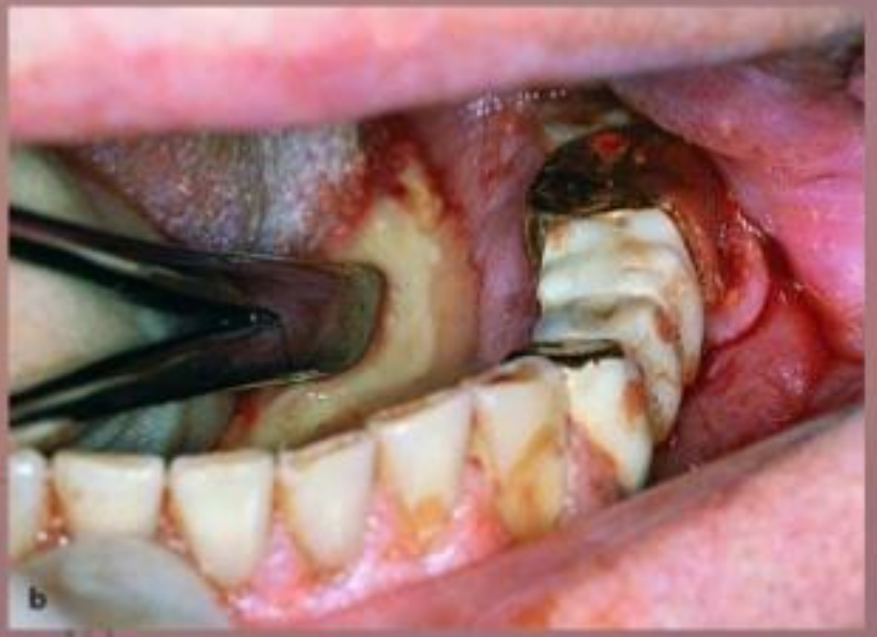
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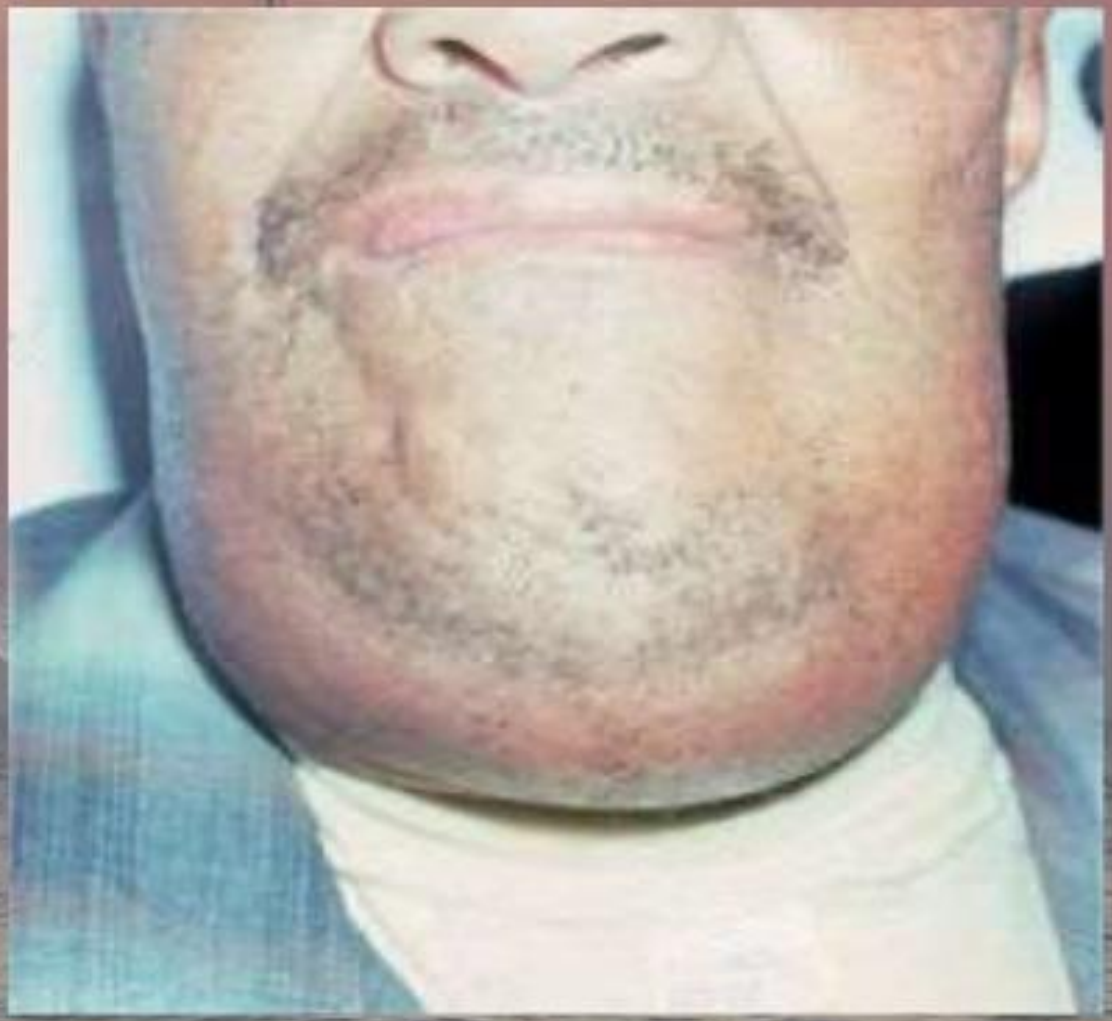
Ludwigs angina



Ludwig's Angina

It is a massive firm cellulitis, affecting simultaneously the submandibular, submental and sublingual spaces bilaterally. It is a very serious condition that require prompt treatment, it was described by Wilhelm Friedrich von Ludwig in 1836.

Causes

- Dental infections in 90% of the cases.
- Submandibular salivary gland infections.
- Mandibular fractures.
- Soft tissue lacerations and wounds of the floor of the mouth

The term angina is related to the sensation of suffocation. If untreated this condition is almost fatal mainly due to posterior extension of the infection into the epiglottis causing epigllotic edema and respiratory obstruction.

Signs and Symptoms:

There is a firm extensive bilateral submandibular swelling, intraorally there is swelling of the floor of the mouth that raises the tongue which may protrude from the mouth in extreme cases. The patient is toxic, feverish and there is dyspnea and difficulty in swallowing.

Treatment

- Securing the airway, endotracheal intubation is very difficult in this situation, tracheostomy may be needed, but it is also difficult to perform due to the massive neck edema.
- General anesthesia should be avoided.

Early surgical drainage of all the infected spaces bilaterally under local anesthesia, little pus is obtained since the infection is usually cellulitis.

- Intravenous antibiotic, using a combination of Penicillin and Metronidazole.

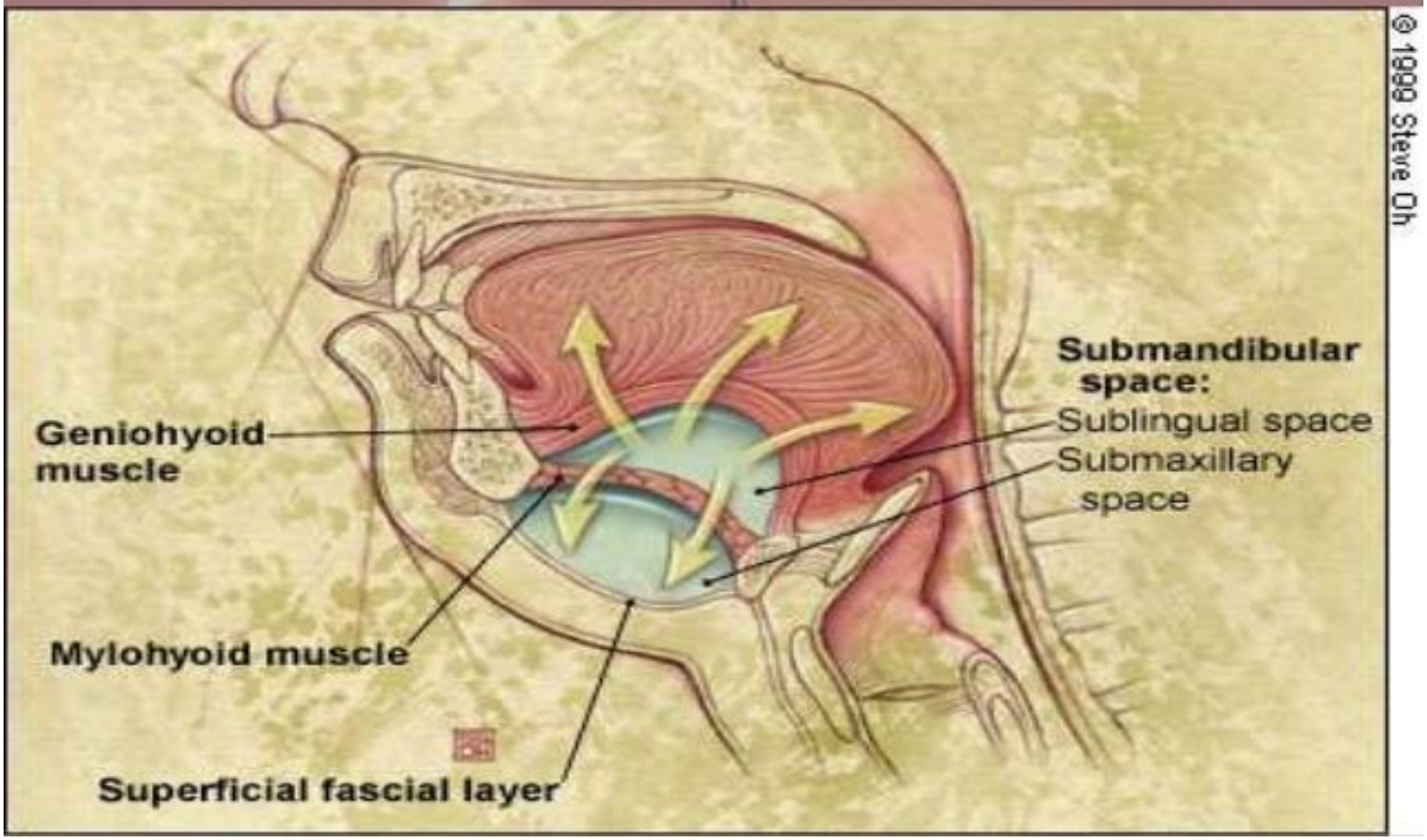
Some disciplines advocate high dose of antibiotics without surgery until fluctuation develops.

Ludwigs angina

- Causes

- Dental infections(90%)
- Sunmandibular gland infections
- Mandibular fractures
- Soft tissue laceratios and wounds in the floor

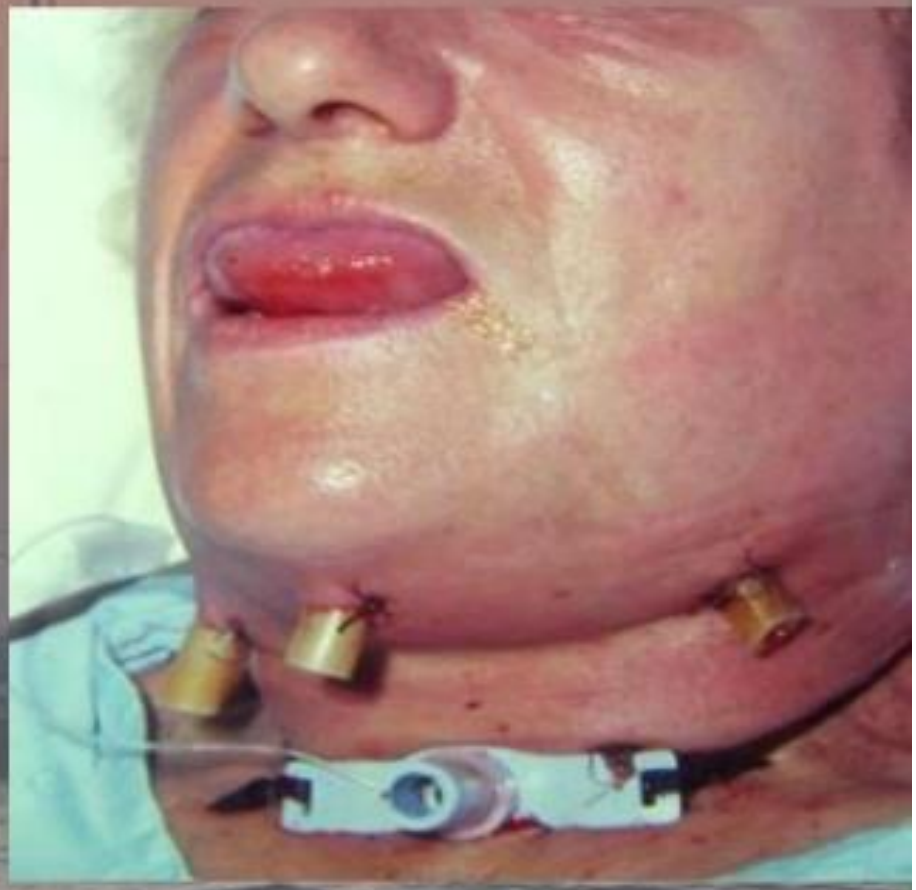
Signs and symptoms



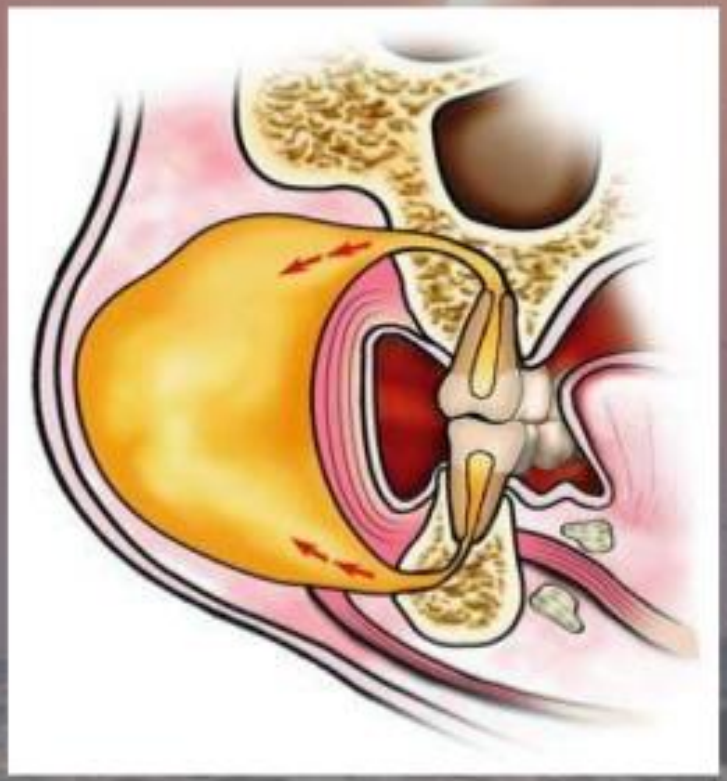
Classical s/s in addition to dyspnea and dysphagia

