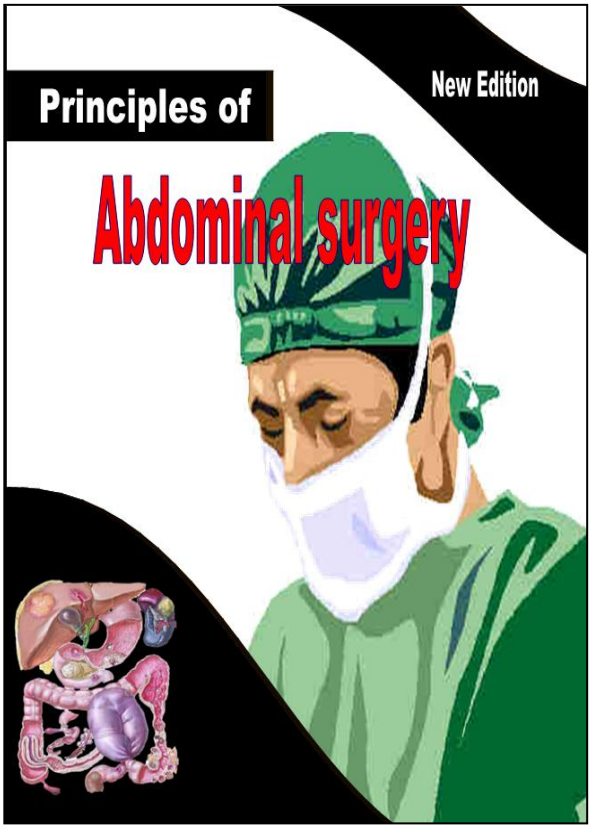
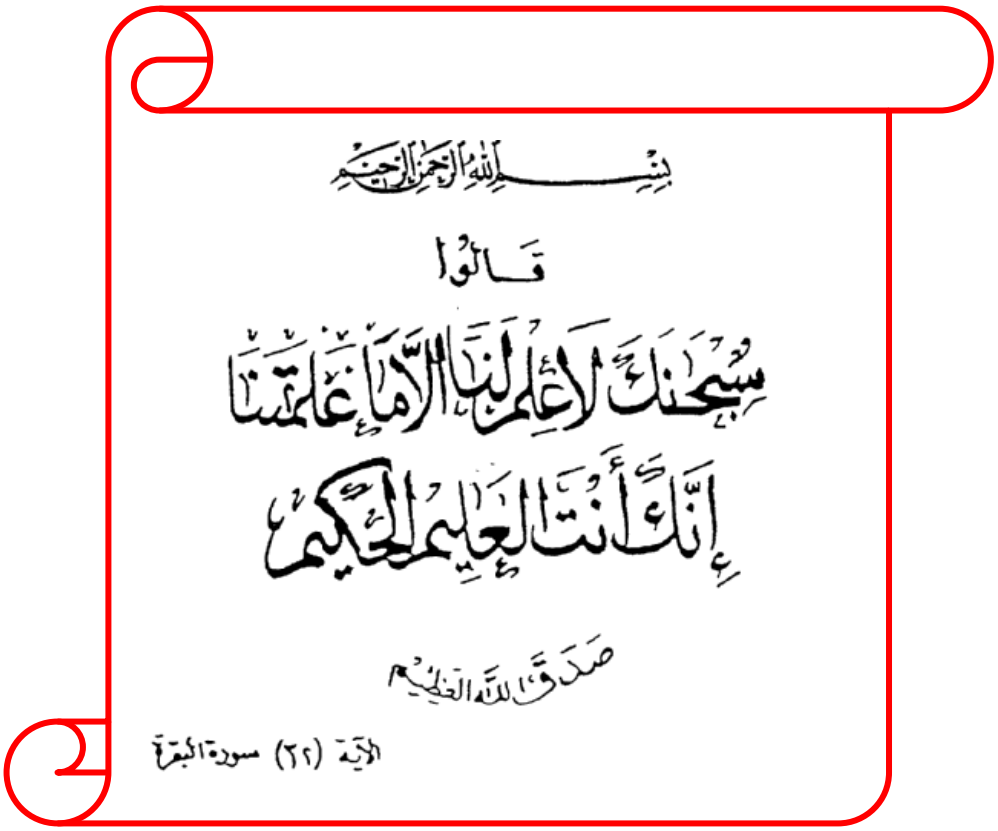
PRINCIPLES OF

ABDOMINAL SURGERY



Dr. WAEL METWALY



اللهـــم

- اللهم اجعل هذا العمل خالصًا لوجهك الكريم
- اللهم زدنى علما .... واجعل هذا العلم رافعاً 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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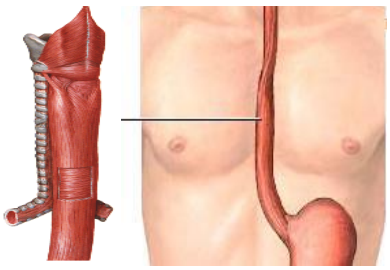
The Oesophagus

THE OESOPHAGUS

ANATOMICAL CONSIDERATIONS

Oesophagus is a muscular tube

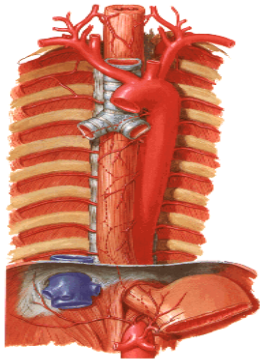


Length **25 cm**.

Begins From the lower border of cricoid cartilage (C_6 Vertebra) as a continuation from pharynx.

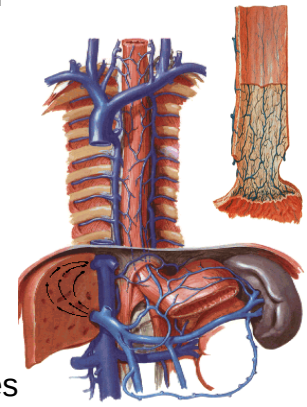
Ends At cardiac orifice of stomach

ARTERIAL SUPPLY



- **Cervical oesophagus :** Supplied by **inferior thyroid artery**.
- **Thoracic oesophagus :** Supplied by branches from the **aorta** & **bronchial branches**.
- **Abdominal oesophagus :** Supplied by oesophageal branches from lt. gastric artery.

VENOUS DRAINAGE



Sub-epithelial venous plexus Drain to **inferior thyroid vein**

Sub-mucus venous plexus Drain to **azygos** & **hemiazygos** vein

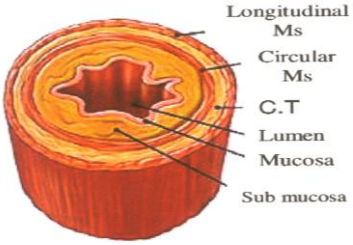
Peri-oesophageal venous plexus Drain to lt. gastric vein

LYMPHATIC DRAINAGE

- **Cervical oesophagus :** Drains into the **cervical** lymph nodes.
- **Thoracic oesophagus :** Drains into the **tracheal** & **tracheo-bronchial** lymph nodes
- **Abdominal oesophagus :** Drains into the **Lt. gastric** & **coeliac** lymph nodes.

HISTOLOGICAL CONSIDERATIONS

Oesophagus is formed of 3 layers



Mucosa Stratified squamous epithelium

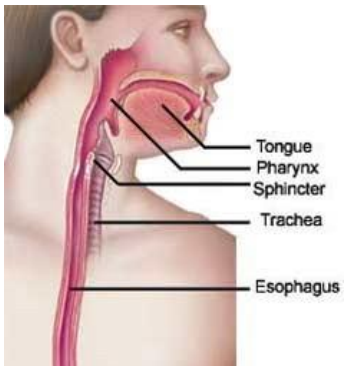
Submucosa Contains vessels & lymphatics.

Musculosa
- **Upper 1/3 :** Striated muscles.
- **Lower 2/3 :** Smooth muscles which is inner circular & outer longitudinal.

N.B: **No serosa** except (the abdominal part)
There is a layer of loose areolar connective tissues.

DYSPHAGIA

DEFINITION It means difficult swallowing



CAUSES It may be ☟

Causes in the mouth

① Stomatitis.

② Glossitis.

③ Neoplasm of tongue & cheek.

Causes in the pharynx

① Pharyngitis.

② Retro-pharyngeal abscess

③ Pharyngeal diverticulum.

④ Post-cricoid carcinoma.

⑤ **Plummer Vinson syndrome** ☟

Plummer Vinson syndrome (Rare)

Incidence Common with post-menopausal female.

Aetiology It is due to Fe deficiency anemia.

Characters **Dysphagia** (spasm) + microcytic anemia, splenomegaly, achlorohydria, glossitis, angular stomatitis & spooning of nails.

Treatment Dilatation for dysphagia & Fe therapy for anemia

Functional causes

① Neuritis of glossopharyngeal or vagus nerve.

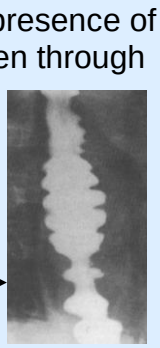
② Myasthenia gravis, tetanus or rabies.

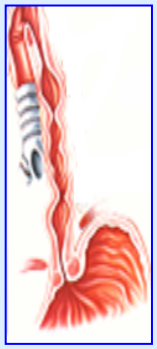
③ Hysterical.

④ Motility disorders e.g. **cork-screw oesophagus** ☟

Cork-screw oesophagus (Rare)

Aetiology The disease differ from achalasia by presence of **multiple areas of spasm** may be seen through the oesophagus.



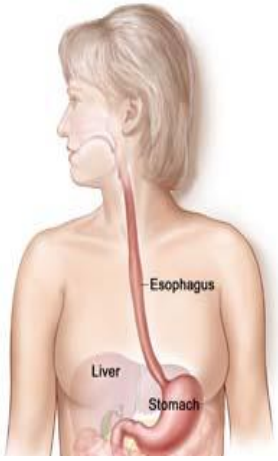


Characters **Dysphagia** (spasm) + painful swallowing felt mainly in the root of the neck.

Investigation **Barium swallow** shows → multiple areas of spasm

Treatment Dilatation or long myotomy

Causes in the oesophagus



- ⇨ **Causes in the lumen :** e.g. Foreign bodies.
- ⇨ **Causes in the wall :**
  - ① **Congenital** as oesophageal atresia
  - ② **Traumatic** as corrosive stricture .
  - ③ **Inflammatory** as reflux oesophagitis.
  - ④ **Neoplastic** as carcinoma.
  - ⑤ **Functional** as achalasia.
- ⇨ **Causes outside the wall**
  - ① Malignant thyroid (rare)
  - ② Aortic aneurysm.
  - ③ Malignant L.Ns.

INVESTIGATIONS According to suspected cause

- **Chest** (X-ray & CT scan)
- **Barium swallow** & **oesophagoscopy**.
- **Manometric** studies.

TREATMENT According to the diagnose.

.....

I- Oesophageal injuries

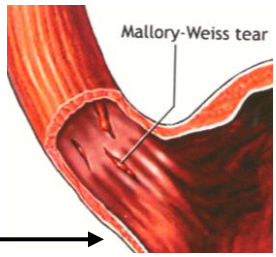
CAUSES

① **Instrumental iatrogenic :**
During oesophagoscopy or dilatation of a stricture

② **Traumatic :**
- Penetrating or blunt injuries to the neck
- **Foreign bodies.**
- Swallowing corrosives .

③ **Pathological** e.g. carcinoma.

④ **Emetogenic :**
Following forcible repeated vomiting with inco-ordinate oesophageal motility i.e **Mallory Wiess syndrome** ⟶

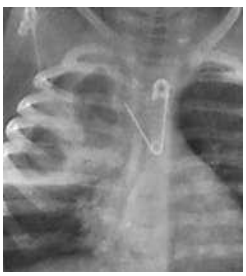


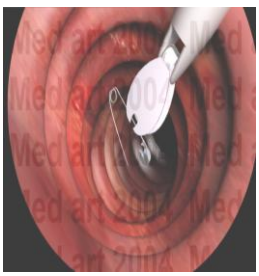
CLINICAL PICTURE

Painful dysphagia

INVESTIGATIONS

X-ray chest : reavels the cause





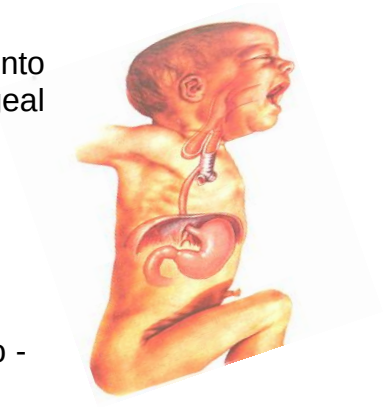
TREATMENT

- ⇨ **Thoracotomy** to repair the tear.
- ⇨ **Gastrostomy** as feeding

II- Oesophageal atresia

DEFINITION

It is a defect in the division of the proximal fore-gut into ventral tracheo-bronchial tube and dorsal oesophageal tube.



INCIDENCE 1 - 4000

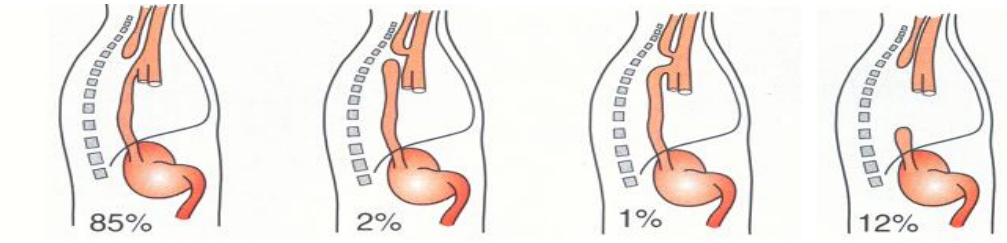
TYPES It may be

I- Atresia with fistula

① **85 %** proximal blind atresia with distal tracheo - oesophageal fistula .

② **2 %** Distal blind atresia with proximal tracheo-oesophageal fistula

③ **1 %** Both ① &② at same time.

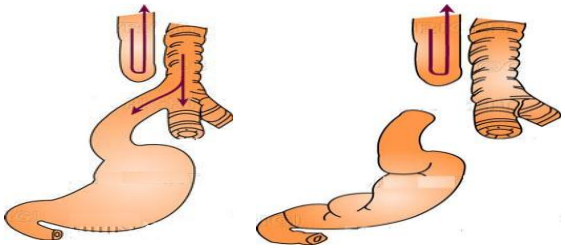


II- Atresia without fistula

④ **12 %** which is blinding both proximal & distal ends .

PATHOLOGY

- The saliva & milk → trachea → bronchopneumonia
- The acidic gastric juice → trachea → acid pneumonia (**fatal**)



CLINICAL PICTURE

Any newborn presenting with frothy saliva should be considered as having **oesophageal atresia** until proved otherwise

1- Ante-natal diagnosis :

- **Mother :** polyhydramnios.
- **Fetus :** U/S shows dilated upper pouch.

2- At birth :

- **General :** ① Milk regurgitation.
  ② Frothy saliva & attacks of cyanosis.
- **Local :** ① ***Chest*** shows pneumonia.
  ② ***Abdomen*** shows distention if atresia with fistula & scaphoid if atresia without fistula.

ASSOCIATED CONGENITAL LESIONS

VACTERL syndrome (**2** of them at least)

Vertebral : Spina bifida.

Ano-rectal : Imperforate anus

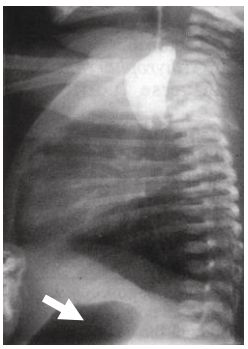
Cardiac : Fallot, ASD or VSD.

Trachea : Treacheo-oesophageal fistula.

Esophagus : Atresia.

Renal : Polycystic kidney.

Limb : Usually anomalies in the upper limb.

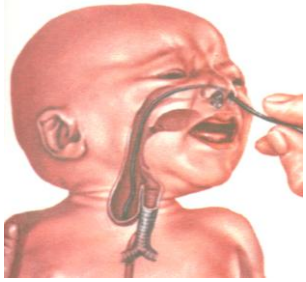


INVESTIGATIONS

1- X-ray (chest & abdomen)

After inserting a nasogastric tube. This will confirm The presence of atresia of the esophagus & presence of gastric air bubble will confirm presence of fistula.

2- Introduce catheter



No.10 through the nose, it will be arrested at about **10 cm** from the nostrils.

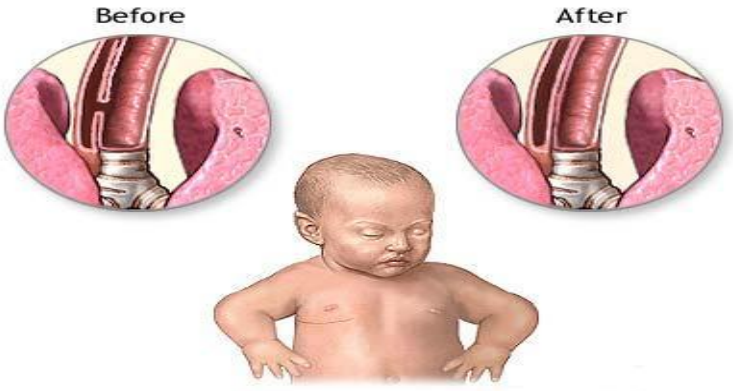
3- Fiber-optic pediatric oesophagoscopy

TREATMENT

It depend on types

1- Atresia with fistula :

Through Rt. thoracotomy. We legate the fistula then 1^{ry} anastomosis of both oesophageal ends.



