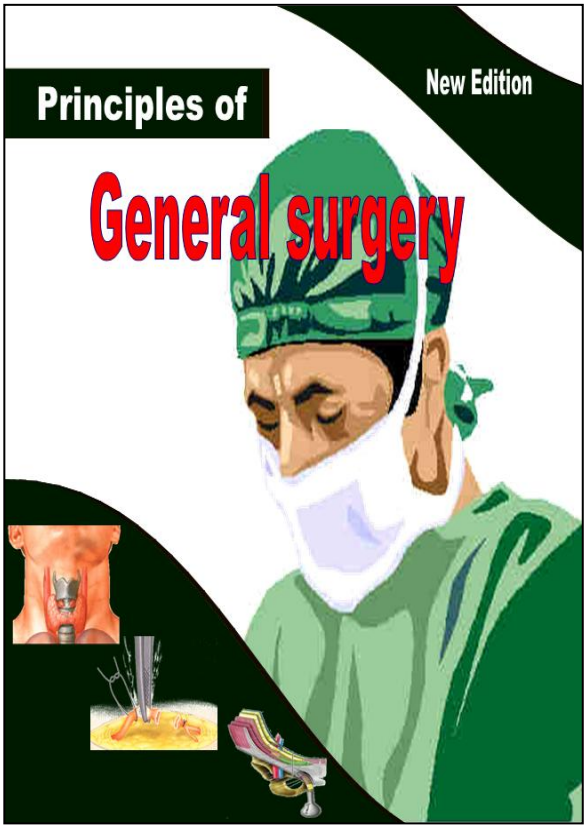
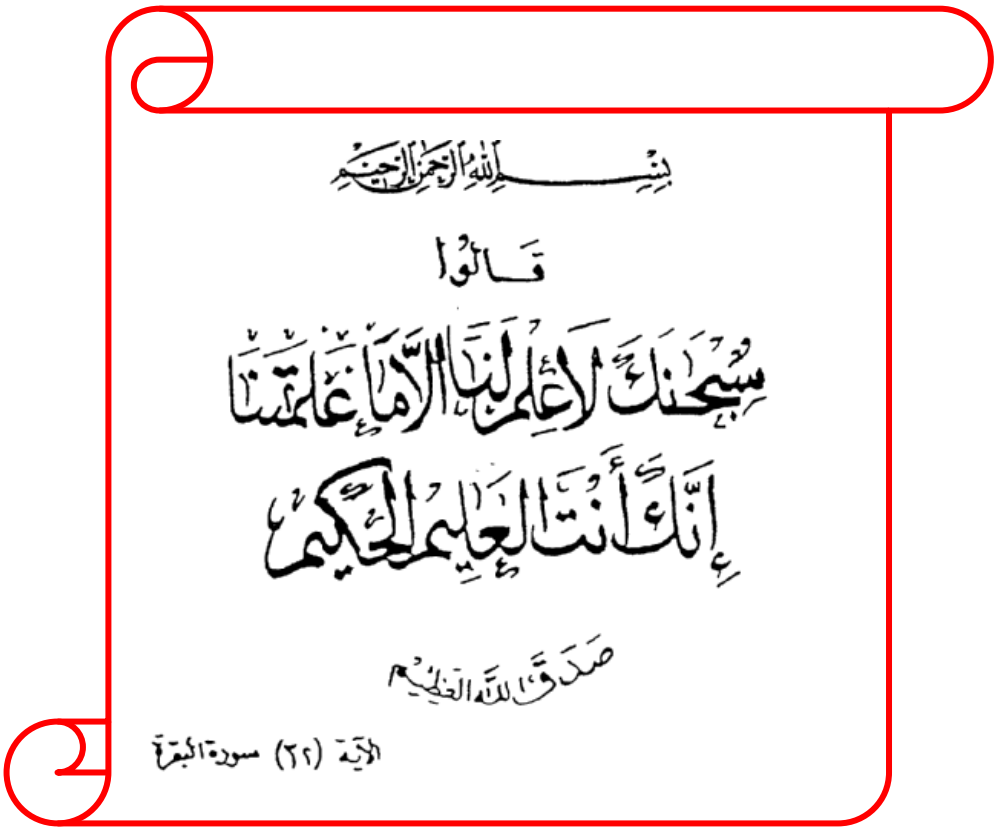
PRINCIPLES OF

GENERAL SURGERY



Dr. WAEL METWALY



اللهـــم

- اللهم اجعل هذا العمل خالصًا لوجهك الكريم
- اللهم زدنى علما .... واجعل هذا العلم نافعاً لكل من يدرسه
- اللهم ارزقنى من هذا العمل رضا ومغفرة وعتقاً من النار ما حييت وبعد الممات
- اللهم اجعل هذا العمل صدقة جارية لا ينقطع بها عملى بعد موتى

اللهم آمين

اللهم آمين

وائل متولى

CONTENTS

With my best wishes

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Wounds

WOUNDS

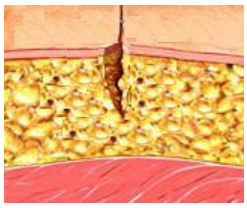
DEFINITION

It is forcible loss of continuity of soft tissues, mainly due to mechanical trauma

CLASSIFICATION

A- Opened wounds " in which the skin continuity is interrupted "

INCISED WOUNDS



- Caused by **sharp** cutting instrument.
- It has **clean cut** edges with **little** or **no** tissue damage.
- It is **more** liable for bleeding & **less** liable for infection.

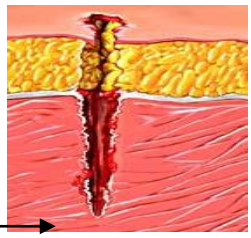
LACERATED WOUNDS

- Caused by **blunt** heavy instrument
- It has **crushed** edges with **severe** tissue damage
- It is **less** liable for bleeding & **more** liable for infection

ABRASIONS

- Partial denuation of the superficial layer of the skin due to friction of skin with rough surface.

STAB OR PUNCTURED WOUNDS



- Caused by **pointed** objects as daggers.
- It is the most dangerous type.

MISSILE WOUNDS

- They have an **inlet** & may have an **exit**.
- **The damage is due to the following factors :**
  1- Direct damage by missile track.
  2- Shock waves preceding missile.
  3- Temporary cavitational effect.
  4- If bone affection; bone fragments act as secondary missiles.

- **The degree of damage depends on its velocity :**
  (a) *Low velocity missile*
  produces direct laceration
  (b) *High velocity missile* It is more lethal.
  produces tissue necrosis several centimeters on either side of the track due to the cavitation effect.

BITES (**Animal** or **Human**)

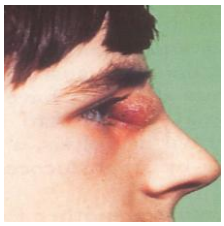
- They are very liable for infection

B- Closed wounds " in which the skin surface is intact "

CONTUSION (**Ecchymosis**)

- It is **bluish skin patches** caused by blunt trauma.
- It is extravasation of blood from injured small vessels with no swelling.
- **Treated by** fomentation
  (**cold** in **1st 24** hours then **hot** after that)

HEMATOMA



- It is a localized extravascular collection of blood

- **Types :** ▪ Subcutaneous hematoma.
  ▪ Sub-periosteal hematoma.
  ▪ Intra-muscular hematoma.

- **Fate :** ▪ Resolution by absorption
  ▪ Localized by fibrosis.
  ▪ Infection → **abscess**.
  ▪ Calcification e.g. **myositis ossificans**

-**Treated by** fomentation + **antibiotics**
+ Pressure bandage (if small) or evacuation (if large)

COMPLICATIONS OF WOUND

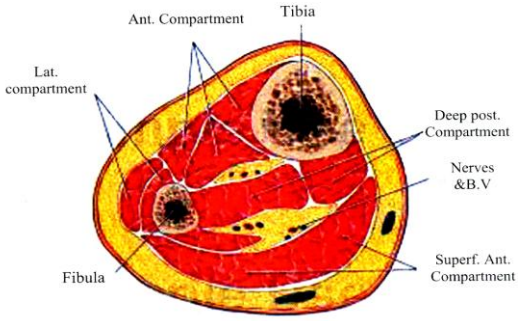
1- General complications

➢ **Shock :** hypovolaemic, septic or neurogenic

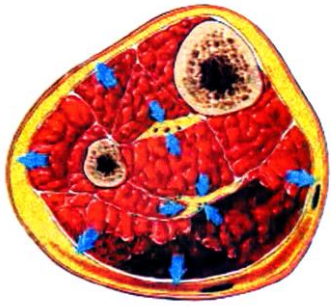
➢ **CRUSH SYNDROME**
[**Traumatic anuria**]

- Results from massive crushing of big muscles → ↑ myoglobin from crushed muscles enter the circulation → acute renal tubular necrosis → **acute renal failure**

- The crushed muscles **swell** (oedema) inside their fascial coverings → ↑ compartmental pressure → compression on vessel → **acute limb ischemia**



Normal cut section of leg



After revascularization

-**Treatment :**
- ▪ Anti-shock measures.
- ▪ Forced diuresis by I.V mannitol.
- ▪ **Fasciotomy** to prevent compartmental syndrome.

2- Local complications

➢ **Infection :**
- ▪ **Non specific :** Staph, Strept ……etc.
- ▪ **Specific :** Tetanus & gas gangrene.

➢ **Injuries of important structures :**
Nerves, vessels, muscles, tendons, solid organs ….etc.

➢ **Retained foreign bodies**.

TREATMENT

A- First aid treatment

" **Pre-hospital management** "

① Ensure patent airway if patient is unconscious
② **Control of bleeding by compression**
③ Sterile dressing to prevent contamination.

Then Transfer the patient to **hospital**

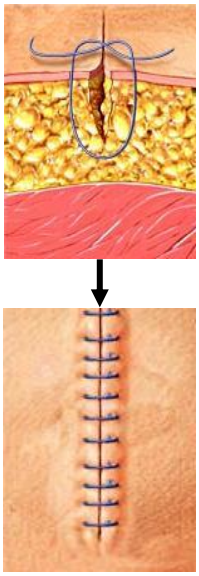
B- Definitive treatment

① Assessment of injury for associated visceral, arterial & nerve injury .
② **3 Anti** (**Anti**-shock, **Anti**biotics & **Anti**-tetanic serum) + **Analgesic**
③ Stabilization of any orthopedic fractures.

Then Transfer the patient to **operating room**

According to the onset of wound

WOUNDS WITHIN 6 HOURS



There is **no** contamination & **no** Risk for infection so we do ⇩

⇨ **Incised wounds**
1ry suture + suture of all structures including nerves & tendons

⇨ **Lacerated wounds**
Excision of the edges then **1ry suture**. Nerves & tendons repair are postponed 4 - 6 weeks

WOUNDS WITHIN 6 - 24 HOURS

The wounds are **potentially contaminated** so we do ⇩

⇨ **Incised or lacerated wounds**
Excision of the edges + systemic A.B + repeated dressing to allow the granulation tissues to fill the wound.
It becomes **clean** within 4 - 6 days → we do **delayed 1ry suture**

WOUNDS AFTER 24 HOURS

The wounds are **contaminated** so we do ⇩

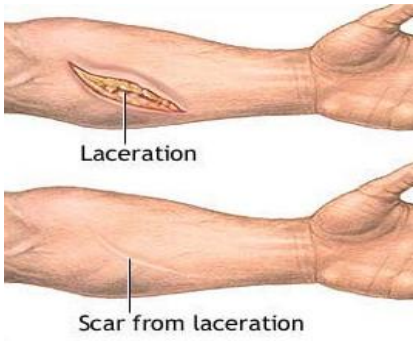
⇨ **Incised or lacerated wounds**
Depridement (**no excision** of edges) + systemic A.B + repeated dressing to allow the granulation tissues to fill the wound.
When it becomes **clean** → we do **2ry suture**

N.B.: **1- Missile injuries :** incise the skin i.e. **NO** excision
Then incise the deep fascia & excise the dead tissues

2- Amputation :
Indicated in severely lacerated wounds, uncontrolled infection or vascular gangrene.

WOUND HEALING

COMPONENTS OF WOUND HEALING



Wound contraction

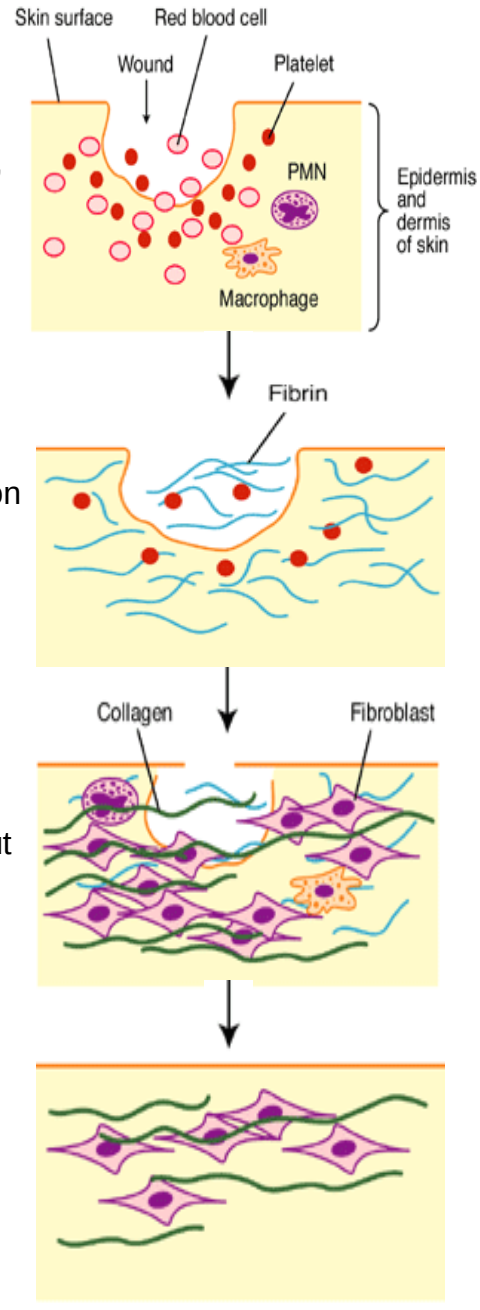
- This process of contraction helps to diminish the size of wound.
- It starts immediately & continues for the next 2 - 3 weeks.

Granulation tissue formation

- This is later on replaced by fibrous tissue

Epithelialization

STAGES OF WOUND HEALING (3 Phases)



Hemostasis & inflammation phase

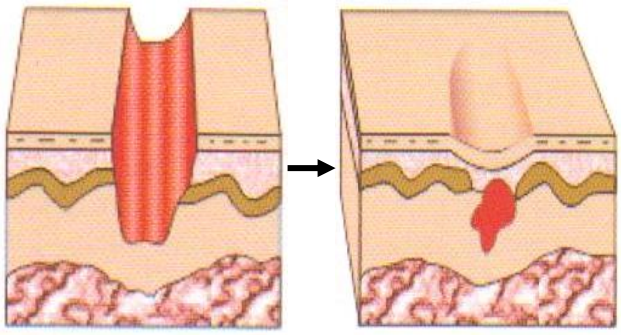
- Injury of blood vessels leads to aggregation of platelets & activation of coagulation
- This is followed by **chemotaxis** of leucocytes, macrophages & lymphocytes.
- **Macrophages** play an important role in phagocytosis & wound debridement.
- This phase lasts for about **5** days, but may be prolonged if there is wound infection

Proliferation phase

- This phase is characterized by the proliferation of **fibroblast** that secrete **collagen** fibers.
- Endothelial cells with fibroblasts form the **granulation tissue**.
- **Epithelial cells proliferation**.

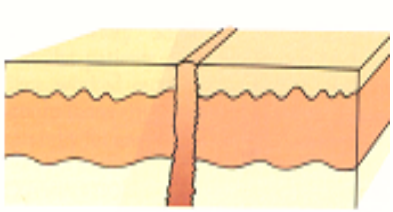
Maturation & remodeling phase

- Deposition of collagen fibers become **thicker**.
- The process of remodeling continues for about **one** year.



TYPES OF WOUND HEALING

Healing by 1ry intention



- This occurs with clean wounds,
- There is **little** amount of granulation tissues & **minimal** fibrosis.

So the final result is a **fine linear scar**.

Healing by 2ry intention

- This occurs with **septic** wounds,
- There is **marked** amount of granulation tissues & **marked** fibrosis.

So the final result is an **ugly scar**.

Healing by 3ry intention

- This occurs after 2ry suture or delayed 1ry suture.
- The aim is to lessen the fibrosis & to obtain a fine linear scar similar to 1ry intention.

FACTORS AFFECTING WOUND HEALING

General factors

① **Age :** Wound healing is slow with elderly due to ↓ protein turn over rate.

② **Nutritional status** as

- Vitamin **C** deficiency → ↓ Proto-collagen maturity.
- **Protein** deficiency → ↓ Collagen synthesis.
- Vitamin **A** deficiency → ↓ Epithelialization.
- Ca^{+}, zinc, copper & manganese play a minor role in wound healing.

③ **Cortisone administration** as it inhibits fibroblast proliferation.

④ **Irradiation** → end arteritis obliterans → ischemic wound.

⑤ **Chronic diseases :** like uremia, diabetes & malignancy delay healing

Local factors

① **Poor vascularity** → ↓ healing e.g. below knee wounds.

② **Tension** by tight sutures or hematoma → ischemia of the edges.

③ **Foreign body** & **necrotic tissues** impair wound healing.

④ **Infection :** Bacteria competes with fibroblasts for oxygen, also bacteria secretes collagenolytic enzymes e.g. hyalurindase enzyme which destroy collagen

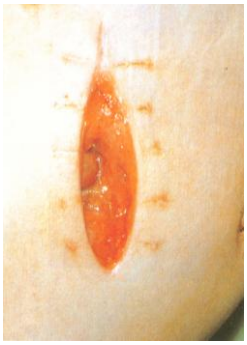
⑤ **Immobilization :** because movement delay healing.

⑥ **Adhesion to bony surface :** prevent wound contraction.

COMPLICATIONS OF WOUND HEALING

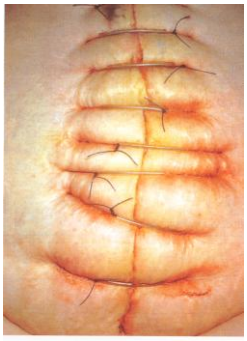
1- Wound failure (wound dehiscence)

BURST ABDOMEN



➢ **Aetiology :**
Failure of abdominal wound to heal

➢ **Pathology :** (at **6 - 8**^{lh} post-operative day)
- Warning (**Red**) sign = serosanginous discharge soaks the dressing.
- If intestine **prolapses** through wound = **Evisceration** .
- If intestine **doesn't prolapse** through wound = **Dehiscence**



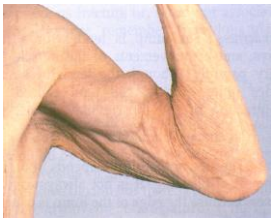
➢**Treatment:**

[A] Preoperative care :
- Cover the prolapsed bowel by a sterile dressing
- Ryle tube for suction and I.V fluids & antibiotics.

[B] Operative :
The protruded intestinal loops are washed with saline & returned to abdomen, the omentum is spread over the intestine, then the abdominal wall is closed as one layer by prolene (**tension sutures**)

[C] Post-operative care : Abdominal binder is used.

2- Stretching of the scar

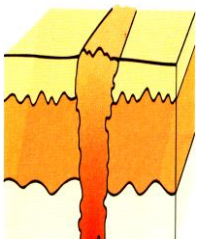


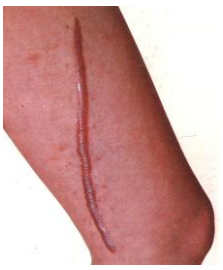
3- Contracture

This is a pathological shortening of scar tissue resulting in deformities

4- Post-operative wound infection

5- Hypertrophic scar



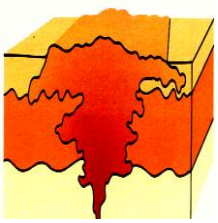


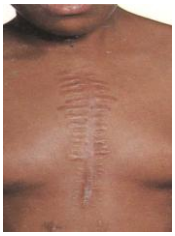
➢ **Definition :** It is an **excessive** amount fibrous tissue **confined** to scar

➢ **Aetiology : Extra-stimulus** to fibrous tissue formation during healing such as infection or excessive tension.

➢ **Treatment :** Excision + plastic repair

6- Keloid formation





➢ **Definition :** It is a **localized overgrowth** of fibrous tissue which extends **beyond** the original wound into normal tissues

➢ **Aetiology :** It occurs after wounds, bums or surgical operations..

➢ **Treatment :** It is difficult because of high rate of recurrence

① Pre & post-operative irradiation to ↓ recurrence.

② Intra-lesional steroid injections.

③ Surgical excision with intra-operative steroid injections.

MAJOR TRAUMA
MULTIPLE-INJURY PATIENT

MECHANISM OF INJURY

1- Penetrating injuries

① **Low velocity injuries :**
caused by knife & other sharp objects.

② **High velocity injuries :**
caused by a missile

2- Blunt injuries

e.g. Road traffic accident

CAUSES OF TRAUMA MORTALITY

1- Immediate deaths

The deaths occur within **few minutes** due to major trauma or injuries of the heart or major blood vessels or rupture of the major airway.

2- Early deaths

The deaths occur within **few hours** due to intra-cranial hemorrhage, massive intra-abdominal or intra-thoracic hemorrhage, or major fractures.

3- Late deaths

The deaths occur within **few weeks** due to sepsis or multiple organ failure.

MANAGEMENT OF MAJOR TRAUMA & THE MULTIPLE INJURY-PATIENT

A- First aid treatment

" **Pre-hospital management** "

① Ensure patent airway if patient is unconscious
② Control of bleeding by compression
③ Sterile dressing to prevent contamination.

Then Transfer the patient to **hospital**

B- Definitive treatment

" **Hospital management** "

The American College of Surgeons developed the
Advanced **T**rauma **L**ife **S**upport (**ATLS**)
ATLS protocol has 3 elements :

1- Primary survey / resuscitation

A = **A**ir way maintenance.

B = **B**reathing.

C = **C**irculating i.e control bleeding.

D = **D**isability i.e. support any fracture.

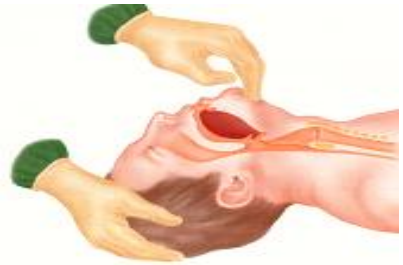
E = **E**xposure of the patients.

2- Secondary survey

3- Definitive treatment of individual injuries

1- Primary survey / resuscitation

A- Airway



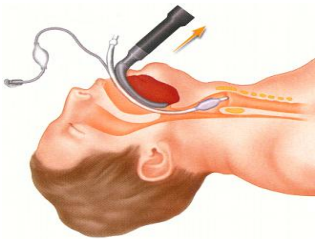
1- Clear airway

- Vomitus, blood or foreign material should be removed, this is followed by chin lift or jaw thrust.

2- Airway control

1- **Oro- pharyngeal tube** to prevents backward falling of the tongue

2- **Endotracheal tube** is indicated with ⇩



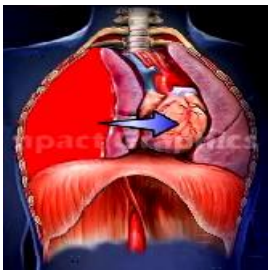
a. Apnea.
b. Inhalation injuries.
c. Maxillofacial trauma
d. Closed head injuries

3- Cervical control

- Cervical spine immobilization is done using a backboard and a rigid collar

B- Breathing

Assessment



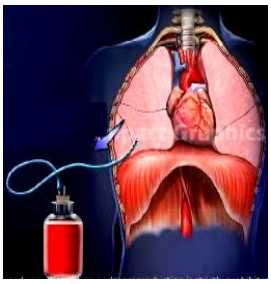
⇨ **Inspection** for chest movement, respiratory rate, cyanosis, open chest wound & expansion.

⇨ **Palpation** for subcutaneous emphysema and flail segments.

⇨ **Percussion** for hyperresonance or dullness

⇨ **Auscultation** for air entry & adventitious sound.

Life - threatening conditions & their treatment



① **Flail chest :** Immobilized by cotton pad & adhesive plaster from sternum to spine

② **Tension pneumothorax :** Deflated by needle which is inserted in **2nd** inter-costal space

③ **Massive hemothorax :** Under water seal drainage

C- Circulation

Action

⇨ **Control of bleeding** by local compression, elevation or packing

⇨ **Anti-shock measures (** discuss **)**

⇨ **ECG monitoring**.

⇨ **CPR** for cardiac arrest.

D Disability

- Common causes of neurological deficits related to trauma are ⇩

Head injury, hypoxia, shock , alcohol or drug abuse

E. Exposure & Environment

- **Clothes** of the trauma victim are removed using a sharp large scissors.
- **Warmth** using blankets to prevent hypothermia.
- **Insert ❶ Urethral catheter** (**Foley's**) to monitor urine output.
  this is contraindicated if there rupture urethra

  ❷ Nasogastric tube (**Ryle's**) to decompresses the stomach
  & to prevents vomiting & aspiration

N.B.: Proper history taking (AMPLE)

- **A**llergies
- **M**edications
- **P**ast medical history
- **L**ast meal (time)
- **E**vents of injury

2- Secondary survey

- **The secondary survey** is to be done after resuscitation efforts, after radiological assessment as plain x-rays, CTscan, MRI …..etc.
- **It includes examination of** ☜
  - ❶ Head & neck
  - ❷ Face & spine
  - ❸ Chest & abdomen (diagnostic peritoneal lavage) are indicated in blunt abdominal trauma
  - ❹ Perineum (including rectal & vaginal examinations if females)
  - ❺ Nervous system
    - ➢ Pupils for size, equality & reaction to light
    - ➢ GCS
    - ➢ Cranial nerves
    - ➢ Sensation & motor activity in limbs
  - ❻ Limbs

3- Definitive treatment of individual injuries

TRIAGE

Definition **Detection of severity of trauma which may be :**

1- **Black triage =** Victim unlikely to survive because of severity
2- **Red triage =** Victim can be helped by immediate intervention
3- **Yellow triage =** Victim transported can be delayed
4- **Green triage =** Victim with relatively minor injuries

Hge, Shock & Blood transfusion

HEMORRAGE

CLASSIFICATIONS

1- Site of bleeding

- **External :** The bleeding is visible as it occurs through the skin as in wounds or from body orifice as in epistaxis.
- **Internal** as in hemothorax or hemoperitoneum.
- **Interstitial :** The bleeding occurs into the tissues forming a hematoma.

2- Types of bleeding vessels

- **Arterial :** The blood is **bright** red in color & comes in **pulsatile jets**. The bleeding is more from proximal than distal end.
- **Venous :** The blood is **dark** red in color & comes in **steady flow** The bleeding is more from distal than proximal end.
- **Capillary :** The blood is **bright** red in color & comes as **oozing**.

3- Time in relation to onset of trauma

- **Primary :** It occurs **at the time** of trauma.
- **Reactionary :** It occurs **within 24 hours** due to slipped ligature.
- **Secondary :** It occurs **within 7-14 days** due to sepsis which dissolves the clot & erodes the arterial wall.

4- Aetiology of the bleeding

- **Traumatic** as accidents " The most common "
- **Pathological** as
  - ❶ Atherosclerotic e.g. ruptured aortic aneurysm
  - ❷ Inflammatory e.g. bleeding peptic ulcer
  - ❸ Neoplastic e.g. hematuria in renal cancer
- **Bleeding tendency** as hemophilia

PHYSIOLOGICAL RESPONSE

CLINICAL PICTURE

CLASSES OF HEMORRHAGE

MONITORING SHOCKED PATIENT

TREATMENT

see Hypovolamic shock

SHOCK

SHOCK is a **patho-physiological condition** that leads to inadequate tissue perfusion through the microcirculation with impaired cellular metabolism

HYPOVOLAEMIC SHOCK

AETIOLOGY

It is due to diminished blood Volume 2ry to ⇘

❶ **Blood loss** as in hemorrhage.

❷ **Plasma loss** as in bum.

❸ **Fluid loss** as in severe vomiting & diarrhea.

PHYSIOLOGICAL RESPONSE

Hemorrhage is the classic example of hypovolaemic shock

There are 2 aims

A- Stopping the bleeding by ⇘

❶ Immediate vasoconstriction.

❷ Retraction of intima of bleeding vessels.

❸ Subsequent clot formation.

B- Maintaining effective circulatory volume by ⇘

❶ **NEURAL FACTORS**

Stimulation of the sympathetic system with the following effects :

- **Constriction of vein** displaces blood to heart.
- **Constriction of arterioles** → ↑ peripheral resistance. It involves the arterioles of the skin not brain & heart.
- ↑ Rate & strength of **cardiac contraction**.

❷ **ENDOCRINAL FACTORS**

Mediated through the following mechanisms :

- **Catecholamine** → ↑ heart rate & myocardial contraction then constriction of arterioles of skin & viscera.
- **The rennin-angiotensin aldosterone system :** Angiotensin II (powerful vasoconstrictor) → ↑ Na & water retention
- ↑ (**ACTH**, **cortisol**, **growth hormone** & **glucagon**) → Hyperglycemia which ↑ extra-cellular fluid

❸ **TRANS-CAPILLARY REFILL**

- **Constriction of arterioles** → ↓ capillary hydrostatic pressure & promotes movement of fluid from interstitium into capillaries.

CLINICAL PICTURE

Hemorrhage is the classic example of hypovolaemic shock

Symptoms

- **Weakness** & **fainting** especially with standing.
- The patient feels **cold** & **thirsty**.

Signs

Patients vary from **anxious** to **drowsy** & usually remain **alert**.

- **PULSE** (**Rapid** & **weak**)
  - **Rapid** due to ↑ Adrenaline → direct stimulation of **S**.**A**.node
  - **Weak** due to hypotension
- **ARTERIAL BLOOD PRESSURE** (**Hypotension**)

  because in hemorrhage → ↓ blood volume → ↓ venous return and so → ↓ cardiac output → ↓ **A**.**B**.**P**
- **TEMPERATURE** (**Subnormal**)

  due to ↓ metabolism as a result from hypoxia & hypotension
- **RESPIRATORY RATE** (**Tachypnea** i.e. Air hunger)

  due to hypoxia which stimulate the respiratory center (**R**.**C**)
- **SKIN** (**Pale**, **cold** & **sweaty**)

  due to sympathetic overtone which leads to
  - Vasoconstriction of skin capillaries i.e. **pale** skin.
  - Vasoconstriction of skin arterioles i.e. **cold** skin .
- **URINE OUTPUT** **Oliguria**

  due to renal hypoperfusion + ↑ ADH

IF **The patient is rapidly treated by adequate volume replacement,**
⇨ These signs will rapidly return to normal

BUT IF **The patient is not rapidly treated by adequate volume replacement,**
⇨The followings will occur

The MOSF (**M**ulti-**O**rgan **S**ystemic **F**ailure)

- **Lung : A**.**R**.**D**.**S** (**A**dult **R**espiratory **D**istress **S**yndrome)
- **Heart :** ↓ Myocardial contractility from coronary ischemia
- **Liver :** Liver hypoperfusion → hepatic dysfunction & Jaundice.
- **Stomach :** Gut hypoperfusion → stress ulceration → hematemesis
- **Small intestine :** Gut hypoperfusion → **T**.**N**.**F** (**T**umor **N**ecrosis **F**actor) which released from hypoperfused ischemic gut
- **Kidney :** Renal hypoperfusion → ↓ **G**.**F**.**R** → acute tubular necrosis → **A**.**R**.**F** (**A**cute **R**enal **F**ailure)

CLASSES OF HEMORRHAGE

Parameter	Class I	Class II	Class III	Class IV
Blood loss	up to **15 %** (**750** ml)	**15 – 30 %** (**750 - 1500** ml)	**30 – 40 %** (**1500 - 2000** ml)	**> 40 %** (**> 2000** ml)
Mental status	Normal to anxious	Anxious to restless	Restless to drowsy	Drowsy to unconscious
Capillary refill	Normal	**> 2 Sec**		
Pulse/min	**90 -100** /min.	**100 -120** /min.	**120 -140** /min.	**> 140** /min.
A.B.P	Normal	Normal in supine only	**L o w**	
Temperature	Normal	**Cold**		
R.R	Normal	**20 - 30** /min.	**30 – 35** /min.	**> 35** /min.
Skin	Normal	**P a l e**		
Urine output	Normal **> 30** ml/h	**30 - 20** ml/h	**20 - 10** ml/h	**10 - 0** ml/h

MONITORING THE SEVERLY SHOCKED PATIENT " I.C.U "

The aim is to check the adequacy of replacement

- **Pulse :** normally = **60 - 90**/min
- **A.B.P :** normally = **90 - 150** / **60 - 100** mmHg
- **Respiratory rate :** normally = **16 - 20**/min
- **Temperature :** normally = **36.5 - 37.2**
- **Urinary output** by Foley's catheter.
- **Central venous pressure** (**C.V.P**) **:** normally = **5 - 10** cm H_2O
- **Pulmonary artery wedge pressure** (**P.A.W.P**)
  by **Swan-Ganz catheter** to estimate pulmonary artery pressure
- **Hb %, haematocrite value. electrolytes, urea & creatinine**
- **Blood gases** as **PO_2** normally = **80 - 100** mmHg
  PCO_2 normally = **35 - 45** mmHg

N.B.: Irreversible shock

➢ **It is a complete vascular collapse** with hypotension unresponsive to volume replacement or drugs intervention.

➢ **It is related to** ❶ The duration & volume of hemorrhage.
❷ The age of patient.
❸ The fitness of patient.

➢ **Before the conclusion that irreversible shock has occurred,**
The cause of unresponsive to therapy, may be due to ☜
❶ Continuous internal blood loss.
❷ Inadequate volume replacement.
❸ Acute myocardial insufficiency.

TREATMENT

Hemorrhage is the classic example of hypovolaemic shock

1- Stop the bleeding

- **1 ry Hemorrhage**
  - ❶ Proximal pressure is applied over the artery against bone.
  - ❷ Cover the wound by clean dressing i.e. packing

    N.B.: Tourniquets are contraindicated because of its complications unless the limb is going to be amputated.
  - ❸ Operative procedures, through control of bleeding points by legation or diathermy coagulation.
- **Reactionary Hemorrhage**
  - Re-exploration & control the bleeding as above.
- **2ry Hemorrhage**
  - ❶ The wound is packed with antibiotics & sterile dressing.
  - ❷ Antibiotics are given systematically.
  - ❸ If the bleeding is **not** controlled in this ways ➪ do re-exploration of the wound & legate the bleeding vessels in the wound itself.

2- Restore the blood volume " **Depending on the classes** "

- **Class II Hemorrhage** (**15 – 30 %** blood loss)
  - ❶ The replacement solution is Ringer's lactate.
  - ❷ The amount is **3 times** the estimated deficient blood.
  - ❸ **The administration : 2** litters are given as a bolus & the response is monitored, then if there is definite improvement the remaining **1** litter is given more slowly.
- **Class III** (**30 - 40 %** blood loss) & **Class IV** (**> 40 %** blood loss)
  - ❶ The replacement therapy is blood transfusion.
  - ❷ The amount is **equal** the estimated deficient
  - ❸ **The administration :**the transfusion is continued till recovery but **if no** improvement search for internal bleeding to be aspirated, then continue the transfusion till recovery.

3- Other procedures

- **Positioning :**
  - Elevate both legs while maintaining the trunk of the patient in supine position.
- **Analgesia :**
  - **Morphia** but it is contraindicated with head injuries or respiratory distress
- **Pulmonary support :**
  - **Oxygen** is administrated through a facemask, nasal catheter or endotracheal tube.

SEPTIC SHOCK

INCIDENCE

- The most serious type of shock & the most difficult to treat.
- The mortality rate > **30 %** & exceeding **80 %** if with **MOSF**

AETIOLOGY

Predisposing factors

❶ Extremes of age.

❷ D.M, malnutrition, malignancy or uremia.

❸ Patient under corticosteroids or immunosuppressive drugs.

Source of gram -ve organism

" **E. coli**, **Klebsiella** & **Pseudomonas** "

❶ Sepsis following operation of genito-urinary, intestinal tract & hepato-biliary especially when surgery is urgent.

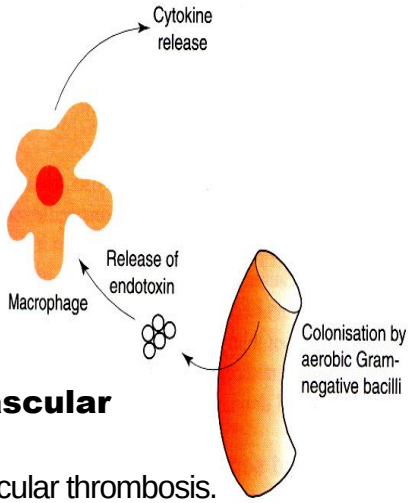
❷ Septic peritonitis.

❸ Major trauma or burn with sepsis.

PATHO-PHYSIOLOGY

Systemic Inflammatory Response Syndrome

Gram -ve organisms produce **endotoxins** which stimulate the **macrophages** to release **Cytokines**



CYTOKINES CAUSE THE FOLLOWING PROBLEMS

I. Platelets & leukocytes adherence to the vascular endothelium which leads to ⇩

- Impairment of microvascular perfusion i.e. microvascular thrombosis.
- More release of **cytokines** & **oxygen free radicals** → more damage to vascular endothelium.

II. Damage of the barrier function of the intestinal mucosa

- Allowing passage of intestinal pathogens into the circulation → Toxemia.

III. Excessive production of Nitric oxide by vascular endothelium

- Which is potent smooth muscle relaxant → peripheral vasodilatation → aggravation of the shock.

Acid-base imbalance

Shock is accompanied by **metabolic acidosis**, which caused by ↑ lactic acid & associated renal failure.

Cellular derangement

Because of hypoxia, cellular functions start to deteriorate & finally cell death

End result	**Poor perfusion of vital organs leads to Multi organ systemic failure i.e. irreversible septic shock**

CLINICAL PICTURE

Hyperdynamic (warm) septic shock

- **It is a systemic inflammatory response.**

  ❶ **Heart rate** = Tachycardia
  ❷ **A.B.P** = Hypotension
  ❸ **Temp.** = > **38** °C " **Fever** " + **chills**
  ❹ **Respiratory rate** = Tachypnea
  ❺ **Skin** = **warm** & **dry** extremities
  ❻ **Urine output** = Oliguria

- **Proper treatment at this stage, the patient will survive.**

Hypodynamic (cold) septic shock

- **It follows the above stage if not treated properly.**
- **The clinical picture** " same as hypovolaemic shock "

  ❶ **Heart rate** = Tachycardia
  ❷ **A.B.P** = Hypotension
  ❸ **Temp.** = **Subnormal**
  ❹ **Respiratory rate** = Tachypnea
  ❺ **Skin** = **Cold** clammy skin
  ❻ **Urine output** = Oliguria

- **Multi-Organ Systemic Failure (MOSF)**

MONITORING THE SEVERLY SHOCKED PATIENT " I.C.U "

As hypovolemic shock +

- Bacteriological studies.
- **Blood picture :** marked leucocytosis.
- **Location of septic source** by x-ray (abdomen - chest), U/S & CT scan.

TREATMENT

It is better to treat these patients in Intensive Care Unite " I.C.U "

- **Immediate recognition** & **early eradications** of the source of sepsis
  e.g. resect gangrenous parts & drain intra-abdominal abscess .
- **Antibiotics** : the choice of drug depend on the possible suspected organism.
  a combination of **Cephalosporin**, **Amino-glycosides**, **Metronidazole** can cover all known organisms.
- **Correction of fluid imbalance** by Ringer's lactate, plasma or blood.
- **Oxygen** mask or mechanical ventilation if **PO_2 < 60** mmHg
- **Medications** as ⇩
  - **Vasopressor** drugs as **Norepinephrine**
  - **Inotropic** drugs as **Dopamine**

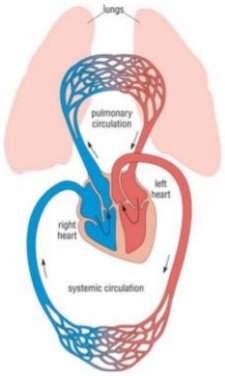
N.B.: Steroids are not indicated in treatment of septic shock

Frequent monitoring by ⇩
Pulse, A.B.P, temp., R.R, urine output, CVP, ECG, P.A.W.P & arterial blood gases

CARDIOGENIC SHOCK

AETIOLOGY

- There is inadequate blood flow to vital organs due to inadequate cardiac output, despite a normal venous return due to ⇘



❶ Acute myocardial infarction (commonest cause)
❷ Massive pulmonary embolism
❸ Severe arrhythmia

CHARACTERIZED BY

❶ Manifestation of the **cause**
❷ Congested neck vein
❸ **High C.V.P**

TREATMENT

❶ Treat the **cause**
❷ Drug to improve myocardial contractility e.g. **inotropics**
❸ **Vasodilators** to ↓ after load & ↑ cardiac output

NEUROGENIC SHOCK

AETIOLOGY

- There is paralysis of vasomotor fibers → peripheral pooling of blood & inadequate venous return due to ⇘

❶ **VASOVAGAL ATTACK**
- due to hearing bad news or sever painful stimuli
- there will be extensive vasodilatation in the splanchnic area & excessive vagal stimulation of the heart leading to bradycardia.

❷ **HIGH TRANSACTION OF SPINAL CORD**
e.g. spine fracture, or following spinal anesthesia

CHARACTERIZED BY

❶ Hypotension
❷ Normal pulse or slight **bradycardia**
❸ Warm dry skin

TREATMENT

❶ The patient should be flat with elevated legs
❷ I.V crystalloid solution as Ringer's lactate
❸ **Vasodilators** may be prescribed.

ANAPHYLACTIC SHOCK

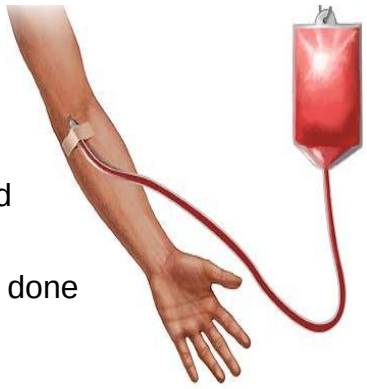
- The patient develops bronchospasm, laryngeal edema & respiratory distress
- There is massive vasodilatation & there is hypotension.
- Treated by **Corticosteroids** + **Antihistaminic** drugs

ENDOCRINAL SHOCK

- This may occur in patient with **Addison** disease or those receiving continuous **Cortisone** therapy.
- They are subjected to any stressful situation

BLOOD TRANSFUSION

PRECAUTIONS



❶ **Blood should be warmed before transfusion** to ↓ the incidence of hypothermia & Arrhythmia.

❷ **Medication should never be added** to blood used

❸ **Blood left out of refrigerator** for **> 30** min should not be used

❹ **Blood typing (ABO)** & **cross matching** must be done

BLOOD COMPONENTS

❶ **Packed red cells**
- Very useful in anemic patients especially with heart diseases as it improve oxygenation ability without over loading the circulation

❷ **Fresh plasma**
- It is rich in platelets & coagulation factors.

❸ **Fresh frozen plasma**
- It is removed from fresh blood then frozen & stored at **40** °C
- It is good source of all the coagulation factors.

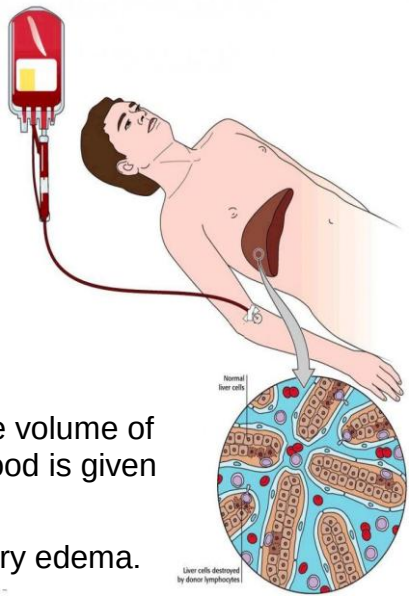
❹ **Platelet concentrates**
- It is rich in platelets & coagulation factors.

❺ **Cryoprecipitate**
- It is prepared from fresh frozen plasma & stored at **40** °C
- It is good source of factor **VIII** & fibrinogen..

INDICATIONS OF BLOOD TRANSFUSION

Product	Indication	Precautions	Storage life
Whole blood	Class **III** & **IV** hemorrhage	ABO & Rh incompatibility	**21** days
Packed RBCs	Severe anaemia	ABO & Rh	**21** days
Fresh frozen plasma	- Bleeding - Hypoalbuminemia - Ascites & burns	ABO	**1** year at (**- 40°** C)
Platelets	Thrombocytopenia	ABO	**24** – **72** hours
Fibrongen	Fibrinogen depletion	ABO	**1** year at (**- 40°** C)
Factor VIII	Hemophilia **A**		**2** years
Factor IX	Hemophilia **B**		
Albumin	- Hypoalbuminemia		**2** years

COMPLICATIONS



I- In the donor

❶ **Neurogenic shock**.

❷ **Anemia** if repeated are taken.

❸ **Local thrombophlebitis** in the veins.

II- In the recipient

A. IMMEDIATE COMPLICATIONS

❶ **Overloading the circulation** " **Heart strain** "

- **It occurs** in elderly persons especially if a large volume of blood is administrated too rapidly or blood is given faster than the heart can accept it
- **Diagnosed by** left side heart failure + pulmonary edema.
- **Treatment :**

  It is recommended to transfuse packed red cells
  but if it occurs do 1- Oxygenation.
  2- Patient is placed in semi-sitting position.
  3- **Drugs** as digitalis, morphia & lasix.

❷ **Hemolytic reactions :**

- **It occurs** due to presence of antibodies in the recipient blood which destroy the donor's R.B.Cs i.e. hemolysis.

  N.B.: Human error is frequently involved in such accident

- **Diagnosed by**
  - **In conscious patient :**
    Fever, rigors, flushing, distended neck veins, chest & flank pain
  - **In anesthetized patient :**
    Tachycardia. hypotension & unexplained bleeding .
  - **In major hemolytic reactions :** Jaundice, A.R.F & hemoglobinuria.
- **Treatment :**
  1- Stop the transfusion immediately then repeat typing
  2- I.V Ringer's lactate + mannitol to wash kidney

❸ **Pyrogenic reactions :**

- **It occurs** due to minor bacterial contamination in the transfusion apparatus.
- **Diagnosed by** chills & fever, headache, malaise & anorexia
- **Treatment :** prophylaxis is the main treatment.

❹ **Allergic reactions :**

- **It occurs** due to recipient response to allergens in the donor's blood.
- **Diagnosed by** mild itching & urticaria to severe laryngeal edema & bronchial asthma.
- **Treatment :**
  1- Stop the transfusion immediately then repeat typing
  2- Anti-histaminic administration.
  3- Corticosteroids administration

❺ Thrombophlebitis :

- **It occurs** in the vein used for transfusion.

❻ Air embolism :

- **It occurs due to** blood transfusion under pressure applied to its surface

B. Complications due to massive blood transfusion

Massive = Transfusion of 2500 ml of blood at one time

❶ Danger of cold blood : Hypothermia & cardiac arrhythmias.

❷ Citrate toxicity :

- **It occurs** in patient with liver disease or in shock.
  the liver cannot metabolize citrate → ↑ citrate which will combine with **Ca^{++}** → hypocalcaemia → tetany.
- **Diagnosed by** tetanic manifestations.
- **Treatment :**
  Administration of **I.V 10 cc** (**10 %**) Ca gluconate.

❸ Metabolic acidosis :

- The PH of 14 days banked blood **6.7** - **6.9** due to hypoxia of **RBCs** → anaerobic metabolism → lactic acid.

❹ Coagulation failure :

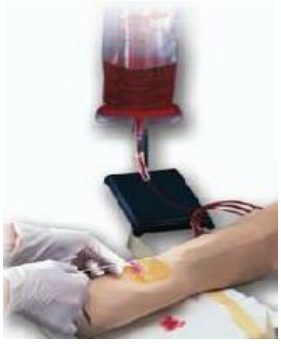
- **It occurs** due to deficiency of coagulation factors & platelets in stored blood → bleeding tendency.

❺ Hyperkalemia :

- **It occurs** due to prolonged storage of blood, there is progressive loss of **K^+** from RBCs into plasma. so with blood transfusion several units of these **K^+** may produce arrhythmia or even cardiac arrest

❻ ↓O^2 carrying capacity of RBCs

C. Late complications



- **Transmission of infections as** ☞

  - Viral hepatitis (**B** or **C**)
  (The most feared complication)

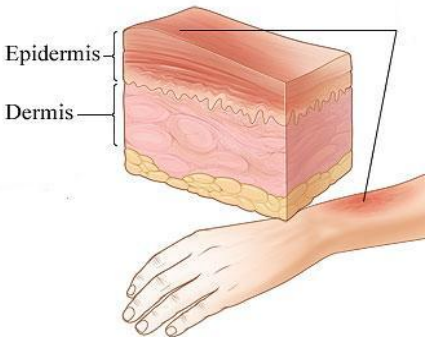
  - AIDS, syphilis, malaria & septicemia.

Burn, Plastic surgery & Surgical nutrition

BURN

DEFINITION

Coagulative destruction of tissues.



AETIOLOGY

1- Physical burns

- **Thermal :** exposure to flame or scald caused by boiled liquids.
- **Electric :** burn due to electricity.
- Exposure to **Irradiations**.

2- Chemical burns

- Acids or alkalis.
- Corrosives as potash.
- Caustics.

3- Inhalation burns

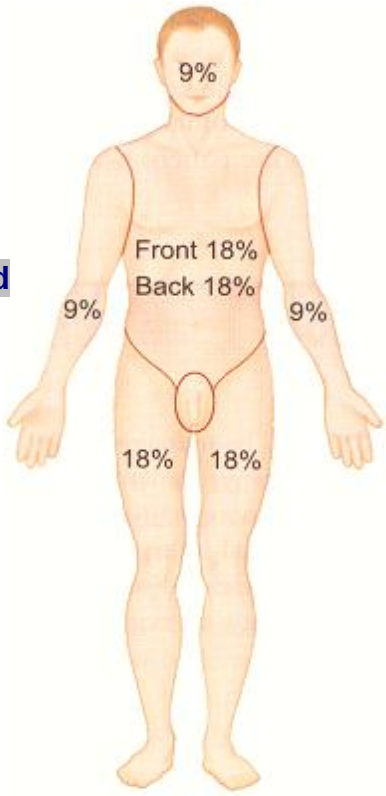
- due to exposure to hot gases.

CLASSIFICATION

1- According to the percentage of surface area involved

Role of 9 **So classified into** ⇩

Head & neck **= 9 %**	Front of trunk **= 18 %**
Each upper limb **= 9 %**	Back of trunk **= 18 %**
Each lower limb **=18 %**	Perineum **= 1 %**

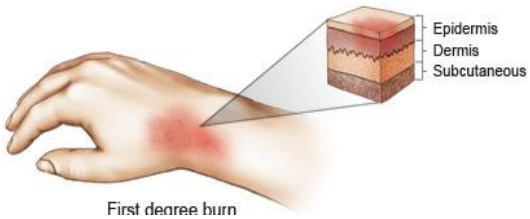


- **Major burn :**
  > 30 % of body surface area.
- **Intermediate burn :**
  (**15 – 30 %** in adult & **10 – 30 %** in children)
- **Minor burn :**
  (**< 15 %** in adult & **< 10 %** in children)

2- According to depth

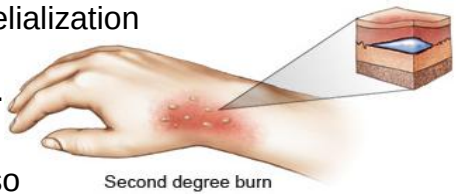
⇨ **1st Degree burns :**

- Only epidermis is damaged → erythema of skin. so they heal rapidly e.g. **sun burns**



⇨ **2ND Degree burns :**

- The epidermis & portion of dermis are damaged
- **If no infection**, the healing occurs by epithelialization from the epithelial remnants of hair follicles & sweat glands.
- **But if infection** occurs, these epithelial remnants will be destroyed & so changed to be a full thickness burn



This degree is subdivided into ⇘

1- SUPERFICIAL PARTIAL THICKNESS BURN

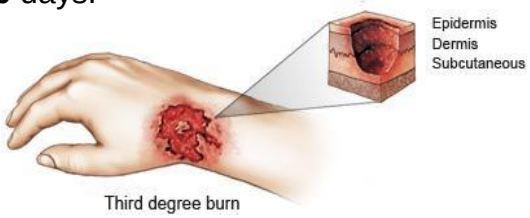
Which is usually heal in **10 -15** days.

2- DEEP PARTIAL THICKNESS BURN

Which is usually heal in **15 - 30** days.

⇨ **3TH Degree (full thickness) burns :**

- This is a complete destruction of epidermis & dermis
- The patient should be prepared for grafting.



Third degree burn

HISTOPATHOLOGY

Temperature > 45°C

- Results in protein denaturation which exceeds the capacity of cellular repair.

Thermal injury results in 3 zones

1- THE CENTRAL INNER ZONE (**Zone of coagulation**)

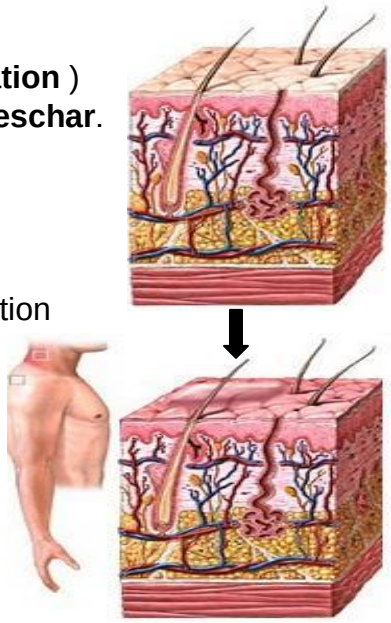
- This forms the **inner** layer of the visible burn **eschar**.

2- THE INTERMEDIATE ZONE **(Zone of stasis)**

- This area surround the zone of coagulation.
- It contains viable tissues that may die over the next 48 hours post-bum, if tissue oxygenation & adequate nutrition are not maintained.

3- THE OUTER ZONE **(Zone of hyperemia)**

- This area contains inflammatory mediators as prostaglandins
- Tissues in this zone normally recover within **7-10** days unless subjected to infection



PATHOPHYSIOLOGY

Pathological sequelae of burns

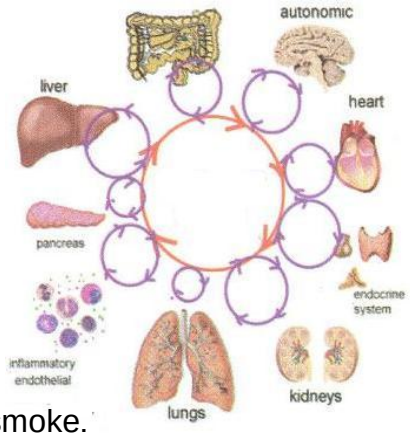
- ↑ Capillary permeability → loss of fluid & protein in the damaged area
- Excessive loss of water by evaporation.
- The burnt area will be colonized by bacteria

D.D Between **partial** & **full thickness** burn

	Partial Thickness burns	Full Thickness burns
Appearance	**Moist** due to exudated plasma	**Dry**
Pain	Very painful	Painless
Pin-prick test	**+ve** " Pin-prick test "	**–ve** " Pin-prick test "

EPILATION TEST If the hair pulls easily & painlessly, the burn is a deep one

COMPLICATIONS



Systemic complications

❶ **Shock :**
- **Neurogenic : Immediately** after burn due to pain from exposed nerve endings.
- **Hypovolaemic : 1st 48 hours** due to plasma loss from burnt surface.
- **Septic : After one week** from infection.

❷ **Respiratory system :**
- Asphyxia & laryngeal edema from inhaled smoke.
- Pneumonia, emphysema & pulmonary edema.
- **Finally ARDS** (**A**dult **R**espiratory **D**istress **S**yndrome)

❸ **CVS :**
- Anemia & hypoproteinemia.
- ↓ Cardiac output up to **50 %** from fluid loss → ↑ tissue hypoxia

❹ **Renal system :**
- Oliguria from renal hypoperfusion.
- Fluid & electrolytes imbalance.
- **Finally ARF** (**A**cute **R**enal **F**ailure)

❺ **GIT :**
- Acute true stress ulcer of stomach & duodenum
- Acute gastric dilatation & paralytic ileus
- **Finally MOSF** (**M**ulti-**O**rgan **S**ystemic **F**ailure)

❻ **Endocrinal system :**
- ↑ Catecholamine from stress.
- ↑ Cortisol & A.D.H secretions → Na^+ & H_2O retention

Local complications

EARLY COMPLICATIONS

❶ **Infection** due to bacterial colonization → septicemia or septic shock

❷ **Burns in face & neck**
→ Edema & suffocation, so urgent **tracheostomy** is done

❸ **Bums in limb & chest**
→ Constricting eschar, so urgent **escharectomy** is done

❹ **Burns in perineum**
→ Reflex urine retention, so **urinary catheter** is inserted

LATE COMPLICATIONS

❶ **Contractures** across joints

❷ **Scar :** Hypertrophy or Keloid formation

❸. **Marjolin's ulcer** which is malignant transformation

MANAGEMENT OF BURNS

A- First aid treatment

" **Pre-hospital management** "

① Ensure patent airway if patient is unconscious
② **Burn is washed with saline or tap water to ↓ pain**
③ Sterile dressing to prevent contamination.

Then Transfer the patient to **hospital**

B- Definitive treatment

" **Hospital management** "

① Assessment of burn " minor, intermediate or major"
② **3 Anti** (**Anti**-shock, **Anti**biotics & **Anti**-tetanic serum) + **Analgesic**
③ Stabilization of any orthopedic fractures.

Then According to the assessment there are " **2 possibilities** "

MINOR BURNS **No** hospitalization but

① Wash of burnt area with saline or tap water to ↓ pain
② Puncture of vesicles.
③ Remove necrotic epithelium.
④ Clean with anti-septic solution (**savlon** or **betadine**).
⑤ Apply **silver sulphadiazine** ointment
⑥ Dressing with **vaseline** gauze

INTERMEDIATE OR MAJOR BURNS

A- General treatment

I- Resuscitation & prevention of shock:

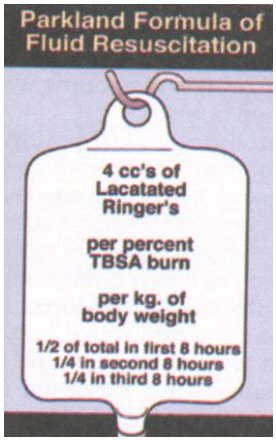
1- **A wide bore I.V cannula** is inserted rapidly for fluid therapy
2- **Ryle's tube** for suction
3- **Foley's catheter** to check the urine output.

II- Resuscitative fluid therapy

1- Evans formula

➡ **1st Day :**
1 m/Kg normal saline X % burn
+ 1 ml/Kg colloid " plasma or blood" X % burn
+ 2000 cc glucose for caloric requirement

➡ **2nd Day :**
0.5 m/Kg normal saline X % burn
+ 0.5 ml/Kg colloid " plasma or blood" X % burn
+ 2000 cc glucose for caloric requirement



2- Parkland's formula

4 ml/kg Ringer's lactate x % burn /**day**

N.B: ● **In all Formulas,** the maximum percentage of burn calculated is **50 %** otherwise serious over-perfusion will occur

● **$^1/_2$ of the above calculated amount** is given during 1st 8 hours then **$^1/_4$** at 2nd 8 hours, then **$^1/_4$** at 3rd 8 hours

● **How to judge the adequacy for resuscitation?**

① Regular follow up of vital signs.

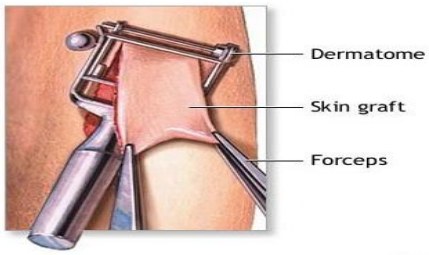
② The urine output should be **> 30** ml/hour.

③ C.V.P in critical cases.

B- Local treatment

I- Early excision :

● **In full thickness burn :**
Excision of burnt tissues
+ covering by suitable skin graft →



● **In circumferential burn**
with constricting eschar as chest & limb
linear excision is indicated i.e. Escharectomy.

II- The exposure (opened) method :

● **Technique :** The exposure method requires isolation of the patient in completely aseptic atmosphere.

● **Indications :** ① Burns of the face, neck & perineum
② Burns involving one side of Trunk.

● **Advantages :** ① More comfortable to patient.
② Avoid repeated dressing.
③ ↓ Bacterial growth by surrounding dry air.

III- The occlusive (closed) method :

● **Technique :** ① Clean the burnt area with aseptic solution.
② Apply **silver sulphadiazine** ointment
③ Dressing with **vaseline** gauze

● **Indications :** ① Circumferential burns.
② Burns in the limbs.

● **Advantages :** ① ↓ Fluid loss by evaporation.
② ↓ Edema of the tissues by compression.
③ ↓ Pain by covering exposed nerves.

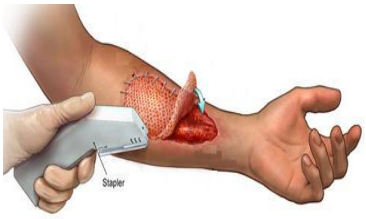
N.B.: Management of special sites of burn

➡ **Burns of head & neck :**
Steroid to ↓ laryngeal odema ± tracheostomy

➡ **Burns of perineum :**
Urinary catheter to avoid reflex urine retention.

C- Later care

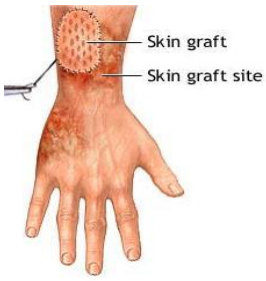
I- Autologous skin grafting :



- **Thiersch grafts** are commonly used to cover the large raw areas.

II- Biological dressings :

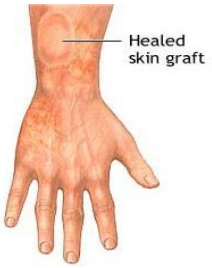
- **Indication :** If autograft are not enough .
- **Advantages :** ① The wound will be less painful.
  ② Minimize fluid & protein loss.
  ③ Control infection.
- **Examples :** ① **Allograft** (cadaver's skin)
  ② **Xenograft** (pig's skin)
  ③ **Amniotic membrane** .



PROGNOSIS OF BURNS

1- Burn factors

- **Extent :** Mortality is about **50 %** if the extent of burn is **50 %**
- **Depth :** Mortality is high with deep burn.
- **Site :** Burns of the face are the worst
- **Type :** High voltage electric burns are the worst



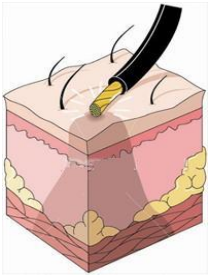
2- Patient factors

- **Age :** Extremes of age (children & elderly) have bad prognosis.
- **Concomitant diseases** e.g. DM & coronary heart disease.

3- Treatment factors

- Patients who are treated in **specialized centers** have better prognosis

Special types of burns



1- Electric burns (**A form of thermal injury**):

➡ **Tissue damage** is due to the passage of the electric current through blood & bones.

➡ **Severity of the burn** is divided into high & low tension injuries according to the voltage responsible for injury

2- Chemical burns

➡ **Tissue damage** is due to corrosive as potash .

➡ **Severity of the burn** is determined by the concentration of the agent & duration of skin contact & its mechanism of action.

3- Inhalation burns

➡ **Tissue damage** is due to **hot** gases & suspected in facial burns

➡ **Severity of the burn** is depends on site of affection
- Upper respiratory tract → laryngeal edema
- Lower respiratory tract → pneumonia

PLASTIC SURGERY

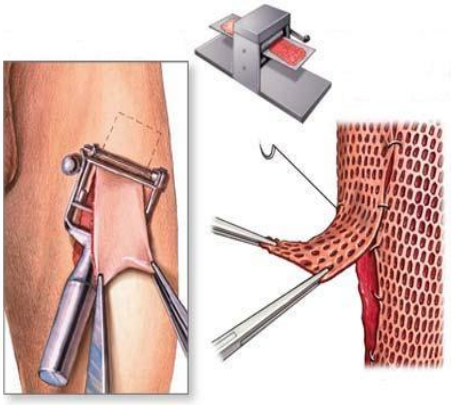
1- Aesthetic surgery

➡ **To improve the appearance**

➡ **Examples : 1-** Rhinoplasty **2-** Face lifting **3-** Eyelids surgery
4- Liposuction **5-** Breast reconstructive surgery

2- Principles of skin coverage

I- SKIN GRAFTS



DEFINITION

A segment of skin, including the epidermis & variable thickness of dermis, separated from its blood supply in donor area & then transplanted to raw recipient area.

AETIOLOGY

① Burns & trauma
② Follow surgical resection of tumors

D.D

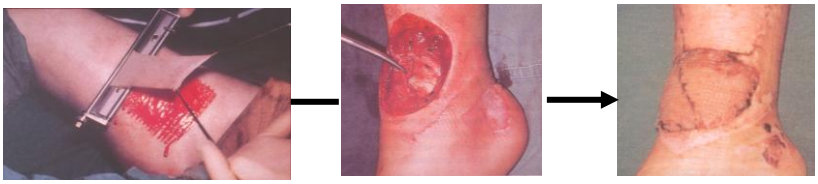
	Split thickness " Thiersch graft "	Full thickness " Wolfe graft "
	Epidermis & **part** of dermis	Epidermis & **whole** dermis
Advantages	- **Good** intake. - Donor sites heal spontaneously	- Cosmetically better. - Better sensation. - Resist to trauma **strongly**. - **Minimum** contracture.
Disadvantages	- Cosmetically poor. - Poor sensation. - Resist to trauma **weakly**. - **Maximum** contracture.	- **Bad** intake. - Donor sites must be closed surgically

FACTORS AFFECTING THE SURVIVAL OF SKIN GRAFT

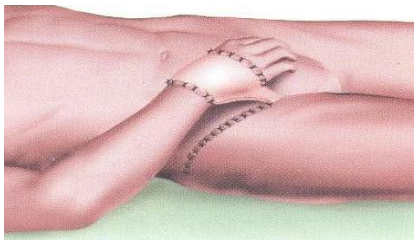
① **The vascularity** of the recipient raw area.
② **Graft thickness :** The thinner is the better.
③ **Graft size :** The smaller is the better.

EXAMPLE

• **Skin graft** is done after subfascial legation of perforators in leg venous ulcers to accelerate healing.



II- SKIN FLAPS



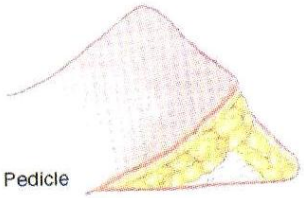
DEFINITION

A segment of skin transferred from one side of the body to another area with their blood supply.

TYPES

1- Skin flaps

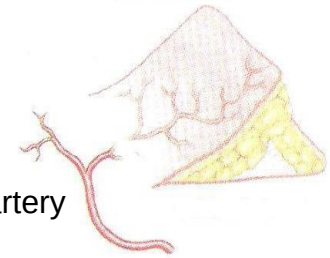
RANDOM PATTERN FLAP



No anatomically recognized blood supply,

So it should has a length **:** width **2 :1**

AXIAL PATTERN FLAP



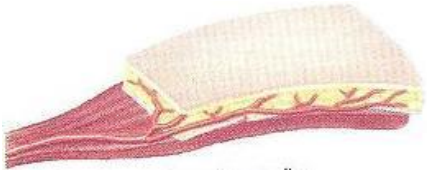
Supplied by a known artery & have no limitation as regard the length : width ratio,

So they have a wider area for rotation
e.g. forehead flap based on superficial temporal artery

DISTANT PEDICLE FLAPS

These keep the recipient site attached to the donor site by a pedicle for **2 - 3 weeks** in order to allow revascularization, before separating the base from the donor site e.g. cross finger flap

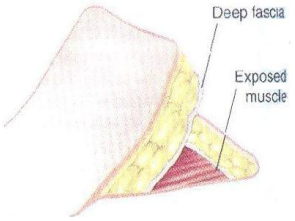
2- Myocutaneous flaps



They are flaps which receive their blood supply from underlying muscle.

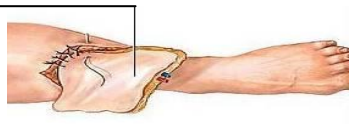
e.g. pectoralis major myocutaneous flap in head & neck

3- Fascio-cutaneous flaps



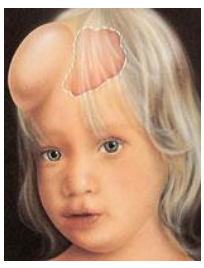
To avoid the functional deficit of muscle transfer & the bulky flap. Fascio-cutaneous flaps are used in the same way as above.

4- Microvascular free flaps



Transfer a flap totally by microvascular anastomoses of its artery & vein to a new vessels at recipient area.

III- TISSUE EXPANDERS



- **Tissue expanders** are inflatable silicon implants.
- **Over several weeks** the expander is gradually inflated with saline through a subcutaneous port.
- **The overlaying skin** is gradually stretched to accommodate a larger area.
- **Finally**, the tissue expander are removed.

SURGICAL NUTRITION

PHYSIOLOGICAL CONSIDERATIONS

Maintaining a healthy nutritional status requires the following daily balanced supplementation

	Kg	For (70 Kg) person
Water (ml)	**35**	**2340**
Carbohydrate (gm)	**2**	**140**
Fat (gm)	**3**	**210**
Protein (gm)	**0,7**	**50**
Nitrogen (gm)	**0,7**	**7**
Na^+ (mmol)	**1**	**70**
K^+ (mmol)	**1**	**70**

MALNUTRITION IN SURGICAL PATIENT

CAUSES OF MALNUTRITION SURGICAL PATIENT

Starvation

① **Social** causes as poverty.

② **Dysphagia** e.g. carcinoma of the esophagus.

③ **Loss of appetite** e.g. carcinoma of the stomach.

④ **Repeated vomiting** e.g. pyloric stenosis.

⑤ **Malabsorption** e.g. extensive inflammatory bowel disease.

⑥ Extended postoperative restriction of oral intake.

Hypercatabolism

① Major trauma & burns

② Major surgical operations

③ Severe acute pancreatitis

④ Major sepsis e.g. peritonitis & septicemia

EFFECTS OF MALNUTRITION ON THE OUTCOME OF SURGERY

① Impairment of wound healing may lead to burst abdomen

② Immunosupression with ↑ susceptibility to infection

③ Delay physical recovery & ↑ hospital stay

④ ↓ Tolerance to radiotherapy & chemotherapy

DIAGNOSIS OF MALNUTRITION

Anthropometric measures

① Recent nutritional **weight loss ≥ 10 %**

② **Body weight < 80 %** of the ideal for height

③ **Triceps skin fold thickness** measured by a caliber is an indication of fat loss

Laboratory tests

① Serum **albumin < 3.5 gm/L** = severe protein loss

② Measurement of **daily nitrogen balance**.

Immune functions

① Total **lymphocyte** count **< 1.2 X 10^9/**L.

② Impaired hypersensitivity reaction.

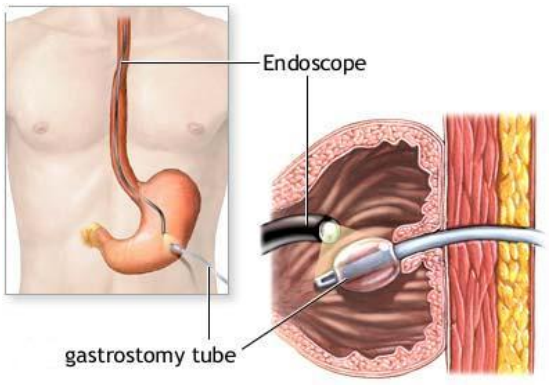
NUTRITIONAL SUPPORT

A- Enteral nutrition

INDICATIONS

patients in whom oral intake is inadequate

as (comatosed patients, severe dysphagia, burns or head & neck surgery)



ROUTE OF ADMINISTRATION

① Nasogastric tube.

② Gastrostomy tube.

③ Jejunostomy tube.

ADMINISTRATION FORMULAE

① Through **gastrostomy** → liquid diets, Juice or milk

② Through **Jejunostomy** → isotonic sterile formula at a slow rate, otherwise the patient develops colic, distention & diarrhea

COMPLICATIONS

① **Mechanical complications :**
- Pharyngeal or esophageal mucosal irritation or ulceration by feeding tube.
- Obstruction of the feeding tube .
- Tube displacement

② **G.I.T complications :**
- Nausea, vomiting, aspiration pneumonia, distention, colics & diarrhea

③ **Metabolic complications :**
- Glucose intolerance, electrolyte imbalance & malnutrition

B- Parenteral nutrition

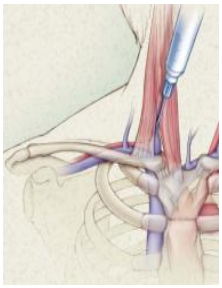
INDICATIONS

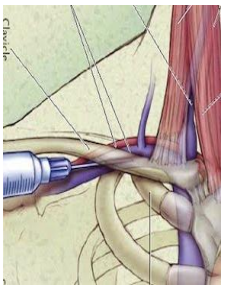
① **In malnourished or hypercatabolic patient** in whom the intestine fails to absorb nutrients e.g. inflammatory bowel disease

② **Preoperative administration** to severely debilitated patients for 2 weeks to ↓ postoperative morbidity & mortality.

ROUTE OF ADMINISTRATION

Central venous line through ① Internal jugular vein
or ② subclavian vein





ADMINISTRATION FORMULAE

① **Carbohydrates : 25 %** glucose.

② **Protein :** L-amino acids solution.

③ **Fat emulsion :** Intralipid **10 %** & **20 %** isotonic produced from **soya oil**

N.B: The amount needed daily or weekly vary according to ;
the degree of malnutrition, age & acid-base balance.

MONITORING

① **Measuring the body weight daily**

② **Balance of daily input & output**

③ **Daily laboratory :** - Full blood picture, blood urea, K+, Na+ & CL^-
- Serum albumin & sugar.

④ **Twice weekly laboratory :** - Liver functions tests.
- Blood coagulation studies.
- Serum trace elements.

COMPLICATIONS

① **Nutritional & metabolic complications :**

- **Over** or **under**feeding of the patient
- **Hypo**natremia, **hypo**kalemia, **hyper**glycemia.

② **Catheter complications :**

- **Displacement** of the catheter outside the vein
- **Puncture** of pleura → pneumothorax .
- **Injury** to subclavian or carotid arteries ± brachial plexus
- **Air embolism** if the infusion set accidentally detached from the catheter .
- **Venous thrombosis**.
- Central venous catheter **infection** → thrombophlebitis → septicemia.

Fluid, electrolyte balance & Acid-base regulation

FLUID BALANCE

PHYSIOLOGICAL CONSIDERATIONS

BODY WATER

- **Total body water** varies from **45 -75 %** of body weight.
  (**2/3** of the water is intracellular & the other **1/3** is extracellular)
- **Water source** is either exogenous or endogenous.
- **Water loss** is usually through lungs, skin, feces & urine.

BODY WATER IMBALANCE

	Water depletion (Pure dehydration)	**Water excess (Water intoxication)**
Causes	① ↓ **Intake :** as difficulty of swallow or comatosed patient. ② ↑ **Loss :** as in fever & uses of osmotic diuresis ③ **Unreplaced losses :** as ↑ loss from lungs after tracheostomy	① ↑ **Administration** of electrolyte free water to Na depleted patient. ② **Over-infusion** of **5 %** glucose to post-operative patient. ③ ↑ **Intake** as neurosis. ④ **Colo-rectal washout** by water enema instead of saline
Pathology	- If water ↓↓ → ↓ volume & ↑ osmolarity → ↑ADH & finally ↑ water re-absorption from kidney.	- If water ↑↑ → ↑ volume & ↓ osmolarity → ↓ADH & finally ↑ water excretion from kidney.
Clinical picture	① Thirst & weakness. ② ↓ Tissue turgor. ③ Oliguria	**(A) Moderate (water excess)** ① ↑ Urine volume. ② ↑ body weight. ③ ↓ Serum Na^+ concentration. ④ ↓ Haematocrite Value. **(B) Marked (water excess)** ① Brain edema → from drowsy to convulsions up to coma. ② Nausea & vomiting
Treatment	⇨ **Either** ① or ② ⇦ ① Replaced the deficit by equal volume of Na free water **In severe cases** at least ½ the estimated deficit replaced over 12hours. ② Replaced by I.V glucose **5 %.**	⇨ **According to** ⇦ ① **Moderate :** only restriction of water intake. ② **Marked :** forced diuresis by mannitol ⇨ **In severe cases** **IV** 5% Nacl solution

ELECTROLYTE BALANCE

PHYSIOLOGICAL CONSIDERATIONS

SODIUM

- **The main extracellular cation**
  that plays main role in maintaining blood volume .
- Adrenal corticoids, mainly aldosterone control the reabsorption of sodium.