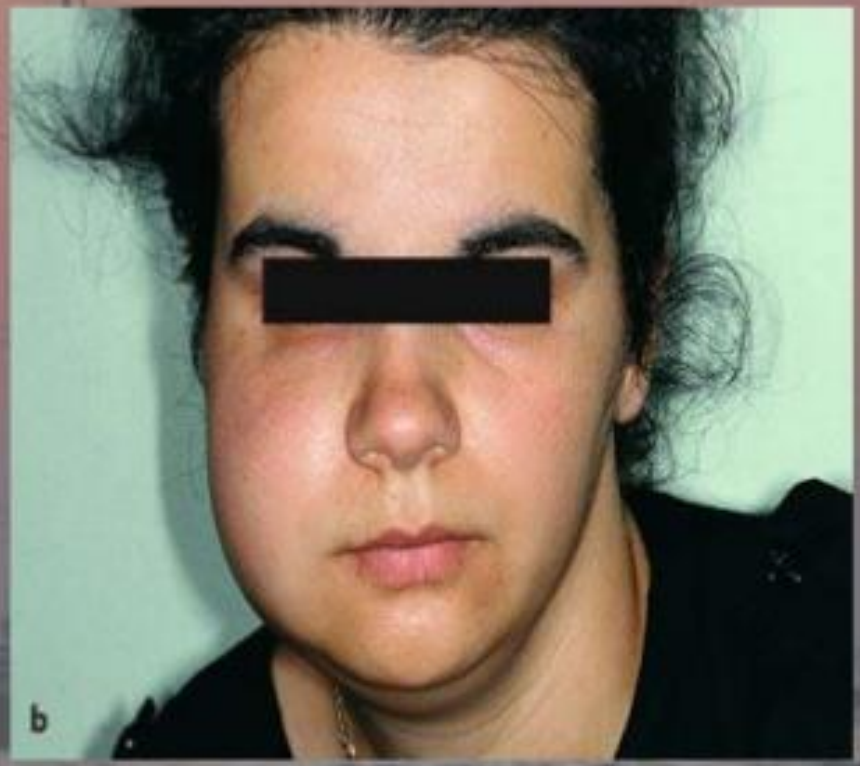
Treatment

- Securing airway
- GA should be avoided
- Surgical drainage
- AB



Buccal space infection





Buccal space infection

Anatomic boundaries bounded by the Buccinator muscle and buccopharyngeal fascia medially, skin of the cheek laterally, labial musculature anteriorly, zygomaticarch superiorly, the inferior border of the mandible inferiorly and the pterygomandibular raphe posteriorly.

It contains the buccal pad of fat, facial artery and the parotid duct.

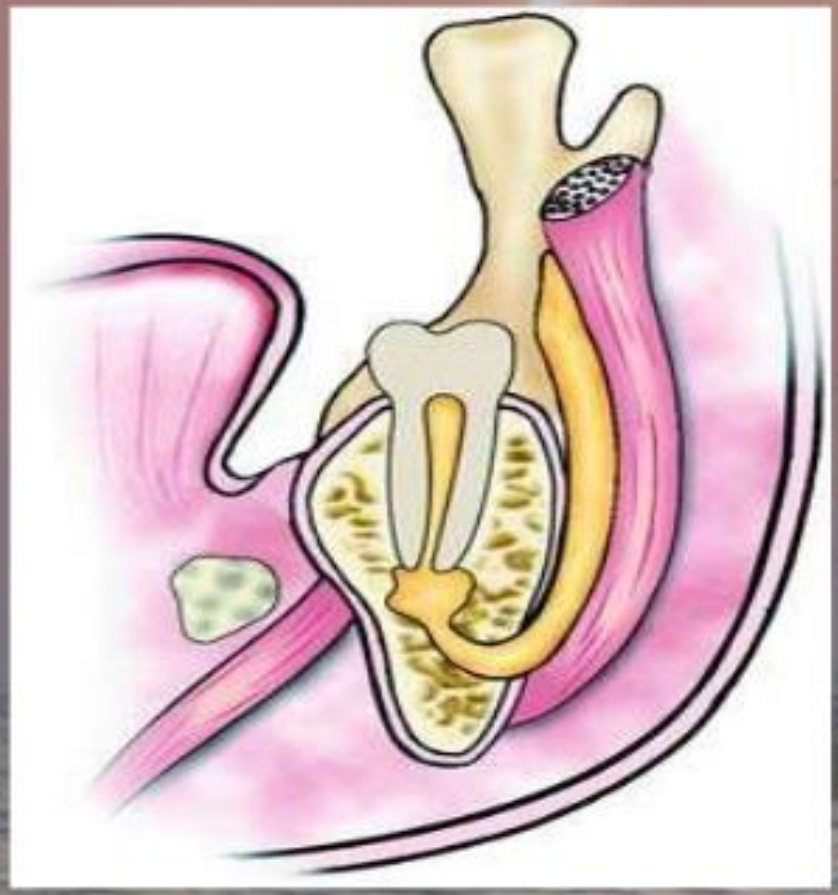
Source of infection of this space can be related to both jaws. The relationship of the origin of the Buccinator muscle from the alveolar bone and the species of the upper and lower premolars

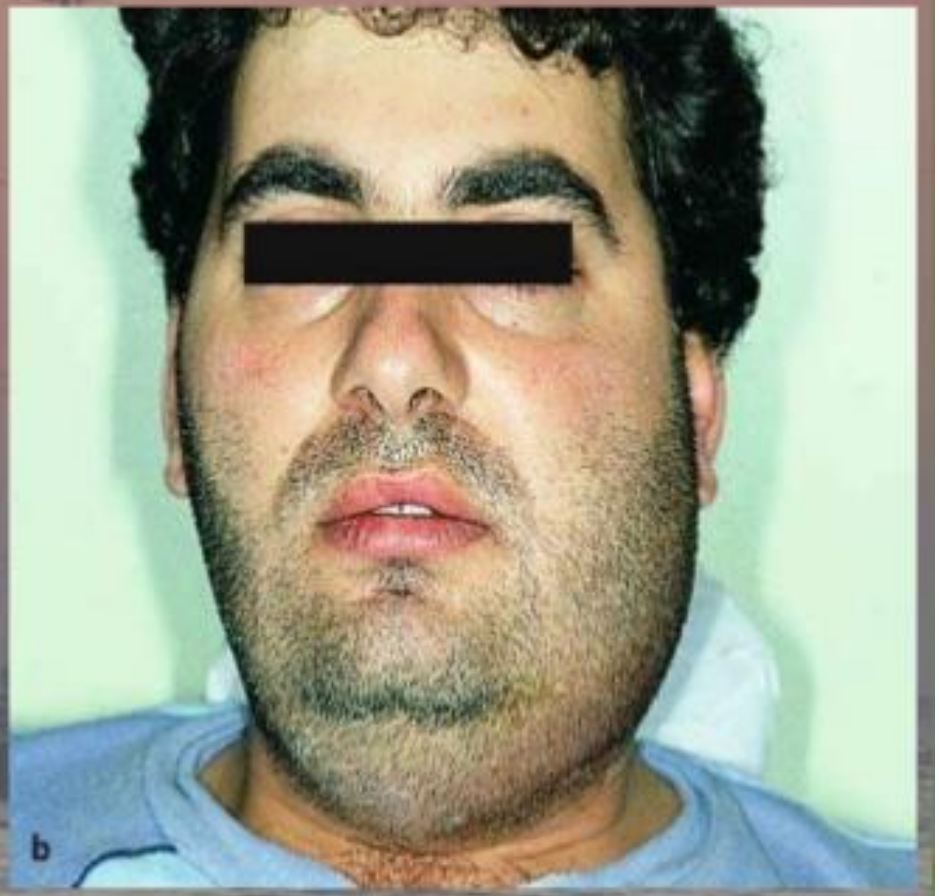
and molars determines the direction of the spread of infection from these teeth. If the infection exits the alveolar bone above the attachment of the muscle in the upper alveolus or below the attachment in the lower alveolus, the infection spreads to the buccal space. Otherwise the infection spreads intraorally into the vestibule where it can be drained easily. Usually the swelling appears in the cheek, the inferior border of the mandible can still be palpated.

Site of incision and drainage intraorally by a horizontal incision in the buccal mucosa below the parotid duct, the incision should be through the mucosa only, the space should be entered bluntly using an artery or sinus forceps through the Buccinator muscle to avoid damage to the facial arterv and nerve.

The incision can be placed extraorally if the pus points cutaneously.

Submassetric space infections





. **Masticator spaces infection**

These are well differentiated spaces but they communicate with each other as well as with the buccal, submandibular and pharyngeal spaces. They are:

- Masseteric space.
- Pterygornandibular space.
- Temporal space.

Masseteric space Infectjon (also called submasseteric space)

Anatomic boundaries this space lies between the outer surface of the ascending ramus of the mandible medially, the Masseter muscle laterally and the parotid gland posteriorly.

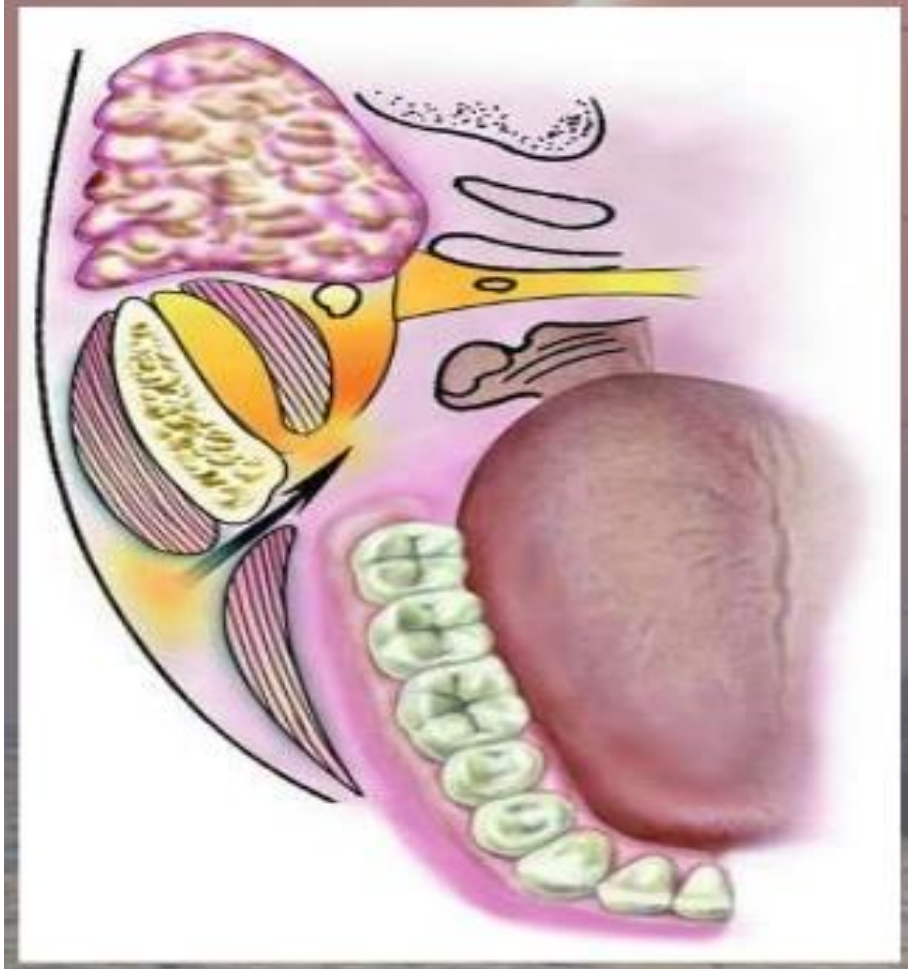
Source of infection usually from molar teeth especially lower third molars, it can also occur after fracture of the angle of the mandible or it can also spread from buccal space.

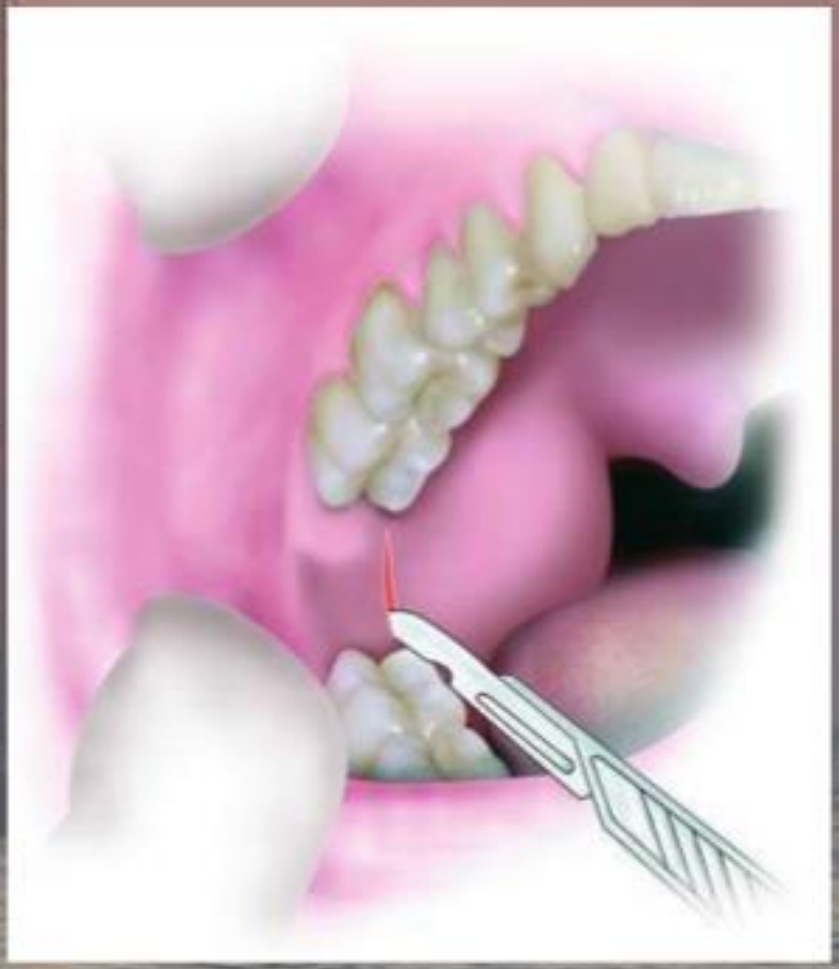
The swelling is moderate in size over the ascending ramus and the angle of the mandible region. This infection is characterized by a marked trismus. Chronic abscess can run a protracted course and can spread to the muscle itself or it can cause osteomyelitis of the ram us of the mandible.

Site of incision and drainage extraorally below and Behind angle of the mandible, the incision is carried through the skin and the subcutaneous tissue then by blind dissection through the platysma muscle and the deep fascia, after incising the attachment of the muscle at the angle the periosteal elevator is inserted beneath the muscle and in close contact with the outer surface of the ramus of the mandible to drain all the pus.