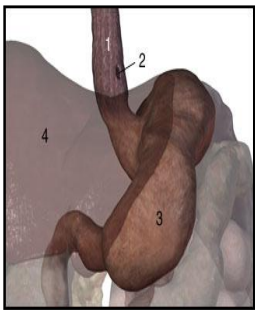
2- Atresia without fistula :

(**Usually the gap is long** i.e. **difficult anastomosis**)

So we do gastrostomy at birth then further surgery to restore oesophageal continuity is done months later.

III- Corrosive injury of the oesophagus

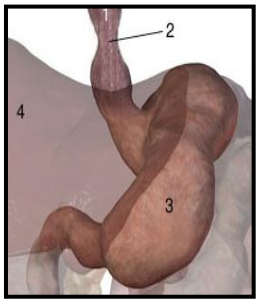
AETIOLOGY



It is a **chemical** burn due to swallowing of corrosive acids or alkalis (the common example is **potash**)

PATHOLOGY

- The extent of damage depend on the concentration or type of chemical burn & the duration of tissue contact
  - **Alkalis** ➔ liquefactive necrosis of **all** layers.
  - **Acids** ➔ coagulation necrosis of **superficial** layer.



- **The end result** is replacement of esophageal wall by fibrous tissue which leads to stricture.

CLINICAL PICTURE

- **General :** High fever ± shock.
- **Local :** Inflammation & edema of lips, tongue & oesophagus

COMPLICATIONS

① Shock.
② Late malnutrition due to dysphagia.
③ Chest infection
④ Dehydration.
⑤ Perforation ➔ mediastinitis
⑥ Malignant transformation.

MANAGEMENT **It is an emergency case**

First aid treatment

⇨ **Washing** the corrosive by water or egg or white starch.

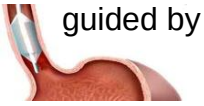
⇨ **Medical treatment** such as 1- Antibiotics to avoid 2ry (chest) infection.
2- Steroids for 6 weeks ➔ ↓ fibrosis
3- Pain killer

THEN WE DO INVESTIGATIONS

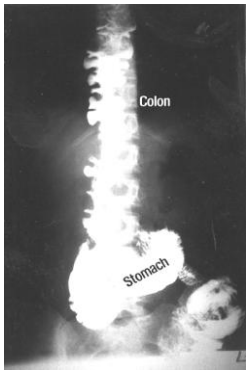
⇨ **Oesophagoscopy** (after 24 hours)

⇨ **Barium swallow** shows multiple strictures.

Subsequent treatment

⇨ **Regular dilatation :**
after 1st week when the acute stage has subsided, dilate stricture with a **BOUGIE**  guided by oesophagoscopy

⇨ **Surgical treatment :**

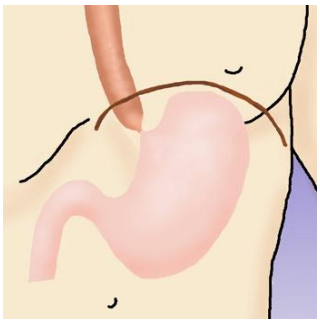


- **Indications :**
  1- Complete stenosis or failure of dilatation.
  2- Tracheo-broncho-oesophageal fistula occur.

- **Procedures :**
  Preoperative gastrostomy or I.V hyper-alimentation
  Then we do either ⇩
  - **Oesophagectomy** if short segment
  - **Colon by-pass** if long segment

IV- Achalasia of the oesophagus

DEFINITION



It is a **functional disorder** characterized by **2** defects :

1- Weak peristaltic wave in the body of the oesophagus.

2- Failure of the cardiac sphincter to relax during swallowing resulting in functional obstruction with progressive dilatation of the oesophagus.

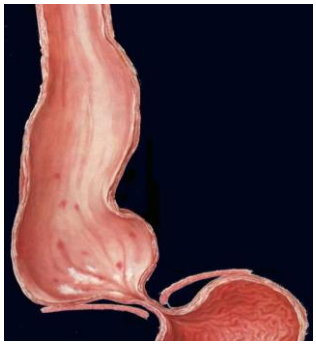
INCIDENCE

- **Age :** 20 - 40 years
- **Sex:** Male **=** female (but more common in **neurotic female**)

AETIOLOGY

- **Unknown** but may be due to degeneration of vagal fibers & the ganglia of Auerbach's plexus of the oesophagus itself.

PATHOLOGY



Because of failure of the cardia to relax in response to swallowing this leads to progressive dilatation which ends in a "**narrow**" neck below the hiatus of the diaphragm.

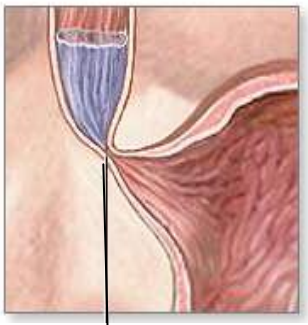
So **Stages are** 1- **Funnel** shaped oesophagus.

2- **Club** shaped oesophagus

3- **Sigmoid** shaped oesophagus.

If **Infection** ➔ fibrosis ➔ **organic** obstruction.

CLINICAL PICTURE



- **Dysphagia :** • Painless, slowly progressive.
  - More to fluid than solid
  - At 1st intermittent, then constant.
- **Regurgitation :** • **Alkaline** fluid.
  - Foul smell fluid & halitosis
  - More Nocturnal.
- **Retro-sternal discomfort** from retention oesophagitis.
- **C/P of complications :**

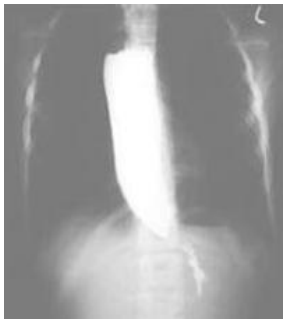
  as • Aspiration pneumonia.
  - Oesophagitis & ulceration ➔ hematemesis.
  - Malignant transformation (**5 %**) from chronic mucosal irritation
  - Pulsion diverticulum of the oesophageal wall.

DEFERENTIAL DIAGNOSIS

- **Other causes of dysphagia**

INVESTIGATIONS

A- Barium swallow

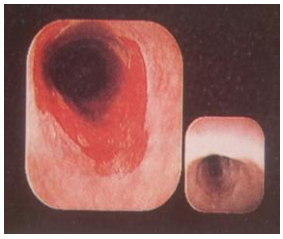


1- Moderate to huge dilated oesophagus. It may also show tortuosity called sigmoid oesophagus.

2- Smooth rounded termination of the lower end of the oesophagus (**Hen's beak**) shaped

3- Lack of gas bubble in the fundus of the stomach due to continuous stagnation of fluids in the oesophagus.

B- Oesophagoscopy **The aim is exclusion of carcinoma**



1- Huge proximal segment
2- Detection of fluid level .
3- Detection of retention oesophagitis .
4- Cardia does not relax & may be eccentric in position

C- Manometric studies (**More specific**)

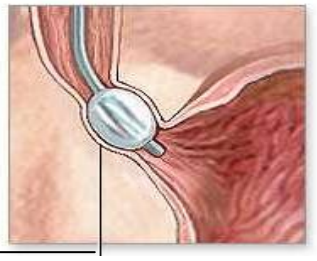
1- Weak peristaltic waves in the body of esophagus after deglutition.
2- Failure of relaxation of cardia in response to swallowing.

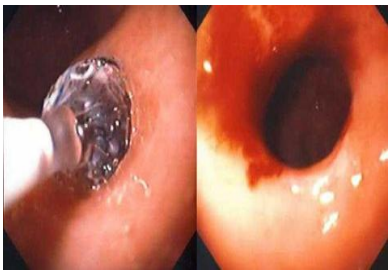
TREATMENT

I- Drugs **e.g.** Nitrates are of **limited** clinical value

II- Dilatation **By PLUMMER HYDROSTATIC BAG**

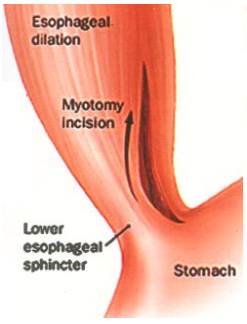
- The idea is to place a special balloon to the cardia then inflate it to produce rupture of circular muscle fibers to relieve the distal oesophageal obstruction

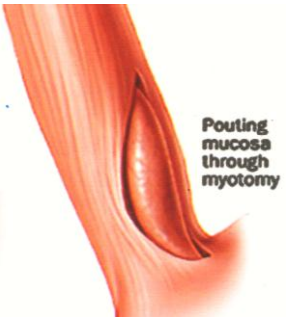




III- Heller's cardiomyotomy

- **The idea** is to expose the lower part of the oesophagus & then cut the muscle fibers completely until the mucosa bulges through





- **Done via** abdominal or thoracic approach.

- **The main disadvantage** is reflux Oesophagitis. So some surgeons prefer to do anti-reflux procedure as **Nissen's fundoplication** with this operation.

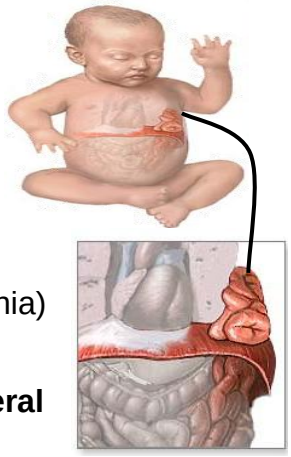
IV- Recently

- **Injection of botulinum toxin** in the wall of the esophagus at the spastic segment **is tried**

V- Congenital diaphragmatic hernia

INCIDENCE **1 - 4000**

TYPES



1. Posterior or Bochdalek (**90 %**)
It is a herniation through foramen of Bochdalek

2. Anterior or Morgagni (**not** important)
It is a herniation through foramen of Morgagni

3. Eventeration of diaphragm (**Rare** & not a true Hernia)

PATHOLOGY

- The main problem in diaphragmatic hernias are **ipsilateral** lung hypoplasia & **contralateral** lung compression

CLINICAL PICTURE

Any newborn presenting with cyanosis & scaphoid abdomen in absence of heart disease should be considered as having **congenital diaphragmatic hernia** until proved otherwise

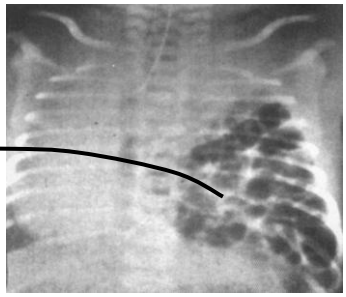
1- Ante-natal diagnosis :

- **Mother :** polyhydramnios.
- **Fetus :** U/S is diagnostic..

2- At birth :

- **General :** Cyanosis & tachycardia..
- **Local :** ① ***Chest*** **:** good heart sounds.
  ② ***Abdomen*** **:** scaphoid

INVESTIGATIONS



1- X-ray (chest)
Reveals bowel in the thorax.

2- Blood gases to assess the hypoxia.

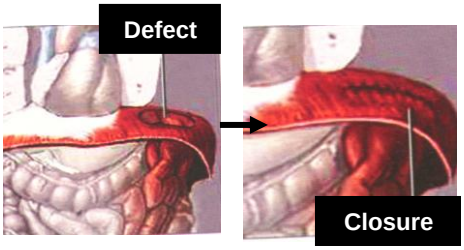
TREATMENT

1- 1st aid treatment :

① Naso-gastric tube insertion to deflate the stomach.
② Endo-tracheal tube.

2- Surgical treatment :

Through an abdominal approach, the herniated abdominal contents are reduced and the defect is closed directly or by a mesh.



3- Modern trends in treatment :

① **Intra-uterine surgical correction** has limited success

② **Extra Corporeal Membranous Oxygenation (ECMO)**
To facilitate oxygenation until the lung mature

VI- Oesophageal Hiatus Hernia

Hernia at the oesophageal hiatus

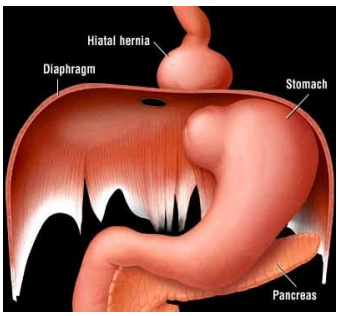
Sliding (85 %) | **Para- oesophageal (10 %)** | **Mixed (5 %)**

A. Sliding Hiatus Hernia

GORD = Gastro-Oesophageal Reflux Disease

DEFINITION

Herniation of the oesophago-gastric junction upwards in the posterior mediastinum through the oesophageal hiatus

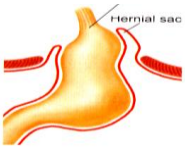


AETIOLOGY

① ↑ **Intra-abdominal pressure**
as in pregnancy, large ovarian cyst, tight corset.

② ↓ **Elasticity of the Rt. crus of diaphragm**
as in obesity & old age.

PATHOLOGY



➪ Herniation is associated with a small peritoneal sac on the Lt. side of stomach.

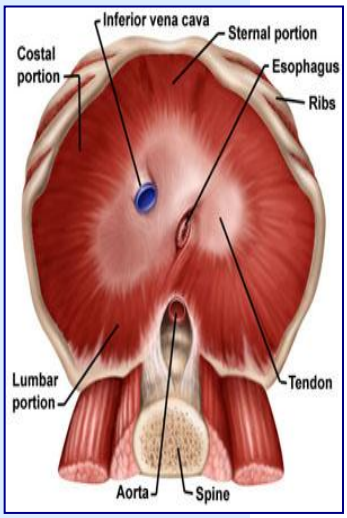
➪ Disturbance of the oesophago-gastric junction ➔ regurgitation from the stomach to lower oesophagus ➔ reflux oesophagitis.

➪ Prolonged oesophagitis ➔ replacement of oesophageal mucosa by columnar cells ➔ **Barrett's oesophagus** which is precancerous.

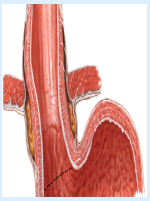
N.B : OESOPHAGEAL SPHINCTERS

Upper end : Anatomical sphincter formed by crico-pharyngeus muscle.

Lower end : Non anatomical (physiological) sphincter i.e. oesophago-gastric junction which formed by ⇘

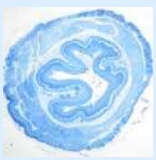


➢ **Factors prevent the reflux :**



① Presence of an intra-abdominal segment of the oesophagus which is subjected to +ve intra abdominal pressure.

② The valvular affect by (**angle of His**) at the cardio-oesophageal junction



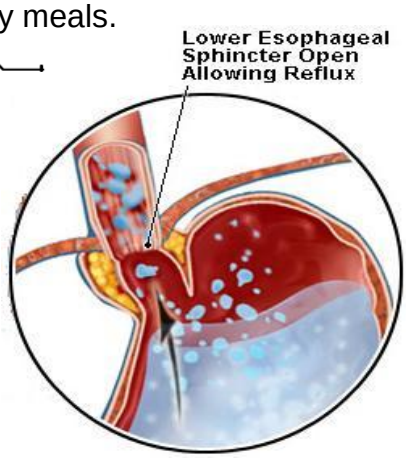
③ The **rosette-like** arrangement of the cardiac gastric mucosa

④ The **pinch-cock** action of the Rt. crus of the diaphragm .

CLINICAL PICTURE

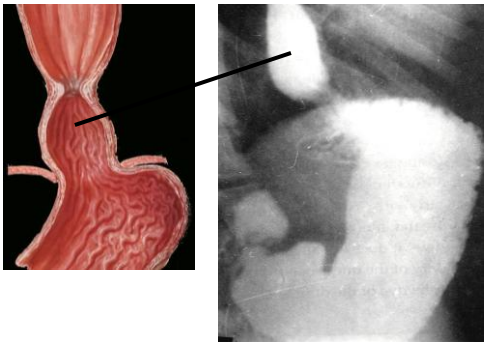
Patient is usually (> 40 years & **obese** female **)**

- **Retro-sternal burning pain** due to reflux oesophagitis which is **characterized by :** • ↑ by lying flat & heavy meals.
  • ↓ by standing upright



- **Regurgitation :** • **Acidic** & bitter fluid.
  • Water brush
- **Dysphagia :** 1st due to reflex spasm of the oesophagus but **later** due to fibrous stricture 2ry to ulceration.
- **C/P of complications :**
  as • Aspiration pneumonia.
  • Bleeding from ulcer → Chronic anemia..
  • **Barrett's oesophagus** which is pre-cancerous
  • Stricture oesophagus.

INVESTIGATIONS



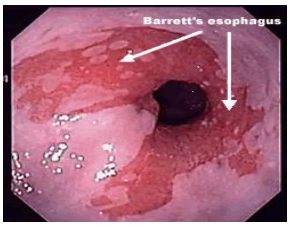
A- Barium meal [**Trendlenburg's position**]

1- Part of stomach protruding through the hiatus towards the chest.