POTASSIUM

- **The main intracellular cation**

ELECTROLYTES IMBALANCE

	Hyponatremia	**Hypernatremia**
Causes	① G.I.T loss :as vomiting & diarrhea ② ↑ Na loss in urine : with renal failure, salt losing nephropathy ③ E.C.F loss : - **Externally** with burns - **Internally** e.g. peritonitis, ascites, tissue edema.. .etc ④ Hypovolemia . ⑤ Adreno-cortical insufficiency ⑥ Restricted Na* in diet	① Excessive saline transfusion in the early post-operative ② Na^+ resorption with ↑ (Aldosterone or cortisone) ③ Inability to excrete Na^+ with sever illness. ④ Abnormal renal retention of Na+ with Heart or liver failure
Clinical picture	① Sunken eyes & depressed fontanels in infants. ② Dry coated tongue. ③ Dry skin & lax S.C tissue. ④ Collapsed skin veins. ⑤ Hypovolemia → tachycardia, hypotension & shock. ⑥ **↓↓C.V.P** ⑦ Oliguria.	① Slight puffiness of the face as early signs. ② The only reliable clinical sign is **EDEMA** & **ASCITES** ③ Weight gain ④ Hypertension 
Treatment	⇨ **Replacement** by Normal saline (Nacl 0.9 %) or Ringer's lactate ⇨ **If associated blood loss :** Blood transfusion is given.	⇨ **Na^+** restriction in diet ⇨ Careful use of **diuretics**

	Hypokalemia	Hyperkalemia
Causes	① **Vomiting** e.g. pyloric obstruction ② **Diarrhea** e.g. ulcerative colitis ③ External GIT fistula. ④ K^+ losing diuretics e.g. furosemide ⑤ Alkalosis & hyperaldosteronism	① Renal failure. ② Acidosis. ③ Diabetic patient
Clinical picture	① Most patients are asymptomatic. ② Malaise & weakness ③ Paralytic Ileus & distention ④ **ECG changes :** Prolonged **QT** interval, depressed **ST** segment & inverted **T** wave	① ↑ Cell excitability for muscle ② Hypotension ③ Bradycardia ④ **ECG changes :** Wide **QRS** complex & peaked **T** wave
Treatment	⇨ **Estimation of K^+ deficit** can be obtained from the total body K^+ capacity ⇨ It should be consider that K^+ is dangerous as hyperkalemia → cardiac arrhythmias, so it must be given slow IV infusion	⇨ **I.V Ca gluconate :** as ca antagonized K^+. ⇨ **I.V Na H_2CO_3** → Alkalosis → Intracellular K^+ ⇨ 20 gm **glucose** + 10 units regular **insulin** infusion.

	Hypocalcaemia
Causes	① Hypoparathyroidism ② Acute pancreatitis ③ Acute alkalosis.
Clinical picture	① **latent** (Ca+ =7 – 9 mg %) → **Chevestic's** sign & **Trousseau's** sign ② **manifest** (Ca+ < 7 mg %) → carpo-pedal spasm . ③ **ECG changes :** Prolonged **QT** interval 
Treatment	slow I.V ca gluconate 10 cc 10 %.

ACID-BASE REGULATION

PHYSIOLOGICAL CONSIDERATIONS

- **The normal** PH is (**7.4 ± 0.04**)
- **Renal regulation** by H_2CO_3 reabsorption & H+ excretion.
- **Respiratory regulation** by CO_2 elimination

Metabolic acidosis

DEFINITION This is a condition where there is a **base deficit** or **acid excess** other than H_2CO_3

AETIOLOGY

A- Over production of an organic acid occurs in ⇩

① Diabetic ketoacidosis

② Lactic acidosis of sepsis &shock.

B- Impaired renal excretory mechanism as in ⇩

① Acute renal failure

② Chronic renal failure

C- Abnormal loss of bicarbonate as in ⇩

① Diarrhea, pancreatic or small intestinal fistula.

② Uretero-sigmoid anastomosis .

DIAGNOSIS

- Nausea, vomiting & drowsiness.
- **Air hunger** " Kussmaul's respiration "

TREATMENT

⇨ **If mild to moderate acidosis** → treat the cause

⇨ **If Severe acidosis** → I.V H_2C0_3 **= Body weight (Kg) X 0.3 X base deficit**

Metabolic alkalosis

DEFINITION This is a condition where there is an **acid deficit** or **base excess**.

AETIOLOGY

① **GIT loss of H^+** as excessive vomiting or suction of gastric secretion.

② **Renal loss of H^+** with aldosterone or hypoparathyroidism.

③ **Bicarbonate retention :** either a or b

a- Na H_2CO_3 administration

b- Milk-alkali syndrome.

DIAGNOSIS

- **Cheyne-stokes** respiration (slow & deep) with periods of **apnea**
- **Tetany** i.e ↓ level of ionized Ca^{+}

TREATMENT

⇨ **If mild with** (no hypokalemia) → I.V **Nacl** infusion

But if hypokalemia → I.V **KCL**

⇨ **If severe** (Not responding to the above measures)

Give I.V Ammonium chloride & hydrogen chloride very slowly

⇨ **If Tetany** → (10 %) Ca. gluconate I.V slowly.

	Respiratory acidosis	**Respiratory alkalosis**
Definition	The condition is due to ↑ **Co^2** associated with ↓ **pH**	The condition is due to ↓ **Co_2** associated with ↑ **pH**
Causes	**(1) Respiratory depression :** ① Drug as opiate ② CNS lesion ③ Cardiac arrest **(2) Respiratory muscles disorders :** ① Myasthenia ② Flail chest ③ Obesity. **(3) Impaired alveolar functions :** ① COPD ② Pulmonary edema	**(1) Hyperventilation :** ① Hysteria ② Hyper-pyrexia **(2) Brain stem lesion :** ① Bacterial sepsis ② Ventilators **(3) Patient undergo neurosurgical procedures** may be hyperventilated → cerebral blood flow.
Clinical picture	● Restless & cyanosed patient. ● Post-operative hypertension, tachycardia & hypercapnia. **The best for sure diagnosis is serum PH & PC02**	● Tetany in severe alkalosis ● Respiratory arrest. **The Best for sure diagnosis is serum PH & PCO2.**
Treatment	⇨ **Improve** ventilation **+** ⇨ **Aiding** renal compensation	⇨ **If hysteria** → Instruct the patient to breath into a bag. ⇨ **Sometimes** → CO_2 addition to inspired gas mixture but the danger is in mistake of a compensatory metabolic acidosis.

Surgical haemostasis

SURGICAL HAEMOSTASIS

DEFINITION

The mechanism by which the body attempt to stop bleeding after injury or cutting of blood vessel

PHYSIOLOGY

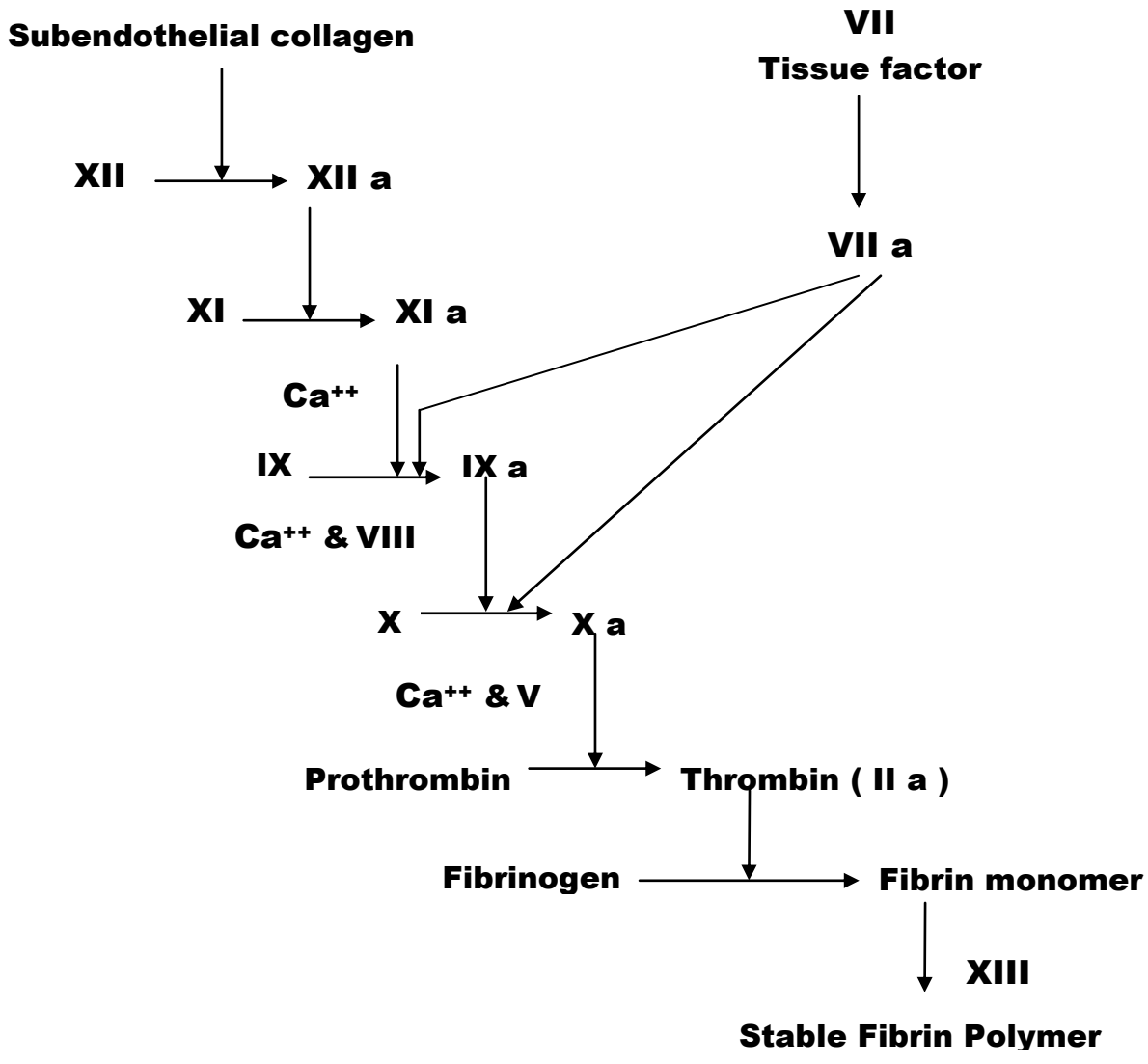
A- 1ry Haemostasis

① Vasoconstriction of disturbed vessel
② Platelets plug formation
③ Tamponade of bleeding by surrounding tissue tension

B- 2ry Haemostasis

Coagulation is activated by 2 mechanism

Intrinsic pathway | **Extrinsic pathway**



DEFECTS OF HAEMOSTASIS

A- Congenital disorders

Hemophilia A & B

- These are due to deficiency of factors **VIII** & **IX** respectively.
- Hemophilia **A** is the most common congenital coagulopathy.
- Inheritance in (both sex) & linked from (females to males)
- **Management :**
  Infusion of factor concentrate within **1** hour before surgery and for **10** days thereafter

B- Acquired disorders

These are more common than congenital ones

- **Hepatic disorders.**
- **Vitamin K deficiency.**
- **Disseminated intravascular coagulation**
- **Anticoagulants.**
- **Massive blood transfusion.**
- **Platelets disorders.**

Examples of acquired haemostatic disorders

Hepatic disorders

AETIOLOGY

Coagulation factors deficiency

① ↓ **Concentration of all clotting factors** except factor VII which is synthesized in other organ

② **Dysfibrinogenaemia :** defective polymerization of the fibrin clot.

③ ↓ **Antithrombin III** level, this contributes to intravascular coagulation in cirrhotics.

Platelets deficiency

① **Thrombocytopenia** due to destruction e.g. hypersplenism

② **Abnormal function** due to less active platelets .

Fibrinolysis

① ↓ synthesis of inhibitors of fibrinolysis,
e.g., alpha 2 anti-prothrombin.

② Impaired clearance of plasminogen activators .

TREATMENT

① **Vitamin K** administration.

② **Fresh frozen plasma** (2-3 units) replaces the missing coagulation factors

③ **Desmopressin** (0.3 ug/k) can raise the levels of factor **VIII**

④ **Tranexamic acid** or other fibrinolysis inhibitors may be useful in upper GIT hemorrhage.

Vitamin K deficiency

AETIOLOGY

① Inadequate diet

② In debilitated patients given prolonged broad spectrum antibiotics (reduce colonic bacteria)

③ Cholestatic jaundice.

④ Malabsorption

⑤ Oral anticoagulants.

FUNCTION Vitamin K is a co-factor in the synthesis of factor **II, VII**, **IX & X.**

INVESTIGATION Both the **PT** & **PTT** are prolonged.

TREATMENT

① **Vitamin K** by slow IV infusion (5-10 mg), or daily injections for 3 days (10-20 mg/day).

② In an emergency, factor concentrates (**II**, **VII, IX & X).**

③ Fresh frozen plasma may be needed.

Disseminated intravascular coagulation (D.I.C)

AETIOLOGY

① Septicemia.

② Severe shock, trauma & burns.

③ ABO incompatible transfusion.

④ **Malignancies,** especially metastatic carcinoma of the lung, pancreases, prostate & leukemia.

⑤ **Obstetric accidents** (eclampsia, amniotic embolism & retained dead fetus)

DIAGNOSIS Suspected by

① Diffuse bleeding from wounds, incisions, drain and venopuncture sites.

② Widespread bruising, purpura & mucosal bleeding.

INVESTIGATION

① Thrombocytopenia
② Both the **PT** & **PTT** are prolonged

TREATMENT

① **Treatment of the underlying cause**
to stop the cycles of coagulation/fibrinolysis،
e.g. draining an abscess & antibiotics for infection

② **Replacement** of consumed coagulation factors & platelets with fresh frozen plasma & platelet transfusion ،

③ **Blood transfusion** to restore circulating blood volume & oxygen carrying capacity since hypoxia exacerbates DIC .

④ **Heparin** to stop the thrombotic component is not widely advocated.

Anti-coagulants therapy

Can cause bleeding if the dose is not properly adjusted

Massive blood transfusion

With stored blood (look chapter blood transfusion)

Platelets disorders

Thrombocytopenia

Disorders of platelets functions

① **Drugs** as aspirin & NSAID inhibit cycloxygenase & prostaglandin synthesis، thus they interfere with platelet adhesiveness .

② **Dipyridamole** (Persantin) reduces platelet adhesiveness .

③ **Uremia** & **hypothermia** can cause platelet dysfunction.

PRE-OPERATIVE EVALUATION OF HAEMOSTASIS

HISTORY

Personal history

Age e.g. childhood (Hereditary disorder)

Present history

History of abnormal bleeding from multiple sites

Past history

History of liver disease, chrome renal failure, massive blood transfusion & drug intake.

Family history

Suggests **Hemophilia** (**A** or **B**)

N.B.: Characters of the bleeding

- **Defect 1^{ry} hemostasls :** The bleeding is superficial
  e.g. skin, mucous membrane... etc.
- **Defect 2^{ry} hemostasls :** The bleeding is deep
  e.g. muscle... etc.

EXAMINATION

① **Cutaneous signs** of liver disease, e.g. jaundice & spider naevi.

② **Skin** & **mucous membranes** are examined for bleeding،

③ **Musculoskeletal system** e.g. muscle hematoma & hemarthrosis.

④ **Abdomen** e.g. hepatomegaly & splenomegaly.

⑤ **Lymph node enlargement** may be caused by lymphoma, chronic lymphocytic leukemia & IMN

TESTS OF HAEMOSTASIS

Tests for 1^{ry} haemostasis

① Platelet count & bleeding time

② Bone marrow aspiration & biopsy:

③ Tests of platelet function (adhesion, release, aggregation)

Tests for 2^{ry} haemostasis

① **Prothrombin time** (**PT**) measures the time of clotting though the extrinsic pathways which involve factor **VII** & factors **X**,**V**,**II** and fibrinogen

② **Partial thromboplastin time** (**PTT**) measures the time of clotting through the intrinsic pathway which involve factors **XII**,**XI**,**IX** & **VIII**

Surgical infections

SURGICAL INFECTIONS

ACUTE NON SPECIFIC INFECTIONS

1- **Post-operative wound Infection (SSIs)**
2- **Boil (Furuncle)**
3- **Carbuncle .**
4- **Cellulites .**
5- **Erysipelas.**
6- **Bacteremia & septicemia**
7- **Abscess**
8- **Necrotizing fasciitis**
9- **Hydradenitis suppurativa**

1. Postoperative wound infection

SURGICAL SITE INFECTIONS (SSIS)

- **Contaminated microbe may be derived from :**
  - The patient himself (endogenous)
  - The patients environment (surgical team, Instruments, dressing ...etc)

PREDISPOSING FACTORS

General factors

① **Poor general** condition.

② **Systemic diseases** that impair host defense as diabetes or uremia .

③ **Drugs** that cause immunosuppression as corticosteroids .

Local factors

① **Poor** blood supply

② **Poor** surgical Techniques e.g. rough handling of tissues

③ **Presence of foreign bodies** as prosthetic implants.

④ **Nature of the operation** e.g. operationfor unprepared colon.

⑤ **Defect** in the sterilization technique in the operating theatre.

TYPES OF SURGICAL WOUNDS

1- Clean

There is **no** gross contamination from endogenous or exogenous sources
e.g. herniorrhaphy or thyroidectomy wounds .
⇨ **The risk of infection is 1 - 2 %**

2- Clean contaminated

Wounds involve regions of that may contain low numbers of resident organisms
e.g. urological procedures or surgery on prepared colon.
⇨ **The risk of infection is 2 – 5 %**

3- Contaminated

An unprepared region of the body with numbers of endogenous organism
e.g. surgery on unprepared colon.
⇨ **The risk of infection is 5 – 30 %**

CLINICAL PICTURE

Usually appears between the 5th & 10th days post operatively

① Postoperative fever.
② The wound is swollen tender and red.
③ Fluctuant areas or crepitus can occasionally be felt.

DIFFERENTIAL DIAGNOSIS

A- Other causes of postoperative fever

e.g. chest infections, DVT ...etc.

B- Other causes of wounds swelling

e.g. hematoma.

PROPHYLAXIS

① **Any predisposing factor** should be controlled e.g. diabetes, malnutrition
② **Prophylactic antibiotics** are indicated in ⇨
- Clean contaminated or contaminated surgery.
- In wounds where foreign material is implanted

③ **Before any elective colorectal surgery**.
Mechanical & chemical preparations of the bowel are recommended

④ **Proper surgical technique :**
e.g. gentle handling of tissues, adequate haemostasis.

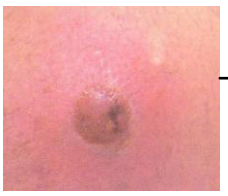
TREATMENT

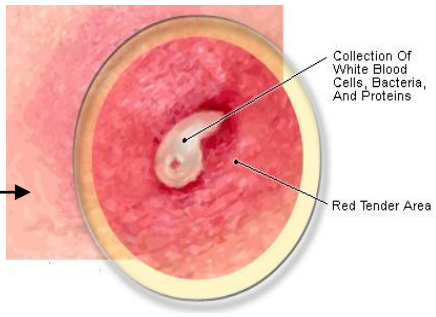
⇨ **The wound** should be opened & stitches removed to allow pus drainage.
⇨ **Antibiotics** guided by culture & sensitivity tests.
⇨ **Possible sources** of hospital infection should be traced & corrected.

2. BOIL (FURUNCLE)

INCIDENCE

More common in diabetics & whenever there is lack of personal hygiene





AETIOLOGY

This is a **staphylococcal** infection of a hair follicle or a sebaceous gland

PATHOLOGY

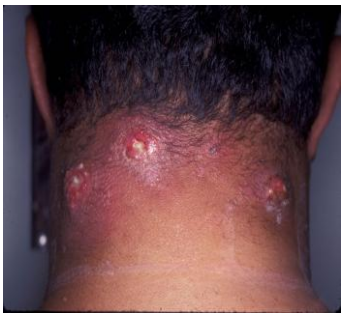
Necrosis of the central part occurs & it is discharged together with pus

TREATMENT

⇨ **Antibiotics** effective against staph. organisms. (Incision may be needed).
⇨ **Icthiol** ointment & **warm** foments
⇨ **Painting the surrounding skin** with an antiseptic to prevent infection of the neighboring glands or hair follicles

Always suspect diabetes mellitus in patients who develop recurrent boils

3. CARBUNCLE



INCIDENCE

Acute non specific infection of skin & S.C tissue ending by infective gangrene .

AETIOLOGY

- **Organism :** Staphylococcus aureus
- **Predisposing factors :** Patient > 40 years with DM or Immunosuppressed.
- **Mode of infection :** Lack of cleanliness at hairy areas of face, nape of neck, back & dorsum of hand .

PATHOLOGY

- Infection start in hair follicle then extends to S.C. tissue.
- Staph. aureus releases potent **necrotoxin** → necrosis & sloughing of overlying skin → **gangrene**

CLINICAL PICTURE

➢ **Symptoms :**

① Patient complains of furuncle that resist treatment & extends to other hair follicles
② Severe pain aching in nature (not throbbing)
③ Multiple discharging openings.

➢ **Signs :**

① The skin is dusky in color.
② Indurated swelling with marked diffuse tenderness.
③ The swelling shows multiple sinuses discharging sloughs (no pus).

COMPLICATIONS

① **Spread of infection** leads to cellulites & lymphangitis
② **Septicemia** & **pyaemia**.
③ **Cavernous sinus thrombosis,** if the carbuncle is in the face.
④ **Meningitis** & **epidural abscess,** if the carbuncle is in the back.

INVESTIGATION

Culture & sensitivity test for the discharge.

TREATMENT

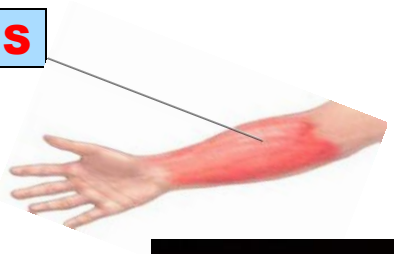
General treatment

① **Improve** general health (diet, vitamins…. etc.).
② **Control** of D.M if present.
③ **Systemic antibiotics** according to culture & sensitivity test.

Local treatment

① Local **antibiotics**
② **Glycerin magnesia** to help separation of sloughs.
③ **Excision** of sloughs.

4. CELLULITIS

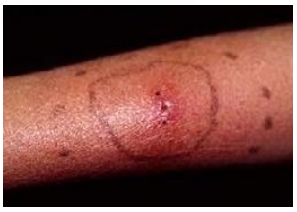


DEFINITION

This is an invasive **non** suppurative infection of the **loose connective tissue**

AETIOLOGY

- **Organism :** Gram +ve organism mostly streptococci into the superficial skin structure.
- **Mode of infection :** May be trivial, e.g. scratch or prick

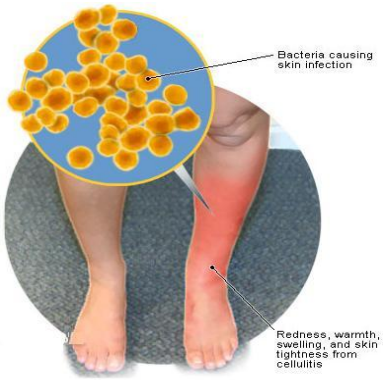


CLINICAL PICTURE

① The affected area is **red**, indurated, hot & painful

② It spreads rapidly & the Advancing edge is **ill** defined.

③ **No** suppuration except at portal of entery.



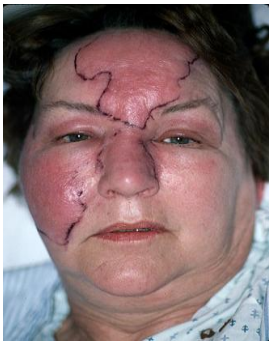
TREATMENT

① Rest & elevation.
② Local hot packs.
③ Antibiotics (penicillin group)

5. ERYSIPELAS

DEFINITION

This is a rapidly spreading **non**-suppurative infection of the **lymphatics** of the skin.



AETIOLOGY

- **Organism :** Specific strains of hemolytic streptococci
- **Mode of infection :** Through minute scratch or abrasion.

CLINICAL PICTURE **Similar to cellulitis but** ☟

① The affected area is **rose-pink**

② The Advancing edge is **well** defined, slightly raised & often shows minute vesicles just behind the spreading margin..

③ There may be islets of inflammation beyond the spreading margin separated from the main area by apparently normal skin.

TREATMENT **Similar to cellulitis but** ☟

The patient **must be isolated** because the disease is very contagious

6. BACTEREMIA & SEPTICEMIA

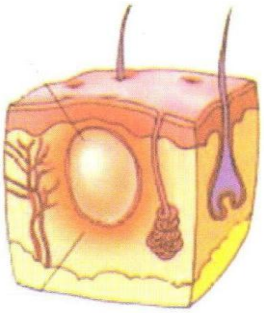
Bacteremia Presence of bacteria that are **not multiplying** in the blood.

Septicemia Presence of **multiplying** organisms in the blood stream + leucocytosis

N.B. **Toxemia** Presence of toxins in the circulation

Pyaemia Presence of septic emboli in the circulation.

7- ABSCESS



DEFINITION

An abscess is a localized **suppurative** inflammation.

AETIOLOGY

- **Organism :** The commonest are staphylococci
- **Mode of infection :** Through producing **coagulase enzyme** that helps localization of the acute inflammatory processes.

PATHOGENESIS

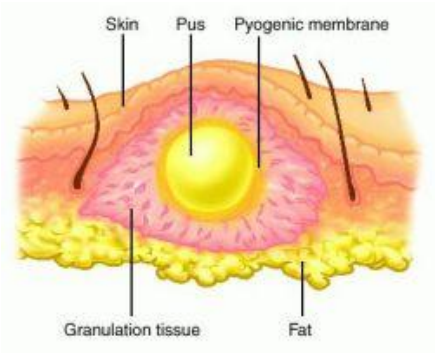
The organism reach the tissues by ⇩

- **Direct spread** through wounds, scratches & abrasions
- **Lymphatic spread** from a septic focus in their drainage area.
- **Blood spread** as in bacteremia or pyaemia.

PATHOLOGY

An abscess consists of **3** zones ⇩

① **A central zone** of coagulative necrosis
② **An intermediate zone** of granulation tissue forms a proactive layer against the spread of bacteria & their toxins.
③ **A peripheral zone** of acute inflammation fades gradually into healthy surrounding tissues.

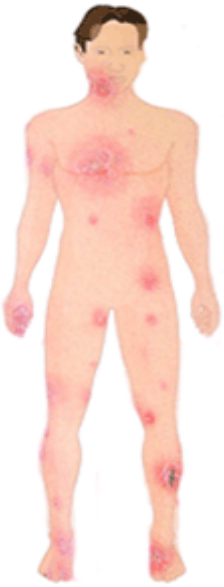


FATE

An acute abscess **may end in one of the followings** ⇩

① **Resolution** occurs if resistance is high or treatment is started early
② **Pointing** & **rupture** is the commonest.
③ **Spread of infection** either locally, lymphatics or blood stream.
④ **Chronicity :** A dense fibrous tissue around the incompletely resolved abscess

CLINICAL PICTURE



General **F**ever, **h**eadache, **m**alaise & **a**norexia

Local

- Starts as painful mass with edematous red skin.
- **The draining L.Ns :** Firm & tender
- **When pus formed :**
  ① The pain becomes **throbbing.**
  ② The fever becomes **hectic**.
  ③ The covering skin shows **pitting edema**.
  ④ **Fluctuation** can usually be elicited.

TREATMENT

Before suppuration

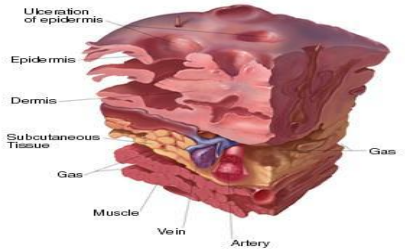
Proper **antibiotic** therapy, rest & hot application

After suppuration

Once pus forms, **the only treatment should be drainage**.

8- NECROTIZING FACIITIS

AETIOLOGY

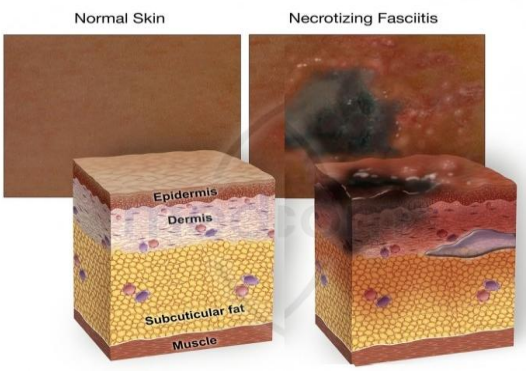


- **Organism :** Mixed microbial flora as staphylococci & streptococci
- **Predisposing Factors:** Immunocompromised patients e.g. diabetic patient

.

- **Mode of infection :** Through a puncture wound, leg ulcer or a surgical wound.

PATHOLOGY



- The infectious process spreads alone the fascial planes which leading to thrombosis of the vessels
- There is ischemic changes lead to superficial skin necrosis

CLINICAL PICTURE

General

- **F**ever, **h**eadache, **m**alaise & **a**norexia
- The patient is alert.
- Tachycardia.

Local

- **The skin shows**
  ① Hemorrhagic bullae & necrosis**.**
  ② Odema & inflammation.
  ③ Anaesthetic parts

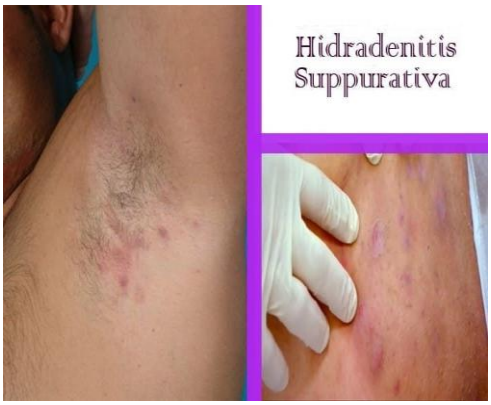
TREATMENT

Surgical Debridement (under anesthesia)

Medical Penicillin together with Gentamycin or Amikacin

9- HYDRADENITIS SUPPURATIVA

AETIOLOGY



- **Organism :** Mixed microbial flora as staphylococci & streptococci

CLINICAL PICTURE

- It affects the **apocrine** sweat glands, perineum or the axilla, produces multiple abscesses

TREATMENT

- **drainage** of abscesses followed by careful hygiene, painting with disinfectants and antifungal applications may be enough.

Otherwise, **excision** of the apocrine sweat-bearing skin followed by skin grafting

ACUTE SPECIFIC INFECTIONS

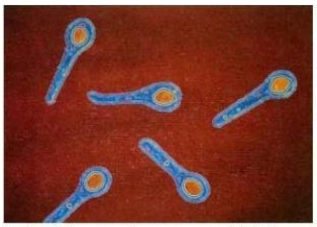
1- TETANUS

DEFINITION

Tetanus is acute specific anaerobic infection that mediated by neurotoxin of **Clostridium tetani** & leads to nervous irritability & muscular contractions

AETIOLOGY

Organism **Clostridium tetani**


Clostridium tetani spores, magnified about 3,000 times their actual size.

- Gram +**ve** anaerobic bacilli, spore forming with drum stick shape
- Naturally the organism living in small intestine of horses.
- The spores present in street dirt. This spores resist to antiseptics, heat or boiling for 5 min.

predisposing factors

The anaerobic organisms flourish more in wounds with **low oxygen** tension As ① Deep wounds especially if contused.

② Tissue anoxia from hemorrhage & shock.

③ Associated pyogenic infections which consumes local oxygen

Mode of infection

① The organism exist normally in bowels of animal

② **Tetanus neonatorum** from infected umbilical stump.

PATHOGENESIS

The organism produce powerful **neurotoxin** ➔ blood ➔ B.B.B ➔ CNS ➔ motor end plate & motor cells ➔ hyper-excitability of motor cells.
So, any minor stimuli ➔ violent generalized spasm

CLINICAL PICTURE

The incubation period in non immunized patient = 24 hours - 15 days
but in immunized patient = 11 days - several weeks

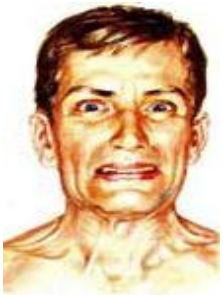
A- Stage of toxemia

↑ Temperature i.e. rigors, irritability, headache & G.I.T disturbance.

B- Stage of tonic rigidity

① Pain & tingling in the area of injury.

② Spasm of facial muscles " **risus sardonicus** " = Bitter smile & limitation of jaw movement " **trismus** " = lock Jaw.



③ Neck stiffness, difficulty in swallowing due to affection of muscles of deglutition.

C- Stage of colonic spasm " convulsion "

① Severe muscle contraction with incomplete muscle relaxation due to minor stimuli as bright light, noise ……etc

② Back is arched backwards i.e. " **opisthotonus** "

③ Spasm of diaphragm & Intercostals muscles leads to longer periods of apnea.

④ Marked tachycardia is a grave sign.

⑤ ↑ Temp, with profuse sweating.

SPECIAL TYPES OF TETANUS

- **Acute tetanus :** It affects the **uni**immunized patient with **short** incubation period, **marked** toxemia & **death** in few days.
- **Chronic tetanus :** It affects the **immunized** patient with **long** incubation period, **mild** toxemia.
- **Local tetanus :** It occurs in **immunized** patient, the spasms are restricted to the muscles around the wound.
- **Splanchnic tetanus :** It affects only muscles of **deglutition** ➔ dysphagia or muscles of **respiration** ➔ dyspnea.
- **Cephalic tetanus : It is a rare type due to wound of face or scalp**, the toxin is absorbed by the facial nerve which becomes edematous & compressed within the bony canal, so that paresis of facial muscles occurs.
- **Cryptogenic tetanus :** This is a mild form may occur without an overt wound.
- **Surgical tetanus :** It occurs postoperative due to **catgut** used in the sutures.

COMPLICATIONS

① Tear in the muscles & avulsion fracture of bone.

② Toxic myocarditis & toxic nephritis.

③ Liver failure

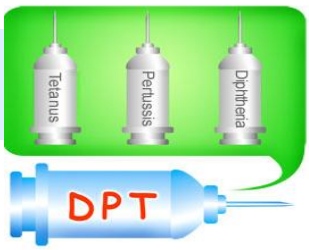
DIFFERENTIAL DIAGNOSIS

- **Trismus :** Differentiated from tempro-mandibular arthritis.
- **Meningitis :** Neck muscles affected first with turbid CSF
- **Strychnine poisoning :** The spasms are entirely colonic with complete relaxation between attacks .
- **Tetany :** Carpo-pedal spasm + ↓ serum Ca .
- **Rabies :** History of dog bite + spasm occurs mainly on seeing or drinking water. The muscles of deglution & respiration are mainly affected

LABORATORY FINDINGS

Leucocytosis may be present.

PREVENTION (Tetanus is a preventive disease)



Every child should be actively immunized by a routine (**D**.**P**.**T**) vaccines at **2**,**4**,**6**, months then a booster dose of tetanus toxoid is taken every **7** - **10** years..

So **According to this facts,**
there are 3 possibilities for prevention :

⇨ **IF patient previously received 3 or more doses of toxoid & the last one within 10 years**

- A booster dose of tetanus toxoid is needed on exposure to tetanus prone wounds ➔ **0.5** ml I.M

⇨ **IF patient previously received less than 3 doses**

- With **clean minor wounds** ➔ only tetanus toxoid is needed on exposure to tetanus prone wounds.
- With **wounds having high risk of tetanus** ➔ both tetanus toxoid & tetanus immunoglobulin (**T**.**I**.**G**) is given .

⇨ **IF Patient (not) previously immunized**

- With **clean minor wounds** ➔ only tetanus toxoid is needed but **3** doses with **4** - **6** weeks interval.
- With **wounds having high risk of tetanus** ➔ both tetanus toxoid (**3** doses) & tetanus immunoglobulin (**T**.**I**.**G**) is given **+ strong antibiotics**.

TREATMENT

A- Neutralize toxins with (T.I.G) **3000** - **6000** unites I.M.

- It is given in the proximal portion of the wounded extremity
- Repeated doses may be needed, since the half life of antibodies 3 weeks & the established tetanus often lasts longer.

B- Excise & debride the wound under anesthesia

- The wound must be left open & washed by " **hydrogen peroxide** "

C- The patient

- **Should be protected from sudden minor stimuli,** so the patient is isolated in a **dark quiet** room & nutrition is maintained by a naso-gastric tube
- **Proper sedation** is used as barbiturates but cautiously as they often cause cardio-respiratory failure.
- **Tracheostomy :** If respiratory problems is associated.
- **Penicillin-G : 10** - **40** million units / day

PROGNOSIS

The mortality rate = **30** – **60** %.

2- GAS GANGRENE

Clostridial myositis

DEFINITION

Gas gangrene is acute specific anaerobic infection caused by **gas forming Clostridium group** leads to gas formation & end by infective gangrene.

AETIOLOGY

Organism Clostridium group

- Gram +**ve** anaerobic bacilli, all of them are motile & Non capsulated **except** clostridium welchii.
- Naturally the organism living in small intestine of animal & **man**.
- **They are 2 groups :** saccharolytic or proteolytic group.

predisposing factors

The anaerobic organisms flourish more in wounds with **low oxygen** tension As ① Deep wounds especially if contused.

② Tissue anoxia from hemorrhage & shock.

③ Associated pyogenic infections which consumes local oxygen

④ Contaminated **above knee stump** by stool if patient suffer from fecal incontinence.

Mode of infection

The organism exist normally in bowels of man & animal

PATHOGENESIS

Systemic effects

- **Blood hemolysis** ➔ pallor & ting of Jaundice.
- **Degenerative changes** of liver & kidney may occur .

Local effects According to type of organism :

SACCHAROLYTIC ORGANISMS

[**Cl. welchii**, **Cl. spticum**, **Cl. edematiens**]

They ferment glycogen of devitalized or (ischemic) muscles ➔ liberation of (CO_2 + H_2) ➔ hemolysis of blood ➔ liberation of blood pigments which stain the dead muscles by a **brick red** color.

PROTEOLYTIC ORGANISMS

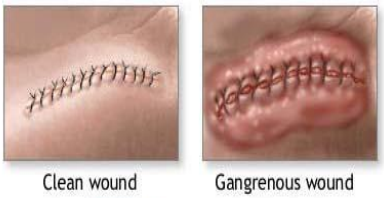
[**Cl. histolyticum**, **Cl. sporogenes**]

They ferment proteins of devitalized or (ischemic) muscles ➔ liberation of (ammonia & (H_2S) Hydrogen sulphide) ➔ hemolysis of blood ➔↑ iron from hemoglobin which combines with the (H_2S) ➔ iron sulphide which stain the dead muscles by **greenish black** color.

CLINICAL PICTURE

The incubation period varies from few hours to few days

General examination



① The patient shows pallor, fever & tachycardia.

② An icteric Jaundice & oliguria.

③ In severe cases patient is shocked

Local examination

① The wound is swollen & seen under tension with crepitus sensation .

② A sanguineous discharge with characteristic foul odor may exude from the wound.

③ The affected muscles don't contract or bleed if cut.

④ The affected muscles show red or greenish discoloration

DIFFERENTIAL DIAGNOSIS **From other clostridial infections**

- **Simple contamination :** Localized to site of infection
- **Gas abscess :** As simple contamination but no muscle affe
- **Clostridial cellulitis :** Superficial & localized to S.C tissue.
- **Localized myositis :** Localized to one group of muscles.

LABORATORY FINDINGS

Gram +ve may on stained smears of exudates.

PREVENTION

① **Adequate debridement** of wounds with excision of dead muscles.

② **Strong antibiotics** especially penicillin.

③ **Adequate circulatory support** in severe injuries is required to avoid tissue hypoxia.

N.B.: Anti-gas gangrene serum **not** used in modern surgery

TREATMENT

General treatment

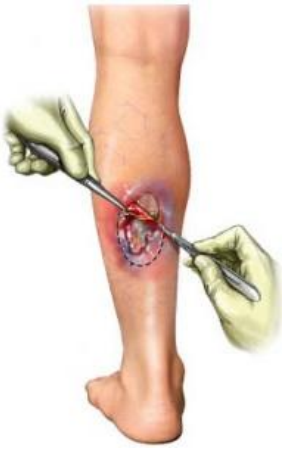
⇨ **Fresh blood transfusion**.

⇨ **Strong antibiotics :** Penicillin **l0** - **40** million Unit/ day I.V.

⇨ **Hyperbaric oxygenation**

- It is oxygen drenching in a pressure chamber.
- It inhibits bacterial invasion.
- It is given at **3** atm for **1** - **2** hours and is repeated every **6** -**12** hours **3** - **5** exposures are usually necessary

Local treatment



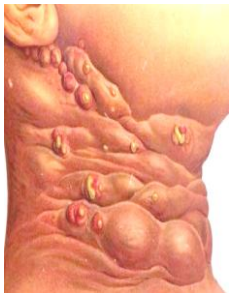
- ⇨ **The wound** is opened & all dead tissues are excised.
- ⇨ **Tight fascial compartment** are decompressed.
- ⇨ **The deep fascia** & **skin** are left open.
- ⇨ **Daily debridement** is necessary under anesthesia.
- ⇨ **Amputation :**
  - When there is diffuse myositis & complete loss of blood supply
  - When adequate debridement leaves a useless limb.
- ⇨ **Diverting colostomy :**
  - When the source of the clostridial infection is an associated injury of the colon or rectum.
  - In an extensive perineal infections.

PROGNOSIS

The mortality rate = **20** %.

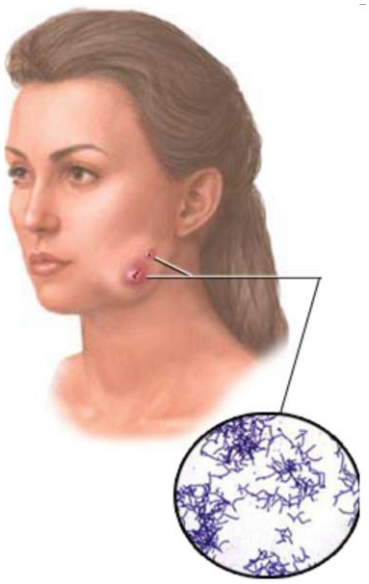
3- ACTINOMYCOSIS

AETIOLOGY



- **Organism : Actinomycetes**, which are gram-positive, They are anaerobes and part of the normal flora of the human oropharynx & tonsils..
- **Mode of infection :** In most cases the endogenous organism gain access through abrasions in the mucous membrane

PATHOLOGY



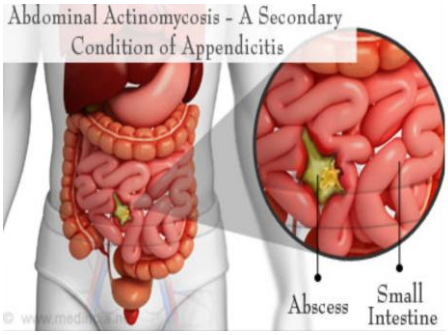
- The disease is characterized by inflammatory nodular masses, which may liquify to form abscesses and sinuses.

CLINICAL PICTURE **4 clinical types :**

1- Facio-cervical type (60 %)

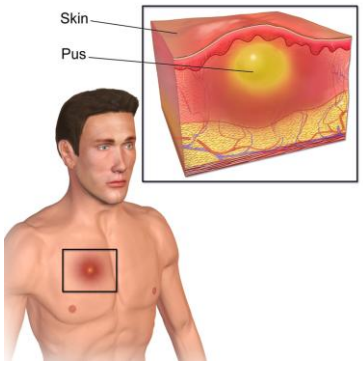
- This is the commonest type.
- A hard painless, non-tender mass appears in the parotid region, associated with trismus & thickening of the lower jaw.
- Multiple nodules then appear with softening and formation of abscesses and sinuses discharging pus and sulphur granules.

2- Abdominal type (20 %)



- An indurated mass affects the appendix or ileocecal region and later invades the abdominal wall resulting in multiple sinuses.
- Occasionally the liver is affected by blood spread from the ileo-cecal region
- The liver becomes enlarged & multiple abscesses are formed.

3- Thoracic type (15 %)



- The disease occurs by aspiration from the upper air passages or by direct spread from the neck.
- Later in the course of the disease the sinuses perforate the pleural cavity & the chest wall, often involving the ribs or vertebrae.

4- Cutaneous type (5 %)

Rarely affected by direct inoculation.

TREATMENT

Surgical **Excision** or drainage

Medical **Penicillin** for many weeks.

.....

COMPLICATIONS OF SURGICAL INFECTIONS

1. **Spread of infections :**
   a) ***Direct spread*** as necrotizing infections & abscesses.
   b) ***Lymphatic spread*** producing lymphangitis
   c) ***Blood spread*** produces bacteremia & septicemia.
2. **Fistulae** & **sinus** tracts
3. **Necrosis** or **gangrene** of the affected part.
4. **Immuno-suppression** so suppressed wound healing.
5. **S**ystemic **I**nflammatory **R**esponse **S**yndrome (**SIRS**)

Hand infections & Antibiotics

HAND INFECTIONS

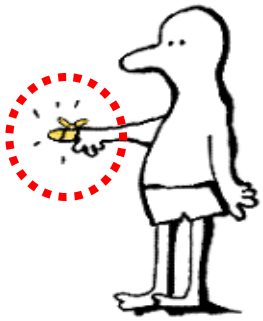
Due to the use of the hand & its exposure to contamination, the frequency of hand infection is high

So **Proper treatment is needed to preserve the function of hand.**

CLASSIFICATION

A- Cutaneous & S.C infections

① **Paronychia** (Acute & chronic)
② **Pulp** space infection
③ **Web** space infection



B- Fascial spaces infections

① **Midpalmar** space infection .
② **Hypothenar** space infection .
③ **Theanar** space infection

C- Synovial sheath infections

① **Acute digital tenosynovitis**
② Ulnar bursitis .
③ Redial bursitis.

D- Bone & joints infections

CLINICAL PICTURE

History Usually manual workers with history of prick or usually house wives.

Generally The condition presents with pain, swelling, & fever.

Locally ① Redness, tenderness, hotness & dorsal edema overlying skin.
② Firm & tender L.Ns.
③ The fingers can't be approximated because adduction pain

INVESTIGATION

① **Plain x-ray** if the presence of a foreign body is suspected.

② **Blood sugar tests** for those with recurrent infections, may reveal the presence of diabetes mellitus.

TREATMENT

⇨ **Early administration** of strong antibiotics.

⇨ **Early drainage** of infection " **don't wait for fluctuation** "

⇨ **Incision** (never crosses hand crease) & under **general anesthesia**.

⇨ **Incision** is done under tourniquet to have bloodless field

⇨ **Early restoration** of function by movement to avoid stiffness of joints.

⇨ **General** & **local** rest in elevated position .

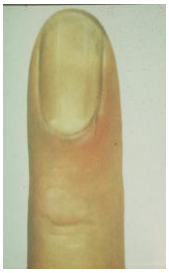
CUTANEOUS & S.C INFECTIONS

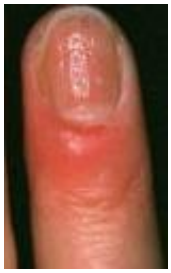
I. Paronychia

1- Acute paronychia

DEFINITION

Acute suppurative infection of the **nail fold**.





INCIDENCE

The commonest type of hand infections.

AETIOLOGY

After trauma or removal of excess skin

CLNICAL PICTURE

As general +

Localized painful swelling of the **nail fold**

TREATMENT

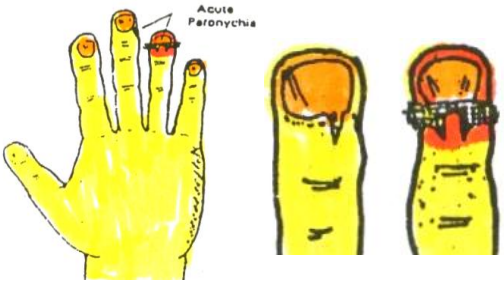
As general +

Unilateral

Triangular incision in the skin fold to raise nail fold.

Bilateral

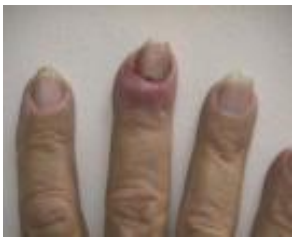
Incision of the skin at the both angles of nail fold, raising the nail fold



2- Chronic Paronychia

DEFINITION

Chronic inflammation of the **nail fold**



INCIDENCE

Common with washer women.

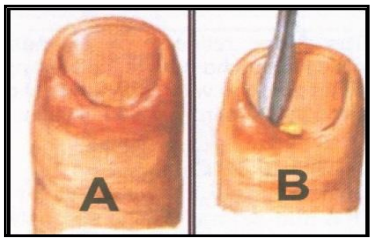
AETIOLOGY

It a fungal infection.

CLNICAL PICTURE

As general +

Localized painful swelling of the **nail fold** with trophic changes.



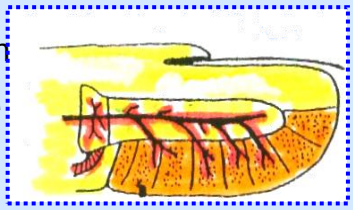
TREATMENT

As general +

① Anti-fungal treatment
② Avoid water.
③ Nail extraction.

II. Pulp space infection

SURGICAL ANATOMY

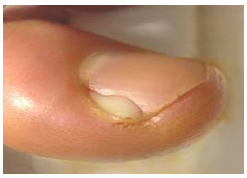


- **Pulp space** is closed compact space between skin & periosteum of terminal phalan
- **It is shut from** the middle pulp by a transverse septum attached to bone.
- **It is filled** with fat & partitioned by incomplete fibrous septa

AETIOLOGY

Infection mainly by **staph**, organism through direct inoculation by a pin prick or extension from **paronychia**

CLNICAL PICTURE



As general +

Localized painful swelling over the **distal pulp**

COMPLICATIONS

① **Tenosynovitis** (tenderness over proximal fingers & palm)

② **Arthritis** (limited movement of all Joint of hand)

③ **Lymphangitis** & **lymphadenitis**

④ **Extension** to mid or proximal pulp spaces

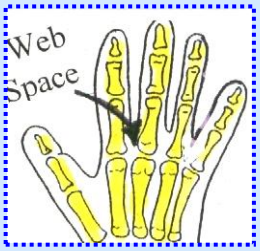
TREATMENT

As general +

⇨ Incision over the inflamed point, & all septa are incised to transform it into one single cavity evacuating all pus inside.

III. Web space infection

SURGICAL ANATOMY



- **Web space** is S.C. spaces between the **4** digital slips of palmar apponeurosis .
- **It is bounded by :**
  - ① Proximal phalanges on each side
  - ② Palmar skin infornt
  - ③ Dorsal skin behind
- **It is filled with** fat & crossed by the lumbrical muscles + neurovascular bundle.

AETIOLOGY

Infection mainly by **staph**, organism through direct inoculation by a pin prick or extension from **mid** or **proximal pulp space infection**.

CLNICAL PICTURE

As general +

Localized painful swelling over the **web space**

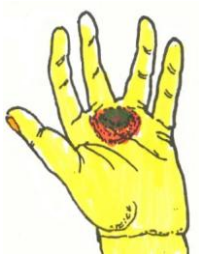
COMPLICATIONS

① **Tenosynovitis** (tenderness over proximal fingers & palm)

② **Arthritis** (limited movement of all Joint of hand)

③ **Lymphangitis** & **lymphadenitis**

④ **Extension** to mid palmar spaces, hypothenar or thenar spaces



TREATMENT

As general +

⇨ **Transverse incision** on palmar surface of web, near its free border, then a sinus forceps are opened in a longitudinal direction (to avoid damage to the digital nerves & vessels)

⇨ **Counter incision** may be done posteriorly if the abscess communicates with a dorsal pocket.

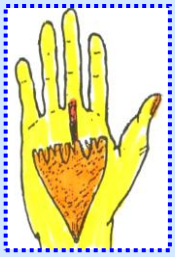
.....

FASCIAL SPACES INFECTIONS

I. Mid-palmar space infection

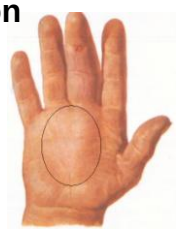
SURGICAL ANATOMY

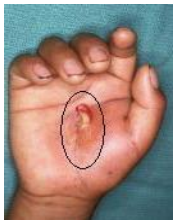
- **It is bounded by :**
  - ① **Anteriorly** by palmer apponeurosis.
  - ② **Medially** by medial septum separating it from the hypothenar space
  - ③ **Laterally** by the lateral septum separating it from the thenar space.



AETIOLOGY

Infection mainly by **staph**, organism through direct inoculation by a pin prick or extension from **web space infection** between **middle** & **ring** fingers.





CLNICAL PICTURE

As general +

Localized painful swelling over the the **mid-palmar cavity** i.e. (**Frog's hand**)

TREATMENT

As general +

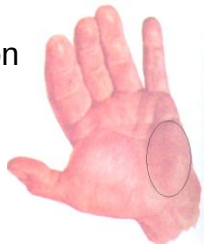
⇨ **Transverse incision** is done in distal palmar crease.

⇨ **To avoid injury of deep important structures**, the incision is made through the skin only & then a sinus forceps is introduced inwards & is gently opened to let out pus (**Hiltons method**)

II. Hypothenar space infection

AETIOLOGY

Infection mainly by **staph**, organism through direct inoculation by a pin prick or extension from **web space infection** between **little** & **ring** fingers.



CLNICAL PICTURE

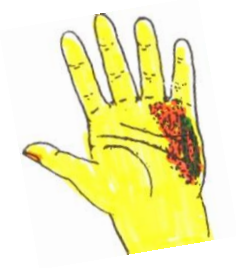
As general +

Localized painful swelling over the the **hypothenar space**

TREATMENT

As general +

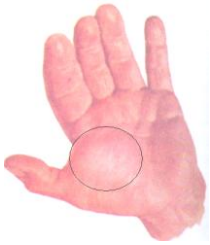
⇨ **Vertical incision** along the medial border of the 5th metacarpal bone is done. The small muscles of the little finger are reflected and the space is entered by a sinus forceps i.e. (**Hiltons method**)



III. Thenar space infection

AETIOLOGY

Infection mainly by **staph**, organism through direct inoculation by a pin prick or extension from **web space infection** between index & **middle** fingers.



CLNICAL PICTURE

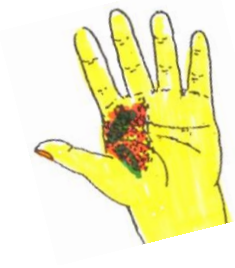
As general +

Localized painful swelling over the the **thenar space**

TREATMENT

As general +

⇨ **Transverse incision** at the distal end of the thenar eminence at the web then the space is open by a sinus forceps i.e. (**Hiltons method**)



⇨ **Vertical incision** on the lateral aspect of the back of the 2nd metacarpal bone, this is the incision commonly used

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SYNOVIAL SHEATH INFECTIONS

Teno-synovitis

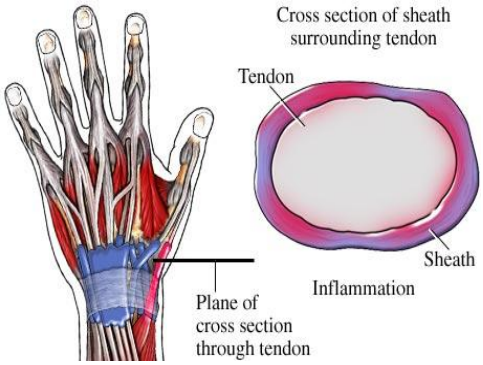
SURGICAL ANATOMY

- **The middle 3 fingers** are surrounded by **3** tendon sheath which extend from the distal phalanx to the head of the corresponding

AETIOLOGY

Infection mainly by **staph**, organism.
extended from cutaneous & S.C tissue

PATHOLOGY



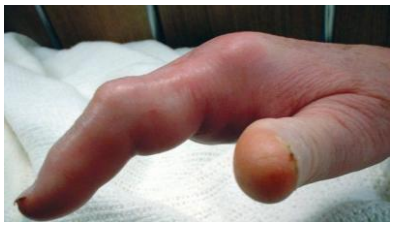
- **Synovitis** is associated with excessive clear synovial fluid which turned to be turbid by pus.
- **The sheath** will be distended.
- The nutritional element to tendons will be impaired by inflammatory thrombosis of vessels

CLNICAL PICTURE

As general +

① Symmetrical swellings of fingers .

② Semiflexion of all joints (**Hook sign**)



③ Extension of inter-phalangeal joints is very painful, while metacorpo-phalangeal joints is slightly painful

TREATMENT

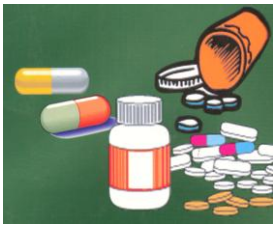
As general +

⇨ **Transverse incision** over distal crease then aspirate the pus by a catheter & inject antibiotics.

.....

ANTIBIOTICS

CHOICE OF THE SUITABLE ANTIBIOTICS



- **2 Factors should be considered;**

1- The patient

① Age, sex if female (pregnancy & lactation)

② The condition of renal & hepatic function i.e sites of antibiotics metabolism

③ History of allergy to antibiotics.

④ Uses of other drugs which ↑ side effects of antibiotics e.g. oral contraceptives.

2- The pathology & causative organism

GUIDE LINES FOR ANTIBIOTICS PRESCRIPTION

① An initial diagnosis is essential before stating antibiotic therapy.

② Length of antibiotics course based on pathology & clinical improvement.

③ To change from antibiotic to another based on culture & sensitivity

COMPLICATIONS OF ANTIBIOTICS

① Hypersensitivity reaction commonly with penicillin including urticaria, fever & asthma .

② Vitamin B deficiency due to alternation of bowel flora especially with prolonged used

③ **Specific toxicities :** e.g. nephrotoxicity, ototoxicity ....etc.

ANTIBIOTICS IN COMMON USE

A- B-Lactam antibiotics

PENICILLINS

- **Penicillin G :** effective mainly against **gram +ve** bacteria such as **streptococci**
- **Ampicillin:** effective mainly against **gram-ve** bacteria & **enterococci**.
- **Amoxicillin** with a **lactamase inhibitor** (**Clavulanate**) serves to extend the spectrum against **gram +ve**, **gram-ve** bacteria, aerobic & some anaerobic organisms.

B - Cephalosporins

1ST GENERATION as **Cephalothin**, have a activity against **gram +ve** bacteria

2ND GENERATION as **Cefuroxime** have less **anti-gram +ve** activity & much better **gram-ve** aerobic activity.

3RD GENERATION as **Ceftazidime,** have **anti-gram -ve** activity & pseudomonas with variable **gram +ve** activity

C- Aminoglycosides

- This group includes **Gentamycin** & **Amikacin** which effective mainly against **gram-ve** aerobic bacteria with little activity against **gram +ve** bacteria
- They all have considerable ototoxicity & nephrotoxicity.

D- Quinolones

- This group includes **Ciprofloxacin** & **Norfloxacin** which effective mainly against **gram-ve** aerobic bacteria, they commonly used in U.T.I

E- Glycopeptides

- These agents (e.g. **Vancomycin**) have activity against **gram +ve** bacteria & more potent than other agents .

F- Tetracycline & Chloramephenicol

- This group has anaerobic activity

G- Trimethoprim-sulhamethoxazole (Septrin)

- These agent against **gram-ve** aerobic bacteria & some infections observed in immunosuppressed patients as with Pneumocystis carnii

H- Metronidazole

- It has excellent anaerobic activity but no effect on aerobic organisms

I- Erythromycin

- It is a **Marcolide** drug that has activity against **gram +ve** bacteria & usually used in patients allergic to B-lactam agents

J- Clindamycin

- These agent have excellent anaerobic activity & some **gram +ve** activity as well

Tumors & Transplantation

TUMORS

HISTOGENIC CLASSIFICATION OF TUMORS

	BENIGN	MALIGNANT
Epithelial tumors		
• Squamous epithelium	- Papilloma	- Squamous cell carcinoma - Basal cell carcinoma
• Columnar epithelium	- Adenoma	- Adenocarcinoma
• Transitional epithelium	- Papilloma	- Transitional cell carcinoma
Connective tissues		
• Adipose	- Lipoma	- Liposarcoma
• Fibrous	- Fibroma	- Fibrosarcoma
• Cartilage	- Chondroma	- Chondrosarcoma
• Bone	- Osteoma	- Osteosarcoma
• Smooth muscle	- Leiomyoma	- Leimyosarcoma
• Striated muscle	- Rhabdomyoma	- Rhabdomyosarcoma
Neuroectoderm		
• Nerve cells	- Ganglioneuroma	- Neuroblastoma
• Melanocytes	- Pigmented nevus	- Malignant melanoma
• Meninges	- Meningioma	- Malignant meningioma
• Nerve sheaths	- Neurofibroma	- Neuroflbrosacoma
Hemopoietic & Lymohoreticular tissue		- Leukemias - Lymphomas
Blood vessels	- Hemangioma	- Hemangiosarcoma
Lymph vessels	- Lymphangioma	- Lymphangiosarcoma

AETIOLOGY OF CANCER

1- Onchogenesis

Agents that damage genes that initiate the malignant transformation.

① Chemical agents ② Physical agents
③ Viruses ④ Diet

2- Another category:

Agents not damage genes but enhance the growth of tumor cells

e.g. Hormones 1- **Estrogen** stimulate growth of cancer breast
2- **Androgen** stimulate growth of cancer prostate

3- Chemical agents

a. Tobacco smoke (mainly of **cigarettes**)

e.g. cancer lung, esophagus, urinary bladder & pancreas.

b. Occupational agents :

e.g. - **Asbestos** ➔ Mesothelioma of lung
- **Aromatic amines** ➔ Transitional cell carcinoma of urinary bladder

4- Physical agents

a. Mechanical irritation :

e.g. **gall stones** ➔ cancer gall bladder.

b. Ionizing radiation :

e.g. **α & β rays** ➔ cancer in man & animal.

c. Ultraviolet rays :

e.g. cancer skin.

5- Viruses

a. Human papilloma viruses :

Sexually transmitted ➔ cancer cervix & anus.

b. Hepatitis B & C ➔ Hepatocellular carcinoma.

6- Diet

a. Fat ➔ Cancer colon & rectum.

b. Alcohol ➔ Cancer upper digestive tract & hepatocellular carcinoma.

7- Idiopathic

STAGES OF CANCER DEVELOPMENT

① **Hyperplasia :** The cells look normal but reproduce to too much cells.

② **Metaplasia :** Change of type of epithelium into another type.

③ **Dysplasia :** The cells becomes a typical in size & shape.

④ **In situ :** The cells not invade the basement membrane.

⑤ **Invasion :** The cells begin to invade the neighboring tissue

⑥ **Metastasis :** The cells tend to reach blood & lymphatics

GRADING OF CANCER

Grading in a measure for tumor aggression

- **Well** differentiated tumors : The least aggression.
- **Moderately** differentiated tumors.
- **Poorly** differentiated tumors : The most aggression

STAGING OF CANCER (T.N.M)

(**T**) = Extent of 1ry tumor in size & depth.

(**N**) = Presence or absence of lymph nodes.

(**M**) = Presence or absence of metastasis.

SPREADING OF CANCER

Properties that allow metastasis

① **Defective cell adhesions :**

Cancer cells lack of adhesive proteins which bind the cells to another.

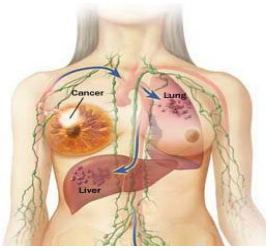
② **Tumor angiogenesis :**

Cancer cells access to circulation through newly formed capillaries.

③ **Production of proteolytic enzymes :**

Which digest the basement membrane allowing invasion.

Mode of metastasis



① **Local spread :** To neighboring organs & tissues.

② **Lymphatic spread : 2 types**

⇨ **Permeation :** Malignant cells invade the endothelium of lymphatics then grow inside the lymph vessels,

⇨ **Embolization :** Malignant cells carried as an emboli to draining L.Ns.

③ **Blood spread :** Malignant cells invade to capillaries as an emboli to **l**ung, **b**one، **l**iver & **b**rain.

④ **Transcoelomic spread :** Travel along cavities e.g. peritoneum

DIAGNOSIS OF CANCER

A. Screening

- Some people may have a higher risk of developing a certain malignant tumor
- So certain screening programs are done to detect the neoplasm as early as possible.
- A common example is to do **soft tissue mammography** for females who have a higher chance of developing breast cancer .

B. Radiological

- Various radiological techniques including **contrast studies**, **ultrasound** & **C.T**

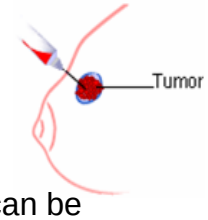
C. Endoscopy

- This is very useful for diagnosis of most lesions of the **respiratory**, **gastro-intestinal** & **urinary**.

D. Histology

- **Needle** or **operative biopsies** essential for tissue diagnosis.

E. Cytological examination



- **Fine needle aspiration cytology** is now a well established line of investigation which is commonly used to diagnose lesions of the thyroid, breast.. .etc

F. Tumors markers

- Many malignant tumors secrete certain **oncofetal proteins** which can be established. This may help in the diagnosis of certain tumors

EXAMPLES INCLUDED

① **α-feto-protein** is raised in **hepatocellular** carcinoma & **testicular** tumor.

② **CEA** (**C**arcino-**E**mbryonic **A**ntigen) is raised with cancer **colon** & **stomach**

③ **Prostatic specific antigen** is raised in **prostatic** carcinoma.

④ **CA 15 - 3** is raised in carcinoma of the **breast**.

⑤ **CA 125** is raised in carcinoma of the **ovaries**.

⑥ **CA 19 - 9** is raised in carcinoma of **colon**.

⑦ **Thyroglobulin** is raised in carcinoma of **thyroid**

TREATMENT OF CANCER

(A) Early (potentially curable, operable) cancer

- ⇨ **Treatment is radical**.
- ⇨ **Adjuvant** (complementary) treatment of systemic modalities such as **chemotherapy** is indicated if there is a high possibility of systemic microscopic spread in distant sites.

(B) Late (incurable, inoperable) cancer

- ⇨ There are **distant metastases**.
- ⇨ Cure is **not** possible.
- ⇨ Treatment aims to **palliate** of the patient's symptoms so as to provide him with a reasonable life quality.
- ⇨ Treatment is also essentially by **systemic modalities** as chemotherapy & hormones.
- ⇨ Surgery or radiotherapy is sometimes needed to palliate local symptoms.

➡ **The individual modalities of treatment include** ⇩

1- SURGERY

Primary tumor

Radical surgery aims at excision of the primary tumor with as wide a safety margin.

Lymph nodes

The treatment of lymph nodes varies from tumor to another

- **G.I.T. malignancies :** Lymph nodes are routinely resected
- **Breast cancers :** They are either resected or irradiated
- **Head & neck malignancies :** The nodes are treated only if they prove to contain malignant deposits.

Advantages

Surgical excision is both quick & effective.

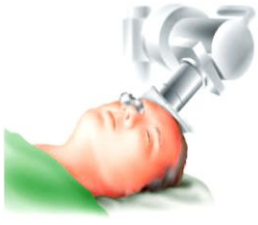
Disadvantages

Surgery may produce functional & cosmetic disabilities.

2- RADIOTHERAPY

Indications

1- Cancer of the larynx so as to preserve the voice.
2- Early cancer prostate & early cancer breast



Radiotherapy uses X-rays to destroy cancer cells.

Methods

1- Powerful **X-rays, gamma rays, electrons,** or **heavy particles** are directed to the tumor.

2- The radiation may be aimed at a tumor from outside the body **(Teletherapy),** or it may be delivered by placing radioactive needles at the cancerous site **(Brachytherapy)**.

Advantages

1- Curing the cancer **without sacrificing** the patient's ability to function.

2- Radiation can **destroy** microscopic extensions of cancerous tissue around that a tumor that a scalpel might miss.

3- Radiation is a **safer** option for older.

Disadvantages

1- Some tumors as squamous cell carcinoma are sensitive to Irradiation. But adenocarinoma is much less sensitive.

2- Radiation is commonly associated with burns of the skin or enteritis, which are difficult to treat.

3- Compared to surgery, radiotherapy is slower as it usually takes **5** to **8** weeks.

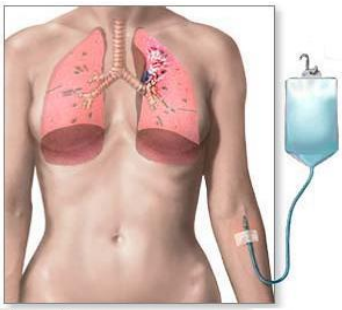
3- CHEMOTHERAPY

Indications

1- Main line of treatment of leukemia.
2- Metastases.

Methods

- Better results are obtained from **combination** chemotherapy rather than using one agent.



Chemotherapy, alone or combined

Advantages

- **The drugs travel the circulation** & can reach malignant cells any where in the body. Many malignancies including leukaemias, lymphomas and testicular cancer are now successfully treated by new conbinantion of chemotherapy.

Disadvantages

- **The available chemotherapeutic drugs** often kill many healthy cells & thus bring on serious effects, so causes anemia, leucopenia & thrombocytopenia.

4- HORMONE THERAPY

Examples are ⇩

- **Anti-estrogen** with cancer breast that is **+ve** for estrogen receptors.
- **Anti-androgen** with cancer prostate.
- **Thyroxin** to suppress TSH for patient with papillary cancer thyroid.

5- IMMUNOTHERAPY

Non-specific

The tuberculosis vaccine **BCG** stimulates the immune system as in transitional cell carcinoma of the urinary bladder.

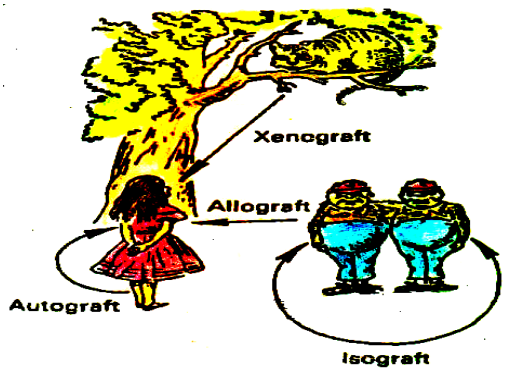
Specific

This method is still of limited use.

TRANSPLANTATION

BASIC IMMUNOLOGY

Types of graft



① **AUTOGRAFT** Same individual.

② **ALLOGRAFT** Same species.

③ **ISOGRAFT** Identical twins.

④ **XENOGRAFT** Different species

Major histocompatibility complex

- Glycoprotein molecules on the surface of all somatic cells act as " **self- markers** " which are responsible for triggering the immune reaction leading to allograft rejection.
- These molecules were originally detected on leukocytes and are therefore, named **H**uman **L**eukocyte **A**ntigens (**HLA**)
- These **HLA** are genetically controlled by loci on the short arm of **chromosome 6**. This area on chromosome 6 is termed **major histocompatibility complex**
- **The important antigens in transplantation are**

CLASS I HLA **A**, **B** & **C** antigens

CLASS II HLA **D** antigens

These antigens are capable of stimulating the recipient immune mechanism & triggering lymphocytes sensitization, starting rejection of the transplanted organs.

TRANSPLANTATION IMMUNOLOGY

Pre-transplantation assessment (**Histocompatibility tests**)

ABO BLOOD GROUP & CROSSMATCHING

- This is essential for all allograft.
- The aim is to prevent hyperacute rejection.

HLA CROSSMATCHING

- This is essential for renal & pancreatic transplantation
- Donor's lymphocytes are mixed with the recipient's serum.
- The aim is to minimize genetic disparity & later graft rejection.

The Donors

LIVING DONOR

- Only if paired organ or vascularised segmental part like the liver.

CADAVER DONOR

- With proved total brain death by

① **Deep coma** (No response to external stimuli).

② Bilateral dilated fixed **pupils** .

③ Absence of all **reflexes** .

④ Inability to maintain the **vital signs** for 3 minutes without artificial means.

⑤ Flat **EEG** in all channels.

Organ preservation

COOLING (0 – 4 °C)

to reduce tissue metabolism

PERFUSION OF A SPECIAL SOLUTION

to maintain a normal metabolic activity.

Immunosuppressive therapy

To prevent rejection

A- INDUCTION IMMUNE SUPPRESSION

Given prior to transplantation in order to avoid rejection.

① Large doses of **corticosteroids**.

② **Azathioprine**.

③ **Anti-thymocyte globulin**.

B- MAINTENANCE IMMUNE SUPPRESSION

① Small doses of **Steroids**.

② **Azathioprine**.

③ **Cyclosporine A**

C- ANTI-REJECTION TREATMENT

① Large doses of **Steroids**.

② **Monoclonal antibodies** again T-lymphocytes

Complications of immunosuppression

1- **Infection :** Bacterial, viral, or fungal in urinary tracts, surgical wounds or catheters .

2- **Nephrotoxicity** with Cyclosporine A.

3- **Bone marrow depression**

4- Complications of **corticosteroids** .

5- **Neoplasia :** Immunodeficiency may predispose to cancer development.

THE REJECTION PROCESS

Cellular mechanism

THESE STEPS CAN BE SUMMARIZED AS FOLLOWING

Allo-antigen (process by macrophage **)** → ↑ **T-helper** → ↑ **Interleukin 2** → ↑ **Cytoxic T lymphocytes** which attack the transplanted tissues directly & causing rejection

Humoral mechanism

The present of **complement-fixing cytotoxic antibodies**, prior totransplant-ation, produces hyperacute allograft rejection

Types of graft rejection

	Onset	Mediators	Treatment
Hyperacute	**Immediate**	Humoral	Graft removal
Acute	**1st** month	Cellular	High dose steroids
Chronic	**Several** months	Cellular or humoral	Retransplantation

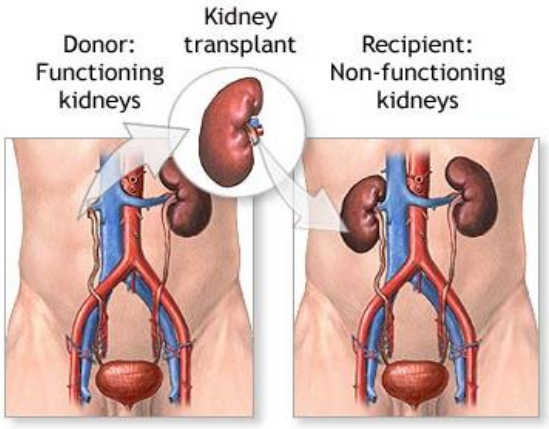
ORGANS TRANSPLANTATION

1. Renal Transplantation

INDICATIONS

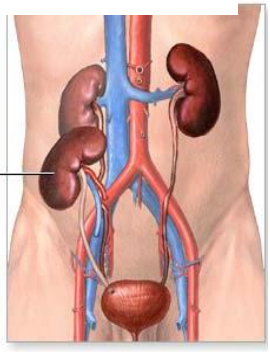
All cases of end-stage renal diseases which may be secondary to

① Glomerulonephritis
② Hypertension .
③ Diabetes
④ Chronic pyelonephritis .
⑤ Lupus nephritis.
⑥ Obstructive uropathy
⑦ Congenital nephrotic syndrome



TECHNICAL CONSIDERATIONS

- **The grafted kidney** is placed in an extra-peritoneal position in the iliac fossa.
- **The arterial anastomosis** is performed between the renal artery & external iliac artery
- **The venous anastomosis** is performed between the renal vein and external iliac vein
- **The ureter** of grafter kidney is anastomosed to patient's U.B



COMPLICATIONS

① Complications of immunosuppression (**see before**)

② Recurrence of original disease in the grafted kidney

③ **Technical complications** as vascular occlusion, urinary leakage، ureteric stricture or wound infection.

2. Hepatic Transplantation

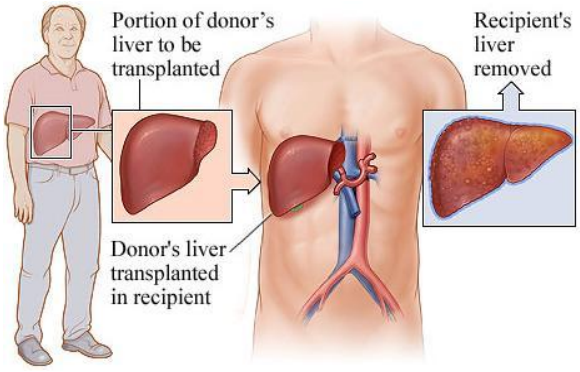
INDICATIONS

In children

① **Cirrhosis** due to biliary atresia, congenital hepatic fibrosis & cirrhosis.

② **Metabolic** as glycogen storage disease & alpha1 antitrypsin deficiency.

In adults

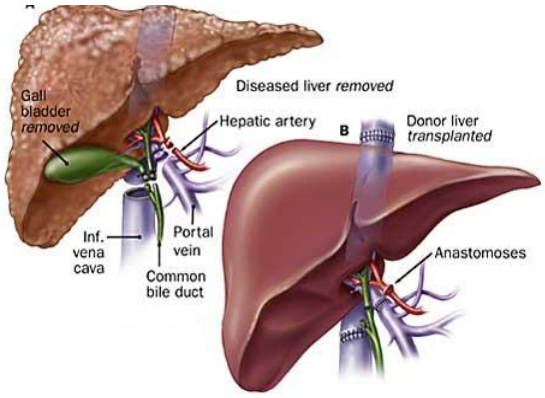


① **Cirrhosis :** 1^{ry} & 2^{ry} biliary cirrhosis, chronic active hepatitis, sclerosing cholangitis & alcoholic cirrhosis

② **Metabolic :** Hemochromatosis, Wilson's disease & Budd-chiari syndrome.

③ **Neoplastic : Fibrolamellar tumor** & **hepatocellular carcinoma** on top of liver cirrhosis.

TECHNICAL CONSIDERATIONS



- The liver of the recipient is removed then the **grafted liver** (cadaveric) is placed in same position.
- **The following anastomoses are performed** ⇩
  - Supra-hepatic & intra-hepatic I.V.C.
  - Portal vein of recipient to donor،
  - Hepatic artery of recipient to donor،
  - Common bile duct of recipient to donor .

N.B. **Nowadays**, **segmental liver transplantation** from living donors are being successfully performed.