Intraorally drainage can be carried out through an incision along the anterior border of the ramus of the mandible, but in this case the drainage can be insufficient as it is not in a dependent point, also intraoral drainage may prove to be very difficult due to the presence of trismus.

Pterygomandibular Abscess





Pterygomandibular space infection

anatomic boundaries it is bounded medially by the Medial Pterygoid muscle, laterally by the medial surface of the ramus of the mandible, Lateral Pterygoid muscle superiorly, parotid gland posteriorly and the pterygomandibular raphe and the Superior Constrictor muscle of the pharynx anteriorly.

Source of infection usually from molar teeth especially lower third molars, it can also result after inferior dental nerve block with contaminated needle or solution.

Infection can spread from submandibular, sublingual and infratemporal spaces.

Swelling is minimal near the angle of the mandible or sometimes there is no swelling at all, but there is a marked trismus.

Site of incision and drainage extraorally, the same as that described in the masseteric space infections but directed to the inner surface of the ramus.

Intraorally can be drained through an incision made just medial to the pterygomandibular raphe and dissecting along the inner surface of the ramus., but the presence of trismus can prevent efficient drainage.

Temporal space infection

Anatomic boundaries the Temporalis muscle divides this space into two spaces:

- Superficial temporal space; between the muscle and temporal fascia.
- Deep temporal space; between the muscle and the temporal bone.

The temporal space is contiguous with the pterygomandibular and masseteric spaces.

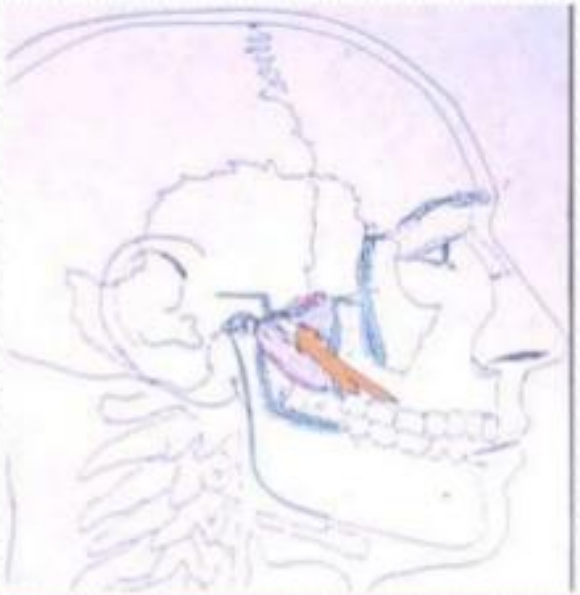
Source of infection upper and lower molars, or by extension from the other masticator spaces.

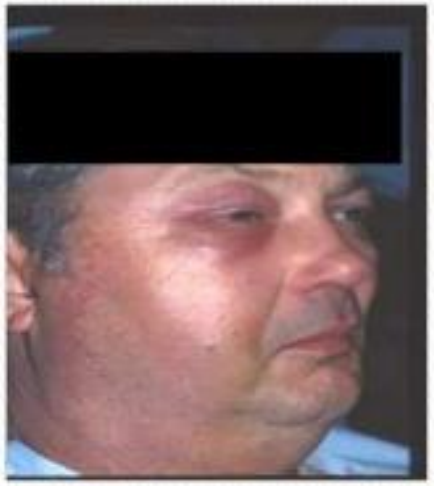
The swelling is behind the lateral orbital rim and above the zygomatic arch, it is almost always associated with trismus.

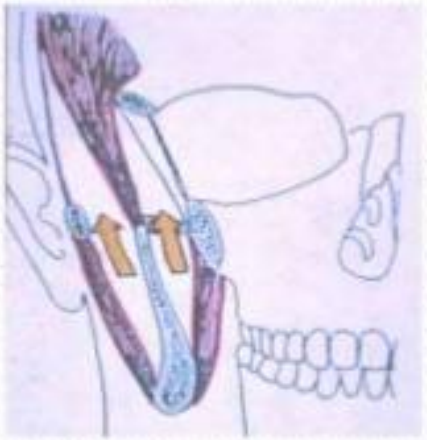
Site of incision and drainage extraoral, through an incision superior and parallel to the zygomaticarch between the lateral orbital rim and the hair line. Intraorally this space can also be drained through an incision along the anterior border of the ascending ramus with the artery forceps directed upwards on the outer aspect of the ramus, but the presence of trismus makes this approach difficult.

Temporal Space Infection

Temporal space:



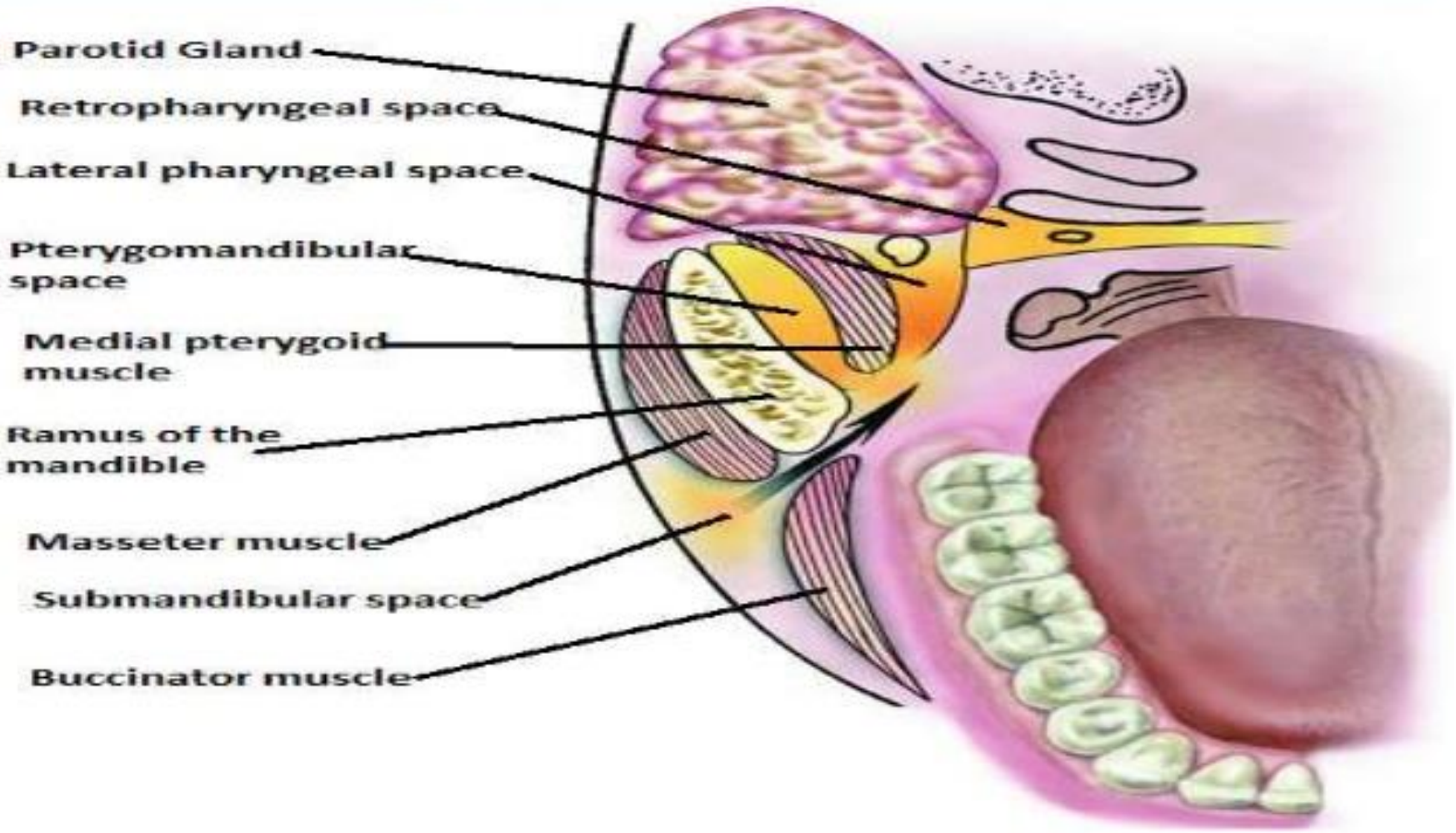




Superficial temporal space:between the temoral fascia and temporalis muscle

Deep temporal space:between temporalis and skull

Lateral Pharyngeal Abscess



Lateral pharyngeal space infection (also termed parapharyngeal space)

Anatomic boundaries this space extends from the base of skull to the hyoid bone, it is conical in shape, the lateral boundaries include the medial surface of the Medial Pterygoid muscle, the medial wall is the Superior Constrictor muscle, Styloglossus muscle, Stylopharyngeus muscle and the Middle Constrictor muscle of the pharynx. Posteriorly by the parotid gland and anterirorly by pterygomandibular raphe.

This space can be divided into two compartments; anterior and posterior, the latter contains the carotid sheath.

Source of infection spread of infection from upper and *lower* molar teeth, most commonly from lower third molar infections by the way of submandibular, sublingua1 and pterygomandibular spaces.

A non-odontogenic infection can spread to this space like tonsillar infections.

Infections of this space are serious, the patient exhibits pain, fever, chills, medial bulge of the lateral pharyngeal wall, extraoral

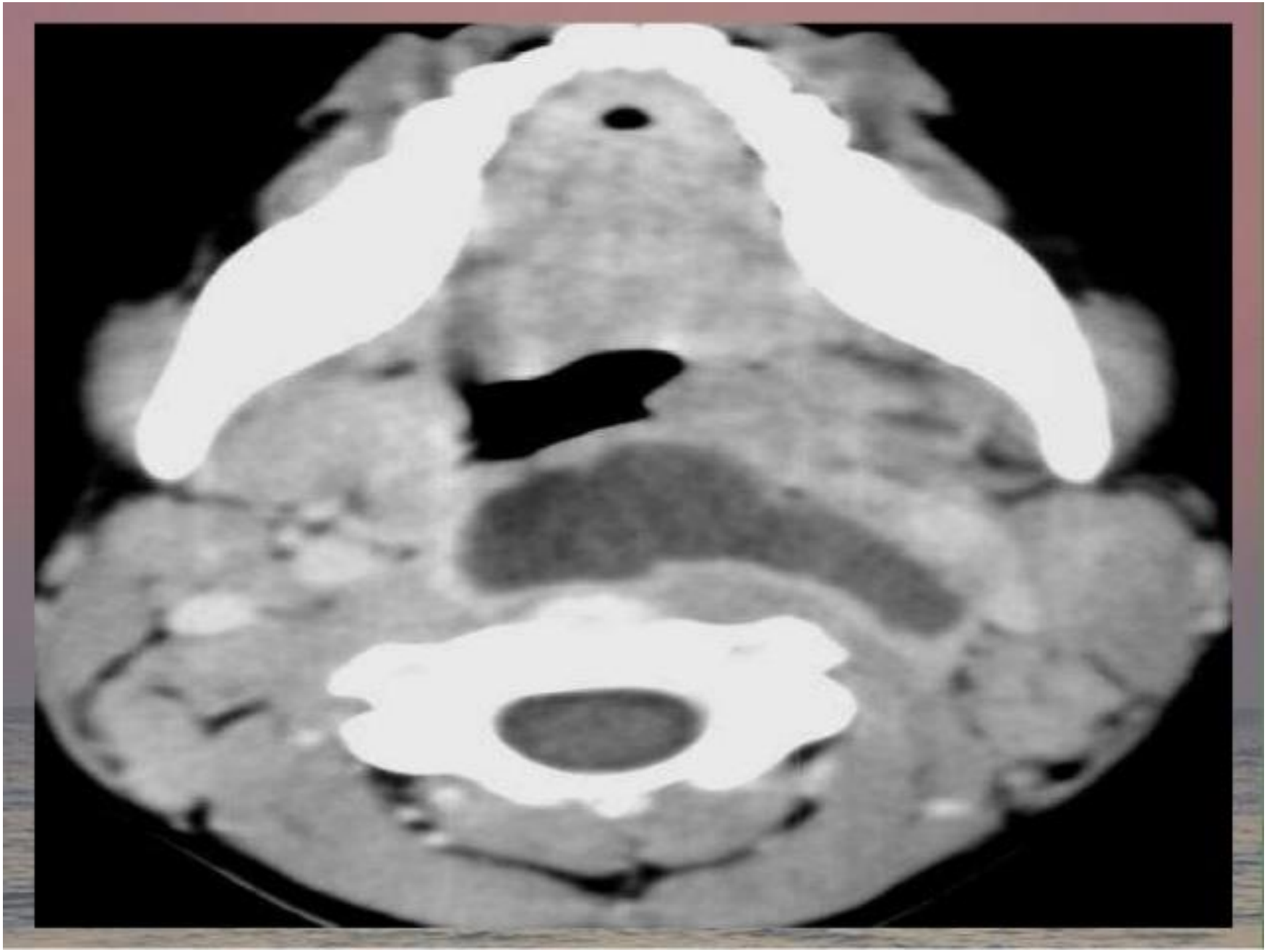
swelling *below* the angle of the mandible and trismus. It may lead to respiratory obstruction, septic thrombosis of the internal jugular

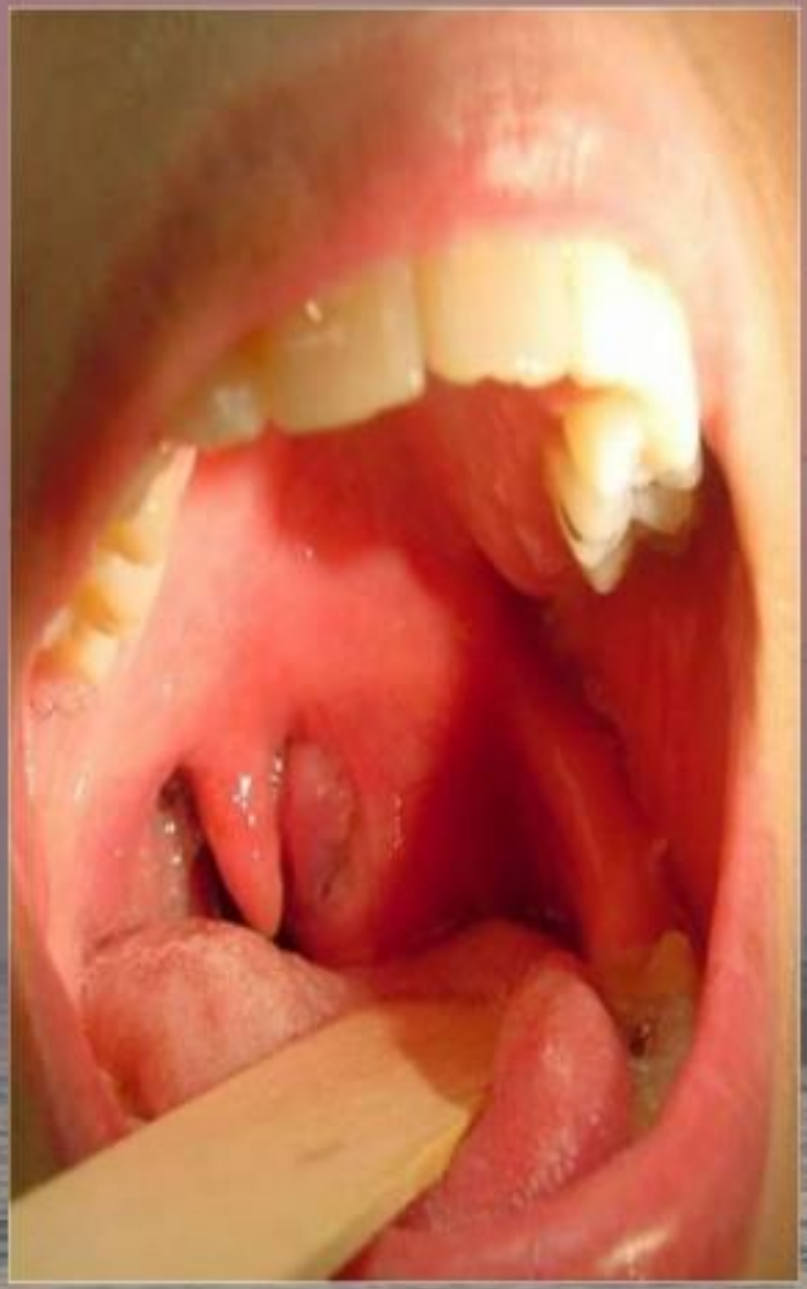
vein and carotid artery hemorrhage.