2- Reflux of barium from stomach to the oesophagus

3- Widening of oesophageal hiatus.

B- Oesophagoscopy



1- The cardia opens on inspiration (normally it closes)

2- Reflux of gastric juice through the cardia.

3- **Complications** as oesophagitis, ulceration or strictures.

4- **Endoscopic grading of reflux :**

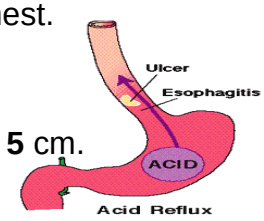
- ***Grade I :*** Hyperaemic mucosa
- ***Grade II :*** Superficial ulceration
- ***Grade III :*** Deep ulceration
- ***Grade IV :*** Stricture or Barrett's oesophagus

C- Manometric studies reveal

Loss of normal (high) pressure zone at the lower end of the oesophagus due to displacement of oesophago-gastric junction into the chest.

D- PH monitoring (**More specific**)

The most diagnostic for reflux oesophagitis **so** it shows (**PH < 4**) which is acidic at lower **5** cm.



TREATMENT

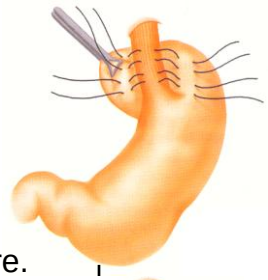
I- Conservative treatment

① Reduction of body weight.

② Semi-sitting position during sleep.

③ Frequent small meals & avoidance of smoking, coffee, …etc.

④ **Drugs :** Antacids + omeprazole (proton pump blocker) or ranitidine (**Zantac**) (H_2 blockers)

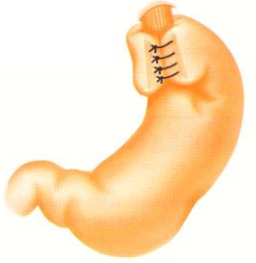


II- Surgical treatment

- **Indications** :

1- Failure of medical treatment.

2- Presence of **complications** as oesophagitis or stricture.



ANTI-REFLUX PROCEDURES ➪

Nissen's fundoplication

- Principle :

- The fundus of stomach is wrapped around the lower oesophagus to create a high pressure zone.

- This operation can be done by open or laparoscopic surgery

B. Para-oesophageal (Rolling) Hernia

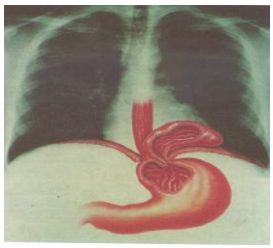
DEFINITION

It is a **TRUE HERNIAL SAC** of the greater curve of the stomach into the posterior mediastinum.

PATHOLOGY

➪ **Unlike** sliding hiatus hernia, the oesophago-gastric junction remain below the diaphragm.
So **no reflux oesophagitis.**

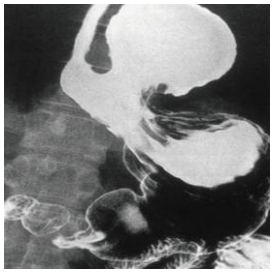
CLINICAL PICTURE



- **Post-prandial chest pain** from distended stomach.
- **Bouts of hiccough** from irritation to the phrenic nerve.
- **Complications :**

① **Strangulation** ➔ rupture ➔ mediastinitis. .
② **Pressure on heart** ➔ cardiac symptoms e.g. arrhythmias

INVESTIGATIONS



A- Barium meal [**Trendlenburg's position**]
Herniation of stomach in the chest & the oesophago-gastric junction remain below the diaphragm.

B- Chest x-ray Shows gastric gas shadow in the chest.

TREATMENT

Stomach is retracted down, the hernial sac is excised & the defect is closed

VII. Carcinoma of the oesophagus

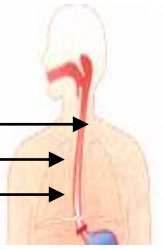
INCIDENCE

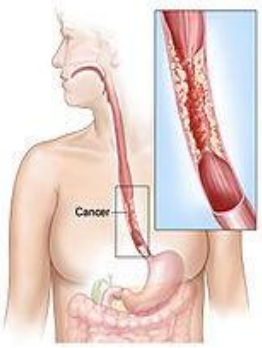
- Less common GIT cancer (about **4 %** only)
- **Age :** > **50** years
- **Sex :** Male **>** female

PREDISPOSING FACTORS

- **Chronic irritations** as **5S** (**s**pirits, **s**pices, **s**moking, **s**epsis & **$**)
- **Corrosive strictures** of the oesophagus
- **Achalasia** of the cardia.
- Long standing **reflux oesophagitis**
- **Barrett's oesophagus**.
- Plummer Vinson syndrome

PATHOLOGY



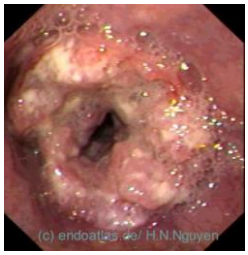


Site

- The upper **1/3** = **17 %** →
- The middle **1/3** = **50 %** →
- The lower **1/3** = **33 %** →

NIE picture

- Annular stenotic type
- Ulcerative type.
- Cauliflower (fungating) type



Microscopic picture

- **Squamous** cell carcinoma **95 %.**
- **Adenocarcinoma 5 %** from cancer fundus or on top of **Barrett's oesophagus**.

SPREAD

Direct Both in (circumferential & longitudinal) direction then Infiltrate the surrounding

- **Cervical** oesophagus ➔ Trachea, thyroid, RLN & carotid sheath.
- **Thoracic** oesophagus ➔ Trachea, pleura, lung & aorta.
- **Abdominal** oesophagus ➔ Lt. lobe liver, diaphragm & stomach.

Lymphatic

- **Cervical** oesophagus ➔ **Cervical** lymph nodes.
- **Thoracic** oesophagus ➔ **Tracheal** & **tracheo-bronchial** lymph nodes
- **Abdominal** oesophagus ➔ **Lt. gastric** & **coeliac** lymph nodes.

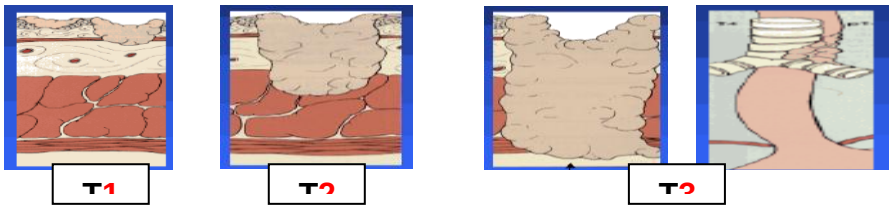
Blood • Late to lung, bone, liver & rarely brain.

Transcoelomic spread

- **Only** with abdominal oesophagus leading to peritoneal nodules & malignant ascites then krukenberg's tumor & nodules in douglas pouch.

STAGING T.N.M

T = Tumor	N = Nodes	M = Metastases
Tis = Cancer in situ T_1 = **< 5** cm & confined to mucosa T_2 = **> 5** cm & confined to musculosa T_3 = Any size with extra-oesophageal spread	N_0 = **No** L.Ns N_1 = Mobile L.Ns (**unilateral**) N_2 = Mobile L.Ns (**bilateral**) N_3 = Fixed L.Ns	**Mo** = **No** distant metastasis M_1 = Distant metastasis



CLINICAL PICTURE

Type of patient (**> 50** years & Male > female)

Manifestations

1. Dysphagia :

- Insidious **onset**, slowly progressive **course** & short **Duration**.
- More to **solid than fluid** but later on patient cannot swallow his own saliva.
- **At the time of its presentation** about 2/3 of circumference of oesophagus has been involved

2. Regurgitation :

- More nocturnal & Alkaline fluid.
- Mixed with saliva & possibly streaked with blood.

3. Pain : Late manifestations.

4. C/P of complications :

- Cachexia, weight loss & metastasis.
- Recurrent laryngeal nerve paralysis ➔ Hoarseness of voice.
- Phrenic nerve paralysis ➔ Hiccough & diaphragmatic paralysis
- Tracheo-oesophageal fistula ➔ Recurrent chest infection.

DEFERENTIAL DIAGNOSIS

	Achalasia	Carcinoma
Age	**Middle** (20 - 40)	**Old** (**>** 50)
Sex	More with **female**	More with **male**
Emotion	**Aggravates** dysphagia**.**	**No** effect.
Dysphagia	More to **fluids.**	More to **solids.**
Pain	May be	**No** or **late**
General condition	**Good**	**Very bad**

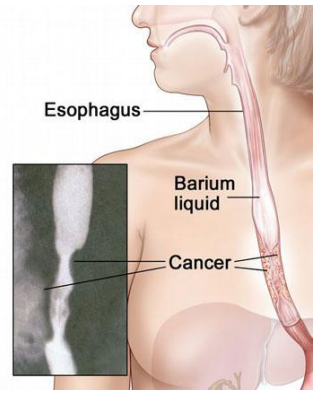
INVESTIGATIONS

A- Laboratory

1- Complete blood picture for anemia.

2- Serum electrolytes & kidney function tests.

3- Serum proteins & liver function tests.

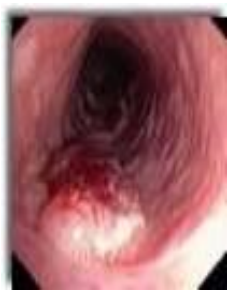


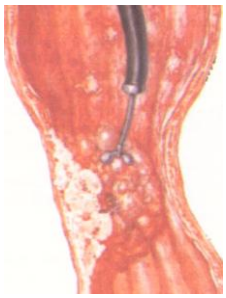
B- Barium swallow

1- Narrow lumen at the site of the lesion with mild proximal dilatation due to short duration (**unlike achalasia**)

2- Irregular filling defect in cauliflower lesion.

3- Annular lesions will show irregular narrowed segment with **shouldering** + **Rat tail** appearance.





C- Endoluminal U/S The most recent.

D- Oesophagoscopy

Demonstrates the lesion + biopsy

E- Metastatic work-up

1- **Bronchoscopy** for tracheal invasion.

2- **Abdominal U/S** for liver metastasis.

3- **C.T scan chest** for extra-oesophageal spread & lung metastasis.

TREATMENT

A- Operable cases

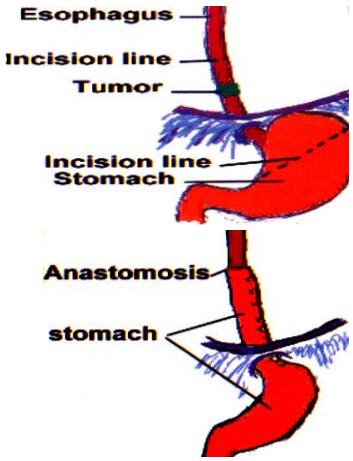
INDICATIONS

Patient in a good general condition with respectable tumor.

THE IDEA

The idea is to resect the lesion with adequate safety margin on either side (**10 cm**) & then to restore the continuity of gastro-intestinal tract.

TYPES OF OPERATION



➢ **For tumors of the lower 1/3 :**

- The oesophagus is excised and the **upper portion** of the stomach, then oesophago-gastric anastomosis is performed

➢ **For tumors of the middle 1/3 (Ivor Lewis) :**

- The oesophagus is excised down to **cardia**. then oesophago-gastric anastomosis is performed

➢ **For tumors of the upper 1/3 :**

- **Total oesophagectomy** and then perform an anastomosis in the neck

Nowadays, many surgeons prefer to do :

Total oesophagectomy(wherever the site)
and then perform an anastomosis in the neck

- Advantage of this technique :

① Total oesophagectomy guarantee adequate safety margin.

② The oesophago-gastric anastomosis is performed in the neck. So, if leakage occurs, it doesn't lead to mediastinitis.

③ No need for thoracotomy.

N.B.: Oesophageal replacement surgery

① Gastric pull-up procedure.
② Colon replacement
③ Pectoralis major myocutaneous flap
④ Free jejunal replacement with microvascular anastomosis.

B- Inoperable cases

INDICATIONS

1- **Unfit patient** e.g. poor cardio-pulmonary status.

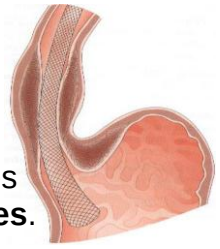
2- Patient with distant metastasis as lung & liver metastasis.

3- Patient with marked complications "see before".

THE AIM

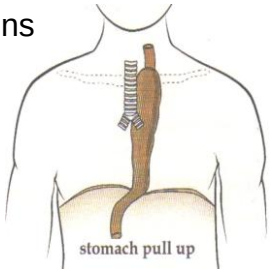
To relieve dysphagia, i.e to allow the patient to swallow.

PALLIATIVE METHODS



- **Intubations :**
  The idea is to insert a rigid tube through the stenosed segment to keep a patent lumen. e.g. the **Souttar** or **Celestin** tubes **there are now also self-expanding tubes**.

- **Palliative surgery**
  As colon - bypass or gastric pull-up but these operations with high mortality for patient with short life span



- **Radiotherapy**
  A dose of 4000 - 4500 rads over period of 4 weeks especially for the **upper** oesophageal carcinoma.

- **Laser photocoagulation (ND:YAG)**
  Laser energy causes tissue coagulative necrosis

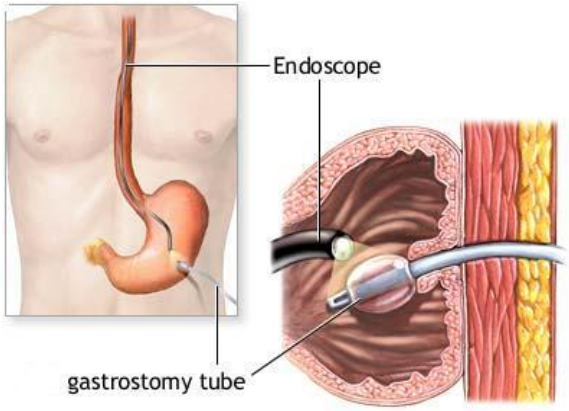
- **Gastrostomy**
  This is performed when there is no other alternative. It doesn't relief the patient from the inability to swallow his saliva

CONTINUOUS FOLLOW UP

To detect any progression occur.

N.B

INDICATIONS OF GASTROSTOMY



1. **Temporary**
   - **Congenital** : Congenital oesophageal atresia.
   - **Traumatic**.: Rupture oesophagus due to Instrumentation.
   - **Inflammatory** : Stricture of oesophagus.
   - **Neoplastic** : Removable tumors of (Mouth, pharynx or oesophagus)
2. **Permanent**

   Irremovable tumors of (Mouth, pharynx or oesophagus)

Stomach
&
Duodenum

THE STOMACH & DUODENUM

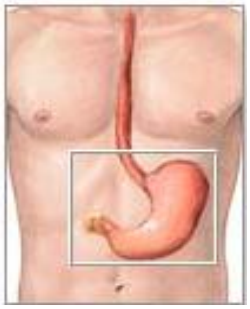
ANATOMY OF THE STOMACH

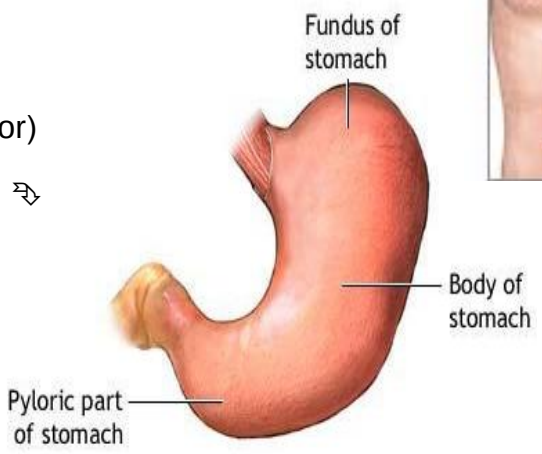
The Stomach is formed of

- **2 Orifices** (cardiac & pyloric)
- **2 Curves** (greater & lesser)
- **2 Surfaces** (anterior & posterior)

The Stomach is divided into

- **Fundus**
- **Body**
- **Pylorus** - Pyloric sphincter.
  - Pyloric canal .
  - Pyloric antrum.

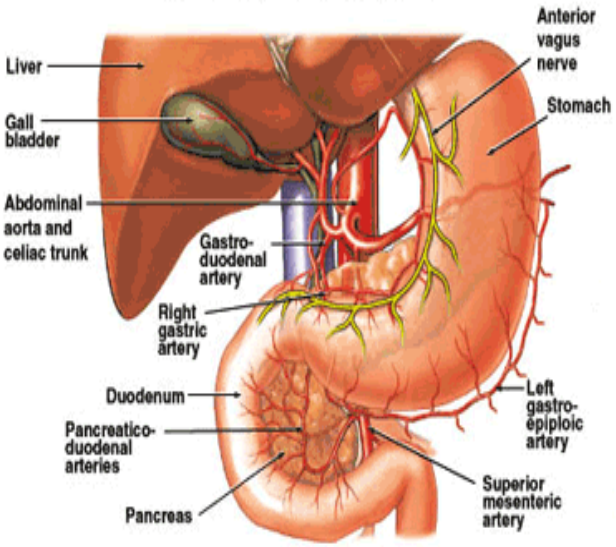




ARTERIAL SUPPLY

Arteries along " lesser curve "

- **Lt. gastric artery**
  - Branch from coeliac trunk.
  - End by anastomosing with Rt. gastric artery.
  - It gives oesophageal branches
- **Rt. gastric artery**
  - Branch from the hepatic artery.
  - End by anastomosing with Lt. gastric artery.