This will overcome the problem of shortage of available liver donors

COMPLICATIONS

① Complications of immunosuppression (**see before**)

② Recurrence of original disease in the grafted liver

③ **Technical complications** as vascular occlusion, bile duct leakage, and bleeding,

Breast disorders

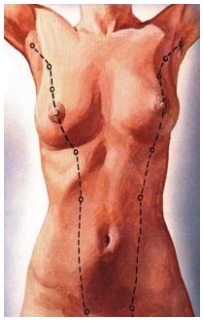
BREAST DISORDERS

EMBERIOLOGY

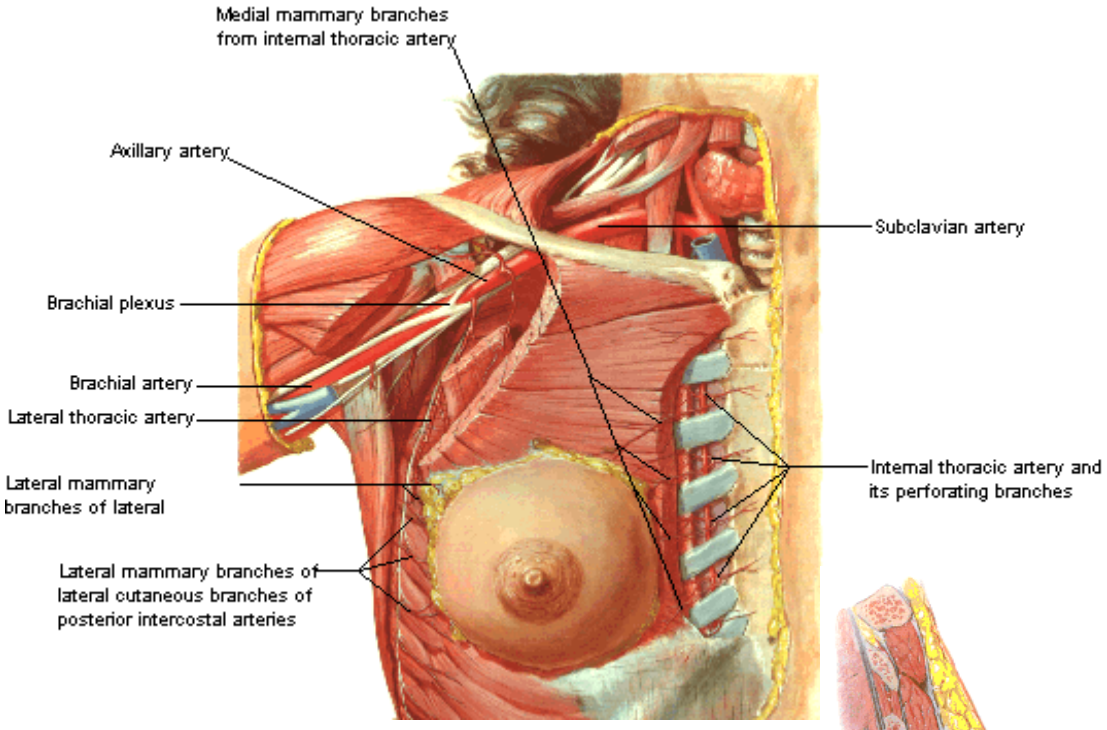
The Breast is a modified sweat gland which is developed from an **ectodermal ridge** " **mammary ridge** " which extends between the anterior pectoral fold & the groin.

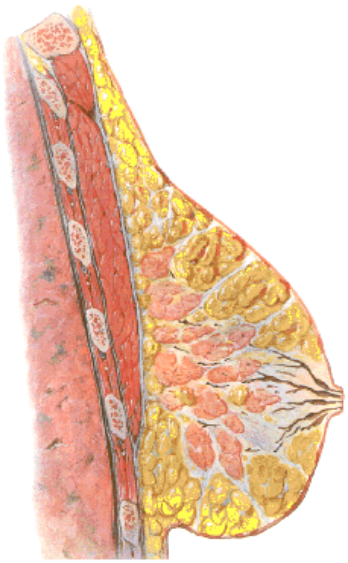
Normally it disappears all through except in the front of the chest where solid columns of epithelia pass deeply

→ **milk duct**



ANATOMY





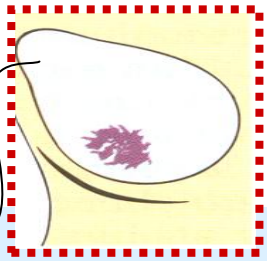
✷ Extent

- **Above :** at **2nd** rib.
- **Below** : at **6th** rib.
- **Medially :** at lateral border of sternum.
- **Laterally :** at anterior axillary line.

The actual extent of the breast is important for the surgeon who aims at removal of the whole breast for malignancy.

SO It actually extends:

- **Above** to the clavicle.
- **Below** to below the costal margin.
- **Medially** to the middle line.
- **Laterally** to the posterior axillary line.

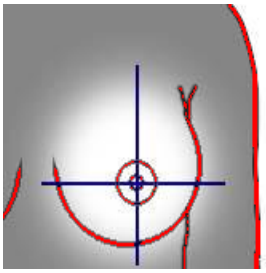


***Axillary tail of Spence* : (3rd rib)**

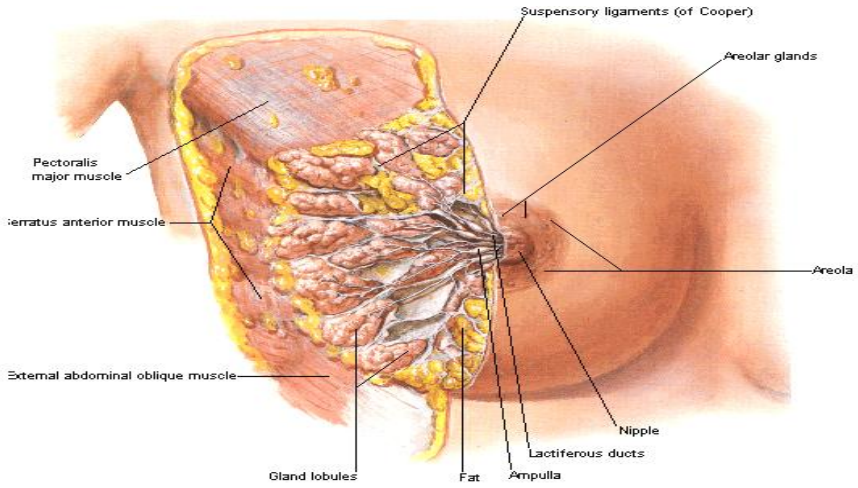
It is a prolongation from upper outer part of gland up to axilla. it is considered the only part which is deep to pectoral fascia through **foramen of Langer**. so it drains directly into posterior axillary L.Ns

✷ Areas (6 areas)

- Upper inner quadrant
- Upper outer quadrant
- Retro-areolar part
- Lower inner quadrant.
- Lower outer quadrant.
- Axillary tail.



✷ Architecture

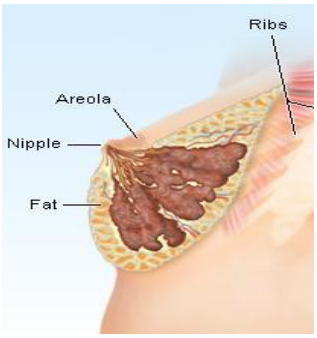


- **Breast consists of** (**15 - 20**) **lobes** which are arranged in radiating manner & each is drained by a lactiferous duct. the ducts converge at the nipple. A lobe is made up of (**20 - 40**) **lobules**, each of which consists of (**10 - 100**) **alveoli**

- **The supporting tissues :**
  - The ducts are attached to underlying pectoral fascia by band of fibrous tissue to the skin called ***(Cooper's ligament)***. This ligament can be involved in fibrotic lesions leading to **skin dimpling**
  - The ducts are surrounded by **contractile myoepithelial cells** which are stimulated by oxytocin & move milk towards the nipple.

- **Nipple : (4th intercostal space)**
  On its top **15 – 20 opening**, its normal direction is downward, forward & laterally

- **Areola :**
  Thick skin, pink in nulipara, blackens brown with pregnancy. contains sweat & sebaceous glands of **montogomory**.



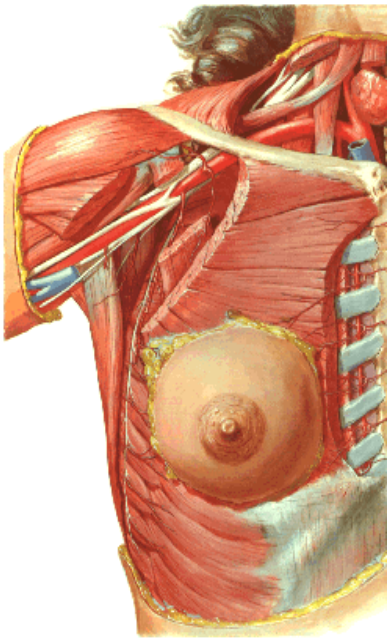
✷ **Muscle flour** It lies on **3 muscles**

1- PECTORALIS MAJOR MUSCLE

- Origin :

- Sterno-costal head :
Ant. surface of sternum, upper 6 costal cartilages.

- Clavicular head :
Medial 1/2 of front of clavicle.



- Insertion :
Lateral lip of biciptal groove.

- Nerve supply :
Medial & lateral pectoral nerve.

- Action :
Flexion, adduction & medial rotation

N.B : PECTORALIS MINOR MUSCLE

- Origin :
3rd ,4th ,5th ribs

- Insertion :
Coracoid process of scapula.

- Nerve supply :
Medial pectoral nerve.

- Action :
draws the scapula downwards & forwards.

2- SERRATUS ANTERIOR MUSCLE

- Origin :
8 digitations with upper 8 intercostal muscles

- Insertion :
Medial border of the scapula .

- Nerve Supply :
Nerve to serratus anterior

- Action :
Keeps the stability of the scapula with use of upper limb

3- EXTERNAL OBLIQUE MUSCLE

- For Anatomy : See chapter Hernia

✷ Arterial supply

- **Axillary artery** → lat. thoracic artery
- **Internal mammary artery** → 2,3,4 perforators.
- **Intercostal perforators**.

✷ Venous drainage

- **Axillary vein**
- **Internal mammary vein**
- **Intercostal veins**

(which drain into Azygos system which communicates with ***valveless*** *vertebral veins)*

***This explains* early vertebral metastasis** *with cancer breast*

✷ Lymphatic drainage

◊ Classic description

1. **Sub-areolar plexus of Sappey :**
   from nipple & areola then drains to deep plexus.

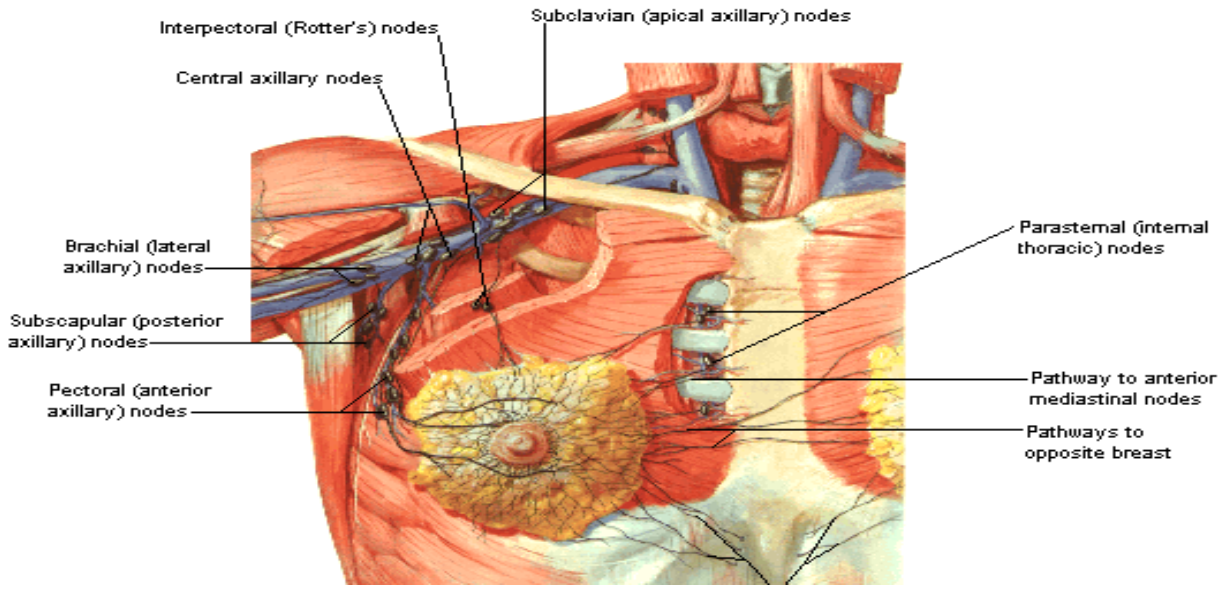
2. **Deep plexus** (over pectoralis)
   from sub-areolar plexus & deep part of the gland then drain to axillary L.Ns & Internal mammary through the pectoralis muscles.

◊ Modern description

Lymphatics drain through axillary L.Ns & Internal mammary L.Ns

I. Axillary L.Ns

These nodes receive about 75 % of breast lymph. There are on average 35 lymph nodes in the axilla that are arranged into :



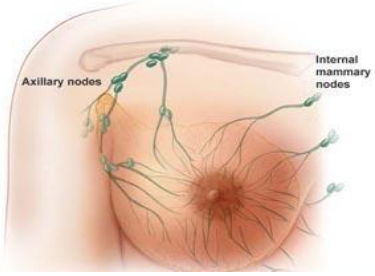
1. **THE ANTERIOR (PECTORAL) GROUP**
   - **SITE :** under cover the pectoralis major along the lateral thoracic vessels at the lower border of the pectoralis.
   - **DRAINS :** - chest wall.
     - whole breast except tail.
     - ant. abdominal wall above level of umbilicus.

2. **THE POSTERIOR (SUB-SCAPULAR) GROUP**
   - **SITE :** along the subscapular vessels.
   - **DRAINS :** - axillary tail.
     - post. abdominal wall above level of umbilicus.

3. **THE LATERAL (HUMERAL) GROUP**
   - **SITE :** along the axillary vein (upper part of humerus).
   - **DRAINS :** all the upper limb.

4. **THE MEDIAL (CENTRAL) GROUP**
   - **SITE :** central part of axilla (embedded in the axillary fat).
   - **DRAINS :** {1},{2},{3}

5. **THE APICAL GROUP**
   - **SITE :** extreme apex of axilla.
   - **DRAINS :** {1},{2},{3},{4}



II. Other associated L.Ns

1. Internal Mammary L.Ns

2. Inter-pectoral L.Ns of Rotter between 2 pectoral muscles

III. Further lymphatic spread

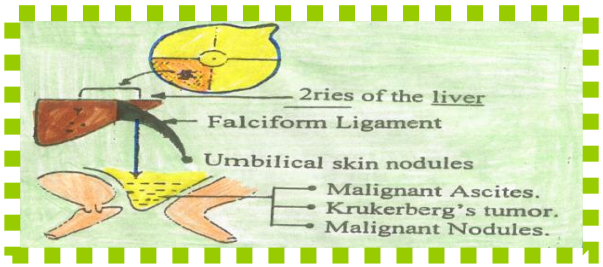
Connection of the lymphatics of the lower inner quadrant of the breast with the peritoneum. Lymphatics pierce rectus sheath → spread to liver leading to liver nodules. then through (falciform ligament)
→ umbilical nodules **(Josef sister's nodules)**

N.B.: Some malignant cells will lead to

Malignant ascites,

Krukenberg's tumor

& **Malignant nodules**
in the douglas pouch.

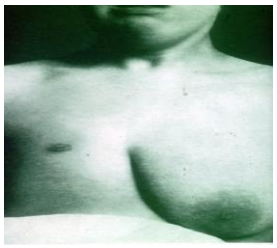


From prognostic point view axillary L.Ns are classified by
Pectoralis minor muscle into **3 levels**
- Level **I** → L.Ns **below** the muscle
- Level **II** → L.Ns **behind** the muscle
- Level **III** → L.Ns **above** the muscle

The prognostic importance with treatment of cancer breast with adjuvant therapy

I- CONGENITAL ANOMALIES

1. The Breast

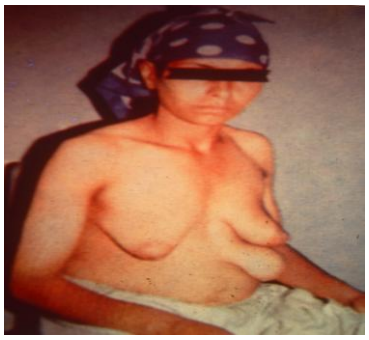


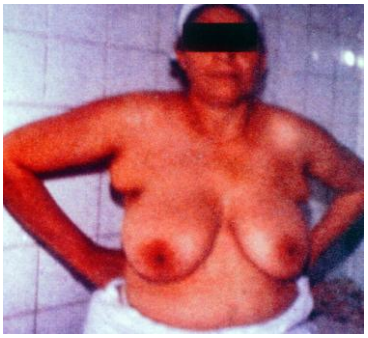
1. Amazia :

absence of breast (unilateral or bilateral)

2. Polymazia :

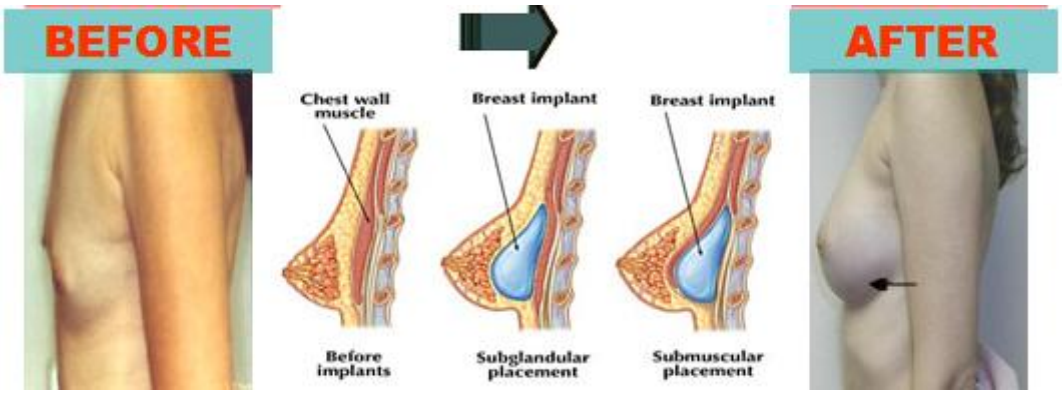
accessory breast along mammary ridge
they may function during lactation





3. Micromasia :

Small breast treated by **augmentation** mammoplasty



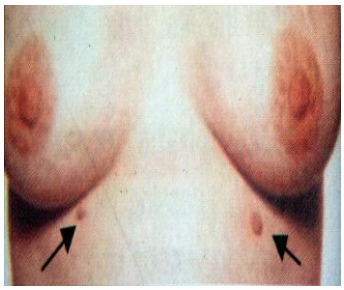
4. Diffuse hypertrophy :

Big breast treated by **reduction** mammoplasty

5. Infantile gynaecomastia :

Diffuse enlargement of the male breast which may be unilateral or bilateral. It is caused by the effect of circulating maternal sex hormones. The condition is usually **reversible within 6 months**, and therefore, requires **no** treatment

2. The Nipple



1. Athelia :

absence of nipple (very rare).

2. Polythelia :

accessory nipple along mammary ridge
an accessory nipple may be mistaken
for a mole or a wart.

3. Congenital retraction of the nipple :

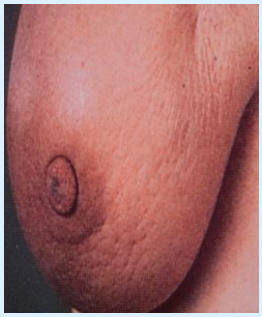
➢ It must be differentiated from **acquired** retraction

	Congenital retraction	Acquired retraction
• **History**	dating **since birth**.	recent.
• **Side**	**bi**lateral > 3/4 of cases	**un**ilateral.
• **Mass**	**no** breast mass	presence of breast mass
• **Sulcus**	absent	present

Don't Forget

[Causes of acquired nipple retraction]
due to **"excessive fibrosis"**

1. Mammary duct ectazia.
2. Chronic breast abscess.
3. Carcinoma of the breast.



II- TRAUMATIC DISEASES

(that may be clinically difficult to DD from carcinoma)

usually follow a **blunt** trauma

1. Traumatic fat necrosis

- ***Trauma*** → death of some fat cells → liberation of fatty acids which combine with calcium from local tissue fluid → **calcium soaps**
- ***Calcium soaps*** **:**
  - **cyst** containing " thick oily fluid "
  - **hard** mass If we do biopsy the cut section will show "characteristic chalky white appearance".
- ***Treatment*** **:** Excision & biopsy.

2. Breast hematoma

- ***Trauma*** → blood clot → organization → **fibrosis**
- ***Fibrosis*** → **hard** mass.
- ***Treatment*** **:** Excision & biopsy.

III- INFLAMMATORY DISEASES

A- Acute inflammatory mastitis

1- Acute lactational mastitis & Acute breast abscess

Mastitis from milk engorgement

- ★ **Incidence :** 1st month of 1st lactation.
- ★ **Aetiology :** due to obstruction of duct by dry inspissated milk or epithelial debris.
- ★ **Clinical picture :**
  - ➢ **Symptoms :**
    - ▪ **General : Toxic** symptoms [**F**ever, **H**eadache, **M**alaise & **A**norexia]

      N.B : Fever *is due to absorption of* ***Milk protein*** *(Ag x Ab)*
    - ▪ **Local :** dull aching pain.
  - ➢ **Signs :**
    - ▪ **Diffuse** tense & tender.
    - ▪ **No** physical signs of inflammation i.e. no hotness or redness.
    - ▪ **No** axillary L.Ns.
- ★ **Fate :**

  [If neglected] → acute bacterial mastitis or acute breast abscess.

Acute bacterial mastitis

- ★ **Incidence :** 1st month of 1st lactation i.e. fate from milk engorgement.

  or when baby is at 6 months i.e. development of incisors.
- ★ **Aetiology :**
  - ➢ **Predisposing factors :**
    - ▪ mastitis from milk engorgement.
    - ▪ abrasions of nipple e.g. cracks or fissures.
    - ▪ lack of breast hygiene.
  - ➢ **Organism :** Staphylococcus aureus (gram +ve).
  - ➢ **Route of entery :** organism from baby's mouth.

    much less common (blood born infection).
- ★ **Clinical picture :**
  - ➢ **Symptoms :**
    - ▪ **General : toxic** symptoms [**F**ever, **H**eadache, **M**alaise & **A**norexia]

      N.B : Fever *is due to absorption of* ***Organism*** *(Ag x Ab)*
    - ▪ **Local :** dull aching pain but gets worse.

➢ **Signs :**

- **Diffuse** tense & tender.
- **Physical signs** of inflammation, e.g. hotness or redness of skin.
- **Axillary L.Ns :** firm & tender (non specific).

★ **Fate :**

[If neglected] → acute breast abscess

Acute breast abscess

★ **Pathology :** milk engorgement + staph**.** infection → **pus**

★ **Clinical picture :**

➢ **Symptoms :**

- **General : toxic** symptoms [**F**ever, **H**eadache, **M**alaise & **A**norexia]

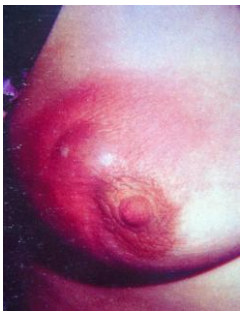
N.B. : Fever is hectic

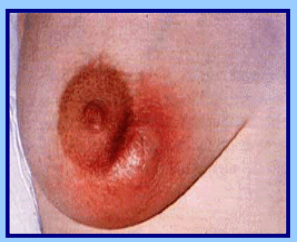
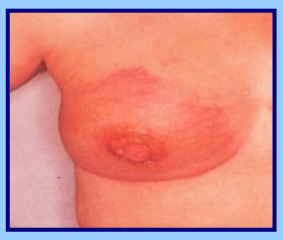
i.e. fluctuant & does not reach the basal line at the same day

- **Local : throbbing** pain which is more at night

➢ **Signs :**

- **localized** tense & tender.
- **physical signs** of inflammation.
  e.g. hotness or redness of skin.
- **axillary L.Ns** : firm & tender (non specific).
- **pitting oedema** of skin overlying the abscess.
- **fluctuation** is very late.



		Acute bacterial mastitis 	**Mastitis carcinomatosa** 
History	• Onset, course & duration	- **acute** onset & **rapidly** progressive course.	- **gradual** onset & **slowly** progressive course.
	• Fever	- **high** grade fever.	- **low** grade fever
Inspection	• Skin over	- **firey** red.	- **dusky** red.
Palpation	• Tenderness	- **markedly** tenderness.	- **mild** tenderness.
	• Axillary L.Ns	- firm & tender.	- hard & not tender
Treatment	• A.B	- cured	- **no** response

TREATMENT OF ACUTE LACTATIONAL MASTITIS & ACUTE BREAST ABSCESS

A- Prophylactic treatment

(1) Correct hygiene of breast during lactation.

(2) Paint the nipple with topical soothing creams.

(3) The breast should be evacuated completely with each lactation.

B- Active treatment

I. STAGE OF MILK ENGORGEMENT & ACUTE BACTERIAL MASTITIS

i.e. before suppuration [*no abscess*]

1. **Local heat** "hot application".
2. **Support of the breast** helps to lessen pain
3. **An antibiotic** against staphylococci e.g. **Flucloxacillin** or **Cephalosporin.**
4. **The Advisability of weaning:**

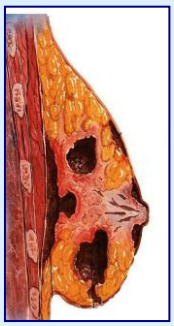
- If baby **> 9M** → **stop** feeding**,** the agent in common use is **"Parlodel"** 2.5 mg twice/day.
- If baby **< 9M** → **continue** feeding with healthy breast & regular evacuation of diseased one by using a **pump**

II. STAGE OF ACUTE ABSCESS FORMATION

i.e. after suppuration [*don't wait for fluctuation*]

- **Anaesthesia :** general anesthesia.
- **Incision :**

N.B : **Incision & drainage according to type of abscess :**



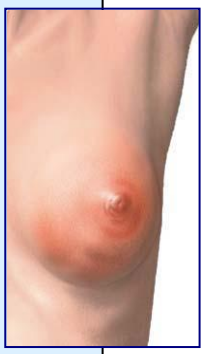
(1) Supra (pre) mammary abscess : *incision any where.*

(2) Intra-mammary abscess : it may *be*

a. Radial*: radiating from areolar*

b. Circum-areolar *: at margin of areola. 1st then radial incision is done so better cosmetic.*

(3) Retro (post) mammary abscess :
incision in sub-mammary fold.

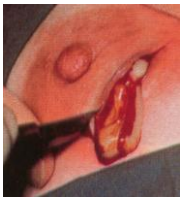


- **Technique :**

1- Surgeon's finger breaks all loculi to form single cavity

2- Pus evacuation for culture & sensitivity.

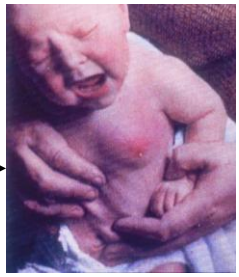
3- Drain is brought out through the most dependent part.



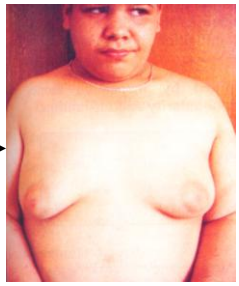
2- Non lactational mastitis

The commonest type of **non** Lactational mastitis is that which complicates mammary duct ectasia

3- Rare types of mastitis



1. **Infected haematoma**.
2. **Infected tumors**.
3. **Mastitis neonatorum** (**female & male**).
   - It is due to retention of mother hormones i.e. (maternal prolactine) stimulates lactation in infant.
   - **C/P :** swollen breasts on 3rd, 4th day with few drops of milk (***witch's milk)***
   - It subsides within 2-3 weeks.



4. **Mastitis of puberty (male only)**
   - The condition affects adolescent boys → pain + swelling of breast. which becomes indurated but (suppuration **never** occur).

B- Chronic inflammatory diseases

1- Mammary duct ectazia

[Plasma cell mastitis]

Definition

Dilatation of major ducts of the breast.

Aetiology

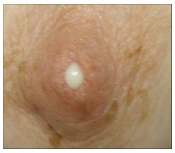
Unknown.

Pathology

Chronic inflammation of duct system leads to dilatation of major ducts which are
- Filled by : **Creamy secretions**. (atrophic epithelium + fatty material).
- Surrounded by : Plasma cells so (called **Plasma cell mastitis**).

Clinical Picture

- **Age :** around or after menopause.
- **Mass : hard** mass, may be associated with nipple retraction, peau d 'orange .. etc **So** similar to cancer breast.
- **Discharge : Creamy white** or may be **blood stained**.

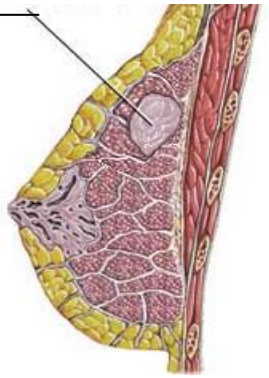


Treatment

Excision & biopsy (to exclude malignancy).

2- Chronic breast abscess

Non specific (**Chronic pyogenic breast abscess**)



★ **Definition :**

fate of improper treatment of acute abscess.

★ **Aetiology :**

prolonged use of antibiotics → killing of bacteria → sterile pus → **Antibioma**

★ **Pathology :**

- **cavity :** containing sterile pus
- **wall :** thick fibrous wall.

★ **Clinical picture :**

- **Mass : hard** mass, may be associated with nipple retraction, peau d 'orange .. etc
  So similar to cancer breast.
- **Discharge: no** discharge**.**

★ **D.D :**

	Chronic abscess	Cancer breast
• Toxaemia.	- **Low** grade fever.	- **Absent**
• Post-surface.	- **Rounded**.	- **Flat**.
• History of A.B	- **+ ve**	- **- ve**

★ **Treatment :**

Excision & biopsy (to exclude malignancy).

Specific (**T.B**)

★ **Definition :**

a rare disease with active pulmonary **T.B**

★ **Aetiology :**

Tubercle bacilli **(T.B)**

★ **Pathology :**

T.B. granuloma.

★ **Clinical picture :**

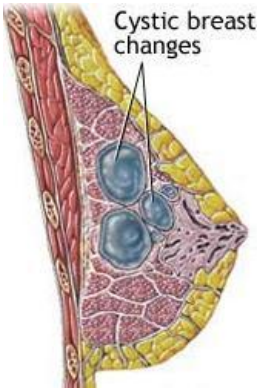
- **History** of (night sweat, night fever, loss of weight & loss of appetite).
- **Mass *:*** multiple nodules of the breast.
- **Axillary L.Ns :** enlarged & matted.

★ **Treatment :**

Anti T.B. drugs + **Excision** for resistant cases.

IV. FIBROCYSTIC DISEASE OF THE BREAST

FIBROADENOSIS



(Other names)

- **Mammary dysplasia.**
- **Mastopathy.**
- **Chronic interstitial mastitis** but misnomer as no evidence of inflammation
- **ANDI** [**A**berration of **N**ormal **D**evelopment & **I**nvolution]

Incidence

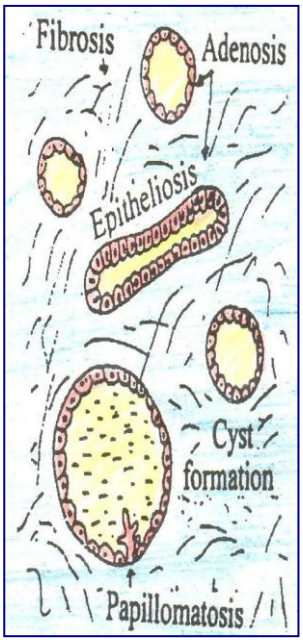
This is the most frequent disorder of the breast. the upper outer quadrant of the breast is the commonest site of affection.

Aetiology

[Unknown] but may be due to oversensitivity of oestrogenic receptors.
i.e. [**Relative hyperoestrogenaemia**]

Pathology

[**An image of pathological action of oestrogen on breast**]



★ **N/E picture :**
- **Site *:*** localized or diffuse.
- **Side *:*** unilateral or bilateral

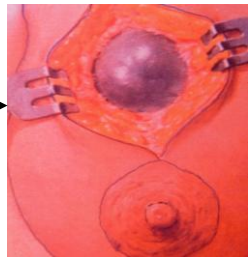
★ **Microscopic picture :** [**Panplasia**]
- **Adenosis :** ↑ number of acini.
- **Epitheliosis :**
  Hyperplasia of epithelial lining the ducts
  → Atypical hyperplasia → Pre-cancerous.

N.B: Duct papilloma
It is a localized form of epitheliosis

- **Fibrosis :** Fibrous tissue replaces the fat
  i.e. Sclerosing adenosis

N.B: Fibroadenoma
It is a localized form of adenosis & fibrosis

- **Cyst formation :**
  (A) *Microcyst* : degenerating cyst.
  (B) *Macrocyst :* retention cyst due to obstruction by :
  - **Epitheliosis** from inside.
  - **Fibrosis** from outside.



Sometimes **papillomatosis** are seen in the cyst from excess epithelial proliferation

Clinical picture

★ ***Age :***

after puberty or **before** menopause

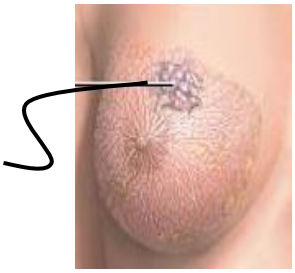
★ ***Symptoms :*** (May be **asymptomatic**)

- **Pain (mastalgia) :**
  dull ache. ↑ before, ↓ after menses.

  N.B.: This pain stops with pregnancy
- **Discharge :**
  clear or yellow but sometimes brown or green.
- **Mass :**
  painful & fixed to breast tissue.

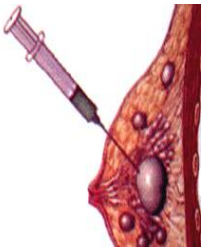
★ ***Signs :***

- **Tender** breast tissues
- **Discharge :** by patient herself.
- **Mass :** firm or fine nodules by **tips** of fingers



Investigations [The Aim is to exclude cancer]

- U/S & soft tissue mammography
- Aspiration & cytology.
- Biopsy & histopathology.



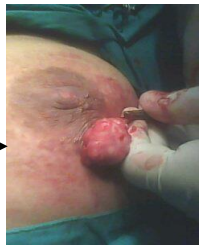
Treatment

(A) MEDICAL TREATMENT ***(The main ttt)***

- Reassurance of the patient
- Advice patient to stop caffeine e.g. stop coffee, tea & chocolate
- Sedatives & tranquilizers.
- Support the breast by tight braces to ↓ pain
- **Parlodel (anti-prolactin) :** 2.5 mg twice per/day.
- **Danazol (synthetic androgen) :** 100 - 200 mg twice per/day.
- **Tamoxifen (anti-estrogen) :** 10 mg once daily.

(B) SURGICAL TREATMENT

Excision & biopsy
indicated with localized mass.



(C) FOLLOW UP

with atypical hyperplasia, discovered by biopsy,
should be instructed to perform a **monthly self examination**

V. BREAST NEOPLASM

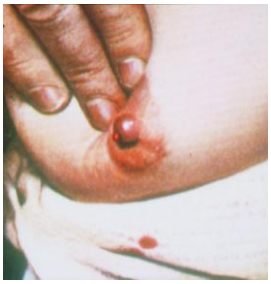
A. BENIGN

- ***Epithelial :*** **Duct papilloma.**
- ***Mixed :*** **(Epithelial** & **Fibrous Tissue) Fibroadenoma.**

B. MALIGNANT

A. BENIGN NEOPLASM

1- DUCT PAPILLOMA



Incidence

Common at **young** women.

Aetiology

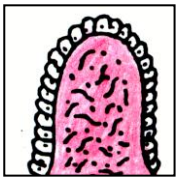
[**Benign tumor of epithelial cells**] It may be

- from the start i.e. de novo.

or on top of excessive localization of **epitheliosis** of fibroadenosis.

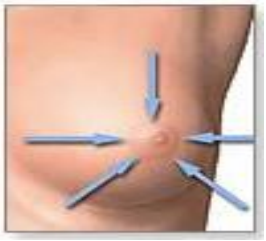
Pathology

- **N/E Picture :** usually single & arises from main lactiferous duct near the nipple
- **Microscopic picture :** core of very **vascular C.T** covered by hyperplastic epithelial layer.



Clinical picture

- **Age : 30 - 40** years.
- **Symptoms : - Bleeding** per nipple
  - **Retro-areolar** mass i.e. retention cyst
- **Signs :** - **Localize** the duct by palpation of each quadrant
  - **Retro-areolar** mass i.e. retention cyst



Complications

- Malignant transformation i.e. **duct carcinoma**.
- Profuse **bleeding** per nipple

Investigation

DUCTOGRAPHY

Retro-areolar **filling defect** in major duct.

Treatment

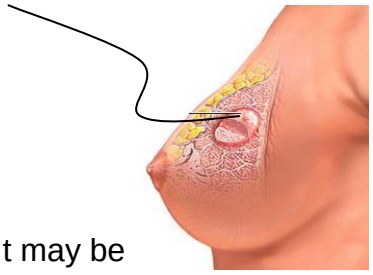
MICRODOCHECTOMY

Excision of the affected duct through circum-areolar incision

- If there is a lump, the excision is easy.
- If there is **no** lump, the duct is identified by inserting a blunt tipped needle

The excised specimen should be histologically examined

2- FIBROADENOMA



Incidence

The commonest breast mass.

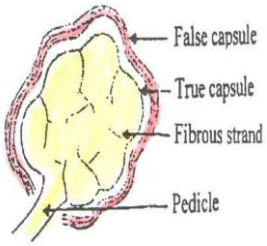
Aetiology

[Benign tumor of epithelial cells + fibrous tissue] It may be

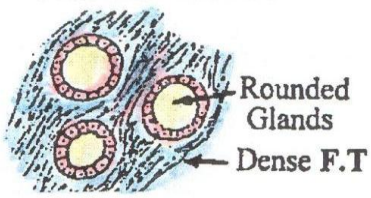
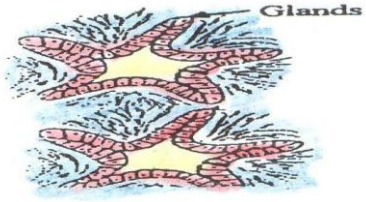
- from the start i.e. de novo.

or on top of excessive localization of **adenosis** & **fibrosis** of fibroadenosis.

Pathology



- **The tumor is (well capsulated)**
  - **True** capsule **:** showing fibrous bands dividing it into lobules
  - **False** capsule **:** formed by compressed breast tissue.
- **There are 2 types :**

	Hard fibroadenoma (Peri-canalicular)	**Soft fibroadenoma (Intra-canalicular)**
• **N/E Picture**	attached to its capsule by **one** pedicle. 	attached to its capsule by **multiple** pedicles. 
• **Microscopic Picture**	ducts are **surrounded** by fibrous tissue 	ducts are **compressed** by fibrous tissue 

Clinical Picture

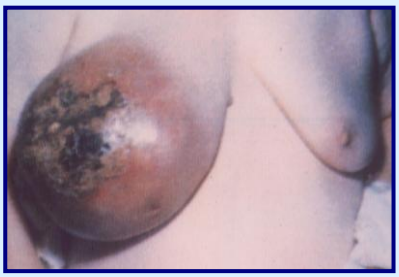
• **Age**	**20 - 30** years	**30 - 50** years
• **Symptoms**	• painless mass. • **slow** rate of growth i.e. malignancy is **never.**	• painless mass. • **rapid** rate of growth i.e. malignancy is **common**
• **Signs**	• **firm** & not tender. • well defined edge. • mobile **(breast mouse)** • **no** L.Ns enlargement	• **soft** & not tender.

Complication

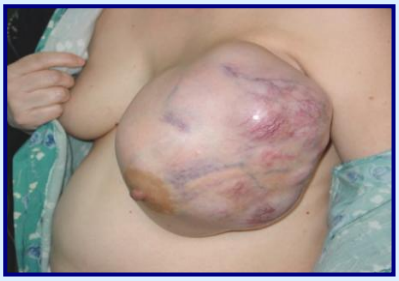
• **Malignancy**	• **Never**	• Liable to turn to **Sarcoma**

N.B: Cystasarcoma phylloides :

- **The name :**
  - The term cystasarcoma, however, is a misnomer as many are **not** cystic & it is **not** sarcoma.
  - It better termed "' **Phylloides tumor** "
  - It was so named by " **Brodie** " who was used the term **Phylloides** because the cut surface resembles a leaf or a fan



- **Pathology :**
  - It is highly cellular type of fibroadenoma that tends to grow rapidly



- **Examination :**
  - It is giant soft fibroadenoma.
  - Ulcerate through skin but not attached to it
  - **No** axillary L.Ns except if infected.
- **Treatment :**
  Wide local excision or Simple mastectomy

Investigations

Soft tissue mammography

Treatment

- **Hard** fibroadenoma **:** Excision & biopsy.
- **Soft** fibroadenoma **:**
  - If small : Excision & biopsy
  - If large : Simple mastectomy

B. CARCINOMA OF THE BREAST

1- Incidence

According to the data of the **N**ational **C**ancer **I**nstitute of Cairo University

About **35 %** of total malignancies of **Egyptian**  **females**.

2- Risk Factors

A - GENETIC FACTORS

- Accounts for **5 – 10 %** of all breast cancer.
- Presence of breast cancer in a mother or sister ↑ risk **3** times. while presence of cancer in both mother & sister ↑ risk **14** times.
- **2 Genes are associated**:
  1. **BRCA I ;** long arm of chromosome **17** associated with breast, ovarian & colon cancer
  2. **BRCA II ;** long arm of chromosome **13** associated with breast & ovarian cancer

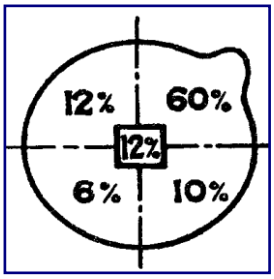
B - Endocrinal factors

- **Early** menarche **< 13** years.
- **Delayed** menopause **> 50** years.
- Female get **1st pregnant > 30** years.
- The relations to **oral contraceptive pills** is not known exactly.
- **Obesity** as adipose tissue converts steroid hormones to estradiol.
- Female with cancer to **one breast.**

C - Precancerous lesions

- Relations to **duct papilloma** ↑ risk **1.5 - 2** times.
- Relations to **atypical hyperplasia** of **fibroadenosis** ↑ risk **2 - 5** times.
- Relations to **l**obular **c**arcinoma **i**n **s**itu **(LCIS)** or **d**uct **c**arcinoma **i**n **s**itu **(DCIS)** ↑ risk **5 - 10** times.

3- Pathology



A - Site

- Upper outer (**60%**) **The commonest**
- Lower outer (**10%**)
- Upper inner (**12 %**)
- Retroarolar (**12%**)
- Lowe inner (**6%**)

The rarest & worst → spread to sub-diaphragmatic lymphatics

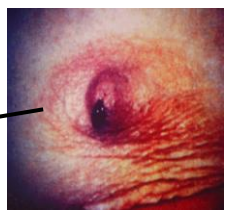
B – Pathological types

(I) Lobular carcinoma

- **Non invasive = In situ (LCIS)**
- **Invasive (7.5 %)** 25 % bilateral (**Mirror Image**)

II) Duct carcinoma

- **Non invasive = Insitu (DCIS)**
  - **Papillary duct carcinoma** → **bleeding** per nipple.
  - **Comedocarcinoma** → **Sebaceous like material** from cut surface of the tumor.



- **Invasive [Subtypes]** ↓ **See next page**

(III) Paget's disease of nipple ↓ **See later**

Invasive Duct Carcinoma

	1. Scirrhous Ca (75 %) Scirrhous = Hard	2. Encephaloid Ca (10 %) Encephaloid = Brain like
• N/E picture	• small, hard & irregular mass. • C.S: → Gritty, concave, pale & non capsulated	• large, soft & irregular mass. • C.S: → soft, convex bulging & non capsulated
• Microscopic picture	• fibrous tissue is more than malignant cells which is undifferentiated hence the name NOS (Not Otherwise Specified) • areas of Hge & necrosis.	• malignant cells more than fibrous tissue • lymphocytic infiltration • areas of Hge & necrosis.
• Prognosis	**good** if early diagnosed	**better than** scirrhous because of lymphocytic infiltration

	3. Mucinous Ca (3 %)	4. Inflammatory Ca (very rare) Mastitis carcinomatosa
• N/E picture	• large, soft gelly like. • very bulky	• large mass. • very rapidly growing tumor.
• Microscopic picture	• spheroidal cells distended with **mucoid** material • **Signet ring** like	**N.B :** • It occurs during pregnancy & lactation. **SO D.D.:** from acute mastitis
• Prognosis	the **best** prognosis.	the **worst** prognosis & consider **T4**

N.B

HORMONAL RECEPTORS

- About **60%** of breast cancers have receptor for **Estrogen** & termed **ER +ve** ,These tumors are respond to hormonal treatment.
- About **20%** of breast cancers exhibit **Herceptin** & termed **HER2/neu +ve**.
  , These tumors are respond to immunological treatment.
- **Recently :** Tumors are stained for **c~erb 52** (growth factor receptor)
  , These tumors are respond to monoclonal antibodies treatment.

III. Paget's disease of the nipple

Incidence

1%

Aetiology

Malignant erosion caused by duct carcinoma

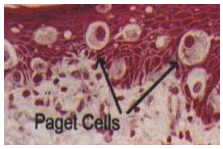
Pathology

- **N/E picture :** [**Malignant eczema**]

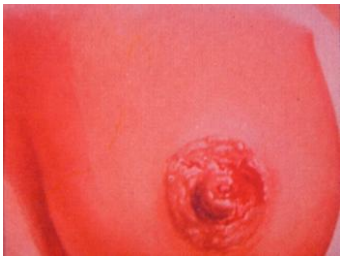
  unilateral with well defined margin.

- **Microscopic picture :**

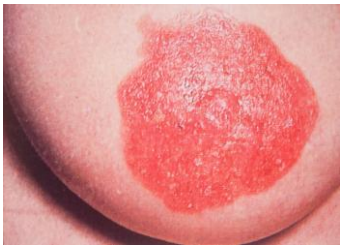
  1. Hyperplasia [**all epidermis**].
  2. **Paget's cells [deep epidermis**] clear vacuolated cells with small **dark** stained nuclei



  3. Round cell Infiltration [**dermis**].



Ulcerative Type



Eczematous Type

Clinical Picture

2 Types may be present

(1) Ulcerative Type.

(2) Eczematous Type.

D.D.

Paget's disease (**Malignant eczema**)	**Dermatitis** (**Ordinary eczema**)
• Usually **menopausal** female.	• Usually **lactating** female.
• **Uni**lateral.	• **Bi**lateral.
• Erosion.	• **No** erosion.
• **No** itching	• Itching
• Start in the **nipple**.	• Start in the **areola**.
• Breast lump may be felt.	• **No** lump.
• **Not** respond to eczema treatment.	• Respond to eczema treatment.

Staging

Paget's disease **alone =** (**stage I**)

Treatment

Radical mastectomy

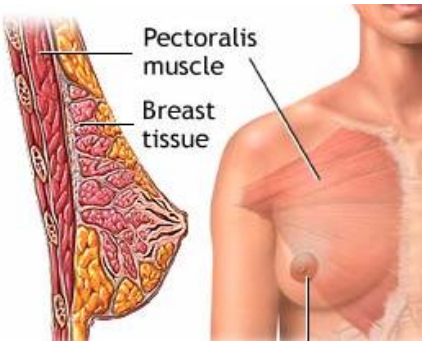
N.B.: Paget's disease is **radio-resistant.**

Prognosis

Paget's disease **alone = good** prognosis

4- Spread

1. **Direct :** [skin, underlying muscle & chest wall].
2. **Blood :** [**L**iver, **B**one, **L**ung & **B**rain].
3. **Lymphatic :**
   [by **Embolization & Permeation**].
   - ➢ through axillary L.Ns → internal mammary L.Ns → supra-clavicular L.Ns.



✯ DON'T FORGET

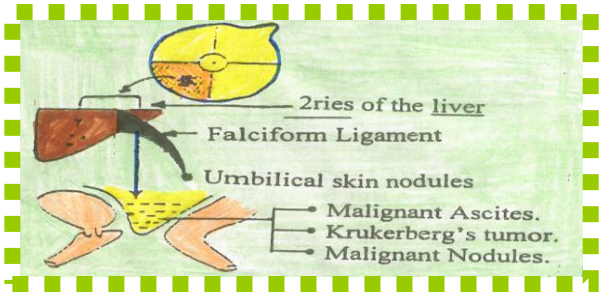
Connection of the lymphatics of the lower inner quadrant of the breast with the peritoneum. Lymphatics pierce rectus sheath → spread to liver leading to liver nodules. then through (Falciform ligament)
→ umbilical nodules **(Josef sister's nodules)**

N.B.: Some malignant cells will lead to

Malignant ascites,

Krukenberg's tumor

& **Malignant nodules**
in the douglas pouch.



5. Staging

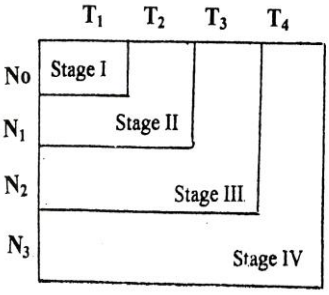
[A] T.N.M Staging

T = Tumor	N = Nodes	M = Metastasis
Tis = **Ca in situ** or Paget's alone	**N0** = **No** palpable L.Ns.	**M0** = **No**
T0 = **No** evidence of 1ry tumor.	**N1** = **Mobile** axillary L.Ns.	distant metastasis
T1 = **< 2** cm.	**N2** = **Fixed** axillary L.Ns.	**M1** =
T2 = **2- 5** cm	**N3** = Palpable **homolateral** supra-clavicular L.Ns.	Distant metastasis
T3 = **> 5** cm		
T4 = **Any size** with extension to chest wall		

[B] U.I.C.C Staging

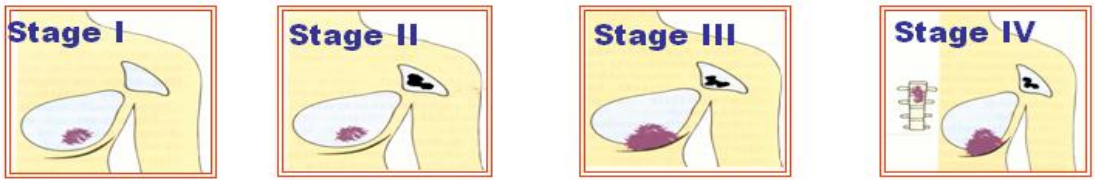
(Union International Cancer Center)

Stage U.I.C.C	Category
Stage I	**Early** breast cancer with **No** L.Ns
Stage II	**Early** breast cancer with **mobile** L.Ns
Stage III	**Advanced** breast cancer with **fixed** L.Ns
Stage IV	**Metastatic**



[C] Manchester Classification
(Clinical classification)

	Tumor	L.Ns	Metastasis
Stage I	• **Mobile** breast mass.	• **NO**	• **NO**
Stage II	• **Mobile** breast mass.	• **Mobile** (at same side).	• **NO**
Stage III	• **Skin** involvement. • **Pectoralis muscles** are involved. • **No** Chest wall involvement.	• **Fixed** (at same side).	• **NO**
Stage IV	• **Skin** involved e.g. cancer en curirasse • **Pectoralis muscles** are involved. • **Chest wall** is involved	• Involved at **opposite** side of axilla.	• **Metastasis**



Stage I & II = Operable & Stage III & IV = Inoperable

6- Clinical picture

★ **Age :**

commonly at **40 - 60** years + **risk factors** (**discuss**).

★ **Symptoms :**

[A] General symptoms

(may be the 1st presentation) i.e. **occult** carcinoma.

- **Lung:** chest pain, cough, dyspnea & haemoptsis.
- **Bone** : mass in skull, backache & pathological fracture.
- **Liver:** pain at Rt. hypochondrium & Jaundice.
- **Brain :** extremely rare.

[B] Local symptoms

- **Hard,** painless mass, **discovered accidentally.**
- **Rapid rate** of growth.
- **Discharge : Blood** If duct carcinoma.

➢ **Signs :**

[A] General signs

- To detect Metastasis (Liver, Bone, PR, PV & ... etc).

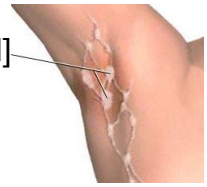
[B] Local signs

(1) Mass :

- **Hard** not tender mass.
- **Circumscribed** edge (hard mass inside soft breast).
- **Flat** under surface (local spread Ant. > Post.).
- **Fixed** to skin & +/- chest wall.

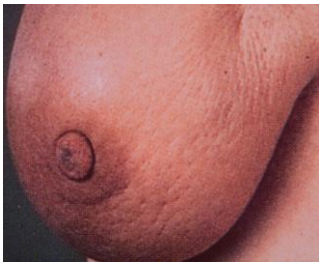
(2) L.Ns :

[Hard, enlarged, 1st mobile later on fixed]



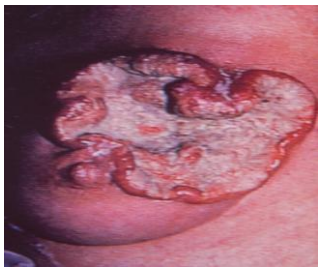
(3) Breast :

SKIN MANIFESTATIONS



1. Dimpling & puckering :

- due to contracture of **Cooper's ligaments**.

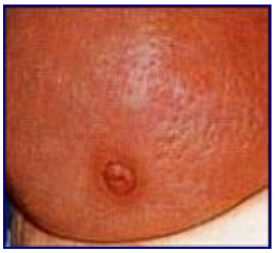


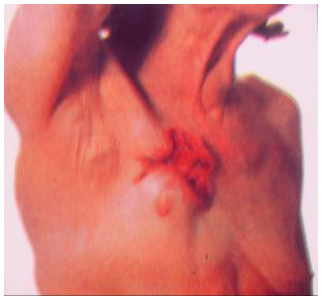
2. Nipple retraction & deviation :

- due to excessive fibrosis [**not pathognomonic**]

3. Peau d'orange : [**Pitting oedema**]

- due to obliteration & compression of lymphatic by excessive fibrosis [**not pathognomonic**]



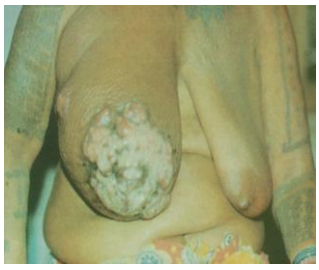


4. Cancerous skin nodules :

- due to lymphatic spread. It may be near or far from tumor e.g. around umbilicus.

5. Cancer en curirasse : [Advanced stage]

- It means hard, thick skin, metallic brown & stretched as **[War-shield]**



6. Ulceration & fungation :

- Raised everted edge with necrotic floor.

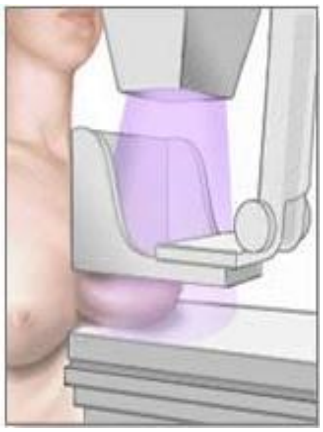
7. Paget's disease of nipple (see before)

8. Mastitis carcinomatosa (see before)

9. Dilated veins over the skin of the breast.

7- Differential diagnosis

1. **D.D. from nipple retraction**
   - **Carcinoma.**
   - **Mammary duct ectazia.** [history of creamy white discharge]
   - **Chronic breast abscess** [history of acute abscess & A.B intake]
2. **DD. from bloody discharge**
   - **Duct carcinoma**
   - **Duct papilloma**
3. **D.D from hard mass**
   - **Carcinoma.**
   - **Mammary duct ectazia.**
     [history of creamy white discharge]
   - **Chronic breast abscess**
     [history of acute abscess & A.B intake]
   - **Traumatic disorders**
     [history of trauma]

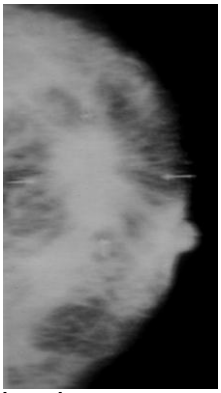


8- Investigations

A. Soft tissue mammography

- Cancer appears as a **dense** opacity.

N.B. : **Mammography is of less value** with **young** female because of similarity of lesion to dense breast



- **Indications :**
  1. Screening for **high** risk group.
  2. Search for **occult** cancer in female with metastatic disease.
  3. Evaluate **non** palpable breast lump.
  4. Evaluate **opposite** breast with cancer in the other breast.
- **Mammagraphic findings suggestive of malignancy :**
  1. Nipple **retraction**.
  2. Irregular outlines from irregular **spicules** penetrate surrounding breast.
  3. Increase **vascularity** of the breast.
  4. Microcalcification

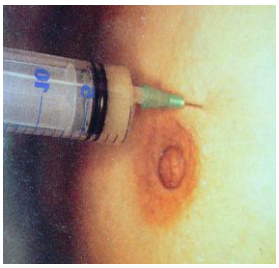
B. Ultrasonography

Both U/S & Mammography are complementary to each other.

- It can differentiate solid from cystic mass.
- It is useful in **young** women .

C. Diagnostic procedures (Biopsy)

- **Excision biopsy :** (The most reliable) but under general anaesthesia.
- **Frozen section biopsy :** diagnosed within 20 min while patient is under anesthesia (**if +ve** → Radical mastectomy).
- **Tru-cut biopsy :** under local anesthesia by a special needle which cuts a core of tumor tissue.
  Its disadvantages are : **1.** take a false tissue.
  2. may disseminate malignancy.
- **Fine Needle Aspiration Cytology (FNAC) :**



  1. ***Advantages*** *:* 90% accurate, very simple & inexpensive.
  2. ***Disadvantages*** *:* a skilled cytologist is needed.

D. MRI of the breast

- It is a gold standard for women with synthetic implants
- Also used for post-operative scar to D.D between fibrosis from local recurrence

E. Detection of distant metastasis

- **Lung** → plain x-ray.
- **Brain** → CT scan & MRI.
- **Liver** → U/S & liver function tests.
- **Bone** → bone scan.

F. Detection of tumor markers

- ***CA*** **15-3 :** Cancer Antigen. (prognostic rather than diagnostic)

9- Early detection

This aims at the detection of breast cancer very early in the asymptomatic females

A. Breast Self Examination (BSE)

- All woman over age 20 should be advised to examine their breasts monthly.
- The physician instructs the women as how to conduct a systematic inspection & palpation.
- The woman suspects the presences of a lump, skin dimpling, or nipple retraction .

B. Screening programs

In some Western countries high risk women are subjected to regular clinical examination & mammography. The frequency of examination is every one, two, or three years, depending on the program

10- Treatment

TRIPLE ASSESSMENT

1. **Clinical examination**
2. **Mammography & Ultrasound**
3. **FNAC**

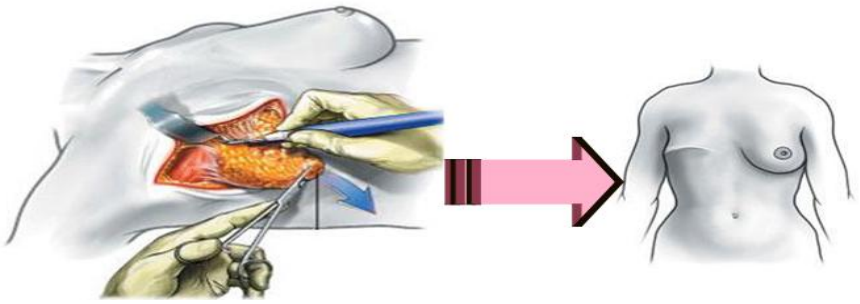
A. Operable (Early) less than T2, N1, M0
or Stage I & II as U.I.C.C

Different surgical options + Adjuvant systemic therapy if +ve Axillary L.Ns

- **Stage I : Modified radical mastectomy of Patey**
  + Follow up

▪ **Follow up :**

- **Aim to detect :** 1. Local recurrence or metastasis.
  2. Any post-operative complications.
- **Time** → after ttt then every 3 months at 1st 2 years
  then every 4 months for the next 3 years.
  then yearly



- **Stage II : Modified radical mastectomy of Patey**
  + Adjuvant systemic therapy

▪ **Radiotherapy :** To [↓ local recurrence]

To 1. Mediastinal region for internal mammary L.Ns.
2. Supraclavicular region for supraclavicular L.Ns.

▪ **Chemotherapy :** To [↓ late blood born metastasis]

By 1. **CMF** : **C**yclophosphamid, **M**ethotrexate & 5 **F**lurouracil
2. Adriamycin.

Indicated *with* **ER -ve** female

▪ **Hormonal :** to [↓ growth of tumor]

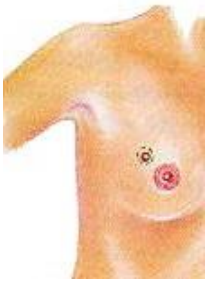
By Tamoxifen or Anastrazole

Indicated *with* **ER +ve** female.

Idea about

SURGICAL OPERATIONS

1. Conservative breast surgery



W.L.E

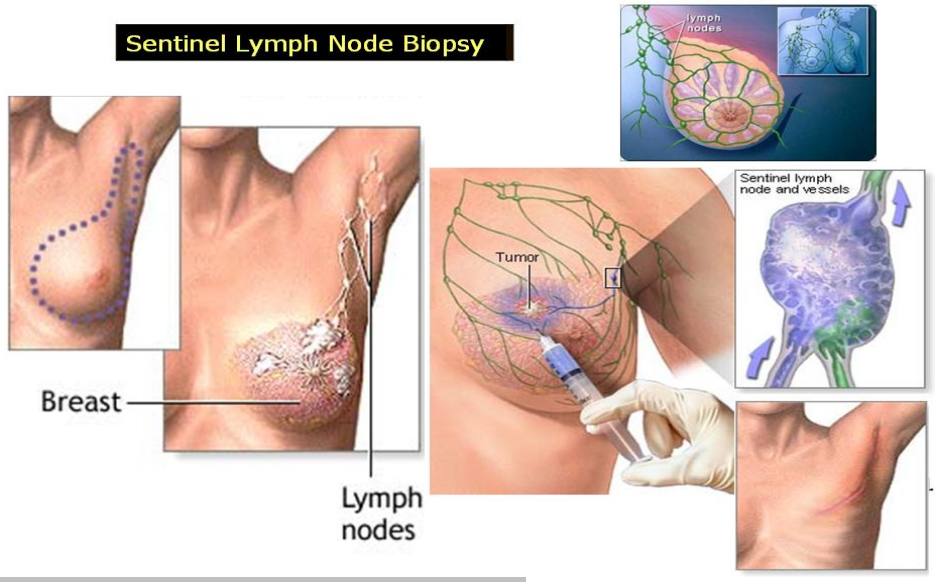
W.L.E = **W**ide **L**ocal **E**xcision with 2 cm safety margin
Then **Sentinel** lymph node biopsy

- Indications : ① Small masses < 4 cm
  ② Big breast
  ③ Young female
  ④ Peripheral lesions

- Contraindications : the reverse of indications +
  ① Pregnancy
  ② Collagen vascular disease (↓ tolerance to radiotherapy)

SENTINEL LYMPH NODE BIOPSY

The sentinel lymph node is localized per-operative by injection of a **blue** dye near the tumor. The dye will pass to the 1ry node draining the tumor area, which can be detected by **gamma** camera. then biopsy & histologically examined.



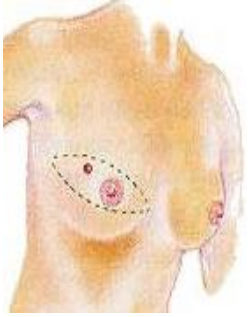
2. Modified radical mastectomy of (Patey)

(Most widely accepted)

Same as Halsted but

① we preserve pectoralis major muscle.

② Pectoralis minor either removed or cut at its insertion, or retracted to expose the axilla.



Radical Mastectomy

3. Radical mastectomy of (Halsted)

- **Removal of :**
  1. Elliptical part of skin with nipple & areola
  2. Whole breast tumor
  3. 2 Pectoralis muscles.
  4. All axillary L.Ns & fat medial to axillary vein

➢ **Preservation of :**

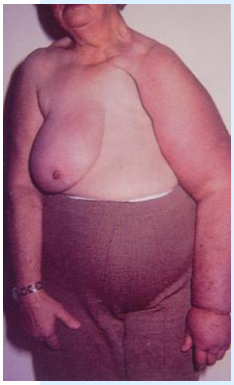
1- Axillary vessels
2- Cephalic vein
3- Nerve to serratus anterior
4- Nerve to latissmus dorsi.

4. Extended radical mastectomy

(Not done nowadays)

Same as Halsted + removal of internal mammary L.Ns., through median sternotomy.

N.B.: 1. Post-operative complications:



[I] HAEMATOMA OR WOUND INFECTION

[II] OEDEMA OF UPPER LIMB

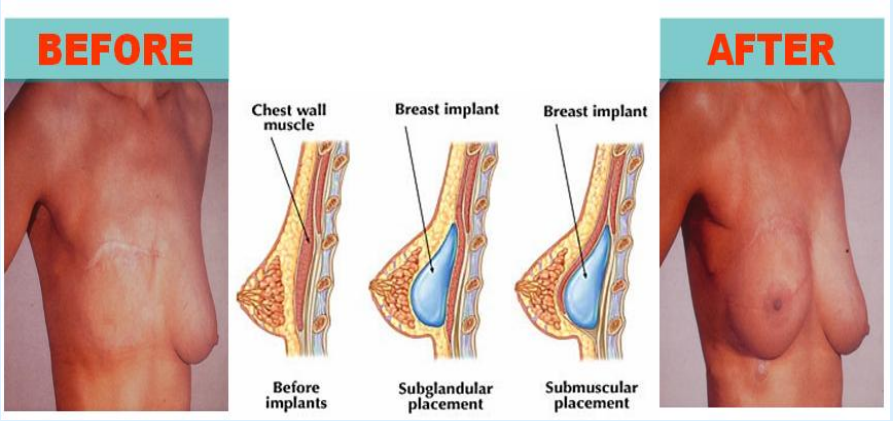
- ***Early pitting odema:*** (within **few days**) due to removal of excess lymphatics,
- ***Late non pitting odema*** (within **few months**) due to 1. Recurrence of axillary L.Ns.
  2. Arm infection.
  3. Axillary radiotherapy.

[III] BRIDLE SCAR

limitation of abduction.

N.B.: 2. Breast reconstruction :

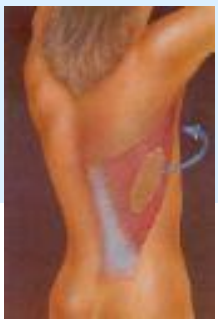
by SILICONE PROSTHESIS



or MYOCUTANEOUS FLAP

as Rectus abdominis

or Latissimus dorsi flap.



B. Inoperable (Advanced) more than T2, N1, M0

or Stage III & (Metastatic) IV as U.I.C.C

➢ **Stage III :**

[1] LOCAL TREATMENT (The main)

- **Radiotherapy :**
  - To 1. Mediastinum
    2. Supraclavicular region
    3. **Axilla**
- **Surgical indication**
  - Through palliative simple mastectomy

[2] SYSTEMIC TREATMENT

- **Chemotherapy** (**CMF** & **Adriamycin**)
  - Indicated especially :
    1. **ER -ve** female
    2. failure of hormonal treatment.
- **Endocrinal treatment :**

60 % cancer breast cases have receptors for estrogen so termed (**ER +ve**) which become :
- **More** active in presence of this hormone
- **Less** active in absence of this hormone

Temporary response for anti-oestrogen after 24 - 30 months occur especially with post-menopausal female or **ER +ve**.

➢ **Endocrinal treatment as :**

- **Tamoxifen (Nolvadex) : 1st line of ttt.**
- **Anastrazole (Aramidex) : 2nd line of ttt** if relapsed after Tamoxifen

➢ **Stage IV :**

[1] LOCAL TREATMENT

- **Radiotherapy** for any malignant deposits
- **Surgical indication**
  - **Excision** of skin nodules.
  - **Internal fixation** for pathological fracture.

[2] SYSTEMIC TREATMENT (The main)

- **Chemotherapy** (**CMF** & **Adriamycin**)
- **Endocrinal treatment :** (**Tamoxifen**, **Anastrazole…. etc.**)

As above

[3] TREATMENT OF METASTASIS

- **L**iver metastasis **: C**hemotherapy.
- **B**rain metastasis **: R**adiotherapy
  + Corticosteroids (↓ Intra-cranial tension)
- **L**ung metastasis **: C**hemotherapy.
  (pleural effusion) chest tube + cytotoxic bleomycine through it. i.e. **pleurodesis**.
- **B**one metastasis **: R**adiotherapy
  + Internal fixation if pathological fracture

11. Prognosis

(The prognostic index is less or equal 2.4 is excellent & has survival rate 95%)

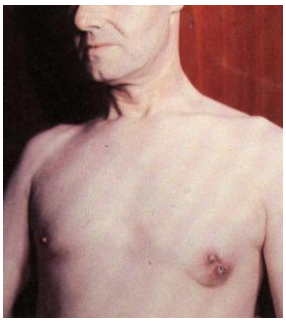
1. **Type** of tumor : Paget's & cancer situ are **better than** Mastitis carcinomatosa.
2. **Stages** of tumor : Stage **I** is **better than** Stage **II**, **III** or I**V**.
3. **Sites** of tumor : Lateral side is **better than** Medial side.
4. **Age** of patient : Old age is **better than** Young [because of sex hormones]
5. **Sex** of patient : Cancer female is **better than** Cancer male.
6. **Hormone receptors :** ER **+ve** are **better than** ER **-ve**.
7. **Size, mobility & number of lymph nodes :** involved (pathology)
   - Patients with **– ve L.N** → 10 years survival = **65%**
   - Patients with **less than 4 +ve nodes** → 10 years survival = **38%**
   - Patients with **more than 4 +ve nodes** → 10 years survival = **13%**

DD OF BREAST PAIN

1. **From Breast**
   - ☆ Breast Fibroadenosis.(أهبهم) or Mastodynia.
   - ☆ Bacterial mastitis or acute breast abscess.
   - ☆ Carcinoma is painless ***except***
     - Advanced
     - Infected
     - Mastitis Carcinomatosa
2. **Referred pain**
   - ☆ Angina Pectoris.
   - ☆ Intercostal neuralgia.
   - ☆ Diseases of bone, muscle and pleura

DISEASES OF MALE BREAST

Carcinoma of male breast



Incidence Male **:** Female = 1 **:** 100 (**1%** of all cancer breast)

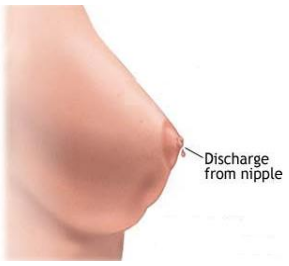
Staging

Treatment **Similar to cancer female breast** but castration is the main hormonal treatment

Prognosis **Worse than** cancer female due to early spread to chest wall (**no** breast fat)

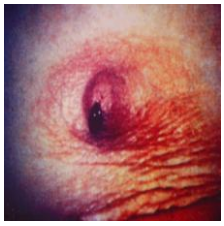
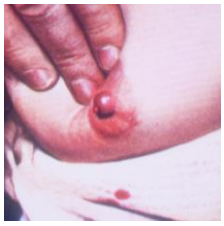
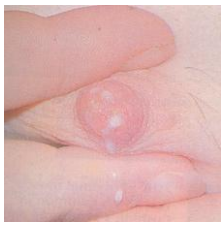
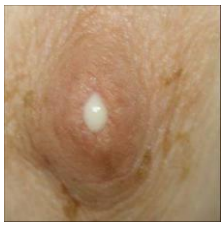
VI. NIPPLE DISCHARGE

Aetiology



- **Physiological**
  1. **Milky** discharge **:** during lactation
  2. **Serous** discharge **:** during pregnancy

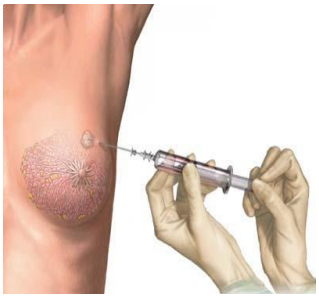
- **Pathological**
  1. **Creamy white** or may be **blood stained** discharge **:** Duct ectazia
  2. **Clear** or **yellow** but sometimes **brown** or **green** discharge : Fibroadenosis
  3. **Bloody** discharge **:** - Duct papilloma
     - Duct carcinoma
  4. **Milky** discharge **:** - Hyper-prolactinemia
     - Contraceptive pills

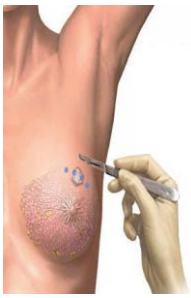
Diagnosis

- **History**
- **General Examination**
- **Local Examination**
  - especially for 1. Nature & side of discharge.
    2. Associated mass
    3. Age of patient
    4. Use of contraceptive pills
    5. Use of drugs as prolactin

Investigations



- **Soft tissue mammography & U/S**
- **Ductography : Lipidol** injection may show filling defect
- **Biopsy** or **Aspiration cytology** for mass
- **Serum prolactin level**
- **Tests for occult blood in discharge** through **[Benzedine test]**

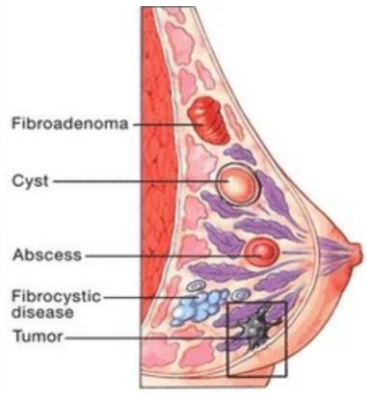


Treatment

- **If mass is associated**
  - Excision & biopsy
- **If No mass is associated**
  - Localized ducts : **Microdochectomy**
  - Many ducts (rare) : **Cone excision** of major ducts.

VII. BREAST MASSES

(1) Breast cysts



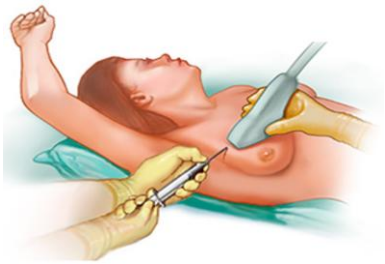
Aetiology

(A) Stroma [inter-acinar cysts]

- ***Traumatic :*** Blood cyst.
- ***Inflammatory :*** Cold abscess (T.B) or acute abscess.
- ***Neoplastic :*** Degeneration carcinoma.
- ***Parasitic :*** Hydatid cyst.
- ***Miscellaneous :*** Skin cyst e.g. sebaceous cyst, lymphatic cyst …..etc.

(B) Duct [Acinar cysts]

- ***Fibrocystic disease :*** e.g. Cyst i.e. macrocyst
- ***Retention cyst :*** e.g. Duct papilloma

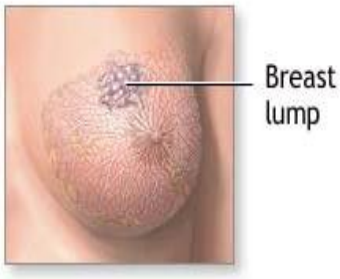


Treatment

(A) Aspiration + Cytology

(B) Excision If : 1. Rapid refilling after aspiration
2. Residual mass after aspiration
3. **Bloody** aspirate

(2) Solid swellings



[A] Hard masses :

1. Traumatic disease.
2. Chronic breast abscess.
3. Mammary duct ectazia.
4. Cancer breast (Scirrhous)

[B] Soft masses :

1. Soft fibroadenoma.
2. Cystasarcoma phylloids.
3. Cancer breast (Encephaloid or Mucinous)

[C] Firm mass :

1. Fibroadenosis.
2. Hard fibroadenoma.

N.B.: Causes of huge breast

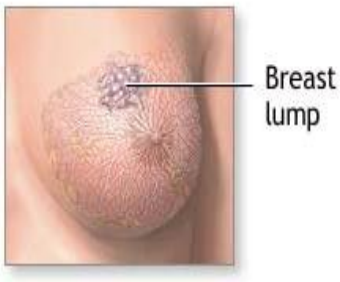
1. Diffuse hypertrophy of breast.
2. Soft fibroadenoma.
3. Cystasarcoma phylloids.
4. Sarcoma.

Q : DISCUSS DD OF BREAST LUMP ?

ANSWER :

LUMP (MASS) MAY BE CYSTIC OR SOLID (FIRM, SOFT & HARD)

(1) Breast cysts



(A) Stroma [inter-acinar cysts]

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- ***Neoplastic :*** Degeneration carcinoma.
- ***Parasitic :*** Hydatid cyst.
- ***Miscellaneous :*** Skin cyst e.g. sebaceous cyst, lymphatic cyst …..etc.

(B) Duct [Acinar cysts]

- ***Fibrocystic disease :***

e.g. Cyst i.e. **(Fibroadenosis)**

- **Pathology :**
  - **N/E** : Localized or diffuse, unilateral or bilateral & common at upper outer quadrant of breast.
  - **M/P** : Adenosis, Epitheliosis, fibrosis & **cyst** formation which may be microcyst or macrocyst
- **C/P** : - **Age** : After puberty or before menopause.
  - **Symptoms** : Pain related to menses with **Clear** or **yellow** but sometimes **brown** or **green** discharge
  - **Signs** : Tender cystic swelling.

- ***Retention cyst :***

e.g. **Duct papilloma**

- **Pathology** : Core of vascular C.T. covered by hyperplastic epithelium
- **C/P :** 30 - 40 years + **bleeding** per nipple with retro-areolar mass.
- **Complications**: Duct carcinoma

(2) Solid swellings

[A] Hard masses :

(1) Traumatic disorders

- **Traumatic fat necrosis** : release of fatty acids which bind to Ca → Ca soap i.e. **Hard** mass
- **Breast hematoma :** blood clot & Fibrosis i.e. **Hard** mass

(2) Chronic breast abscess

- **Aetiology** : Follow improper treatment of acute abscess by antibiotics so it is called Antibioma
- **C/P** : It represents by **hard** mass with nipple retraction, peau d'orange it similar to carcinoma by characterized by history of Antibioma

(3) Mammary duct ectazia

- **Aetiology** : Unknown may be due to excessive fibrosis.
- **C/P** : It represents by **hard** mass with nipple retraction, peau d'orange it similar to carcinoma by characterized by **Creamy white** or may be **blood stained** discharge

(4) Cancer breast (Scirrhous)

- **Commonly** at 40 - 60 years with one or more risk factors.
- **C/P** : - **Symptoms** : **Hard** mass & discovered accidentally.
  - **Signs** : metastasis + L.Ns + Skin manifestations as (Dimpling, puckering, cancerous nodules, Paget's etc.).