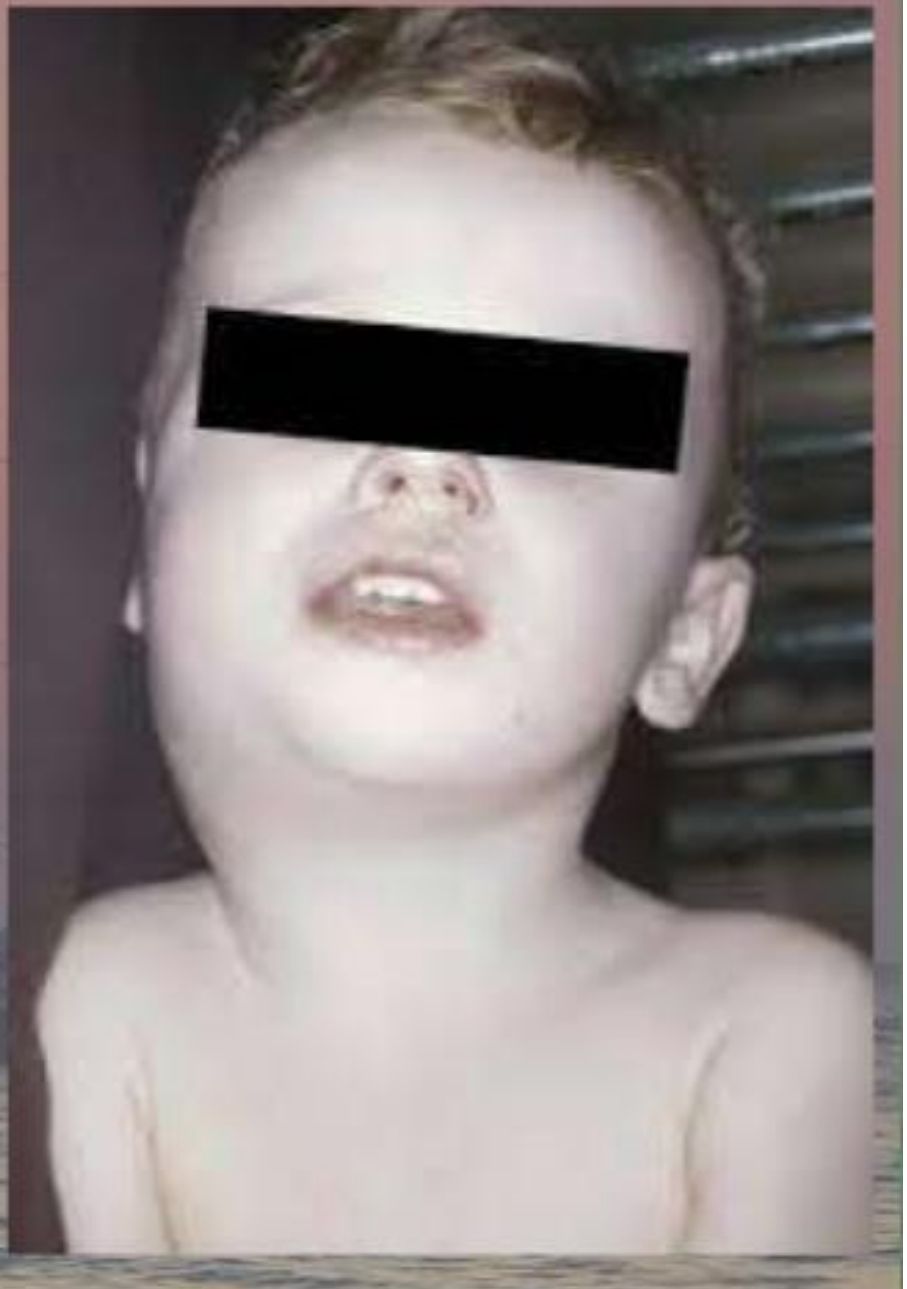
Site of incision and drainage intraoral incision medial to the pterygomandibular raphe with the dissection medial to the Medial Pterygoid muscle.

Extraoral incision at the level of the hyoid bone.anterior to the sternocteidomaetoid muscle.(SCM)and the dissection continued superiorly and medialy between submandibular gland and posterior belly of digatstric muscle.

Through and through driange can also be applied.








Retropharyngeal space infection

Retropharyngealspace infection

Anatomic boundaries extend from the base of the skull to the upper mediastinum (C6-Tl), it is bounded anteriorly by posterior wall of the pharynx and posteriorly by the Alar fascia.

Source of infection upper and lower molar teeth by lateral

pharyngeal space by the way of pterygomandibular,

submandibular, sublingual spaces. It can also result from nasal and pharyngeal infections.

The swelling causes bulge of the posterior pharyngeal wall, there is dysphagia, dyspnea, and fever. Lateral neck radiograph may reveal widening of the retropharyngeal space.

Site of incision and drainage extraorally by an incision anterior to the SCM below the hyoid bone, SCM and the carotid sheath are retracted laterally and blunt dissection is carried out deeply to enter the space.

Some authors advocated intraoral drainage by an incision along the posterior pharyngeal wall in extreme Trendlenburg position and suction. Most anesthesiologists prefer tracheostomy to secure the airway.


Peritonsillar (Quinsy) abscess

Peritonsillar abscess or Quinsy

Anatomical boundaries it is localized between the C.T. bed of the faucial tonsil and the Superior Constrictor muscle of the pharynx.

Source of infection it arises from tonsillitis, but it is occasionally a complication of pericoronitis of the lower third molar.

It causes swelling of the anterior pillar of the fauces and a bulge of the soft palate of the affected side which may reach the midline and push the uvula. Also there is acute pain, dysphagia, the voice becomes muffled, odynophagia, drooling and anorexia.

Site of incision and drainage the incision is placed in the point of maximum fluctuation, this can be done under local anesthesia, if general anesthesia is used the anesthetist should be experienced and good suction be available to prevent aspiration and the patient should be in heead down position.


Infection of spaces related to upper jaw

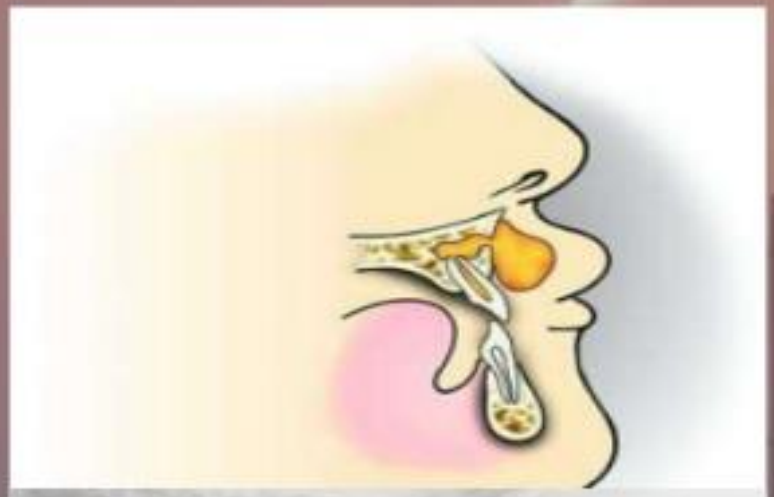
1. **Upper lip infection**

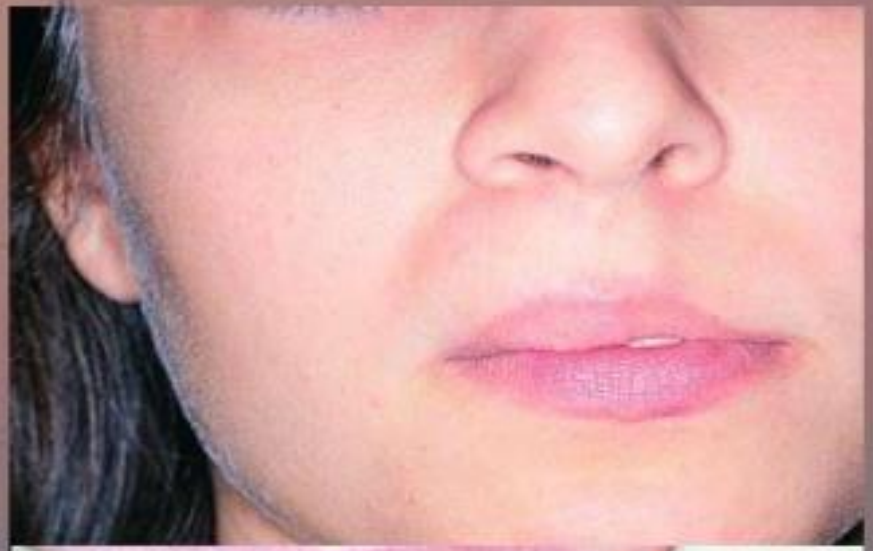
Infections of the upper incisors and canines can spread to the upper lip usually on the oral side of Orbicularis Oris muscle and points in the vestibule.

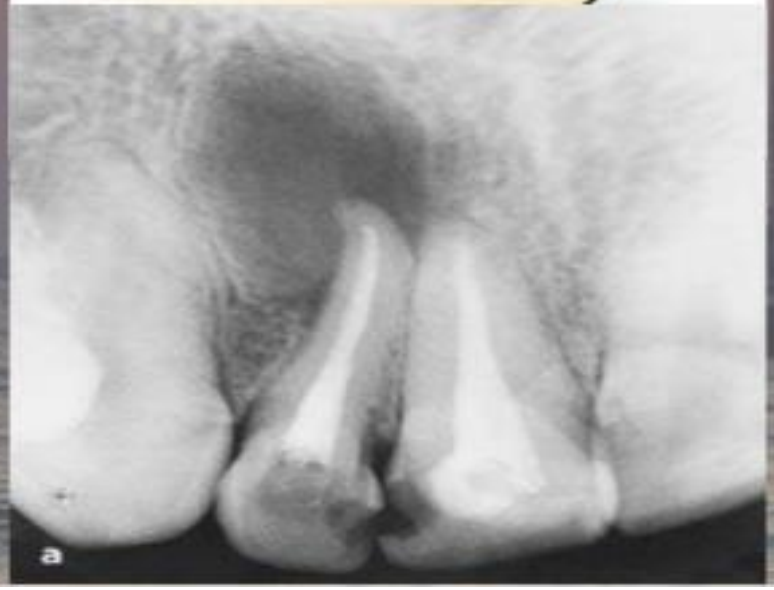
Infection of the upper lip can lead to serious complications like orbital cellulitis or cavernous sinus thrombosis by extension of infection through the superior labial vein to anterior facial vein to ophthalmic vein to cavernous sinus.

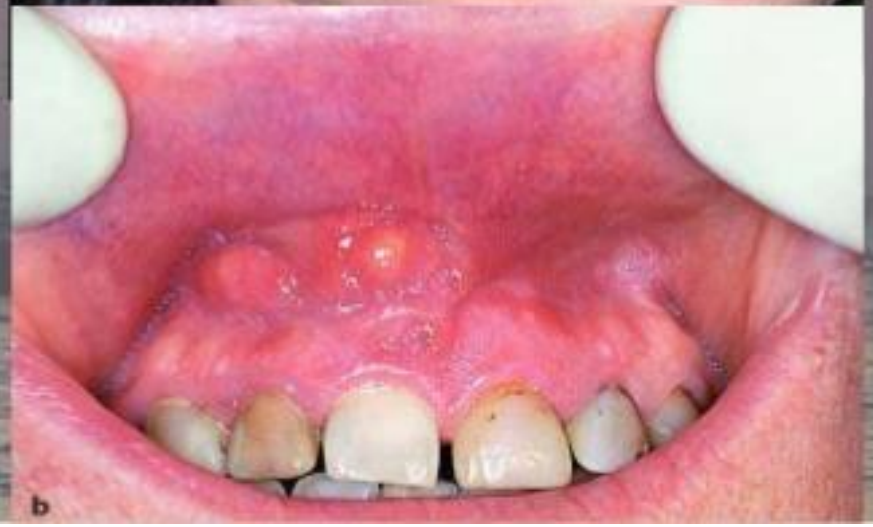
Incision for drainage is made near the vestibule intraorally.