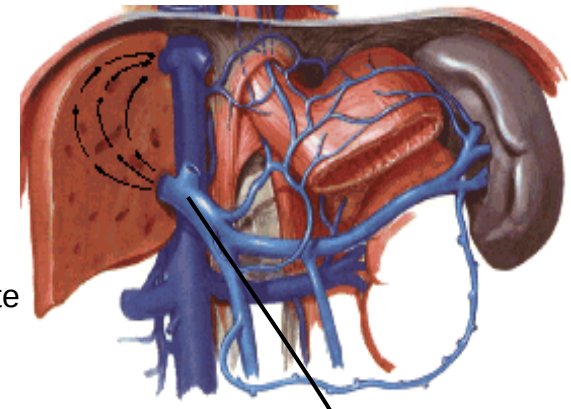


Artery at " fundus of stomach "

- **Short gastric branches**
  - 2-3 branches from splenic artery

Arteries alone the " greater curve "

- **Lt. gastro-epiploic artery**
  - Branch from the splenic artery.
  - End by anastomosing with Rt. gastro-epiploic artery
- **Rt. gastro-epiploic artery**
  - Branch from the gastro-duodenal arte
  - End by anastomosing with Lt. gastro-epiploic artery.



VENOUS DRAINAGE

Corresponding to the arteries then drained into **portal vein**.

NERVE SUPPLY

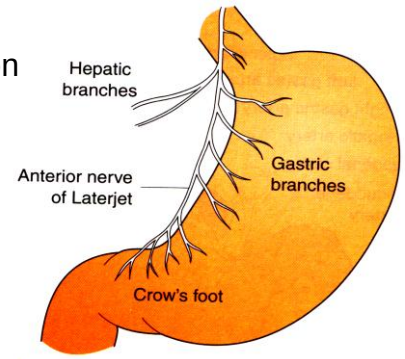
Parasympathetic supply

- **RESPONSIBLE FOR** stimulation of acid secretion & gastric motility

- **REACH THE STOMACH THROUGH**

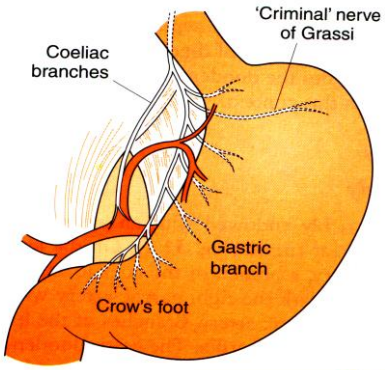
⇨ **Ant. vagus nerve :**

Give **hepatic** branch to liver & gall bladder then continues on the ant, surface of stomach as nerve of **Laterjet** and end at the pylorus for motor power as **Crow's foot**.



⇨ **Post. vagus nerve :**

Give **coeliac** branch to supply intestine then continues on the post, surface of stomach



Sympathetic supply

- **RESPONSIBLE FOR** visceral sensation.

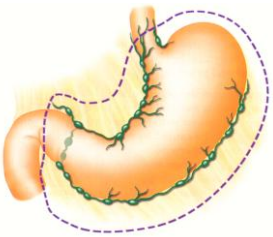
- **REACH THE STOMACH FROM** greater splanchnic nerve from coeliac ganglion.

LYMPHATIC DRAINAGE

- **A sound Knowledge** of the lymphatic drainage for stomach is essential for proper treatment of cancer stomach.

- **For details (see cancer stomach)**

-



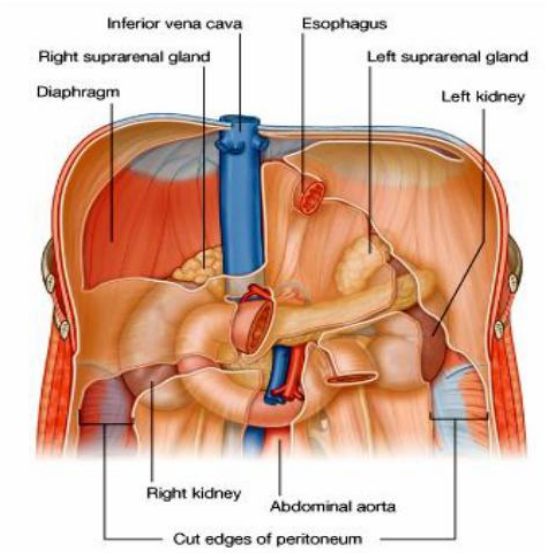
ANATOMY OF STOMACH BED

✷ Definition

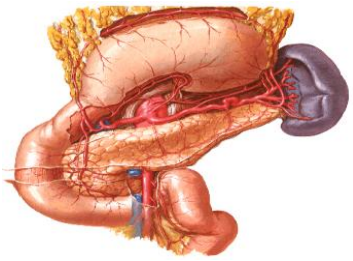
Posterior surface of stomach

✷ Contents

1- Lt. crus of the diaphragm
2- Lt. suprarenal gland
3- Lt kidney
4- Spleen
5- Splenic a.
6- Body of pancreas.
7- Transverse mesocolon.
8- Transverse colon.



ANATOMY OF THE DUODENUM



The duodenum **It is divided into 4 parts**

- **1st. part** is distal to the pylorus
- **2nd part** is receiving the ampulla of Vater which is the union of common bile duct & pancreatic duct
- **3rd part** It runs horizontally behind the superior mesenteric vessels.
- **4th part** It runs superiorly & terminate as duodeno-jejunal (DJ) flexure

ARTERIAL SUPPLY

- **Superior pancreatico-duodenal artery**
  which is a branch of the gastro-duodenal artery
- **Inferior pancreatico-duodenal artery**
  which is a branch of the superior mesenteric artery.

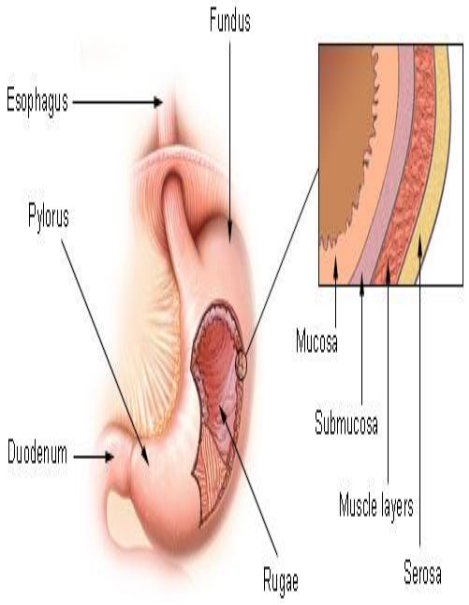
HISTOLOGICAL CONSIDERATIONS

The stomach is formed of 4 layers

1. **Mucosa :**
   Lined by columnar epithelium which secretes mucus lubrication.

   N.B.: Containing special cells :

   - CHIEF CELLS : present at fundus & secretes **pepsin**.
   - PARIETAL CELLS : present at body. & secretes **Hcl**.
   - G. CELLS : present at pyloric antrum. & secretes **gastrin**



2. **Submucosa**.
3. **Musculosa :**
   a. Inner circular.
   b. Middle oblique.
   c. Outer longitudinal .
4. **Serosa**.

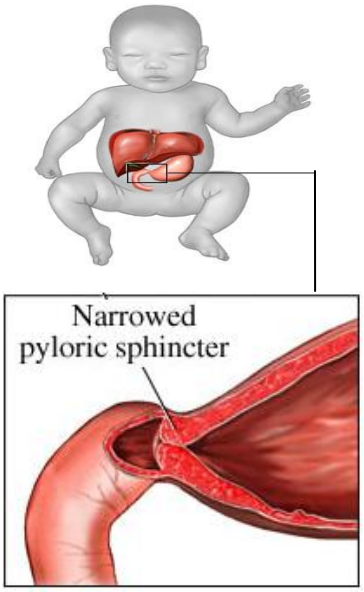
Gastroduodenal mucosal barrier

A group of factors that protect the mucosa from being digested by acid

1. **Mucus secretion** by the mucosa serves as a protective lining.
2. **Bicarbonate secretion** by the mucosa
3. **Rapid regeneration** of mucosal cells.
4. **Good blood flow** of mucosal cells.
5. **Prostaglandins** which maintain the blood supply.

I- CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

CHPS



INCIDENCE

- **Rare** disease (**3 : 1000**)
- **Age : 2 - 3** weeks after birth (never at birth)
- **Sex :** Male **:** female **= 4 : 1**

AETIOLOGY

Unknown but may be due to

1- Genetic factors : (**7** % familial)

2- Pre-natal :

Neuromuscular incoordination leading to failure of pylorus to relax

3- Post-natal :

Hypertrophy of the pyloric muscles.

PATHOLOGY

The pylorus

Hypertrophy of circular muscle layer, which stopped suddenly at the duodenum.

The stomach

At 1st **hypertrophied** to overcome the obstruction, but later on dilated with gastritis from retention.

The intestine Normal & collapsed.

CLINICAL PICTURE

Any newborn presenting with projectile, non-bile stained vomiting should be considered as having **CHPS** until proved otherwise

Symptoms

1- VOMITING :

- Starts 2 - 3 weeks after birth.
- Projectile, non-bile stained vomiting.
- After vomiting, the infant is V. Hungry.

2- PROGRESSIVE CONSTIPATION

3- COMPLICATIONS :

- Dehydration & loss of weight.
- Chest infection (**aspiration pneumonia**)
- Metabolic alkalosis ➔ **Tetany** from repeated vomiting

Signs

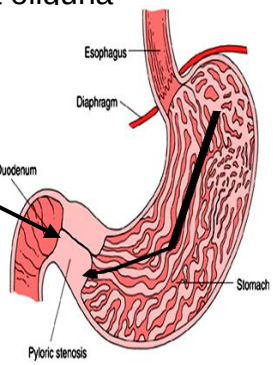
1- GENERAL "Signs of dehydration"

Dry inelastic skin, dry tongue, sunken eyes & oliguria

2- LOCAL : " Test feed "

After a feed & in a good light

- Abdominal distention.
- Visible peristalsis from Lt. to Rt.
- **Palpable**, **firm** & **olive** like mass under costal margin & to the Rt. of the midline.
- Succession splash by auscultation.



D.D From other causes of neonatal vomiting as

① GASTRO-ENTERITIS
② PYLOROSPASM
③ DUODENAL ATRESIA
④ INTESTINAL ATRESIA.
⑤ VOLVULUS NEONATORUM.
⑥ BIRTH HEAD INJURIES

INVESTIGATIONS

1- Laboratory "Serum electrolytes"

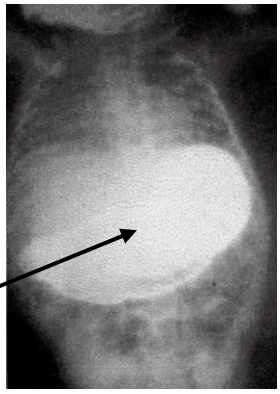
Metabolic alkalosis & ↓ Na

2- Abdominal U/S (**The choice**)

Thickened pyloric muscles.

3- Barium meal

Huge dilated stomach may reach down to the pelvis with fluid level in the stomach.



TREATMENT "Only surgical"

⇨ **1ST LINE OF TREATMENT :** Correct the dehydration & electrolytes imbalance.

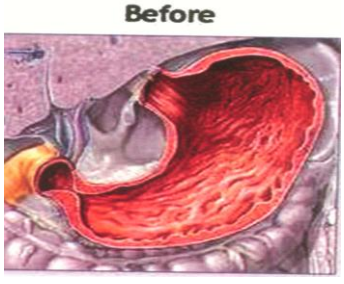
⇨ **PRE-OPERATIVE PREPARATIONS :**

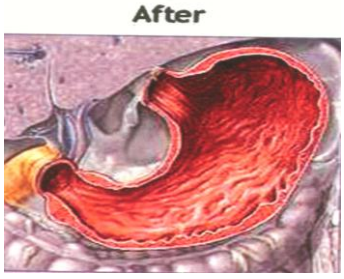
① **Stop feeding**, then naso-gastric suction with normal saline .

② **High protein diet** with I.V hyperalimentation .

③ **Chest antibiotics**.

⇨ **OPERATION :** (**Ramstedt's pyloromyotomy**)





① A small upper abdominal incision
② The hypertrophied pylorus is grasped between index & thumb
③ **Pyloromyotomy** is done until the mucosa of pylorus bulges out.

II- TRAUMATIC DISORDERS

Foreign bodies

TYPES

Ingested F.B. e.g. coins, pin ……..etc

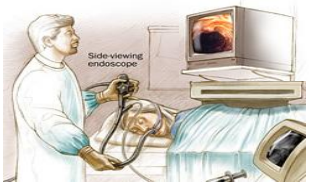
Locally formed F.B. (**Bezoars**)

① **Trichobezoar :** formed from hair which may be eaten by children or mentally retarded girl.
② **Phytobezoar :** from some plant fibers,

TREATMENT

1- Conservative treatment :
We wait for spontaneous passage in stool with **daily x-ray** abdomen

2- Endoscopic removal :
Indicated in **small** ingested F.B in the stomach.



3- Surgical removal : (**Gastrostomy**)
Indicated in **large** ingested F.B in the stomach

.....

III- ACUTE GASTRIC DILATATION

DEFINITION It is a condition where the stomach **loses its tone**

PREDISPOSING FACTORS

① **Abdominal operations** especially (biliary or pelvic)
② **Opiates** ➔ ↓ gastric empting .
③ **Labor**.
④ **Fractures** especially with (femur, pelvis & spine)
⑤ **Ventilators** .
⑥ **Aerophagia**

AETIOLOGY **Unknown** But may be ↑ sympathetic tone over activity ➔ Hypotonia.

CLINICAL PICTURE

Symptoms ① **AT 1ST** hiccough & eructation on the 3rd post-operative day.
② **VOMITING : EFFORTLESS** (large, dark & watery)
③ **COMPLICATIONS :** - **Tetany** from metabolic alkalosis.

Signs ① **GENERAL :** Dehydration, tachycardia & ↓ ABP.
② **LOCAL :** Distended dead silent abdomen.

INVESTIGATIONS

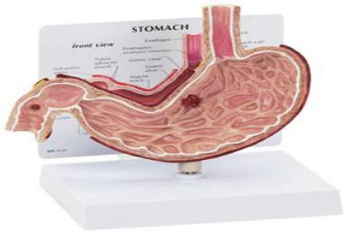
Na^+ & K^+ serum level. **NEVER** barium meal as it worsen the case.

TREATMENT (**No role for surgery)**

Prophylactic Ryle's tube to decompress the stomach.

Curative Continue Ryle's tube + correct electrolytes

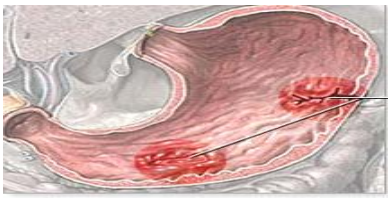
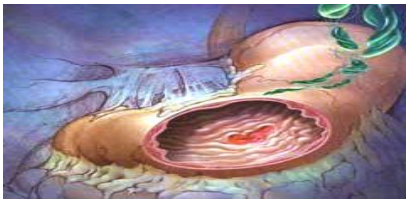
IV- PEPTIC ULCER



SITES **In order to frequency** ⇩

① Duodenum " The commonest "
② Stomach.
③ Jejunum after gastrojejunostomy.
④ Lower oesophagus if reflux oesophagitis.
⑤ Meckel's diverticulum " The rarest."

1. Acute peptic ulcer

	Multiple erosions	True stress ulcer
		
AETIOLOGY	Disruption of gastric mucosa due to uses of **NSAIDs** which leads to ↓ mucosal blood flow by its Anti-prostaglandin effect.	Disruption of gastric mucosa due to mucosal ischemia 2^{ry} to 1- vascular injury. 2- Patient At (ICU) with severe trauma or major burn.
PATHOLOGY	Multiple shallow erosions limited to mucosa and sub-mucosa	Multiple erosions if not recognized and treated they will coalesce to becomes **acute hemorrhagic gastritis**
CLINICAL PICTURE	Mild to moderate Hematemesis	Severe hematemesis so may leads to shock.

TYPES ① **Cushing ulcer :** after cerebral trauma & neurosurgical operations .
② **Curling ulcer :** after major burn.