[B] Firm mass :

(1) Fibroadenosis

As mentioned about cyst of Blood good but it is firm mass felt by Tips of fingers.

(2) Hard fibroadenoma

- **Pathology :** Lobulated mass with one pedicle.
- **C/P :** - **Age** : 20 - 30 years.
  - **Symptoms** : Firm, painless & slowly growing tumor.
  - **Signs** : Firm (breast mouse) with No L.Ns.

[C] Soft masses :

(1) Soft fibroadenoma

- **Pathology** : Lobulated mass with multiple pedicle.
- **C/P** : - **Age** : 30 - 50 years.
  - **Symptoms:** Soft, painless & rapidly growing tumor.
  - **Signs** : Soft, not tender, No L.Ns & if enlarged it must be D.D from **(Cysta-sarcoma phylloides)**

(2) Cancer breast (Encephaloid)

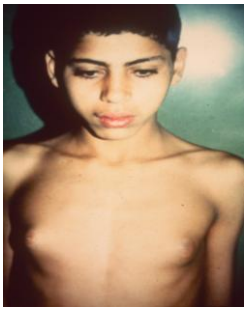
- It is large, soft, irregular & non capsulated mass.
- **Prognosis :** The better in prognosis than scirrhous because of lymphocytic infiltrations.

(3) Cancer breast (Mucinous)

- It is large, soft, irregular & non capsulated mass.
- **Prognosis :** The best prognosis

DISEASES OF MALE BREAST

Gynaecomastia

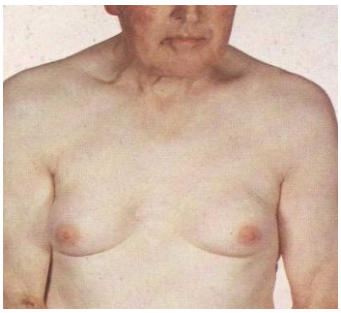


Definition

generalized enlargement of the glandular element of the male breast.

Aetiology

- **Physiological**
  - ***Infantile*** : from maternal sex hormones.
  - ***Pubertal*** : resolves within 2 years when adult testosterone level is reached
  - ***Senile Gynaecomastia :*** from ↓ testicular functions with age.



- **Pathological** 2ry to
  - ↓ ***Testosterone*** : e.g. Orchidectomy.
  - ↑ ***Oestrogen*** : e.g. Supra-renal tumor.
  - ↓ ***Metabolism of oestrogen*** : e.g. Liver cell failure.
  - ***Ectopic hormones :*** e.g. Bronchial carcinoma.
  - ***Drugs***: e.g. Digitalis, Cimitidin, Aldactone
  - ***Chronic renal disease*** may be a cause
  - ***Sertoli cell tumor*** may be a cause

Clinical picture

- ***Symptoms*** **:** unilateral or bilateral, tender mass (i.e. like a **disc**).
- ***Sign*** **:** Enlargement of the male breast with prominent nipple due to hypertrophy of the glandular tissue. .

Investigations

- Hormonal profile & liver function tests .
- **Biopsy** if doubt of cancer.

Treatment

- **Medical (mainly) : 1. Physiological :** Reassurance.
  2. Pathological : Treatment of the cause.
- **Surgical :** If persists → **S.C. mastectomy**.

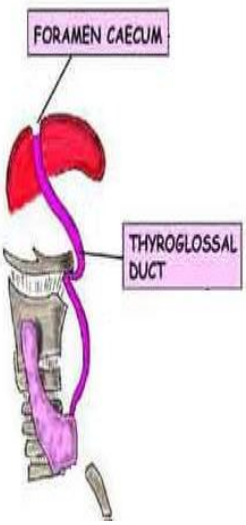
Thyroid disorders

THYROID GLAND DISORDERS

EMBERIOLOGY

Thyroid gland appears at the **3rd** week, as a median epithelial growth at the floor of the primitive pharynx (**foramen caecum**) = junction of anterior 2/3 & posterior 1/3 of the tongue

then descends to the lower part of the front of the neck being connected to the foramen caecum of tongue by **thyro-glossal duct** which disappears later on.



ANATOMY

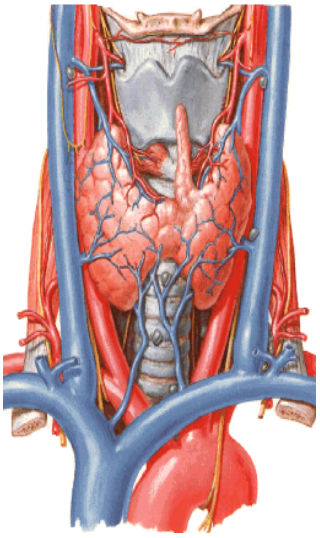
✷ Parts

- **2 Lobes**.
- **Isthmus.**
- **Pyramidal lobe :** 80 % of people is attached to hyoid bone by **levator glandulae thyroidae**

✷ Weight **20 - 25 gm**

✷ Level

- **upwards :** oblique line of the thyroid cartilage.
- **isthmus :** opposite **2nd** & **3rd** tracheal rings.
- **base :** at level of **4th** & **5th** tracheal ring



✷ Arterial supply

- **Superior thyroid artery :** from E.C.A & related to external laryngeal nerve.
- **Inferior thyroid artery :** from thyro-cervical trunk of 1st part of subclavian artery & related to R.L.N.
- **Thyroida ima artery :** from aortic arch (occasionally present & enters lower part of the isthmus).
- **Oesophageal** & **tracheal arteries :** supply the medial aspect of the gland

Surgical importance

✶ **Legature of superior thyroid artery (near) the upper pole (Why ?)**
to avoid injury of (external laryngeal nerve) which supplies crico-thyroid muscle

So if injury occurs :

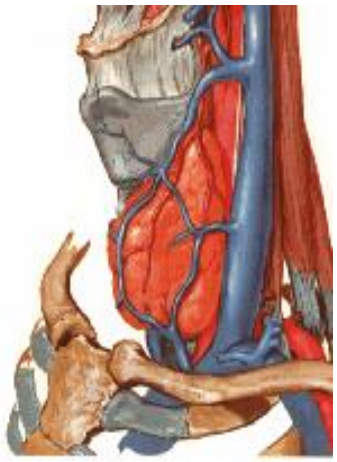
- ***unilateral*** → loss of high pitched voice
- ***bilateral*** → chocking

✶ **Legature of inferior thyroid artery (away from) the lower pole (Why ?)**
to avoid injury of (recurrent laryngeal nerve)

So if injury occurs :

- ***unilateral*** → dyspnea on exertion or hoarseness of voice
- ***bilateral*** → stridor (suffocation) which needs tracheostomy or aphonia.

✷ Venous drainage



- **Superior thyroid vein** which drained to I.J.V
- **Middle thyroid vein** which drained to I.J.V
- **Inferior thyroid vein** which drained to innominate vein

✷ Lymphatic drainage

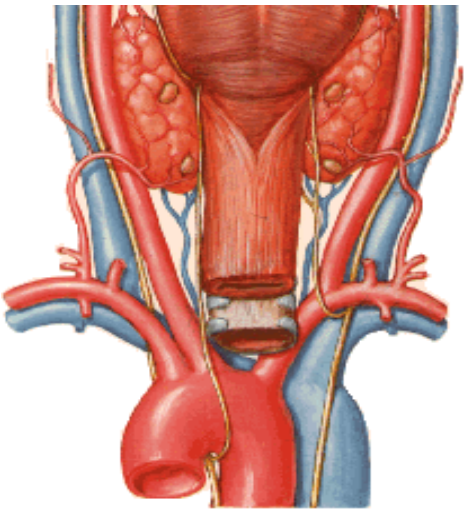
UPPER LEVEL

- **Lateral :** upper deep cervical L.Ns.
- **Medial :** pre-laryngeal L.Ns.

LOWER LEVEL

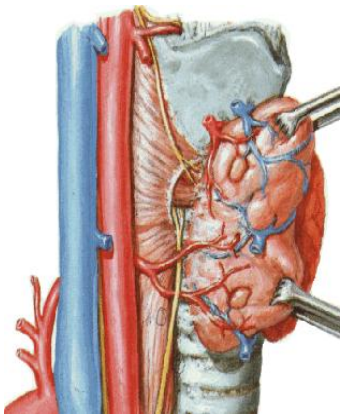
- **Lateral:** lower deep cervical L.Ns.
- **Medial :** pre- tracheal L.Ns.

✷ Nerves related to the gland



- **External laryngeal nerve**
  from superior laryngeal nerve
  from vagus nerve
- **Recurrent laryngeal nerve**
  from vagus nerve
  - **RT. R.L.N :** hooks around Rt. subclavian artery.
  - **LT. R.L.N :** hooks around the arch of the aorta.

✷ Relations of the thyroid gland



A- MEDIAL SURFACE

1. Upper part

Larynx (thyroid & cricoid cartilage) separated from them by 2 muscles

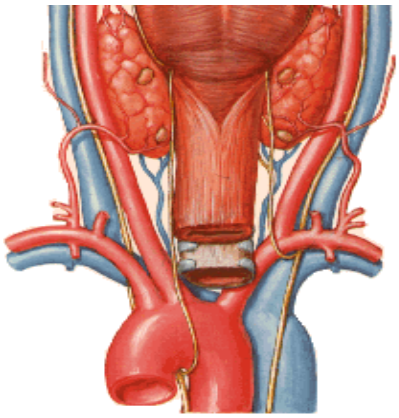
a. inferior constrictor m.

b. crico-thyroid in.

2. Lower part

trachea & esophagus with (R.L.N) in between.

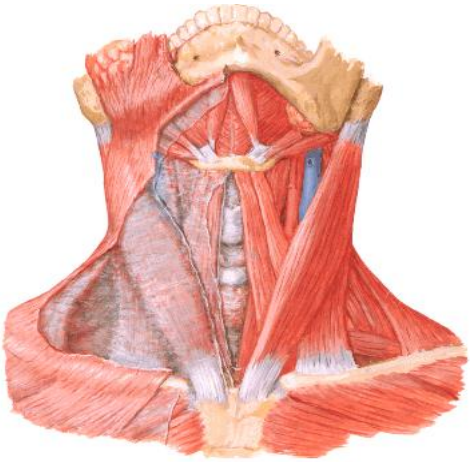
B- POSTERIOR SURFACE

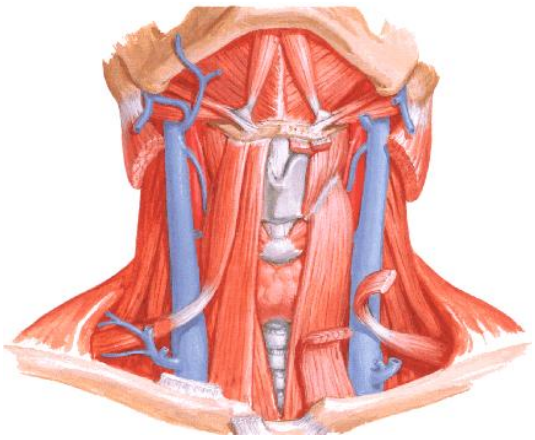


1. carotid sheath & its contents
2. superior & inferior parathyroid gland

C- SUPERFICIAL SURFACE

1. superior belly of omohyoid.
2. sterno-hyoid
3. sterno-thyroid
4. anterior border of sternomastoid
5. platysma
6. skin





✷ Capsules of the thyroid gland

A. True fibrous capsule enveloping the gland

B. False fascial capsule (derived from **pre-tracheal fascia)**

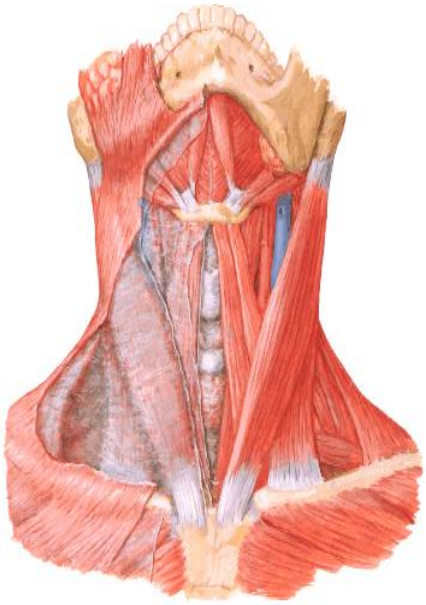
NB.: Attachment of pre-tracheal fascia

- ***Above*** *: Oblique line of thyroid cartilage & hyoid bone*
- ***Below*** *: Superior mediastinum.*
- ***On each side*** *: Carotid* sheath

- The fascia is thickened posteriorly forming the **ligament of Berry** which fixes the back of the gland. So it allows the gland to move up & down during deglutition).

ANATOMICAL CONSIDERATIONS

1- THE STERNOMASTOID MUSCLE



- **Origin :**
  - **Sternal head :** from the manibrium sterni .
  - **Clavicular head :** from the clavicle.
- **Insertion :**
  at mastoid process
- **Nerve supply :**
  Spinal part of accessory nerve.

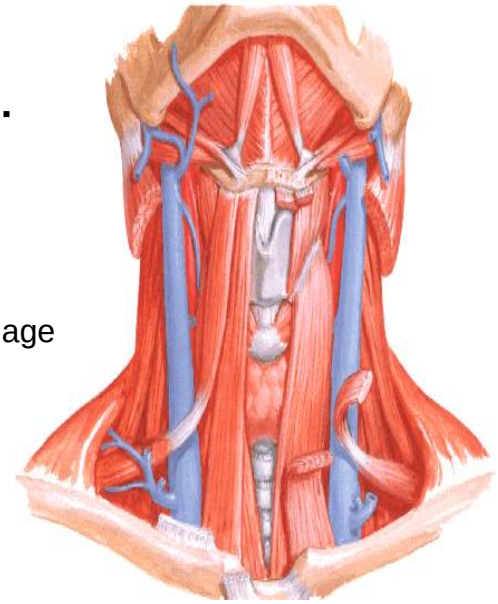
.....

2- THE PLATYSMA MUSCLE

- **Extent :**
  - **Above :** the mandible
  - **Below :** the manibrium sterni & clavicle
- **Nerve supply :**
  Cervical branch of facial nerve
- **Applied anatomy :**
  It must be closed as a separate layer or it will becomes adherent or form ugly scar

.....

3- STRAP MUSCLES



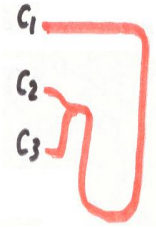
- **Types :**

☆ **STERNOTHYROID MUSCLE**
from sternum to oblique line of thyroid cartilage

☆ **STERNOHYOID MUSCLE**
from sternum to hyoid bone.

☆ **OMOHYOID MUSCLE [2 heads]**
- **One head** from the scapula .
- **The other head** from hyoid bone & joined together by intermediate tendon passing under the sternomastoid

- **Nerve supply :**
Ansa cervicalis which is A LOOP formed of Ant. rami of C**1**,C**2** &**3**
C**1** = Descendus hypoglossi
C**2** & **3** = Descendus cervicalis

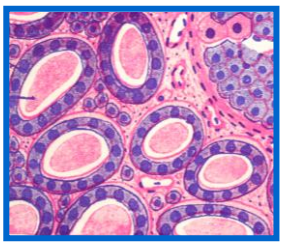


- **Applied anatomy :**
Ansa cervicalis enter the muscles from **below**
SO retraction of these muscles as **high** as possible during thyroidectomy

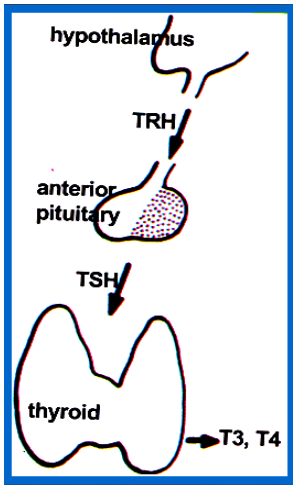
HISTOLOGY

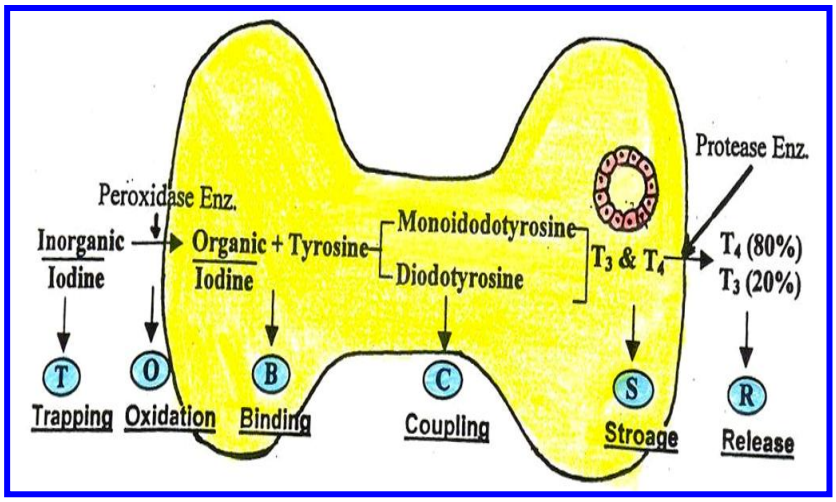
The thyroid gland is formed of acini which are

- **Separated by** connective tissue.
- **Lined by** simple cubical epithelium.
- **Filled with** colloid substance.



PHYSIOLOGY





✷ Physiological functions of the thyroid gland

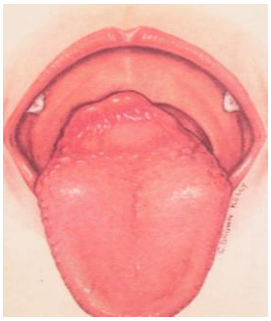
THE EFFECT OF THYROID HORMONES

1. **Catabolic effect :** (lipolysis - proteolysis – glycogenolysis).
2. **Metabolic effect :** (↑ metabolic rate & energy liberation)
   so, thyroid hormones are called **thermogenic hormones** .
3. **Stimulate :** (skeletal growth, sexual maturity & mental development).
4. **Increase sensitivity** of receptors for catecholamine e g. adrenaline.
   so, leads to (heart → ↑ **HR**, hand → **tremors**
   & skin → **sweating**).

.....

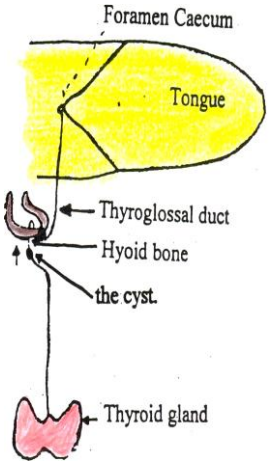
CONGENITAL ANOMALIES

I. Lingual thyroid



- **Ectopic thyroid tissue** occurs at base of tongue
  i.e. Failure of descent of the **thyroid tissue**
- **IF enlarged → dys**pnea, **dys**phagia & **dys**arthria.
- **Investigation :** Thyroid scan will establish the absence of thyroid tissue in the normal site of the neck.
- **Treatment :** wedge excision or radioactive-iodine ablation
  + L-thyroxin for life.

II. Thyroglossal cyst



AETIOLOGY

Remnant of thyroglossal duct.
i.e. midline **tubulo-dermoid cyst**

SITE

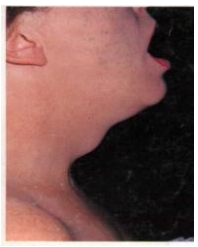
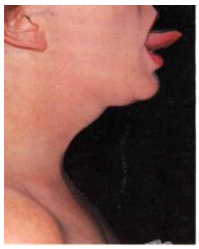
At any point of the course of thyroglossal track
(from foramen caecum to the isthmus of the gland)

N.B.: The commonest site is Just **below** the hyoid bone

PATHOLOGY

- **The wall** of cyst is lined by columnar epithelium.
- **Its wall** is rich in lymphatic tissue which is connected to other neck lymphatics (**so, liable for** recurrent attacks of inflammation)
- **The cyst** contains clear mucoid fluid rich in cholesterol crystals.

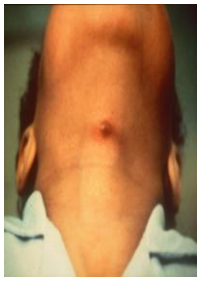
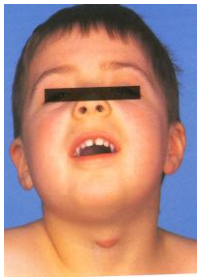
CLINICAL PICTURE

- **Age :** common at childhood.
- **Symptoms :** 1. **mass** at middle line of neck
  2. pain if infected.
  3. fistula if complicated.
- **Signs :** 1. **rounded** mass at middle line of neck.
  2. **tense** & **cystic** in consistency.
  3. **moves** up & down with deglutition & protrusion of tongue.

DD Subhyoid bursitis

COMPLICATION

Thyroglossal fistula

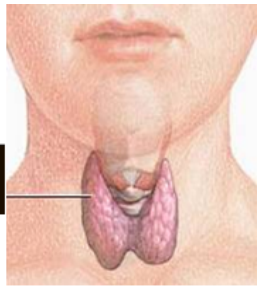
- **Incidence :** acquired **never** congenital.
- **Aetiology :** infection of cyst due to high lymphoid tissue or inadequate removal of the cyst.
- **Manifestations :**
  1. **Discharge :** viscid fluid or pus.
  2. **Firm tract :** from fistula (below) to hyoid bone (above).
  3. **The opening :** crescentic due to fibrosis from infection.

TREATMENT

Sistrunk operation

- **Elliptical incision** over cyst or fistula.
- **Excision** of cyst or fistula.
- **Removal of** 1. the track.
  2. the centre of hyoid bone to prevent recurrence.
  3. core of tissue up to foramen caecum of tongue

GOITER



Enlargement of The Thyroid gland

Classifications

1. **Simple Goitre**
   - ★ Physiological goiter
   - ★ Colloidal goiter
   - ★ Simple Nodular Goiter (SNG)
2. **Toxic Goitre**
   - ★ 1ry or 2ry Toxic goiters
   - ★ Toxic Nodule.
3. **Neoplasm**
   - ★ Benign Neoplasm
   - ★ Malignant Thyroid
4. **Inflammation**
   - ★ Inflammatory
   - ★ Autoimmune

I - SIMPLE GOITRE

Non toxic, **non** inflammatory, **non** neoplastic enlargement of the thyroid gland

(i.e. The patient is euthyroid)

Aetiology

Simple goitre is due to persistent stimulation of thyroid gland by ↑ **T.S.H** which is due to ↓ production of thyroid hormones from :

1- Iodine deficiency which may be

- **Absolute :** endemic as Oases i.e. dietary iodine deficiency.
- **Relative :** as puberty, pregnancy & lactation i.e. ↑ demands.

2- Enzyme deficiency **Pendred's syndrome**

- congenital deficiency of **peroxidase** enzyme which converts inorganic I_2 into organic I_2.
  C/P : goitre + dwarfism + deafness & mutism

3- Goiterogenic substance

- **Diet :** thiocyanates in cabbage & cauliflower.
- **Drugs :** antithyroid drugs as thiouracil which interfere with iodine uptake or hormone synthesis

Types

(1) Physiological (diffuse hyperplastic) goitre .

(2) Colloid goitre .

(3) Simple nodular goitre.

1- Physiological goitre

Diffuse hyperplastic goitre

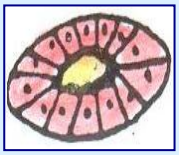
Incidence common in young girls ᵘ **Venus neck** "

Aetiology **Stress** " **relative** I_2 deficiency as puberty, pregnancy & lactation i.e. ↑ demands.

Pathogenesis " **Diffuse hyperplasia** "

Stress → ↑ TSH from ant. pituitary → diffuse hyperplasia & hypertrophy

So the change is

From **cubical**  → to **columnar** 

So cut section shows **fleshy brown** appearance with **little** colloid.

Clinical picture diffuse enlargement + (**s**mooth, **s**oft & **s**ymmetrical)

Fate return back to normal if stress is corrected
But If untreated, it may passes into **S.N.G**

Treatment Reassurance + L.thyroxin 0.2 mg/d (several months)
then 0.1 mg/d (several years).

2- Colloid goitre

[It is Intermediate stage between **diffuse hyperplasic** & **nodular goiter]**

Incidence common in endemic area.

Aetiology patient with (diffuse hyperplasic goiter) **receiving large doses of iodine**.

Pathogenesis " **Hyperinvolution** "

If the aetiology is removed **suddenly** as large dose of Iodine is taken.

this leads to hyperinvolution.

So the change is

from **cubical**  or **columnar**  → to **flat** 

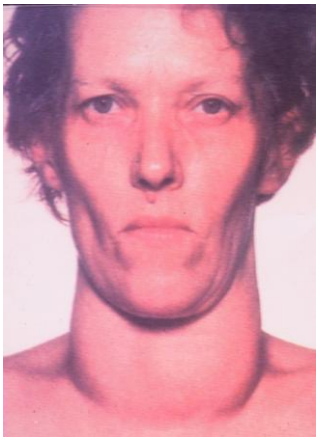
So cut section shows **golden brown** appearance with **excess** colloid

Clinical picture Irregular enlargement & soft in consistency

Fate If untreated, it may passes into **S.N.G**

Treatment Subtotal thyroidectomy.

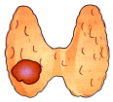
3- Simple Nodular Goitre
(S.N.G)



Incidence (The commonest disease of thyroid gland)

- **Age :** middle aged.
- **Sex :** female > male.

Aetiology See before

Types It may be **single**  or **multinodular** 

Pathology

- **Repeated cycles** of hyperplasia & involution
- **The nodules** are inactive.

Complications

A- In the gland itself

1. **Cyst formation** i.e. Hge in cyst.
   - consider emergency case because of sudden compression on trachea → reflex spasm of pre-tracheal muscles → impending suffocation.
   - **Treatment :** 1. aspiration.
     2. incision in skin & deep fascia allowing the gland releasing the trachea until thyroidectomy is done.
2. **Calcification** on long standing.
3. **Carcinoma** [follicular type **3 %**].
4. **2ry thyrotoxicosis** [**30 %**].
5. **(R.S.E)** Retro Sternal Extension more in males due to short neck & strong neck muscles.
6. **Carotid artery displacement** → fainting attacks due to ↓ blood supply to brain

B- pressure on trachea

1. **unilateral** compression → **kinking** of trachea.
2. **bilateral** compression → antero-posterior slit → **scabbard trachea**
3. **Tracheomalacia (Chondromalacia)** absorption of tracheal rings → **collapse** post-operatively

Clinical picture

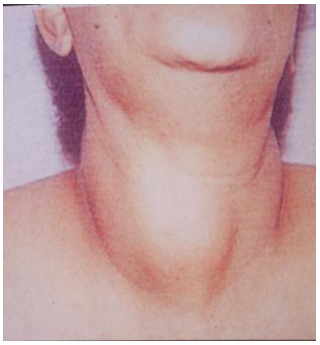
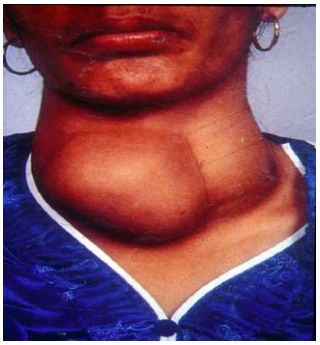
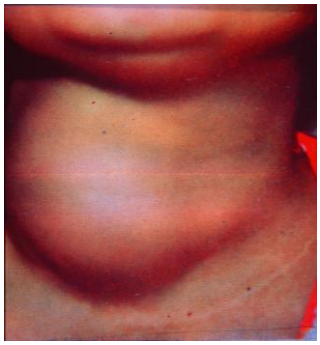
★ **Symptoms : slowly enlarged neck swelling** i.e. disfigurement or pictures of complications.

★ **Signs:**

A- General examination

- Exclude **toxicity** (Pulse, ABP, Eye signs ... etc)
- Exclude **metastasis** (**L**iver, **B**one, **L**ung & **B**rain)

B- Local examination

INSPECTION

usually single in **number**, localized or diffused in **shape**, variable in **size**, nodular **surface**, normal **skin over** (except dilated vein with R.S.E). It can moves up & down with deglutition (except with R.S.E) as **special sign**, well defined **edge** & the lower border can be **seen** (except with R.S.E), the trachea is not shifted (except with R.S.E)

PALPATION

not tender except if infected & the lower border can be **felt** (except with R.S.E), firm in **consistency** (hard if calcified or cystic if Hge in cyst)

PERCUSSION

on **manubrium sterni :** normally resonant (except dull with R.S.E).

AUSCULTATION

No murmur except if complicated by 2^{ry} toxic goiter

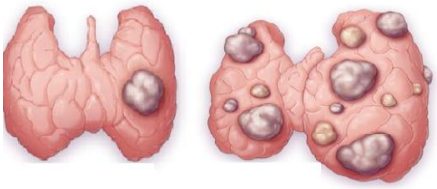
Investigations

1. **Thyroid scan** with **Tc^{99} :**
   show heterogenous uptake all over the gland
2. **Thyroid function tests :**
   to exclude toxicity.
3. **Plain X-ray ;**
   may show calcification & tracheal deviation.
4. **Neck U/S :**
   to detect cystic from solid lesions & can also detect R.S.E

Treatment

★ **Indicated with** ➪

1. cosmetic disfigurement.
2. R.S.E
3. pressure (if huge).
4. suspicion of malignancy.



✯ **So if solitary nodule :** hemi-thyroidectomy = lobectomy + isthmusectomy

but if multinodular : subtotal thyroidectomy, leaving about 8 gm of relatively normal thyroid (size of a normal lobe) on each side.

N.B.:*1.* ***We leave postero-medial part*** *to preserve parathyroid & R.L.N*

2. ***All operations*** *followed by L-thyroxin 50 – 100 ug/d*

3. ***Surgery is not advised below 25 year*** *to avoid recurrence.*

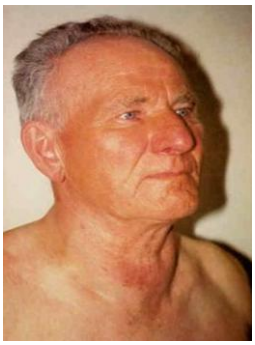
RETROSTERNAL GOITRE

Goiter in superior mediastinum

Types (3 Varieties)

1. **Mediastinal goitre**
   which lie in the superior mediastinum, but **still** derive its blood supply from **thyroid** vessels in the neck.
2. **Intrathoracic goitre**
   which lie in the superior mediastinum, but derive its blood supply from **mediastinal** vessels **i.e. ectopic thyroid**
3. **Plunging goitre**
   which lie in the superior mediastinum, **but rise in the neck** during deglutition & then descend again.

Clinical picture



★ **Type of patient :** more common with male due to short neck & strong muscle.

★ **Symptoms : Mediastinal syndrome**
(dyspnea, dysphagia & congested neck veins)

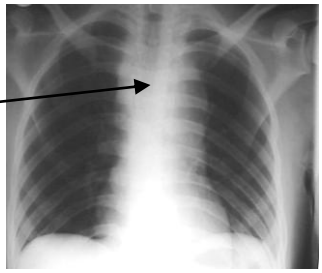
★ **Signs :** ***1. Inspection :***
- **dilated superficial veins** on neck & chest due to obstruction of the innominate veins
- lower border **can't be seen**.
- trachea may be **shifted**.

2. Palpation : lower border **can't be felt**.

3. Percussion : **dullness** over manubrium sterni

★ **Special test : (Pamberton's sign)** ▪▪▪▪▪▪▪▪▪▪▪▪▪▪▪▪▪▶
ask patient to raise up arms & keep this position for a while → congestion of face due to obstruction of great veins & trachea at thoracic inlet

Investigations



1. **X-ray chest :** shows soft tissue shadow & shifting of the trachea.
2. **CT scan chest :** more accurate.
3. **Thyroid scan with Tc^{99} :** diagnostic

Treatment

- **Preoperative preparation** by Inderal, especially with toxic R.S.E

N.B.: Antithyroid drugs *are contraindicated because* → ↑ size of gland

- **Operative procedures**
  1. **Try to deliver it** to neck after devascularisation of the gland & remove it like usual thyroidectomy
  2. **If failed** → piecemeal removal.
  3. **If failed** → median sternotomy (Rarely used)

II - TOXIC GOITRE

Thyrotoxicosis

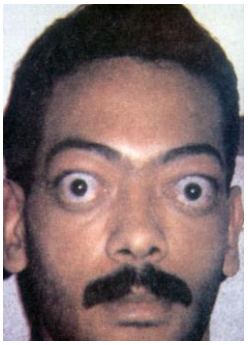
1. 1ry toxic	2. 2ry toxic	3. Toxic nodule	4. Rare types
75 %	15 %	5 %	5 %

1. 1ry toxic = Diffuse toxic = Exopthalmic goitre = Grave's disease

- **Aetiology :**
  - It is an autoimmune disease initiated by IgG antibodies stimulating T.S.H receptors on a previously normal gland → release of C-AMP → ↑ production of T_4

    N.B. : L.A.T.S = **L**ong **A**cting **T**hyroid **S**timulator is one of these IgG

  - The disease is often precipitated by **stress** e.g. psychic trauma, emotional stress & acute infection.



- **Affect : 20 - 40** years.
- **Characterized by diffuse** enlargement + (**s**mooth, **s**oft & **s**ymmetrical)
- **Mass** occurs at **same** time of toxicity.
- +ve **Eye signs** & Reticulo-endothelial system hyperplasia**.** e.g. spleen & L.Ns.

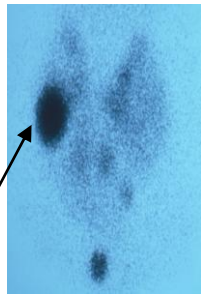
2. 2ry Toxic = Nodular goitre = Plummer's disease

- **Aetiology :**
  - It is a complicated simple nodular goitre

    N.B.: The nodules are **inactive** & inter-nodular tissue is the **only active part**

- **Affect : 40 - 50** years.
- **Characterized by localized** enlargement (firm, nodular & asymmetrical)
- **Mass** occur **before** time of toxicity.
- Extremely rare **Eye signs**.

3. Toxic nodule



- It is a **solitary** (overactive) nodule.
- It is **autonomous** because hypertrophy & hyperplasia **not** affected by T.S.H.
- **Investigation : Thyroid scan** shows **Hot** nodule. because T.S.H secretion is suppressed by the high levels of circulating thyroid hormones → so the normal thyroid tissues surrounding the toxic nodule are suppressed
- **Treatment : Hemi-thyroidectomy** after control of toxicity by **inderal**.

Don't forget

✭ Rare types of toxic goitres

1. **Neonatal thyrotoxicosis :** newborn of **thyrotoxic mothers**.
2. **Thyrotoxicosis factitia :** due to excess intake of **L. thyroxin**.
3. **Jod-Basedow thyrotoxicosis :** due to large dose of **iodine** given to hyperplastic gland → temporary ↑ T_4
4. **Hashitoxicosis :** 5 % of hashimoto's thyroiditis in early stages are thyrotoxic
5. **Decurvan's thyroiditis :** due to libration of hormones from destroyed tissue
6. **Functioning carcinoma.**
7. **TSH secreting adenoma** of the pituitary gland.

Clinical picture

✭ **Age :**

- **1ry** toxic goitre (**20 - 40** years)
- **2ry** toxic goitre (**40 - 50** years)

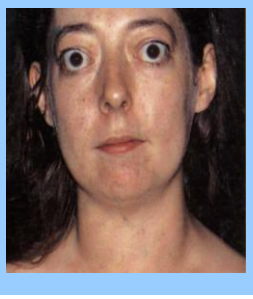
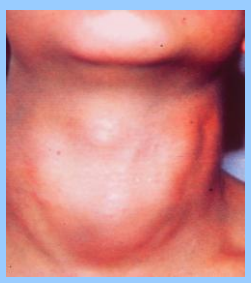
✭ **Symptoms :**

The term Thyrotoxicosis is better used than hyperthyroidism,
as not all manifestations due to ↑ thyroid hormones
e.g. True exophthalmos or pertibial myxodema

- ***Metabolic :*** 1. loss of weight inspite of good appetite
  2. ıntolerance to hot weather.
  3. excessive sweating.
- ***C.V.S :*** Palpitation even (at rest) if there is heart failure, the patient complains of orthopnea & bilateral edema at both lower limbs.
- ***Chest :*** exertional dyspnea.
- ***C.N.S* :** 1. fine tremors of tongue & hand
  2. irritability & anxiety
  3. insomnia.
- ***G.I.T :*** polyphagia ± diarrhea.
- ***Urinary :*** polyuria

***N.B.:* Causes of polyuria** in this case :
- Glucosuria.
- ↑ Metabolic water.
- ↑ Water intake 2ry to polyphagia.
- ↑ Renal blood flow.

- ***Skeletal* :** generalized bone aches, i.e. **osteoporosis**.
- ***General :*** diplopia of eye or corneal ulceration.
- ***Gonadal :***1. impotence in **male**.
  2. menstrual disturbance in **female**.

	1ry Toxic Goitre	2ry Toxic Goitre
		
• **Age**	20 – 40 years	40 -50 years.
• **Exophthalmos**	true	apparent
• **Metabolic** • **C.V.S & Chest** • **C.N.S**	**+++** **+** **+++**	**+** **+++** **+**

★ **Signs :**

A. Vital signs

1. Temp. (↑) With toxic goitre.

2. Pulse rate :

" Tachycardia, irregular, large volume, equal on both side & water hummer pulse as special characters." because of (↑ systole & ↓ diastole).

N.B.: **1. All types of arrhythmia** can occur **except** heart block.

2. Sleep pulse means examination of pulse during sleep = > 90 /min

the value to exclude anxiety & for follow up.

3. A.B.P (↑ systole & ↓ diastole).

4. R.R may be increased

B. General examination

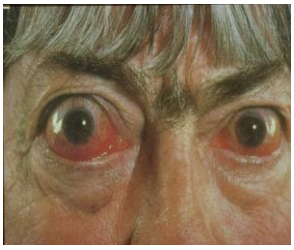
A = Appearance	normal
B = Built	under built
C = Conscious	conscious
D = Decubitus	orthopnea **If heart failure** occur → 
E = Emotion	irritable & alert
F = Face	staring look

C. Systemic examination

I. HEAD

1. **Face :** flushed face.
2. **Tongue :** tremors (**N.B.: unsupported tongue**).
3. **Eye :** 1.**Tremors** in upper eve lid.
   2. **Eye signs** (see later).
   3. **Exophthalmos**

EXOPHTHALMOS



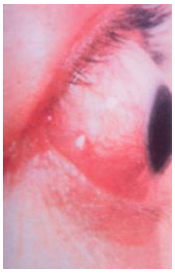
AETIOLOGY

Unknown cause but may be due to **E.P.S.**
(**E**xophthalmos **P**roducing **S**ubstance)

TYPES

1. False :

due to retraction of the upper eye lids due to contraction of **muller's muscle** (part of levator palpebrae superiors muscle) which innervated by sympathetic supply. **so ↑ thyroxin → ↑ sensitivity to catecholamines**



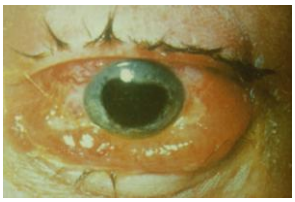
2. True :

(A) Moderate :

- actual protrusion of eye ball.
- due to deposition of retro-orbital fluid
- the condition is aggravated by ophthalmic vein compression → lid oedema & corneal ulceration

(B) Malignant :

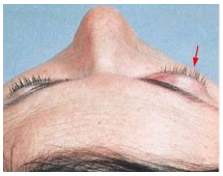
- severe progressive form of exophthalmos.
- due to weakness of extra-occular muscles.
- the condition leads to → corneal opacities, optic atrophy → may end in blindness.



HOW TO EXAMINE EXOPHTHALMOS

A- To show true or false

1. Naffziger test to see (the level of supra & infra-orbital ridges)



the examiner stands **behind** the patient, with the head tilted backwards.

In true exophthalmos :
supra & infra not in same plane

2. Russell Frazer's test to see (the obliteration of sulcus of supra-orbital margin with slight closed eye

.

the examiner stands **at side** of the patient

In true exophthalmos :
obliteration of the supra-orbital margin

3. Ruler test to see (the level of supra & infra-orbital ridge) by using a **Ruler**

In true exophthalmos :
The ruler will reach the cornea

B- To determine the degree

The distance between lateral orbital margin & apex of cornea is measured by a **Ruler** or **Exophthalmometer** (Normally = l5 -17m)

HOW TO EXAMINE EYE SIGNS

1. Stellwag's sign

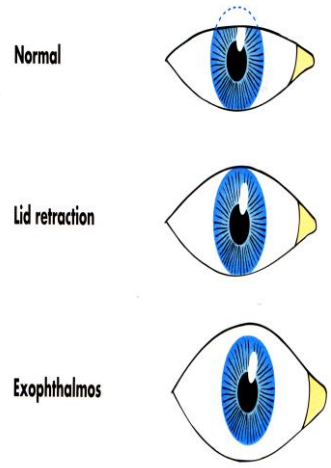
Staring look with Infrequent blinking
(Normally = 5 - 8 Times/min)

2. Von Graefe's sign

Lid lag when the patient looks **down** & while the head is fixed

3. Dalrymple's sign

appearance of **rim of sclera** above the cornea when the patient looks **down** & while the head is fixed



4. Joffroy's sign

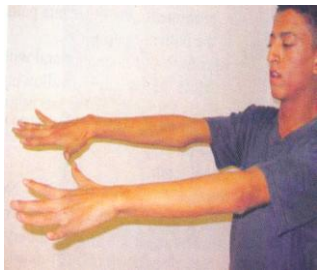
Loss of wrinkling of the forehead when the patient looks **up** & while the head is fixed

5. Moebius sign

Lack of convergence on looking at near object.

II. UPPER LIMB

For 1. Tremors of hands i.e. fine tremors
2. Exaggerated reflexes.
3. Warm sweaty hands.
4. Myopathy of proximal limb muscles.

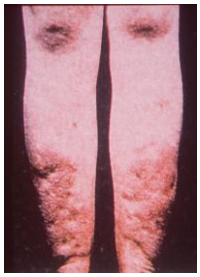


III. LOWER LIMB

For **Pretibial myxedema** which is ⇩

- **Multiple** yellow patches of the skin over shin of tibia.
- due to mucin deposition **not** due to ↑ T_3 or T_4
- may be associated with clubbing of fingers & toes

= Thyroid acropathy (more common with 1ry toxic)



IV. CHEST

For **heart** examination shows ↑ **heart sounds**

V. ABDOMEN

For **liver** & **spleen** enlargement may be associated with **1ry** toxic goitre

D. Local examination

1ry Toxic Goitre Grave's disease		2ry Toxic Goiter Plummer's disease
symmetrical, soft & smooth		Asymmetrical, firm & nodular

INVESTIGATIONS

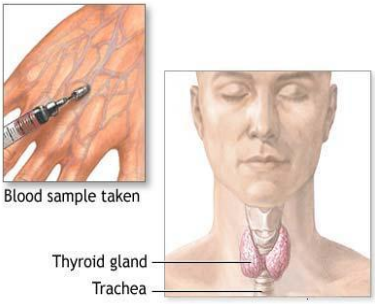
[A] Thyroid function tests.

[B] Radioactive I^{123} or Tc^{99} studies.

[C] Other investigations.

A. Thyroid function tests

[1] MEASUREMENT OF SERUM THYROID HORMONES



- **Total** serum T_4 = 55 – 150 nmol/l
- **Total** serum T_3 = 1.2 – 3.1 nmol/l
- **Free** serum T_4 = 8 – 26 pmol/l
- **Free** serum T_3 = 3 – 9 pmol/l

Total serum hormones are not accurate as they affected by fluctuation of serum level of plasma proteins.

So may by **- False high** with ↑ **ptn** as in pregnancy.

- False low with ↓ **ptn** as in nephrotic syndrome.

So measurement of free T_3 & T_4 are more accurate

N.B: T3 Thyrotoxicosis which is
- **High T3 & Normal T4**

[2] ESTIMATION OF TRH IN SERUM

Depressed levels of TRH are of value in early detection of **hyper**thyroidism

[3] ESTIMATION OF TSH IN SERUM

Depressed levels of TSH are of value in early detection of **hyper**thyroidism

[4] MEASUREMENT OF FREE BINDING SITES FOR THYROID HORMONES IN THE BLOOD (T_3 Uptake test)

Radioactive T_3 is incubated with the patient's serum → T_3 will bind to the free thyroid binding proteins → estimate the number of free binding sites in the serum.

So May by **- Low** with **hyper**thyroidism (thyrotoxicosis).

- High with **hypo**thyroidism (myxedema).

[B] Radioactive I^{123} studies or Tc^{99}

[1] RADIOACTIVE IODINE UPTAKE TEST

- Following an **oral** dose of **5 uci**
- The percentage of uptake of radioactive material by thyroid gland is measured at **4 hours** (normal uptake = **11** – **55** % of tracer dose)

So in Hyperthyroidism :

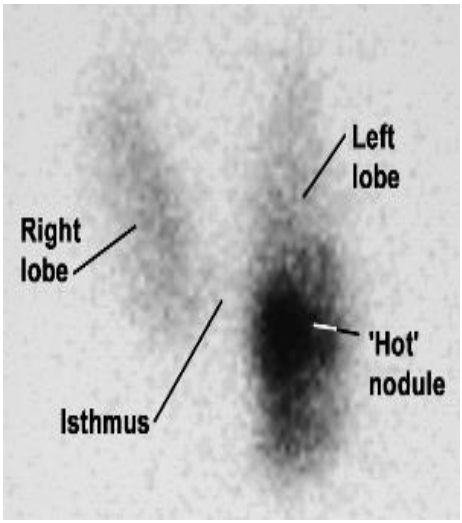
↑ Thyroid uptake **> 55 %** at **4 hours**

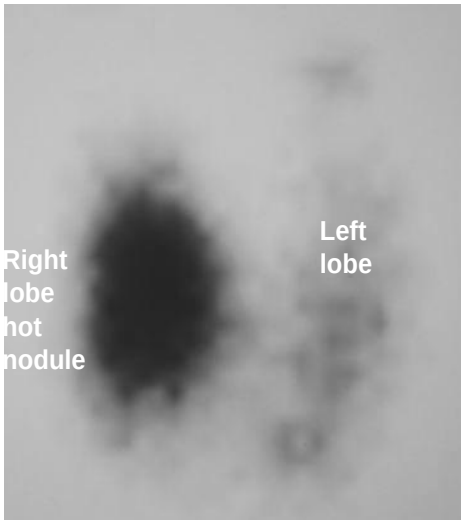
[2] THYROID SCAN

- After **injection** of **I^{123} (half life = 11 hours)**

or **Tc^{99}** a scan of the thyroid gland is obtained.

- **It allows evaluation of the functional activity** of the different areas of the gland, wether normal (**warm**)
  or hyperactive (**hot**)
  or non functioning (**cold**).
- **The principle value is in identifying** an autonomous toxic nodule





e.g. Thyroid scan shows **Hot** nodule.

because T.S.H secretion is suppressed by the high levels of circulating thyroid hormones → so the normal thyroid tissues surrounding the nodule are suppressed

[C] Others

1. **ECG** to exclude arrhythmia.
2. **Blood sugar** & **urine analysis** for glucosuria.
3. **Thyroid antibodies titer** (**L.A.T.S**) increased with 1ry toxic goitre

TREATMENT

	1ry	2ry	Toxic nodule
Medical ttt	**main** treatment	as pre-operative preparation	---------
Surgical ttt	• Failure of med. ttt • Recurrent. • Huge in size.	**main** treatment	Hemi-thyroidectomy (Isthmusectomy + lobectomy)
I^{131} Radiotherapy	If patient > 25 years.	----------	**main** treatment

(A) Medical treatment

INDICATIONS

1. **1ry toxic goitre:** all cases.
2. **2ry toxic goitre :** as pre-operative preparation.
3. **Children : No** surgery because of " ↑ risk of recurrence ".
4. **Refusal** of operation or **bad general** condition.

CONTRAINDICATIONS

1. **Solitary toxic nodule** " because it is non hormonal dependent ".
2. **Retrosternal goitre** " because anti-thyroid drugs leads to ↑ size of the gland ".
3. **Pregnancy**
4. **Lactation**

} because antithyroid drugs affects fetus leads to cretenoid goitre

LINE OF TREATMENT

1. **Mental & physical rest.**
2. **Sedatives & tranquilizers.**
3. **β Blocker**

e.g. Propranolol (Inderal)

- **Action :** - blocks the peripheral adrenergic features of ↑ T_4 .
  - partially ↓ conversion of $T_4 \rightarrow T_3$.
- **Dose :** 10 - 40 mg t.d.s. orally.

4. **Antithyroid drugs :**

A- Carbimazole (Neomercazole)

- **Action :** - prevents **o**xidation of inorganic iodine,
  - interfere with **b**inding of I_2 & tyrosine.
  - immunosuppressive on thyroid antibodies.
- **Dose :** 10 mg / 8h. orally till euthyroid state
  then 5 mg / 8h. orally for 1-1.5 years.
- **Onset :** after 7 - 14 days
  This means the already formed T_3 & T_4 are not affected

➢ **Side effects :**

(1) Aplastic anemia.

(2) **Agranulocytosis** :

- C/P : sore throat & fever are early signs.

- ttt : stop the drug, fresh blood transfusion & vit. B_{12} to stimulate bone marrow.

B- Propyl Thiouracil

➢ **Action :** as carbimazole + ↓ conversion of $T_4 \rightarrow T_3$

➢ **Dose :** 100 mg t.d.s till euthyroid state
then a maintenance dose 50 mg/d.

➢ **Side effects :**

(1) Bone marrow depression.
(2) G.I.T disturbance.
(3) Joint pain.

C- K Perchlorate

➢ **Action :** It interferes with I_2 trapping & very safe with children.

➢ **Dose :** 200 - 300 mg / 8h.

(B) Surgical treatment

INDICATIONS

1. **1ry toxic goitre :** - Failure of medical treatment.
   - Recurrence after medical treatment.
   - Huge in size.
2. **2ry toxic goitre :**The main treatment,
3. **Solitary toxic nodule** i.e. Hemi-thyroidectomy.

PRE-OPERATIVE PREPARATION

A- Neomercazole till reach the euthyroid state (for 2-3 months)

then Lugol's Iodine (5 % Iodine + 10 % KI in water)

➢ **Action :**

↓ protease enzyme → ↑ storage of colloid & ↓ vascularity, so the thyroid is easier to handle (firm & less vascular)

➢ **Dose :**

5-15 drops t.d.s for 10 days before operation .

➢ **Drawback :**

action can't be maintained for > 2 weeks .

➢ **Side effects :**

(1) skin rashes.
(2) excessive salivation.
(3) parotid enlargement.

B- Inderal 40 mg (3 Times / day)
1 week pre-operative & continued for 1 week after

OPERATIONS

- ✯ **Solitary toxic nodule :**
  Hemi-thyroidectomy = lobectomy + isthmusectomy
- ✯ **1ry & 2ry toxic goitre :**
  Subtotal thyroidectomy, leaving 1/8 of the gland

POST - OPERATIVE FOLLOW UP

1. indirect laryngoscope before hospital discharge.
2. serum ca^{+} after 6 weeks.
3. serum **T_3** & **T_4** every 6 months to detect recurrence or myxedema.

(C) Radioactive Iodine

INDICATIONS

1. **1ry toxic goitre** > 25 years.
2. **Solitary toxic nodule :** The main treatment,
3. **Recurrence** after surgical ttt.
4. **Poor** general condition.

CONTRAINDICATIONS

1. **Iodine allergy.**
2. **Young** to prevent risk of carcinoma.
3. **Pregnancy**
4. **Lactation**

} to prevent risk on fetus

LINE OF TREATMENT

Radio-Iodine I131 (half life = 8 days)

- **Action : I^{131}** → destruction of thyroid cells by beta particles
  → sub-lethal damage.
- **Dose :** 160 uci /1gm thyroid tissue.
- **Onset :** 2-5 months.
  If no improvement after 3 months, further dose is given
- **Side effects:**
  (1) Myxedema (80 % after 10 years).
  (2) Thyroid carcinoma (after 10 - 15 years).
  (3) Leukemia.

(D) Special problems

[A] THYROTOXICOSIS WITH PREGNANCY

- **Medical ttt : No** to avoid fetal hypothyroidism.

  N.B: Propyl thiouracil is safe during lactation as it secreted in a very low concentration in milk
- **Surgical ttt :** Indicated in **2^{nd}** & **3^{rd}** trimester.
  so we give minimal dose of propranolol before surgery.
- **Radio-Iodine I^{131} : No** to avoid destruction of fetal thyroid.

[B] THYROTOXICOSIS WITH CHILDREN

- **Medical ttt :** the **Ideal** until the age of 25 years.
- **Surgical ttt : No** to avoid high risk of recurrence.
- **Radio-Iodine I^{131} : No** to avoid high risk of malignancy.

[C] THYROCARDIAC PATIENT

- **Medical ttt :** as preoperative preparation only.
- **Surgical ttt :** the **Ideal**.
- **Radio-Iodine I^{131} : No** except for unfit patient for surgery.

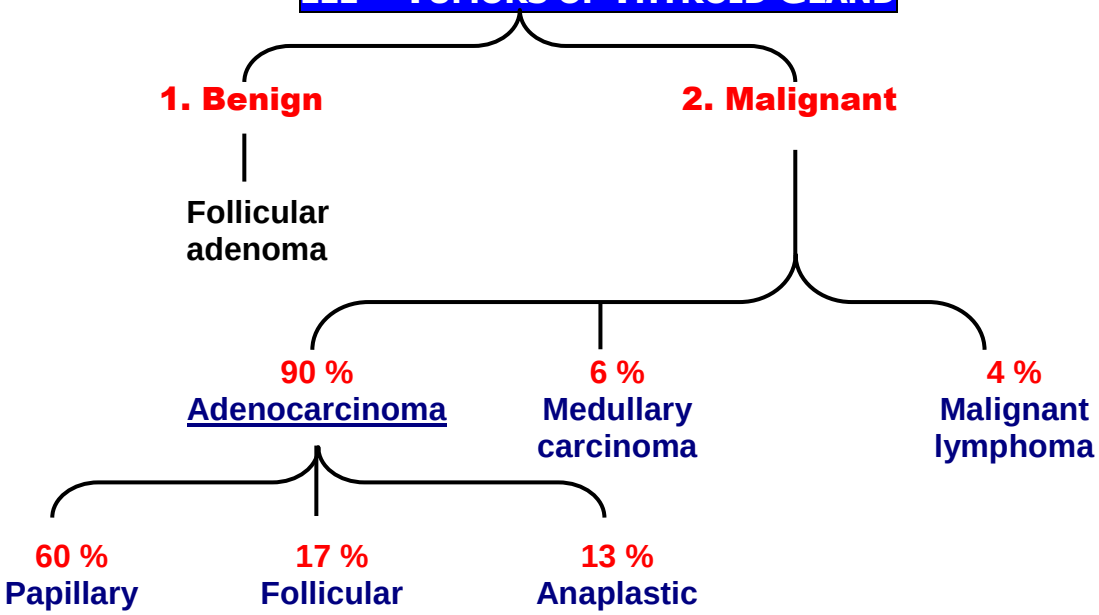
[D] PROGRESSIVE EXOPHTHALMOS

- **It is not advisable to terminate thyrotoxicosis by surgery or radio-iodine** for fear of a theoretical risk of progressing exophthalmos, so give anti-thyroid drugs until the condition is stable for 6 months.
- **Surgical ttt :**
  [**if malignant exophthalmos**]
  lateral tarsorrhaphy to protect the cornea.

[E] THYROTOXIC CRISIS (THYROID STORM)

- **Incidence :**
  Rare nowadays, because of good preparation & control of toxicity before operation.
- **Manifestations:**
  - **Symptoms :** muscular excitability up to convulsion.
  - **Signs :**
    (a) Temp. = hyperpyrexia up to 41°C or more.
    (b) Pulse = irregular & rapid up to 160/min or more.
    (c) A.B.P. = ↑ (systole & diastole) → heart failure.
- **Treatment :**
  [should be urgent]
  - **Ice Packs** → to limb, head & abdomen → ↓ pyrexia.
  - **Morphia** → sedation for convulsions.
  - **A.B.** → to prevent chest infection.
  - **O_2** Inhalation & I.V (**hydrocortisone**) 25 mg.
  - **Beta blocker** (**inderal**) **:**
    I.V drips in glucose under ECG screen till pulse reach normal level
  - **Lugol's I_2** 3cc in I000 cc glucose solution.

III – TUMORS OF THYROID GLAND



1- Benign tumors

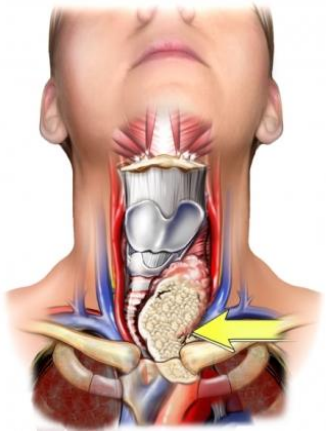
Follicular adenoma

- **Represent** as a solitary nodule.
- **Complicated** by follicular carcinoma.
- **Investigation** [histopathological examination]
  to detect evidence of invasion to capsule i.e. malignancy.
- **Treatment** (**Hemi-thyroidectomy**) lobectomy + isthmusectomy

N.B.: Papillary adenoma (most pathologist consider it as malignant)
so there is nothing called papillary adenoma.

.....

2- Malignant goiter

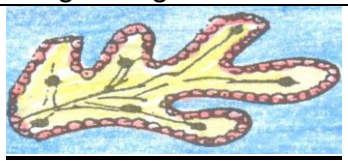
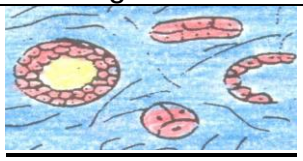
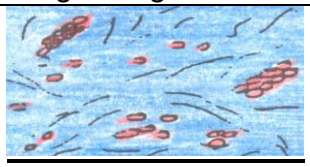


1- PREDISPOSING FACTORS

- **S.N.G** complicated by carcinoma
  i.e. **follicular** carcinoma.
- **Follicular** adenoma → **follicular** carcinoma.
- **Irradiation** to neck in children with T.B. lymphadenitis.
  i.e. **papillary** carcinoma.
- **Hashimoto's thyroiditis** → **papillary** carcinoma
  or **lymphoma**.

N.B.: Anaplastic carcinoma usually occurs de novo.

2- TYPES OF CARCINOMA

	Papillary	Follicular	Anaplastic
• **Incidence**	**60 %**	**17 %**	**13 %**
• **Age**	children & young	middle Age	elderly
• **Sex (F : M)**	**3.5 :1**	**2 : 1**	**1 : 1.3**
• **N/E picture**	**localized** & slowly growing nodule.	**ill defined** & irregular tumor.	**large** & rapidly growing tumor.
• **Microscopic picture**	 - cystic area with papillary projections. - composed of C.T center & covered with malignant cells - scattered ca. called **Psammoma bodies**	 - thyroid follicles with variable degree of differentiation. - vascular & capsular invasion.	 - clusters of spindle cells which are small or giant cells. - separated by little fibrous tissue. - local infiltration is seen
• **Differentiation**	Differentiated	Differentiated	**Un**differentiated
• **Spread**	mainly **lymphatic** "lateral aberrant thyroid" = thyroid with neck L.Ns metastasis.	mainly **blood** especially Bone.	mainly **direct**
• **Multiplicity**	**+ve** - It means multiple foci in same or other lobe. - due to intra-thyroid lymphatic spread i.e. **multicenteric**	**-ve**	**-ve**
• **Behaviour** - Hormonal dependency - I^{131} uptake - Response to irradiation	**+ve** **-ve** **-ve**	**-ve** **+ve** **-ve**	**-ve** **-ve** **+ve**
• **Prognosis**	the **best**	**less** favorable	very **poor**
• **10-year survival**	90%	70 %	die within 1-2 years.

N.B: Medullary carcinoma

- **Incidence : 6** %
- **Origin :** parafollicular (**C**) cells secreting calcitonin.
- **Spread :** 50 % are lymphatic spread.

Some tumors are familial

so form part of **MEN type IIa** (**Sipple's syndrome**) which is

[Medullary ca.**+** Pheochromocytoma **+** Hyperparathyroidism]

N.B : MEN (Multiple Endocrinal Neoplasia) syndrome

1- MEN type I (**Wermer's syndrome**) which is ↓

[Pituitary ca.**+** Gastrinoma **+** Hyperparathyroidism]

2- MEN type II (**Sipple's syndrome**) which is ↓

Type IIa

[Medullary ca.**+** Pheochromocytoma **+** Hyperparathyroidism]

Type IIb

[Medullary ca.**+** Mucosal neuroma **+** Hyperparathyroidism]

- **Manifestation :** diarrhea in 30% due to 5HT (5 hydroxy tryptamine)
  also may be due to ↑ PGs, produced by tumor cells.
- **Investigation :** ↑ level of calcitonin in blood.
- **Treatment :** Total thyroidectomy + selective neck node dissection.

.....

N.B: Lymphoma

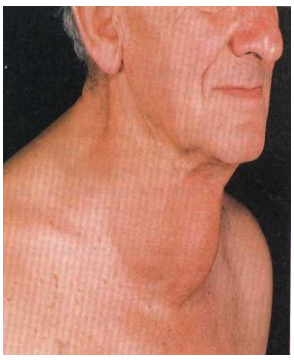
- **Incidence : 4** %
- **Origin : Lymphoid** elements.
  may be predisposed by hashimoto's thyroiditis.
- **Treatment :** Radiotherapy & chemotherapy.
- **Prognosis :** good prognosis.

.....

3. Staging T.N.M Staging

T = Tumor	N = Nodes	M = Metastasis
T1 = **< 1** cm.	**N0** = **No** palpable L.Ns.	**M0** = **No**
T2 = **1- 4** cm	**N1** = palpable L.Ns.	distant metastasis
T3 = **> 4** cm		**M1** =
T4 = Extension to the thyroid capsule		Distant metastasis

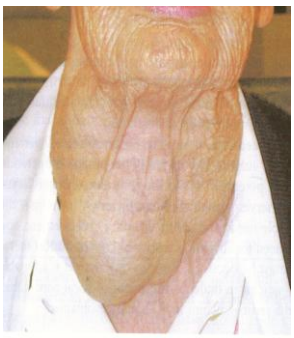
4- CLINICAL PICTURE



★ **Age :** usually **> 50** Years.

★ **Symptoms :**

- **Rapid** increase in size with **short** duration.
- **Pain** is related to swelling or referred to ear.
  i.e. Arnold nerve (branch from vagus nerve)
- **Metastasis** as [**L**iver, **B**one, **L**ung, **B**rain].
- **Symptoms of infiltrations** ⇘



  - **Trachea** → dyspnea.
  - **Oesophagus** → dysphagia but (rare)
  - **Sympathetic chain** → **Horner's syndrome**.
    (ptosis, myosis, enophthalmos, anhydrosis)
  - **Carotid artery** → postural fainting.
  - **Internal Jugular vein** → oedema of face.
  - **Recurrent laryngeal nerve** → may be
    - Hoarseness of voice if unilateral affection.
    - Stridor if bilateral affection.

★ **Signs :**

[A] General examination

- Examine for metastasis e.g. bony swelling.

[B] Local examination

- **Swelling** is **T**ender, **H**ard, **I**rregular, **E**nlarged & **F**ixed
- **"Berry's sign"** = **absent** carotid pulsation, due to infiltration of carotid sheath in advanced cases.
- **L.Ns** (enlarged, hard, 1st mobile later on fixed)

N.B.: ***Some patients represent 1st*** by L.Ns in the neck
i.e. occult carcinoma = papillary carcinoma.

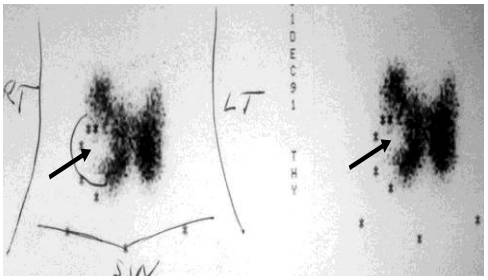
5- INVESTIGATIONS

[A] Laboratory for tumor markers

- ↑ Serum **calcitonin** = medullary carcinoma.
- ↑ **Thyroglobulin** = differentiated carcinoma.

[B] Diagnostic procedures

1- Radioactive I^{123} scan → cold nodule.

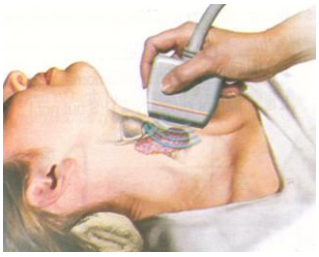


N.B.: ***DD cold nodule on scan :***
1. malignant tumor.
2. benign tumor.
3. simple nodule.

2- C.T scan & M.R.I

3- Neck U/S :

To DD cyst (papillary carcinoma) from solid.

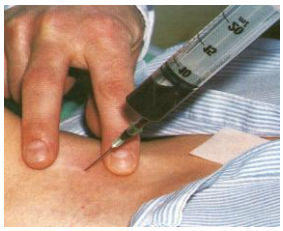


If cystic do aspiration ⇘

N.B.: ***Criteria of malignant aspirate :***

1- hemorrhagic.
2- residual mass.
3- rapid re-accumulation of fluid.
4- **+ve** cytology for malignant cells.

4- Biopsy :



- **Pre-operative L.N biopsy** :
  For enlarged cervical lymph node.
- **FNAC** : accurate with papillary carcinoma.
- **Excision biopsy** i.e. Hemi-thyroidectomy.
- **Frozen biopsy** i.e. during the operation

[C] Detection of distant metastasis

- **Lung :** X-ray.
- **Bone :** Bone scan (mainly with follicular carcinoma).
- **Liver :** Abdominal U/S.
- **Brain :** (Extremely rare) so no investigations are done.

[D] Detection of complications

- **Laryngoscopy** for R.L.N invasion.
- **Bronchoscopy** for tracheal invasion.
- **Oesophagoscopy** for oesophageal invasion.

6- TREATMENT

I. Operable " Total thyroidectomy "

★ **Indications :**

1. **Papillary** carcinoma **:** because it is multicenteric.
2. **Follicular** carcinoma **.**
3. **Early anaplastic** carcinoma**.**
4. **Medullary** carcinoma**.**

★ **Post-operative :**

L.thyroxin 0.1-0.2 mg/day as replacement therapy.

★ **Preservation of :**

1. R.L.N.
2. at least one of parathyroid gland.

★ **Removal of L.Ns :**

1. If **no** L.Ns or If **few** L.Ns : selective picking of L.Ns.
2. If **extensive** L.Ns : block dissection of L.Ns.

★ **Post-operative follow up :**

every 6 months by thyroid scanning, clinical exam. & tumor marker to detect local recurrence.

II. inoperable

★ **Criteria of inoperability :**

1. unfit for surgery e.g. cardiac disease.
2. patient with metastasis.

★ **Indications :**

1. **anaplastic** carcinoma.
2. infiltration to vital structures with **papillary** or **follicular** carcinoma.

MANAGEMENT

★ **Palliative isthmusectomy** (rarely) if tracheal compression

★ **According to types ;**

- **Papillary** : give L.thyroxin.
- **Follicular** : I^{131} uptake
- **Anaplastic** : give Ext. radiation.

★ **Treatment of complications as :**

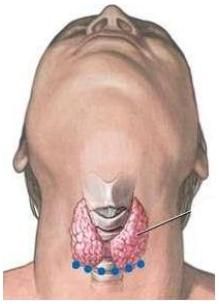
- **Tracheostomy** if tracheal invasion.
- **Gastrostomy** if oesophageal invasion.

7- PROGNOSIS **Bad prognosis if**

1. Age of patient : male > 40 years & female > 50 years.
2. Size of lesion > 5 cm.
3. Distant metastasis.
4. Presence of capsular or vascular invasion microscopically.

THYROIDECTOMY

✱ INDICATIONS



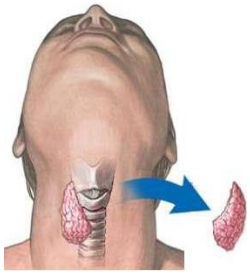
I. Subtotal thyroidectomy (Removal of 7/8 of the gland) →

① Main treatment of **2ry toxic** goiter.

② **1ry toxic** goiter with a. Failure of medical ttt.
b. Recurrent after medical ttt.
c. Huge in size .

③ **SNG** i.e Multi-nodular goiter

④ **Hashimoto's** thyroiditis



II. Hemi-thyroidectomy (Unilateral lobectomy) →

① **Toxic** Nodule.

② **Adenoma** of thyroid gland .

③ **SNG** i.e. Single nodule.

III. Total thyroidectomy **Malignant** goiter.

IV. Isthmusectomy

① **Anaplastic** carcinoma.

② **Riedel's** thyroiditis.

③ **Nodule** in the isthmus

V. Near total thyroidectomy

It means (Lobectomy + isthmusectomy + near total Lobectomy)

Complications after thyroidectomy

1. Recurrent laryngeal nerve injury :

- **causes :** pressure on the nerve by
  1. oedema & blood clot.
  2. accidentally ligated.
- **manifestations :**

  If • unilateral paralysis → hoarseness of voice & dyspnea.

  • bilateral paralysis → stridor & suffocation.

2. External laryngeal nerve injury ;

- **cause :**

  accidentally ligated during ligation of superior thyroid vessels
- **manifestations :** loss of high pitched voice.

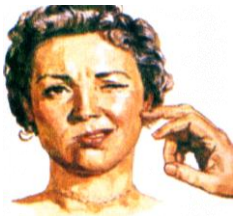
3. Respiratory obstruction :

- **causes :**
  1. Laryngeal edema due to trauma by the endo-tracheal tube.
  2. Bilateral recurrent laryngeal nerve injury.
  3. A tension hematoma in the deep cervical space.
  2. Tracheal collapse due to tracheomalacia.

4. Post-operative thyroid crisis (Thyroid storm) (see before)

5. Hypo-parathyroidism :

- **cause :**
  1. removal of all parathyroid gland.
  2. interrupt their blood supply.
- **manifestations :**
  - **latent** (Ca+ =7 – 9 mg %)
    → **Chevestic's** sign
    & **Trousseau's** sign
  - **manifest** (Ca+ < 7 mg %)
    → carpo-pedal spasm .



- **treatment :** slow I.V ca gluconate 10 cc 10 %.

6. Hypothyroidism :

Removal of too much thyroid tissue. (It needs replacement therapy)

7. Reactionary hemorrhage :

- **cause :** result from continuous oozing (bad haemostasis).
- **manifestations :** suffocation.
- **treatment :** urgent opened wound in bed.

8. Adherent Scar :

scar adherent to the trachea.

9. Increase of exophthalmos i.e. progressive

10. Recurrence of hyperthyroidism :

because of inadequate removal or hyperplasia of that has been left.

IV- THYROIDITIS

A- Inflammatory Thyroiditis

ACUTE BACTERIAL THYROIDITIS

- **Cause :** rare follows bacterial infection of mouth, pharynx & L.Ns.
- **Manifestations :** acute onset of severe neck pain & fever (**chills**).
- **Investigations :** - ↑ T.L.C (**T**otal **L**eucocytic **C**ount).
- **Treatment : Antibiotics** + draining of abscess which is rarely formed.

SUBACUTE VIRAL THYROIDITIS " De Quervain's thyroiditis "

- **Cause :** viral infection e.g. mumps or measles.
- **Manifestations :** 1. incidious onset of neck pain, fever (**no chills**)
  2. the gland is slightly tender (**pain radiates to ear**)
- **Investigations :** - ↓T.L.C (**T**otal **L**eucocytic **C**ount).
  - Thyroid function tests : ↑ production of T_4
- **Treatment : Anti-inflammatory** with sever cases

B- Autoimmune Thyroiditis

HASHIMOTO'S THYROIDITIS

- **Manifestations :** mild hyperthyroidism due to destruction of thyroid follicles → release of T_4 in blood.

N.B.: ***It may be complicated by***

- **Hypofunction** i.e. excess follicular destruction.
- **Carcinoma** i.e. papillary carcinoma or lymphoma.

- **Investigations :** 1. ↑ Thyroid antibodies titer.
  2. **Biopsy** to DD from carcinoma.
- **Treatment :** 1. Replacement therapy by L.**thyroxin** if hypofunction
  2. **Subtotal thyroidectomy** if leading to pressure symptoms.

C- Riedel's Thyroiditis

COLLAGEN DISEASE only 0.5 %

- **Manifestations :** 1. hard (**woody**) gland from excessive fibrosis.
  2. infiltration to surroundings.
- **Investigation : Biopsy** to DD from anaplastic carcinoma.
- **Treatment :** 1. Replacement therapy by L.**thyroxin** if hypofunction
  2. **Palliative isthmusectomy** to free the trachea from compression

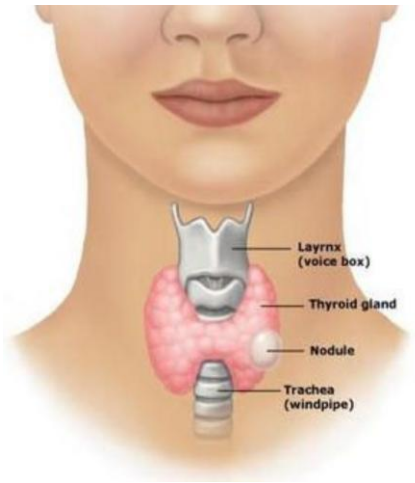
V- SOLITARY THYROID NODULE

DEFINITION

A goitre which clinically appears to be a **single nodule.**
which may be

1. **True solitary :** If one nodule is felt & the rest of the gland is **not** felt.
2. **Dominant :** If one nodule is felt & the rest of the gland is **slightly** felt.

AETIOLOGY



1. **Simple** nodule.
2. **Toxic** nodule.
3. **Adenoma**.
4. **Carcinoma**.
5. **Colloid** nodule.

FINDINGS THAT RAISE SUSPICION OF MALIGNANCY

1. **History** of previous irradiation.
2. **Elderly** patient.
3. **Recent** onset & **rapid** growth.
4. **Pain**.
5. If the nodule is hard, irregular, with limited mobility.
6. Presence of local invasion or lymphatic or blood metastasis.

INVESTIGATIONS

[A] Laboratory :

- Thyroid function tests : ↑ in thyrotoxicosis & functioning carcinoma.

[B] Thyroid scan :

- **Hot** nodule = Toxic nodule and possibility of malignancy is excluded.
- **Cold** nodule = The possibility of malignancy is 10 - 16 %
- **Warm** nodule = Adenoma & the possibility of malignancy is 3.5 %

[C] Neck U/S :

- To differentiate cyst from solid

[D] Biopsy :

1. **FNAC :**
   It is very useful in papillary tumor but can't differentiate benign adenoma from carcinoma as they need histo-pathology.
2. **True cut needle biopsy** (another option).
   It obtains a core of tissue for histo-pathology.
   But It may cause hematoma
3. **Excision biopsy** :
   The only diagnostic with Hemi-thyroidectomy

TREATMENT **According to the underlying aetiology :**

1. If <u>cyst</u> → aspiration → if malignant criteria as ⇘

1- hemorrhagic. 3- rapid re-accumulation of fluid.

2- residual mass 4- **+ve** cytology for malignant cells

→ proceed to **total thyroidectomy**.

2. If <u>benign condition</u> → Lobectomy is enough.

a- Simple nodule = hemithyroidectomy

b- Toxic nodule = Hemithyroidectomy after control of toxcicity by indral only

Propranolol (Inderal)

➢ **Action :** Blocks the peripheral adrenergic features of ↑ T_4

➢ **Dose :** 10 - 40 mg t.d.s. orally.

3. If <u>malignant condition</u> → Total thyroidectomy

I. Operable " Total thyroidectomy "

★ **Post-operative : L.thyroxin** 0.1-0.2 mg/day as replacement therapy

★ **Preservation of :** . R.L.N. & at least one of parathyroid gland.

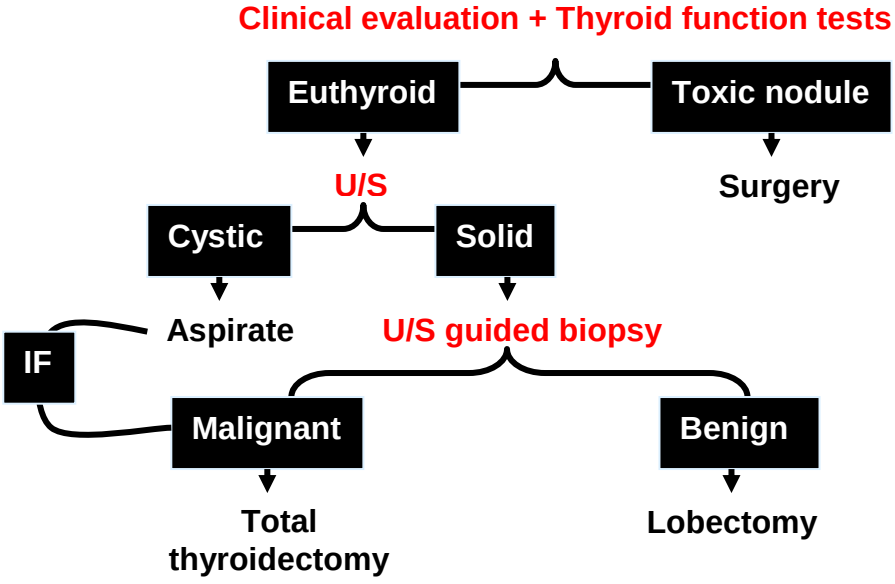
★ **Removal of L.Ns :** selective picking of L.Ns.

II. inoperable ★ **According to types ;**

- **Papillary** : give L.thyroxin.
- **Follicular** : I^{131} uptake
- **Anaplastic** : give Ext. radiation.