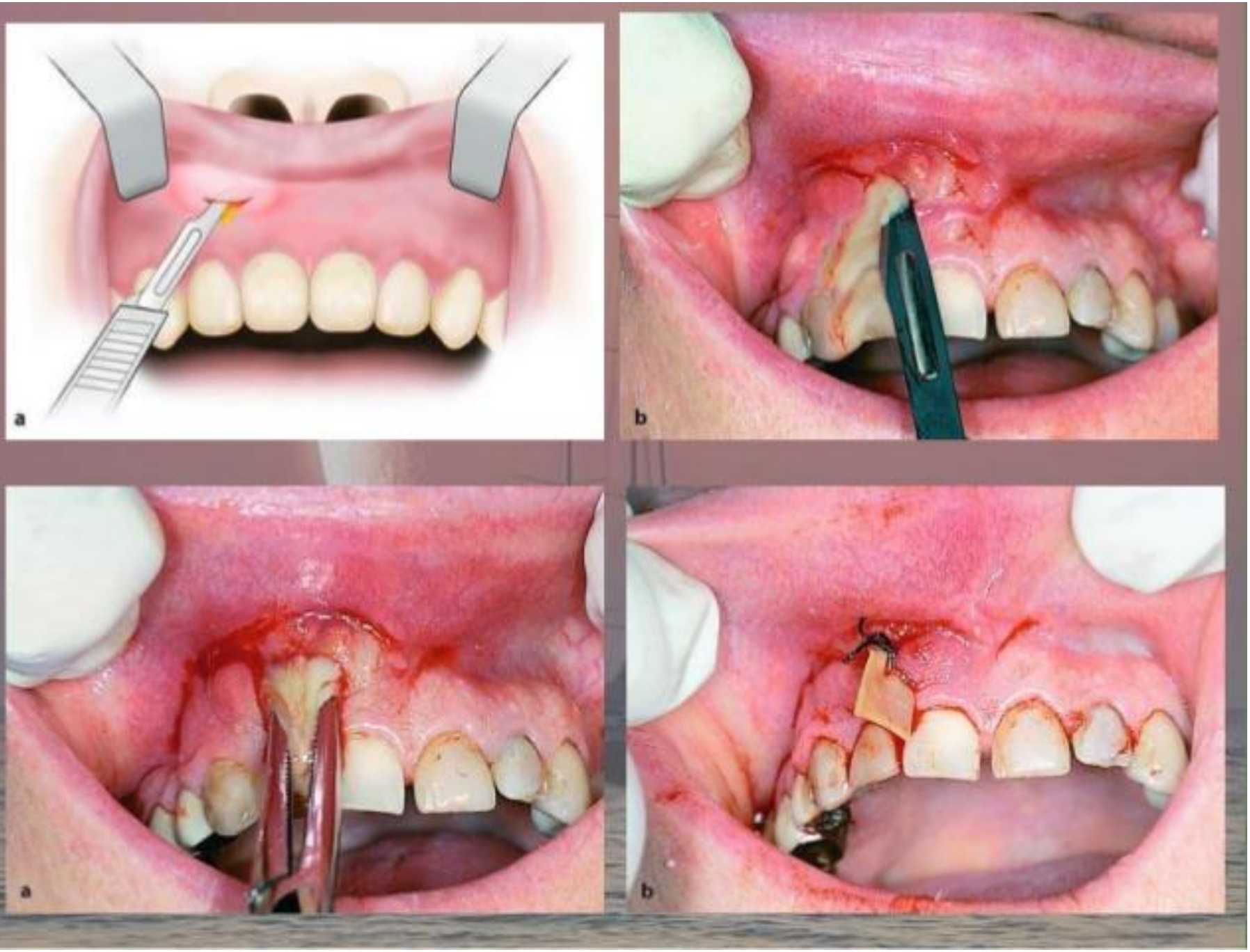
Upper lip infections







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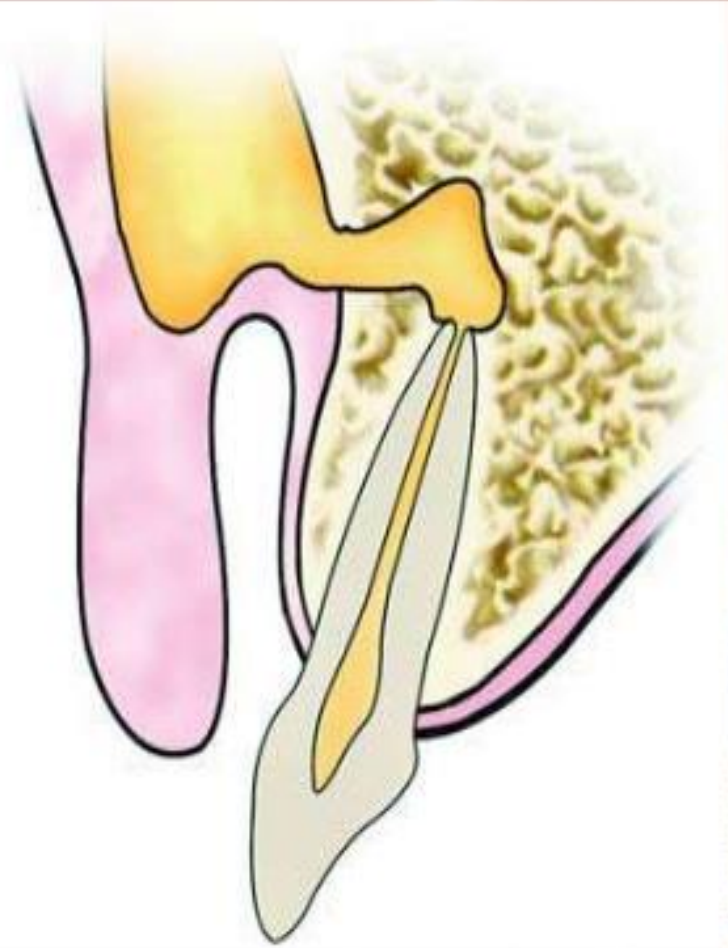
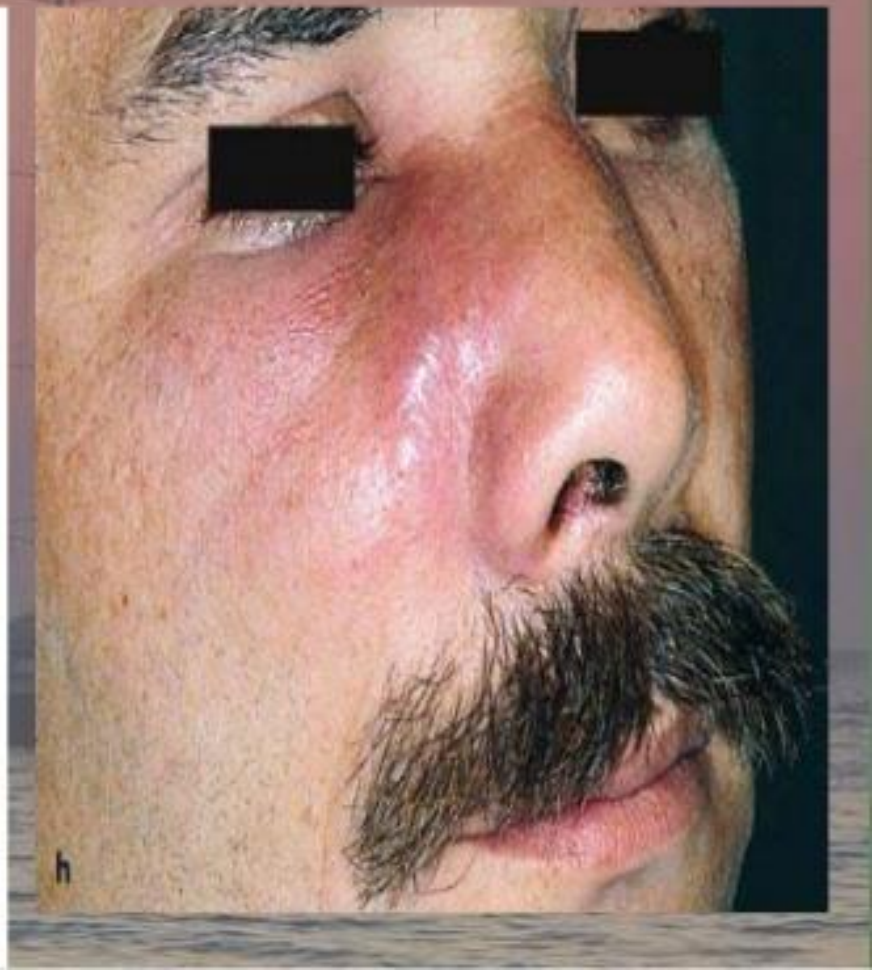
Canine fossa infections





Canine Cossa infections

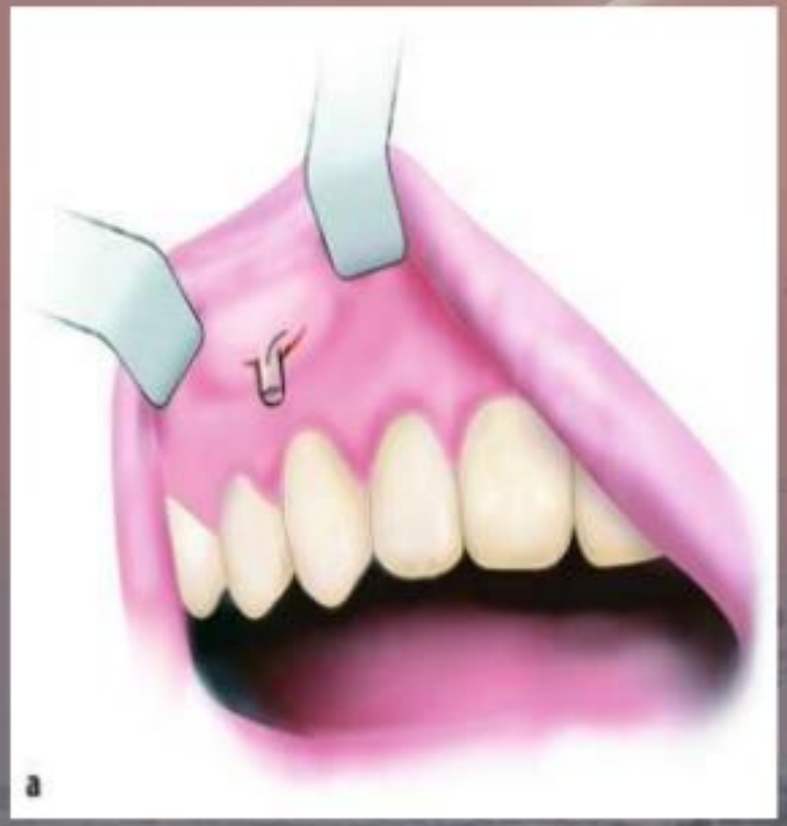
Anatomic boundaries it lies between the canine fossa and the muscles of the facial expression.

Source of infection mostly is the canine and first premolar but the infection can spread from upper incisor teeth.

Infection occurs when it spreads in the area above the origin of the Levator Anguli Oris and is directed toward the medial edge of the Levator Labii Superioris.

The swelling is lateral to the nose leading to obliteration of the nasolabial fold and may lead to periorbital cellulitis, there is risk of cavernous sinus thrombosis.