DIAGNOSIS (**An emergency case**)
So do endoscopy (barium meal is **useless** as ulcer is superficial)

TREATMENT

Conservative treatment

① **Withdrawal** of NSAIDs .
② **Blood transfusion** to correct shock .
③ I.V. **antacids** e.g. Omeprazole (proton pump blocker)

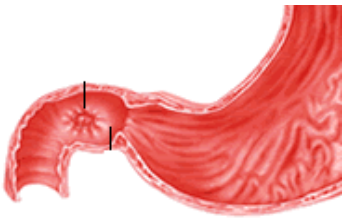
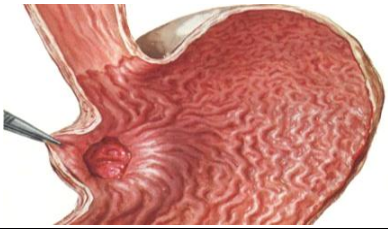
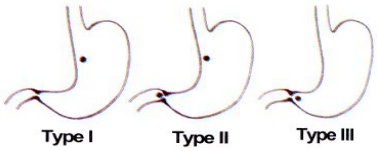
Recent treatment

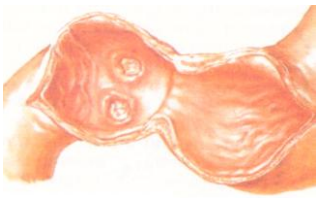
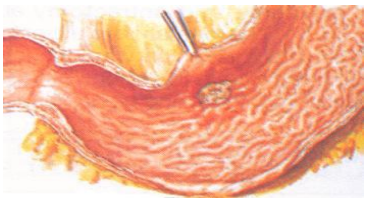
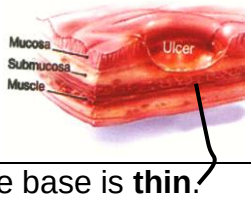
① Endoscopy then local injection of **vasoconstrictors** .
② Endoscopy then **LASER** photocoagulation
③ **Ice cold saline wash** mixed with adrenaline.

Surgical treatment

Subtotal or **total** gastrectomy (if all the above are failed)

2. Chronic peptic ulcer

	Duodenal ulcer	Gastric ulcer
INCIDENCE	 **25 : 1**	
• Age	Usually young (**30** - **40**) years	Usually elderly (**50** - **60**) years
• Sex	**5** Male **: 1** Female	**2** Male **: 1** Female
TYPES	Only **one** type Present at the **1st inch** of the **1st** part of duodenum & it shows hyperacidity	There are **three** types  **Type I :** ulcer at lesser curve **Type II :** GU & DU **Type III :** ulcer at the antrum
AETIOLOGY	**Increased gastric acidity**	**Decreased mucosal barrier**
	1. Genetics : acts by producing large parietal cell mass. so leads to High HCL secretions. **2. ↑ Vagal tone :** due to mental stress & nervousness. **3. Increased gastrin hormone :** - ↑ SECRETION due to Zollinger Ellison's syndrome i.e. gastrinoma. - ↓ INACTIVATION due to liver cirrhosis. **4. Helicobacter pylori** which is gram (-**ve**) ➔ antral gastritis ➔ ↑ gastrin release. **5. The uses of (NSAIDs)** **6. Other causes** as irritant food. **N.B.:** Blood group (**O**) & **+ve** family history are also risk factors	⇨ **↓ Protective layer of mucosa** **1. Duodeno-gastric reflux :** Due to disturbed pyloric sphincter so it will leads to damage of gastric mucosa. . **2. Antral stasis :** Due to defect in gastric empting ➔ atrophy of gastric mucosa. **3. Helicobacter pylori** which split urea ➔ ammonia so ↑ PH ➔ epithelial damage **4. The uses of (NSAIDs)** **5. Other causes :** as irritant food. ⇨ **↓ Regenerative power of the gastric mucosa** **6. Atherosclerosis** ➔ ischemia at the gastric mucosa

	Duodenal ulcer	Gastric ulcer
PATHOLOGY		
• **Number**	• Usually **single**, but may be **2** kissing ulcers	• Usually **single**
• **Site**	• In the **1st** inch of **1st** part of the duodenum.	• At **junction** of the body & the antrum on lesser curve
• **Shape**	• **Rounded** or **oval** shape.	• Usually **irregular** shape.
• **Size**	• Usually **small < 2 cm.**	• Usually **large > 2 cm.**
• **Edge**	• **Sloping** (healed) or **punched out** (unhealed).	
• **Margin**	• The surrounding mucosa is thrown into folds which converge on the ulcer, i.e. **regual convergence**. 	
• **Floor**	• Filled with granulation tissue with **no** muscle layer.	
• **Discharge**	• **+ve Stippling sign (** at emergency operation **)** By rubbing the site of ulcer with a **gauze** → **minute petechial hemorrhage** 	
• **Base**	• The ulcer is **deep,** penetrate the muscle coat & the base is **thin**.	
C/P (**Pain**)		
• **Site**	• **Epigastric** & to the **Rt.** of middle line i.e. pointing sign..	• **Epigastric** & to the **Lt.** of middle line, i.e pointing sign.
• **Character**	• **Burning** pain.	• **Burning** pain.
• **Time**	• **2** hours after meal. **N.B. :** - May be $^{1}/_{2}$ hour before meal i.e. **hunger pain** - Maximum at night i.e. **nocturnal pain**	• **1/2** hour after meal. **N.B. : no** hunger pain & **no** nocturnal pain.
• **↑↑ by**	• Irritant food**,** stress & hunger.	• Irritant food.
• **↓↓ by**	• **Eating** & antacids (patient has always some biscuits).	• **Vomiting** & antacids
• **Referred**	• **Boring pain** at back i.e. penetration of the pancreas	
• **Appetite**	• **Good**	• **Bad** (afraid to eat)
• **Nausea & Vomiting**	• **Rare**	• **Common** (may be self Induced) to bring alkaline bile to stomach.
• **Periodicity**	(pain occur at periods of ulcer activity)	
	• **Marked**	• **Less marked.**

COMPLICATIONS " May 1st presentation "

① **Perforation :** with penetration to nearby organs as pancreas .

② **Bleeding :** - **Duodenal ulcer :** melena is common.
- **Gastric ulcer :** hematemesis is common

③ **Stenosis :** - **Duodenal ulcer** ➔ Pyloric stenosis.
- **Gastric ulcer** ➔ Tea-pot stomach or hourglass stomach

④ **Malignancy :** " **Never** with duodenal ulcer
& occur with gastric ulcer **only** "

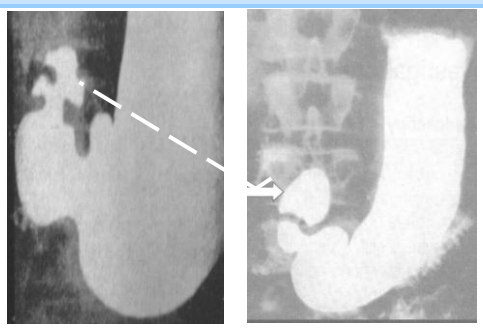
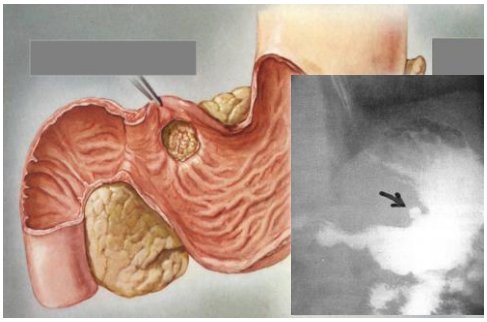
INVESTIGATIONS

A- Endoscopy

For visualization of ulcer & follow up

N.B. : Biopsy to exclude carcinoma with gastric ulcer **only**

B- Barium meal

DUODENAL ULCER	GASTRIC ULCER
	
• **Ulcer niche** full of barium in duodenal cap which is normally (triangular shape with rounded corners).	• **Ulcer niche** full of barium on the lesser curve of stomach.
• **Duodenal cap** is deformed from spasm & later fibrosis.	• **Ulcer notch** may be seen at the greater curve(**opposite to ulcer niche**) from spasm & fibrosis of circular muscles.
• **Persistent deformity** of the duodenal cap detected by (At least 3 serial films with **15 minute** Interval).	• **Post-evacuation film** show convergence of mucosal fold towards the ulcer i.e. barium at ulcer & seen radiating from it.
• **Rapid** gastric emptying	• **Delayed** gastric emptying
• **Localized** tenderness **(under screen)**	

C- Investigation for H. pylori

As serological test & culture from biopsy.

TREATMENT

1- Treatment of chronic duodenal ulcer

A. Conservative treatment

THE AIM

Reduction of gastric acidity to allow the ulcer to heal

THE MEASURES

1- GENERAL MEASURES :

① Physical & mental rest.

② **Diet :** Frequent small meals & avoid irritant food.

> **N.B.: Milk** are given as a some only.
> because inspite of its neutralizing effect,
> it is a potent stimulus for Hcl

③ **Stop** smoking, coffee, tea & alcohols

2- DRUG THERAPY :

I- H2 receptor blockers such as

1- Cimitidine (Tagamet)

- **Dose : 800** mg at bed time.
- **Side effects :** (Rare) only **5 %**
  - ① ***Anti-androgenic :***
    causing (impotence, gynaecomastia & oligospermia)
  - ② Risk of ***gastric carcinoma***
  - ③ ***Confusion :*** If high dose because it can passes B.B.B.

2- Ranitidine (Zantac)

150 mg/ twice daily.

3- Famotidine (Antodine)

40 mg at bed time

II- Proton pump blocker such as

Omeprazole (Losec)

- **Dose :** 20 mg orally in the morning.
- **Effect :** It Reduces gastric acidity up to **Zero** by **99 %.**

III- Antacids

- **Combined** (Aluminum hydroxide gel + Magnesium oxide)
- **It can help** in the rapid relief of pain.

IV- Specific treatment for Helicobacter pylori

1- Metronidazole (Flagyl)

2- Tetracycline or **Amoxicillin**

3- Colloidal bismuth (De nol)

They are given for **2 weeks**
to give good result (**90 %**)

B. Surgical treatment

THE INDICATIONS

① **Complications** of ulcer

② **Combined** gastric & duodenal ulcers.

③ **Poor** patient (**financial factor**) i.e. can't afford medical treatment.

④ **Poor** compliance i.e unwilling to take tablets.

⑤ **Resist** to medical treatment i.e. no response.

⑥ **Recurrent** after adequate medical treatment.

THE TECHNIQUES

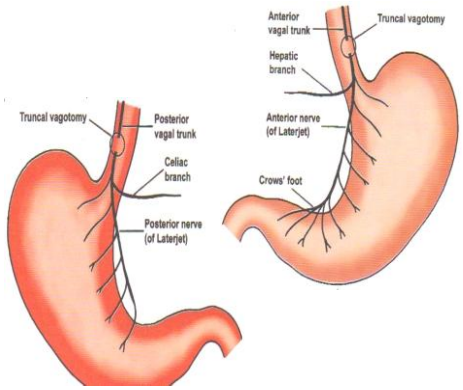
1- Abolishing the nervous phase of gastric acid secretions by **Vagotomy**

2- Abolishing the hormonal phase of gastric acid secretions by **Antrectomy**

N.B.: Some surgeons **combine vagotomy** & **antrectomy**

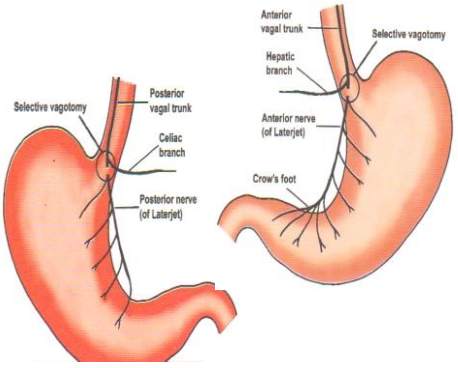
I- Vagotomy

1. Truncal vagotomy



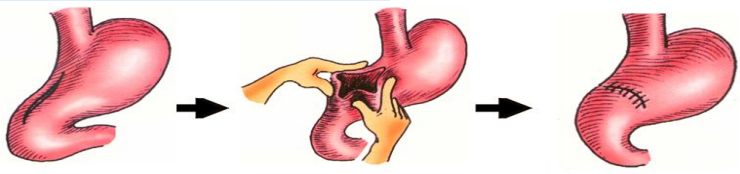
- **Dividing both ant. & post. vagal trunks**
  At the lower end of esophagus
  But complicated by ⇘
  - **1- Denervation of pylorus**
    ➔ **pylorospasm** ➔ antral stasis
    ➔ ↑ gastrin release ➔ ↑ Hcl secretions ➔ recurrence of ulcer
  - **2- Denervation of whole abdomen**
    ➔ Distension & biliary dyskinesia.
- **80 % reduction of Hcl** secretions but recurrence rate (**5 -10 %**)
- **Drainage procedures** is done as pyloroplasty or gastrojejunostomy.

2. Selective vagotomy



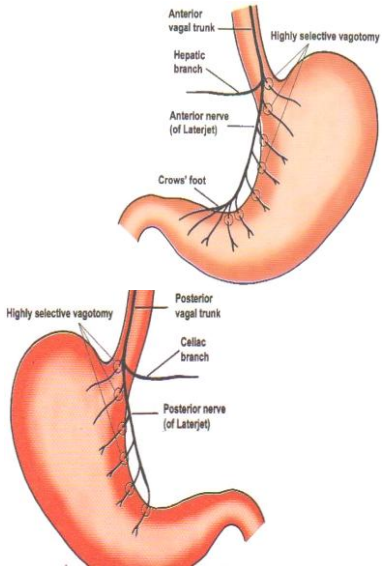
- **Dividing both ant. & post. vagal trunks**
  Sparing both coeliac & hepatic branches
- **There is still pylorospasm only** .
- **Drainage procedures** is still done.

N.B. Drainage procedures :
- ⇨ Gastro-jejunostomy
- ⇨ Pyloroplasty



The pylorus is divided longitudinally & sutured transversely

3. Highly [Super] selective vagotomy



Also called " **Parietal cell vagotomy** "

& " **Proximal gastric vagotomy** "

- **The denervation** occur only for acid secreting part (body & fundus) so we preserve the nerve of Laterjet supplying motor power of pylorus.
- **No drainage procedures** are needed.

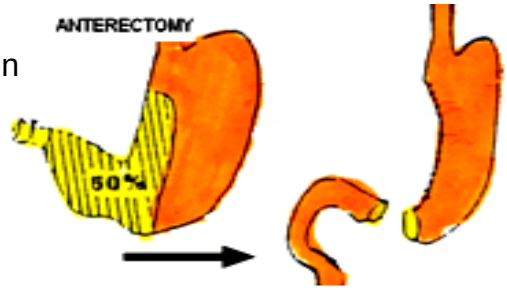
4. Lesser curve seromyotomy

As " **Proximal gastric vagotomy** "

- **Most surgeons** perform it anteriorly **only** adding a posterior truncal Vagotomy

2- Antrectomy

" Hemi-gastrectomy "



- **It is designed** to remove the pyloric antrum which is the site of production of gastrin hormone
- **About 50 %** of distal part of the stomach is removed.
- **It is followed by** gastro-duodenal anastomosis.

N.B: Antrectomy may be add to vagotomy to abolish both nervous & hormonal phase.

.....

2- Treatment of chronic gastric ulcer

A. Conservative treatment

AS CHRONIC DUODENAL ULCER

⇨ **But with the following considerations :**

1- Because of the risk of being malignant , it is essential to exclude malignancy before deciding medical treatment.

2- Endoscopy should be carried out after **6 weeks** regardless of symptomatic improvement,

(a) If ulcer shows (no attempt) of healing

➔ It should be considered carcinoma & surgery is performed.

(b) If ulcer shows (partial) healing

➔ Continue the medical treatment for another **2 months** and repeat endoscopy.

⇨ **We can use cytoprotective drugs :**

1- Colloidal bismuthate

De nol

- **It form** protective coat on the mucosa.
- **Bacteriostatic** for Helicobacter pylori.
- **Dose :** 2 tab. before breakfast & dinner.

2- Sucralfate

Gastrofate

- **Pepsin-antagonist**.
- **Increase prostaglandins activity** which protect gastric mucosa.
- **Dose :** I gm before meal.

B. Surgical treatment

THE INDICATIONS

① **Complications** of ulcer

② **Combined** gastric & duodenal ulcers.

③ **Poor** patient (**financial factor**) i.e. can't afford medical treatment.

④ **Poor** compliance i.e unwilling to take tablets.

⑤ **Resist** to medical treatment i.e. no response.

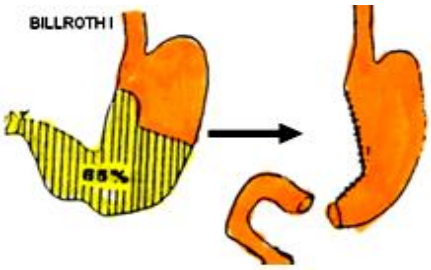
⑥ **Recurrent** after adequate medical treatment.

THE TECHNIQUE

Billroth I

" Partial gastrectomy "

- **It is designed** to remove the gastric ulcer
- **About 65 %** of distal part of the stomach is removed.
- **It is followed by** gastro- duodenal anastomosis



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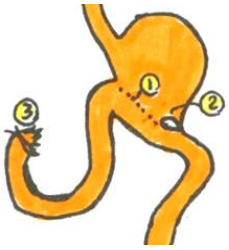
COMPLICATIONS AFTER GASTRECTOMY

I. EARLY COMPLICATIONS

❶ **Haematemesis :** Due to bleeding from suture line of anastomosis.