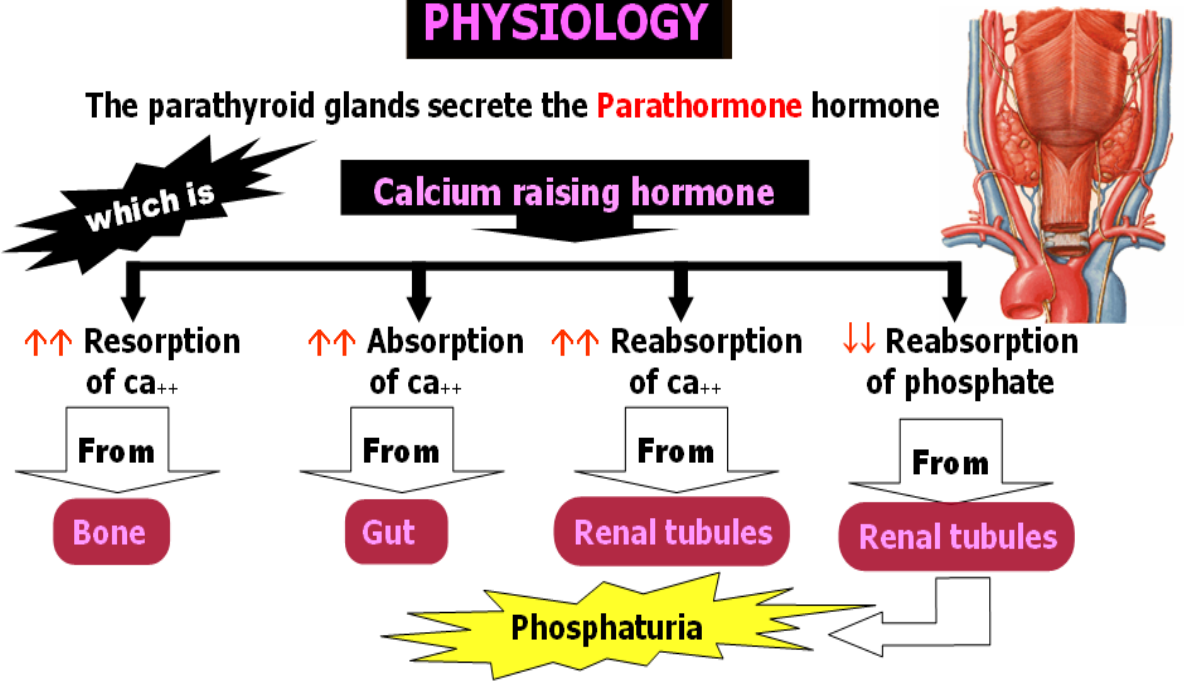
.....

Algorithm for the management of a solitary thyroid nodule



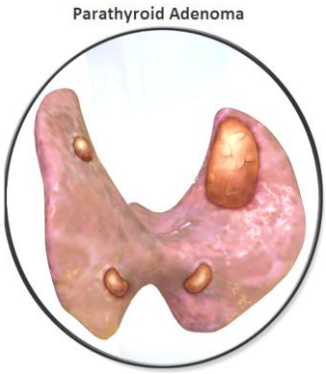
PARATHYROID GLANDS

PHYSIOLOGY



HYPERPARATHYROIDISM

AETIOLOGY



(3 Types)

1. **1ry hyperparathyroidism** (The commonest variety)
   it is due to single adenoma **85 %,** multiple adenoma **4 %** or hyperplasia **10 %** or carcinoma **1 %** .

2. **2ry Hyperparathyroidism**
   2ry parathyroid hyperplasia due to hypocalcaemic states e.g. chronic renal failure, malabsorption syndrome

3. **3ry hyperparathyroidism**
   with prolonged stimulation the reactive hyperplasia acquires **autonomy** it secretes excess hormone **without stimulation**.

CLINICAL PICTURE

(The disease of Bones, Stones, Abdomen & Psychic moans)

1. **Bones :**
   Generalized decalcification of bones = **ostitis fibrosa cystica**
   → multiple bone cysts → pathological fractures.
   e.g. (1) sub-periosteal resorption of **phalanges**
   → tufting of terminal phalanges
   (2) **skull** is the 2nd common site**.**

2. Renal stones :

Hypercalcuria & phosphaturia cause recurrent renal stones, bilateral renal stones (30 – 80 %) & nephrocalcinosis (5 – 10 %) the latter is irreversible & may lead to renal failure.

3. Abdomen :

Increases gastric & pancreatic secretion by hypercalcaemia may precipitate peptic ulceration or acute pancreatitis.

4. Psychic moans : common in women.

Restlessness, irritability, personality changes & even neurosis.

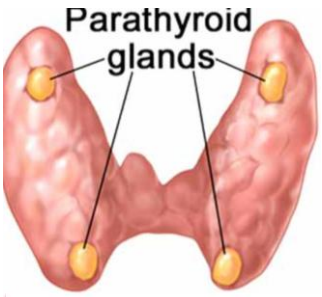
5. Articular & Soft tissues manifestations : Chondro-calcinosis

i.e. **Ca^{++}** deposition in articular cartilages & menisci

6. Hyperparathyroid crisis :

occurs with ↑ serum **Ca^{++}** & presents by muscular weakness, nausea, vomiting, weight loss, fatigue & drowsiness.

INVESTIGATIONS



1. Laboratory diagnosis

- ↑ **Serum Ca** level .
- ↑ **Excretion** of **Ca** & **Phosphate** in urine.
- **Immune assay for parathormone :**
  ↑ plasma level of **PTH**

2. Localization modalities

- **High resolution ultrasound :** with accuracy 76 %
- **CT scan :** with accuracy 50 %
- **<u>Thallium^{201} Technetium^{99} subtraction scan</u> :** with accuracy 72 %
  The Idea of this test is that **Tc^{99}** is taken only by thyroid gland while **Thallium^{201}** is taken by both thyroid & parathyroid gland by computerized subtraction of the two captured images, the parathyroids appear as **hot** spots.

3. Radiological skeletal survey

- **Skull** shows multiple small osteolytic lesions
  (**salt & pepper appearance**) fig.1
- **Hands** shows sub-periosteal resorption of **phalanges** fig.2
- **Long bones (pelvis & spine)** shows multiple osteolytic lesions
  (**ostitis fibrosa cystica**) fig.3

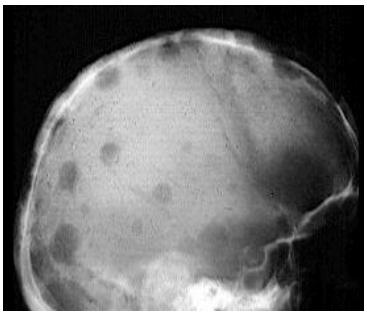


Fig.1

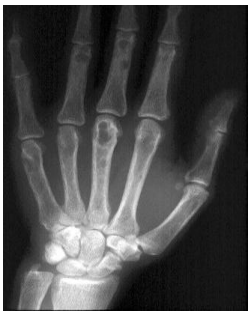


Fig.2

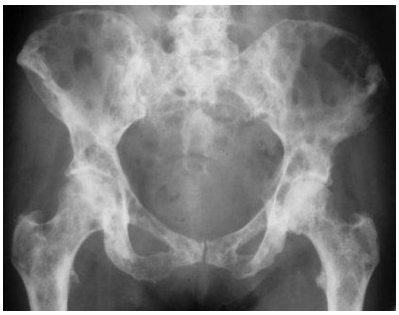


Fig.3

TREATMENT

1ry hyperparathyroidism

1. **If one gland enlarged by an (adenoma)**
   It should be excised, together with exposure of other glands to ensure that they are of normal size & biopsy of one of them.

2. **If parathyroid (hyperplasia)**
   Subtotal parathyroidectomy, i.e. excision of 3.5 glands

Another alternative surgical strategy

Total parathyroidectomy, with auto-transplantation of very thin slices in the forearm muscles.

2ry & 3ry hyperparathyroidism

Essentially medical

- **Vitamin D**
- **Calcium & phosphate binder**

Arterial disorders

ARTERIAL DISORDERS

ISCHEMIA

Diminished arterial blood supply

Limb ischemia

DEFINITION

Impairment arterial blood supply sufficient to interfere with nutrition and functions of the limb.

AETIOLOGY

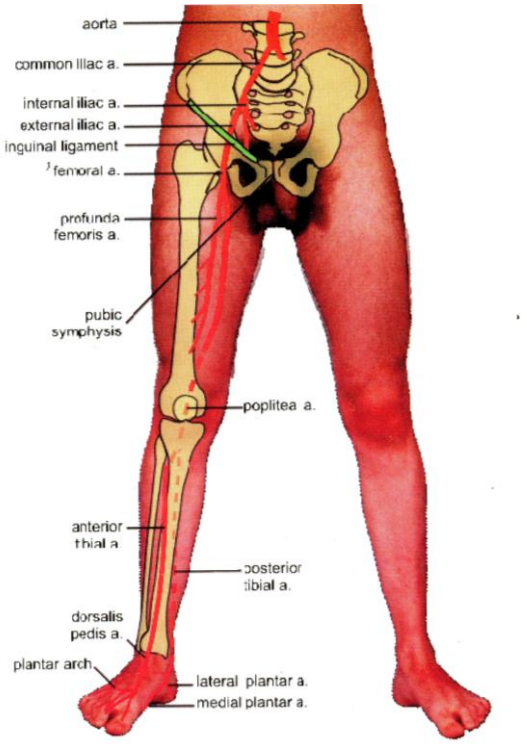
due to arterial occlusion by trauma or vascular disease.

TYPES

It may be :

- Acute ischemia.
- Chronic ischemia

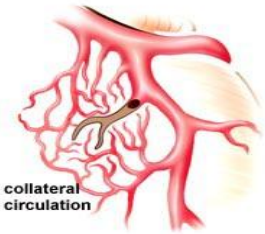
THE EFFECT OF ISCHEMIA DEPEND UPON



1. **The degree of arterial occlusion :**
   - Partial **:** mild ischemia.
   - Complete **:** severe ischemia.
2. **The rate of arterial occlusion :**
   - Acute **:** sudden ischemia.
   - Chronic : gradual ischemia.

N.B.: Acute ischemia more serious
because **no** time for development of collaterals.

3. **The type of artery for (collateral circulation)**
   - some arteries have **good** collaterals as subclavian artery & iliac artery.
   - other arteries have **poor** collaterals as brachial artery & popliteal artery.



4. **Heart failure** → ↓ cardiac output → ↑ ischemia.
5. **Anaemia** → ↓ O_2 → ↑ ischemia.
6. **Smoking** → ↑ ischemia.
7. **Cold weather** → spasm → ↑ ischemia.
8. **Alcohol** → **improve** collateral circulation → ↓ ischemia.

I - ACUTE ISCHEMIA

DEFINITION

Sudden total occlusion of a previously patent artery supplying a limb.

AETIOLOGY

1. **Embolism** " the commonest cause " .
2. **Causes in the wall :**
   - Arterial injuries.
   - Acute thrombosis on top of atherosclerosis.
   - Dissecting aneurysm.
3. **Compression on artery :**
   e.g. tourniquet, fractured bone…etc.
4. **Phlegmasia alba dolens :**
   extensive ilio-femoral D.V.T

PATHOLOGY

a. Sudden occlusion of an artery
→ stagnation of the nearby vein
→ tissues will be loaded with fluid
→ If gangrene occur, it will be moist aseptic.

b. Different tissues tolerate ischemia to variable extent :
- Muscle **:** irreversible damage within **6 - 8** hours.
- Skin **:** moist aseptic gangrene within **24** hours.

CLINICAL PICTURE [6PS]

➢ **Pain :**
- sudden onset.
- bursting or burning (nerve ischemia)
- starts al point of occlusion.
- shoots distally.

> **N.B.: Pain** may be **absent** in some patient :
> because of rapid onset of anesthesia.

➢ **Pallor :** which is replaced later on by mottled **cyanosis** due to accumulation of deoxygenated blood then finally **black** (if irreversible damage).

➢ **Pulselessness :** below the level of occlusion.

➢ **Parasthesia :** cutaneous hyposthesia progresses slowly to be frank anesthesia.

➢ **Paralysis :** due to muscle ischemia.

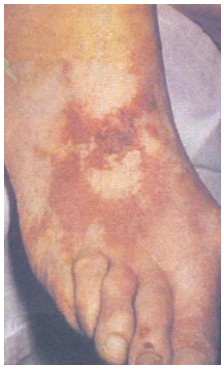
➢ **Progressive coldness :** decreased energy production due to cellular hypoxia.

N.B.: Irreversible lower limb ischemia :

(1) Fixed color changes " **blue staining** ".

(2) Signs caused by **muscle necrosis**.
- tense calf.
- fixed plantar flexion of the foot.
- bulging anterior leg compartment.

(3) **Acute paraplegia** may occur in case of saddle aortic embolism.



COMPLICATIONS

(Pathological sequelae)

- **2ry distal thrombosis :** after circulatory arrest → wide spread of distal intravascular thrombosis
- **Peripheral oedema :** due to wide spread D.V.T of the affected limb, also the clot may dislodge after revascularization → ↑ risk of pulmonary embolism.
- **Nerve ischemia :** impaired nerve conduction due to ischemia of vasa nervosa.

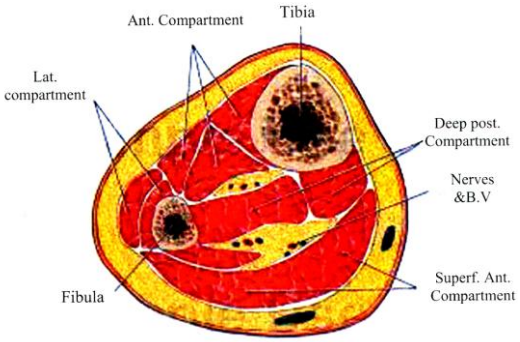
N.B.: Nerve damage never occur

- **Muscle :**

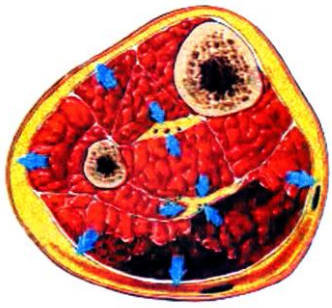
[**Compartmental syndrome**]

Muscle oedema that result from prolonged ischemia → ↑ pressure inside the fascial compartments of the leg especially after revascularization → more ↑↑ ischemia.

N.B.: Irreversible muscle damage within **6 - 8** hours



Normal cut section of leg



After revascularization

- **Skin :**

Gangrene occurs if the condition is neglected (moist gangrene)

N.B.: Gangrene occurs within **24** hours

Incomplete recovery

leads to development of clinical picture of chronic ischemia from inadequate blood flow.

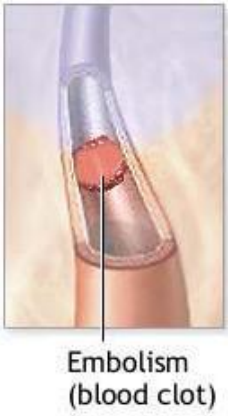
1- Arterial Embolism

Embolic ischemia

DEFINITION

Sudden impaction of an embolus in a narrow blood vessels.

PATHOLOGY see before +



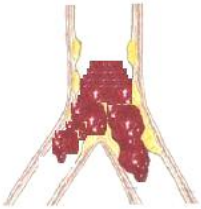
A. Source of an Embolus

(90 % from the heart) due to ⇘

1. Lt Atrium : MS with AF.
2. Lt Ventricle : after recent myocardial infarction
3. The aorta : from an aneurysm.
4. Valves : If subacute bacterial endocarditis (S.B.E)
5. Paradoxical embolism : (rare) If A.S.D or V.S.D.
6. Atheromatous plaque from atheromatous vessels

B. Site of impaction

(bifurcation of vessels) due to ⇘



1. Decreasing in diameter.
2. Slowing in blood circulation.
3. Turbulence of blood flow.

CLINICAL PICTURE see before **[6Ps]** +

Examination of heart may reveal the cause.

COMPLICATIONS see before +

gangrene is more common because of poor collaterals due to ⇘

1. Reflex V.C of collaterals.
2. 2ry thrombosis including collaterals.
3. Showers of embolism may block collaterals.

D D

Acute arterial thrombosis i.e. Thrombotic ischemia

	Embolic ischemia	Thrombotic ischemia
✪ **Personal history** ➢ age	● common with **young**	● common with **elderly**
✪ **Present history** ➢ onset ➢ embolus	● sudden. ● **present**	● gradual. ● absent
✪ **Past history** ➢ heart disease ➢ claudication	● **present**. ● absent	● absent ● **present**
➢ **Trophic changes**	● absent	● **present**
➢ **Radial pulse**	● **irregular** with A.F.	● regular
➢ **Angiography**	● **minimal** collaterals	● **marked** collaterals

INVESTIGATIONS

(A) **ECG & Echocardiography** for evidence of valvular heart disease.

(B) **Doppler U/S & duplex scanning** to detect the level of obstruction.

(C) **Arteriography :** but may cause a delay for 2-3 hours, therefore it's **not done** in a threatened limb.

(D) **Laboratory studies :** Acidosis & ↑ C.P.K indicate extensive muscle necrosis.

TREATMENT

A- Urgent Embolectomy

✪ **Anaesthesia :** better under local anesthesia (may be spinal anesthesia).

✪ **Pre-operative :** Immediate heparin start with 5000 – 10000 IU then 5000 IU every 2 hours to prevent propagation of thrombosis.

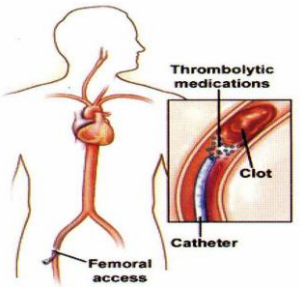
✪ **Operation : [Fogarty balloon catheter]**

- **It must be within 6 - 8 hours to save the limb.** but if done after that time it will be called delayed embolectomy
- **The value of delayed embolectomy** is lowering the level of amputation because of irreversible muscle damage which has occurred.
- **Complications of embolectomy** & **its treatment**

COMPLICATIONS	TREATMENT
(1) **Sudden death** due to pulmonary embolism, 2ry to detachment of a large thrombus.	1. **Thrombectomy** of nearby vein at same time of embolectomy. 2. Putting **I.V.C filter**
(2) **Compartmental syndrome** See before	● **Fasciotomy** is done to save the vitality of the limb i.e. ↓ tension.
(3) **Reperfusion injury** of kidney & heart due to passage of large amount of **myoglobin** & K^+ from ischemic muscle → renal shutdown or cardiac arrest	1. I.V **mannitol** to induce diuresis. 2. I.V **insulin + glucose** to stimulate intracellular shift of K^+ 3. **dialysis** if anuria developed.

B- Fibrinolysins

- **Streptokinase** & **T**issue **P**lasminogen **A**ctivator (**T.P.A**).
- They dissolve an acute thrombosis without surgery.
- They are given through a catheter.
- **Heparin** should not be used at same time.



C- Amputation If established gangrene.

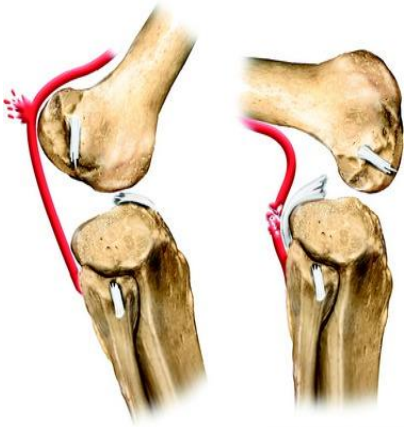
2- Arterial injuries

DEFINITION

Sudden interruption of arterial supply of the limb by injury

INCIDENCE

Common nowadays due to ⇘
[car accidents, war injuries
& iatrogenic as invasive investigations]



AETIOLOGY

➢ **Open injury :**
- Stabs, bullets or iatrogenic following arterial canulation
- **Intra-arterial drug injection** (see later)

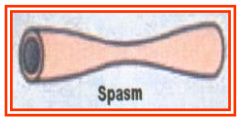
➢ **Closed injury:**
- Direct : ① Plaster or tourniquet compression.
② Road traffic accidents.
- Indirect : Fracture or dislocations of bones.
e.g. Supra-condylar fracture humerus with **brachial** artery injury,
or Supra-condylar fracture femur with **popliteal** artery injury,

TYPES → ARTERIAL INJURIES

without division (No bleeding)

A. Arterial spasm

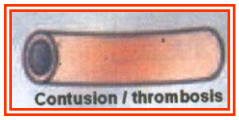
- Due to arterial irritation by missile near the artery or canulation.



- Associated with intimal tear but not diagnosed as a cause except after exploration of the artery.

B. Arterial contusion

- The outer coat remain intact



- If associated with intimal tear ± superadded thrombosis.

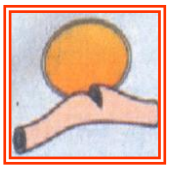
with division (bleeding)

A. Complete division

- Both ends bleed profusely but soon the bleeding decreases because of ⇘
  - intima curls.
  - media contracts.
  - divided stumps retract.
- The pulse is **lost** distally.

B. Partial division

- The bleeding will continue → **(pulsating haematoma)** the bleeding does not stop because of contraction & retraction of arterial wall.
- The pulse is **weak** distally



CLINICAL PICTURE

- **History** of trauma
- **General Exam**
  as vital signs to evaluate the blood loss.
- **Local Exam :**

  (A) **Hard signs :** see before **[6Ps] +**
  external bleeding, pulsating or expanding hematoma.

  (B) **Soft signs :** Less specific (**equivocal**) signs
  small or moderate sized not pulsating & not expanding hematoma.

INVESTIGATIONS

- **In patients with hard signs :**
  Immediate surgical exploration is indicated without investigations.
- **In patients with soft signs :**
  (1) **Plain X ray** to detect foreign bodies (bullets) or fractures.

  (2) **Doppler & duplex** to detect collaterals.

  (3) **Angiography** (the most accurate)
  to localize site of traumatic A/V fistula

TREATMENT

A- First aid treatment

- **Temporary control of bleeding :**
  by external compression or elevation or tourniquet of the limb
- **Anti-shock** measures, **antibiotics** & **analgesics**.
- **Associated fractures** must be fixed to stabilize the repair

B- Definitive treatment

Immediate exploration & **fasciotomy**
to prevent (compartmental syndrome)

Then according to type of arterial injury

1. ARTERIAL INJURY WITHOUT DIVISION :

[A] Arterial spasm :

Intra-arterial injection of papaverine.

- ☞ **If not successful :**
  forcible dilatation by **Fogarty catheter** is performed.
- ☞ **If not successful :**
  Arteriotomy to detect intimal tear. If present →
  excise the spastic segment then graft is performed.

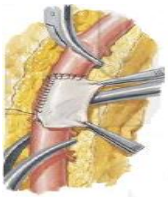
[B] Arterial contusion :
excision of contused segment then graft is performed.

2. ARTERIAL INJURY WITH DIVISION :

[A] Complete division:

- **No gap :** direct end to end anastomosis.
- **Gap :**
  - ***Small*** *artery :* natural (saphenous vein) graft.
  - ***Large*** *artery :* synthetic (dacron or teflon) graft.

[B] Partial division :



- **Longitudinal tear :**
  - ***Small*** *artery :* vein patch graft.
  - ***Large*** *artery* : direct suture.
- **Transverse tear :**
  - ***< 1/2*** *circumference :* direct anastomosis
  - ***> 1/2*** *circumference :* turn it to complete then treat.

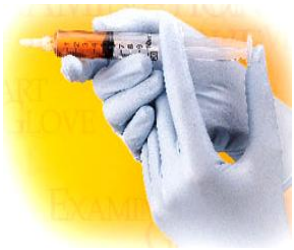
N.B.: 1- Damaged main vein should be repaired

2- Damaged nerves can be repaired or left for another setting

.....

Intra-arterial drug injection

AETIOLOGY



1- **Drug addicts** may wrongly inject drugs into arteries instead of veins .

2- **Anesthetists** may also wrongly inject thiopentone into an artery during induction of anesthesia .

TYPE OF ARTERY

The most commonly punctured arteries are **brachial** & **radial** arteries.

CLINICAL PICTURE

- **Burning discomfort**
  extending from the point of injection to the tips of the fingers
- **Coldness** & **cyanosis** soon follow.
- **Digital gangrene** may then develop .

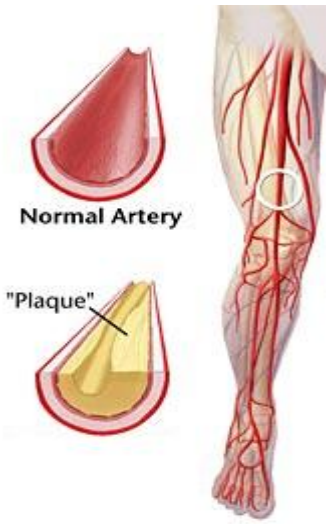
TREATMENT

- **Heparin** 10.000 IU intravenously
  to prevents thrombosis of small vessels.
- **Dexamethazone** 4 mg I.V / 6 hours
  to limits oedema
- **Low molecular weight dextran** (dextran 40)
  to minimizes platelet aggregation.
- Strong **analgesics**

II - CHRONIC ISCHEMIA

DEFINITION

Slowly progressive arterial obstruction that gives enough time for collaterals to develop and, therefore gangrene does not occur rapidly.



AETIOLOGY

[A] Above 45 years :

Atherosclerosis is the commonest cause .

[B] Below 45 years :

- In non diabetics :
  - I. In Males : **Buerger's** disease
  - II. In Females : **Raynaud's** disease
- In diabetics : **Diabetic** foot & gangrene.

I- Atherosclerosis

DEFINITION

Degenerative arterial disease due to aging process affecting the whole arterial system.

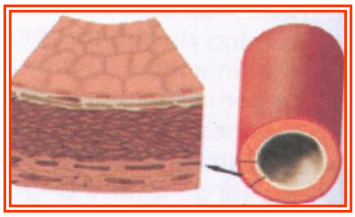

Atherosclerosis

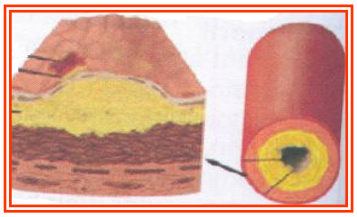
INCIDENCE

- **Age :** > 45 years.
- **Sex :** male > female
- **Risk factors :**
  - hypertension
  - hypercholesterolemia
  - hyper triglycerides.
  - obesity
  - +ve family history.
  - heavy smokers

PATHOLOGY

- **It is a generalized disease :**
  affecting large & medium sized vessels.
- **The 1ry pathology is called " Atheromatous plaques "**
  ① started as an elevated yellow plaque on the intimal surface of the artery.
  ② subintimal layer shows accumulation of lipid & C.T matrix.
  ③ the media & adventitia shows flbrosis.


Normal

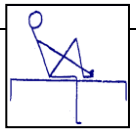

Atheroma

CLINICAL PICTURE " Press And See How Colour Fades "

- **P** (**P**ain)
- **A** (**A**rterial pulsation)
- **S** (**S**kin trophic changes)
- **H** (loss of **H**otness)
- **C** (**C**olour changes)
- **F** (**F**unctional changes)

P = PAIN

	Intermittent claudication " Muscle ischemia "	**Rest pain** " Nerve ischemia "
➢ **Characters**	• **Cramp** like pain	• **Burning** pain
➢ **Site**	• **Aorto-iliac occlusion** → both buttocks ± thighs ± calves • **Ilio-femoral occlusion** → thigh ± calf • **Femoro- popliteal occlusion** → calf ± sole	• **Foot (dorsum > sole)** because of dorsum of foot less vascular than sole, so more affected than sole.
➢ ↑ **by**	• Walking	• Elevation & warmth
➢ ↓ **by**	• Rest	• Lowering or uncovering the limb or rubbing the dorsum of foot.



***N.B. : Intermittent claudication pain** also characterized by :*

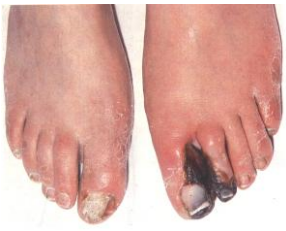
- **Claudication distance :** distance after which the pain is felt.
  the shorter the distance is the more the ischemia.
- **Claudication time :** time after which the pain is felt.
  the shorter the time is the more the ischemia.
- **Rest time :** time of rest needed to start walking again.
  the longer the time is the more the ischemia.

A = ARTERIAL PULSATION

- pulse below the level of obstruction are absent. They may be weekly felt at rest & disappear on exercise (**disappearing pulse**)
- the wall of the artery may be felt rigid from calcification.
- **bruit** can be heard if arterial stenosis is present.

S = SKIN TROPHIC CHANGES :

- loss of hair, brittle nail & dry scaly skin.
- inter-digital infection, i.e. tinea pedis & tapering digits .
- ischemic ulcers over pressure sites e.g. ball of big toe, heal & tips of toes. finally **dry** gangrene.



H = COLDNESS (LOSS OF HOTNESS)

- examination of temp. should be bilateral starting with normal limb.
- before examination of temp, let the limb uncovered for 5 min. to avoid false covered warm limb.

N.B.: Causes of false warm ischemic limb :

1. covered limb.
2. infected limb.
3. under ttt by sympathectomy
4. D.M. "Auto-sympathectomized patient"

- detect the level of change of temp **= level of obstruction**. by palpation from distal to proximal

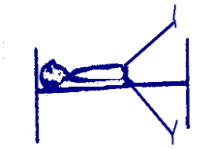
C = COLOUR CHANGES :

[The patient is lying down & exposing his both LL from groin downwards]

- Normal colour indicate → **Mild** ischemia.
- Postural changes indicate → **Moderate** ischemia.

✪ **Bureger's test**

- ➢ Normally, limb is not affected by elevation.
- ➢ Elevation of ischemic limb causes **pallor**
- ➢ Lowering of ischemic limb causes **cyanosis**

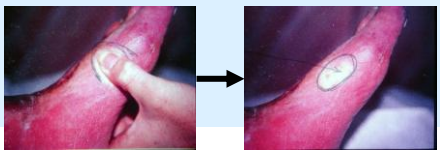


N.B.: Bureger's angle : It is angle at which. limb becomes pale on elevation from horizontal.
So the smaller the angle is the more the ischemia.

- Fixed colour indicate → severe ischemia **=** Pre-gangrene.

N.B.: Capillary circulation test : (press on nail bed 2 sec)

- **Normally :** blanching then rapid return to normal colour.
- **If delayed :** ischemia.
- **If no return :** gangrene.



N.B.: Harvey's venous refilling time :

- **Elevate** the limb till vein empties then allow dependency and record time of filling veins.
- **Normally :** 10 -15 sec.
- **IF** - **mild** =15 -30 sec.
  - **moderate** = 30 -120 sec.
  - **severe** = >120 sec. i.e. > 2 min. **=** Pre-gangrene

F = FUNCTIONAL CHANGES :

✪ **Motor disturbance**

- Gradual atrophy & loss of strength of the muscles.

✪ **Sexual disturbance** = Impotence **[Le Riche syndrome]**

- Aorto-iliac block with occluded both internal iliac arteries
  → occlusion of median sacral artery which supplies nerve erigent ($S_{2\&3}$) which is responsible for erection.

✪ **Systemic disturbance**

- Anginal pain = coronary atherosclerosis.
- Transient ischemic attacks = cerebral atherosclerosis.
- Abdominal pain after meal = mesenteric arteries atherosclerosis.
- Renal pain = renal artery atherosclerosis.

N.B.: Clinical tests to determine the degree of ischemia :

- **Degree of pain :**
  ① Claudication time.
  ② Claudication distance.
  ③ Rest time.
- **Colour changes :**
  ① Bureger's test & angle.
  ② Capillary circulation test.
  ③ Venous refilling time.

N.B.: Clinical tests to determine the level of ischemia :

- **Site** of claudication pain.
- Level of **absent pulse.**
- Level of **coldness.**
- **Le Riche** syndrome**.**

CLINICAL STAGING OF CHRONIC ISCHEMIA

Stage I Asymptomatic

Stage II Intermittent claudication

Stage III Rest pain

Stage IV Ulceration or gangrene

INVESTIGATIONS

I- Laboratory investigations

1. Blood picture for anaemia.
2. Fasting blood sugar & glycosylated hemoglobin.
3. Kidney function tests to exclude renal insufficiency.
4. Serum lipid (cholesterol, triglycerides & lipoproteins)

II- Radiological investigations

1. ARTERIOGRAPHY

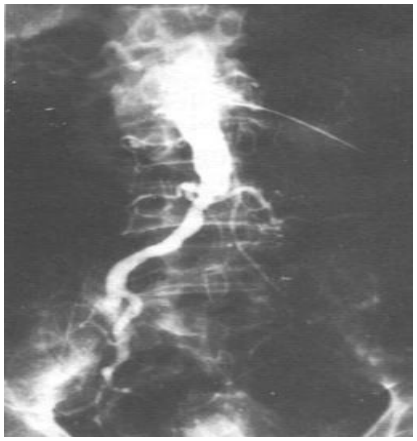
- **Indication :**
  It is done only if direct arterial surgery is considered.
- **Values :**
  shows ① State of the vessel wall.
  ② Site & length of obstruction.
  ③ Collateral circulation.
  ④ Distal **Run off**
  i.e. distal flow beyond the occlusion.
- **Methods :**
  ① Direct trans-femoral arterigraphy.
  ② Direct trans-lumbar aortography.
  ③ Ante-grade trans-brachial aortography.
  ④ Retro-grade trans-femoral aortography.

N.B.: The needle which is used is called **seldinger needle**

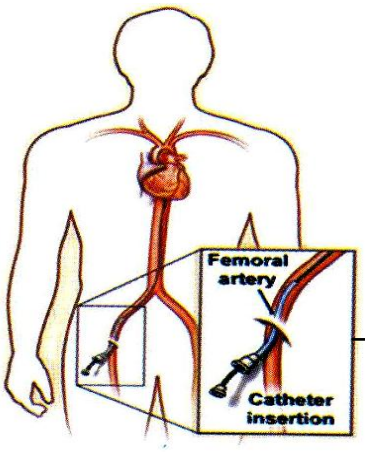
- **Hazards :**
  ① Dissecting aneurysm.
  ② Haemorrhage.
  ③ Spasm & thrombosis.
  ④ Dislodgement of plaques i.e. embolism.
  ⑤ Sensitivity to the dye.

. complete occlusion of Lt .iliac arteries

. occluded Rt. external iliac artery



Trans-lumbar Aortography

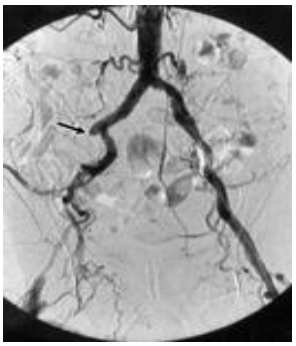


Trans-femoral Arteriography

. complete occlusion of Superficial femoral artery

2. DIGITAL SUBTRACTION ANGIOGRAPHY [D.S.A]

- **Less** Invasive.
- **X-ray picture** is taken & the image is introduced to the computer.
- **The dye** is injected I.V to reach the heart then to the arteries.
- **Further X-ray picture** is taken & the image is introduced to the computer.
- **By subtracting** the 1st image from the 2nd an arteriogram can be obtained.



blocked Rt. external iliac artery (arrow)

3. COMPUTED TOMOGRAPHIC ANGIOGRAPHY [C.T.A]

- It can give **good quality** imaging of arteries

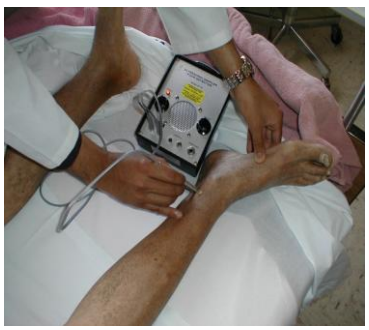
4. MAGNETIC RESONANCE ANGIOGRAPHY [M.R.A]

- It can visualize arteries **without** injection of any contrast

III- Instrumental investigations

1. ECG & ECG STRESS TEST (**demonstrating**)

- The coronary artery disease.
- The condition of myocardium.



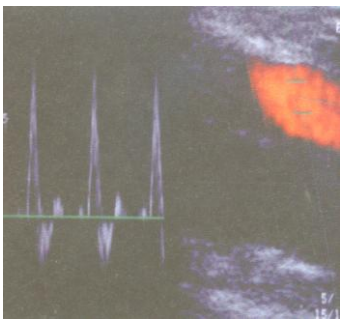
2. DOPPLER ULTRASOUND

- **It is an ultrasonic waves** directed to the vessel. their reflection by the moving R.B.Cs can give an idea about the flow pattern & the pattern of an artery.

- **To detect :**
  1- Stenosed or occluded segments.
  2- Post-stenosed or post-occluded segments.
- **To measure (Ankle / Brachial pressure index)**
  which is the ratio between pressure in both ant. tibial artery & brachial artery which is normally **> 1**.

So
- **If < 0.9 =** mild to moderate ischemia .
- **If < 0.7 =** severe ischemia .
- **If < 0.3 =** impending gangrene .

3. COLORED DUPLEX



The most important investigation

- It combines the benefits of **Doppler u/s** & **colored images** of vessels
- **Colored image** shows
  1. blood flow.
  2. degree of ischemia.
  3. site of obstruction
  4. state of collaterals

TREATMENT

A- Conservative (**No** Rest pain + distal Run off)

B- Endovascular surgery

- Percutaneous trans-luminal balloon angioplasty (**P.T.A**)
- Arterial **stent.**
- **Laser** angioplasty.

} **(See later)**

C- Operative

(Rest pain)

Distal Run off

(Arterial reconstruction)

- **Thrombo-end arterectomy**
- **Arterial by-pass**
  - **Fit** → **Anatomical by-pass**
  - **Unfit** → **Extra-anatomical by-pass**

No Distal Run off

(Indirect arterial surgery)

- **Sympathectomy**
  - **Fit** → **Surgical**
  - **Unfit** → **Chemical**
- **If gangrene & toxemia** → **Amputation**

A- Conservative treatment

INDICATIONS

Mild Ischemia (**No** Rest pain & distal Run off **)**

METHODS

A. Relief of symptoms

1. Improve general health & tissue oxygenation :

- correction of anaemia & any associated lesion e.g. heart lesion.

2. Protection of ischemic parts :

- carefully wash, dry & powder ischemic parts with use of suitable shoes.
- nails are cut continuously.

3. Improve blood supply :

- V.D. drugs as **Trental**
- Anti-platelets as **Aspirin** (small dose)

B. ↓ Progression of the disease

1. Stop smoking.

2. Control of D.M, hypertension ….etc

B- Endovascular surgery

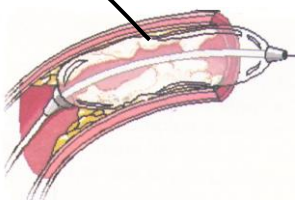
INDICATIONS

Day case surgery (No hospital stay)

1. Suitable only for localized occlusion in a large & medium sized vessels.
2. Suitable for unfit patient for surgery.

METHODS

1. Percutaneous trans-luminal balloon angioplasty (PTA)

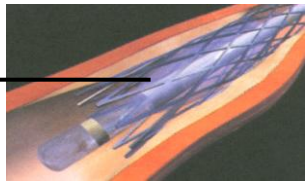


- A special balloon catheter is introduced at site of narrowing of the blood vessel.
- The balloon is inflated to dilate the stenosed segment.

2. Arterial stent

(**After balloon dilatation**)

- A stent is introduced to keep the lumen patent.



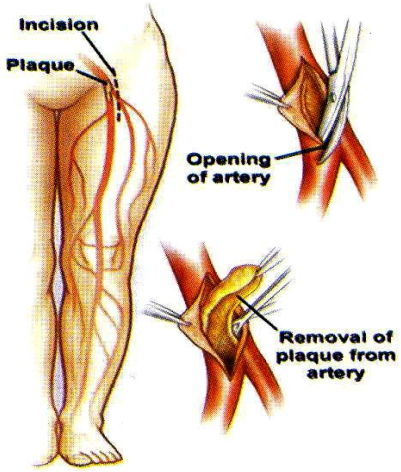
3. Laser angioplasty

- Destruction of the atheromatous plaque by laser.

C- Surgical treatment

1. Arterial reconstruction

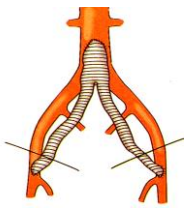
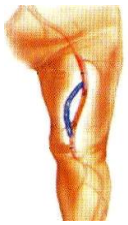
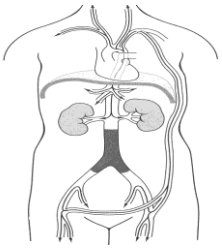
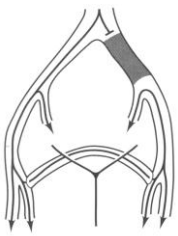
A. Thrombo-end-arterectomy



- **Indications :** 1. Rest pain + distal Run off
  2. **localized** obstruction.
  3. **large** Arteries.
- **Technique :**
  1. **Arteriotomy** then removal of thrombus & thickened atherosclerotic intima leaving a patent lumen.
  2. **Before closing,** the distal intima should be attached to arterial wall by interrupted sutures to prevent it's dissection later on.
  3. **After closing,** the patent lumen will be endothelialized.

B. Arterial by-pass

- **Indications :** 1. Rest pain + distal Run off
  2. **multiple** lesions.
  3. **large** & **medium** sized arteries.
- **Technique :**
  The idea is to by-pass the obstruction by inserting a graft from the health artery above the obstruction to distal run off below

Anatomical by-pass (**Fit patient**)		**Extra-anatomical by-pass** (**Unfit patient**)	
e.g. Aorto- iliac block	e.g. Femoro-popliteal block	e.g. bilateral common iliac block	e.g. unilateral common iliac block
			
Aorta-iliac graft	Femoro-popliteal graft	Axillo-femoral graft	Femoro-femoral graft

- **Types of grafts :**

1. Synthetic grafts e.g. Aorta-iliac graft
- They may be : (a) Teflon or Dacron.
  (b) Polytetra Flouroethylene (PTFE).

2. Natural grafts e.g. Femoro-popliteal graft
- They may be : (a) **reversed** long saphenous vein
  reversed to avoid obstruction by its valves

  (b) **In situ** long saphenous vein
  valves are destroyed by a **stripper**

2. INDIRECT ARTERIAL SURGERY — Sympathectomy

- **Indications :**

1. Vascular conditions

1. Rest pain + **No** Run off (**Buerger's** disease & **Raynaud's** disease)
2. After amputation to help wound healing.

2. Hyperhidrosis Excessive sweating of the hands

- **Contraindications :**
  1. Intermittent claudications
  2. Established gangrene.

- **Principles of <u>lumbar</u> sympathectomy**
  1. Fit **:** (**surgical sympathectomy**)
     - Preganglionic section of L_1 ,L_2 & L_3 ganglia.
  2. Unfit **:** (**chemical sympathectomy**)
     - Para-vertebral block of L_1 , L_2 & L_3 ganglia.
       5% phenol in water is injected beside bodies of $L_{1,2\&3}$ ganglia under screen to destroy these ganglia.

N.B : The 1st. lumbar ganglion should be preserved on one side, otherwise failure of ejaculation occurs

- **Principles of <u>cervicodorsal</u> sympathectomy**
  1. Fit **:** (**surgical sympathectomy**)
     - Preganglionic section of T_2 & T_3 ganglia.
  2. Unfit **:** (**chemical sympathectomy**)
     - Para-vertebral block of T_2 & T_3 ganglia.
       5% phenol in water is injected beside bodies of $T_{2\&3}$ ganglia under screen to destroy these ganglia.

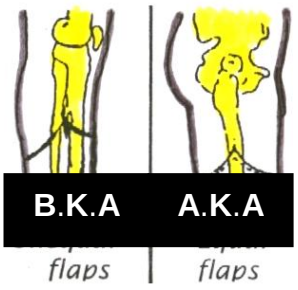
3. AMPUTATION

[I] Conservative amputation

- **Indications :**
  1. If good blood supply to adjacent gangrenous tissues.
  2. If line of separation & demarcation are well defined.
- **Technique :**
  1. Excision at line of demarcation.
  2. The skin should be neither redundant nor undertension.

[II] Urgent high amputation

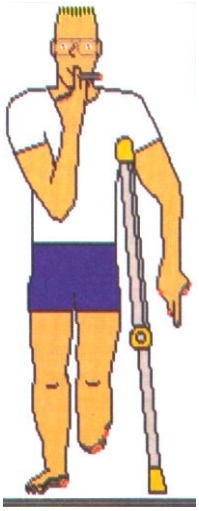
- **Indications :**
  1. If spreading gangrene.
  2. If uncontrolled infection & toxaemia.
- **Technique :**
  1. **Above knee amputation** (**A**.**K**.**A**)
     The stump will be supplied by the profunda femoris.
  2. **Below knee amputation** (**B**.**K**.**A**)
     If popliteal pulse is felt.



ATRERITIS

II- Buerger's disease

Thromboangitis obliterans



DEFINITION

Inflammation & thrombosis of small arteries associated with perivascular fibrosis which blends artery, vein & nerve into one mass, causing early neuritis & severe rest pain.

AETIOLOGY

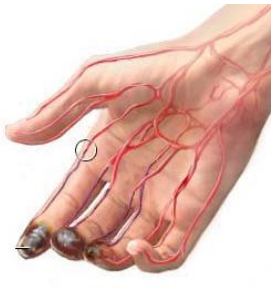
Unknown but may be initiated by spasm.
the spasm is mainly due to **smoking**

D.D

	I. Atherosclerosis	II. Buerger's disease
(1) Incidence	• Common (> 45 years). • Commoner in male with risk factors as D.M…..etc.	• Rare (20 - 40 years). • Only male & exclusively in **smokers**
(2) Pathology	• Atheroma & thrombosis. • Calcification.	• Inflammation & thrombosis • **No** calcification
(3) Clinical picture	• **No** upper limb ischemic symptoms • **No** migrating superficial thrombophlebitis • Calf claudication. • Popliteal pulse (**absent**) • **Late** rest pain & **massive** gangrene • **No** Raynaud's phenomenon	• Upper & lower limb are involved. • Migrating superficial thrombophlebitis. • Sole claudication • Popliteal pulse (**present**) • **Early** rest pain & **limited** gangrene • **Raynaud's** phenomenon.
(4) Investigations ➢ **X-ray** ➢ **Arteriography**	• Calcification. • Irregular narrowing of main arteries with distal run off.	• **No** calcification. • **Not** needed because of distal block i.e. no run off.
(5) Treatment	• Stop smoking → ↓ disease. • Arterial by-pass (the main). • Sympathectomy (**No** Value). • **Urgent high** amputation.	• Stop smoking (the main). • Arterial by-pass (**No** Value). • Sympathectomy (the best). • **Conservative** amputation.

VASOSPASTIC DISORDERS

III- Raynaud's disease



DEFINITION

Vasospastic disease affecting digital arteries.

INCIDENCE

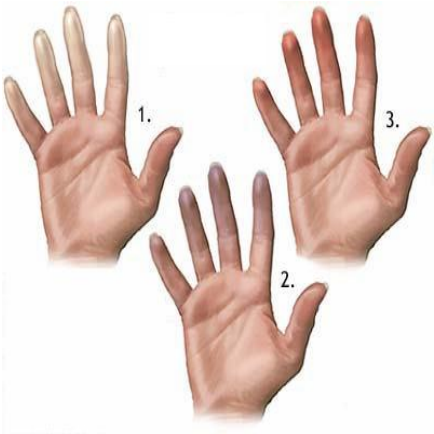
It affects young female, bilaterally & in cold weather.

AETIOLOGY

Arteriolar over-sensitivity to cold, precipitated by presence of cold agglutinins in the blood which cause agglutination of R.B.Cs on exposure to cold

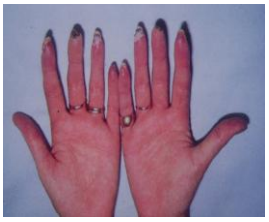
N.B. : the spasm is **not** due to sympathetic over activity

CLINICAL PICTURE



- **During the attacks (3 phases)**
  - **Pallor :** due to arteriolar spasm
    so the pain is **dull aching**
  - **Cyanosis :** due to spasm → ↑ metabolites
    → congestion & cyanosis.
    so the pain is **burning**
  - **Redness :** as attack end & arterioles relax.
    so the pain is **disappeared**
- **Between the attacks:** normal skin & no pain
- **In late cases :**
  - Trophic changes : brittle nail, loss of hair & dry scaly skin
  - Superficial ulcers ± dry gangrene of tips of fingers

GRADES



- **1st** degree : presents only with Raynaud's phenomena
- **2nd** degree : mild trophic changes in tips of fingers & nails
- **3rd** degree : dry gangrene of tips of fingers

MANAGEMENT

1- Conservative treatment successful in early cases.
Vasodilator drugs as Trental or Aspirin (small dose) & avoid cold.

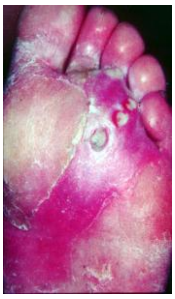
2- Sympathectomy with severe cases.
Good result immediately occur but after few months, mild sensitivity to cold returns. but at least, not severe.

N.B. : **Raynaud's phenomenon** 2ry to
- Occupations using vibrating tools as drills, typists or pianists.
- Associated with certain diseases as bureger, thoracic outlet syndrome & collagen disease as rheumatoid arthritis.
- **Treat the cause** (**No** value of sympathectomy).

IV- Diabetic foot infection & gangrene

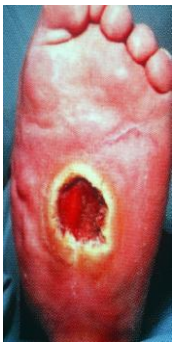
Diabetic infective gangrene

PREDISPOSING FACTORS



Diabetic patients are susceptible to serious foot infections & gangrene due to ⇘

- **Vascular affection :**
  - Major vessels
    as atherosclerosis i.e. diabetic macro-angiopathy.
  - Minor vessels
    as arteritis i.e. diabetic micro-angiopathy.



- **Peripheral neuropathy :**
  - This makes the patient unaware of injuries.
    & also makes him neglect the trophic changes.
- **Infection :**
  - Favored by ↑ blood sugar which acts as good media for infection & If severe → moist septic gangrene.

AETIOLOGY

may be due to one of the followings :

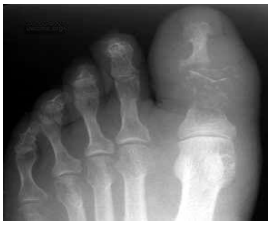
- Pure **ischemia**
- Pure **neuropathy**
- Pure **infection**
- **Mixed** : there is severe infection in addition to major vascular & neuropathic affection

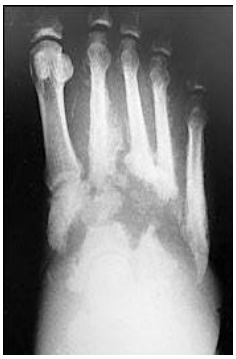
CLINICAL PICTURE

- **History** of minor trauma to diabetic foot.
- **Foot** infection, offensive odour & black discoloration if gangrene.
- **Complication** as chronic osteomyelitis (if infection reaches the bone)

INVESTIGATION

- Urine analysis & blood sugar curve for **D.M**
- Culture & sensitivity for **discharge**.
- **X-rays** on foot for **osteomyelitis**.
- **MRI** on foot for condition of **soft tissues**.





MANAGEMENT

1- Prevention

1. Proper control of D.M
2. Careful trimming of toe nail.
3. Avoidance of walking bare footed.
4. Avoidance of tight shoes.
5. **Daily foot care :**
   washing, drying, powdering & inspecting them for wounds or interdigital infection.

2- Active treatment

Diabetic foot infection is not successfully controlled except after draining of pus

1. Draining of pus.
2. Proper control of D.M
3. Antibiotics according to culture & sensitivity test.
4. Excision of gangrenous parts.
5. Glycerin magnesia dressings → adsorb water → dryness of the infected part + help separation of necrotic tissues.

3- Treatment of complications

- **Conservative** amputation if osteomyelitis.
- **Urgent high** amputation if marked gangrene & toxaemia.

.....

Gangrene

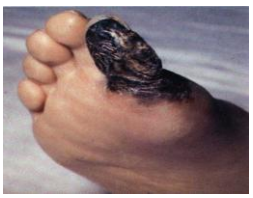
DEFINITION

Macroscopic death & putrefaction of gross part of tissues

AETIOLOGY

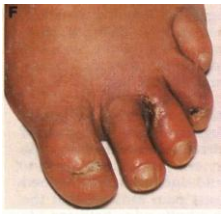
1. **Arterial** gangrene :
   i.e. acute or chronic ischemia.
2. **Venous** gangrene : massive ilio-femoral thrombosis
   i.e. phlygmasia cerulae dolans
3. **Neuropathic** gangrene : leprosy or D.M
4. **Infective** : - specific : as gas gangrene
   - non specific : as carbuncle
5. **Traumatic** : - direct as bed sore
   - indirect as injury of a main vessels.
6. **Physiochemical** : as burn or frost bite.

N.B.: The commonest 2 causes are :
diabetic foot then ischemia of lower limb

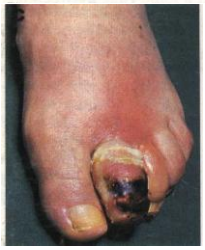


CLINICAL TYPES

- **Dry gangrene :**
  - occurs with chronic ischemia.
  - characterized by : dry, shrunken in size, wrinkled in shape, hard in consistency & no odour.

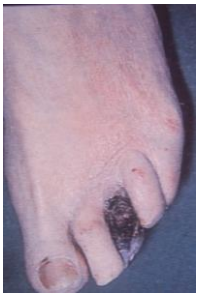


- **Moist aseptic gangrene :**
  - occurs with acute ischemia
    i.e. reflex spasm of nearby vein.
  - characterized by : swollen in size, mild toxaemia, offensive odour **without** pus & gangrene spreads **rapid**.



- **Moist septic gangrene :**
  - occurs with infected gangrene i.e. diabetic foot
  - characterized by : swollen in size, marked toxaemia, offensive odour **with** pus & gangrene spreads **very rapid**.

CLINICAL PICTURE **THREATENED GANGRENE** are



The sentence **" Press & See How Colour Fades "** is a good reminder of these signs.

1. Loss of **P**ulsation .
2. Loss of **S**ensation.
3. Loss of **H**eat.
4. Fixed **C**olor changes.
5. Loss of **F**unction.

TRAUMATIC GANGRENE **BED SORES (DECUBITUS ULCER)**

Aetiology : Pressure over bony prominences.

Commonest sites : ischeal tuberosity, greater trochanters, Sacrum, heels, big toes & scapular blades.

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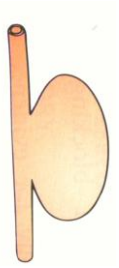
III - ANEURYSM

DEFINITIONS



Pathological definition

Sac filled with blood & communicating with the lumen of an artery.

Practical definition

A permanent localized dilatation of an artery, having at least 1.5 times the normal diameter.

CLASSIFICATIONS

(I) Aetiology of the aneurysm

CONGENITAL

- commonest in circle of willis **(Berry's aneurysm)** → Sub-arachnoid Hge.

PATHOLOGICAL

- **Atherosclerosis** : the commonest cause nowadays
- **$** that affect the aortic arch.
- **Subacute bacterial endocarditis** → infected emboli
- **Collagen disease** e.g. Behcet disease & Marfan's syndrome.

TRAUMATIC

- weakening of the wall of the artery i.e. **True aneurysm**
- interruption of the wall leading to pulsating hematoma i.e. **False aneurysm**.

(II) Structure of the aneurysm

TRUE ANEURYSM the wall of the aneurysm is formed by (**3** layers).

FALSE ANEURYSM the wall is formed of fibrous tissue due to pulsating haematoma.

(III) Shape of the aneurysm

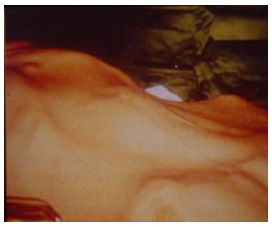
CLINICAL PICTURE may be **silent**

A- General examination

- to detect the cause e.g. $, Atherosclerosis, SBE ...etc

B- Local examination

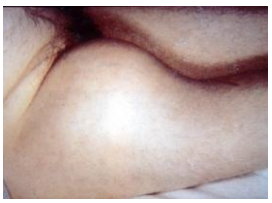
- usually single, along the course of an artery, variable in size & rounded in shape with smooth surface.
- compression on adjacent structures (see complications)



Aortic aneurysm

INSPECTION

- Expansile pulsations are **seen**.
- Proximal pressure **reduces** the size of the aneurysm.
- Distal pressure **increases** the size of the aneurysm.



Femoral aneurysm

PALPATION

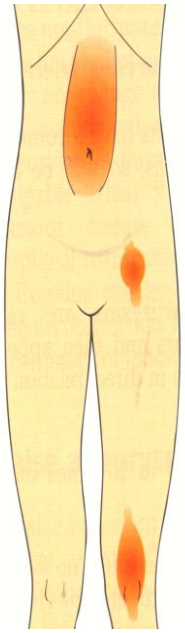
- Expansile pulsations are **felt**
- Moves side to side across but not along the course of an artery.
- Compressible (but absent if filled with a thrombus).

AUSCULTATION Systolic murmur.

COMPLICATIONS

A- Intrinsic complications

1. **Rupture** is the most serious complications.
2. **Infection** → suppuration → rupture → haemorrhage.
3. **Thrombosis** or **embolization**
4. **Distal ischemia** due to
   (a) thrombosis of the aneurysm.
   (b) compression on main artery.
   (c) embolization.
   (d) associated atherosclerosis.



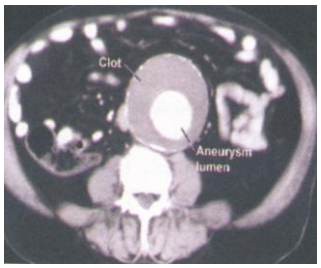
B- Extrinsic complications

1. Compression on **vein**
   → oedema & varicosities.
2. Compression on **artery**
   → ischemia changes.
3. Compression on **nerve**
   → impaired sensation ± paralysis.
4. Compression on **bone**
   → erosion may occurs with abdominal aneurysm.

D.D

- **Transmitted pulsation :**
  which is characterized by ⇩
  not expansile, pulsation disappears if you push it away from the artery & does not ↑ in size if you compress the artery distally
  e.g. pancreatic pseudo-cyst.
- **Pulsating tumor :** e.g. Vascular sarcoma.
  irregular in shape, ill defined, not compressible & does not overlie the line of main artery.
- **A-V fistula :**
  which is characterized by ⇩
  machinery (continuous) murmur, tachycardia & +ve Branham's sign.
- **An abscess**.

INVESTIGATIONS



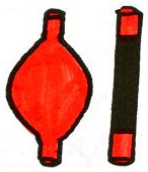
- **Duplex scanning** is very useful.
- **C.T. scan** is the choice.
- **Arteriography** useless if clotted aneurysm.
- **Plain X-ray** for calcified plaques or eroded bone.

TREATMENT

Aneurysms are liable to rupture.
so any aneurysm should be **treated surgically**.

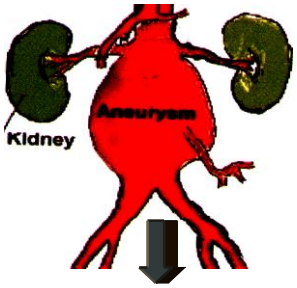
1. The classic treatment

Excision + graft



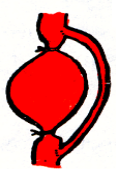
2. Aortic aneurysm > 5 cm

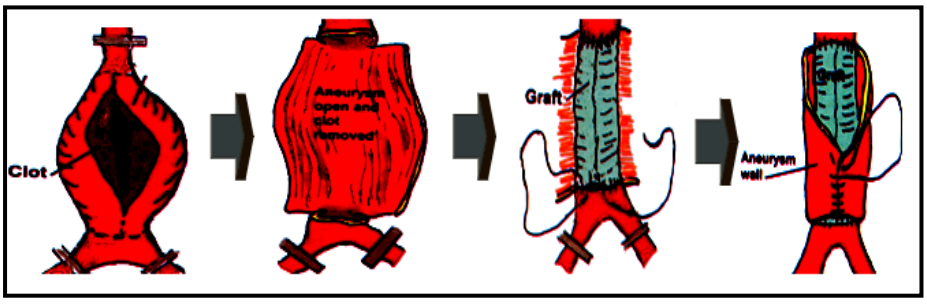
Graft inserted inside the aneurysmal sac



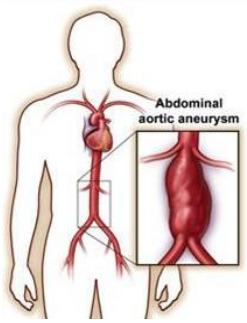
3. If surrounded by important structures

Exclusion + by-pass graft





Abdominal Aortic Aneurysm (AAA)



AETIOLOGY

- Atherosclerosis is the commonest cause (95%)

CLINICAL PICTURE As general **+**

Types of rupture If **> 5 cm**

1. **Intraperitoneal rupture (20 %)** which is fatal .
2. **Retroperitoneal rupture (80 %)** the patient can be saved .

Symptoms of rupture classic triad of (AAA)

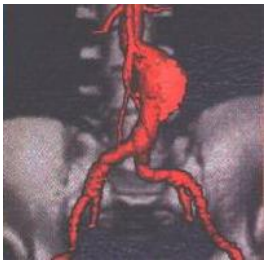
1. Acute upper abdominal **pain** : which is present in flanks.
2. **Pulsatile** abdominal **mass** : which is usually tender.
3. **Shock** : which is present at time of rupture.

COMPLICATIONS

Rather than rupture, the aneurysm can erodes the spine these may be wrongly diagnosed as lumbar disc prolapse.

D.D Pancreatic pseudo-cyst (see before)

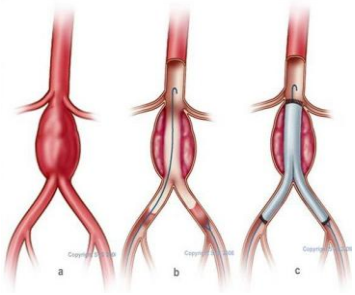
INVESTIGATIONS



- **Ultrasound :** the screening investigation of choice
- **Spiral (3D) C.T. scan :** the investigation of choice to diagnose elective & complicated cases.

TREATMENT

- **Regular follow up / 6 months** If **< 5 cm** in diameter
- **Urgent surgery** for complicated cases.



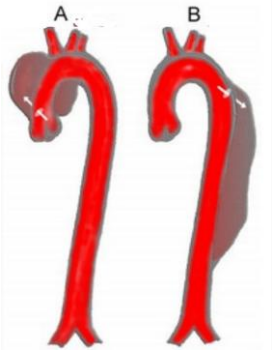
Techniques :

- Open surgery by opening the aneurysm then graft inserted inside the aneurysm
- Endoluminal stented graft through bilateral femoral arteriotomies for high risk patient.

Aortic dissection

AETIOLOGY

- Atherosclerosis & hypertension



TYPES

- **Type A :** Dissection start in **ascending** aorta (more serious)
- **Type B :** Dissection start in **descending** aorta (less serious)

TREATMENT

- **Type A : Urgent surgery** (as above)
- **Type B : Antihypertensive** drugs

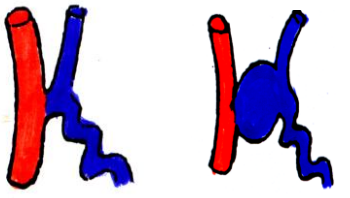
Arterio-venous Fistula

DEFINITION

Abnormal connection between artery & vein.

PATHOLOGICAL TYPES

- **Aneurysmal varix** i.e. direct communication
- **Varicose aneurysm** i.e. false sac communication



Aneurysmal varix | **Varicose aneurysm**

AETIOLOGY

- **Congenital :** local gigantism
  - Usually small & multiple
  - common at lower limb → giant limb.
- **Acquired :**
  - Trauma : stab, bullet... etc
  - Artificial : during haemodialysis in chronic renal failure.

CLINICAL PICTURE

Systemic signs

As tachycardia, water hummer pulse ...etc

Local signs

A small, rounded & pulsating swelling along the course of the vessels.

Distal signs

The veins become dilated, tortuous & pulsating varicose vein.

Auscultation

Machinery (continuous) murmur.

Special test **Branham's sign**

Slowing of heart rate as soon as the fistula is compressed.

COMPLICATIONS

Chronic ischemia, 2ry V.V & if ruptured → bleeding

INVESTIGATION

- **Colored duplex :** diagnostic & non invasive.
- **Arteriography :** will show the site, size & nature of A-V communications.

TREATMENT

According to the cause

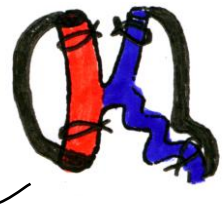
- **Congenital :**

  No surgery except if single (excision + vascular repair)

- **Acquired :**

  Excision + vascular repair by restoration of the continuity of both artery & vein.

  if failed → legate proximally & distally i.e. quadriple ligation. then by-pass graft.



ENDOVASCULAR SURGERY

DEFINITIONS

Endovascular surgery is the management of vascular disease **percutaneously** through a puncture to deal with a lesion in a remote site.

CLASSIFICATION

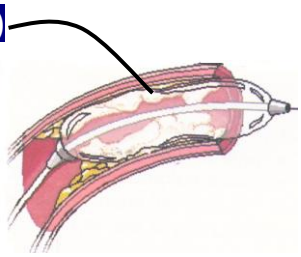
A- System (Arterial or Venous)

B- Purpose (Diagnostic or Therapeutic)

THERAPEUTIC PROCEDURES

A- Arterial

1. Percutaneous trans-luminal balloon angioplasty (PTA)

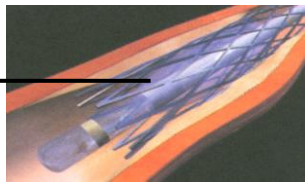


- A special balloon catheter is introduced at site of narrowing of the blood vessel.
- The balloon is inflated to dilate the stenosed segment.

2. Arterial stent

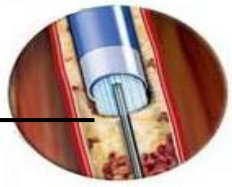
(**After balloon dilatation**)

- A stent is introduced to keep the lumen patent.



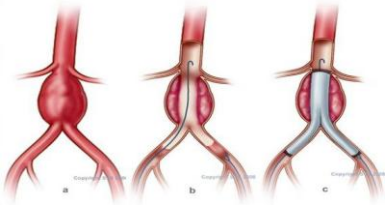
3. Laser angioplasty

- Destruction of the atheromatous plaque by laser.



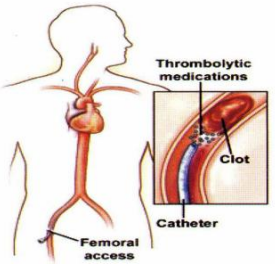
4. Endoluminal stented graft

- For example (treatment of A.A.A) bilateral femoral arteriotomies for high risk patient.



5. Thrombolytic therapy

- **Streptokinase** & **T**issue **P**lasminogen **A**ctivator (**T.P.A**)
- They dissolve an acute thrombosis without surgery.
- They are given through a catheter.
- **Heparin** should not be used at same time.



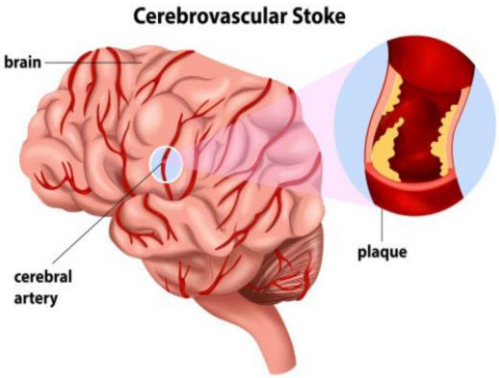
B- Venous

Inferior vena cava filter

- To avoid showers from recurrent D.V.T

EXTRACRANIAL CEREBROVASCULAR DISEASE

AETIOLOGY



- **Embolization** is the commonest cause of TIA's & ischemic stroke

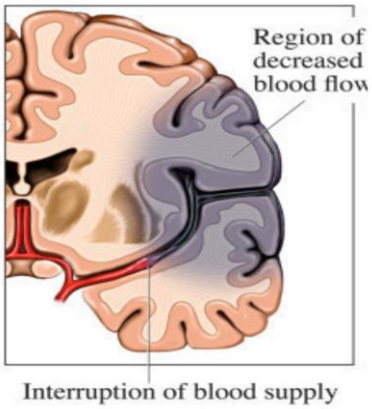
PATHOGENESIS

- **The embolus** may arise from the heart, but more commonly it arises from an ulcerating atherosclerotic plaque at the carotid bifurcation.

CLINICAL PICTURE

May be asymptomatic

1. **Patients with TIA's** (See internal medicine)
2. **Patients with stroke** (Neurologic deficits)



INVESTIGATION

- **Colored duplex :** diagnostic & non invasive.
- **CT scan** of the brain

TREATMENT

- **Surgical :**
  Carotid endarterectomy
- **Medical :**
  1. Control risk factors.
  2. **Antiptatelet** drugs such as aspirin
  3. **Anticoagulation** in patients with cardiac embolic disease.

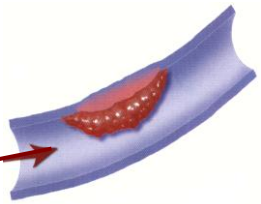
Venous disorders

VENOUS DISORDERS

I. DVT **II. Varicose vein** **III. Venous ulcer**

I. DEEP VENOUS THROMBOSIS

D.V.T



DEFINITION

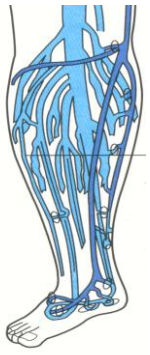
DVT means thrombosis in the deep veins

TYPES

	Phlebothrombosis	Thrombophlebitis
definition	thrombosed **un**inflammed veins	thrombosed inflammed veins
causes	stasis or hyperviscosity	draining inflammed organs
site	common with **leg** veins	common with **pelvic** veins
size of 1ry thrombus	small	large
emboli	common & sterile	rare & infected

INCIDENCE **Risk factors**

- Common (> 50 %) **after operations**
  as post prostatectomy & fracture neck of femur.
- Commonest site is **calf veins**
  because of being valveless & sinusoids.



AETIOLOGY **Vircow's Triad**

1- Changes in vessel wall i.e. endothelial damage.

e.g. trauma to venous wall (during pelvic operations)
or inflammatory process near the vein (pelvic sepsis)

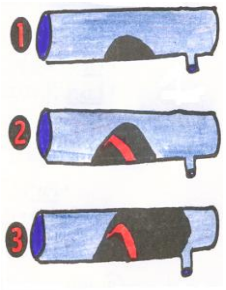
2- Changes in blood flow i.e. venous stasis

e.g. prolonged recumbency as shock or heart failure.
or venous compression by tumor or pregnant uterus.

3- Changes in blood composition i.e. hypercoagulability

e.g. oral contraceptive pills or polycythaemia.
or deficiency of (anti-thrombin III)
or deficiency of protein C & S which are normal anti-coagulant
or deficiency of factor V

PATHOGENESIS



1. The process usually starts in the calf venous sinuses by adherence of platelets to the endothelial surface forming a **grey cluster**.
2. Fibrin & R.B.Cs are deposited as a layer between platelets giving **line of Zahn**
3. When the vein is totally occluded. the **Jelly-like** propagated thrombus spreads upwards as far as the near major tributary.

N.B.: at this stage, **pulmonary embolism** occurs because of loosely attached thrombus.

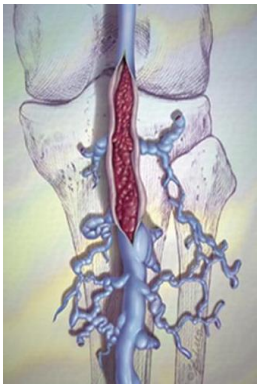
4. The thrombus becomes tightly adherent to the venous wall producing destruction of valves **+** occlusion of lumen **= Post-phlebitic limb**.
5. Later on the process of fibrinolysis & phagocytosis start and help **recanalization** of the vein but the valves are permanently destroyed.

CLINICAL PICTURE

Asymptomatic group

- There are **no** local symptoms & the patient may present later with either pulmonary embolism or post-phlebitic limb.
  However, it may be suspected by the presence of unexplained fever or tachycardia.

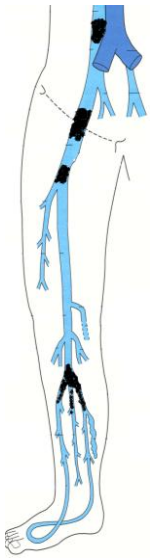
Symptomatic group



- **The classic picture :**
  ① **Pain** : there is usually aching discomfort in the involved calf or thigh.
  ② **Swelling** i.e. oedema
  the most reliable physical sign.
  ③ **Tenderness** on pressing muscles against bone.

N.B.: The level of swelling in D.V.T
differs according to the site of obstruction

So there are [4 possibilities]



1- DEEP CALF THROMBOSIS

- **Site :** affects venous sinuses of calf muscles especially soleus.
- **Signs :** tenderness & tense oedema affecting calf muscles.
  Homan's sign sudden dorsiflexion
  → calf pain by stretching veins

N.B.: Homan's sign (not done)
to avoid spread of thrombus to circulation.

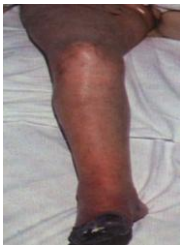
2- FEMORAL VEIN THROMBOSIS

- **Site :** affects calf & lower part of the thigh.
- **Signs :** tenderness affecting calf & distal thigh muscles
  femoral vein is felt as tender & firm cord like in femoral Δ.

3- ILIO-FEMORAL THROMBOSIS

- **Site :** affects all lower limb.
- **Signs :** according to the severity ⇩

	Phlegmasia alba dolans " **Painful white limb** "	**Phlegmasia cerulae dolans** " **Painful blue limb** "
severity	**Partial** venous obstruction **less** severe	**Complete** venous obstruction **more** severe
color changes	**Pallor** (associated arterial spasm **)**	**Cyanosis** (no venous return)
complications	Coldness & ↓ **P**ulsation	Venous gangrene.



4- I.V.C OBSTRUCTION

- **Site :** affects inferior vena cava.
- **Signs :** manifestations affect both legs.
  dilated veins cross the groin.

D.D

1. Contusion of calf muscles.
2. Rupture of plantaris muscle.
3. Other causes of **leg swelling :**
   e.g. local gigantism, A.V fistula, cellulites,
   venous oedema or lymphatic oedema.
4. Other causes of **leg pain :**
   - **A**rterial **=** ischemic pain.
   - **V**enous **=** D.V.T & V.V.
   - **L**ymphatic **=** tender L.Ns.
   - **N**erve **=** sciatica.
   - **M**uscle **=** myopathy.
   - **B**one **=** osteomyelitis.
   - **J**oint **=** osteoarthritis & osteoarthrosis
   - **L**igament **=** flat foot.

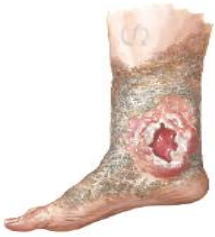
COMPLICATIONS

Early Complications

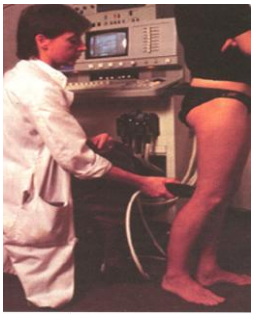
1. **Pulmonary embolism (see Cardiothoracic)**
2. **Venous gangrene** with phlegmasia cerulae dolans .

Late Complications

1. **2ry varicose vein.**
2. **Chronic venous insufficiency** (**Post-phlebitic syndrome**)
   - Cause : It follow ilio-femoral thrombosis
     because of ↑ pressure in deep veins → reflux of blood
     from occluded deep veins to superficial veins
   - Characters :
     ① high pressure in superficial veins occurs during walking
     ② non pitting oedema from fibrosis
     ③ venous ulcer ± its complications as ⇩
     - malignancy " marjoline ulcer "
     - periosteitis If fixed to tibia.
     - talipes equinous.



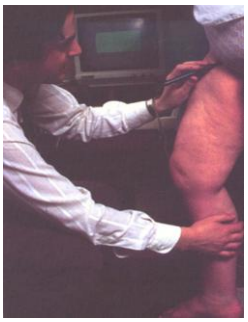
INVESTIGATIONS



1. Doppler U/S :
(accuracy 85 %)

- If the probe is applied over the femoral or popliteal veins & a roar sound (venous hum) is accentuated, this means the veins are patent. but if there is thrombosis the roar does not occur.

2. Colored duplex
(accuracy 90 -100 %)



- The standard test for diagnosis of D.V.T
- It employs color flow imaging which permits determination of flow direction & turbulence & to detects partly occlusive thrombi.

3. Enhanced 3D helical C.T

- The most recent, it shows thrombi even in small veins.

4. Radio-iodine I_{125} labeled fibrinogen (Not used)

- ***before technique*** **:** give KI to block iodine uptake by thyroid gland.
- ***technique*** **:** labeled fibrinogen with I_{125} will be incorporated in the newly forming thrombi which can be detected by scanning over it [so can't detect already formed thrombi]
- ***value*** **:** it can be repeated daily, so that it is useful for follow up.

TREATMENT

A- Prophylactic treatment

MEASURES TO PREVENT STASIS & IMPROVE VENOUS RETURN

- **Before operation :**
  - stop oral contraceptive pills.
  - regular walking.
  - any heart lesion must be controlled.

- **During operation :**
  - galvanic stimulation to calf muscles.
  - pneumatic calf trousers.
  - legs are slightly raised.
- **After operation :**
  - adequate hydration by I.V or oral fluid.
  - early ambulation from bed.
  - leg elevation 15-20 degree.