Site of incision and drainage intraoral horizontal incision in the buccal vestibule .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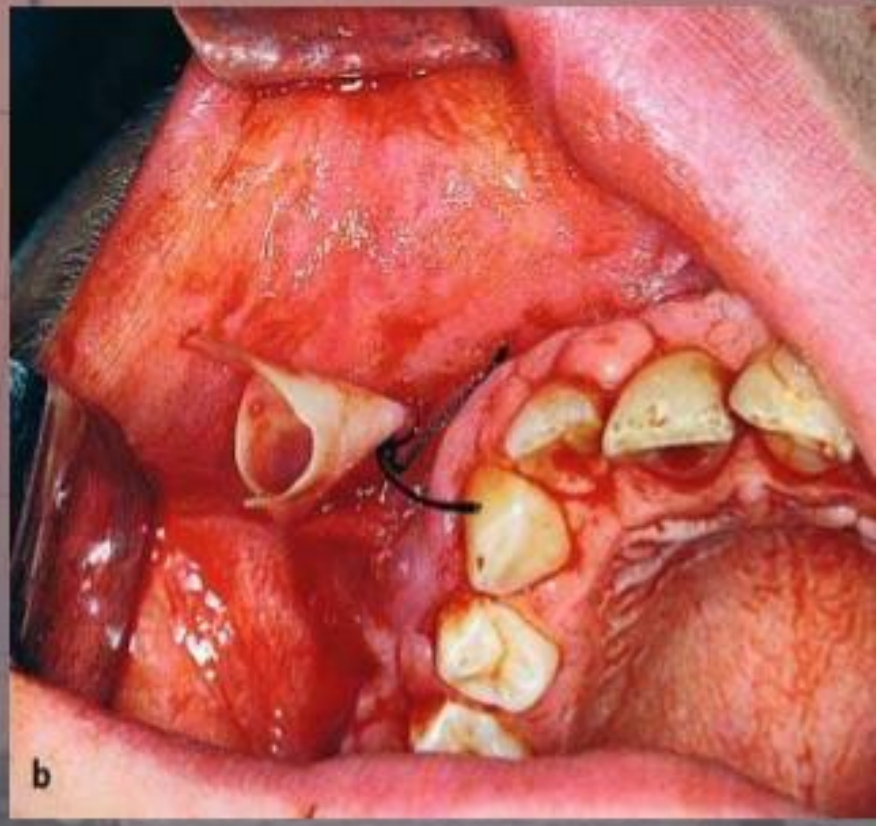
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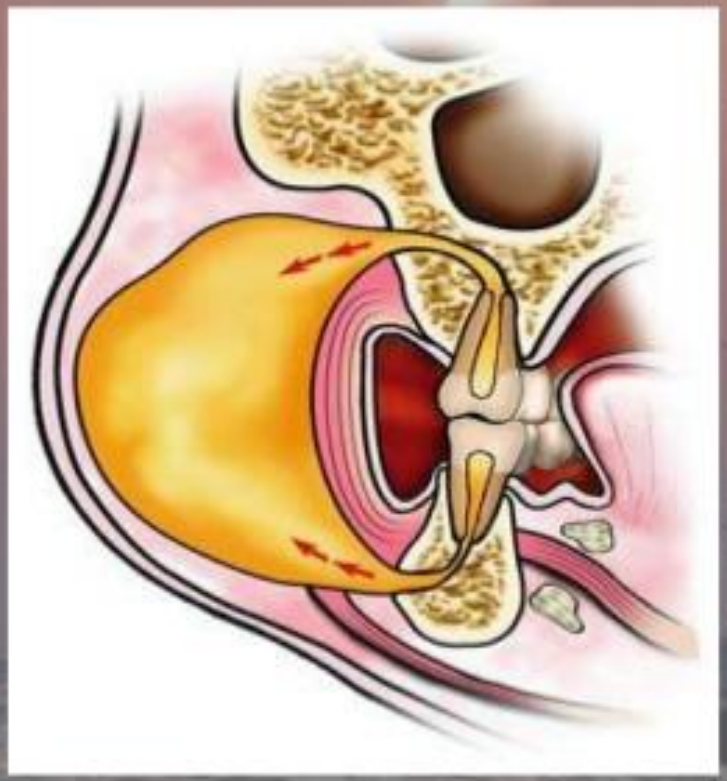
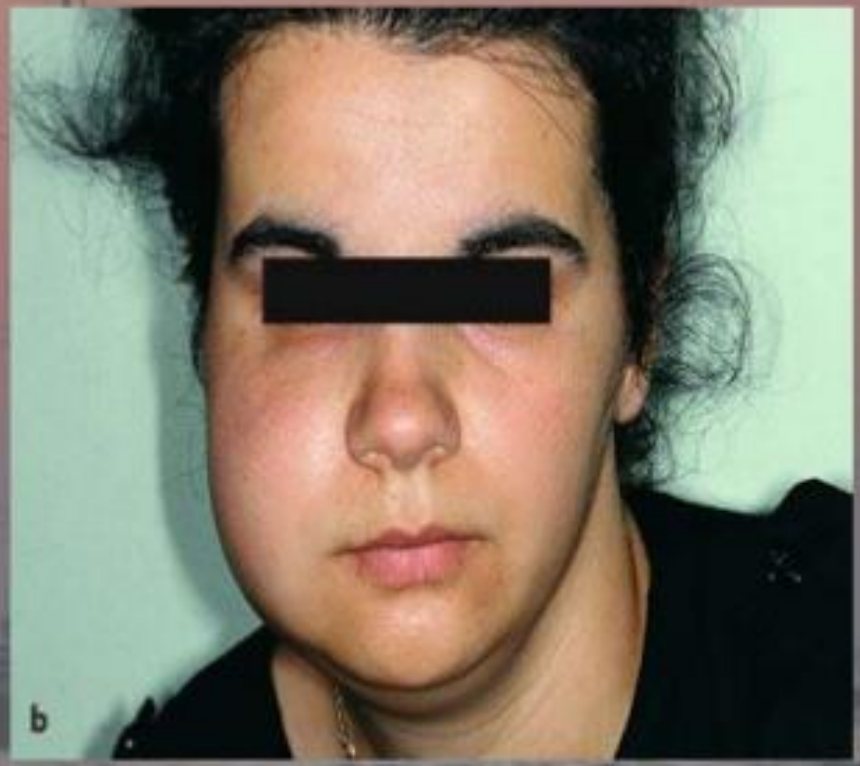
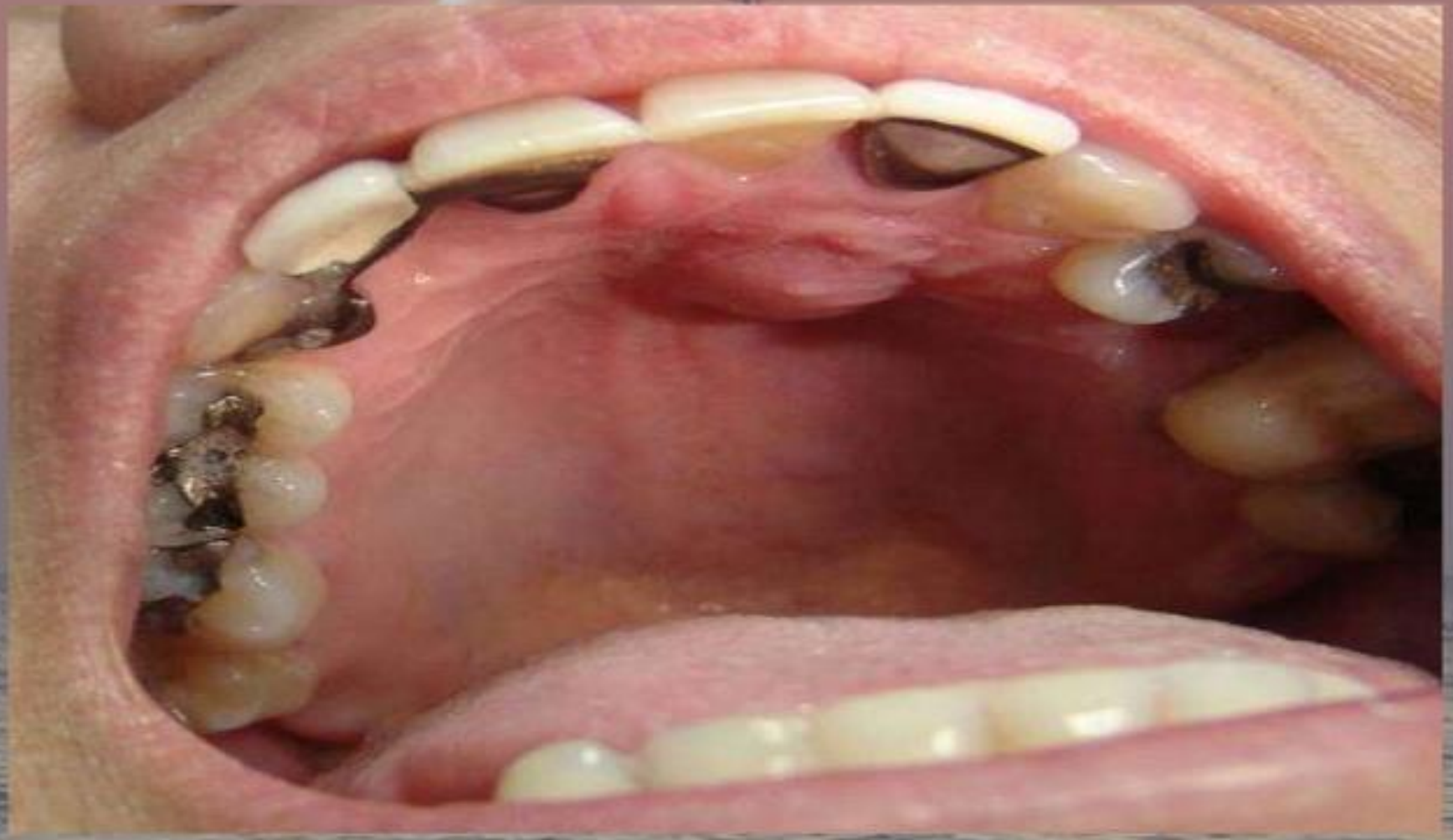
Buccal space infection





Subperiosteal in the palate



Subperiosteal abscess in the palate

This potential space lies between the palatal mucoperiosteum and the underlying bone, the mucoperiosteum is strongly attached in the midline and at the gingival margin, pus may accumulate beneath the mucoperiosteum leading to its separation from the underlying bone.

Source of infection it may spread from the apex of the lateral incisor which is close to the palatal bone. Also infection can spread from the palatal root of multirooted upper molars. It can also originate from periodontal pocket.

The swelling causes palatal bulge between the gingival margin and the midline, confined to one side.

Site of incision and drainage anteroposterior incision parallel to greater palatine vessels.

Maxillary antrum

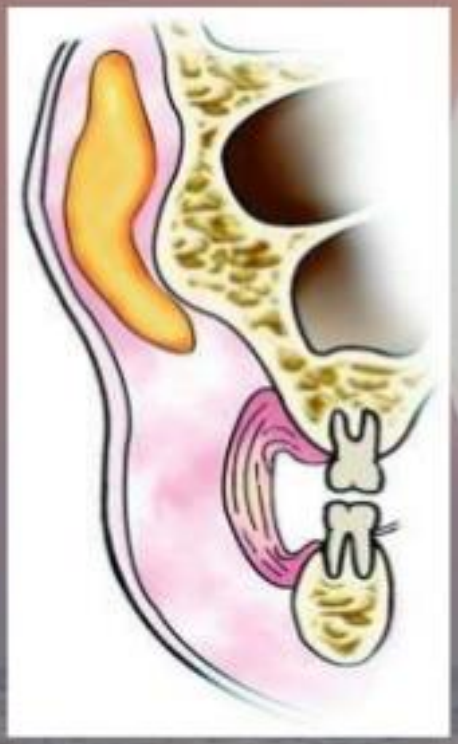
Infection from upper molars and less frequently premolars may spread to the maxillary antrum, this depends on the size of the maxillary antrum and the length of the root.

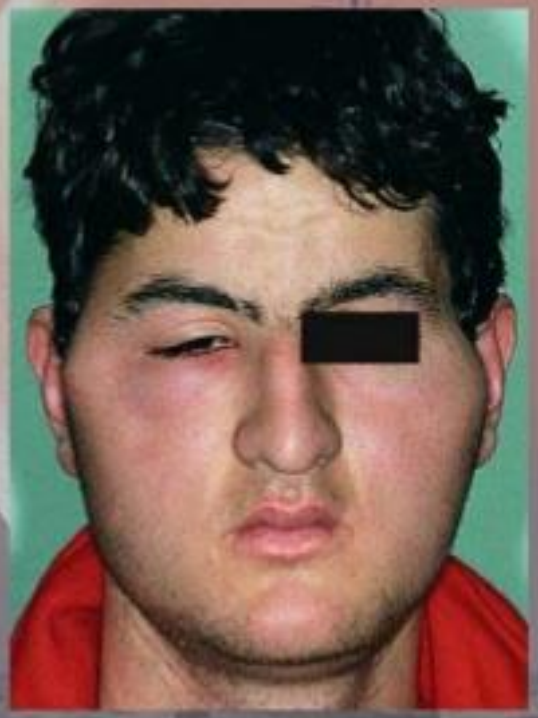
It causes acute sinusitis with facial pain that worsens on bending or leaning forward, the infection may lead a chronic course leading to mucosal thickening and polyps.

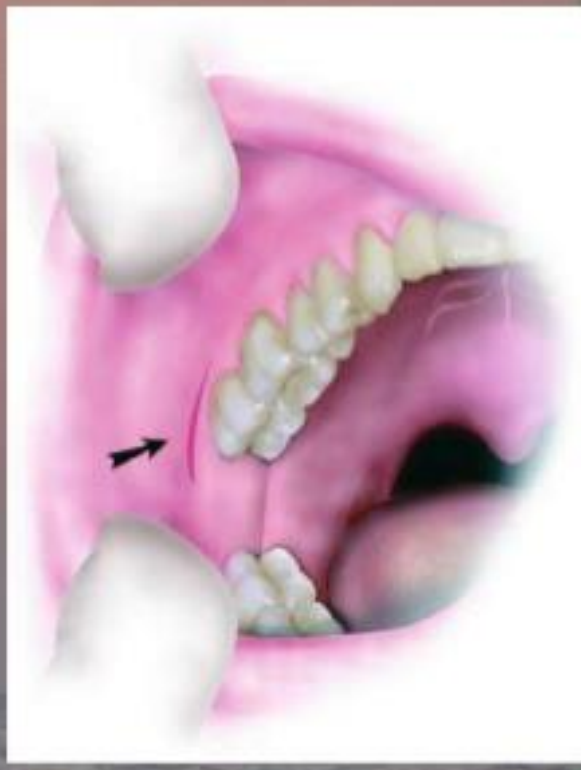
Occipitomental radiograph shows opaque maxillary sinus or fluid level.

Pus may drain partially through the sinus osteum, extraction of the causative tooth leads to drainage of pus but it may leave a defect in the floor of the sinus and cause oroantral fistula. If the defect is small and with antibiotic treatment the socket may heal uneventfully, but larger defects may require further management.

Infra temporal space Abscess







Infratemporal space infection

Anatomic boundaries this space is bounded laterally by the ramus mandible and the Temporalis muscle, medially by lateral pterygoid plate, superiorly by infratemporal surface of the greater wing of the sphenoid.

It is traversed by the maxillary artery and contains pterygoid venous plexus. It represents the upper extremity of the pterygomandibular space.

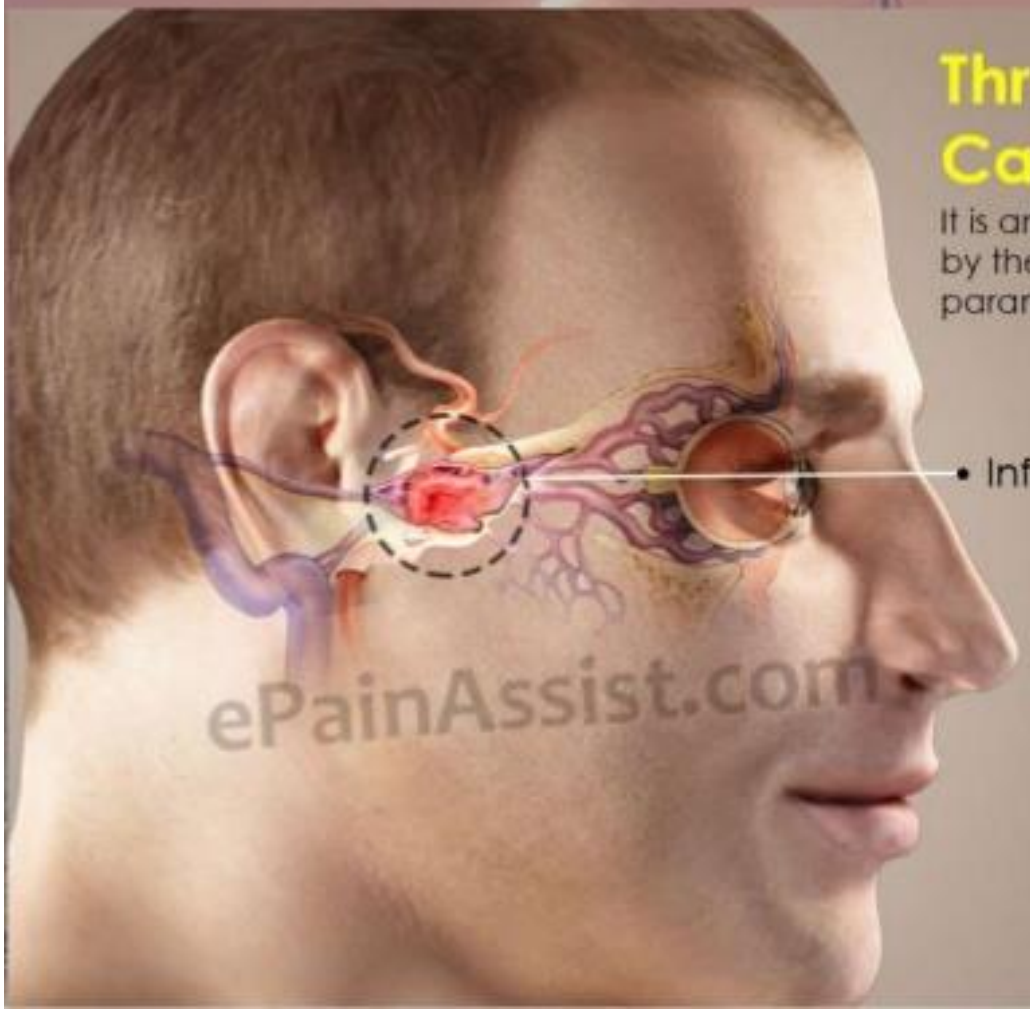
Source of infection directly from upper molar teeth or through contaminated needle from the pterygomandibular space. Infection may spread to the temporal space.

There could be moderate swelling in the temporal region with trismus, usually the patient is toxic with high temperature. These infections are serious since they can spread through the pterygoid venous plexus to the cavernous sinus through emissary vein or it can spread to the middle cranial fossa with headache, photophobia, irritability, vomiting and drowsiness.

Site of incision and drainage intraorally through an incision buccal to the upper third molar followingthe medial surface of the corronoid upward and backward, but with the presence of trismus this approach is difficult. Extraorally through an incision in the upper and posterior edges of the Temporalis muscle within the hair line passing downward, forward and medially.

Infection related to the maxillary teeth can spread to the masticator spaces and pharyngeal spaces and these were already discussed

Cavernous sinus thrombosis



Thrombosis of the Cavernous Sinus

It is an infection leading to blood clot caused by the complication of an infection in the paranasal or central face sinuses.