❷ **Stomal obstruction :** i.e. obstruction at line of anastomosis, by odema at stoma

❸ **Duodenal blow out :** after gastro-jejunostomy the blind duodenal stump may be distended with pancreatic and biliary juice → ↑ pressure → disruption of sutures → Biliary peritonitis.



II. Late complications

1. Recurrent ulceration

✱ **Aetiology**

A. Inadequate surgery : e.g. Missing a vagal nerve

B. Other causes as Zollinger-Ellison syndrome i.e. Gastrinoma.

C. Uses of ulcerogenic drugs : Corticosteroids, Aspirin, NSAIDs ... etc.

2. Dumping [Post-cibal Syndrome]

- It is a syndrome with vasomotor & G.I.T symptoms after meal.
- It may be :

1. Early	2. Late
If symptoms occur within 1st **1/2 hour** after meal	If symptoms occur within **2-3 hours** after meal

I. Early Dumping

✱ **Aetiology :**

Rapid gastric empting with the delivery of a hyperosomolar solution to the proximal small gut with the result of shift of fluid from circulatory plasma to the small gut leading to ↑ Intestinal activity & ↓ blood volume.

✱ **Clinical picture :**

- G.I.T. symptoms as epigastric fullness & ending by explosive diarrhea.
- Vasomotor symptoms as sense of weakness, flushing & palpitation.

II. Late Dumping

✱ **Aetiology :**

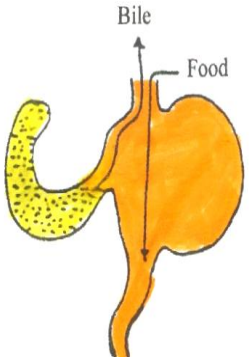
overshot of insulin which is caused by rapid delivery of large amounts of carbohydrates to the small intestine.

✱ **Clinical picture :** (**Hypoglycaemia**)

Sweating, palpitation & confusion relieved by carbohydrate ingestion.

3. Biliary gastritis

Alkaline reflux gastritis.



4. Afferent loop syndrome

It is a mechanical obstruction of the long afferent jejunal loop so that the bile and pancreatic juice accumulate until the obstruction is suddenly relieved.

5. Gastro-jejuno-colic Fistula

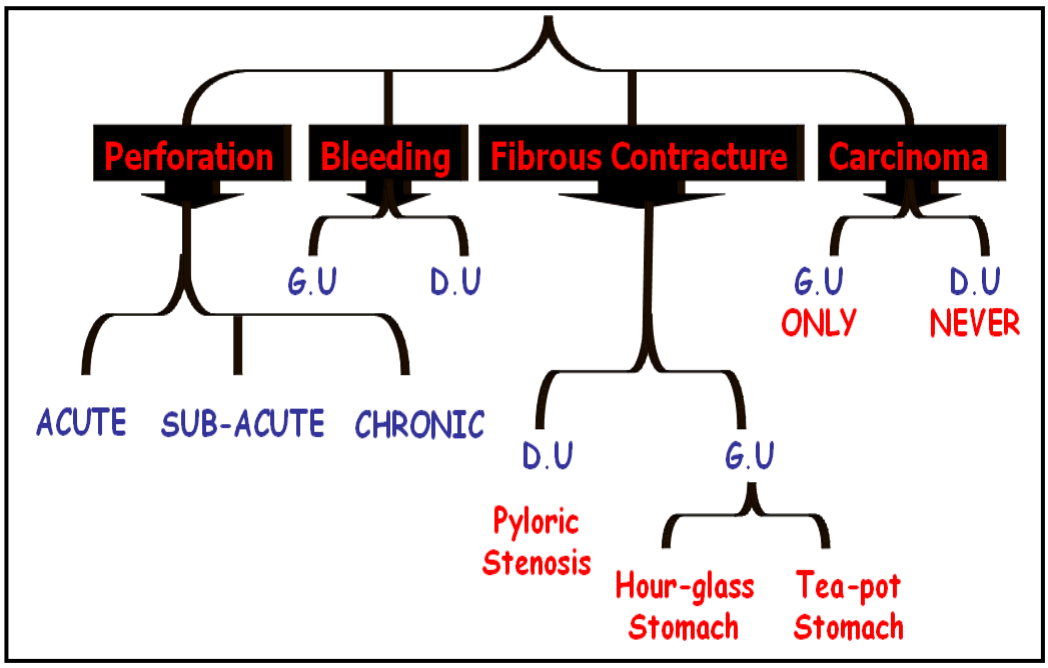
6. Intestinal obstruction

7. Gall stone formation

8. Post-gastrectomy nutritional disturbances

1. Weight loss.
2. Steatorrhea & diarrhea :
3. **Vitamin D deficiency :** Treated by Vit. D supplement.
4. **Ca deficiency :** Treated by Ca supplement.
5. **Fe deficiency anemia :** Treated by I.M Vit. B12.

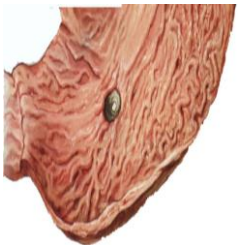
Complications of chronic peptic ulcer



I. Perforation

1. Acute perforated peptic ulcer

INCIDENCE

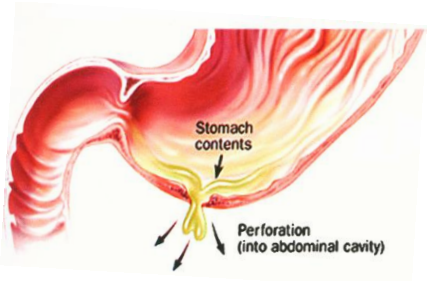


- Anteriorly situated ulcers perforate more commonly.
- Duodenal ulcer perforates more commoner than gastric ulcer.

AETIOLOGY

- Perforation occur during periods of exacerbation of an ulcer.
  i.e. **w**orry, **w**ork, **w**ines & **w**eather ……etc.
- Perforation occur due to relapse precipitated by irritant food.
- Perforation occur due to continuous using of ulcerogenic drugs.

PATHOLOGY [**3 stages**]



Stage of perforation

- Escape of gastro-duodenal contents into the peritoneal cavity.

Stage of chemical peritonitis (Illusion)

- The peritoneum will react by production of alkaline fluid neutralizing the acidity
  ➔ Peritonitis which is sterile so called "**chemical peritonitis**".
- This stage called (**lucid interval**), this stage lasts from **3 - 6 hours**

Stage of septic peritonitis

- It occurs when the body resistance fails.
- Micro-organisms invade peritoneal cavity then pus is formed.

CLINICAL PICTURE (**Corresponding to pathological stages**)

About **80 %** of cases with "**+ve**" history for peptic ulcer but only **20 %** of cases with "**silent**" chronic ulcer that perforate

Stage of perforation

- Sudden sever upper abdominal pain & collapse from pain
  i.e. Neurogenic shock.

- **GENERAL :** - Pallor & sweating.
  - **Pulse :** increased.
  - **Temp. :** subnormal.
- **LOCAL :**
  Upper abdomen shows tenderness & rigidity.

Stage of chemical peritonitis

- Patient is relieved from shock & he comes walking to the hospital
  i.e. Lucid interval

- **GENERAL :** - **No** pallor or sweating.
  - **Pulse :** still increased.
  - **Temp. :** normal.
- **LOCAL :**
  - **Inspection :** No abdominal movement with respiration.
  - **Palpation :** Tenderness & rigidity of the abdomen.
  - **Percussion :** Obliterated liver dullness due to escape of gas from perforated gut.

Stage of septic peritonitis

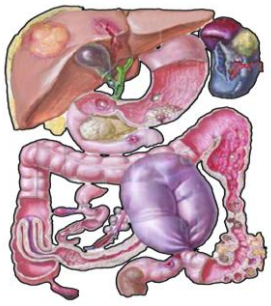
- Patient shows toxic symptoms then finally septicemia & shock
  i.e. Septic shock

- **GENERAL :** - Patient is toxic.
  - **Pulse :** marked increased.
  - **Temp.:** increased.
- Patient also complains of constipation, distension & vomiting

- **LOCAL :**
  - Dead silent distended abdomen on auscultation.
  - Patient will die in few days if untreated

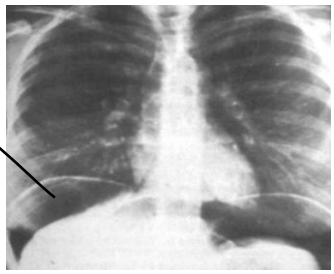
D.D "**Other causes of acute abdomen**" as ⇩

① **Acute perforated peptic ulcer**.
② Acute cholecystitis.
③ Acute pancreatitis.
④ Acute sub-hepatic appendicitis
⑤ High intestinal obstruction.



INVESTIGATIONS AFTER RESUSCITATION

1- Plain X-ray "abdomen" "In **erect** position"
- Reveal **air** under diaphragm.



2- Gastro-graffin swallow
- Reveal escape of **dye** through the perforation.

3- U/S - Reveal intra-abdominal **fluid**.

4- Peritoneal tapping
- Reveal **bile**

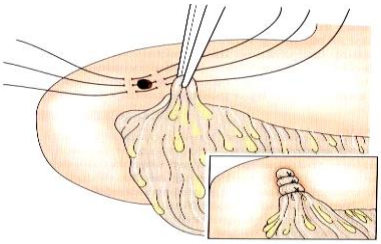
TREATMENT

A- Preoperative (**Urgent resuscitation**)

① A Ryle's tube for suction & careful attention to respiration.
② I.V fluids guided by electrolyte & PH estimations.
③ I.V A.B. & omeprazole.

N.B.: Blood transfusion is indicated with late cases with toxemia

B- Urgent operation (**Laparotomy**)



⇨ **The simplest procedure :**
Closure of perforation with covering omental patch, then peritoneal toilet then abdominal closure with drain.

⇨ **Some surgeons perform definitive operation :**
Provided that good general condition of patient.

- **IF DUODENAL ULCER** ➔ Vagotomy + drainage procedures.
- **IF GASTRIC ULCER** ➔ Partial gastrectomy + gastro-duodenostomy.

N.B.: Biopsy is taken to exclude malignancy **if G.U**.

C- Post- operative

The patient is treated medically for his ulcer disease.

2. Sub-acute perforated peptic ulcer

- This is a small perforation allowing only a minimal amount of contents to enter the peritoneal cavity and is rapidly sealed.

- If diagnosed correctly it can be treated conservatively.

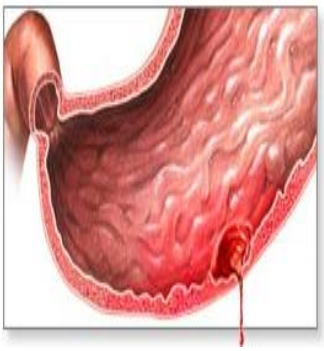
3. Chronic perforated peptic ulcer

- This is a penetration into adjacent structures as the pancreas.

- It diagnosed when the pain referred to the back.

II. Bleeding

INCIDENCE

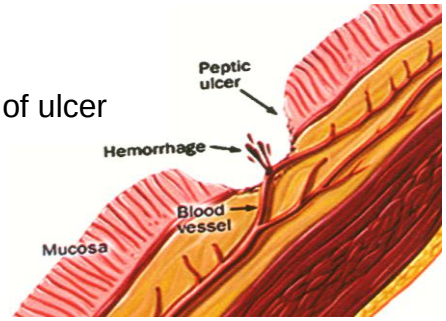


- **Hematemesis :** more common with gastric ulcer.
- **Melena :** more common with duodenal ulcer

AETIOLOGY

- It occurs during periods of exacerbation of an ulcer.
  i.e. **w**orry, **w**ork, **w**ines & **w**eather ……etc.
- It occurs due to relapse precipitated by irritant food.
- It occurs due to continuous using of ulcerogenic drugs.

PATHOLOGY (**3 degrees**)



1- Mild degree

Bleeding from granulation tissue of the floor of ulcer caused from trauma by solid food.

2- Severe degree

Erosion of a small vessel in the ulcer.

3- Fatal degree

Penetration of a large extra-gastric vessels as splenic artery or gastro-duodenal artery running near the ulcer

CLINICAL PICTURE

- **Hematemesis :** Dark red & acidic with food particles.
- **Melena :** Altered blood in stool
- **Sign of progressive hypovolemic shock :**
  ① Tachycardia & tachypnea (air hunger)
  ② Hypotension & hypothermia.
  ③ Pale cold skin & oliguria.

D.D " **Other causes of upper G.I.T hemorrhage** "

I- General causes

Hypertension, purpura or hypoprothrombinaemia.

II- Local causes

- **OESOPHAGEAL CAUSES :**
  ① Oesophageal varices.
  ② Oesophageal carcinoma.
  ③ Oesophagitis (reflux)
- **GASTRO-DUODENAL CAUSES :**
  ① Chronic peptic ulcer
  ② Gastric carcinoma.
  ③ Acute gastric erosions & acute peptic ulcer

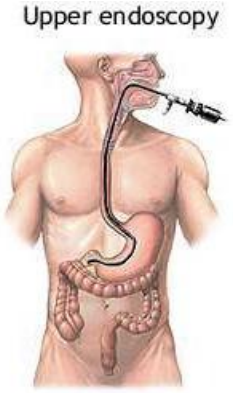
In Egypt **The commonest 3 causes are** ⇦

① Oesophageal varices
② Chronic peptic ulcer .
③ Acute gastric erosion.

	HEMATEMESIS	HAEMOPTSIS
HISTORY	G.I.T troubles.	Chest troubles.
PRECEDED BY	Vomiting.	Cough.
FOLLOWED BY	Melena.	Blood stained sputum.
THE BLOOD	Dark red, acidic with food particles.	Bright red, alkaline with frothy sputum.

	BLEEDING ULCER	BLEEDING VARICES
HISTORY	Dyspepsia relieved by antacids.	Liver cirrhosis & hepatosplenomegaly
ENDOSCOPY	**+ve** ulcer.	**+ve** varices.
PORTAL PRESSURE	Normal pressure	High pressure
SENGESTAKEN TUBE	No effect.	Control bleeding

INVESTIGATIONS AFTER RESUSCITATION



1- HB% & haematocrite value

- For proper replacement of blood & plasma

2- Endoscopy

- The ulcer may show blood clot at the floor.

3- Blood examination

- To exclude blood diseases as purpura

TREATMENT

A- Conservative treatment

⇨ RESUSCITATION :

- The aim is to restore the blood volume & arresting the bleeding

1- Absolute bed rest under opiate sedation.

2- Ryle's tube :

To ① Asses the amount of bleeding.
② Prevent it's propagation to the gut .
③ Irrigate the stomach with ice-cold saline

3- I.V blood transfusion to correct the hypovolaemic.

⇨ DIAGNOSIS :

- **When the condition of the patient becomes stabilized.** investigations are carried out (as before)

⇨ SUB-SEQUENT TREATMENT

1- The patient is given the routine treatment for peptic ulcer including Cimitidine (Tagamet) I.V & antacids.

2- Frequent & light diets.

3- Observation charts : for pulse, A.B.P., temp., urinary output & amount of bleeding …etc.

B- Surgical treatment

⇨ INDICATIONS :

① If patient is already under medical treatment.

② If the bleeding is **recurrent** while patient still in hospital

③ If Initial bleeding is severe & equal **2** liter or more

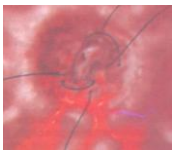
④ If the bleeding is continued inspite of transfuse **1** litter or more per day to maintain stability

⑤ If old atherosclerotic patient

⑥ If the patient with long history of ulcer disease.

⇨ OPERATIVE PROCEDURE (CONTROL OF BLEEDING ONLY)

If bleeding coming from ⇩



- **THE ULCER FLOOR :**
  Obliterate ulcer by sutures.
- **ERODED VESSELS AT THE EDGE :**
  Under running on either side of ulcer.
- **EXTRA-GASTRIC VESSELS :** legate them.

⇨ OPERATIVE PROCEDURES WITH FIT PATIENT

The decision should be taken early (within **48** - **72** hours)

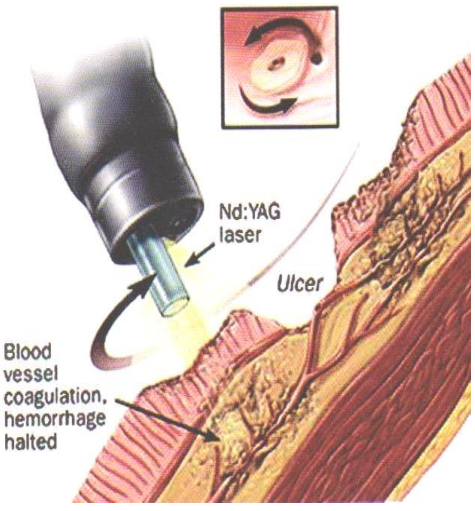
- **IF DUODENAL ULCER** → Vagotomy + drainage procedures.
- **IF GASTRIC ULCER** → Partial gastrectomy + gastro-duodenostomy.