PROPHYLACTIC ANTICOAGULANTS FOR HIGH RISK PATIENTS

- **Low dose heparin :**
  5000 IU S.C 2 hours before operations & then every 12 hours for 7 days.
- **Low molecular weight (LMW) heparin :**
  It is given once daily & has lower risk of bleeding so more popular.

B- Curative treatment

1- CONSERVATIVE TREATMENT

- **Aim :** to prevent clot propagation, formation of new thrombi & avoid detachment or embolization.
- **Regimen :** 1- leg elevation 15 -20 degree & elastic bandage.
  2- anti-coagulants

Anti-coagulants

Heparin I.V or S.C

- **Action :**
  it acts as co-factor for anti-thrombin III.
- **Methods of administrations :**
  ① **I.V**. bolus therapy (5000 I.U) every 4 - 6 hours.
  the dose is controlled by making **C**lotting **t**imes
  ② **I.V infusion** of glucose 5% containing low molecular weight heparin at a rate of 20 - 30 I.U/Kg/hour after giving initial dose of 5000 IU & the dose is monitored by **A**ctivated **P**artial **T**hromboplastin **T**ime
  ③ **S.C** heparin (**Clexan**) 1mg/kg every 12 hours.
  the dose does not require a blood test for adjustment.
- **Antidote : Protamine sulphate**
- **Complications :** 1- bleeding from overdose
  2- failure to response i.e. heparin resistant.

Oral Anticoagulants (Warfarin)

Warfarin (oral)

- **Action :**
  blocking synthesis of (Prothrombin & factors **VII**, **IX** & **X**)
- **Methods of administrations :**
  10 mg Initially then 5 mg for 5 days.
  the dose is monitored by **P**rothrombin **t**ime & **c**oncentration
- **Antidote : Vit. K.**
- **Complications :** 1- bleeding from overdose
  2- interaction with drugs as aspirin.

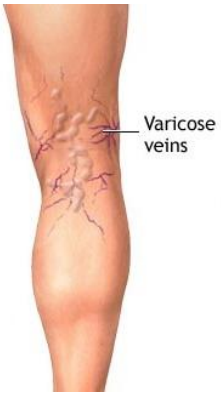
2- FIBRINOLYSINS

- as Streptokinase, Urokinase & Tissue plasminogen activator

3- SURGICAL TREATMENT

- **Venous thrombectomy** with phlegmasia cerulae dolans by Fogarty catheter.
- **I.V.C interruption** by filter insertion through the jugular vein to prevent recurrent pulmonary embolism.

II. VARICOSE VEINS



Varicose veins are multiple, dilated, elongated, tortuous, soft, bluish & compressible veins of superficial veins of lower limb.

Anatomical considerations

Veins of lower limb

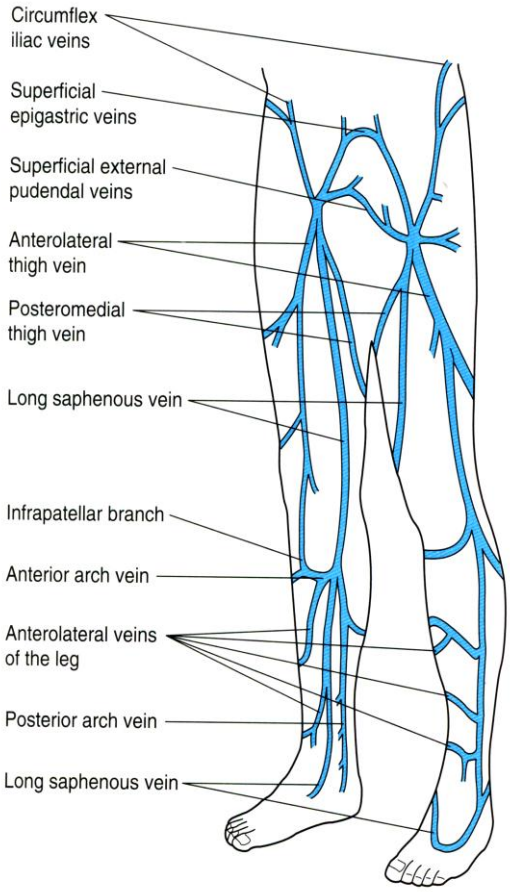
The lower limb is drained by the following venous systems

I- Superficial system (superficial to deep fascia)

It includes ⇘

LONG SAPHENOUS VEIN

It begins at the medial aspect of the dorsal venous arch of the foot and ascends infront of the medial maleolus to the medial aspect of the leg then behind the knee to the inner aspect of the thigh till the saphenous opening (1.5 inch below & lateral to the pubic tubercle) where it arches to join the femoral vein.



✶ **Tributaries of the long saphenous vein**

A. In the thigh :

1. Superficial circumflex iliac vein
2. Superficial epigastric vein.
3. Superficial pudendal Vein.
4. Antero-lateral vein.
5. Postero-medial vein.

B. In the leg :

6. Anterior vein of the leg.

C. In the foot :

7. The dorsal venous arch.

Don't Forget

① **All vein** are containing valves except at soleus muscle.

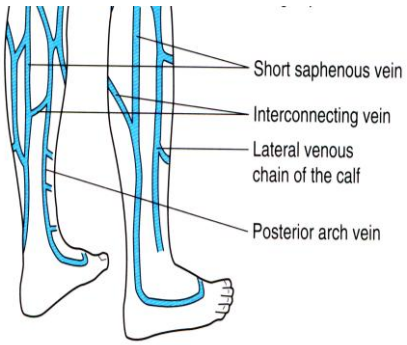
② **Saphena varix :**

- Saccular dilatation at sapheno-femoral junction.
- Saphena **=** clear
- Varix **=** dilatation.

③ **Long saphenous vein** is the longest vein all over the body.

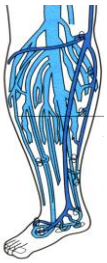
④ **Sapheno-femoral Junction =** Trendlenburg valve.

LESSER (SHORT) SAPHENOUS VEIN



It begins at the lateral aspect of the dorsal venous Arch. ascends below & behind the lateral maleolus to run along the lateral edge of tendo-achilles in the posterior midline of the leg to the middle of popliteal fossa where it pierces the deep fascia to join the popliteal vein.

II- Deep system (deep to deep fascia)



It includes

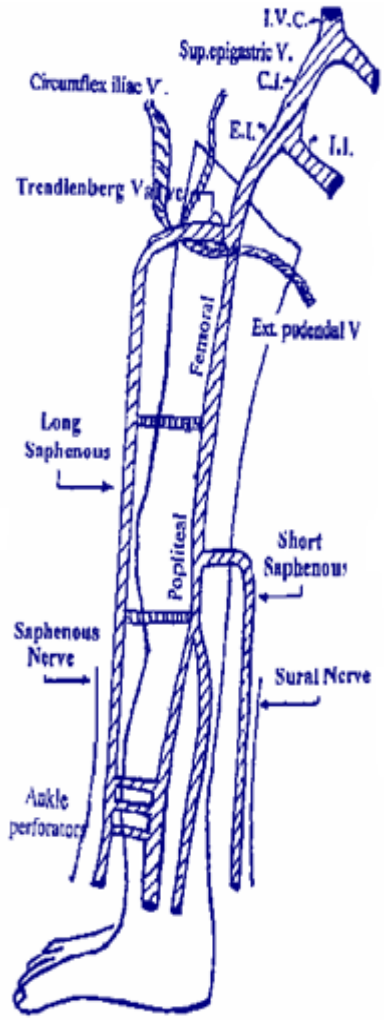
BELOW THE KNEE

They consists of venae commitantes of the arteries + the venous sinuses inside the calf muscles (soleus).

THE LEVEL OF THE KNEE

They unite to form the popliteal vein which ascends to the thigh to become the femoral vein at the adductor canal then passes deep to the inguinal ligament to change its name into the external iliac vein.

III- The connecting system



✪ These veins connect the superficial to deep veins (They have valves which allow a uni-directional blood flow from superficial to deep veins).

✪ **They are either**

DIRECT COMMUNICATORS = PERFORATORS

THE PERFORATORS OF THE LONG SAPHENOUS :

- **3 ankle perforators** (**2**, **4** & **6** inches) above medial maleolus they drain blood directly from the venous plexus of the skin to the deep system.
- **1 perforator** just below the knee.
- **1 perforator** at the mid thigh.
- **The sapheno-femoral Junction.**

THE PERFORATORS OF THE SHORT SAPHENOUS :

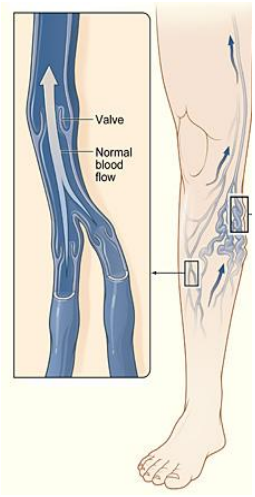
- **1 lateral perforator** (**5** inches) above the lateral maleolus.
- **The sapheno-popliteal Junction.**

INDIRECT COMMUNICATORS

Veins passes from superficial vein to the muscles & another vein passes from the muscles to the deep veins.

Venous pathophysiology

- **blood from the muscles** of the leg returns to deep veins.
- **blood from the skin & superficial tissues** drains via the long & short saphenous veins. then through the connecting system to the deep veins.



SO

1RY VARICOSE VEINS

- The superficial system is weak wall or absent valves or incompetent valves → high pressure (heaviness pain) with standing
- But with walking or exercise → shift of blood from superficial to deep system. so the pain is **decreased**

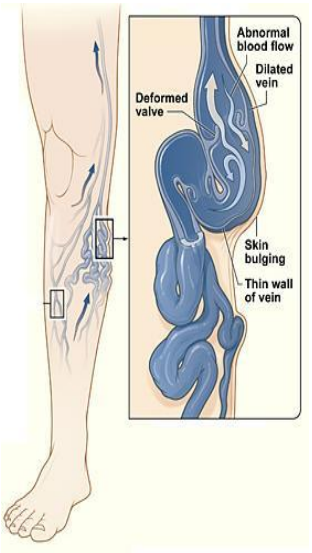
2RY VARICOSE VEINS

The superficial system is **normal** but the deep system is occluded or compressed → high pressure (bursting pain) **not only** with standing **but also** walking & exercise → so the pain is **increased**

CEAP CLASSIFICATION OF VARICOSE VEIN

C STANDS FOR CLINICAL CLASSIFICATION

C0 No visible or palpable signs of venous disease.
C1 Reticular veins less than 3 mm.
C2 Reticular veins more than 3 mm.
C3 Oedema
C4 Skin & subcutaneous changes
C5 Healed venous ulcer
C6 Active venous ulcer



E STANDS FOR ETIOLOGY

En No venous cause
Ec Congenital
Ep Primary
Es Secondary.

A STANDS FOR ANATOMY

An No location identified.
As Superficial veins:
Ap Perforator veins.
Ad Deep veins.

P STANDS FOR PATHOPHYSIOLOGY

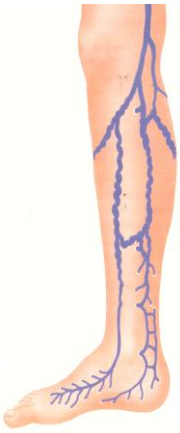
Pn No pathophysiology identified.
Pr Reflux
Po Obstruction
Pr Reflux and obstruction

VARICOSE VEINS

Varicose veins are multiple, dilated, elongated, tortuous, soft, bluish & compressible veins of superficial veins of lower limb.

AETIOLOGY

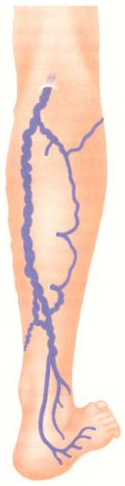
A- 1ry varicose vein due to ⇘



- Congenital weakness of venous wall.
- Congenital absence or incompetent valves.
- This is precipitated by prolonged standing as surgeons, hair dressers, ……etc.
- **Other manifestations of weak mesenchyme :**
  - ① kyphosis ② flat foot.
  - ③ visceroptosis ④ hernia.
  - ⑤ varicocele ⑥ piles

N.B : 1ry V. V is associated with **minimal** complications

B- 2ry varicose vein due to ⇘



- **DVT** (the commonest cause) especially with chronic venous insufficiency i.e. postphlebitic limb.
- **Deep venous compression :**
  - Pelvic or abdominal swellings as pregnancy, tumor ….etc.
- **Arterio-venous fistula**
  - congenital
  - acquired : due to trauma e.g. bullet or stab wound in femoral Δ.

N.B : 2ry V. V is associated with **marked** complications

COMPLICATIONS

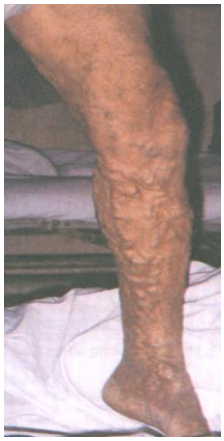
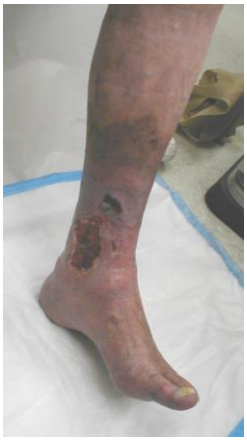
A- Venous complications

- **Hge** from minor trauma.
- **Superficial thrombophlebitis**.

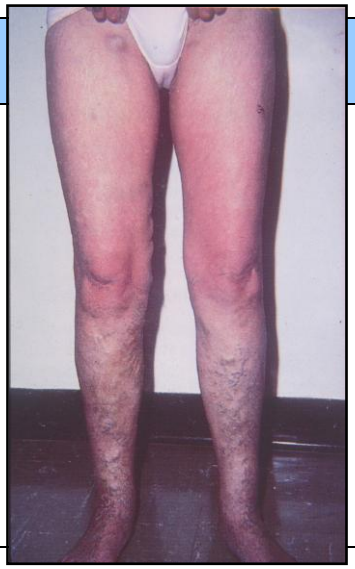
B- Skin complications

- **Brown pigmentation :** by extravasated haemosiderin from ruptured S.C venules.
- **Dermatitis** (redness & itching) from haemosiderin irritation.
- **Eczema** follows scratching of dermatitis.
- **Oedema** involving the lower 1/3 of the leg.
- **Ulceration** due to local hypoxia from venous stasis.
- **Liposclerosis :** S.C fats are replaced by fibrous tissue.

CLINICAL SHEET

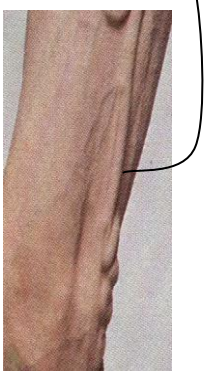
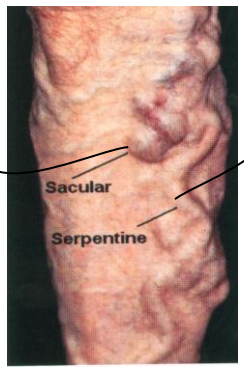
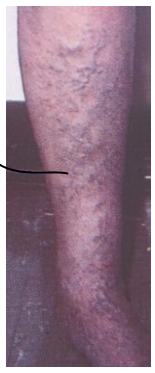
	1ry V.V.	2ry V.V.
Personal history - Age. - Occupation. - Marital status. - Special habits	 • commonly **adult** • surgeons, hair dressers, …etc. • ---------- • ----------	 • commonly **old** • ---------- • multiple pregnancy. • tight corset.
Complaint - Pain	• commonly **bilateral**	• commonly **unilateral**
Present history **1- PAIN** - severity - characters - ↑ by - ↓ by	• **mild** • **heaviness** pain ± **burning** pain due to superficial thrombophlebitis. • ↑ with prolonged standing • ↓ by elevation of the affected limb & by **walking**	• **severe** • **bursting** pain due to D.V.T. • ↑ with prolonged standing or by **walking**. • ↓ by elevation of the affected limb only
2- OEDEMA	• **mild** & appear at evening then resolute after sleep.	• **marked** & persist not related to time.
3- COMPLICATIONS	• **minimal**	• **marked**
4- ASSOCIATED SWELLING	• **groin** e.g. saphena varix • abdomen or pelvis : **no mass**	• **groin** e.g. L.Ns • Abdomen or pelvis : **±** **mass**.
Past history	• **no** history suggest DVT • DM & hypertension …etc	• **+ve** history suggest DVT • pelvic or abdominal mass • trauma (A/V fistula) - bullet. - stab wound • DM & hypertension …etc
Family history	**congenital weak mesenchyme**	----------

GENERAL EXAMINATION

1ry V.V		2ry V.V
Manifestations of weak mesenchyme 1. kyphosis 2. visceroptosis 3. hernia 4. lt. varicocele 5. flat foot & halux valgus		**Look for the cause :** 1. ↑ HR if A/V fistula 2. organomegaly. 3. dilated veins cross groin 4. bilateral varicocele if I.V.C. obstruction 5. tallipus equinous as a complication of ulcer

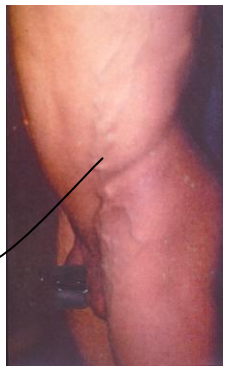
LOCAL EXAMINATION

A- Inspection

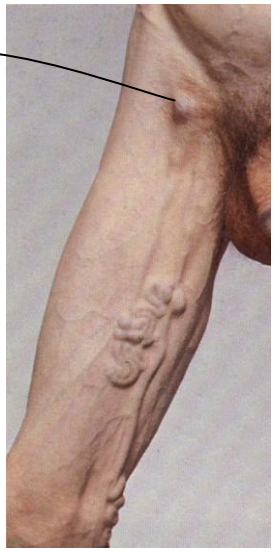
	1ry V.V.	2ry V.V.
	Multiple, bluish, tortuous & visible swellings	
1. Side	• usually **bilateral**	• usually **unilateral** but bilateral if I.V.C. obstruction.
2. Site	Along course of veins (long or short saphenous).	
	• V.V. **never cross** the groin	• V.V. **cross** the groin
3. Shape	• **Tubular** • **Saccular** • **Serpentine** • **Spider**.   	
4. Skin over	• **minimal** complications.	• **marked** complications.
5. Swollen limb	• **minimal** oedema.	• **marked** oedema.
6. Skeletal deformity	• flat foot or halux valgus.	• tallipus equinous.
7. Look for inguinal region	• saphena varix. • hernia as weak mesenchyme	• L.Ns

B- Palpation

1- THE PATIENT IN STANDING POSITION



1. Palpate **soft** & **compressible** varicose veins.
2. Palpate tender **nodule** for thrombosis.
3. Palpate tender **cord** for superficial thrombophlebitis,
4. **Direction of blood of dilated veins**
   across inguinal region **(if 2^{ry} V.V)**
5. **Thrill** if A. V fistula.
6. Impulse on cough at sapheno-femoral junction.
7. **Saphena varix : (if 1^{ry} V.V)**
   Saccular & compressible dilatation shows expansile impulse on cough at sapheno-femoral junction
   .



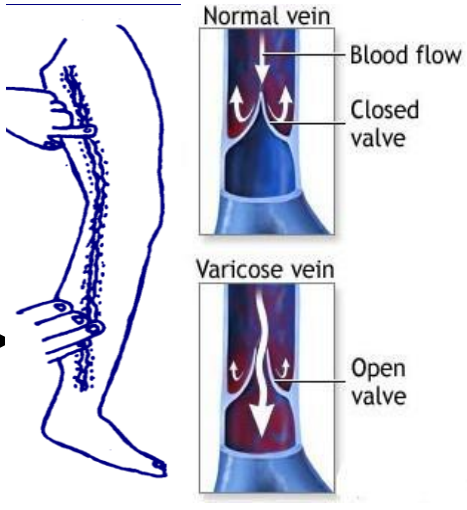
2- THE PATIENT IN SUPINE POSITION

1. **Skin :** for venous ulcer
2. **S.C.** tissue **:** for oedema
   - **1ry** → pitting
   - **2ry** → non pitting
3. **Muscle :** for tender calf muscle
   i.e. **Homan's test** (not done)
   sudden dorsiflexion → calf pain by stretching veins

   N.B.: Homan's sign (not done)
   to avoid spread of thrombus to circulation.
4. **Bone :** for periosteitis of tibia.
5. **Vein :** for defect in deep fascia.
   i.e. **Fegan's test**
6. **Artery :** for arterial pulsation as dorsalis pedis artery
   to exclude ischemic ulcer
7. **L.Ns :** for inguinal L.Ns.

C- Percussion Schwartz percussion

- The vein is percussed by index of one hand & palpate **distally** by fingers of other hand.
- If **1ry V.V** ,the valves are incompetent
  SO the wave is transmitted **distally**



D- Auscultation

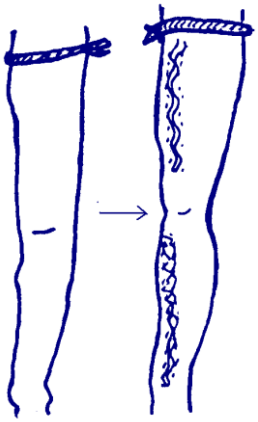
If A/V fistula = continuous **machinery** murmur.

E- Special tests

A. Test to detect (blow out) = Incompetent perforator

① TRENDLENBURG TEST

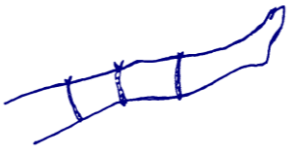
1. Patient lies down & his leg is raised.
2. massage to empty veins.
3. tourniquet just below saphenous opening
4. ask pt, to stand up

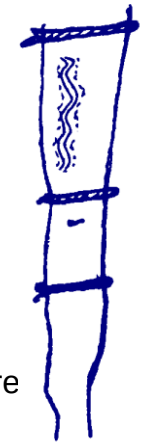


- **The result**
  - ⇨ If slowly filling from below **=** normal
  - ⇨ If rapid filling **= blow out**
  - ⇨ If we remove tourniquet & fill from above **=** incompetent sapheno-femoral junction

② MULTIPLE TOURNIQUET TEST

1. Patient lies down & his leg is raised.
2. massage to empty veins.
3. tourniquet
   - Just below Saphenous ring
   - below Knee
   - above Knee
4. ask pt, to stand up





- **The result**
  - ⇨ If rapid filling of a segment means that there is **blow out**

N.B : for more localization do more tourniquet in the segment

③ MANUAL LOCALIZATION TEST " 2 FINGERS TEST "

- Patient stand & the 2 index are pressed at a point on long saphenous vein then empty at opposite direction.

- **The result** ⇨ If vein fill between two fingers **= blow out**

④ FEGAN'S TEST

- 1st patient stand & then mark the varicosities . then patient, lies down & detect the defect of deep fascia i.e. **blow out** then mark by **(x)**

B. Test to differentiate between occluded & patent deep vein

① PERTHE'S TEST (not done)

1. The patient lies on his back & the lower limb is elevated.
2. An elastic bandage is applied firmly from the toes to the upper thigh.
3. The patient is then asked to stand & walk in situ for 5 minutes.

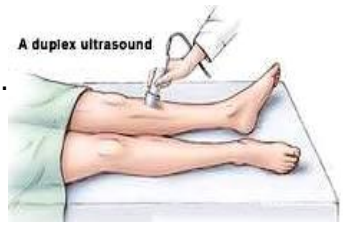
- **The result** ⇨ If the deep system is **occluded,** the patient will complain of pain in the leg.

② MODIFIED PERTHE'S TEST

1. The patient is standing.
2. A tourniquet is applied just below sapheno-femoral junction. .
3. The patient is asked to walk quickly in situ 5 minutes.

- **The result**
  - ⇨ If the varicose veins disappear **=** the deep system is **patent.**
  - ⇨ If the veins become more engorged **=** the deep system is **occluded**

INVESTIGATIONS



1. **Doppler & duplex U/S** to detect incompetent perforators & ensure patency of the deep system.
2. **Abdominal & pelvic CT scan** to detect pelvic masses

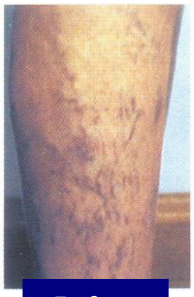
TREATMENT

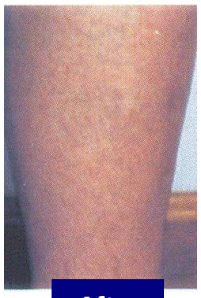
1- Conservative treatment

- **INDICATIONS :** if early 1ry V.V, patient is pregnant, unfit, waiting for or refusing operations.
- **METHODS :** ① avoid prolonged standing
  ② below or above knee elastic stocking.
  ③ periodic leg elevation to prevent stasis.

2- Injection-compression sclerotherapy

- **AIM :** occlusion of lumen by fibrosis & not by thrombosis.
- **INDICATIONS :** ① minor varicosities i.e. spiders
  ② residual varicosities after operations.
- **CONTRAINDICATIONS :** ① 2ry V.V with D.V.T
  ② pregnancy
  ③ acute septic thrombophlebitis.
- **SCLEROSING MATERIALS :** ① **3 %** Na Tetradecyle sulphate.
  ② **5 %** Ethanolamine oleate.


Before


After

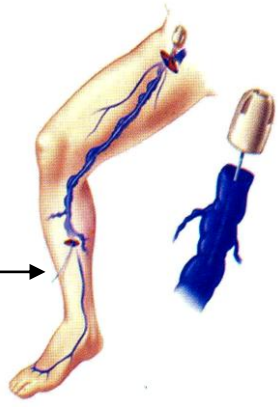
- **TECHNIQUE :** segment injected should be empty of blood i.e isolated by 2 fingers. then firm elastic bandage is applied for 6 weeks.
- **PRECAUTIONS :** ① small dose (1 ml).
  ② one is done only then others at other visits.
  ③ immediate walking after injection to prevent venous stasis.
- **COMPLICATIONS :** extravasation of sclerosing agent → discoloration & sloughing of skin.

3- Operative treatment

1. TRENDLENBURG OPERATION

- **INDICATED :** with sapheno-femoral incompetence i.e. saphena varix.
- **PRINCIPLE :**. legation of long saphenous & it's tributaries.

2. SUBCUTANEOUS STRIPPING OF LONG SAPHENOUS →



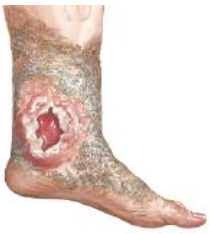
- **INDICATED :** if whole system is severely affected
- **PRINCIPLE :** trendlenburg operation then S.C stripping of whole long saphenous vein.

3. SUB-FASCIAL TRIPLE LIGATION OF INCOMPETENT PERFORATORS

- **INDICATED :** with incompetent perforators i.e. blow out
- **PRINCIPLE :** 3 ligatures are applied on the perforator & saphenous vein above & below then the segment between are removed

III. VENOUS ULCER

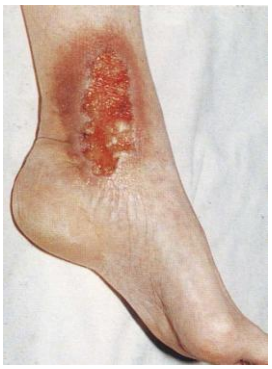
Varicose ulcer

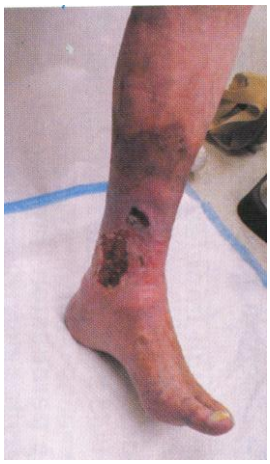


DEFINITION Chronic leg ulcer complicating 2^{ry} V.V
(very rare with1^{ry} V.V)

CLINICAL PICTURE

Inspection	
• **Number**	• usually single.
• **Site**	• above the medial malleolus.
• **Shape**	• oval or rounded.
• **Size**	• variable (cm x cm).
• **Edge**	• punched out edge.
• **Margin**	• brownish pigment & varicosities .
• **Floor**	• unhealthy granulation tissue
• **Discharge**	• pus if infected
Palpation	
• **Temp.**	• slightly higher if infected.
• **Tenderness**	• tender if infected.
• **Skin around**	• thick, brown with varicosities.
• **Base**	• hard base if chronic ulcer.
• **Artery**	• normal pulsation.
• **Vein**	• oedema.
• **L.Ns**	• not enlarged except if infected.
• **Nerve**	• intact sensation.





COMPLICATIONS

1. **Malignancy : Marjolin ulcer**
   which is raised everted edge, hard & fixed base with hard L.Ns.
2. **Periosteitis**.
3. **Talipes equinous**.

INVESTIGATION

- **L**aboratory (blood, urine & stool)
- **A**spiration **B**iopsy **C**ytology (**A**.**B**.**C**)
- **B**iopsy (must include the edge)
- **S**pecific e.g. X-ray to exclude periosteitis

TREATMENT

A- Conservative treatment for **all** cases (Usually **successful**)

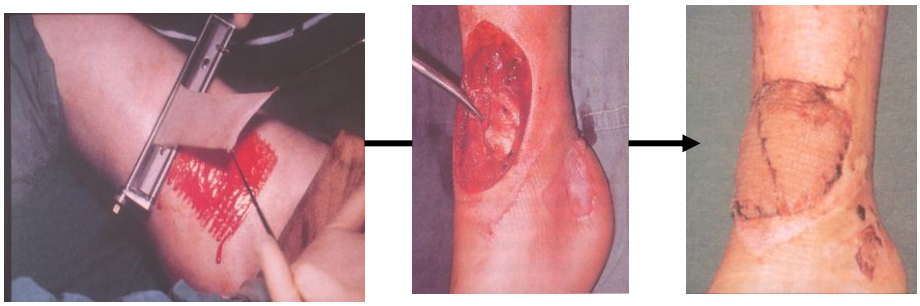
① elevation of the foot in bed.

② daily dressing & systemic antibiotics.

B- Surgical treatment for **chronic** cases

1. COVERING THE ULCER

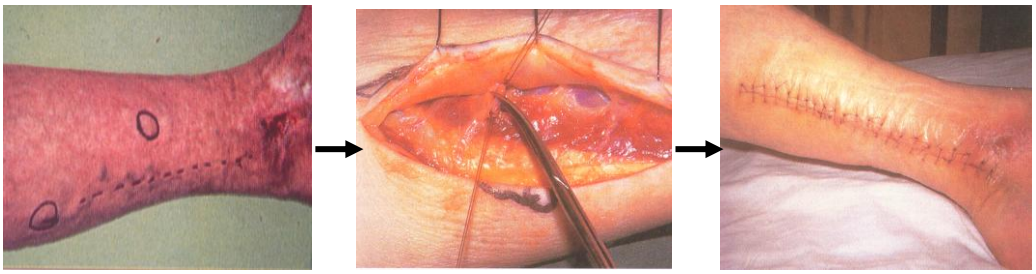
- **Skin graft** is done after subfascial legation in big ulcers to accelerate healing.



2. SUB-FASCIAL LIGATION OF PERFORATOR

(Cockett 's operation)

- through postero-medial incision behind tibia by passing from muscles to perforate the deep fascia
- complicated with ugly scar & high rate of recurrence.



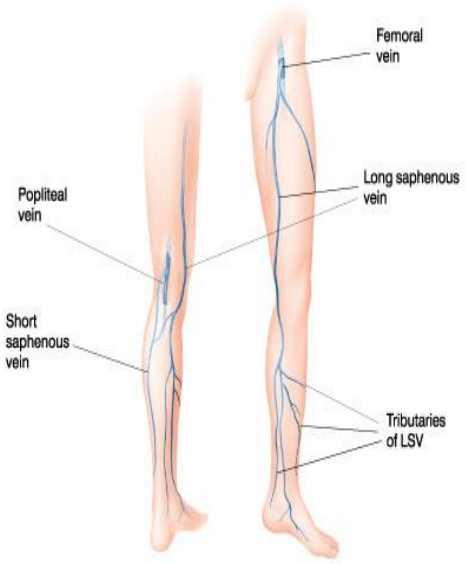
3. TREATMENT OF COMPLICATIONS

- **Malignancy :** excision with safety margin 1 cm & block dissection of L.Ns.
- **Periosteitis :** saucerization.
- **Talipes Equinous :** physiotherapy.

SUPERFICIAL THROMBOPHLEBITIS

DEFINITION

Inflammation & thrombosis of superficial veins.



AETIOLOGY

Varicose veins, trauma, bureger's disease,
I.V infusion & venous cut down.

N.B.: Migrating thrombophlebitis (Trousseau sign)
due to ↑ blood viscosity with internal carcinoma as cancer stomach & cancer pancreas.

CLINICAL PICTURE

- **Symptoms :**
  Pain + **F**ever, **H**eadache, **M**alaise & **A**norexia.
- **Signs :**
  Tender cord like structure with overlying skin redness

TREATMENT

1. Compression by crepe bandage.
2. Antibiotics & anti-inflammatory drugs.

CHRONIC LEG ULCERS

Classification

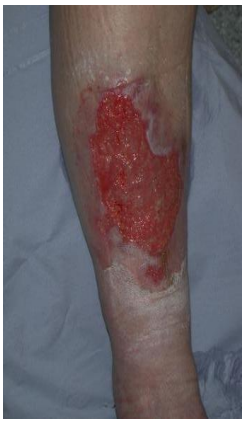
- **Congenital ulcer :**
  1- Sickle cell anaemia
  2- Congenital spherocytosis
- **Traumatic ulcer :**
  Bed sore or trauma.
- **Inflammatory ulcer :**
  1-T.B ulcer
  2- $ ulcer
- **Neoplastic ulcer :**
  Marjolin ulcer
- **Venous ulcer :**
  Varicose ulcer
- **Arterial ulcer :**
  Ischemic ulcer.
- **Lymphatic ulcer :**
  Lymphoedema ulcer
- **Nervous ulcer :**
  Neurotrophic ulcer

1. Congenital ulcer

e.g. **Sickle cell anaemia** & **congenital spherocytosis**

2. Traumatic ulcer

• **Number**	• usually single
• **Site**	• **middle 2/4 of tibia**
• **Shape**	• variable.
• **Size**	• variable
• **Edge**	• punched out
• **Margin**	• ecchymosis.
• **Floor**	• granulation tissues - healthy if healed. - unhealthy if recent
• **Discharge**	• purulent
• **Base**	• indurated at margin



3. Inflammatory ulcer

	T.B.	$
• **Number**	• single	• single or multiple
• **Site**	• **metaphysis of tibia**	• **middle 2/4 of tibia**
• **Shape**	• variable	• variable
• **Size**	• variable	• variable
• **Edge**	• undermined	• punched out.
• **Margin**	• cyanotic	• skin rashes.
• **Floor**	• casseous material	• granulation tissues
• **Discharge**	• serous	• ooze pus & blood.
• **Base**	• soft	• indurated at margin

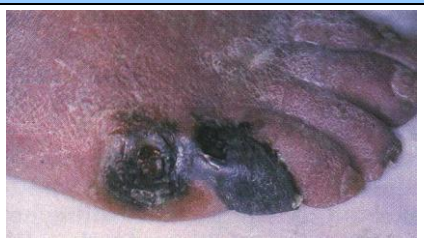
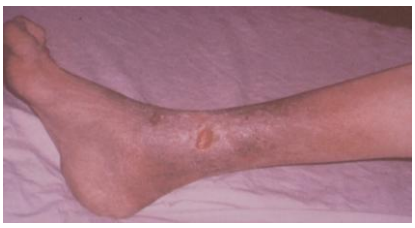
4. Malignant ulcer

- **Marjolin ulcer**
  on top of venous ulcer
  which is raised everted edge, hard & fixed base with hard L.Ns.

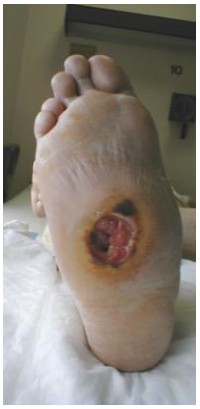
5. Venous (varicose) ulcer

See before

6. Arterial (ischemic) ulcer

Ischemic ulcer	Varicose ulcer
	
• deep ulcer of **foot at pressure area** • ischemic disease • ischemic manifestations. • **no** leg swelling i.e. no oedema. • ↓ **Pain** by foot dependency	• superficial ulcer **above medial maleolus** • post-phlebitic syndrome. • V.V manifestations. • leg swelling i.e. oedema. • ↓ **Pain** by foot elevation

7. Neurotrophic ulcer



DEFINITION

Occur at area deprived from its nerve supply

CAUSE

Peripheral neuropathy as D.M.

MECHANISM

The foot is anaesthetized & the patient is unaware of trauma → ulcer.

CLINICAL PICTURES

- **Site : sole** of foot.
- **Size :** variable.
- **Margin :** corns & callosities.

TREATMENT

1. **Conservative :** rest, elevation & dressing.
2. **Excision** of callosities.
3. **Amputation** if destroyed bone i.e. osteomyelitis

8. Lymphoedema ulcer

- Commonest site at **dorsum** of foot.
- Due to rupture infected bulla.
- Associated with : 1. papillary projections.
  2. lymphorrhaea.
  3. swollen limb oedema.

Lymphatic disorders

LYMPHATIC DISORDERS

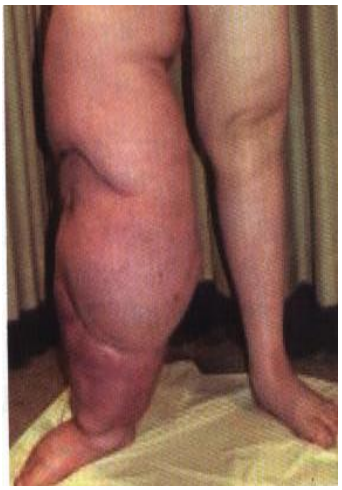
I. DISEASES OF LYMPHATIC VESSELS

Lymphoedema

DEFINITION

Lymphoedema is a hypertrophic condition of skin & S.C tissue caused by chronic lymphatic obstruction.

SITE S.C tissues of limbs, breast, scrotum & vulva.



AETIOLOGY

A- Congenital (1ry type) Rare

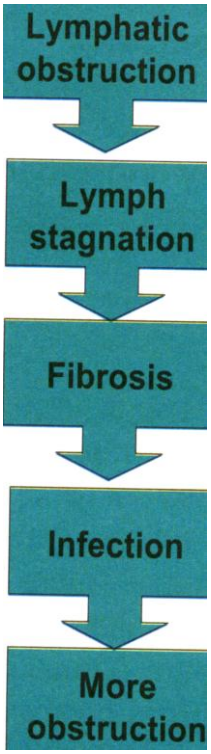
- congenital aplasia or hypoplasia.
- It may be familial (**Milroy's** disease)
- It may be manifested
  - at birth → lymphoedema **congenita**
  - at puberty → lymphoedema **precox**
  - at adult → lymphoedema **tarda**

B- Acquired (2ry type) Common

- **Traumatic :**
  - circumferential skin loss of the limbs, e.g. burn.
  - block dissection of inguinal or axillary lymph nodes.
- **Inflammatory :**
  - chronic specific lymphangitis as **Filariasis**
  - chronic non specific lymphangitis.
- **Neoplastic :**
  - metastasis occluding the lymph nodes.
  - lymphoma but rare.

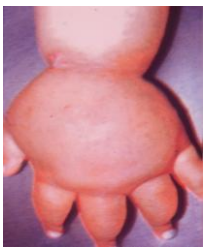
PATHOLOGY

Lymphoedema → lymph stasis in s.c lymphatics → recurrent streptococcal lymphangitis , each attack obliterates more lymphatics → **4 stages**



1. **Stage of soft pitting oedema :** early
2. **Stage of lymphorrhoea :** due to rupture of lymphatic vesicles which leads to discharge of their lymph.
3. **Stage of fibrosis (non-pitting oedema) :** extravasated fluid with its high protein content excites fibrosis of skin & subcutaneous tissue.
4. **Stage of warty pseudo-papillomatous formation** : i.e. **Elephantiasis,** the skin is roughened, puckered & non pitting like elephant skin.

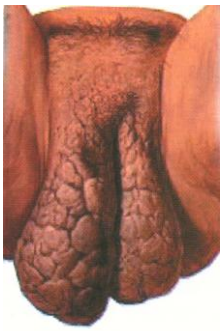
COMPLICATIONS



- Recurrent cellulites & lymphangitis.
- Skin blebs & lymphoedema ulcers.
- Huge & heavy limb interfere with activity.
- Lymphangio-sarcoma (very rare).

D.D

Causes of swollen limb

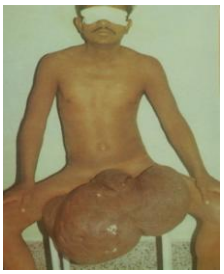


➢ **Bilateral** i.e. generalized oedema

- **Renal** oedema.
- **Hepatic** oedema.
- **Cushing syndrome**
- **Cardiac** oedema.
- **Allergic** oedema.
- **Myxedema**

➢ **Unilateral** i.e. localized oedema

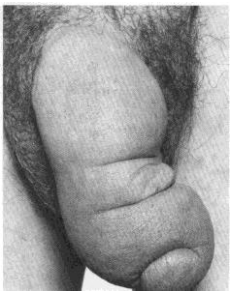
- **Venous** oedema.
- **Lymphatic** oedema.
- Congenital **A.V fistula** (local gigantism).
- Elephantiasis **neurofibromatosis.**



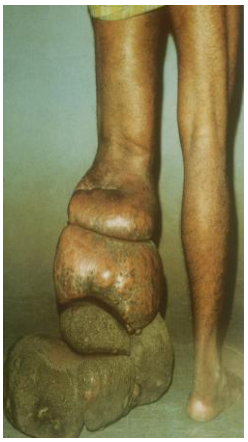
CLINICAL PICTURE

FILARIAL LYMPHOEDEMA

- **Age :** adult or elderly.
- **Residence :** Endemic area for **Filariasis**
  e.g. Rasheed, Damietta, Giza, Imbaba & Sharkeya.



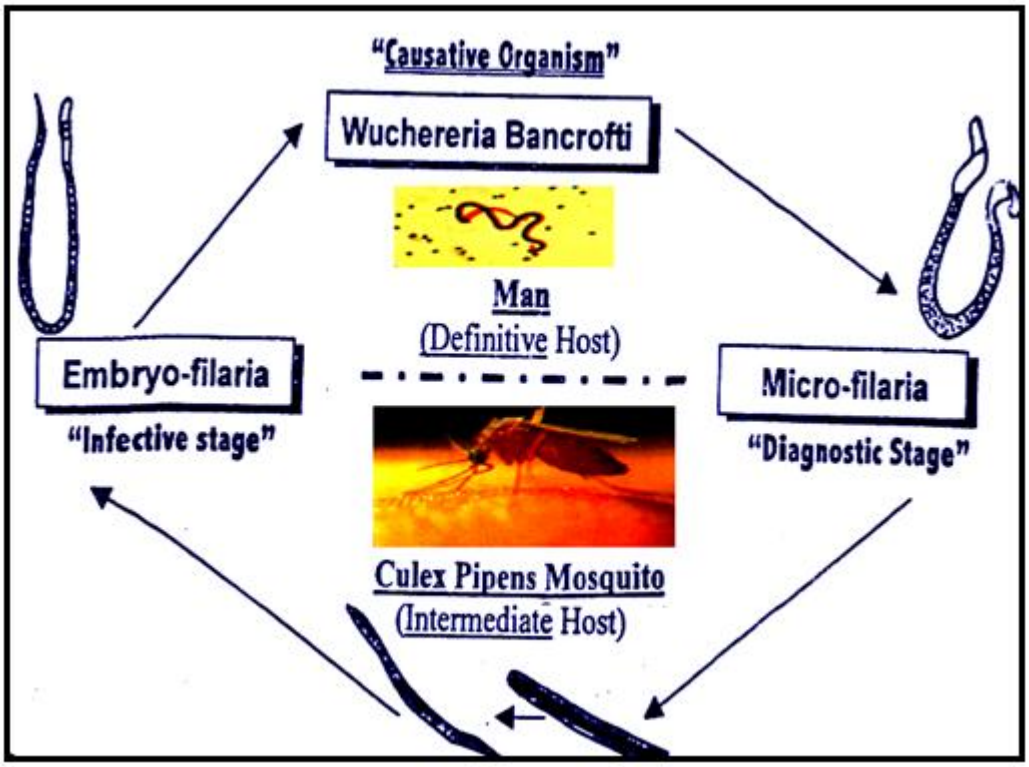
- **Symptoms :**
  - Progressive **leg swelling** with exacerbation & partial remission from recurrent lymphangitis.
  - Elephantoid **fever**
  - **In late cases :** dark, thick skin with callosities i.e. Elephantiasis.
- **Signs :**
  - **Oedema**
    ranges from pitting to non pitting according to its stage



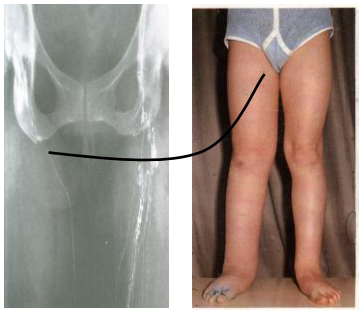
N.B : No oedema in the following sites :
- **Sole :** because of planter aponeurosis.
- **Crease:** because it drains directly to the ankle joint i.e. deep lymphatics.

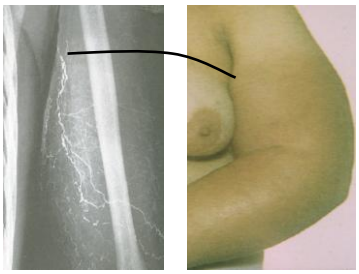
  - **Inguinal L.Ns**
    may be enlarged, firm & tender
  - **Other filarial manifestations**
    1. **Scrotum :**
       → mechanical impotence (sunken penis)
    2. **Spermatic cord & epididymis :**
       → funiculo-epididymitis.
    3. **Tunica vaginalis :**
       → 2ry hydrocele i.e. chylocele

LIFE CYCLE OF FILARIASIS



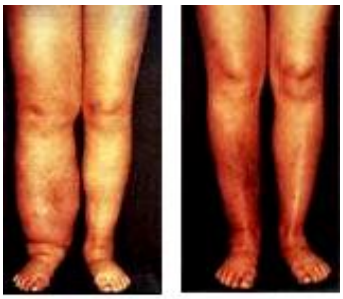
INVESTIGATIONS





1. **Lymphangiography (** not done nowadays **)**
   by injecting ultra-fluid lipidol in lymphatics on the dorsum of foot.
   - It shows the state of lymphatic vessels.
   - It asses extent of L.Ns affection in tumors.
2. **Investigations for filariasis**
   - Night blood film shows micro-filarial.
   - Lymph node biopsy shows dead filarial worms
3. **Lymphocintigraphy :**
   Scanning of lymphatic vessels & nodes using radioactive **TC^{99}** labeled with colloidal particle.
4. **CT scan :** to visualize pelvi-abdominal mass
5. **MRI :** provides clear image of lymphatic vessels & lymph nodes.

TREATMENT


Before After

A. Conservative treatment with early cases

1. Rest & elevation of foot.
2. Massage & elastic stocking.
3. Diuretics, which are of **controversial value**
4. antibiotics e.g. **Penicillin**
   1.200.000 units every 3 weeks.
5. Anti-filarial drugs : **Diethyl carbamazine**

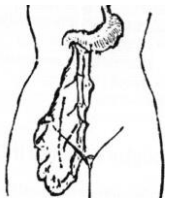
B. Surgical treatment with chronic cases.

I. PHYSIOLOGICAL (BRIDGING) OPERATIONS

- **Aim :** creating new pathway for lymphatic drainage.
- **Disadvantage :** they leave the thick subcutaneous tissues.
- **Types :**

1. Enteromesenteric bridge operation

A segment of the ileum with its mesentery are separated & brought under the inguinal ligament.



2. Omental pedicle flap

A segment of the greater omentum is mobilized with intact blood supply, & brought under the inguinal ligament to laid in the thigh muscles to develop new lymphatics

3. Micro-lymphatico-venous anastomosis

The dilated obstructed lymph trunks are anastomosed to nearby veins.

4. Micro-lymphatic transfer operation

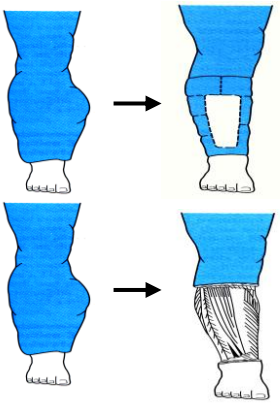
The healthy lymph trunks are harvested from the normal limb & anastomosed to bypass the obstructed lymphatics.

5. Lympho-venous anastomosis

The L.Ns are bisected & anastomosed to nearby vein.

II. EXCISIONAL OPERATIONS

- **Aim :** Excision of skin & thick S.C tissue for cosmetic reasons.
- **Types:**



1. Sistrunk operation

Excision of an ellipse of skin & thick S.C tissue and closure of the defect.

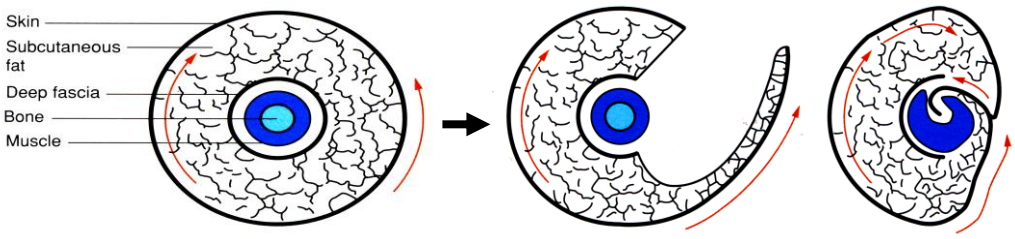
2. Charle's (flaying) operation

Excision of skin, thick S.C tissue & deep fascia (flaying) then covered by skin graft.

III. PHYSIOLOGICAL & EXCISIONAL OPERATION

Thompson's (Swiss roll) operation

Excision of S.C Tissue & then implantation of a shaved flap of skin between the muscles near the deep lymphatic vessels **so that** physiological drainage may be improved.

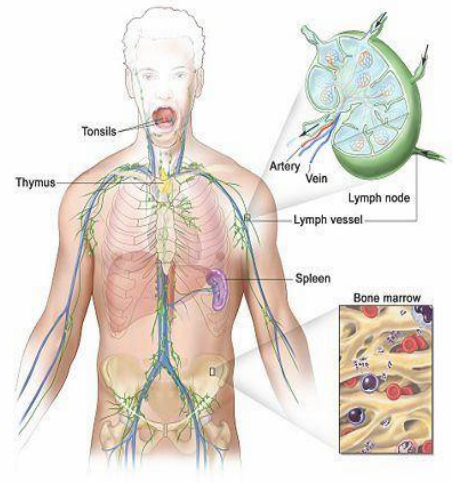


N.B : AMPUTATION indicated with **lymphangiosarcoma**

II. DISEASES OF LYMPHATIC NODES

Lymphadenopathy

The causes of lymph node enlargement are



1. Inflammatory

Acute

- Septic lymphadenitis.
- Infectious mononucleosis (**I.M.N**)

Chronic

NON SPECIFIC

- Child with tonsillitis or pharyngitis.
- Pediculosis capitis.

SPECIFIC

- **Bacterial : - T.B** (**see later**)

  - $ - 1ry $ (chancre)
  - 2ry $ (skin rashes + mucous patches) + generalized lymphadenopathy.
  - 3ry $ (gamma).

- **Parasitic : Filarial** (**see before**)

- **Viral : - Lymphogranuloma inguinal**
  - It is a venereal disease, carry from person to another by contact.
  - Few weeks from contact, papule appears near the genitalia with inguinal L.Ns.

  - Cat scratch disease :
  - Unknown but may be history of cat scratch with lymphadenopathy 1-2 weeks later.

  - AIDS

- **Protozoal : Toxoplasma**.

2. Lymphomas

1. **Hodgkin's** lymphoma.
2. **Non Hodgkin's** lymphoma. } **(see later)**
3. **Burkitt's** lymphoma.

3. Blood diseases

1. **Acute** leukemia.
2. Chronic **myeloid** leukemia.
3. Chronic **lymphatic** leukemia.

4. Metastases

T.B LYMPHADENITIS

A. Lymph borne 1ry T.B

- **fibrocaseous** type.
- **localized** L.Ns
- affect the **cortex** of L.Ns

B. Blood borne 2ry T.B

- **lymphadenoid** type.
- **generalized** L.Ns
- affect the **Medulla** of L.Ns

A. Lymph borne (1ry T.B)

Fibrocaseous T.B

AETIOLOGY

- **Source of infection :** infected milk.
- **Organism :** mycobacterium tuberculosis.
- **Route of infection :** lymphatic vessels.

PATHOLOGY

- **T.B ingested** with milk then filtered through the **tonsils** → **cortex** of upper deep cervical L.Ns.
- **The body reacts** by sending macrophages to engulf the organism.
- **The end result** will depend on ☜

The body resistance & the virulence of organism

I. BODY RESISTANCE > VIRULENCE OF ORGANISM

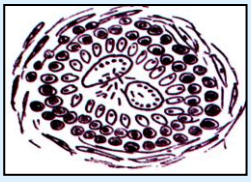
- Macrophages phagocytose the bacilli → Epitheliold Cells

II. BODY RESISTANCE = VIRULENCE OF ORGANISM

- Rare & affect **adult**.
- Fibrosis (no caseation) occur.

III. BODY RESISTANCE < VIRULENCE OF ORGANISM

- Common & affect **children**.
- The macrophages fail to abort the lesion so they fuse together → **Lanqhans giant cells** surround the lesion, further cells like (lymphocytes, plasma cells & fibroblasts) will surround the lesion as outer manner to form finally → **Tubercle follicle**

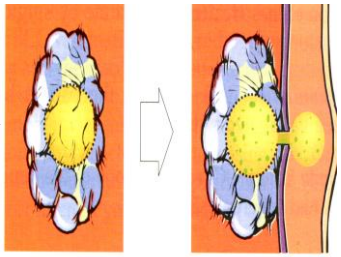


- **Within 2 weeks**
  Necrosis (caseation) occur in the center due to
  1. Hypersensitivity to bacilli toxins.
  2. Ischemia i.e. cellular proliferation without vascular proliferation

CLINICAL PICTURE

- **No** T.B toxaemia
- **Localized lymphadenopathy :**
  - **Site :** Upper deep cervical L.Ns.
  - **Consistency :** Firm, cystic (abscess) or hard (calcification)
  - **Characters :** Painless, **matted** (adherent to each other) i.e. periadenitis or **Rosary beads** due to associated thick lymphangitis.

- **Pictures of complications :**
  1. **Cold abscess :** due to caseation.
     (It is actually **not** cold & **not** abscess).
     - **Not** cold → because it is clinically **warm**.
     - **Not** abscess → because the content is casseous **not** pus.
  2. **Calcification :** It becomes **hard** mass.
  3. **2ry bacterial infection** → Pyogenic abscess.
  4. **Rupture through deep fascia**
     i.e. S.C abscess (Collur studd abscess)
  5. **Rupture through skin**
     i.e. T.B sinus



INVESTIGATIONS

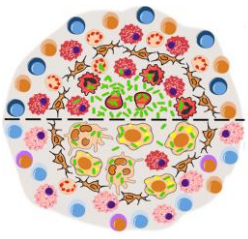
Laboratory

- **Blood picture** (anaemia, leucopenia & relative lymphocytosis).
- **Tuberculin test** serves as good -ve test.
- **Aspiration** of cold abscess for bacteriology :
  - **Ziehl-Nelson stain** → acid fast, alcohol fast bacilli (72 % accuracy)
  - Culture on **Lowenstein media** → (98 % accuracy)
  - **Guinea pig** Inoculation → (94 % accuracy)

Radiological Chest **X-ray**.

Surgical (L.N biopsy) → **Tubercle follicle**

1. **Central zone :** caseation with tubercle bacilli.
2. **Mid zone :** epithelioid cells + langhans giant cells.
3. **Peripheral zone :** small rounded cells as (lymphocytes, plasma cells & fibroblasts).



TREATMENT

A- General treatment

1. Improve the general condition by proper diet & vitamins.
2. **ANTI-TUBERCULOUS DRUGS**
   1st 3 months **Streptomycin** 1 gm IM day after day then a combination of 2 anti-tuberculous drugs at least 9 months
   Rifampicin + **Isonicotinic acid hydrazide (INH)**

B- Local treatment

SURGICAL EXCISION OF L.NS

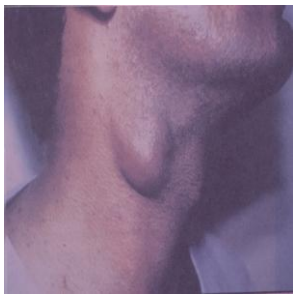
If localized group of L.Ns persist inspite of medical treatment.

TREATMENT OF COLD ABSCESS

- **Repeated aspiration** with injection of streptomycin.

  - Technique :

  1. through healthy skin
  2. under complete aseptic technique.
  3. the site of puncture should be in a non dependant area
  4. wide bore needle.
  5. in a valvular manner.



  - Stop aspiration when blood comes out.

- **Incision & drainage** for 2ry infected abscess.

TREATMENT OF T.B SINUS

- **Repeated dressing** with streptomycin powder.
- **If resists** to treatment **:**
  Excision of underlying lymph node together with the sinus

B. Blood borne (2ry T.B)

Lymphadenoid T.B

AETIOLOGY

- Occurs in adult due to spread of T.B from a 1ry focus e.g. lung

PATHOLOGY

- T.B. reach the L.N through the artery → **medulla** is affected & **not** the cortex. so L.Ns show hyperplasia **not** caseation.
- There is **no** periadenitis, **no** matting, **no** caseation, **no** cold abscess, **no** calcification & **no** sinus.

CLINICAL PICTURE

- T.B toxaemia
  " Night sweat, night fever, loss of weight & loss of appetite "
- **Generalized lymphadenopathy :**
  - **Site :** Start as cervical group of L.Ns then becomes generalized
  - **Consistency :** Firm.
  - **Characters :** Painless, mobile discrete & **uniform** in size

INVESTIGATIONS

"**as before**"
but L.Ns biopsy show hyperplasia

TREATMENT

- **General treatment :** as before
- **Local treatment :** No role of surgery except for L.N biopsy.

LYMPHOMAS

Malignant neoplasm that arise in the lymph nodes or extra-nodal tissues

A- Hodgkin's lymphoma **B- Non-Hodgkin's lymphoma**

A. Hodgkin's lymphoma

Lymphadenoma

INCIDENCE

The commonest type of lymphoma.

PATHOLOGY

SITE

Whenever there is lymphoid tissues, Hodgkin's disease may occur
i.e. L.N, thymus, spleen, liver, bone marrow…… etc..

N.B.: Hodgkin disease may be

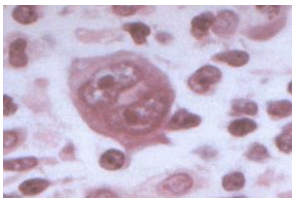
- **Nodal** (common) starts in lower deep cervical L.Ns
  then becomes generalized
- **Extra-nodal** (rare) in late cases affect spleen, liver, … etc.

N/E PICTURE

- The L.Ns are replaced by **pinkish** neoplasm which
  doesn't infiltrate the surroundings except very late.
- **Very late cases** there are deposits in spleen, liver, bone marrow…… etc..

MICROSCOPIC PICTURE

- Variable lymphocytes but the characteristic is
  Reed Sternberg cells (pathognomonic giant cell)
  which is multinucleated 2-8 nuclei
  arranged in mirror images



HISTOLOGICAL CLASSIFICATION

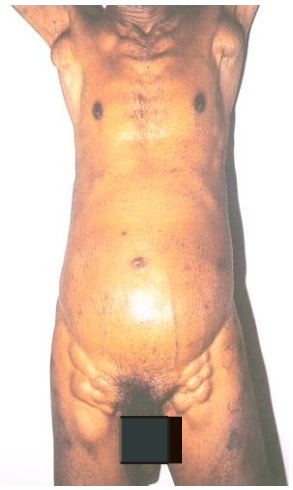
- **Lymphocyte predominant** type **:** the best prognosis.
- **Nodular sclerosis** type **:** the cell nodules surrounded by fibrosis.
- **Mixed cellularity** type.
- **Lymphocyte depleted** type **:** the worst prognosis

CLINICAL PICTURE

NODAL PICTURE

- **Site :** Start as **one** group (**lower** deep cervical L.Ns)
  then becomes generalized.
- **Consistency :** Firm **(Rubbery).**
- **Characters :** Painless, mobile discrete & **different** in size

EXTRA-NODAL PICTURE



- **Systemic :**
  - Night sweat, loss of weight, pruritis, anaemia.
  - **Pel-Ebstein** fever (characteristic fever) which is **2** weeks of fever alternating with **2** weeks freedom.
  - **Alcoholics** →↑ pain at site of Hodgkin's disease.
- **Local :**

  i.e. **Pressure symptoms**

Abdominal manifestations

- Slight splenomegaly.
- Hepatomegaly & Jaundice.
- Ascites from hepatic dysfunctions.
- I.V.C & ureteric compression by para-aortic L.Ns.

Chest manifestations

- Dyspnea, cough & chest pain i.e. mediastinal syndrome.

STAGING Ann Arbor system

STAGE (I) **one** group only of L.Ns.
- ***stage I E*** : if associated single extra-nodal organ.

STAGE (II) **> one** group of L.Ns at **same** side of diaphragm.
- ***stage II E*** : if associated single extra-nodal organ.

STAGE (III) L.Ns are involved **above** & **below** diaphragm.

STAGE (IV) Nodal & extra-nodal spread
spleen, liver, bone marrow…… etc..

N.B: Each stage is further subdivided into

"A" → No systemic symptoms.

"B" → One or more of systemic symptoms as night sweat, loss of weight , pruritis.... etc.

INVESTIGATIONS

LABORATORY

- **Blood picture :** anemia + ↑ ESR
- **Liver function tests :** ↑ alkaline phosphatase with liver involvement.
- **Bone marrow aspirate :** may show bone marrow involvement.

RADIOLOGICAL

- **Chest X-ray & CT scan :** to detect mediastinal L.Ns affection.
- **Abdominal U/S & CT scan :** to detect para-aortic L.Ns affection. & organs affection as liver &spleen.

SURGICAL

- **L.Ns biopsy :** to detect type & staging of lymphoma.
- **Staging laparotomy :**
  - Splenectomy : for staging & avoids the need of its irradiation.
  - Biopsy of both liver lobes.
  - Biopsy of all intra-abdominal lymph node, which are marked by metal clips to help future localization by the radiotherapist.
  - Bone marrow biopsy from the iliac crest.

N.B.: Staging laparotomy is **not** preferred in many centers because of ① ↑ accuracy of CT scan & MRI

② The risk of overwhelming post-splenectomy (OPSI), that is fatal in children.

TREATMENT **" depend on staging "**

- stage **IA**&**B** and stage **IIA** : **Radiotherapy alone**
- stage **IIB** : **6** cycles **chemotherapy** (MOPP) supplemented by **radiotherapy**.
- stage **III** & stage **IV** : **12** cycles **chemotherapy** (MOPP) supplemented by **radiotherapy**.

N.B. : MOPP
Mustin (I.V)
Onchovine (I.V)
Procarbazine (oral)
Prednisone (oral)

ROLE OF SURGERY

① **Splenectomy** if hypersplenism.

② **Decompression** of ureter or IVC by L.Ns.

③ **In young female :** ovaries are sutured retro-uterine to be away from radiotherapy.

PROGNOSIS

Relatively **good** prognosis

(5 years survival rate of 80 % of patients with proper treatment)

B. Non-Hodgkin's lymphoma

N H L

INCIDENCE

Rare type of lymphoma.

but common with the following diseases :

1. Sjogren's disease.
2. Systemic lupus erythromatosus (S.L.E).
3. Immunosuppression after organ transplantation.
4. AIDS

PATHOLOGY

SITE

- **Nodal :** starts in the cervical group of L.Ns then becomes generalized.
- **Extra-nodal :** (more likely to present extra-nodal than Hodgkin's lymphoma)
  e.g. spleen, liver, skin " mycosis fungoids "
  & GIT mucosa so may leads to
  - **Gastric lymphoma**
    which produce manifestations similar to carcinoma.
  - **Intestinal lymphoma**
    which may produce intestinal obstruction, bleeding or perforation.

N/E PICTURE

- The L.Ns are replaced by **white** neoplasm which **infiltrate** the surrounding tissues.
- **Similar** deposits in spleen, liver, bone marrow…… etc..

MICROSCOPIC PICTURE

- The normal architecture of nodes are completely **lost** & replaced with malignant cells of different shapes.

HISTOLOGICAL CLASSIFICATION **" based on cell of origin "**

- **B cell** lymphoma **:**
  (a) **small** cell lymphoma.
  (b) **large** cell lymphoma.
  (c) **mixed** small & large cell lymphoma.
- **T cell** lymphoma**.**
- **Lymphoblastic** lymphoma**.**
- **Histocytic** lymphoma.

CLINICAL PICTURE

NODAL PICTURE

- **Site :** Start as **one** group (cervical group of L.Ns) then becomes generalized.
- **Consistency :** Firm, Soft (if degenerated) or Hard (if calcified)
- **Characters :** Painless & **amalgamated** (can not be counted)

EXTRA-NODAL PICTURE **as** Hodgkin's lymphoma

STAGING — **as** Hodgkin's lymphoma

INVESTIGATIONS **No** staging laparotomy

TREATMENT **" depend on staging "**

- **Radiotherapy & Chemotherapy :**
  The commonly used drugs are ⇩
  Cyclophosphamid, **A**driamycin, **V**incristine & **B**leomycin.

ROLE OF SURGERY

① **Gastrectomy** if gastric lymphoma.

② **Intestinal resection** if intestinal lymphoma.

PROGNOSIS

Very bad prognosis

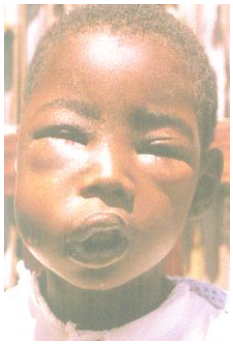
(The prognosis of gastric lymphoma is better than gastric carcinoma)

.....

Burkitt's lymphoma

INCIDENCE

The rarest type of lymphoma



AETIOLOGY

Unknown, but may be related to infection with Ebstein Barr (EB) virus which cause I.M.N in healthy people & Burkitt's lymphoma in patient with chronic malaria.

PATHOLOGY

Burkitt's lymphoma is a malignant tumor of the B lymphocytes.

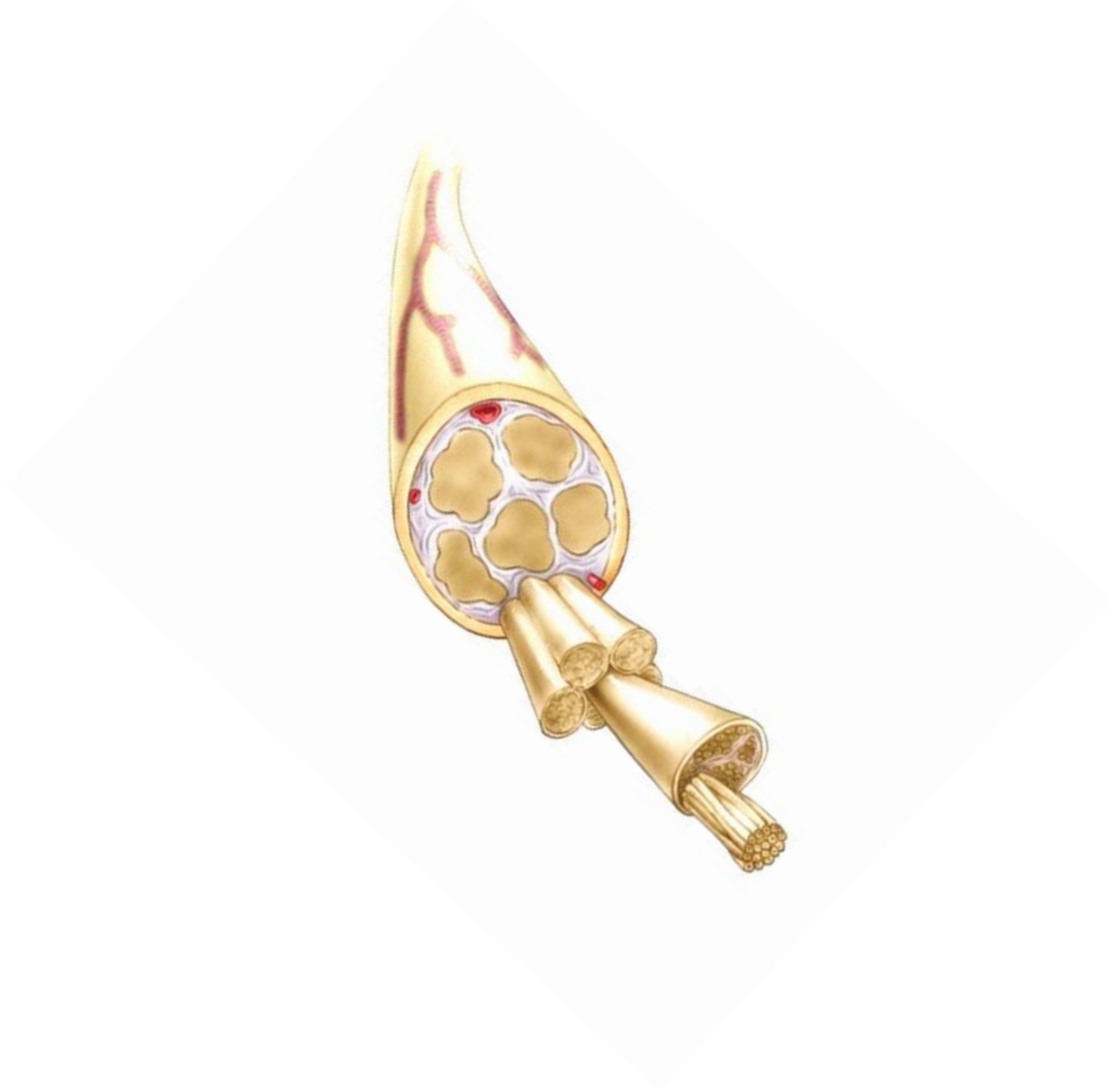
CLINICAL PICTURE

- Common among children **< 12** years
- Common in central Africa which is known be endemic for malaria.
- **The usual presentation** :
  - Painless, progressively enlarged jaw swelling, which may distort the face, may displace the eye & partially occludes the mouth
  - It may also affect the kidneys, ovaries, long bone & central nervous system.

TREATMENT

- **Chemotherapy :**
  The commonly used drugs are ⇩
  Cyclophosphamid & **C**ytosine arabinoside.

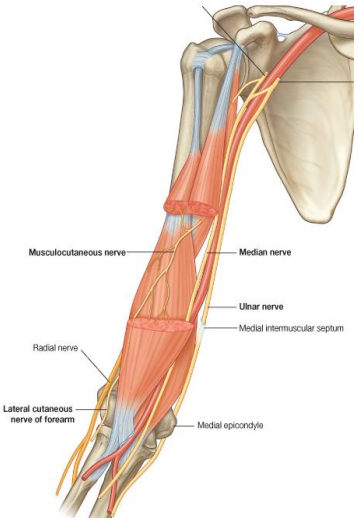
PERIPHERAL NERVE INJURY



Anatomy
of
Peripheral nerves

1- Anatomy of Ulnar nerve

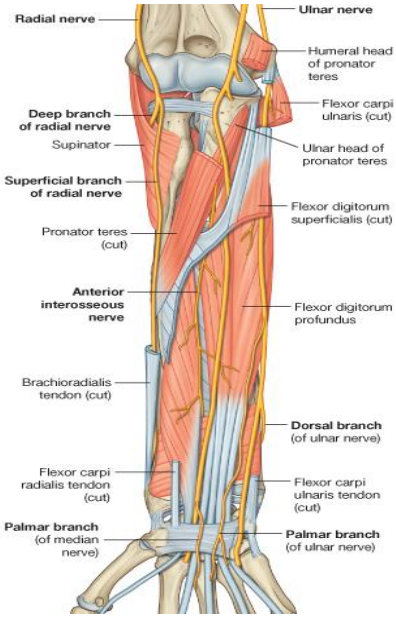
✷ Course & relations



In the axilla & arm

- It arises as the termination of **medial** cord of brachial plexus
- It descends medial to brachial artery
- Then directed medially & downwards to pierce the medial intermuscular septum, to reach the posterior compartment of the forearm.

In the forearm



- It enters the forearm by passing behind the medial epicondyle
- It passes between the 2 heads of flexor carpi ulnaris, & descends between flexor carpi ulnaris & flexor digitorum profundus
- Continues downwards superficial to the flexor retinaculum.

In the hand

- Terminates by dividing into superficial & deep branches.

✷ Branches

In the forearm

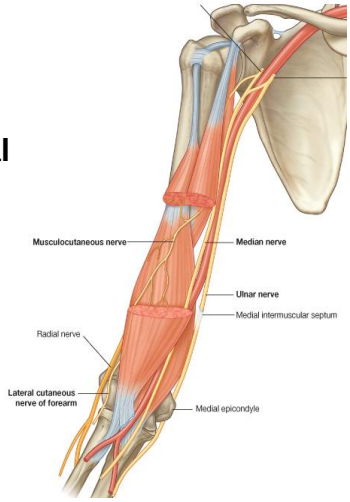
- **Motor :** Supply flexor carpi ulnaris + medial 1/2 of flexor digitorum profundus.
- **Sensory :** Supply medial 1/3 of the palm & medial 1/3 of the back of the hand & back of the medial I & 1/2 fingers

In the hand

- **Motor :** Supply
  a. Adductor pollicis
  b. Intreosseous space
  c. Hypothenar eminence
    - Abductor digiti minimi.
    - Flexor digiti minimi.
    - Opponens digiti minimi.
- **Sensory :** sensation at palmar surface of medial 1.5 finger only

2- Anatomy of Median nerve

✷ Course & relations

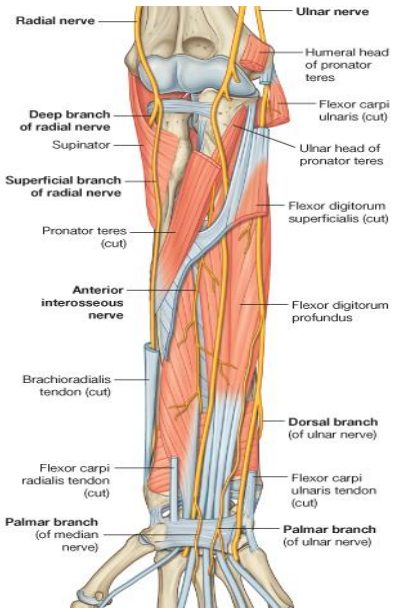


In the axilla & arm

- It arises by 2 braches from **medial** cord & **lateral** cord of brachial plexus (**C5, C6, 7, 8 & T1**).
- It crosses in **front** of the brachial artery

In the forearm

- It leaves the cubital fossa by passing between the 2 heads of pronator teres.
- Passes deep to flexor digitorum superficialis
- Passes under cover the carpal tunnel



In the hand

- By dividing into lateral & medial divisions

✷ Branches

In the forearm

- **Motor :** All flexors of the forearm **except** flexor carpi ulnaris **+** medial 1/2 flexor digitorum profundus
- **Sensory :** Supply lateral 2/3 of the palm

In the hand

- **Motor :** Supply
  a. Abductor pollicis brevis.
  b. Flexor pollicis brevis.
  c. Opponens pollicis.
- **Sensory :** sensation at palmar surface of lateral 3.5 finger **only**

3- Anatomy of Radial nerve

✷ Course & relations

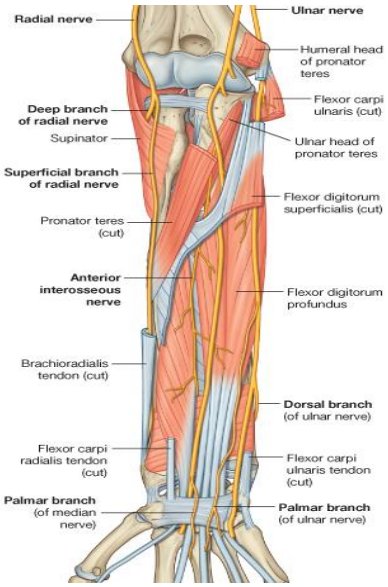
In the axilla & arm

- It arises as the terminal branch of **posterior** cord (**C5,6,7,8 & T1**)
- It passes **behind** the axillary artery.
- It passes between medial & lateral heads of triceps inside the spiral groove
- **After leaving the spiral groove :**

It pierces the lateral intermuscular septum to enter the anterior compartment
Here, the nerve lies in the groove between the brachialis & brachioradialis.

IN THE FOREARM

- It passes In **front** of lateral epicondyle
- It gives the **posterior interosseous** & the **superficial radial nerve**.



IN THE HAND

- It reaches the back of the hand by crossing over the anatomical snuff box

✷ Branches

IN THE AXILLA

- **Motor :** to long head of **triceps**.
- **Sensory :** Posterior cutaneous nerve of the arm

IN THE SPIRAL GROOVE

- **Motor :** to lateral & medial heads of **triceps**
- **Sensory :** Posterior cutaneous nerve of the forearm

IN THE LATERAL SIDE OF ARM

- **Motor :** A branch to **brachioradialis**.
  & A branch to **extensor carpi radialis longus**.

IN THE FOREARM **Posterior interosseous nerve (pure motor)**

Extensors of forearm (12 muscles)

I. Superficial group: (7 muscles)

1. **Brachioradialis.** → Flexion of elbow in mid prone position.
2. **E.C.R. longus** → Common action + abduction.
3. **E.C.R. brevis** → Common action + abduction.
4. **Extensor digitorum** → Common action + extension of M/P of med. 4 fingers
5. **Extensor digiti minimi** → Extension of M/P of little finger.
6. **Extensor carpi ulnaris** → Common action + adduction.
7. **Anconeus** → Common action except extension of wrist.