- Inflamed Cavernous sinus

Signs and Symptoms of Thrombosis of Cavernous Sinus

1. Fatigue
2. Seizures
3. Vomiting
4. Impaired vision
5. Boil on the face
6. Drooping eyelids
7. High temperature
8. Sinusitis- an infection in the skull
9. Severe pain or numbness in the face
10. Infection in eyes- redness, swelling or irritation around the eyes

Cavernous sinus thrombosis

It is a very serious ascending infection, although not a fascial space infection but it can be caused by odontogenic infections especially of upper teeth . It can also result from upper lip, nasal and orbital two routes:

Infection can spread to cavernous sinus through two routes

Anterior route: through the valveless angular vein and inferior ophthalmic vein.

Posterior route; through the pterygoid venous plexus and transverse facial vein.

This infection has a high mortality rate.

Clinical features:

Marked edema and congestion of the eyelids and conjunctiva which can be bilateral due to the spread of infection to the other side.

- Proptosis (exophthalmos) and ptosis.
- Ophthalmoplegia and dilated pupil.
- Papilloedema with multiple retinal hemorrhage.
- Fever:
- Depressed level of consciousness.

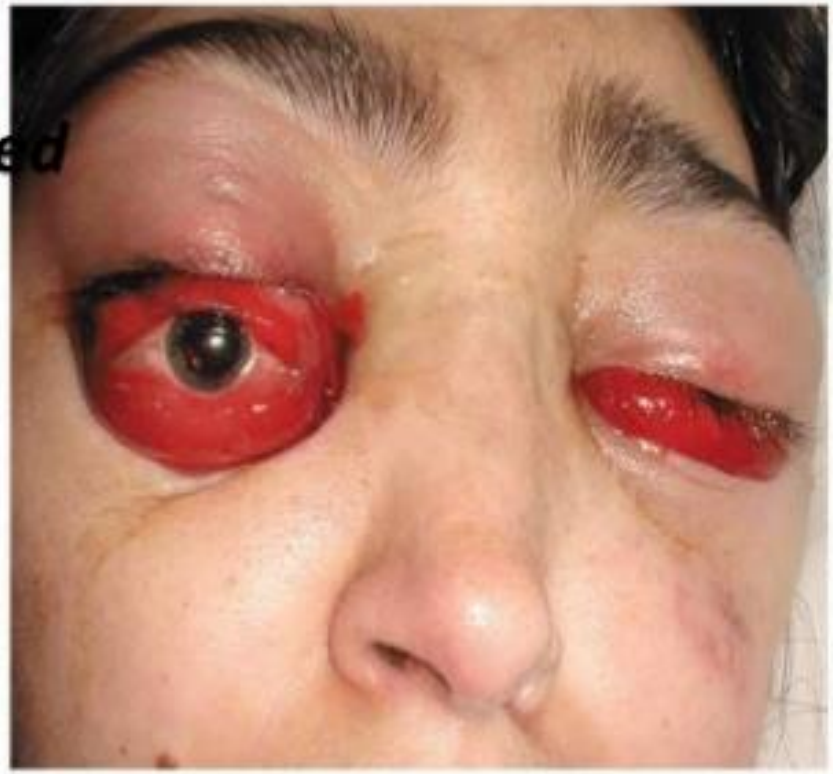
Treatmen:t

It is an emergency that requires a neurosurgical consultation, the lines of treatment include:

- Antibiotic treatment
- Heparinization to prevent extension of thrombosis.
- Treatment of the odontogenic cause.

Clinical features

- Marked edema and congestion of the eyelids and conjunctiva
- Proptosis and ptosis
- Ophthalmoplegia and dilated pupil
- Papilloedema
- Fever
- Deprressed level of consciousness




Treatment
• AB
• Heparin
• Treatment of odontogenic causes

Principles of treatment of odontogenic infections

- Dental treatment
- Surgical treatment

Principles of treatment of odontogenic infections

In treating odontogenic infections, the clinicians need to identify the presence of infection through the presence of the local and systemic signs and symptoms of the infection and whether it is cellulitis or abscess, also to determine the state of the host defenses, as these can be depressed by many factors such as; physiologicfactors, disease related factors, immune-system related factors and drug suppression related factors.

Treatment of odontogenic infections requires medical, surgical or dental therapy or a combination.

Dental treatment; it aims to eliminate the source of infection through endodontic treatment, periodontal treatment or extraction of the offending tooth. These factors should be taken in consideration:

- The extent of infection.
- Patient general health status.
- Degree of trismus.
- Biomechanical necessity of retaining the tooth.

Surgical treatment: it aims to drain the accumulated pus to rid the body of the toxic purulent material and to relieve and decompress the tissues allowing better perfusion of blood to the , infected area

Methods of drainage

- Through the root canal
- Through the socket by extraction
- Through fenestraion of the alveolar bone
- Through incision

Indications for incision and drainage

- Signs of pus accumulation
- When the involved compartment are inaccessible
- Serious and rapidly evolving infections of neck and floor of the mouth

Principle of incision and drainage

1. Should put on healthy skin and mucosa as possible , in area of maximum fluctuancy

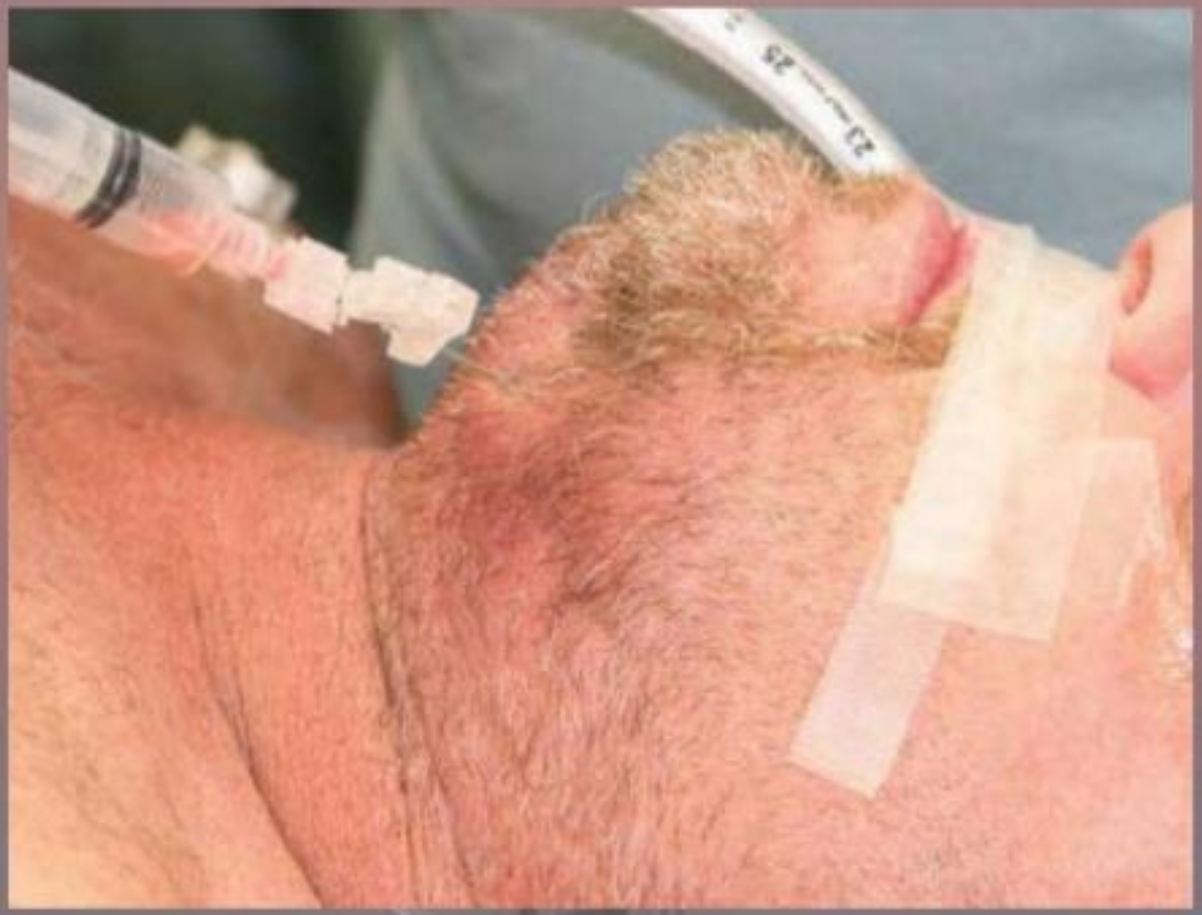
2. Esthetically accepted area

3. should include only skin and subcutaneous tissues and dissection through deeper tissue should be done bluntly

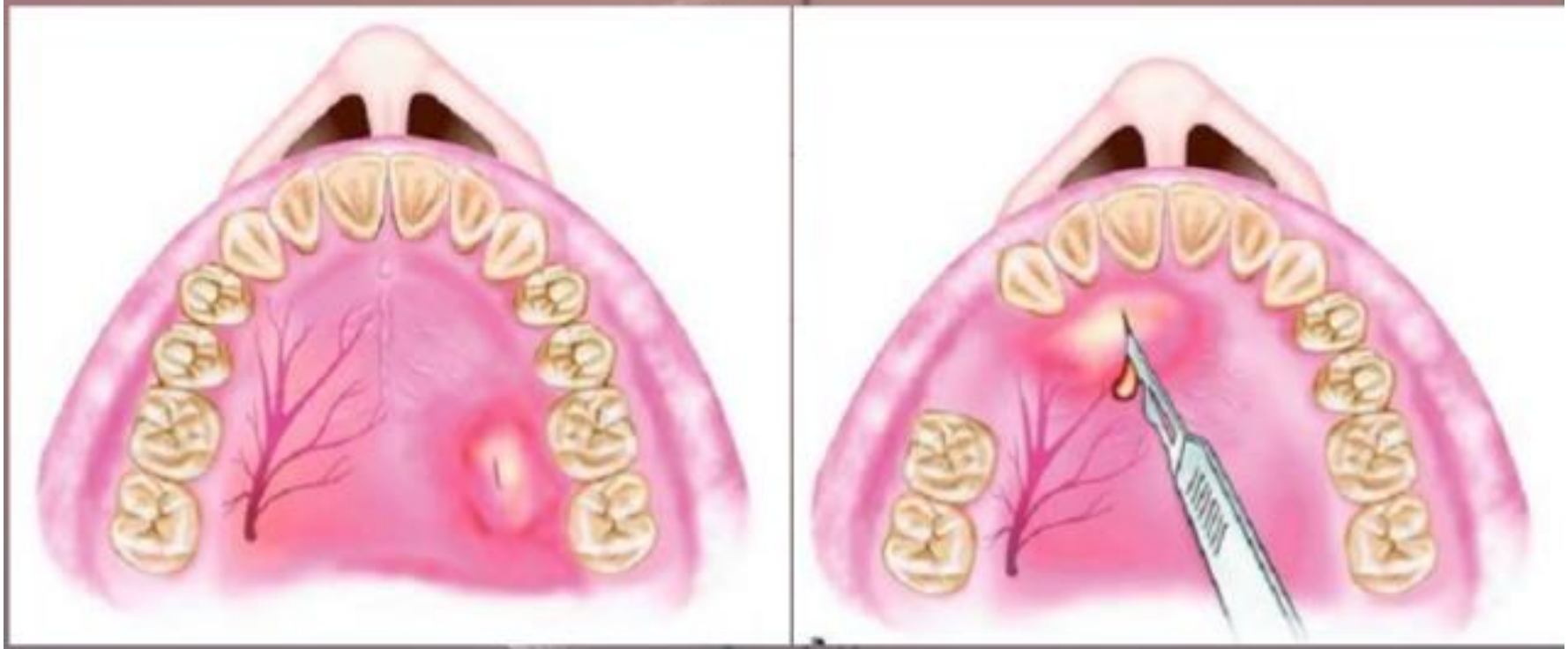
4.Drain should be used after after evacuation of the pus

5. Wound margins should be cleaned daily to remove clots and debris

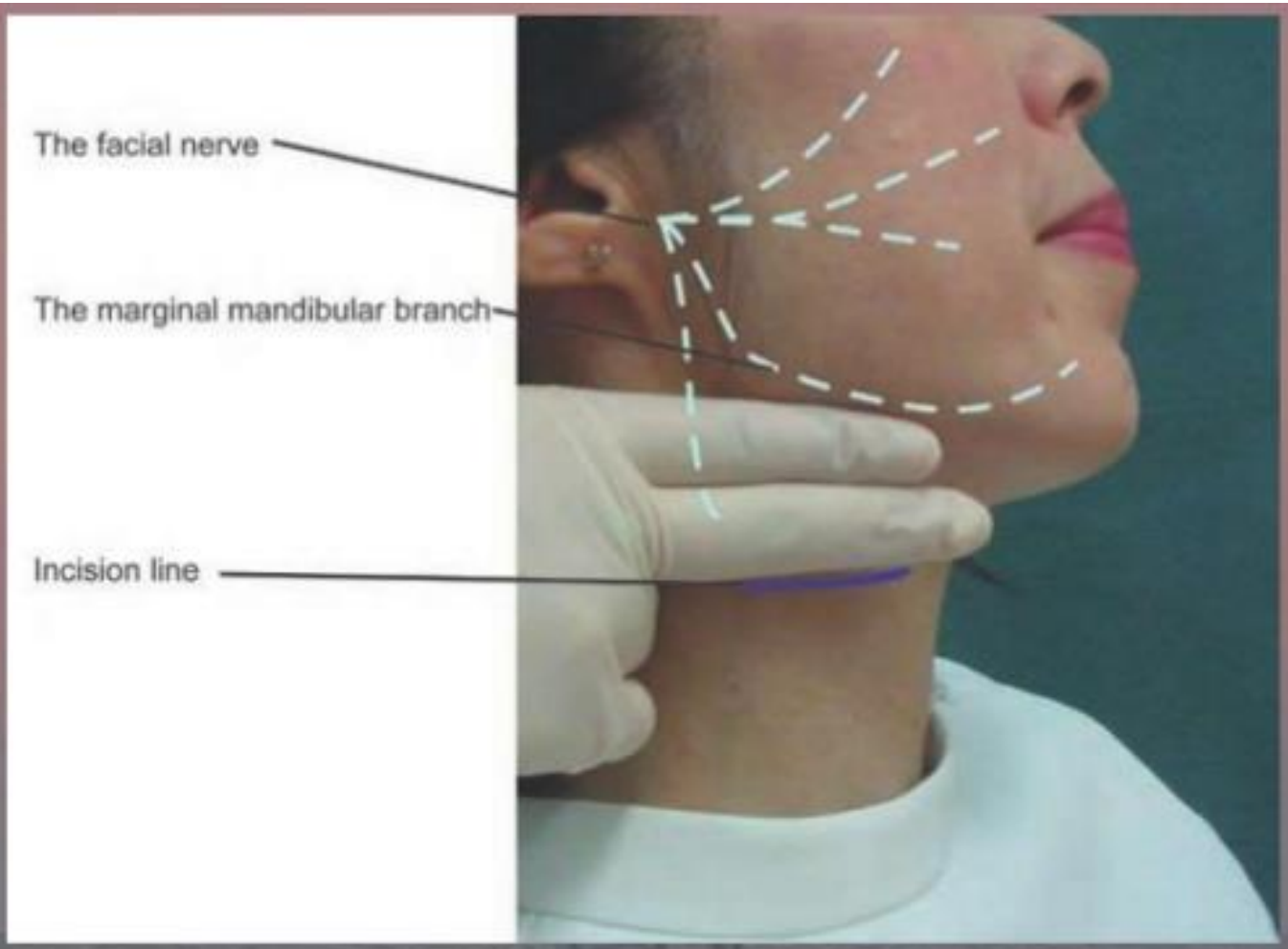
6. Sample of pus should be obtained and sent to the laboratory for culture and sensetivity test