N.B.: Biopsy is taken to exclude malignancy if **G.U**.

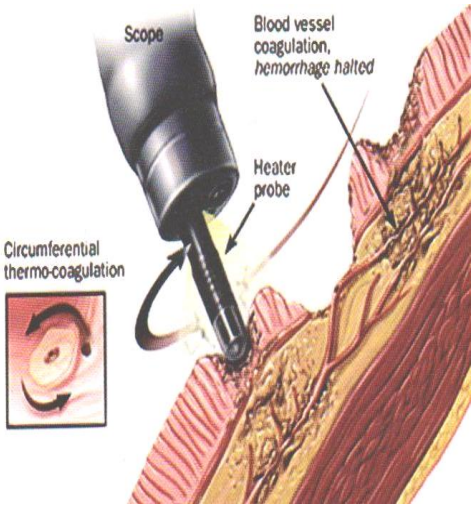
C- Endoscopic Treatment (**The most recent**)

⇨ BY USING :

LASER PHOTOCOAGULATION ⇩

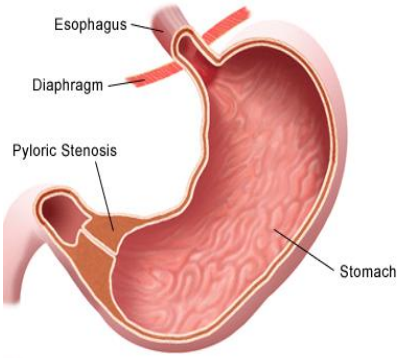


DIATHERMY COAGULATION ⇩



III. Fibrous contractures of peptic ulceration

1. Pyloric stenosis



DEFINITION

It is a narrowing of pyloric canal interfering with the downwards passage of gastric content

AETIOLOGY

Due to excessive fibrosis around the duodenal ulcer

PATHOLOGY

The Pylorus Cicatrized & stenosed.

The Stomach At Ist hypertrophied to overcome the obstruction, but later on dilated (may reach the pelvis) with gastritis from retention.

The Intestine Normal & collapsed.

CLINICAL PICTURE

Patient with **long history of ulcer dyspepsia,** or it may be the **1st presentation**.

Symptoms

1- VOMITING :

- Occurs once/day at the evening.
- Projectile, non-bile stained & containing food from previous meal with foul odor
- After vomiting, the initial periodic ulcer pain is lost and replaced by epigastric discomfort.

2- PROGRESSIVE CONSTIPATION

3- COMPLICATIONS :

- Dehydration & loss of weight.
- Chest infection (**aspiration pneumonia**)
- Metabolic alkalosis ➔ **Tetany** from repeated vomiting

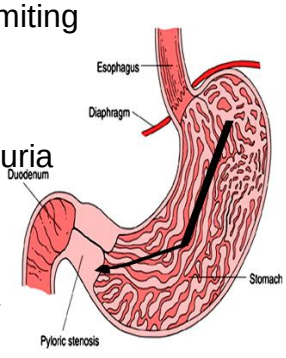
Signs

1- GENERAL "Signs of dehydration"

Dry inelastic skin, dry tongue, sunken eyes & oliguria

2- LOCAL :

- Upper abdominal distention.
- Visible peristalsis from Lt. to Rt. ⟶
- Succession splash by auscultation.



D.D From other causes of (Gastric outlet obstruction) as

① **CONGENITAL : C**ongenital **H**ypertrophic **P**yloric **S**tenosis (**CHPS**)

② **TRAUMATIC :** Impacted F.B (**bezoars**) or badly performed pyloroplasty.

③ **INFLAMMATORY :** Cicatrized duodenal ulcer & rarely T.B

④ **NEOPLASTIC :** Cancer pylorus .

⑤ **FUNCTIONAL:** Pylorospasm or achalasia of pylorus

⑥ **PRESSURE FROM OUTSIDE :** lymphoma or T.B lymph nodes or cancer head pancreas.

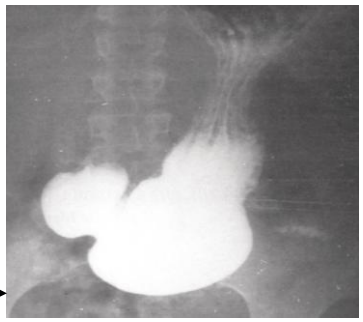
The commonest causes are : Cicatrized D.U. & cancer pylorus

INVESTIGATIONS

1- Laboratory "Serum electrolytes"
Metabolic alkalosis & ↓ Na

2- Endoscopy
Stenosed pyloric ring & retention gastritis.

3- Barium meal
Huge dilated stomach may reach down to the pelvis with fluid level in the stomach.
so it has **Soup dish appearance**



TREATMENT "Only surgical"

⇨ **1ST LINE OF TREATMENT :** Correct the dehydration & electrolytes imbalance.

⇨ **PRE-OPERATIVE PREPARATIONS :**

① **Stop feeding**, then naso-gastric suction with normal saline .

② **High protein diet** with I.V hyperalimentation .

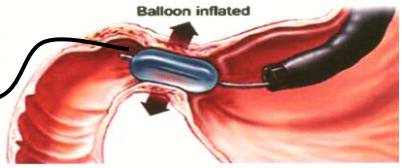
③ **Chest antibiotics**.

⇨ **OPERATION :** (**Trunkal vagotomy** & **gastrojejunostomy**)

OTHER OPTIONS ⇩

① **Endoscopic balloon dilatation**

② **Gastro-jejunostomy** alone in elderly.



2. Hour glass stomach

- **It is due to** cicatrized ulcer on lesser curve of the stomach.
- **The stomach is divided into 2 pouches** (proximal & distal).
- **It is treated by** partial gastrectomy i.e. removal of distal pouch.

3. Tea pot stomach

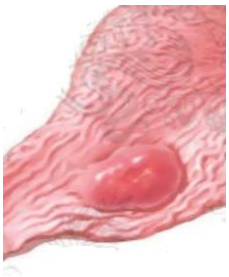
- **It is due to** shortening of lesser curve, thus causing the pylorus becomes higher & represented by features of pyloric obstruction.

IV- Malignant ulcer

- **DUODENAL ULCER : Never** to turn malignant.
- **GASTRIC ULCER : Very rare < 1 %** to turn malignant

V- NEOPLASMS OF THE STOMACH

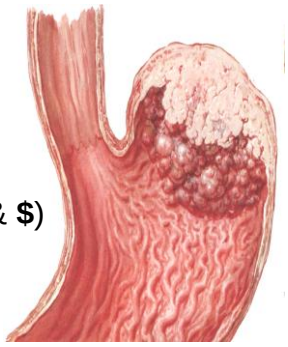
A- Benign neoplasm



- **Incidence :** The commonest is **leiomyoma**.
- **Clinical picture :** Vague symptoms or epigastric mass
- **Investigations (Barium meal)** shows **smooth** filling defect
- **Treatment :** Excision & biopsy

B- Cancer stomach

INCIDENCE



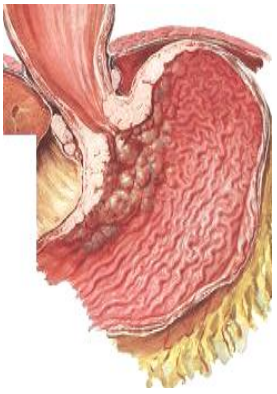
- The commonest **upper** G.I.T carcinoma.
- **Age :** > **40** years
- **Sex :** Male **>** female

PREDISPOSING FACTORS

- **Chronic irritations** as **5S** (**s**pirits, **s**pices, **s**moking, **s**epsis & **$**)

N.B Other Factors as

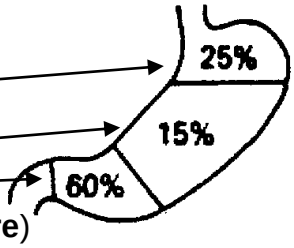
① **Diet** containing carcinogens as hydrocarbons
② Blood group (**A**) & also common in **Japan**



- **Pernicious anemia**
- **Helicobacter pylori** (not sure)
- **Achlorohydria**.
- **Benign gastric neoplasm** as Leiomyoma
- **Benign gastric ulcer** (very rare < 1 %)

PATHOLOGY

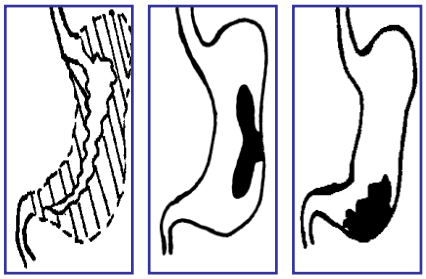
Site



- The cardia = **25 %**
- The body = **15 %**
- The pylorus = **60 %**
- The whole stomach (**very rare**)

NIE picture 2 types

CLASSIC DESCRIPTION

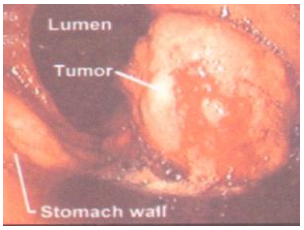


- **Diffusely infiltrating type** "**linitis plastica**"
  The wall of stomach is greatly thickened & indurated while the lumen is greatly reduced. so it is also called "**leather bottle stomach**"
- **Ulcerative** type.
- **Cauliflower** (fungating) type

RECENT DESCRIPTION

- **Protruding** type - **Superficial** type - **Excavated** (penetrated) type

Microscopic picture



- **Adenocarcinoma** carcinoma **95 %.**
  N.B.: Colloid cell adenocarinoma (**poor** prognosis)
- **Squamous** cell carcinoma **5 %** from cancer oesophagus

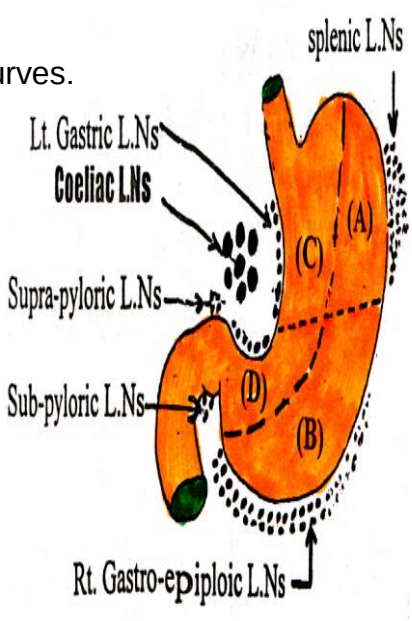
SPREAD

Direct

Both in (circumferential & depth) of the wall of stomach then infiltrates the surroundings as liver, spleen, colon & pancreas.

Lymphatic By both (**embolization** & **permeation**)

THE STOMACH IS DIVIDED INTO 4 AREAS



- **A line parallel** (in between) greater & lesser curves.
- **Another perpendicular line pass** through this line to the lesser & greater curves.

AREA (A) drains into the splenic LNs then Lt. gastric L.Ns ➔ **coeliac** LNs.

AREA (B) drains into Rt. gastro-epiploic LNs ➔ sub-pyloric L.Ns ➔ supra-pyloric LNs ➔ **coeliac** LNs.

AREA (C) drains into Lt. gastric LNs ➔ **coeliac** LNs.

AREA (D) drains into supra-pyloric LNs ➔ **coeliac** LNs.

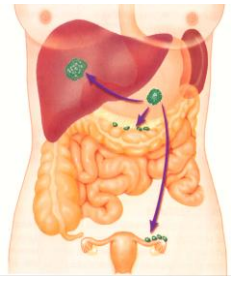
- **FINALLY :** The **coeliac** L.Ns drain into para-aortic LNs ➔ Thoracic duct ➔ Lt. supra-clavicular (**Virchow's gland**) ➔ **+ve TROISIER SIGN.**

Blood

Mainly liver & rarely lung & bone.

Trans- peritoneal

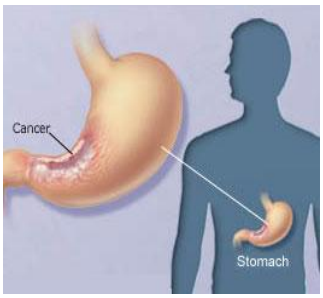
The malignant cells set free in the peritoneal cavity so may spread to the ovaries in young female ➔ **Krukenberg's** tumor & may spread to the douglas pouch leads to what is known as (**Plummer's shelf**)



STAGING T.N.M

T = Tumor	N = Nodes	M = Metastases
T_{is} = cancer in situ	N_0 = LN cannot be assessed	M_0 = **No** metastases.
T_1 = confined to mucosa.	N_1 = peri-gastric LNs within **3 cm**	M_1 = distant metastases.
T_2= involved submucosa.	N_2 = LNs **> 3cm.**	
T_3 = penetrate serosa.	N_3 = involvement of other central LNs. as (coeliac)	
T_4= diffuse involvement of stomach wall.		

CLINICAL PICTURE



Type of patient (**> 40** years & Male > female)

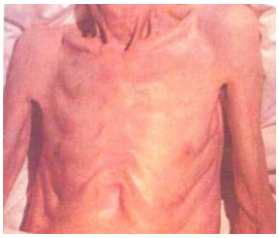
Manifestations

- The disease is usually diagnosed at a late stage because of vague & mild symptoms.
- It may be represented by ⇩

1- DYSPEPSIA GROUP

- A person > 40 years who start to complain dyspepsia **should be** fully investigated for possibility of cancer stomach.
- **It differs from chronic gastric ulcer as in** ⇩
  - ① **Pain** is severe & continuous.
  - ② **Not** relieved by antacids.
  - ③ Vomiting is **blood stained**

2- INSIDIOUS GROUP



- **Vague symptoms as** ⇩
  - ① **A**nemia (pallor & easy fatigability)
  - ② **A**thenia (wasting & weakness)
  - ③ **A**norexia (especially to meat)

3- OBSTRUCTIVE GROUP

- **Patient with manifestations due to obstruction as** ⇩
  - ① Dysphagia with tumor of cardia
  - ② Pyloric obstruction with tumor of pylorus

4- MASS GROUP

- **Patient with epigastric mass** for (D.D) **30 %** of cases.

5- METASTATIC GROUP (i.e. Occult carcinoma)

- **Patient with metastasis** as hard irregular liver, jaundice, **Virchow's** gland, **Troisier** sign & **Krukenberg's** tumors.

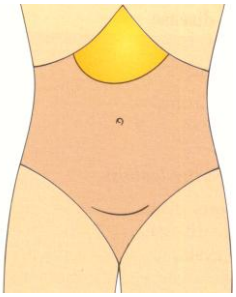
6- OTHER MANIFESTATIONS

- **Patient with** ① Hematemesis & melena (uncommon)
  ② Superficial thrombophlebitis i.e. **Trousseaus** sign.

DEFERENTIAL DIAGNOSIS

A- Causes of epigastric mass

1- Abdominal wall masses, e.g. lipoma.



2- Intra-abdominal masses :

- ① **Stomach :** pyloric obstruction or cancer .
- ② **Lt. lobe liver :** tumor or cyst
- ③ **Transverse colon :** carcinoma or intussusception
- ④ **Pancreas :** cancer pancreas or pseudo-cyst
- ⑤ **Aorta :** aneurysm or para-aortic L.Ns.

3- Epigastric hernia.

B- Causes of athenia as presentation

e.g. cancer stomach, cancer liver & cancer caecum.

INVESTIGATIONS

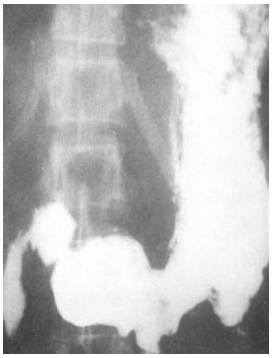
A- Laboratory

1- **Complete blood picture** for pernicious anemia.

2- **Tumor marker** (**C.E.A**) **C**arcino-**e**mbryonic **a**ntigen

B- Barium meal (**75 %** accuracy)

1- **Irregular filling defect** in the pyloric antrum or the body of the stomach.

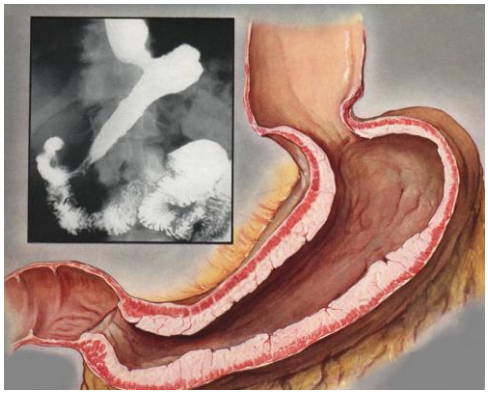


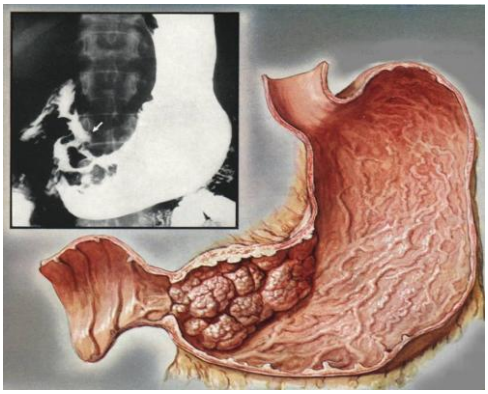
2- **In fundal carcinoma**, patient should be examined in **trendlenburg position** to see the tumor in the fundus.