II. Deep group : (5 muscles)

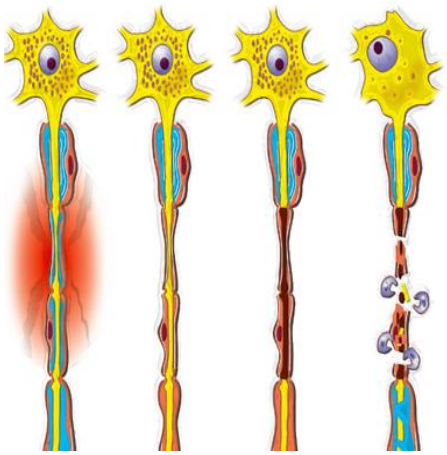
8. **Supinator** : Suppination of extended pronated forearm.
9. **Abductor pollicis longus** → Abduction of adducted thumb.
10. **Extensor pollicis brevis** → Extension of proximal phalanx of thumb.
11. **Extensor pollicis longus** → Extension of all joints of thumb.
12. **Extensor indices** → Extension of proximal phalanx of index

Nerve injury

PERIPHERAL NERVE INJURY

INTRODUCTION

AETIOLOGY (**Trauma**)



A- Open injuries as ⇩

Open wound, bullets, deep burns ... etc.

B- Closed injuries as ⇩

① Fracture & dislocation.
② Contusions by a direct blow
③ Compression by tourniquet
④ Birth injuries.
⑤ Ischemia from injuries of main vessels.

PATHOLOGY (**Types**)

A- Neuropraxia

Temporary loss of nerve function with **no** changes in nerve axons or sheaths so it has the **best** prognosis.

B- Axontmesis

It is due to interruption of the axon with **intact** neurolemmal sheaths so it has a **good** prognosis.

C- Neurotmesis

It is due to **interruption** of both axon & neurolemmal sheaths so it has a **bad** prognosis.

CLINICAL PICTURE

1- Injury of motor part

1- Deformity (malposition).

2- Paralysis (loss of function).

3- Muscle atrophy & **wasting**

2- Injury of sensory part

1- Loss of superficial sensation as pain, temp & touch.

2- Loss of deep sensation as sense of position & movement.

3- Injury of autonomic part

1- Vasomotor changes as redness of skin.

2- Sudomotor changes as loss of sweating.

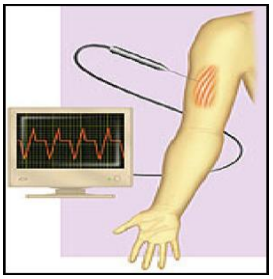
4- Trophic changes.

As loss of hair, brittle fissured nail & dry scaly skin.

DIAGNOSIS OF NERVE INJURY

A- Clinical examination

History of trauma & its type



B- Tests

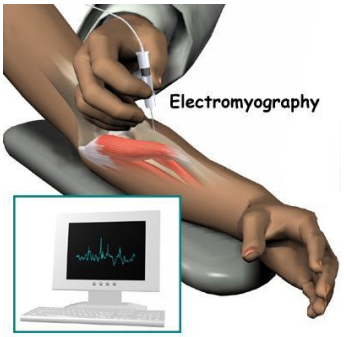
(A) NERVE CONDUCTION TEST (N.C.T) :

- **Neuropraxia** conduct electrical impulse.
- **Axontmesis** & **neurotmesis** can't conduct it.

(B) QUINIZARINE POWDER TEST :

= **Sweating test** to detect anhydrosis

- Put the **white** powder at skin affected then observe the change of its color.
- If remain **white** it means **anhydrosis**
- If changed to be **pink** it means **sweating**,



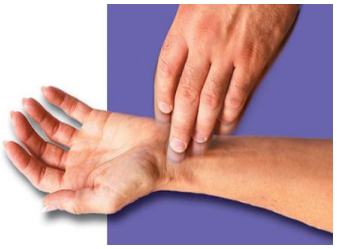
(C) ELECTROMYOGRAPHY :

- Detect response of denervated muscles to electric stimulation.
- **Fibrosed** muscles shows **no** response

N.B.: Plain x-ray :
May detect any foreign bodies or fractures.

(D) TINEL'S PERCUSSION :

- To detect the rate of nerve regeneration,
- The course of the nerve is percussed from below, upwards.
- **Tingling sensation** is experienced when the level of regeneration is reached.



TREATMENT

A- Conservative treatment

INDICATIONS

1- CLOSED INJURIES as ➪
neuroaprexia & axontmesis.

2- BEFORE or **AFTER** nerve repair to prevent deformity.

CONSISTS OF ➪

1- SPLINTAGE to prevent over-stretching of paralyzed muscles.

2- ACTIVE & **PASSIVE** exercise to prevent stiffness.

3- PROTECTION of the skin.

B- Operative treatment

INDICATIONS

1- ALL OPEN INJURIES

2- CLOSED INJURIES after failure of conservative treatment.

CONSIST OF

1- Nerve repair :

- **1^{ry} nerve suture** (Immediate suture of divided nerve) with clean & incised wounds present within **6** hours of injury.
- **2^{ry} nerve suture** (3 - 4 weeks later) If any gap is present it should be overcomed **by the followings :**

① **Dividing** unimportant branches to gain length.

② **Nerve transposition** e.g. bringing radial nerve in front of humerus or ulnar nerve in front of medial epicondyle.

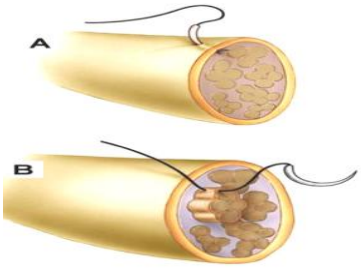
③ **Bone shortening**, which is done only in radial nerve with un-united fracture humerus.

④ **Nerve grafting** by small cutaneous nerve as sural nerve in the leg.

N.B. : Types of nerve repair :

A- Epineural : The suture passes through the nerve sheath

B- Inter-fascicular the suture passes through the nerve bundles



2- Orthopedic treatment :

- **It is used** when one fail to make the nerve to recover
- **It consists of**
  - ① **Arthrodesis :** Fixation of the Joint.
  - ② **Tendon transplantation**

PROGNOSIS

1- Neuropraxia : It is the best prognosis.

2- Motor or **sensory nerve :** Better prognosis than mixed nerves.

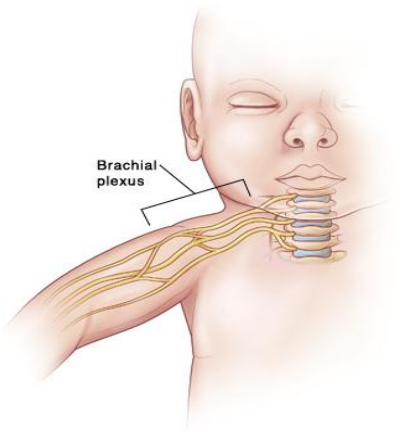
3- Nerve supply bulky muscle : Better prognosis than which supply fine muscles.

4- Good apposition of the cut ends of the nerve = Better prognosis

5- Asepsis : Because sepsis interfere with nerve generation due to fibrosis & loss of nerve tissue.

I- BRACHIAL PLEXUS INJURIES

AETIOLOGY



A- Open injuries as ⇦
Open wound, bullets, deep burns ... etc.

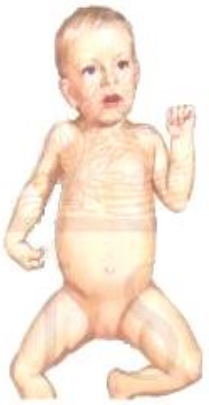
B- Closed injuries as ⇦
① Birth traction injury.
② Fracture clavicle.
③ Hyperabduction of arm under anesthesia.

CLINICAL PICTURE & TYPES

1- Complete brachial plexus injury (Rare)

- **Motor : Paralysis** of all the upper limb muscles.
- **Sensory : Anesthesia** of whole the upper limb
  Except ① **Medial side of arm** which is supplied by intercosto-brachial nerve (**T_2**).
  ② **Over the deltoid** which is supplied by supra-clavicular nerve (**C_3 & C_4**).
- **Horner's syndrome :**
  (Ptosis, myosis, anhydrosis, enophthalmos)

2- Upper trunk injury (Erb-Duchenne paralysis) (C_5 & C_6)



- **Motor : - Paralysis** of shoulder abductors as (deltoid & supra-spinatus), external rotators as (infra-spinatus), forearm flexors & supinators.
  - **So** the limb hangs by the side being adducted, internally rotated, extended, pronated with flexed wrist (**Policeman's tip position**)
- **Sensory : No** effect if **C_5** alone is injured.
  If **C_6** also suffers, there's anesthesia over deltoid.

3- Lower trunk injury (Klummpke's paralysis) (C_8 &T_1)

- **Motor : Paralysis** of flexors of wrist & fingers (**C_8**) & intrinsic muscles of hand (**T_1**) producing (**complete claw hand**)
- **Sensory : Anesthesia** along the inner side of forearm & inner **1½** finger (ulnar distribution).
- **Horner's syndrome :**
  (Ptosis, myosis, anhydrosis, enophthalmos)

II- ULNAR NERVE INJURY

The nerve is either injured at the wrist or elbow

1- Injury at the wrist

AETIOLOGY

① Cut wrist

② Colle's fracture.

③ Carpal tunnel syndrome.

CLINICAL PICTURE

I- Inspection

1- Deformity : (Partial) **ulnar claw hand**
due to paralysis of med, 2 lumbricals.

2- Wasting :

a. Adductor pollicis

b. Intreosseous space

c. Hypothenar eminence

- Abductor digiti minimi.
- Flexor digiti minimi.
- Opponens digiti minimi.

3- Vasomotor or sudomotor changes

4- Trophic skin changes

} **Minimal**

5- Scar at wrist joint

II- Palpation

6- Skin sensation :
Loss of sensation at palmar surface of medial **1.5** finger **only**

2- Injury at the elbow

AETIOLOGY

① Fracture & dislocation of elbow.

② Wounds at the elbow.

③ Stretching & friction of nerve against the medial epicondyle as in **cubitus valgus** deformity.

CLINICAL PICTURE

All of the above +

I- Inspection

1- Deformity : Decreased because of extension of distal I/P joint

- Because of paralyzed medial 1/2 of flexor digitorum profundus.

i.e. Ulnar paradox

- Also there is **radial deviation** because of paralyzed flexor carpi ulnaris

2- Wasting : At medial side of ulna because wasting of flexor carpi ulnaris & medial 1/2 of flexor digitorum profundus.

3- Vasomotor or sudomotor changes

4- Trophic skin changes

} **Marked**

5- Scar : At forearm or cubital fossa.

II- Palpation

6- Skin sensation :

Loss of sensation at palmar & dorsal surface of medial **1.5** finger & loss at palmar & dorsal surface of medial aspect of the hand

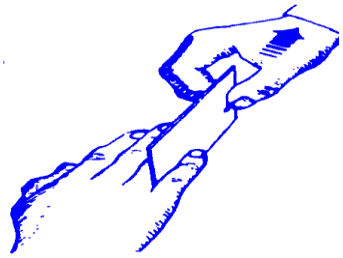
DIAGNOSIS

As general +

Special tests

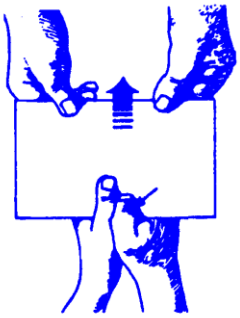
(A) CARD TEST :

- Due to paralysis of **interossei** which adduct the fingers.
- The patient can't hold a card between his extended fingers.



(B) FROMENT TEST :

- Due to paralysis of **adductor pollicis**
- The patient is asked to grasp a paper between his thumb & sides of index fingers.
- The terminal phalanx of the affected thumb is flexed to hold the paper by the flexor pollicis longus which supplied by median nerve.



TREATMENT

As general

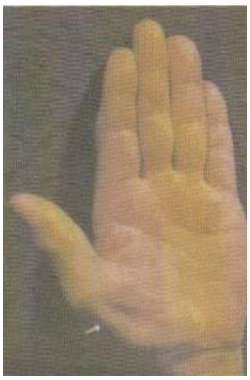
PROGNOSIS

As general

III- MEDIAN NERVE INJURY

The nerve is either injured at the wrist or forearm

1- Injury at the wrist



AETIOLOGY

① Cut wrist

② Colle's fracture.

③ Carpal tunnel syndrome.

CLINICAL PICTURE

I- Inspection

1- Deformity : (Ape hand)
due to paralysis of **ab**ductor pollicis **brevis** and contraction of **ad**ductor pollicis which supplied by ulnar nerve.

2- Wasting :

a. Abductor pollicis brevis.

b. Flexor pollicis brevis.

c. Opponens pollicis.

3- Vasomotor or sudomotor changes

4- Trophic skin changes

} **Minimal**

5- Scar : At wrist joint

II- Palpation

6- Skin sensation :
Loss of sensation at palmar surface of lateral **3.5** finger **only**

2- Injury at the forearm

AETIOLOGY

① Fracture & dislocation of elbow.

② Wounds at the elbow.

CLINICAL PICTURE

All of the above +

I- Inspection

1- Deformity : The same but there is **ulnar deviation** because of paralysis of flexor carpi radialis.

2- Wasting : At flexor surface of forearm because of wasting of their muscles

3- Vasomotor or sudomotor changes

4- Trophic skin changes

} **Marked**

5- Scar : At forearm or cubital fossa.

II- Palpation

6- Skin sensation :

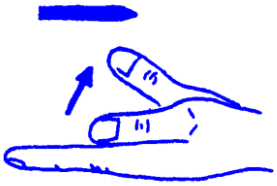
Loss of sensation at palmar surface of lateral **3.5** finger & loss at palmar surface of lateral aspect of the hand

DIAGNOSIS

As general +

Special tests

(A) PEN TOUCHING TEST :



- The thumb can't be abducted to touch a **pen** in front of it with back of hand on table to avoid action of flexors

(B) PRAYER'S POSITION TEST :

- The tip of the thumb of the affected side touching the palmar aspect of the pulp of the non affected thumb.

(C) OCHNER'S CLASPING TEST :

- The **index** on the affected side is pointed, extended & tapered if the patient is asked to clasp his hands together.
- Due to loss of action of lateral 1/2 of flexor digitorum profundus and lateral 2 lumbricals.

TREATMENT

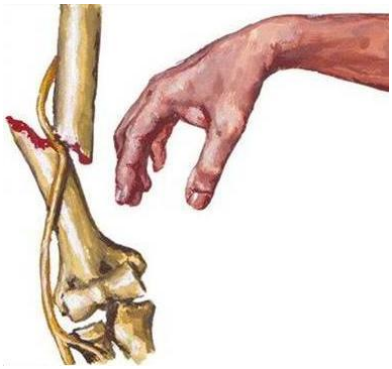
As general

PROGNOSIS

As general

IV- RADIAL NERVE INJURY

AETIOLOGY



A- INJURY AT THE AXILLA :

① Pressure by crutches or chair.

② Fracture & dislocation of shoulder.

B- INJURY AT THE SPIRAL GROOVE :

① Fracture mid-shaft humerus.

② Prolonged application of tourniquet.

③ Injection of irritant drug in back of arm

④ Falling a sleep with the arm lying across the edge of chair (**saturday- night paralysis**).

⑤ Operations in which the out-stretched arm has rested on the edge of the table

EXAMINATION DEPENDING ON SITE OF INJURY

I- Injury at head of radius i.e. **Post intreosseous nerve injury**

- **MOTOR :** Paralysis of all extensors of wrist & fingers
  so (Finger drops deformity)
  no wrist drop because there is weak extension of wrist by brachioradialis & extensor carpi radialis longus.
- **SENSORY : No** changes
  because post. intreosseous n. (purely motor).

II. Injury at lower 1/3 of arm

- **MOTOR :** As above
  + (Wrist drop deformity)
- **SENSORY :** Loss of small area on dorsum of thumb.

III. Injury at spiral groove

- **MOTOR :** As above
  + (Weak extension of elbow)
- **SENSORY :** Anesthesia over lower lateral arm & back of forearm.

IV. Injury at axilla

- **MOTOR :** As above
  + (Complete loss of extension of elbow)

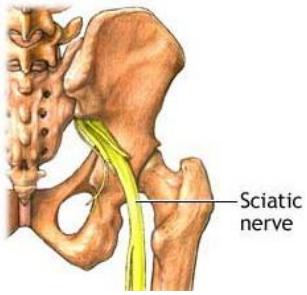
TREATMENT

As general

PROGNOSIS

As general

V- SCIATIC NERVE INJURY



AETIOLOGY

① Wounds, fractures or dislocation of hip

② False site of IM injection of drugs.

③ It may be paralyzed by pelvic tumors

CLINICAL PICTURE

1- Motor effects

1. Paralysis of **flexors** muscles leading to **dropped foot**

2. Complete paralysis below knee.

2- Sensory changes

Complete sensory loss **below knee** with the exception of skin supplied by saphenous nerve (inner side of leg, inner border of foot & big toe).

VI- LATERAL POPLITEAL NERVE INJURY

AETIOLOGY

① Fracture neck fibula or supracondylar fracture femur.

② Pressure from plasters or splints.

CLINICAL PICTURE

1- Motor effects

Complete paralysis of the extensors & peroneal muscles leading to **Talipes eguino-varus** deformity.

2- Sensory changes

Over the **dorsum** of the foot.

VII- MEDIAL POPLITEAL NERVE INJURY

AETIOLOGY (**Rare**)

Since the nerve is deeply placed & well protected by the surrounding muscles

CLINICAL PICTURE

1- Motor effects

Paralysis of muscles of calf & sole leading to **Talipes calcaneo-valgus** and **clawing** of toes.

2- Sensory changes

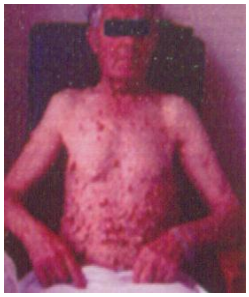
Sensory loss over the **sole**

.

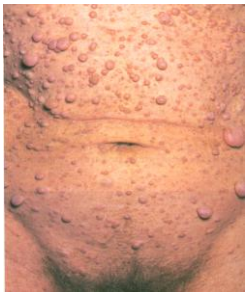
Neurofibromatosis

It is a congenital disease affecting nerves
the tumor arises from fibrous tissue of the neurilemmal sheath.

1- Generalized neurofibromatosis "Von Recklinghousen's disease"

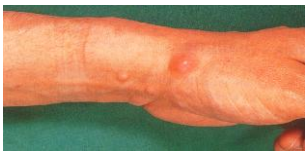


- **It is multiple tumors** of the body along the course of cranial & spinal nerves.
- **It is diagnosed by**
  - The swelling is painless but tender due to pressure (not infiltration) on the affected nerve.
  - **Fusiform** in shape, **firm** in consistency and mobile across but not along the course of nerve.
  - Associated with brown pigments **"Café au lait"** patches.
  - **No** interference with nerve function



- **Treatment :**
  - Removal only of tumors causing pain or pressure effect.

2- Solitary neurofibroma

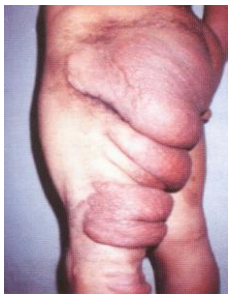


- As generalized but **single**.

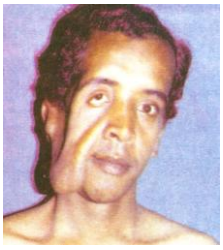
3- Acoustic neuroma

- **Neurofibroma** arising from **acoustic nerve** leading to deafness & cerebellar symptoms.
- It may be single or part of generalized type.

5- Elephantiasis neruomatosa



- It is elephantiasis of the limb →
  + mechanical block of the joint
  + **"Café au lait"** patches.



6- Plexiform neuroma "Pachydermatocele"

- It affect **S.C nerve plexuses** leading to thickening of the nerves & redundant thickened skin

N.B.: NEUROFIBROSARCOMA

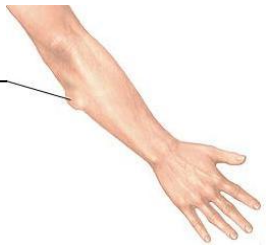
- It a **fibrosarcoma** arising from fibrous tissue of the neurilemmal sheath.
- The tumor grows rapidly with early local infiltration ± metastasis.
- There is pain & interference with nerve function.
- **Treatment :** Wide excision or amputation

Diseases of
Skin & S.C tissues

DISEASES OF SKIN & S.C TISSUES

I. BENIGN LESIONS

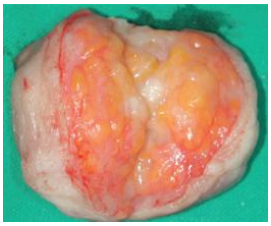
1- Lipoma



DEFINITION

- **Benign tumor** composed of fatty tissue arranged in lobules.

PATHOLOGY

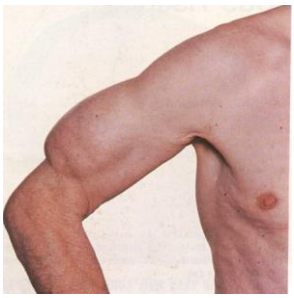


- It is enclosed in a thin fibrous capsule, which sends fibrous tissue septa divide the tumor.
- Yellowish lobulated aggregations of fat cells.

PATHOLOGICAL TYPES

① **Pure** lipoma (the commonest)

② **Fibro**lipoma i.e. contain excess fibrous tissue.



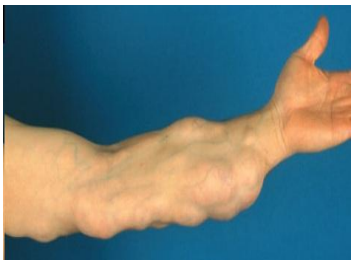
③ **Angio**lipoma i.e. contain angiomatous tissue.

④ **Myxo**lipoma i.e. contain myxomatous tissue.

CLINICAL PICTURE **According to site**

N.B.: Never in brain & eye lid

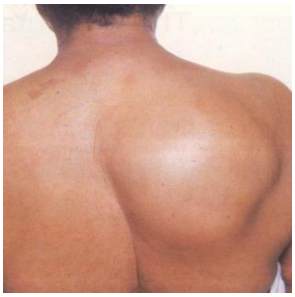
1- Subcutaneous lipomata the commonest.
They have the following characters :



(a) **It may present as :**
- A solitary mass
- multiple lipomatosis
- Diffuse lipomatosis = **Dercum's disease**.

(b) **Lobulated** surface, painless mass, attached to skin at multiple points i.e. **dimpling**

(c) **Soft** in consistency, sometimes gives **pseudo-fluctuation** due to mobility of lipoma in its bed & because fat at warm temperature is liquefied.



(d) It has a well defined **slippery** edge because it runs in its false capsule.

(e) It is mobile & superficial to muscles.

2- Subfascial lipomata
This type is deep to deep fascia. It is not attached to the skin & it does not have a slippery edge.

3- Subserous lipomata i.e. retroperitoneal lipomata
This type may turn to sarcoma.

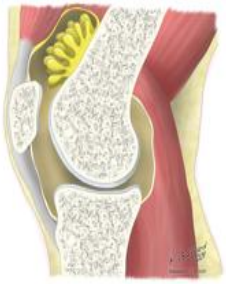
4- Submucous lipomata e.g. larynx or intestine.
This type may cause obstruction.

5- Parosteal lipomata
This type arise under the periosteum of the skull.

6- Extradural lipomata
This type found within the spinal cord
& may cause paraplegia.

7- Intermusclar lipomata
This type found in between the muscles,
commonly at thigh, it becomes more firm
& ↓ in size on muscle contraction.

8- Intra-articular lipomata
This type arise in relation to the capsule of the joint →

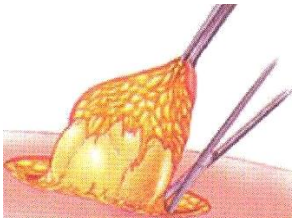


COMPLICATIONS

① **Compression** manifestations.

② **Degenerative changes** leading to liquefaction & calcification.

③ **Malignant transformation,** it can occur with retroperitoneal lipoma.



TREATMENT

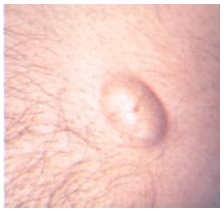
Enucleation of the tumor from plane between (true capsule & false capsule of compressed surrounding tissues)

2- Sebaceous cyst

(**Epidermoid cyst**)

DEFINITION

- **Retention cyst** of the sebaceous gland due to obstruction of its duct by inspissated sebum or dirts.



PATHOLOGY

- **The cyst** is lined by stratified squamous epithelium.
- **The contents** are foul smelling, white, creamy sebum.

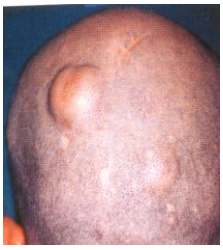
CLINICAL PICTURE **Hairy areas**

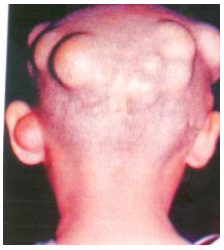
N.B.: Never in palm & sole

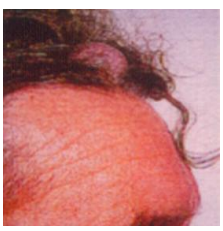
(a) **The commonest sites :**
scalp, face & scrotum

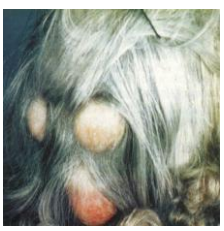
(b) Small, well defined, cystic swelling.

(c) Attached to skin at one point.
this is the site of occluded duct
= Punctum or **black head**.









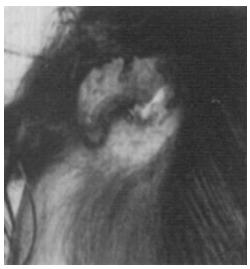
COMPLICATIONS

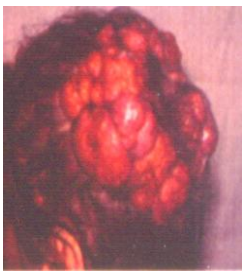
① Infection & suppurations

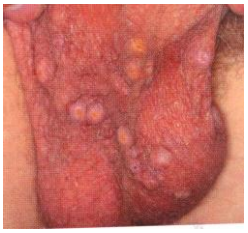
② Localized **alopecia :** hair loss due to pressure atrophy on hair follicle

③ **Sebaceous horn :**
the contents become inspissated in successive layers over the base.

④ **Ulceration (Cock's pecular tumor)**
Infected cyst may undergo ulceration with raised everted edges. It is not a tumor but mistaken for a carcinoma.







TREATMENT

- **Excision** with skin ellipse over it containing the punctum to avoid recurrence
- **IF 2ry infection :** drainage through skin incision**.**

3- Dermoid cyst

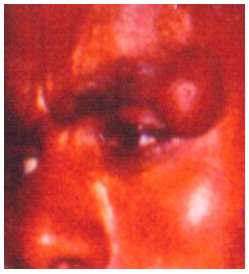
DEFINITION

- It is a cyst lined by stratified squamous epithelium & contains sebaceous material

TYPES

1. Sequestration dermoid cyst

- **It is a congenital inclusion** of a piece of epithelium in the S.C. tissue at line of fusion of the body during the fetal life
- **The commonest sites :**
  1. **Face :** external angular dermoid *&* root of nose.
  2. **Neck & trunk :** middle line (ant. & post.)



N.B: Never appears in upper & lower limbs as they appears as buds & not by fusion.

- **The cyst :** well defined, globular & not attached to the skin

N.B.: In children with sequestrated dermoid in the scalp. It is better to wait until closure of skull sutures because some cysts may communicates with the dura.

2. Tubulo-dermoid

- It is from distension of remnants of **embryonic ducts** as thyroglossal cyst & branchial cyst

3. Teratomatous dermoid

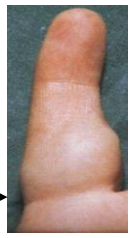
- It is a benign teratoma contains teeth, hair, bone, cartilage. & It occurs mainly in ovary *&* testis.

4. Inclusion dermoid

- It is due to inclusion of epidermis during closure of a cavity as supra-sternal cyst.

5. implantation dermoid

- It is 2ry to puncture wounds which displace some epithelial cells into S.C. tissue → cyst formation.
  It occurs mainly in the sole, palm & fingers →



TREATMENT

- All cases are treated by **excision**

4- Hemangioma

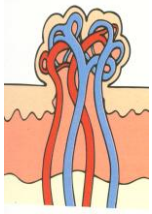
(**Vascular malformations**)

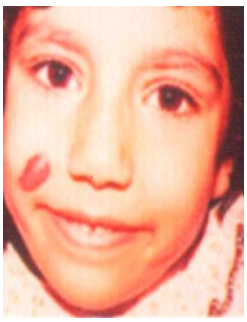
[A] Capillary **[B] Arterial** **[C] Venous**

It is not a true tumor, but tumor like i.e. hamartoma
Hamartoma = congenital malformation of vessels

A. Capillary Hemangioma

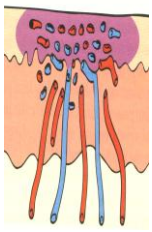
1. Strawberry naevus

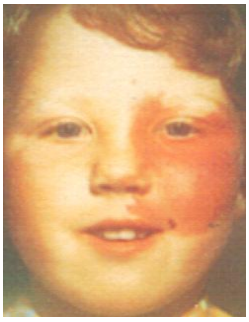




- **Site :** the commonest site is face & head
- **Colour :** bright **red**.
- **Surface :** slightly **raised** above the surface.
- **Course :** present few weeks birth then after one year it starts to undergo **involution** over the next 7 - 8 years.
- **Treatment : not** required because of its spontaneous involution
  but if bleeding occur from friction : surgery or laser are indicated.

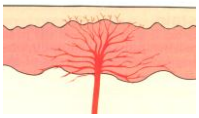
2. Port wine Stain (Nevus flammeus)

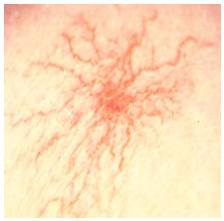




- **Site :** along the distribution of trigeminal nerve of the face & never crosses the middle line.
- **Colour :** dark **purple**.
- **Surface :** usually **flat**.
- **Course :** present since birth & **doesn't** undergo involution.
- **Treatment :** - Laser application is the choice.
  - Excision & grafting is very difficult as it may involve a large area of the face.

3. Spider naevi

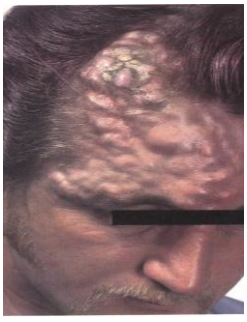




- It occurs with patients having liver cirrhosis & supposed to be due to hyperoestrinism.
- It consists of multiple dilated arterioles from which radicals of dilated capillaries are radiating in the distribution of S.V.C.

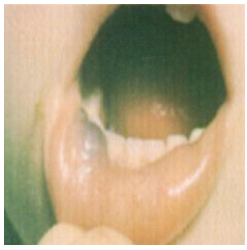
B. Arterial Hemangioma

" Cirsoid aneurysm "

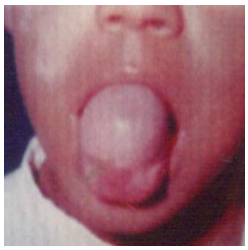


- It is a sort of arterio-venous fistula occurring mostly in the scalp " temporal & occipital " regions.
- It appears as soft compressible & pulsating mass with marked bruit over it
- **Treatment :** - Embolisation of feeding vessels by gel foam then operative legation of feeding vessels.

C. Venous (Cavernous) Hemangioma



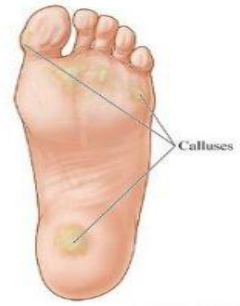
- It consists of multiple large intercommunicating sinus like vascular spaces (**venous**)
- **Clinical features :**
  - Since birth with no involution
  - Reddish swelling of mucus membrane of lips & tongue.
  - Soft, compressible but non pulsating mass.
  - It may be complicated by bleeding due to mild trauma



- **Treatment :**
  - Laser therapy
  - Injection of sclerosing material as 20 % Nacl
  - Surgical excision.

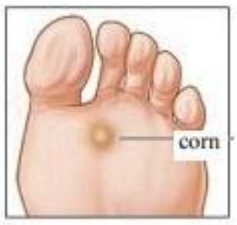
5- Tumor like conditions of the skin

1. CALLOSITY



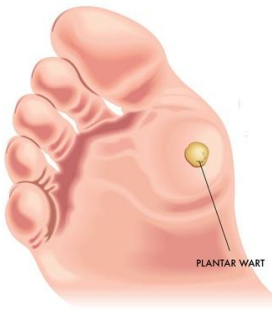
- **Aetiology :** Localized thickening of the skin due to repeated friction
- **Clinical picture :**
  Area of thickened skin which they are painless, elevated above the surface.
- **Treatment :**
  Shaving & application of ***keratolytic agent*** e.g. salicylic acid.

2. CORN



- **Aetiology :** Neglected callosity with down growth of hard horny plug pressing on sensory nerve endings.
- **Clinical picture :**
  Conical mass which is very tender. .
- **Treatment :**
  Like callosity but excision is usually needed.

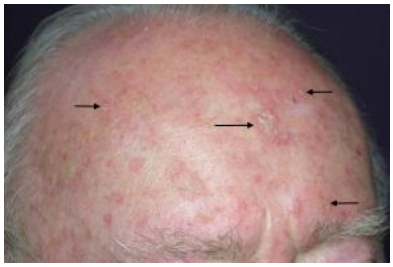
3. WART



- **Aetiology :** Localized epithelial proliferation due to viral infection.
- **Clinical picture :**
  - Small horny projection
  - Common in the dorsum of the hands & feet.
  - Only the plantar warts are painful.
- **Treatment:**
  1. Surgical excision with diathermy.
  2. Cryosurgery by freezing the wart using liquid nitrogen.

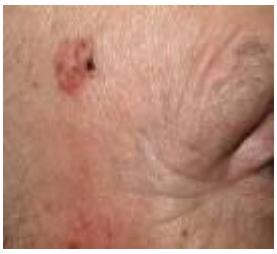
6- Premalignant conditions of the skin

1. Actinic (senile) keratosis



- It is due to prolonged exposure to sun as farmers & fishermen
- Multiple lesions on the face & backs of hand.
- **Investigation** : **biopsy** may be needed.
- **Treatment**
  1. If **Superficial lesion** → Freezing with liquid nitrogen.
  2. If **Horny lesion** → diathermy curettage.
  3. If **Indurated lesion** → Excision.

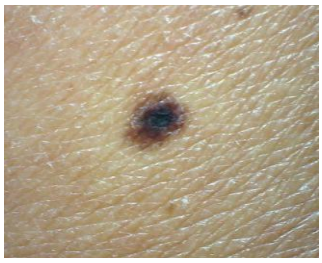
2. Bowen's disease



- it is a potentially malignant disease (seen with elderly).
- Usually red, flat, scaly or crusted mistaken with keratosis
- **Investigation** : **biopsy** may be needed.
- **Treatment**
  1. If **Superficial lesion** → Cryotherapy, curettage or cauterization.
  2. If **Small lesion** → Chemotherapy.
  3. If **Indurated lesion** → Excision

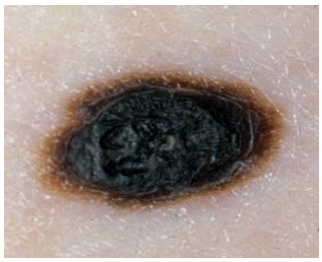
7- Melanocytic tumors

1. Lentigo



- The melanocytes replace the basal layer of epidermis in certain sites.
- **Clinically** : It appears as flat black or brown spots.

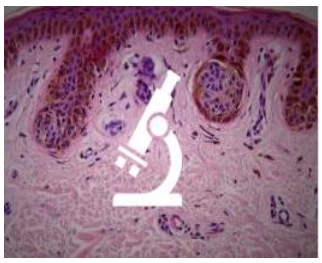
2. Junctional naveus



- More proliferation of melanocytes → Small nodules of epidermis & bulge in the dermis.
- **Clinically** : Like Lentigo

3. Compound naveus

- Present in both epidermis & dermis.
- **Clinically** : It appears as raised brown to black nodule.



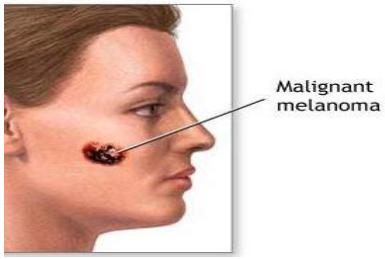
4. Intra-dermal naveus

- Present mainly in dermis
- **Clinically** : Like compound naveus.

II. MALIGNANT & LOCALLY MALIGNANT LESIONS

1. Malignant melanoma

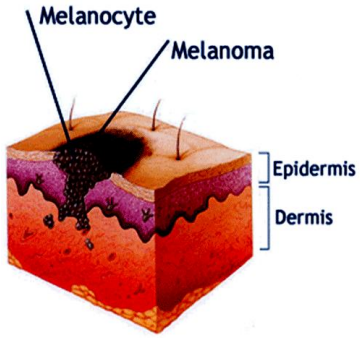
INCIDENCE



- **Age :** 30 - 60 years.
- **Sex :** male > female
- **Commoner** in western countries due to defective ozone layer.

PREDISPOSING FACTORS

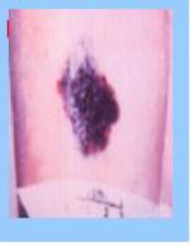
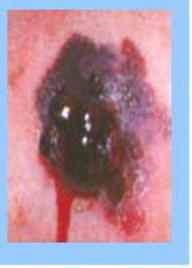
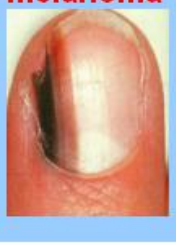
- Pronged exposure to ultraviolet rays of the sun.
- Albinism & xerodermia pigmentosa.
- **On top of benign naevus**



N.B.: Criteria of malignant transformation of benign naevus are :

1. Increase in size or thickness
2. Increase in pigmentation
3. Occurrence of itching, tingling, ulceration or bleeding.
4. Development of satellite nodules.

PATHOLOGY & CLINICAL TYPES

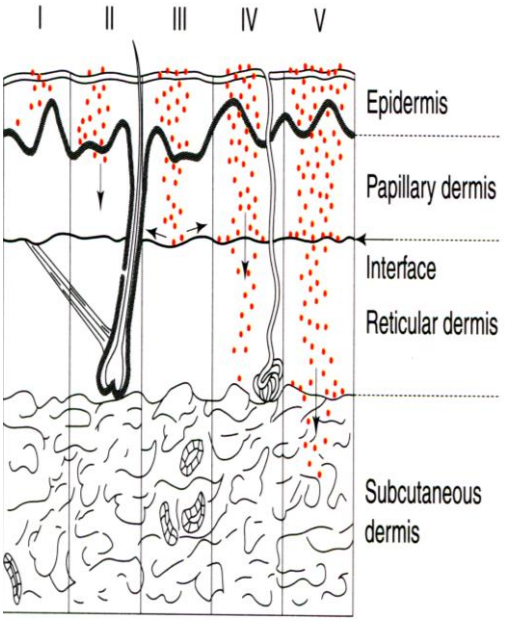
	Superficial spreading	Nodular melanoma	Lentigo maligna	Acral lentigious melanoma	Amelanotic melanoma
					
Incidence	64 %	12 - 25 %	1 -15 %	rare	rare
Age	middle	young	elderly	---------	---------
Site	any part of the body	any part of the body	usually of **face**	**palm** & **sole** may beneath the **nail**	---------
N/E	the lesion is raised above surface with irregular edge	the lesion is nodular, dark in color & **very liable to ulcerate**	the lesion is begins as a flat brown macule which growth very slowly	---------	---------
M/P	highly malignant cells arranged in sheets, melanin granules inside the cells				
Prognosis		bad	good	poor	very poor

CLASSIFICATIONS i.e. prognosis factors

A- Breslow classification

" depends on **thickness** of tumor "

Stage I (< 0.75 mm)
Stage II (0.75 mm - 1.5 mm)
Stage III (1.5 mm - 2.25 mm)
Stage IV (2.25 mm - 3 mm)
Stage V (> 3 mm)



B- Clark's classification

" depends on **depth** of invasion**"**

- **Level I :** Epidermal.
- **Level II :** Dermo-epidermal junction.
- **Level III :** Superficial papillary dermis.
- **Level IV :** Deep papillary dermis.
- **Level V :** Subcutaneous tissue.

SPREAD

- **Direct spread :** to surrounding tissues
- **Lymphatic spread :** by both
  Permeation (i.e. satellite nodules) & embolization .
- **Blood spread :** very rare
  - lungs, liver, bones & brain
  - 2ry deposits are usually **black**

CLINICAL PICTURE see pathological types

DIFFERENTIAL DIAGNOSIS

1. Pigmented basal cell carcinoma.
2. Granuloma or haemangioma.
3. Compound or junctional Naevus.

INVESTIGATIONS

- The only sure method is **biopsy** *&* histological examination.

TREATMENT

- The lesion is **radioresistant** so

The 1ry lesion should be excised with safety margin of skin & S.C tissue.

- IF the tumor thickness is **< 1 mm** → safety margin is **1 cm**
- IF the tumor thickness is **1 – 4 mm** → safety margin is **2 cm**
- IF the tumor thickness is **> 4 mm** → safety margin is **3 cm**

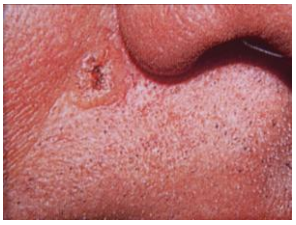
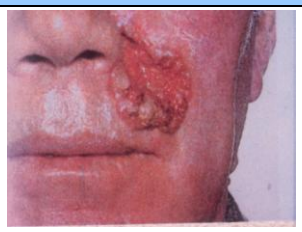
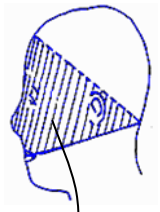
N.B: The deep fascia should **never** be excised

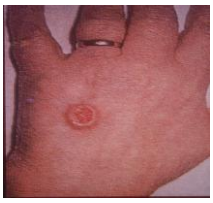
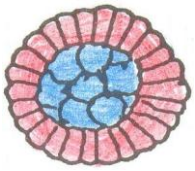
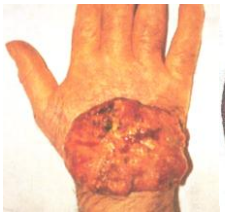
The L.Ns Prophylactic block dissection **no** longer performed.

So if not clinically involved
Fine needle aspiration is done to ensure that they are free

But if clinically involved
Radical block dissection must be done.

The metastasis
Treated by chemotherapy, interferons & interlekin-2

	2. Basal cell carcinoma Rodent ulcer	3. Squamous cell carcinoma Epithelioma
		
DEFINITION	**Locally malignant** tumor that arises from basal layer of skin	**Malignant** tumor that arises from stratified squamous epithelium of the skin
INCIDENCE • **Age** • **Sex** • **Race**	" **Common** " • **> 40** years • male > female • white > Black	" **Less common** " • **> 50** years
PREDISPOSING FACTORS	1. Prolonged exposure to sun rays i.e. ultraviolet rays. 2. Albinism. 3. Xerodermia pigmentosa 	1. Prolonged exposure to sun rays i.e. ultraviolet rays. 2. Albinism. 3. Xerodermia pigmentosa 4. Previous irradiation 5. Long standing irritation as chronic ulcer, sinus or burn scar 6. Carcinogens as tar derivatives.
PATHOLOGY • **Site**	• **90 %** in the **face** especially above a line from lobule of the ear to the angle of mouth. • **Other sites** as dorsum of the hand & perianal regions.	• Upper part of **face**, scalp, lip, gums & tongue. • **Other sites** as dorsum of the hand, oesophagus & anal canal
• **N/E** - Number - Site - Shape - Size - Edge - Margin - Floor - Discharge - Base	• Usually single • As above • Oval or rounded • Usually small • **Rolled in & beaded** • Dilated capillaries. • Covered by **crusts**. • Blood ± pus • Indurated but **not** beyond the edge.	• Usually single • As above • Irregular in shape. • Usually large • **Raised everted**. • …… • **Necrotic** • Blood ± pus • Indurated **fixed** & beyond the edge.

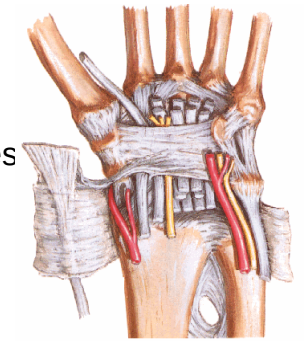
	2. Basal cell carcinoma Rodent ulcer	3. Squamous cell carcinoma Epithelioma
	 	 
• **M/P**	1. Peripheral columnar cells arranged in **palisade** manner. 2. Central **polyhedral** cells with basophilic nuclei. 3. **No** tendency for keratinization	1. Epithelial pearls or cell nests 2. Mass of malignant epithelial cells. 3. Central **keratin**
SPREAD	1. **Direct** spread : the only spread to adjacent structures. 2. **No** Lymphatic spread : but if enlarged L.Ns. it is due to 2ry infections or epitheliomatous transformation 3. **No** blood spread.	**1. Direct** spread : the tumor rapidly infiltrates the adjacent structures. **2. Lymphatic** spread : by embolization to the regional L.Ns. which are hard & stony in consistency **3. Blood** spread : late & rare.
TYPES	• **2 TYPES OF ULCER** - **Excavating** type **:** the ulcer erodes deep into the under lying structures leading to destruction of the nose. - **Field-fire** type **:** the ulcer is rapidly spreading with advancing periphery and healing center. • **LESS COMMON TYPES** as flat superficial or pigmented type.	**GRADING** **BRODER'S CLASSIFICATIONS** • **Grade I :** 75 - 100 % keratin • **Grade II :** 50 - 75 % keratin • **Grade III :** 25 - 50 % keratin • **Grade IV :** 0 - 25 % keratin
CLINICAL PICTURE	- Patient represents by a small **nodule** which later on ulcerate. - **No** draining L.Ns - Examine the ulcer "as before"	1. Patient represents by an **ulcer** which grows rapidly. 2. Hard draining L. Ns 3. Examine the ulcer "as before"
COMPLICATIONS	1. 2ry infections → meningitis & cavernous sinus thrombosis 2. Erosion of big vessels→ severe hemorrhage 3. Epitheliomatous transformation	**METASTASIS**

	2. Basal cell carcinoma Rodent ulcer	3. Squamous cell carcinoma Epithelioma
D.D	**DD OF ULCERS OF THE FACE** **1. Squamous cell carcinoma**	**1. Basal cell carcinoma**
	2. Malignant melanoma	
	3. Keratoacanthoma (Molluscum sebaceum)	
	• It is progressive then <u>regressive</u> course • It forms a red firm rounded papule that enlarges rapidly in one week then regresses slowly to heal within 3 to 6 months. • There is a, **keratin plug** in the center. 	
INVESTIGATIONS	• **L**aboratory (blood, urine & stool) • **A**spiration **B**iopsy **C**ytology (A.B.C) • **B**iopsy (must include the edge) • **S**pecific e.g. X-ray skull	
TREATMENT	**1- SURGICAL EXCISION** • **Excision** with **0.5** cm safety margin	**1- SURGICAL EXCISION** • **Excision** with **2** cm safety margin
	• **Indicated in :** 1. Small lesion as it easy to close the defect. 2. Ulcer infiltrating bone or cartilage because malignant cells are hidden so efficient dose will lead bone necrosis. 3. Recurrence after radiotherapy. 4. Resistant to radiotherapy.	
	2- RADIOTHERAPY • **B.C.C & S.C.C** are **radiosensitive**	
	3- OTHER FORMS • **CRYOSURGERY** • **5 FLUOROURACIL** Cytotoxic ointment (**Effudex**)	**3- IF REGIONAL L.NS INVOLVED** • **BLOCK DISSECTION** of metastatic L.Ns.
PROGNOSIS	**Cure rate** **=** 100 % but recurrent	**Cure rate** **=** 90 % with early treatment

Diseases of Muscle, Tendons & Fasciae

DISEASES OF MUSCLES, TENDONS & FASCIAE

1- Carpal tunnel Syndrome

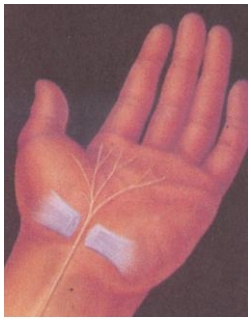


SURGICAL ANATOMY

Carpal tunnel is formed by the flexor retinaculum over the carpal bones, it transmits the long flexor tendons & the median nerve, but the palmar cutaneous branch of median nerve passes on the flexor retinaculum, so it is spared in this syndrome.

CAUSES

- Rheumatoid arthritis.
- Myxedema & pregnancy
  (due to increase tissue fluid deep to flexor retinaculum)
- Colle's fracture

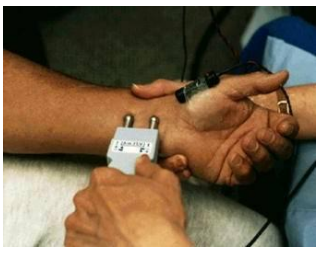


PATHOLOGY

- Manifestations are due to compression of blood supply of median nerve → **ischemic neuritis**.

CLINICAL PICTURE

- **Type of patient :** middle aged female.
- **SYMPTOMS :**
  - **Pain :** In the distribution of the median nerve in the hand, relieved by hanging the hand over the edge of the bed.
  - **Wasting** of thenar muscles
  - **Paraesthesia** over the lateral 3 ½ fingers.
- **SIGNS :**
  - **Tenderness** over the carpal tunnel by percussion.
  - ↑ pain (if fingers & wrist are held fully flexed for few minutes)

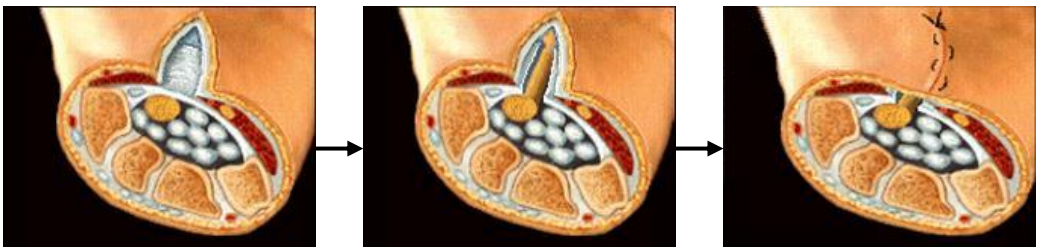


INVESTIGATIONS

Nerve conduction study
on median nerve shows delay at the carpal tunnel

TREATMENT

- **Mild** → Anti-inflammatory + corticosteroids.
- **Severe** → Surgical splitting of the flexor retinaculum



2- Bursitis

INTRODUCTION

☆ **Bursae** are fluid-filled cavities, lined with flattened epithelium
☆ **It contains** clear mucin fluid to **minimize** friction.
☆ **Types :**

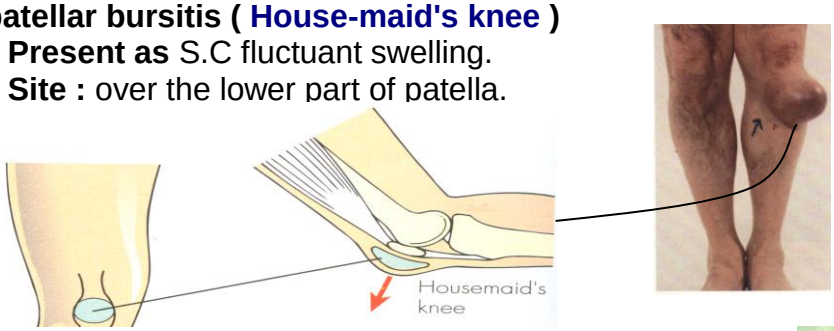
(A) ANATOMICAL BURSAE
present where tendon passes over bony surface or where superficial fascia & skin covering bony prominence.

(B) ADVENTITIAL BURSAE
developed over areas of repeated friction i.e. newly formed.

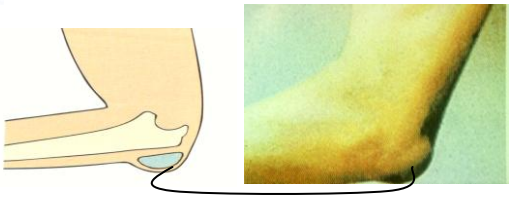
Chronic anatomical bursitis

TYPES OF CHRONIC BURSITIS

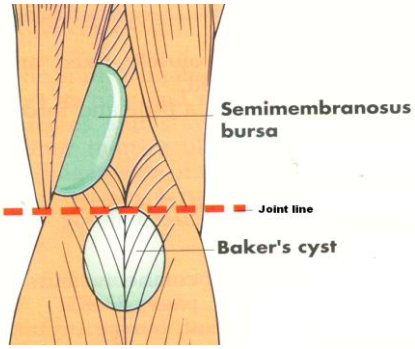
1. Pre-patellar bursitis (House-maid's knee)
- **Present as** S.C fluctuant swelling.
- **Site :** over the lower part of patella.



2. Olecranon bursitis (Student's elbow)
- **Present as** lax fluctuant swelling.
- **Site :** over the olecranon process



3. Semi-membranosus bursa :
- **Present as** tense, fluctuant swelling & characterized by tense on extension of knee & flaccid on flexion of knee.
- **Site :** medial part of popliteal fosse & **above** the joint line.



N.B: D.D from Baker's cyst
which is herniation of synovial membrane of knee joint with osteoarthritic patients & present at center of popliteal fossa **below** joint line.

N.B: D.D of swellings of Popliteal fossa

A- Cystic swellings :
Abscess, hemangioma, lymphangioma, popliteal aneurysm
A/V fistula , saccular varicosity or semi-membranosus bursa

B- Solid swellings :
Lipoma, neurofibroma, fibrosarcoma or osteomyelitis

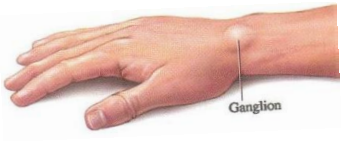
TREATMENT **Excision** of bursa

3- Ganglion

A. Simple ganglion

DEFINITION

- This is a small cyst that contains a clear gelatinous fluid

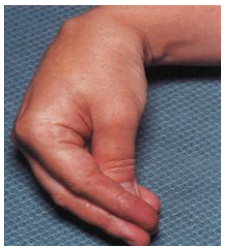


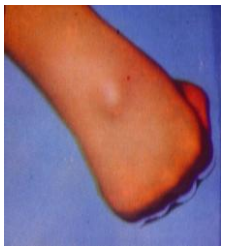
PATHOLOGY

- It is a cyst that contains jelly like mucin,

CLINICAL PICTURE

- Localized, tense, cystic, painless mass & related to tendon
- The commonest site is back of wrist.
- It is mobile across but not along and it's mobility restricted with tendon action.





TREATMENT

- Excision

B. Compound ganglion

DEFINITION

- It is a T.B synovitis of the tendons passing under flexor retinaculum.

CLINICAL PICTURE

- Localized, fluctuant swelling.
- At the lower part of flexor retinaculum.
- Characterized by " **Cross fluctuation test** "

TREATMENT

- Anti-tuberculous drugs
- Immobilization in plaster cast to provide rest of the involved sheath.
- **If no response :** Excision of the diseased sheath

4- Chronic tendinitis

TYPES OF CHRONIC TENDINITIS

1. Tennis elbow :

- The patient complains of pain in the elbow at rest when he uses the hand (**extensors**)

2- Golfer's elbow :

- Similar to tennis elbow, but when he uses the hand (**flexors**)

3- Supra-spinatus tendinitis :

- Pain is felt in the shoulder especially on abduction & rotation. There is localized tenderness over the insertion of the supra-spinatus tendon.

TREATMENT

- **Ask the patient to avoid** powerful contraction of the involved muscles.
- **Local injection** of Hydrocortisone & local anesthetics.
- **Surgical release** if supra-spinatus tendon is calcified

5- Volkmann's ischemic contracture

DEFINITION

Massive infarction of the muscles of forearm from injury of brachial artery in case of supra-condylar fracture humerus

CLINICAL PICTURE

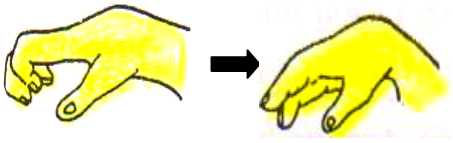
Early **Picture of acute ischemia** of forearm & hand (**6Ps**)
Pain, **P**allor, **P**aralysis, **P**araesthesia, **P**ulselessness & **P**rogressive coldness.

Later on

① **Deformity :** Flexion of wrist & inter-phalangeal joints & extension of metacarpo-phalangeal joints.

② **Atrophy** of forearm muscles.

N.B. : Special test :
The flexors of fingers are short so when the wrist is flexed, the fingers can be passively extended



TREATMENT [**Muscle sliding operation**]

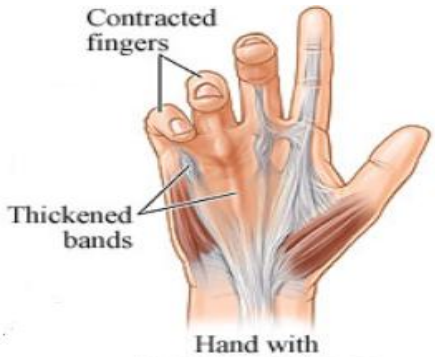
Sliding the origin of flexor muscles from the medial epicondyle downwards to the ulna.

6- Dupuytren's contracture

This is an **idiopathic** disorder that is also known as **palmar fasciitis**

PATHOLOGY

The disease is characterized by progressive thickening and contraction of the palmar aponeurosis.



Hand with Dupuytren's disease

AETIOLOGY

Idiopathic (some cases are familial)

CLINICAL PICTURE

- **Dupuytren's contracture** affects the medial side of the palmar aponeurosis.
- It starts as a **nodule** at the base of the ring or little finger. This is followed by contracture
- **The flexion deformity** affects the metacarpo-phalangeal and the proximal inter-phalangeal joints. The distal inter-phalangeal joints are **free** .
- The palm reveals a **firm nodule**, 1-2 cm proximal to the base of the ring finger.

TREATMENT

- ***Early cases :*** Physiotherapy.
- ***Late cases :*** Subcutaneous fasciotomy

7- Ingrowing toe nail

INTRODUCTION

DEFINITION

- Nail side curls inward causing injury and infection of nail fold.

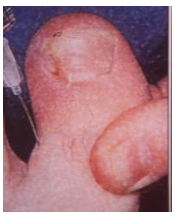
CAUSES

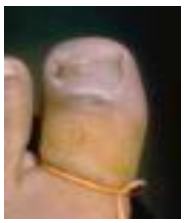
- It may result from tight shoes or cutting the nail short & in convex pattern.

CLINICAL PICTURE

- Mainly affect the **big toe**
- Patient represents by painful red swollen nail fold which may show infected granulation tissue

✱ ANAESTHESIA





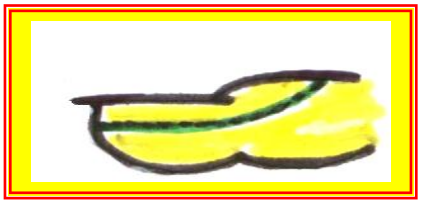
"**Ring anesthesia**"

without Adrenaline

(Local infiltration at the root of the big toe)

✱ INCISION

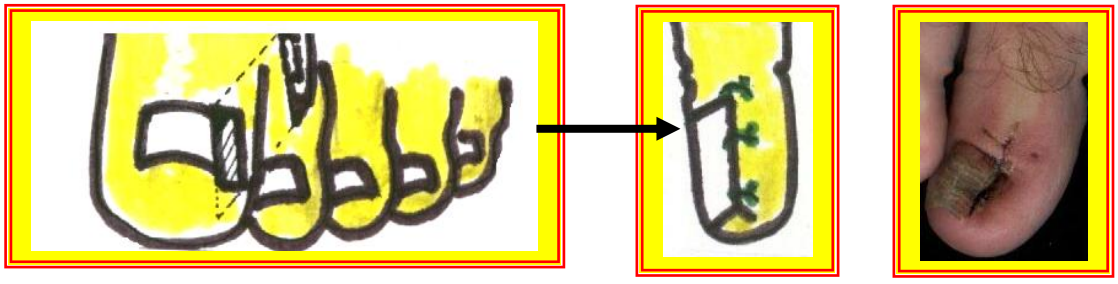
① **Longitudinal one** via the affect side of the nail deep to the bone & extended proximally to the nail root.



② **Another one** is made through the skin by the side of lesion down

✱ STEPS

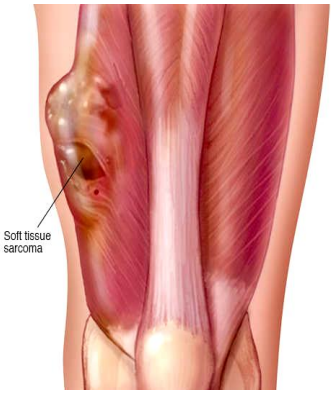
1. **Excise** a wedge of tissue between the 2 incisions.
2. **The gape** may be left open to be healed by granulation tissue or closed by 2-3 interrupted sutures



8- Soft tissue sarcoma

INCIDENCE

- Account for **1%** of all malignancies



RISK FACTORS

- **Lymphangiosarcoma**
  my develop on top of post-mastectomy
- **Neurofibrosarcoma**
  may develop on top of neurofibromatosis

PATHOLOGY

SITE

- Arises in the limbs, pelvic girdle or retroperitoneum

N/E

- Well defined false capsule from compressed surrounding tissue
- Fleshy in consistency with central areas of hemorrhage and necrosis

M/P

Primitive multinucleated mesenchymal cells which may differentiate into :

- **Lipo**sarcoma
- **Angio**sarcoma
- **Fibro**sarcoma
- **Leiomyo**sarcoma
- **Rhabdomyo**sarcoma
- **Synovial** sarcoma

SPREAD

- ***Local spread :*** by infiltrating the surrounding
- ***Blood spread :*** mainly to the lung.

CLINICAL PRESENTATIONS

1. Gradual **enlarging swelling** over months
2. The swelling is **painless**
3. Tumor consistency may be **soft** or **firm**
   depending on the amount of deposited collagen.

DIFFERENTIAL DIAGNOSIS

1. Benign soft tissue tumors like **lipoma**
2. Deep seated **hematoma**
3. **Bone tumors** and **lymphomas**

INVESTIGATIONS

1. ***Biopsy :*** FNAC or open
2. ***CT scan :*** to assess the extent of infiltration
3. ***Chest x-ray :*** for pulmonary metastasis

TREATMENT

A. Operable cases

- Radical surgical excision & Post-operative radiotherapy

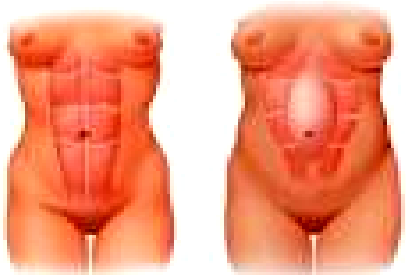
B. Inoperable cases

- Palliative surgery ± Chemotherapy

9- Desmoid tumor

DEFINITION

- **Locally** malignant tumor
  arising from the fibers of anterior rectus sheath.



INCIDENCE

- Multipara female (**80 %**).
- Common with (**Gardener's syndrome**)

N.B. : Gardner's syndrome : Familial polyposis coli + osteoma + sebaceous cyst + desmoid tumor.

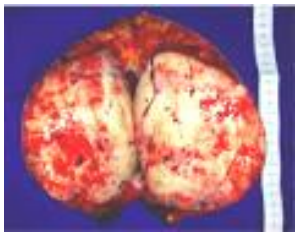
AETIOLOGY

- Due to previous surgical incision or overstretching of anterior rectus sheath.

PATHOLOGY

Site

- It arises in the rectus sheath.
- It is usually arises to one side **never** in midline.



N/E

- Cut section shows parallel fibers like tendon and hence the name (Desmoid tumor).

M/P

- It resemble to fibroma but with giant body cells

SPREAD

- **Only local**, early to mother muscle then to surrounding.

CLINICAL PICTURE

1- Slowly growing, hard (painless swelling in the lower abdomen with ill defined edge.
2- At site of a previous abdominal scar.
3- It can moves side to side & becomes fixed on contraction of the muscle.

INVESTIGATIONS

CT scan & MRI

TREATMENT

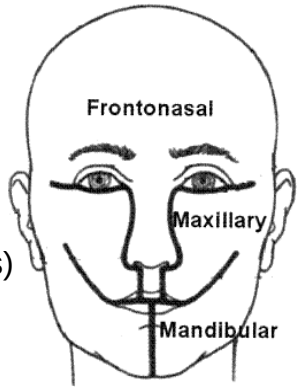
Surgical excision with 1 inch safety margin including the whole thickness of the muscle & the defect in the abdominal wall is repaired with Prolene mesh.

Head Surgery

HEAD SURGERY

DEVELOPMENT OF THE FACE

THE FACE DEVELOPED FROM 5 PROCESSES



Frontonasal process

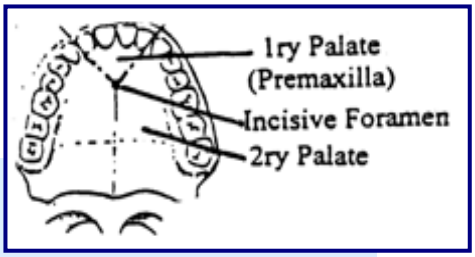
① Forehead & nose
② **Philtrum** (midline depression of upper lip)
③ **Pre-maxilla** = **1ry palate**
(V-shaped at ant. part of upper jaw carry **4** incisors)

2 Maxillary processes

① Cheeks (upper parts)
② Upper lip (except philtrum)
③ **2** Palatine processes on each side fuse together → **2ry palate**

2 Mandibular processes

① Part of cheeks (that cover mandible)
② Lower lip
③ Mandible



Don't forget

- **THE PALATE** is formed by fusion of
  - **1ry palate** (pre-maxilla) from frontonasal process
  - **2ry palate** from **2** maxillary processes

N.B The incisive foramen mark the junction of the 2 palates

- **THE LIPS** are formed of
  - **Upper lip** → **Philtrum** from frontonasal process
    → **Other parts** from **2** maxillary processes
  - **Lower lip** from **2** mandibular processes

CONGENITAL ANOMALIES

A- Abnormalities due to failure of fusion

1- Facial clefts :

① **Craniofacial cleft :** Rare due to failure of fusion between frontonasal & maxillary processes.
② **Cleft lip**
③ **Cleft palate**

See later

2- Macrostomia

3- Pre-auricular sinus

B- Abnormalities due to excessive fusion

1- Narrow palpebral fissures

2- Microstomia

C- Dermoid cyst

1. Cleft lip & Cleft palate

Cleft Lip

Predisposing factors

- Prenatal exposure to x-rays or viral infections or drugs as alcohol, anticonvulsants ….etc.
- **+ve** Consanguinity & familial tendency.

Effects on function (**Complications**)

Cleft lip does not interfere with suckling but may be associated with

① Abnormal teeth growth.

② Psychological upset of the parents .

Pathological types

Unilateral Complete

Bilateral Complete

1- Cleft upper lip

Bilateral or unilateral

1- **Bilateral 85 % :** Due to failure of development of the philtrum

2- **Unilateral 15 % :** Due to failure of fusion between maxillary process on one side & frontonasal process on other side.

Partial (Incomplete) or complete

Whether the cleft extends in the floor of the nostril or not

Unilateral Incomplete

Simple or alveolar

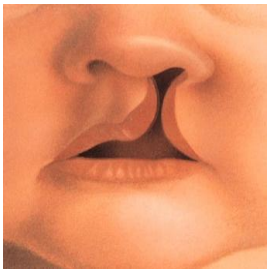
i.e. Associated with cleft palate or not

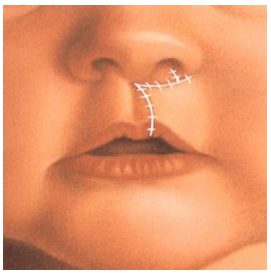
2- Cleft lower lip (**Very rare**)

Median type Due to failure of fusion between the 2 mandibular processes

Investigation **U/S in utero** during ante-natal care

Treatment (**Plastic operative repair**)





Aim of treatment

To improve appearance & to prevent complications

Timing of repair

10 gm % (Hb), **3 - 6** months (age) & **10** pound (weight)

Principles

① Paring the edges.

② Releasing incision in the gingivo-labial sulcus to have lax flaps.

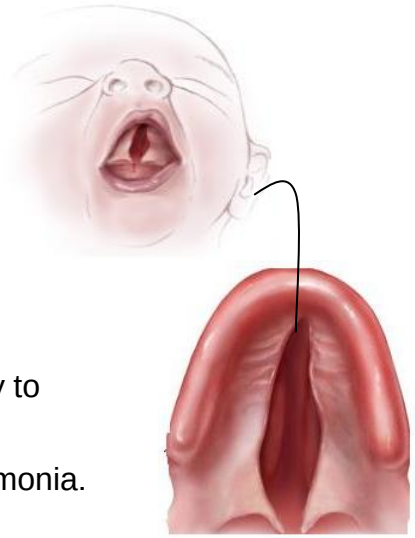
③ Symmetry of lip without vertical shortening with minimal scaring using **Z-plasty**

④ Suture in (**3** layers) of lip (skin, muscle & mucous membrane)

N.B :
- **Incomplete cleft lip** should be transformed into complete
- **If cleft lip is associated with a cleft palate**. cleft lip is repaired first

Cleft Palate

PREDISPOSING FACTORS



- Prenatal exposure to x-rays or viral infections or drugs as alcohol, anticonvulsants ….etc.
- **+ve** Consanguinity & familial tendency.

EFFECTS ON FUNCTION (**Complications**)

Cleft palate may be associated with

① Abnormal teeth growth.

② Psychological upset of the parents .

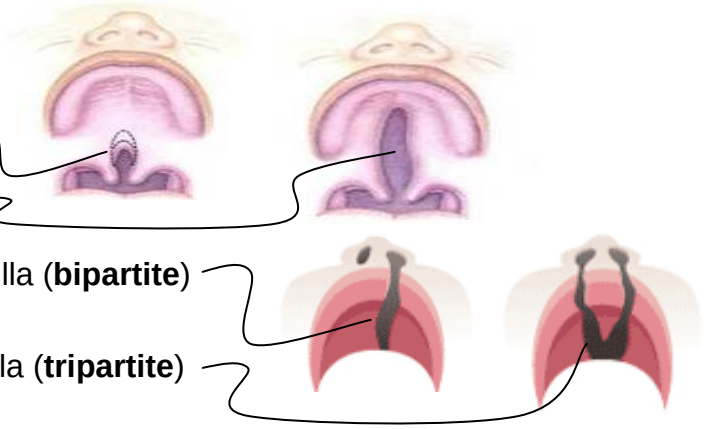
③ Impairment of normal suckling, due to inability to create - ve intra-oral pressure

④ Regurgitation predisposes to aspiration pneumonia.

⑤ Recurrent otitis media ➔ hearing loss

⑥ Speech defect 2^{ry} to hearing loss or nasal tone .

PATHOLOGICAL TYPES



① Cleft uvula.

② Cleft soft palate.

③ Cleft soft & hard palate.

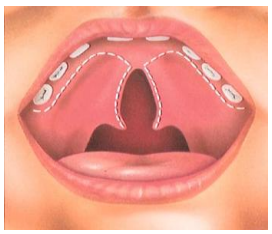
③ Complete cleft palate + **one** side of pre-maxilla (**bipartite**)

③ Complete cleft palate + **two** sides of pre-maxilla (**tripartite**)

TREATMENT (**Plastic operative repair**)

AIM OF TREATMENT

To achieve adequate speech & dentition.



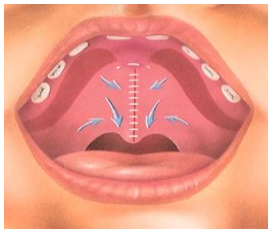
TIMING OF REPAIR

At the age of **1 - 1.5** years before phonation

PRINCIPLES

① Paring the edges.

② Releasing incision in the mucoperiosteum creating lax flaps



③ Fracture of the head of hamulus to relax the tensor palati muscle .

④ Suture the defect

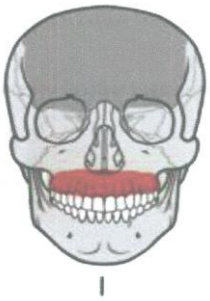
POST-OPERATIVE

Speech therapy

2. MAXILLOFACIAL INJURIES

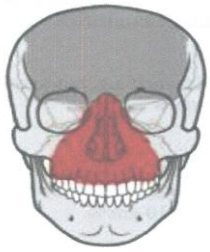
CAN BE DIVIDED INTO (**3 Parts**)

- **The upper face :** The frontal bone & frontal sinus.
- **The mid-face :** The nasal, zygomatic & maxillary bones.
- **The lower face :** The mandible.



A- Fractures of the maxilla

LE FORT CLASSIFICATION (**3 Varieties**)

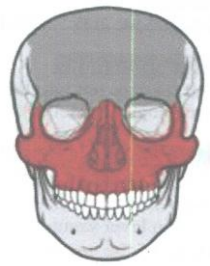


LE FORT I A transverse fracture above the level of the teeth.

It is treated by inter-maxillary fixation to the inferior orbital margin by wire.

LE FORT II A pyraniidai fracture, traversing the base of the nose through the maxillary antrum crossing the orbit

It is treated by inter-maxillary fixation to the zygomatic

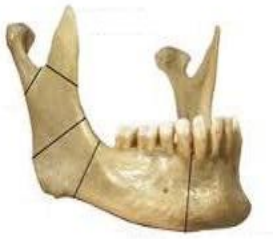


LE FORT III A cranio-facial disjunction i.e., separation of the facial bones from their cranial attachment.

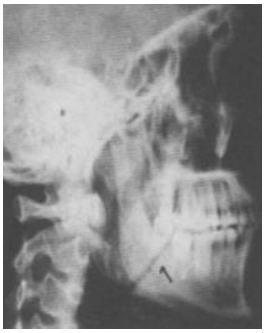
It is treated by correction of nasal & zygomatic fracture of the maxilla as in **Le Fort II**.

B- Fractures of the mandible

SITE



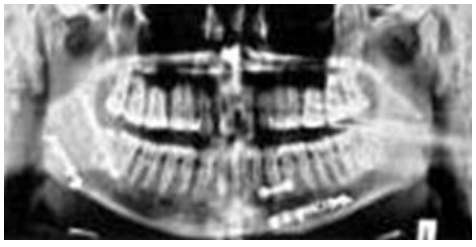
- **The body :** Fracture close to the mental foramen.
- **The coronoid process :** Least important, As the temporalis muscle prevent displacement
- **The condyle :** Indirect fracture caused by blow to the chin
- **The angle :** Minimal displacement, As the fracture line is splinted by the masseter and pterygoid muscles.



CLINICAL PICTURE

1- **Pain** on attempts to open the mouth.
2- **Dribbling** of blood stained saliva
3- **Impairment** of speech & swallowing.
4- Swelling & hematoma + tenderness & crepitus.
5- **Irregularity** of the line of the teeth.

INVESTIGATION

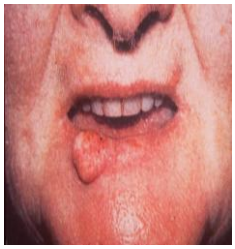


Plain X-ray shows the fracture site

TREATMENT

- **Reduction & fixation** (**3** weeks) By interdental wire & plate with screws.
- Then **mouth wash** by anti-septic solutions.

3. CARCINOMA OF THE LIP



INCIDENCE

- **Age :** > 60 years
- **Sex :** Male > female
- Smokers > non smokers

PREDISPOSING FACTORS

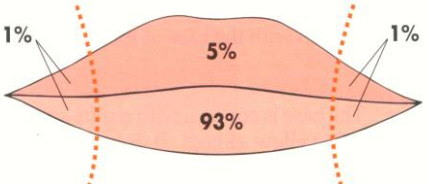
- **Chronic irritations** as **5S** (**s**pirits, **s**pices, **s**moking, **s**epsis & **$**)
- **Benign tumors** as squamous cell papilloma.
- **Leukoplakia :**

  It is a case of **hyperkeratosis** characterized by circumscribed **white** plaques & involving **wide** areas of oral mucosa

PATHOLOGY

Site

- The upper lip = **5 %**
- The angle of mouth = **2 %**
- The lower lip = **93 %**



NIE picture

- Malignant **ulcer**
- Malignant **nodule**
- Malignant **fissure**.

N.B. Diffuse infiltrating type = **woody lip**

Microscopic picture

- Squamous cell carcinoma.

SPREAD

⇨ **Direct :** In the **lip** then surroundings as mandible, maxilla ……etc

⇨ **Lymphatic : Embolization** (rare, slow & late)

① **Submental L.Ns** with central cancer lip
② **Submandibular L.Ns** with lateral cancer lip
③ **Upper deep cervical L.Ns** which drain ① & ②
or if cancer involves the angle of mouth.

⇨ **Blood :** Extremely rare & late.

CLINICAL PICTURE

Type of patient Elderly male.

Malignant ulcer The commonest

- **No :** Usually single
- **Site :** (see above)
- **Shape :** Variable.
- **Size :** Variable.
- **Edge :** Raised & everted
- **Margin :** Indurated
- **Floor :** Necrotic floor.
- **Discharge :** Bloody discharge
- **Base :** Indurated.

Involved L.Ns Stony hard, painless & 1st mobile, later on fixed.

COMPLICATIONS

- Bleeding or infection.
- Dysphagia & dysarthria.
- Upper respiratory tract infection from inhaled necrotic tissues.

INVESTIGATION Excisional biopsy.

TREATMENT

Treatment of 1ry lesion

IRRADIATION

SURGICAL EXCISION

- **Indications :**
  ① **Small** lesion
  ② Cancer on top of **$**
  ③ **Others :**
  - Recurrent lesion after irradiation
  - Resistant lesion to irradiation.
  - Lesion infiltrating bone.