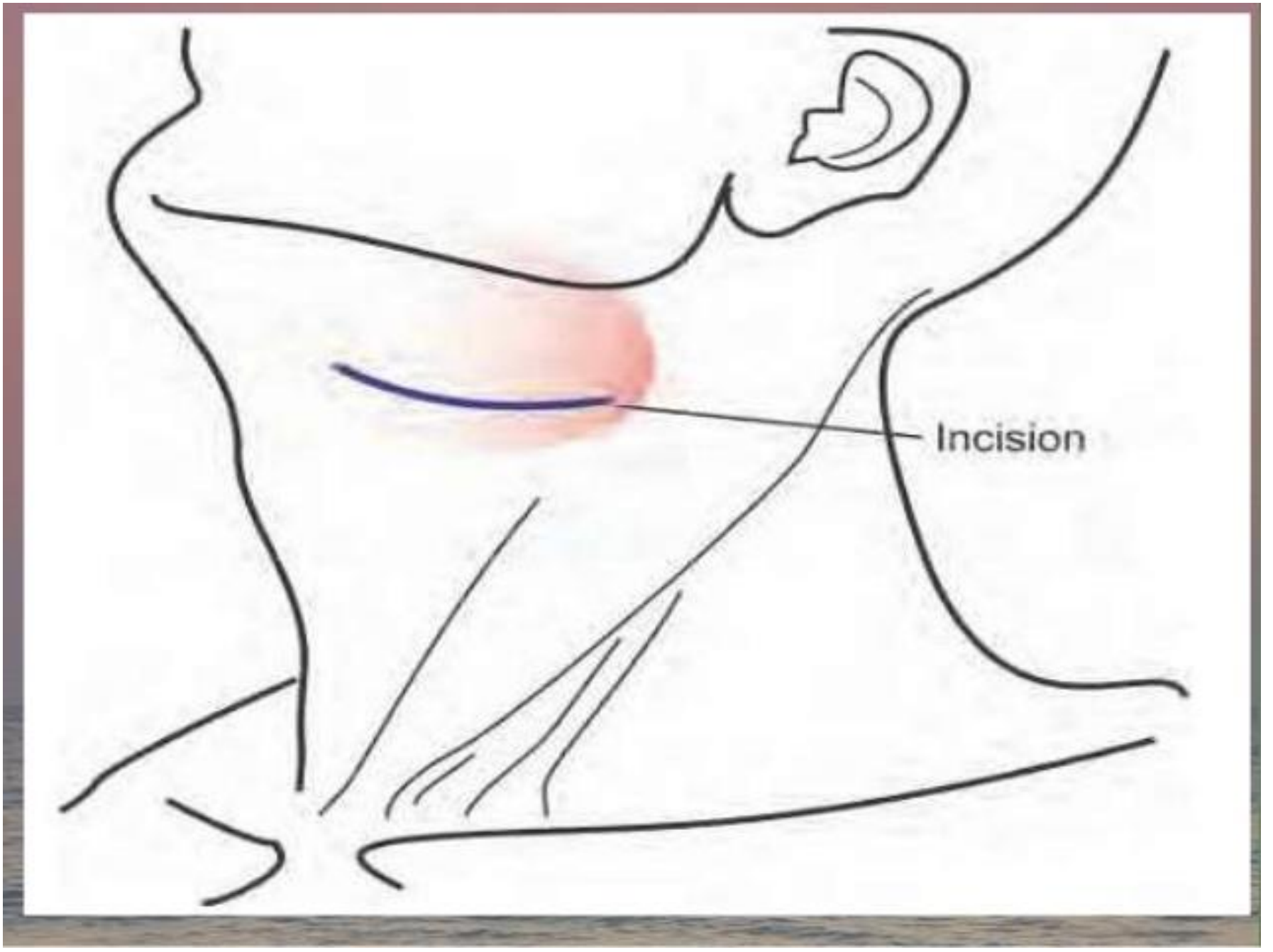
Prior to surgical drainage,
a sterile aspirate is obtained for culture and sensitivity

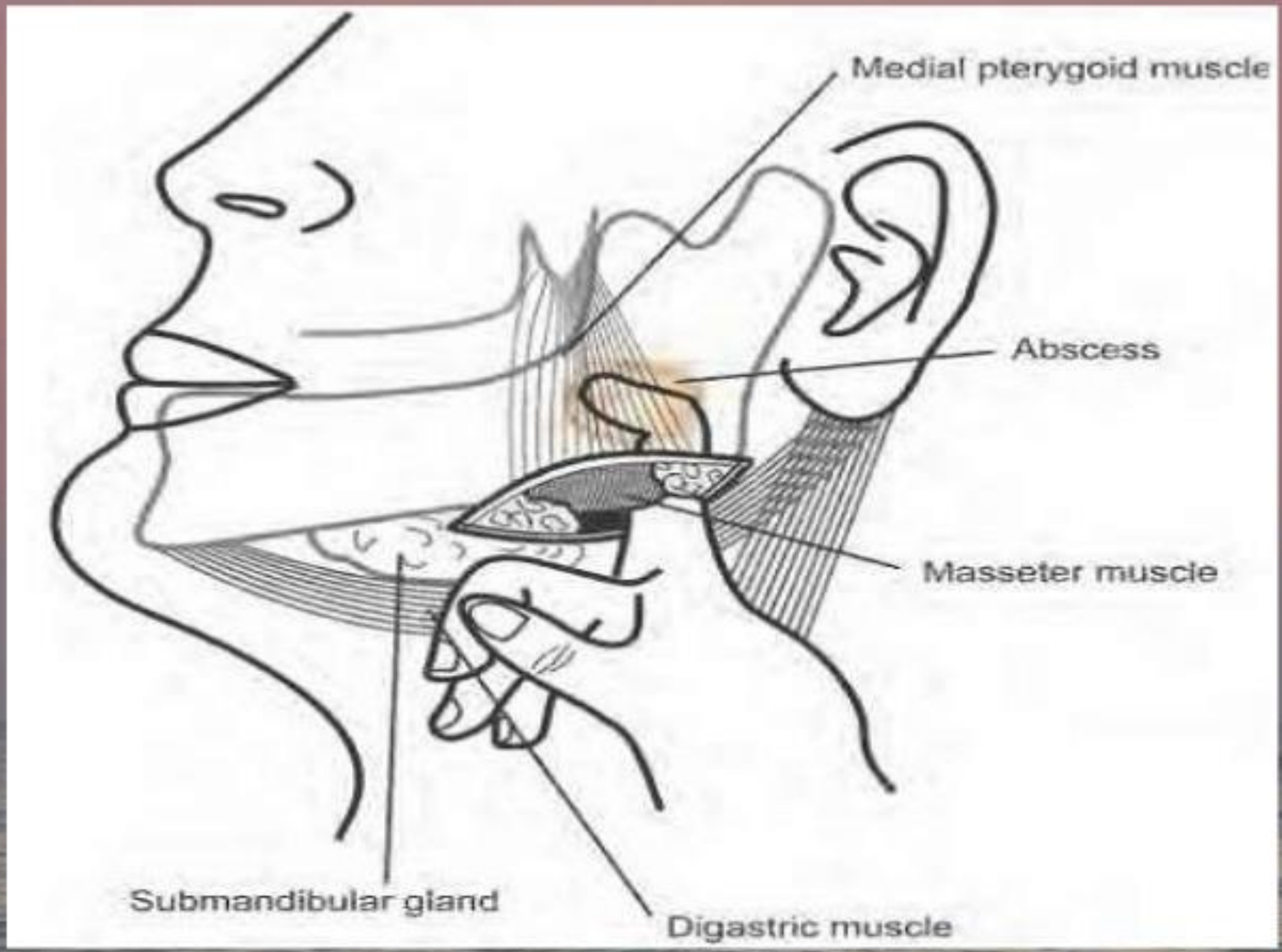


Palatal abscess , incision parallel to the greater palatine vessels



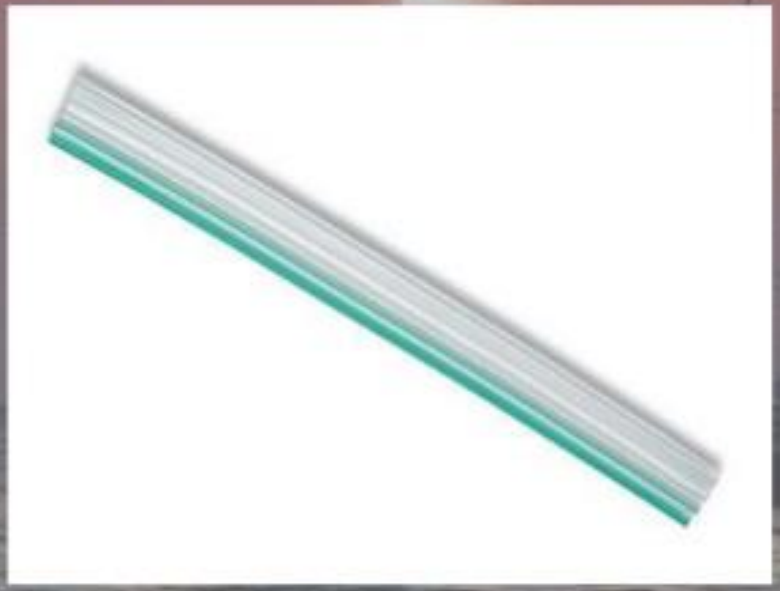
An incision at **submandibular region** should be placed two fingers' breadth below the lower border to avoid injury of the marginal mandibular branch of the facial nerve


Incision


Medial pterygoid muscle
Abscess
Masseter muscle
Submandibular gland
Digastric muscle

5) PLACEMENT OF DRAIN

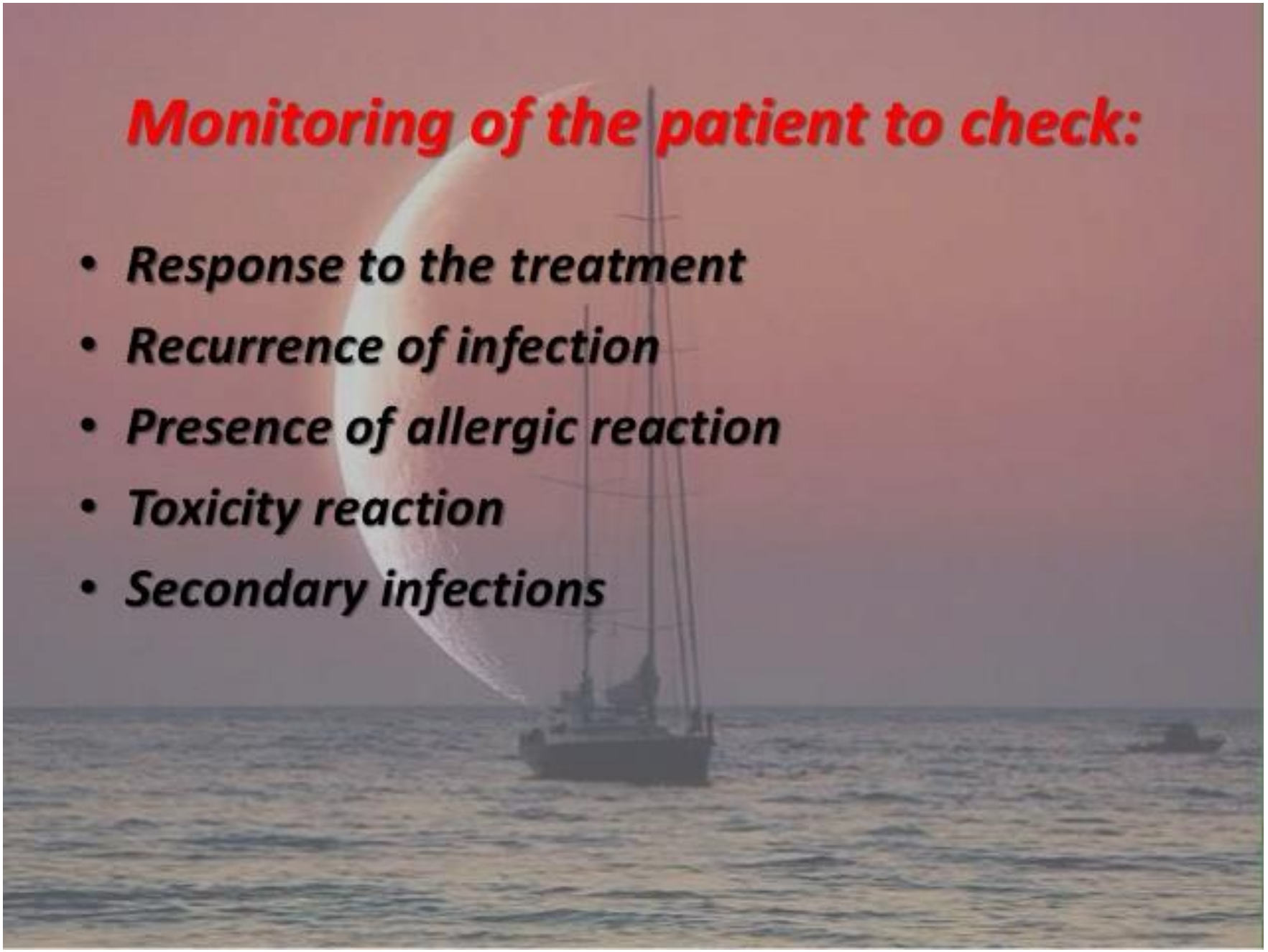
- **Purpose :**
  - to allow the discharge of tissue fluids and pus from the wound by keeping it patent
  - allows for debridement of the abscess cavity by irrigation
- **Types :**



corrugated

Penrose


Medical treatment
Hydration
Soft diet
Analgesics
Maintain good oral hygiene
AB


Monitoring of the patient to check:
Response to the treatment
Recurrence of infection
Presence of allergic reaction
Toxicity reaction
Secondary infections

Indications for the use of AB in odontogenic infection

- Acute cellutitis
- Acute pericoronitis with fever and trismus
- Deep fascial space infections
- Compromised patients


Thank you for listening