- **Technique :**
  Excision with safety margin **1.5 cm** all around & plastic reconstruction of the lip .

Treatment of L.Ns

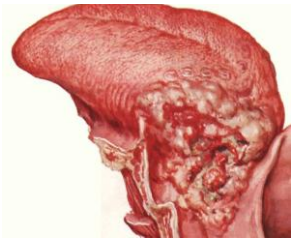
- **If no lymph nodes :** No surgery.
- **If palpable lymph nodes :** Supra-hyoid block dissection done by removal of submental, submandibular & upper deep cervical L.Ns on both side as one mass i.e. (**en block**)

PROGNOSIS

Extremely good in early cases without L.Ns metastasis

.....

4. CARCINOMA OF THE TONGUE



INCIDENCE

- **Age :** > 60 years
- **Sex :** Male > female
- Smokers > non smokers

PREDISPOSING FACTORS

- **Chronic irritations** as **5S** (**s**pirits, **s**pices, **s**moking, **s**epsis & **$**) + **s**harp teeth
- **Benign tumors** as squamous cell papilloma.
- **Leukoplakia :**

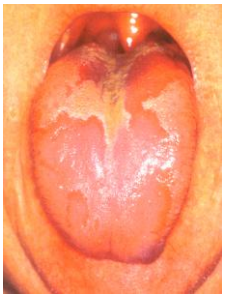
  It is a case of **hyperkeratosis** characterized by circumscribed **white** plaques & involving **wide** areas of oral mucosa

- **Erythroplakia :**

  It is a case of mucosal **atrophy** characterized by irregular **reddish** plaques & involving **small** areas of oral mucosa

- **Chronic superficial glossitis :**

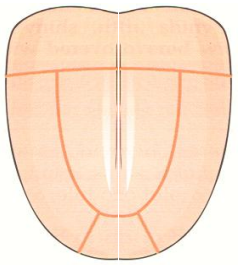
▪ **Aetiology :**
- It is a case of chronic irritation of tongue.



▪ **Pathology :**
- **Site :** It affects the anterior **2/3** of the tongue
- **N/E :** It may be ↓
  Beefy glazed tongue, leukoplakia.
  & fissuring.
- **M/P :** It may be ↓
  Hyperkeratosis,
  Acanthosis (proliferation of prickle cell layer)

▪ **Treatment :**
- Treatment of the cause of irritation .
- Non irritant diet & mouth wash
- Excision & biopsy if localized

PATHOLOGY



Site

- Lateral margin of anterior **2/3** = **50 %**
- Posterior **1/3** = **20 %**
- Less common ventral & dorsal surface of anterior **2/3** of the tongue & rarely the tip of the tongue

NIE picture

- Malignant **ulcer**
- Malignant **nodule**
- Malignant **fissure**.

N.B. Diffuse infiltrating type = **woody tongue**

Microscopic picture

- Squamous cell carcinoma.

SPREAD

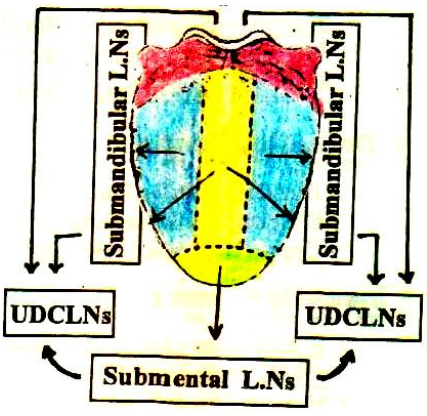
⇨ **Direct :** In the **tongue** then surroundings as mandible, gums … etc.

⇨ **Lymphatic : Embolization** & **permeation** (common, rapid & early)

① **Tip :**
Spread to submental L.Ns then to the upper deep cervical L.Ns on both sides.

② **Lateral margin of anterior 2/3 :**
Spread to the ipsilateral submandibular L.Ns then to the ipsilateral upper deep cervical L.Ns.

③ **Central part of anterior 2/3 :**
Spread to both submandibular L.Ns then to both upper deep cervical L.Ns.



④ **Posterior 1/3 :**
Spread to upper deep cervical L.Ns directly

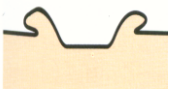
⇨ **Blood :** Extremely rare & late.

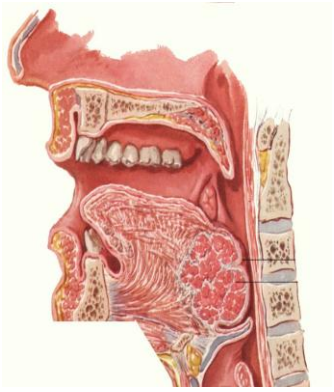
STAGING

T = Tumor	N = Lymph Nodes	M = Metastasis
T_{is} = carcinoma in situ T_0 = no evidence of tumor T_1 = < 2 cm T_2 = 2 - 4 cm T_3 = > 4 cm T_4 = base involvement (posterior 1/3)	N_0 = no evidence of L.Ns N_1 = L.Ns **< 3** cm N_{2a} = Ipsilateral & single **< 6** cm N_{2b} = Ipsilateral & multiple **< 6** cm N_{2c} = Bilateral **< 6** cm N_3 = L.Ns **> 6** cm & fixed	M_0 = no metastasis M_1 = distant metastasis

CLINICAL PICTURE

Type of patient Elderly male.



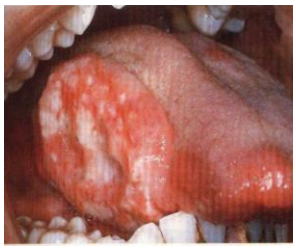


Malignant ulcer The commonest
- **No :** Usually single
- **Site :** (see above)
- **Shape :** Variable.
- **Size :** Variable.
- **Edge :** Raised & everted
- **Margin :** Indurated
- **Floor :** Necrotic floor.
- **Discharge :** Bloody discharge
- **Base :** Indurated.

Involved L.Ns Stony hard, painless & 1st mobile, later on fixed.

Also **Late presentation**

- **Pain :** localized to tongue or referred to ear through the auriculo -temporal branch of mandibular nerve.
- **Profuse salivation** which may be bloody stained .
- **Fetor oris** due to necrosis & infection.
- **Dysphagia** especially with cancer posterior 1/3
- **Dysarthria** i.e. difficulty of speech.
- **L.Ns metastasis** of the neck.



COMPLICATIONS

- Bleeding or infection.
- Dysphagia & dysarthria.
- Upper respiratory tract infection from inhaled necrotic tissues.

INVESTIGATION

- **Biopsy** from the edge of the tumor.
- **F**ine **n**eedle **a**spiration **c**ytology from cervical L.Ns.
- **CT scan :** neck & mandible.

TREATMENT

Treatment of 1ry lesion

IRRADIATION

SURGICAL EXCISION

- **Indications :**
  ① **Small** lesion
  ② Cancer on top of **$**
  ③ **Others :**
  - Recurrent lesion after irradiation
  - Resistant lesion to irradiation.
  - Lesion infiltrating bone.

- **Technique :**
  Excision with safety margin (according to the site of tumor) ⇦
  ⇨ **Carcinoma in situ :**
  - Excision with 1 cm safety margin + 0.5 cm depth.

  ⇨ **Carcinoma of the anterior 2/3 of tongue :**
  - Excision with 1.5 cm safety margin reaching up to hemi-glossectomy or near total glossectomy.

  ⇨ **Carcinoma of the posterior 1/3 of tongue :**
  - Total glossectomy (removing the whole tongue) is done.

  ⇨ **If the tumor infiltrates the mandible :**
  - **COMMANDO** operation is done.
    Combined glossectomy
    + **mand**ibulectomy and neck dissection **o**peration
  - The resulting defect is closed by pectoralis major myo-cutaneous flap + rib graft (contralateral 5th rib)

Treatment of L.Ns

- **Total block neck dissection** is done whether nodes are palpable or not as nodal involvement is early & common.

Palliative treatment (for inoperable patient)

- **Indications :**
  ① **Unresectable** 1^{ry} tumor.
  ② **Fixed lymph nodes** in the neck.
  ③ Distant **metastasis.**

- **Methods of palliation :**
  ① Palliative resection of 1^{ry} tumor if possible.
  ② Chemotherapy & radiotherapy are recommended.
  ③ Analgesic, naso-gastric feeding or tracheostomy may be required.

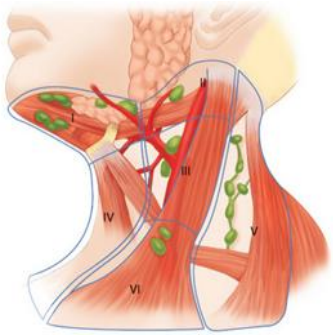
PROGNOSIS

5 years survival rate depending on ⇦
① Presence of L.Ns or not.
② Site (anterior better than posterior)
③ T.N.M staging.

N.B

RADICAL BLOCK NECK DISSECTION

DEFINITION



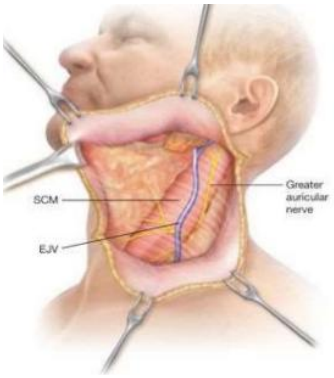
Removal of all lymph nodes on one side of the neck, in one mass **(En block).**

INDICATIONS

1- Operable 1ry malignancy of the head or neck,
with palpable malignant cervical lymph nodes

2- Prophylactic radical block neck dissection,
with malignancies that are known for their high tendency to lymphatic spread. These include carcinoma of the tongue.

TECHNICAL CONSIDERATIONS



- **Incision : GOBLET** is the most popular ⟶
- **Structures that are <u>removed</u> in radical block neck dissection :**
  - ① All lymph nodes on one side of the neck.
  - ② Other structures are also removed because of their close proximity to the cervical nodes.
    - The sternomastoid muscle.
    - The carotid sheath.
    - The internal jugular vein.
    - The sub-mandibular salivary gland.
    - The lower part of the parotid gland.
- **Structures to be <u>preserved</u> radical block neck dissection :**
  - ① The Carotid artery.
  - ② The Vagus nerve.
  - ③ The Accessory nerve.

VARIATIONS OF RADICAL BLOCK NECK DISSECTION

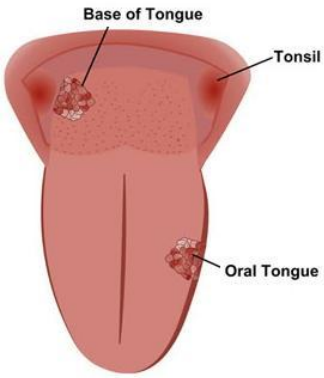
1- **Supra-hyoid block dissection**
Indicated with
- Cancer lip

2- **Radical block neck dissection**
Indicated with
- Cancer tongue

N.B : Bilateral block neck dissection :
Preservation of one jugular vein on the less affected side is
Necessary to provide adequate venous drainage from brain

5. TONGUE ULCERS

Traumatic ulcers



1- FRENULAR (POST-PERTUSSIS) ULCER
- It usually affects children with whooping cough.

2- DENTAL ULCER (See table)

Inflammatory ulcers:

⇨ **Acute**

1- DYSPEPTIC ULCER (See table)

2- HERPETIC ULCER Painless, multiple & small ulcers.

3- LICHEN PLANUS Auto immune disease affects the skin & oral mucosa.

⇨ **Chronic**

1- T.B ULCER (See table)

2- $ ULCER (See table)

3- CHRONIC SUPERFICIAL GLOSSITIS (See before)

Malignant ulcer (See before)

	DYSPEPTIC ULCER	DENTAL ULCER
• **Causes**	G.I.T troubles or follows influenza	Sharp tooth.
• **Number**	• Multiple	• Single
• **Site**	• Tip & side of the tongue.	• Side of tongue opposite sharp tooth
• **Shape**	• Rounded	• Oval
• **Size**	• Small	• Moderate.
• **Edge**	• Sloping	• Punched out
• **Margin**	• Hyperemia	• Hyperemia
• **Floor**	• Yellowish	• Unhealthy granulations
• **Discharge**	• **No** discharge	• Blood & pus
• **Base**	• **Soft**	• Indurated
• **Regional L.Ns**	• **No** lymph nodes	• Firm & tender lymph nodes
• **Treatment**	• Painting by gention vilot	• Removal of sharp tooth.

	T.B ULCER	$ ULCER
• **Causes**	Open pulmonary T.B	3ry stage (Gumma)
• **Number**	• Multiple	• Single
• **Site**	• Tip & base of the tongue.	• Dorsum of tongue in the midline
• **Shape**	• Rounded	• Oval
• **Size**	• Small	• Large.
• **Edge**	• Undermined	• Punched out
• **Margin**	• Bluish (cyanotic)	• ------
• **Floor**	• Casseation	• Leathery sloughs
• **Discharge**	• Serous	• Scanty
• **Base**	• **Soft**	• Indurated
• **Regional L.Ns**	• **Matted** lymph nodes	• ------
• **Treatment**	• Anti-T.B. drugs • Improve oral hygiene	• Anti-syphilitic drugs • Improve oral hygiene.

6. CYSTIC SWELLINGS OF THE FACE

AETIOLOGY

1- Sebaceous cyst

2- External angular dermoid

3- Mucocele of the lacrimal sac

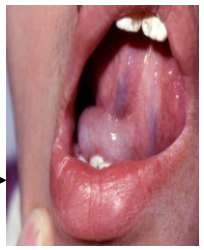
4- Meningocele

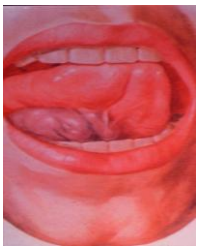
7. CYSTIC SWELLING OF THE FLOOR OF THE MOUTH

Ranula

AETIOLOGY

It is a retention cyst or extravasation cyst of sublingual gland



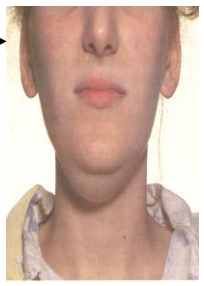


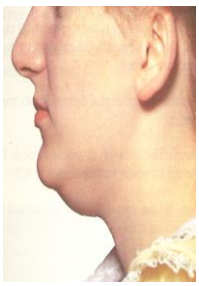
PATHOLOGY

Site

1- Simple ranula :

In one side of the floor of the mouth pushing the tongue to the other side





2- Plunging or dissecting ranula :

Dissect its way behind the posterior border of the mylohyoid muscle to appear in the neck i.e. submandibular region (**Thompson's ranula**)

Composition

It contains jelly-like material rich in mucin

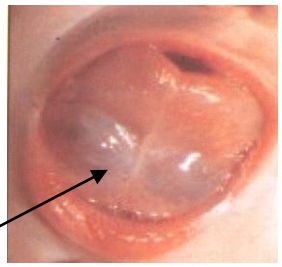
CLINICAL PICTURE

Age

Usually in **infant** or **early childhood.**

Characters

Bluish translucent large cystic swelling pushing the tongue to one side & it may cross the midline & become constricted by the franulum to give an **hourglass appearance**.



COMPLICATIONS

It may cause **dys**phagia, **dys**arthria & **dys**pnea.

TREATMENT

1- Simple ranula :

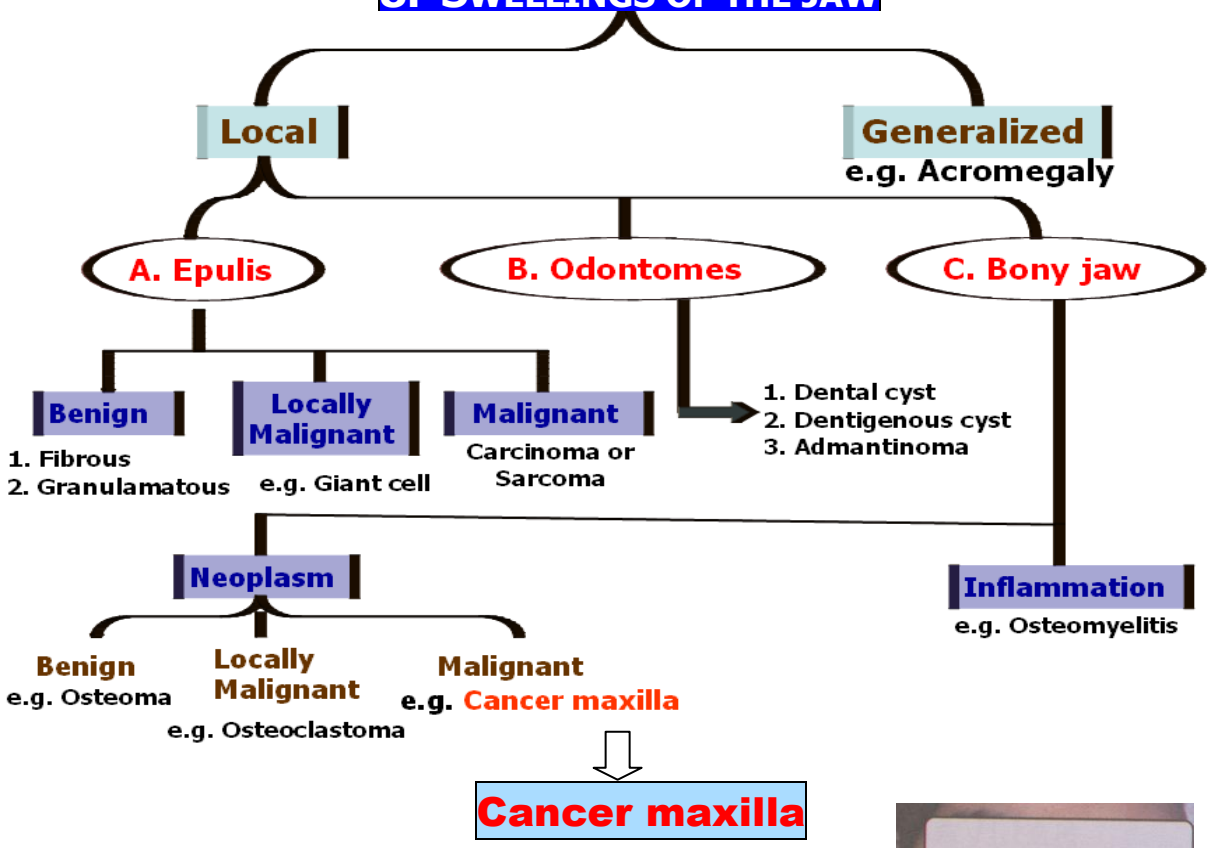
Surgical excision is very difficult. **Marsupilization** is the treatment of choice by deroofing of the cyst & suturing the wall to the oral mucosa.

2- Plunging or dissecting ranula :

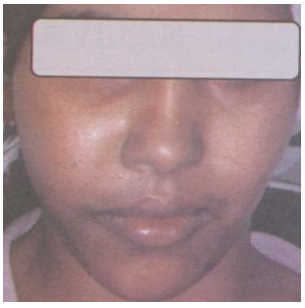
It may need cervical incision for its dissection

.

8. SWELLINGS OF THE JAW



Cancer maxilla



PREDISPOSING FACTORS

Multiple maxillary polyps & chronic sinusitis.

INCIDENCE

Male > Female & 40 years

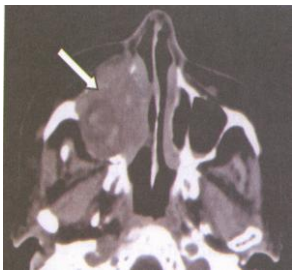
PATHOLOGY

- **Site :** At maxillary antrum.
- **N/E :** It invades by a papillary growth & rarely malignant ulcer.
- **M/P :** Squamous cell carcinoma or columnar cell carcinoma**.**

CLINICAL PICTURE

(Depends on which wall of the **maxillary antrum** is involved)

- ⇨ **Medial wall :** Unilateral nasal obstruction.
- ⇨ **Roof :** Unilateral proptosis & diplopia.
- ⇨ **Floor :** Bulging in the roof of oral cavity.
- ⇨ **Antero-lateral wall :** Bulging & swelling of the cheek.
- ⇨ **Post wall :** Encroaches on the naso-pharynx
  → change of voice & difficulty of breathing.



INVESTIGATIONS

CT scan & **MRI :** Diagnostic

TREATMENT

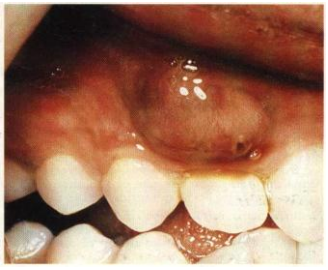
Either radical **surgical excision** or **irradiation**.

1. Epulis

An epulis = mass riding over gums

A- Benign epulides

1. Fibrous epulis



PATHOLOGY

- **Site :** Between **2** teeth.
- **N/E :** Arising from the outer **fibrous** layer of periosteum.
- **M/P : Fibrous** tissue with spindle cells & plasma cells.

CLINICAL PICTURE

The swelling is painless, well defined edge, pedunculated shape.
firm in consistency, pinkish in color & covered with intact mucous membrane.

TREATMENT

Excision of tumor with adjacent tooth + **removal** of wedge of bone with its mucoperiosteum.

2. Granulomatous epulis

PATHOLOGY

- **Site :** Around a **carious** tooth.
- **N/E :** Mass of **granulation** tissue.
- **M/P : Granulation** tissue (capillaries & fibroblasts)

CLINICAL PICTURE

The **granulation** tissue may be ⇩

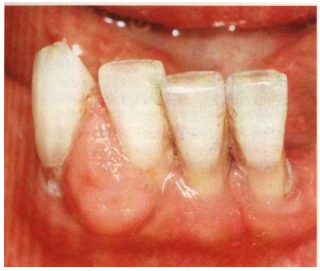
- **Healthy :** Pink, painless doesn't bleed or ooze easily.
- **Unhealthy :** Yellow, painful, bleed or ooze easily.

TREATMENTS

Extraction of the carious tooth + **curettage** of unhealthy granulation tissue.

B- Locally malignant epulides

Giant cell (myeloid) epulis



PATHOLOGY

- **Site :** Undergums (lower jaw **>** upper jaw)
- **N/E :** Arising from **osteoclastic** layer of periosteum
- **M/P :** Giant cells in a matrix of fibrous tissue.

N.B It is **not** malignant but **locally** malignant

CLINICAL PICTURE

The swelling is painless, sessile in shape, soft in consistency, violet in color from vascularity so ulceration leads to severe hemorrhage..

TREATMENTS

Wide excision with part of bone which carries the epulis.

C- Malignant epulides

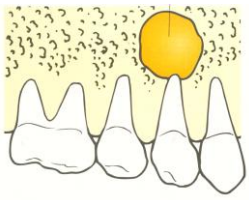
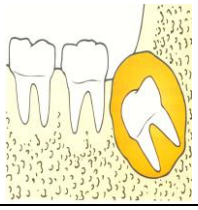
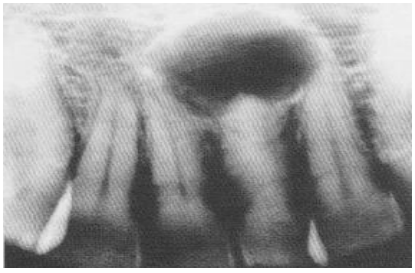
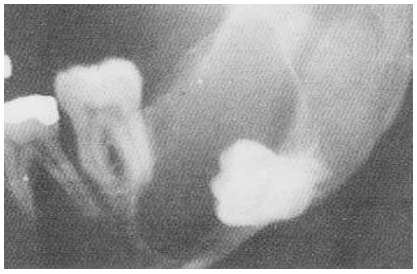
Carcinoma Squamous cell carcinoma of gums.

Sarcoma Parosteal fibrosarcoma or periosteum of mandible.

.....

2. Odontoma

These are cysts related to teeth remnant

	A. Dental cyst	B. Dentigerous cyst
INCIDENCE		
- Age	• Old	• Adult & young
- Sex	• Male > Female	• Male > Female
- Site	• Upper Jaw 	• Lower Jaw 
AETIOLOGY	• Chronic **infected** root of a tooth.	• **Non erupted** permanent tooth.
PATHOLOGY		
- Mechanism	• Chronic infection ➔ stimulation of epithelial debris of the **Malassez** ➔ proliferation ➔ mass which undergo central degeneration ➔ **dental** cyst.	• Cystic degeneration of a dental follicle ➔ **dentigerous** cyst.
- N/E	• Small, unilocular cyst. which is slowly growing expansion of the jaw on both sides **equally**.	• Expanded **outer** table of the jaw only.
- M/E	• Lined by squamous epithelium & contains **mucoid fluid** rich in cholesterol.	• Lined by squamous epithelium & contains a **missed tooth** and viscid fluid
CLINICAL PICTURE	• Painless, well defined mass related to **infected tooth** at upper jaw	• Painless, ill-defined mass related to **missed tooth** at lower jaw.
INVESTIGATION	• **Plain X-ray** shows Radiolucent area at upper jaw 	• **Plain X-ray** shows Cyst with **tooth inside** it 

TREATMENT

1- DENTAL CYST :

Extraction of the affected tooth & **excision** of the wall of the cyst.

2- DENTIGEROUS CYST :

De- roofing of cyst & lining epithelium with **missed** tooth.

C- Adamantinoma

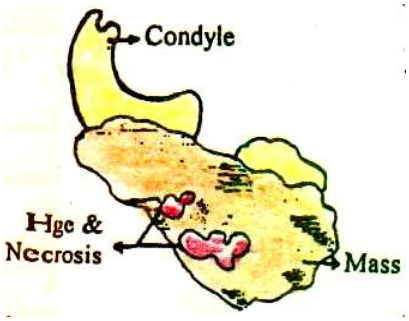
Eve's tumor

Ameloblastoma

ORIGIN " Locally malignant tumor "
Arising from (Ameloblast) = Para-dental cells of **Malassez**.

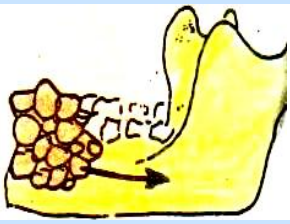
PATHOLOGY

- **Site :** Starts at angle of mandible & grows slowly in both vertical & horizontal directions.
- **N/E :** - Pink or white in color & well encapsulated.
  - Solid mass with cystic areas & almost equal lobulations.
  - Fibrous tissue trabeculations in between.
- **M/P :** Basal cell carcinoma.



CLINICAL PICTURE

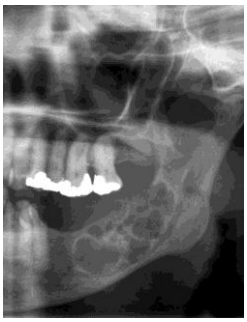
- **Age :** 20 - 40 years.
- **Sex :** Female > male.
- **Presentation :**
  - **Symptoms :**
    - Painless, lobulated, slowly growing swelling in the lower jaw .
  - **Signs :**
    - Expansion with intact overlying skin & mucus membrane
    - Egg shell crackling sensation if enlarged with no L.Ns .
    - **Later on** ① Ulceration & infection
      ② Bleeding from mucus membrane.
      ③ Falling of teeth

D.D	Adamantinoma Eve's tumor	Osteoclastoma Giant cell tumor
		
Site	• Angle of mandible.	• Symphysis menti
Growth	• Both horizontal & vertical.	• Only horizontal
Shape	• **Equal** lobulations & expanded more at outer side.	• **Unequal** lobulations & expanded equally on both sides

INVESTIGATION

1- X-ray shows fine soap bubbles appearance related to angle of mandible.

2- C.T scan & MRI



TREATMENT

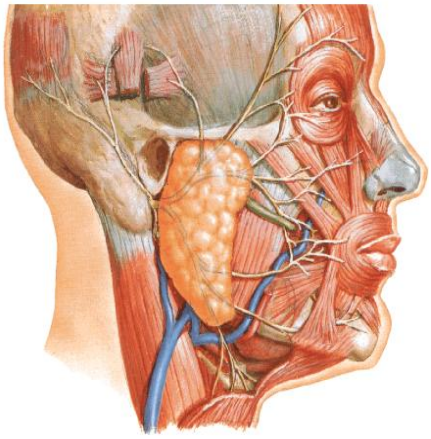
Hemi-mandiblectomy
+ **rib graft** from contra-lateral 5th rib.

9. SALIVARY GLAND DISEASES

I- Anatomy of the parotid gland

✷ **Site** between the angle of mandible & sternomastoid muscle

✷ **Parts**



(1) PAROTID GLAND

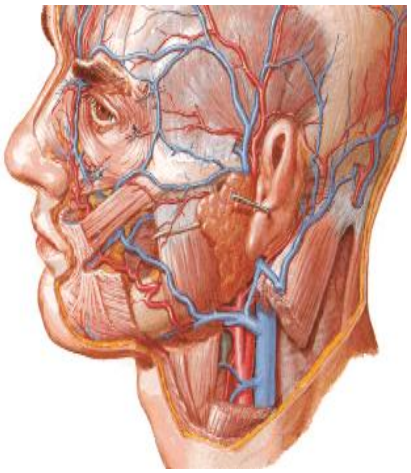
- **Relations :**

(a) Upper end is related to :

1- Superficial temporal vessels.
2- Auriculo-temporal nerve.
3- Cartilaginous part of E.A.M
4- Temporal branch of facial n.

(b) Lower end is related to :

1- Cervical branch of facial nerve.
2- Retromandibular vein.
3- It overlaps the posterior belly of digastric muscle.
4- ECA



(c) Anterior border is related to :

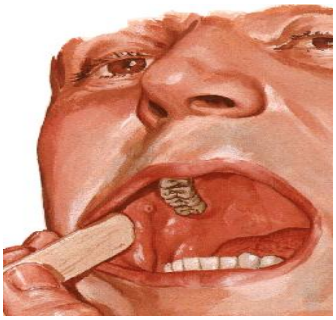
1- Temporal branch of facial nerve.
2- Zygomatic branch of facial nerve.
3- Parotid duct.
4- Buccal branch of facial nerve.
5- Mandibular branch of facial nerve.

(d) Lateral surface is related to :

1- Skin & fascia.
2- Parotid LNs.

(e) Antero-medial surface is related to :

1- Ramus of the mandible.
2- Masseter muscle



(f) Postero-medial surface is related to :

1- Mastoid process.
2- Sternomastoid muscle

(2) PAROTID (STENSEN'S) DUCT

- It is a **5** cm long
- It emerges from the anterior border
- It runs superficial to masseter m. then pierces the buccinators to opens in the vestibule of mouth, opposite the upper 2 molar tooth.

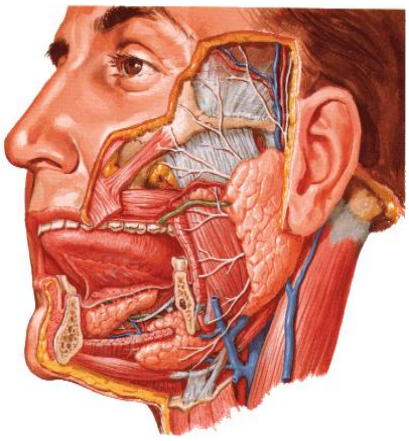
✷ **Structures within the gland**

1- External carotid artery (deepest).
2- Retromandibular vein (superficial to artery).
3- Facial nerve (superficial to the vein).

✷ Blood supply

ARTERIAL SUPPLY

External carotid artery inside the gland



VENOUS DRAINAGE

Retromandibular vein inside the gland

✷ Lymphatic drainage

Parotid L.Ns → upper deep cervical L.Ns.

✷ Surface Anatomy

(1) PAROTID GLAND (by 3 points)

A • Point on tragus.
B • Point on mastoid process.
C • Point on (one inch below & behind angle of the mandible).

(2) PAROTID DUCT *(Middle 1/3 of a line between)*

A • Tragus.
D • Mid-point between ala of nose *&* angle of mouth

✷ Points of Surgical importance

PAROTID ABSCESS should be drained by **Hilton technique**
(to avoid injury of the facial nerve).

.....

II- Anatomy of the facial nerve (VII)

✷ Site

- It passes in auditory meatus & emerges through the stylomastoid foramen.
- It enters the **parotid gland** where it gives its **5** terminal branches.

✷ Branches

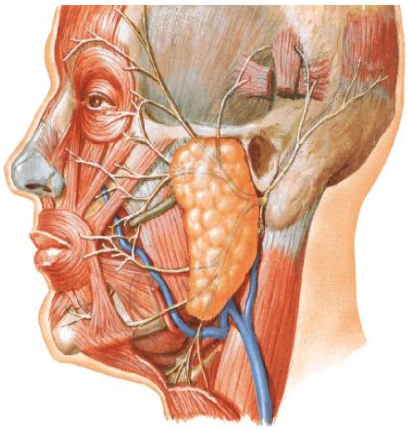
(1) IN FACIAL CANAL It gives :

Chorda tympani which carries taste sensation to the ant 2/3 of the tongue.

(2) AFTER ITS EXIT FROM STYLOMASTOID FORAMEN It gives :

1- Nerve to stylohyoid.

2- Nerve to posterior belly of digastric.



(3) INSIDE THE PAROTID GLAND It gives 5 braches :

1- **Temporal** branch

2- **Zygomatic** branch

3- **Buccal** branch

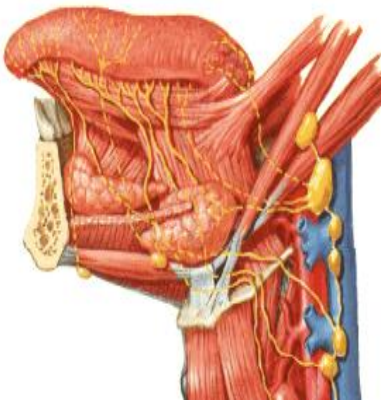
4- **Mandibular** branch

5- **Cervical** branch (which supplies the platysma)

III- Anatomy of the submandibular gland

✷ **Site** in the digastric triangle, partly below & partly deep to the mandible

✷ **Parts**



(1) SUPERFICIAL PART

Wedge shaped, extending :

Posteriorly : to the angle of mandible.

Superiorly : to mylohyoid line of mandible.

Inferiorly : it overlaps the 2 bellies of digastric muscles.

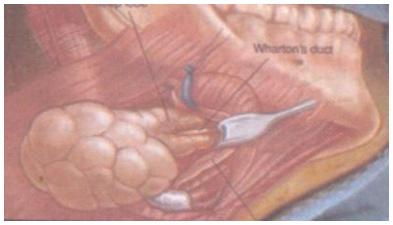
(2) DEEP PART

Small part lying deep to mylohyoid muscle

It lies between lingual nerve **above** & hypoglossal nerve **below**.

(3) SUBMANDIBULAR (WARTON'S) DUCT

5 cm long, has the following course & relations :



It arises from the deep part & passes deep to mylohyoid muscle

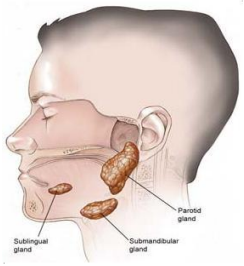
It ends by opening into the floor of the mouth

✷ **Lymphatic drainage**

Submandibular L.Ns → upper deep cervical L.Ns

.....

SALIVARY GLAND DISEASES



1. Sialectasis

Degenerative disease

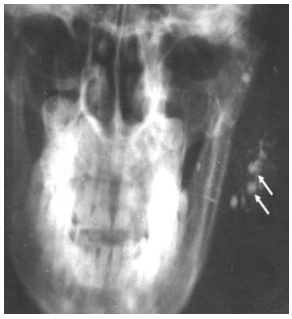
DEFINITION

Abnormal dilatation of the small branches of the salivary ducts.

AETIOLOGY

Not known but the childhood type is known to be a familial disease.

CLINICAL PICTURE



Unilateral recurrent attacks of sialadenitis.

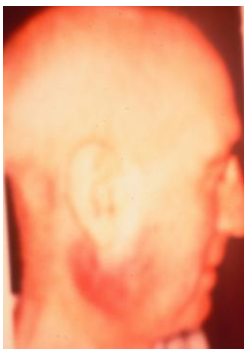
INVESTIGATIONS

Sialography : Snow storm appearance.

TREATMENT

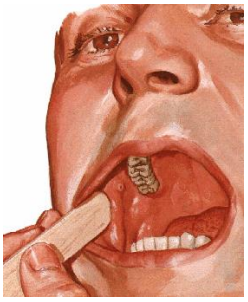
- **Conservative :** Massage the affected gland & antibiotics
- **Surgical :** Excision of the gland

2. Acute parotitis



AETIOLOGY

- **Organism :** Usually staph. aureus.
- **Route of infection :**
  - ① **Retrograde infection** from the mouth through the duct
  - ② **Blood borne** infection
- **Predisposing factors :**
  - ① Dehydration & reduced saliva with fasting or medications with atropine.
  - ② Lack of oral hygiene.
  - ③ Obstruction of the parotid duct e.g. stone.
  - ④ Typhoid & cholera (dry infected mouth)

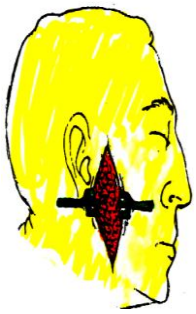


CLINICAL PICTURE

General Toxic symptoms (fever, headache,... .etc)

Local ① Painful swelling at parotid region.
② The opening of the duct is red, raised with possibility of purulent discharge

TREATMENT



- **Prophylactic :** Correct dehydration & care of oral hygiene
- **In early cases :** Antibiotic therapy
- **In fulminating cases :** i.e. **parotid abscess**

  Don't wait for fluctuation

  So - A vertical skin incision is done in front of ear .
  - The deep fascia is incised transversally to avoid injury of facial nerve & its branches.
  - A sinus forceps is introduced closed & then opened to drain the pus i.e. **Hilton's method**.

.....

3- Salivary fistula

TYPES

Internal opens in the mucus membrane.
External opens in the skin.

AETIOLOGY

1- Traumatic : Usually operative or penetrating facial injuries.
2- Inflammatory : Acute abscess + Rupture. chronic inflammation with stone.
3- Neoplastic : Malignant tumors infiltrating the skin.

C/P **Watery discharge** from ectopic site over the gland or duct

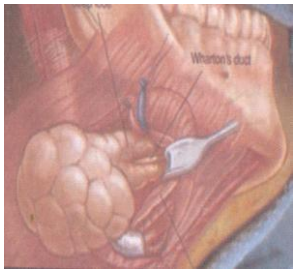
INVESTIGATIONS **Sialogram**

TREATMENT

1- If submandibular (duct or gland) : Submandibular sialadenectomy.
2- If parotid gland : Superficial conservative paratidectomy.
3- If parotid duct : Excision & end to end anastomosis

4. Salivary calculi

INCIDENCE



The **submandibular glands : parotid gland** ratio is **50 : 1**

because ① The submandibular secretion is more viscid.

② The submandibular duct lies in the floor of mouth so liable to be blocked by food particles

③ The submandibular gland drainage is inadequate as it ascends upwards.

PATHOGENESIS

Obstruction ➔ stasis ➔ infection ➔ change of pH of saliva ➔ stone

PATHOLOGY

- **Site :** The stone are impacted inside the gland or in the duct.
- **Number :** The stone may be single or multiple.
- **Nature :** The stone composed of calcium, magnesium phosphate & carbonate (radio-opaque)

CLINICAL PICTURE

Symptoms

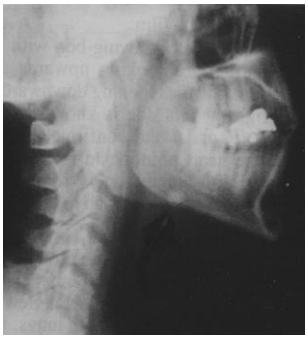
Attacks of pain (at submandibular swelling) during meals,

Signs

① Pain & size of gland are increased when patient is given a piece of lemon to suck i.e. (**Lemon test**).

② Enlarged, tender, palpable gland & **can not** be rolled over the lower border of the mandible = **D.D. from submandibular LNs**

③ The stone may be felt in the duct



COMPLICATIONS

① Salivary fistula.

② Sialectasia i.e. dilated duct.

INVESTIGATIONS

- **Plain x-ray :** Stones are 100 % radio-opaque
- **Sialography :** Dilated ducts + filling defect of stone.

TREATMENT

" **According to site of impacted stone** "

⇨ **Stone at orifice :** Removed through meatotomy.

⇨ **Stone in the duct :** Cutting directly over it through the floor of mouth.

⇨ **Stone in the gland :** Total excision of the gland i.e. Sialadenectomy.

5. Salivary tumors

CLASSIFICATIONS

Benign tumors	Malignant tumors
❶ Pleomorphic adenoma (**Mixed tumor**)	❶ Muco-epidermoid carcinoma
❷ Adenolymphoma = Adenocystic lymphoma (**Warthin's tumor**)	❷ Adeno-cystic carcinoma. (**Cylindroma**)
❸ Oxyphil adenoma (**Oncocytoma**)	❸ Acinic cell carcinoma.
❹ Monomorphic adenoma	❹ Miscellaneous adenocarinoma
	❺ Lymphoma.
	❻ Carcinoma on top of pleomorphic adenoma

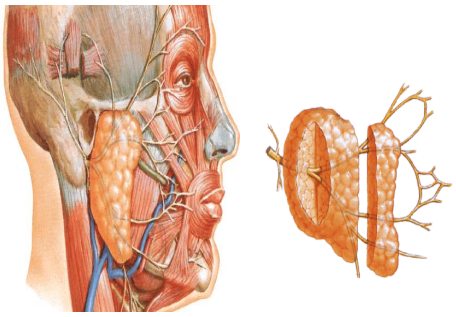
A. BENIGN TUMORS

1. Pleomorphic adenoma

Mixed parotid tumor

The commonest salivary gland tumor occurs at any age in both sex

PATHOLOGY



- **Site :** Usually arises in the **superficial part** of the parotid gland.
- **N/E :** Lobulated, well encapsulated mass. cut surface is grayish white
- **M/P :** Epithelial cells arranged in sheets with blue stroma (**thought to be cartilaginous**)

SPREAD

Strands of tumor tend to penetrate the capsule.
So there is high rate of recurrence after enucleation.

CLINICAL PICTURE

Symptoms

Unilateral, painless & slowly growing swelling

Signs

① The swelling elevate the lobule of the ear.

② The consistency is firm (Never hard)

③ Facial nerve is Not affected

④ The superficial temporal artery pulsation is felt

⑤ **If it affects the deep part**, it will push the tonsil medially.

N.B : It turns into malignancy in **3 %**

DIFFERENTIAL DIAGNOSIS (SWELLINGS IN THE PAROTID REGION)

1- Extra-parotid swellings

① Lipoma & sebaceous cyst may resemble pleomorphic adenomas.

② Pre-auricular or parotid L.Ns

③ Mandibular or maxillary tumors.

④ Hypertrophy of masseter muscle.

2- True parotid enlargement

- It is caused by salivary gland disease.

INVESTIGATIONS

- **Biopsy :** FNAC is usually enough to reach the diagnosis.
- **C.T scan & MRI** are useful in imaging the deep lobe.

TREATMENT

Enucleation of pleomorphic adenoma is easy but followed by high rate of recurrence

So the standard operations

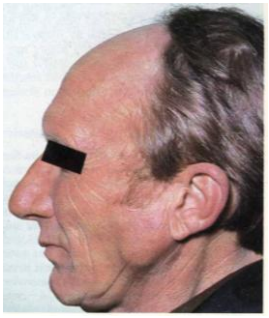
1- **Conservative superficial parotidectomy :**
Removal of superficial part of gland with preservation of facial nerve.

2- **Conservative total parotidectomy :**
As above + removal of deep part.

2. Adenolymphoma

Adenocystic lymphoma = Warthin's tumor

INCIDENCE



- Account **10 %** of parotid tumors.
- It affects males **> 40** years.

PATHOLOGY

- **Site :** Lower pole superficial part of the parotid gland.
- **N/E : Cystic** encapsulated mass & variable in size.
- **M/P :** - **Cystic** spaces with papillary projection.
  - Lined by epithelial cells surrounded by lymphoid stroma.

CLINICAL PICTURE

- It presents as a mass which is **cystic** in consistency & **not raising** the ear

INVESTIGATIONS

- **Biopsy :** FNAC is usually enough to reach the diagnose.

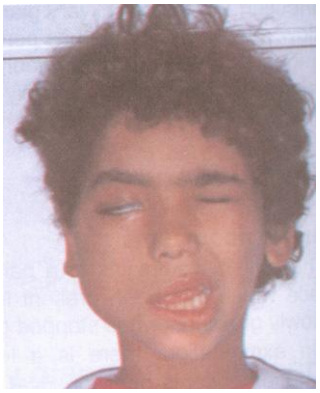
TREATMENT

- **Conservative superficial parotidectomy**

B. MALIGNANT TUMOR

INCIDENCE

- Parotid gland **>** submandibular gland



PREDISPOSING FACTORS

- **De novo** or **on top of** mixed parotid tumor (**3 %**)

PATHOLOGY

- **Site :** Superficial or deep parts
- **N/E :** Infiltrating, non capsulated mass with areas of hemorrhage & necrosis.
- **M/P : Pathological types**

① **MUCO-EPIDERMOID CARCINOMA** (**Rare** but common with **children**)
- Arises from epithelial lining the ducts of salivary gland.
- It is a malignant tumor composed of columnar (**mucoid**) & squamous (**epidermoid**)
- **3 grades of malignancy** (low, intermediate & high).
- **Treated by** removal of gland + radiotherapy.

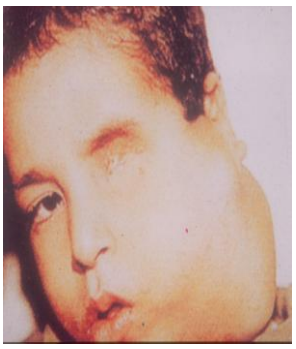
② **ADENO-CYSTIC CARCINOMA (CYLINDROMA)** (The **commonest**)
- Arises as layers of cylinders of cells surrounded by hyaline material forming **cylindroma**.

③ **ACINIC CELLS CARCINOMA** (**uncommon**)
- Arises resembling the acinic cells of the parotid gland

④ **MISCELLANEOUS ADENOCARCINOMA**
- Arises by different histologic pattern e.g. anaplastic or mucous.

SPREAD

- **Direct :** To surroundings e.g. mandible, masseter …….etc.
- **Lymphatic :** Parotid L.Ns ➔ submandibular L.Ns ➔ upper deep cervical L.Ns.
- **Blood :** Late & rare to lung, bone …….etc.



CLINICAL PICTURE

Criteria of malignancy

① **Tender** mass which may be referred to ear

② **Hard** in consistency

③ **Irregular** surface with ill defined edge

④ **Enlarged** i.e. rapid rate of growth

⑤ **Fixed** i.e. infiltration of skin, muscles, vessels & nerves

N.B.: Facial nerve is affected with parotid carcinoma but **hypoglossal** & **lingual nerves** are affected with submandibular carcinoma.

Involved L.Ns Stony hard, painless & 1st mobile, later on fixed.

COMPLICATIONS

① Ulceration, hemorrhage & infection

② **Facial palsy** with cancer parotid.

DIFFERENTIAL DIAGNOSIS (SWELLINGS IN THE PAROTID REGION)

1- Extra-parotid swellings

① Lipoma & sebaceous cyst may resemble pleomorphic adenomas.

② Pre-auricular or parotid L.Ns

③ Mandibular or maxillary tumors.

④ Hypertrophy of masseter muscle.

2- True parotid enlargement

- It is caused by salivary gland disease.

INVESTIGATIONS

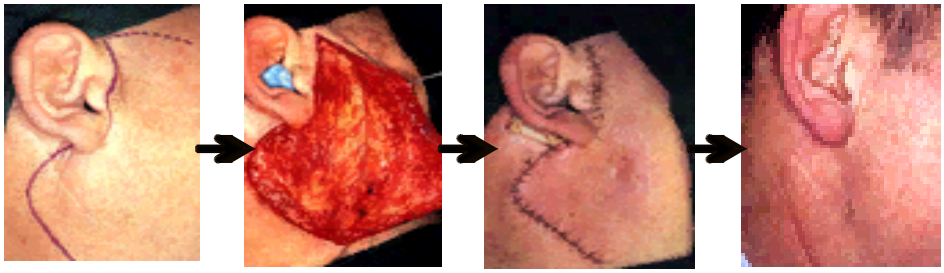
- **Biopsy :** FNAC is usually enough to reach the diagnose.
- **C.T scan & MRI** are useful in imaging the extent of the tumor.
- **Isotopic scan** with **Tc^{99} :** Salivary neoplasm shows as a **cold** spot.
- **Metastatic work up** (C.T brain, chest x-ray, bone scan & liver U/S)

TREATMENT

1- Operable

⇨ **CANCER PAROTID GLAND**

Total radical parotidectomy
+ total block dissection of neck nodes



N.B : The facial nerve is sacrificed
we will do
① Grafting by great auricular nerve

② Transposition of hypoglossal nerve then anastomosing it to the peripheral branches of facial nerve.

⇨ **CANCER SUBMANDIBULAR GLAND**

Total radical submandibular sialadenectomy
+ total block dissection of neck nodes

2- Inoperable

⇨ Palliative resection + radiotherapy

Neck Surgery

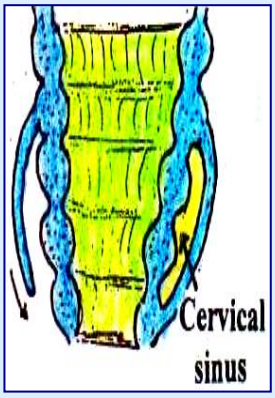
NECK SURGERY

1- BRANCHIAL CYST & FISTULA

Introduction

IN EMBRYO

- The neck starts to develop between the head & the developing heart
- **5 Ridges** develop on each side of the neck called **branchial arches**
  - The **1st** arch forms the mandible & the ear
  - The **2nd** arch forms the hyoid region
  - The **3rd** arch forms the neck over the thyroid region.



DURING EMBRYOLOGICAL DEVELOPMENT

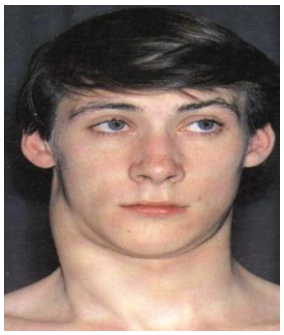
- The **2nd** arch grows rapidly covering the **3rd** & **4th** arches then it fuse with the **5th** arch
- The space between the **2nd** arch & the rest of arches turns into a **cervical sinus** which soon disappears

So **if persists it becomes branchial cyst**.

Also **Branchial fistula :** if the **2nd** arch doesn't completely fuse with the 5th arch

1. Branchial cyst

DEFINITION

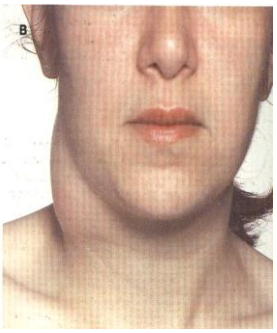


Persistent **cervical sinus**

PATHOLOGY

- The cyst is lined by squamous epithelium
- It is surrounded by lymphoid tissues which explain its frequent inflammation
- It contains mucus rich in cholesterol crystals

CLINICAL PICTURE



- **Age :** congenital but may represented at childhood or later at age of **20** years
- **Site :** Upper part of side of neck just below the angle of mandible deep to anterior border of **upper 1/3** of sternomastoid.
- **Characters :**
  ① Moderate in size about **5** cm
  ② Globular, smooth, well-defined, tense cystic & opaque
  ③ **On contracting sternomastoid**, mass bulge out

TREATMENT

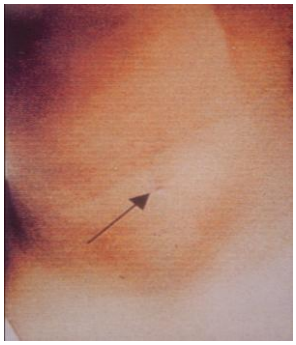
Complete excision through a transverse incision

2. Branchial fistula

Congenital branchial fistula

DEFINITION

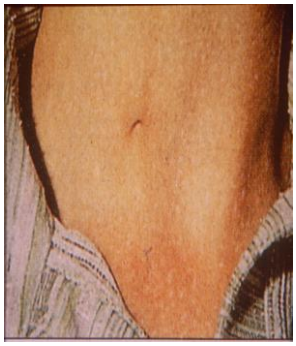
If the **2nd** arch doesn't completely fuse with the **5th** arch



PATHOLOGY

- The track is lined by squamous epithelium & extends up to the side wall of naso-pharynx i.e. " **fossa of Rosen Muller** "
- It is surrounded by lymphoid tissues which explain its frequent inflammation
- It contains mucus rich in cholesterol crystals

CLINICAL PICTURE

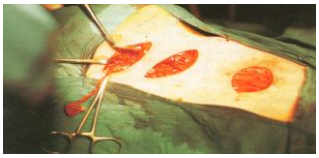


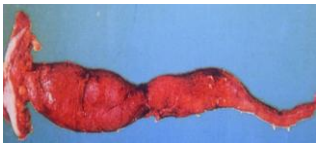
- **Age :** Present since birth.
- **Site :** External opening lies deep to **lower 1/3** of sternomastoid near its anterior border.

N.B. : The fistula passes between E.C.A & I.C.A to end highly in the pharynx behind tonsil, usually blind or rarely opened into it

- **Characters :**
  - ① It represents as a **pin point opening**.
  - ② Fistula is felt as **a thread** passing up & deeply through anterior part of sternomastoid .
  - ③ It discharges mucus or pus if infected.

N.B. : The fistula may be confused with T.B sinus





TREATMENT

Complete excision of the whole track through, multiple transverse neck incisions ; a **small one** around the external opening and **the other** at a higher level just below the Jaw.

Acquired branchial fistula

DEFINITION

Due to rupture of inflamed branchial cyst or incomplete removal of cyst.

CLINICAL PICTURE

Clinically, it differs from congenital fistula in ☜

- ① It appears in adulthood, on top of pre-exiting cyst.
- ② External opening lies high on side of neck (cyst level)

TREATMENT

Complete excision through a transverse incision.

2- CAVERNOUS LYMPHANGIOMA

Cystic Hygroma

It is not a true tumor, but tumor like i.e. hamartoma
Hamartoma = congenital malformation of vessels

AETIOLOGY

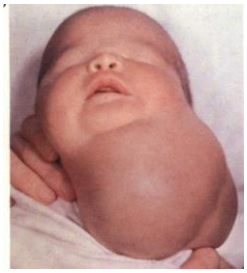
➢ **Normal development :**

The lymphatic system develops by the coalescence of multiple small lymph vesicles. A large accumulation of theses lymph vesicles are present lateral to jugular vein & called **jugular lymph space**.

➢ **Abnormal development :**

If some of the lymph vesicles of the jugular lymph sac fail to join the lymph system they become sequestrated & form a **CYSTIC HYGROMA**.

PATHOLOGY

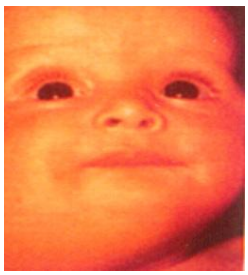


- It **consists of** multiple intercommunicating cystic lymph space.
- It is **lined by** endothelial cells.
- It **contains** clear lymph.

CLINICAL PICTURE

- **Age :** Since birth or shortly after.
- **Site :** ① Common at lower part of the post. triangle.

② The next common site is axilla alone or with neck.



- **Clinical features :**
  - The swelling is translucent to light.
  - Bluish swelling as the overlying skin is thin.
  - Soft, compressible but non pulsating mass.

TREATMENT

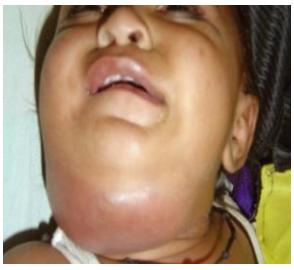
- **Excision** as early as possible.

N.B.: This could be facilitated by **preoperative injection of boiling water** in the swelling to induce fibrosis to make it smaller.

3- CELLULITES OF THE NECK

Ludwig's angina

AETIOLOGY



- It is inflammatory swelling of the **submandibular region** due to **streptococcal** infection.

CLINICAL PICTURE

- The swelling is painful, red, warm, tender and the overlying skin is edematous .

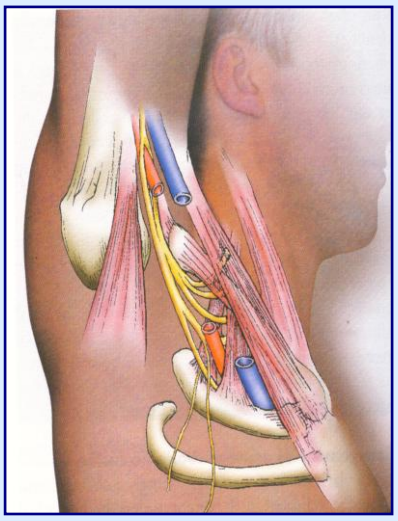
TREATMENT

- **Antibiotics & drainage** through an incision beneath the jaw

4. THORACIC OUTLET SYNDROME

Introduction

ANATOMY



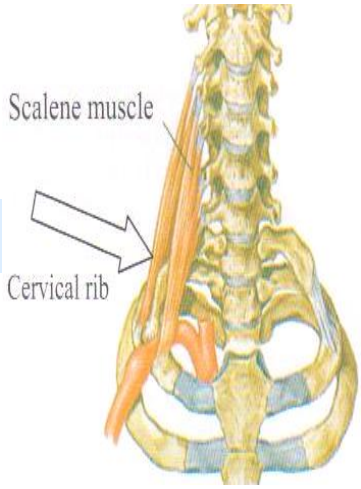
- The brachial plexus & the subclavian artery pass to the upper limb through a narrow triangle in the base of the neck.
- **This triangle is made up of** :
  - **Anterior :** Scalenous anterior muscle.
  - **Posterior:** Scalenous medius muscle.
  - **Inferior :** The 1st rib.
- **At this narrow space** compression of nerves & artery may occur.

AETIOLOGY

1- Cervical rib :

Which may be complete or incomplete. this bony structure extends from the **7th** cervical vertebra to the **1st** rib.

N.B : A **fibrous band** extending from an incomplete cervical rib & ending at **1st** rib



2- A tight scalene muscle.

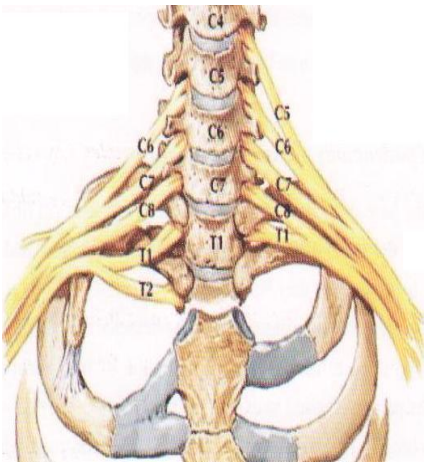
3- Post-fixation of brachial plexus :

In this case the lower root of brachial plexus arises from **T2** instead of **T1**, Thus, this nerve becomes excessively bent over the 1st rib.

COMPLICATIONS

Post-stenotic dilatation

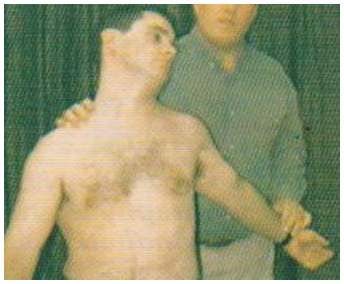
It is an aneurysmal dilatation of subclavian artery, which may send a shower of emboli to the index & middle fingers as they are the direct continuation of the brachial artery.



CLINICAL PICTURE

- **Age : > 20** years.
- **Sex :** Female > male.
- **Symptoms :**
  - **vascular :** Intermittent claudication.
  - **Vasomotor :** Raynaud's phenomenon due to irritation of sympathetic fiber
  - **Nervous :** Tingling & numbness especially in the medial aspect of forearm & hand due to compression on lower root of plexus.

- **Signs :**
  - **Bony swelling** at base of posterior triangle i.e. cervical rib.
  - **Hypothesia** & **wasting** of hand muscles may be noticed.
  - **Adson's test :**
    - ⇨ **1st palpate** the radial pulse then ask patient to turn his head & elevate his chin, then take deep inspiration.
    - ⇨ **Palpate again**, if the pulse becomes weak = **+ve** test for cervical rib.



DIFFERENTIAL DIAGNOSIS

1- Other causes of localized pressure

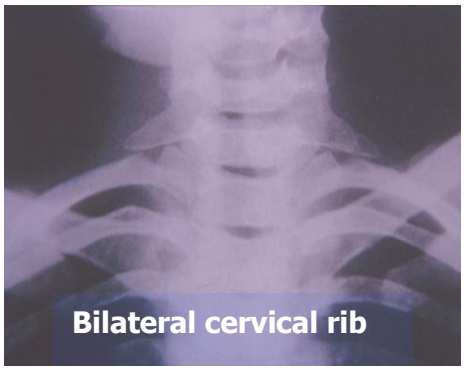
① Cervical spondylosis.
② Carpal tunnel syndrome

2- Other causes of Raynaud's phenomenon

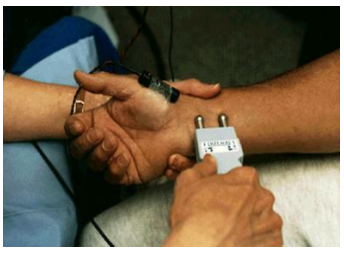
① Systemic lupus
② Collagen disease

INVESTIGATIONS

- **Plain X-ray** (neck & chest)
  may detect **bony cervical rib**.



- **Arteriography** as subclavian angiography.
- **Nerve conduction Study :**
  To detect delayed conduction between neck & forearm so help in **D.D** between the thoracic outlet syndrome & carpal tunnel syndrome

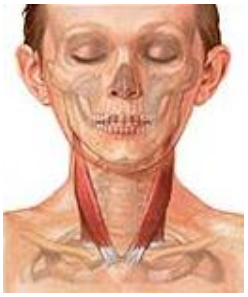


TREATMENTS

- **Asymptomatic cases :** No treatment.
- **Physiotherapy** to strengthen the shoulder muscles.
- **Surgery :**
  - ⇨ **Excision of bony cervical** including its periosteum.
  - ⇨ **Excision of 1st rib** to relieve the lower compression .
  - ⇨ **Incision** of scalenous anterior muscle i.e. **scalenotomy**.

5. CONGENITAL TORTICOLLIS

Sternomastoid tumor

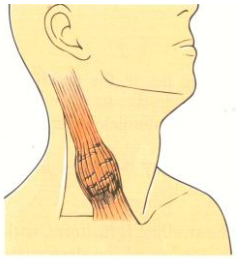


EMBRYOLOGY

- Sternomastoid muscle develops through the union of **3** somites each with its blood supply.

AETIOLOGY

- **Sometimes at birth**, an interruption of blood supply to the central portion occurs causing muscle infarction. the infarcted portion becomes swollen, hence the name **sternomastoid tumor**



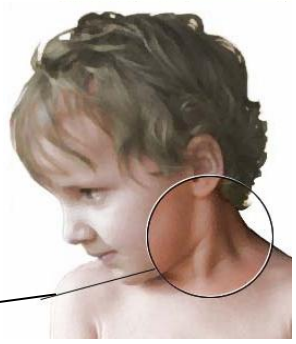
- **After a while** the infracted portion is replaced by fibrous tissue that contracts causing **congenital torticollis**.

CLINICAL PICTURE

① **At birth** there will be a swelling, which is firm in consistency, at the middle portion of the sternomastoid muscle.

② **Later,** when torticollis develops, the head will be tilted to the side of the lesion with the face looking to the opposite side.

③ **Facial asymmetry** will later occur with flattening of the side of the face at the side of the lesion



DIFFERENTIAL DIAGNOSIS

This condition should be differentiated from (**Wry neck**)

- Which is fibrositis causing spasm of the sternomastoid muscle.
- This is a temporary disorder lasting for a day or two and responds to anti-inflammatory drugs.

TREATMENT

- **Early after birth** (**Physiotherapy**)
  an attempt to prevent the development of the deformity by stretching the neck.
- **If the deformity is established :**
  Division of the sternomastoid at its lower part should be done.

6. PNEUMATOCELE

INCIDENCE

- It occurs with emphysematous patient.

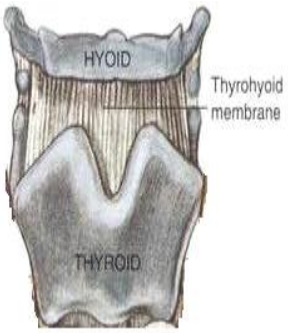
AETIOLOGY

- Herniation of **pleura** through **Sibson's fascia** which cover the pleura. due to ↑ Intra-thoracic pressure

CLINICAL PICTURE

- Cystic, compressible, resonant swelling shows expansile impulse on cough at posterior triangle of the neck.

7. LARYNGOCELE



INCIDENCE

- It occurs with with glass bowers or trumpet players or singers

AETIOLOGY

- Herniation of **laryngeal** mucosa through **thyro-hyoid** membrane due to ↑ Intra-laryngeal pressure.

CLINICAL PICTURE

- Cystic, compressible, resonant swelling shows expansile impulse on cough at midline of the neck.

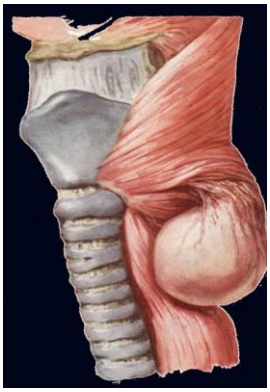
8. PHARYNGEAL DIVERTICULUM

Pharyngo-esophageal diverticulum

DEFINITION

Herniation of **pharyngeal** mucosa through a weak area in the posterior pharyngeal wall

AETIOLOGY

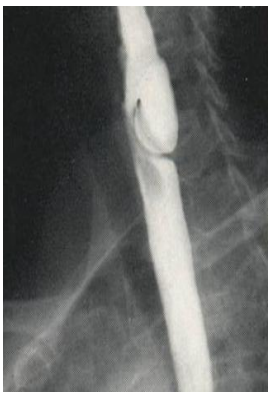


Achalasia of crico-pharyngeus muscle i.e. fails to relax during swallowing → ↑ Intra-pharyngeal pressure → herniation of the mucous membrane posteriorly.
So, food will enter the diverticulum.

CLINICAL PICTURE

Symptoms

- Progressive dysphagia with regurgitation of non digested food after meals.
- Sense of foreign body in the throat.



Signs

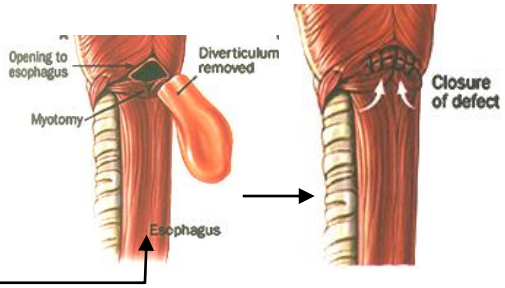
- Swelling characterized by ⇩
  ① Soft & compressible at posterior triangle
  ② Dull or resonant on percussion.
  ③ Gurgling sound can be elicited if patient swallows several glups of air.

INVESTIGATIONS

- **Manometric studies** for achalasia of crico-pharyngeus muscle
- **Barium swallow**
- **Endoscopy** (not done) because of high risk of perforation.

TREATMENT

According to the size of diverticulum ⇩



⇨ **Small :** Repeated dilatation of crico-pharyngeus muscle.

⇨ **Moderate :** Diverticulopexy by invagination & plication.

⇨ **Large :** Diverticulectomy

9. POST-CRICOID CARCINOMA

DEFINITION

Carcinoma of the **hypopharynx** at the level of cricoid cartilage.

INCIDENCE

It may occur on top of **Plummer Vinson syndrome** which is much more common in **female**.

PATHOLOGY

Site : It arises from pharyngeal mucosa.

N/E : Usually fungating mass.

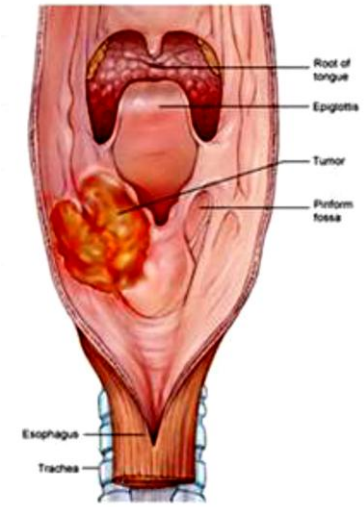
M/P : Squamous cell carcinoma.

SPREAD

Direct : To surrounding tissues.

Blood : Mainly to lung.

Lymphatic : To cervical L.Ns.



CLINICAL PICTURE

Symptoms

The early symptom is **pain** in the throat referred to side of the neck or the ear due to stimulation of Arnold nerve.

Signs

- **Inspection :** Bulge of thyroid cartilage & trachea.
- **Palpation :** Loss of laryngeal click.

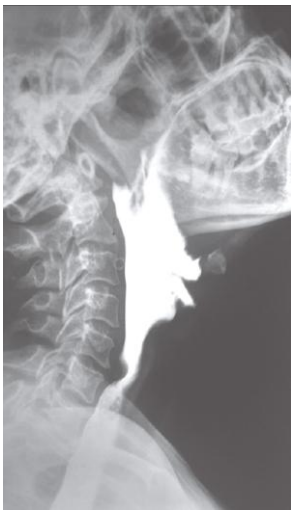
COMPLICATIONS

1- Obstruction : - Oesophageal = Dysphagia
- Laryngeal = Stridor & dyspnea

2- Hoarseness of voice.

3- Ulceration, bleeding & infection..

4- Metastasis.



INVESTIGATIONS

1- Pharyngoscopy & Laryngoscopy :

It shows the tumor & take a biopsy.

2- Barium swallow :

It shows filling defect in the pharynx.

TREATMENT

Operable

Total laryngeopharyngectomy with block dissection of L.Ns + permanent tracheostomy + oesophageal replacement.

Inoperable

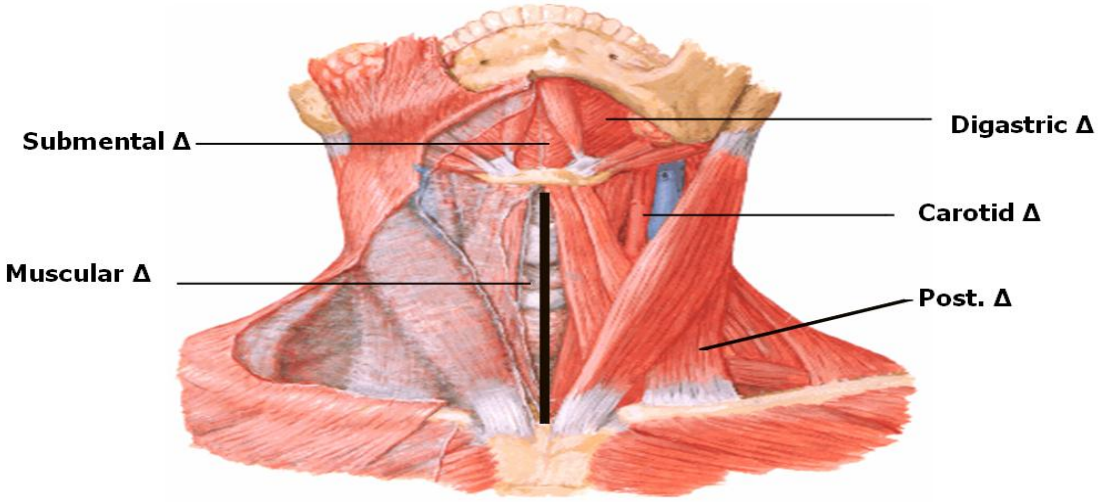
Radiotherapy.

10. DD OF A MASS IN THE NECK

Anatomy of triangles of the neck

Each side of the neck is divided into anterior & posterior triangles by sternomastoid.

Posterior triangles



BOUNDARIES

Anteriorly : Posterior border of sternomastoid muscle.

Base : The clavicle

Posteriorly : anterior border of trapezius muscle.

ROOF

1- **Skin**.

2- **Superficial fascia** (contains platysma).

3- **Deep fascia**

FLOOR

Muscles : levator scapulae & scalenus medlius.

DD OF MASS

Solid swellings

① Cervical rib. (**discuss**)

② Sternomastoid tumor (**discuss**)

③ Neurofibroma arising from brachial plexus.

④ Enlarged L.Ns.

Cystic swellings

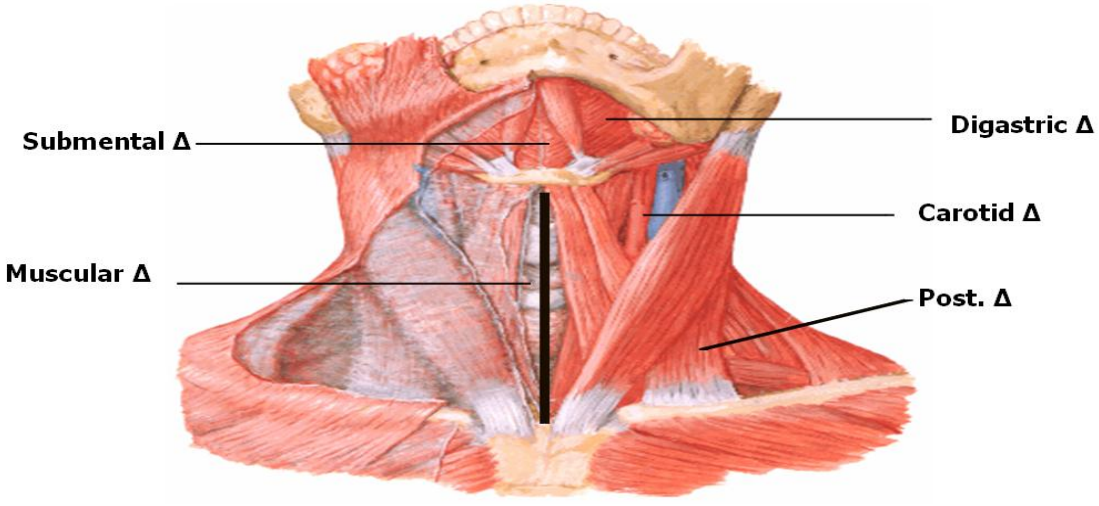
① Cold abscess.

② Pharyngeal diverticulum (**discuss**)

③ Lymphangioma (Cystic hygroma) (**discuss**)

④ Pneumatocele (**discuss**)

Anterior triangles



BOUNDARIES

Anteriorly : Midline of the neck.

Base : lower border of mandible.

Posteriorly : anterior border of sternomastoid. muscle.

SUBDIVISIONS

The digastric muscle & superior belly of omohyoid muscle divide the anterior triangle into **3.5 Δ**

1- **0.5 Submental Δ**

2- **Digastric (Submandibular) Δ**

3- **Muscular Δ**

4- **Carotid Δ**

1- Submental triangle

BOUNDARIES

Apex : Symphysis menti.

Base : hyoid bone.

On either side : anterior bellies of digastric muscle .

FLOOR

Muscles : mylohyoid muscle .

DD OF MASS

Enlarged **submental LNs**.

2- Digastric triangle

SUBMANDIBULAR TRIANGLE

BOUNDARIES

Above : lower border of the mandible.

Below : 2 bellies of digastric muscle.

DD OF MASS

1- Enlarged **submandibular L.Ns**.

2- Enlarged **submandibular salivary gland**.

For D.D : The submandibular L.Ns are multiple & can be rolled over edge of mandible **unlike** the submandibular gland.

3- Carotid triangle

BOUNDARIES

Superiorly : Posterior belly of digastric.

Inferiorly : superior belly of omohyoid.

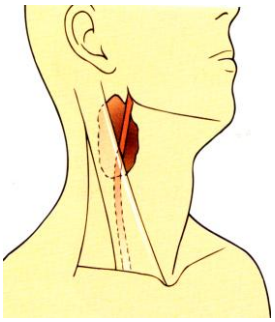
Posteriorly : anterior border of sternomastoid

DD OF MASS

Solid swellings

① Enlarged upper deep cervical L.Ns.

② **CAROTID BODY TUMOR** (**Potato tumor**)



- Its a **rare** slowly growing malignant tumor.
- It **arises** from the chemoreceptors which present at bifurcation of carotid artery
- **Its characterized by** ☟
  - Oval hard swelling with smooth surface
  - Moving from side to side but not along.
  - It is pulsating from high vascularity.
  - Pressure on swelling may cause fainting attack i.e. **Carotid sinus syndrome**
- **Investigation :** Angiography which proves widening of carotid bifurcation.
- **Treatment :** Excision of tumor with preservation of internal carotid artery.

Cystic swellings

① Cold abscess at upper deep cervical L.Ns (**discuss**)

② Branchial cyst (**discuss**)

③ Aneurysm of carotid artery.

4- Muscular triangle

BOUNDARIES

Anterior : midline of the neck.

Postero-superiorly : superior belly of omohyoid.

Postero-inferiorly : anterior border of sternomastoid.

FLOOR

Muscles : Infrahyoid muscles (**strap muscles**)

Sternohyoid, sternothyroid & thyrohyoid muscles.

DD OF MASS

Enlarged **thyroid gland**.

.....

DD OF A MASS IN THE MID LINE OF NECK

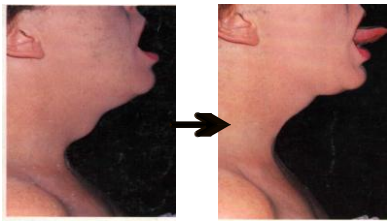
Solid swellings

① Submental L.Ns enlargement.

② Pre-tracheal & pre-laryngeal L.Ns enlargement.

③ Nodule in the isthmus of the thyroid gland .

Cystic swellings



① Cold abscess .

② Thyroglossal cyst (**discuss**)

③ Dermoid cyst : sublingual or supra-sternal.

④ Subhyoid bursitis : (It is a rare disease)

Tender & oval swelling which moves up and down with deglutition and protrusion of the tongue.

⑤ Laryngocele (**discuss**)

⑥ Cystadenoma of thyroid isthmus .

GOOD LUCK

تم بحمد الله