3- **Large ulcer** outside ulcer bearing area.

4- **Linitis plastica :** marked narrowing of the lumen of the stomach

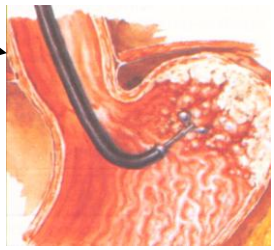
5- **Pyloric obstruction** with proximal dilatation





C- Gastroscopy & biopsy

Demonstrates the lesion (**most accurate**)



D- Metastatic work up

1- **Liver (U/S)** for liver metastasis.

2- **Chest (X-ray)** for chest metastasis

3- **Abdomen (CT scan)** for L.N. involvement.

TREATMENT

A- Operable cases

RADICAL EXCISION

① Removal of tumor with safety margin at least **5 cm** above & **1.5 cm** of the duodenum .

② Both omenta.

③ All draining L.Ns

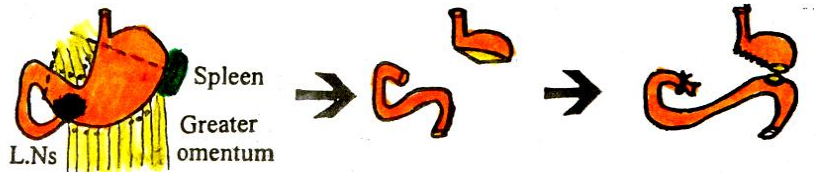
④ Ligation & division of corresponding vessels.

So ACCORDING TO THE SITE of neoplasm **the extent of resection will be determined :**

A- Tumor of the pylorus

SUBTOTAL LOWER RADICAL GASTRECTOMY

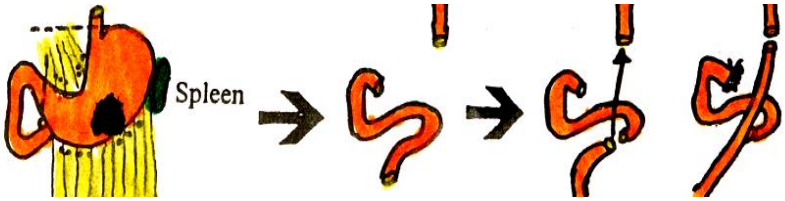
THEN anastomosis of the remaining upper stomach to the jejunum



B- Tumor of the body

TOTAL RADICAL GASTRECTOMY

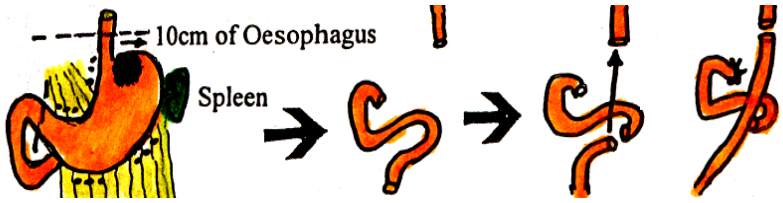
THEN anastomosis the oesophagus to " **Roux-en-Y**" loop of jejunum



C- Tumor of the fundus

OESOPHAGO-GASIRECTOMY "**10 cm from esophagus**"

THEN anastomosis the oesophagus to " **Roux-en-Y**" loop of jejunum



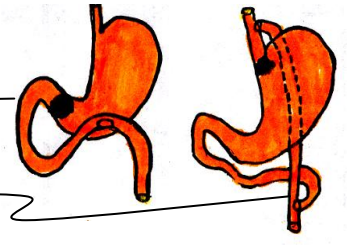
B- Inoperable

PALLIATIVE PROCEDURES

① Palliative gastrectomy

② Palliative anterior gastrojejunostomy
for irremovable tumors in the pyloric region.

③ Palliative oesophago-jejunostomy
for irremovable tumors in the cardiac region



N.B.: Radiotherapy & **chemotherapy : (little value)**
Used in irremovable, incompletely resectable or recurrent cases

PROGNOSIS " **Very bad** "

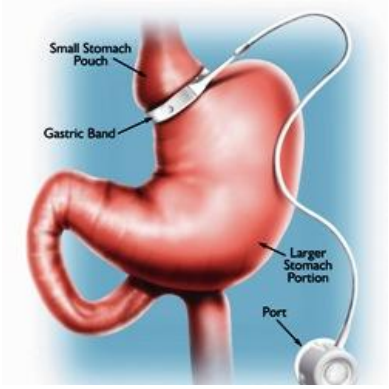
- Only about **40 %** are operable & only **5 %** are alive **5** years after surgery.
- But cure rate **80 - 90 %** in **Japan** because of early detection

VI- BARIATRIC SURGERY

DEFINITION

- **Bariatric surgical procedures** to cause weight loss by restricting the amount of food to the stomach

GRADES OF OBESITY



The degree of obesity can be measured by calculating the **b**ody **m**ass **i**ndex (**BMI**).

BMI = Body weight in kg/Height in metres

Ideal weight 18-25

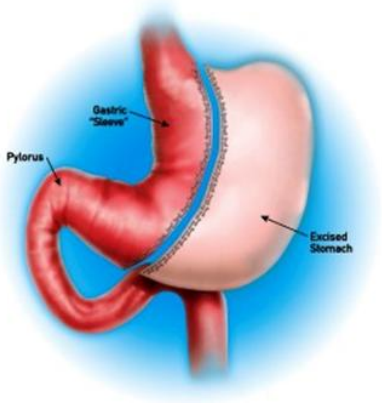
Overweight 25-30

Mild obesity 30-35

Moderate obesity 35-40

Severe (morbid) obesity > 40

TYPES OF PROCEDURES



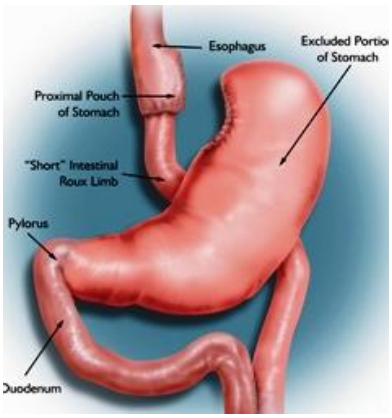
1- Restrictive procedures

(decrease size of stomach)

① **Gastric Stapling** which may be :
- **a. Vertical**
- **b.Transverse**

② **Gastric Banding** which may be:
- **a. Ordinary**
- **b. Adjustable**

③ **Sleeves Gastrectomy** which may be :
- **a. Open surgery**
- **b. Laparoscopic**



2- Malabsorpative procedure

AS **Jeujono-ileal bypass**

3- Combined procedure

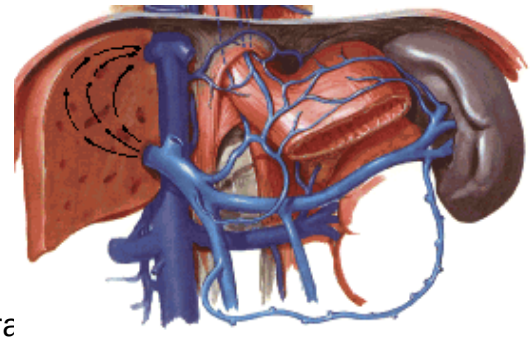
AS **Gastrectomy & Gastrojeujonostomy**

Portal hypertension

PORTAL HYPERTENSION

ANATOMICAL CONSIDERATIONS

Portal vein



Begins by union of superior mesenteric vein & splenic Vein

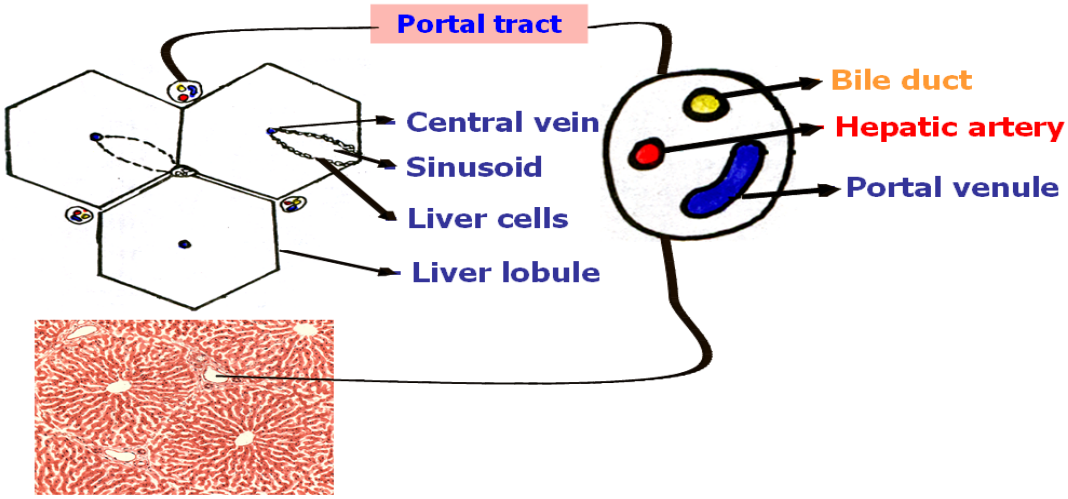
End by divided into Rt. & Lt at the liver,

It drains G.I.T from lower oesophagus to the 1st part of anal canal.

Surgical importance See porto-systemic collatera

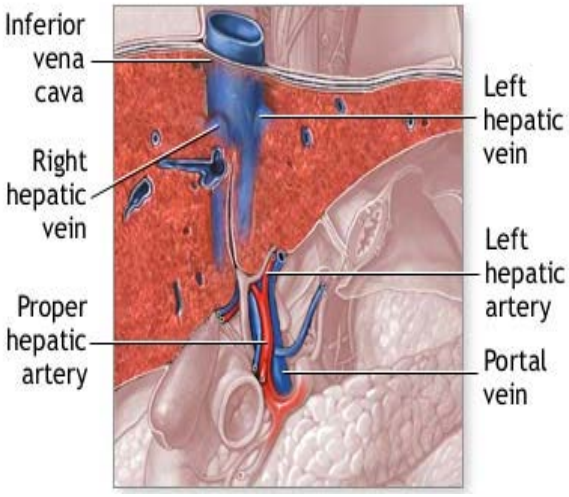
HISTOLOGICAL CONSIDERATIONS

Histology of liver lobules



PHYSIOLOGICAL CONSIDERATIONS

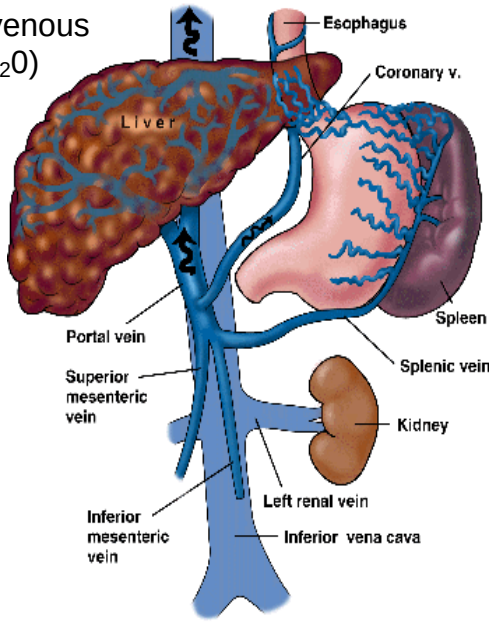
- Normal portal vein pressure
  = 7 mmHg (**8 - l2** cm H_20).
- The liver receives blood from
  both hepatic artery & portal vein so it derives 1/2 of its oxygen from hepatic arterial blood & the other 1/2 from the portal venous inflow
- The average blood flow to the liver is **1500 ml/min** which is
  2/3 from portal vein &
  1/3 from hepatic artery.



PORTAL HYPERTENSION

DEFINITION

- **Portal hypertension** is present when portal venous pressure exceeds 20 mmHg (25 - 30 Cm H_20)



AETIOLOGY

- **Portal hypertension** occurs mainly due to resistance to the portal venous flow.

- **It is classified as** ⇩

I- Pre-hepatic causes

① Congenital atresia of portal vein.

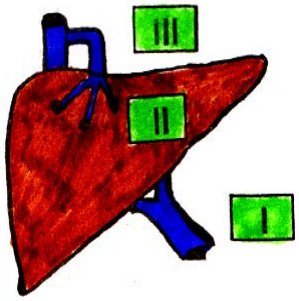
② Portal vein thrombosis due to ⇩
1- Neonatal umbilical sepsis.
2- Intra-abdominal sepsis .
3- Oral contraceptive pills.

③ Extrinsic compression
e.g. cancer head pancreas.

II- Intra-hepatic causes

① Pre-sinusoidal e.g. **Schistosomiasis** through peri-portal fibrosis.

② Sinusoidal e.g. **Liver cirrhosis** due to ⇩
1- Obliteration & compression of sinusoids.
2- Compression of the radicals of portal & hepatic vein by regenerating nodules.



③ Post- sinusoidal e.g. **Veno-occlusive disease** through obliteration of small hepatic venules by sub-endothelial inflammation & edema

III- Post-hepatic causes

① Budd-chiari syndrome (**rare**) which is caused by obstruction of main hepatic veins at their opening into I.V.C due to ⇩
1- Malignant invasion by tumors.
2- Spontaneous thrombosis or 2^{ry} to polycythemia.

② Constrictive pericarditis & pericardial effusion.

③ RVF & T.I.

CLINICAL PICTURE

1- Congestion of whole G.I.T tract

Leads to "anorexia, dyspepsia, indigestion & malabsorption."

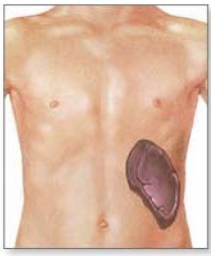
2- Liver

① **In pre-hepatic** causes : usually ***normal*** liver.

② **In inter-hepatic** causes : ***shrunken*** as cirrhosis.
or ***enlarged*** as schistosomiasis.

③ **In post-hepatic** causes : ***Congested*** liver.

3- Splenomegaly



CAUSES

① **Congestion** due to portal venous pressure.

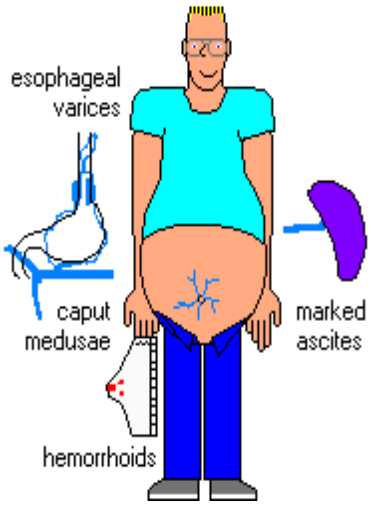
② **Hyperplasia** of R.E.S due to bilharzial toxins

③ **Deposition** of living worms & ova at spleen.

PRESENTATION

- Firm mass at Lt. hypochondrium.
- Pain which may be ⇩
  - ➢ **Dull ache** i.e. stretching the of capsule.
  - ➢ **Dragging** i.e. pulling on the ligaments.
  - ➢ **Stitching** (**stabbing**) i.e. perisplenitis.

N.B.: Peri-splenitis :
due to t attacks of splenic infarction
(Not inflammation)



- Pressure symptoms
  e.g. **dyspepsia** due to pressure on stomach
  or **dyspnea** due to pressure on lung.

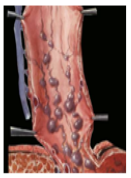
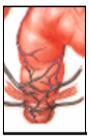
2RY HYPERSPLENISM (**may occur**)

- Splenomegaly

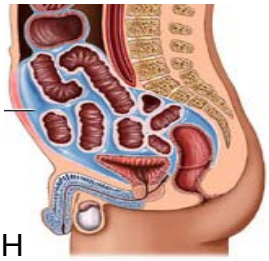
+ pancytopenia (anemia, leucopenia & thrombocytopenia) due to over activity of spleen

+ Active bone marrow

4- Porto-systemic collaterals

Site	Between		Result
	Portal vein	Systemic vein	
☛ Lower end of Oesophagus & Fundus of Stomach	• Oesophageal veins from Lt. Gastric veins & short gastric vein	• Oesophageal Veins from Azygos & Hemi - Azygos	• Oesophageal Varices 
☛ Lower end of Rectum & Anal Canal	• Superior Rectal Vein (Inf. Mesenteric Veins)	• Middle &Inferior (Internal Iliac Veins)	• Ano-rectal varices i.e. Piles 
☛ Umbilicus	• Para- umbilical vein	• Superior & Inferior epigastric veins	• Caput Medusa 

5- Ascites



It is "MULTI-FACTORIAL" the most common causes are ↯

① PORTAL HYPERTENSION ➔ localized filtration of fluid into the peritoneal cavity

② HYPO- ALBUMINEMIA : if associated (**L.C.F**) i.e. liver cell failure

③ LIVER CONGESTION ➔ failure of liver to inactivate A.D.H & aldosterone ➔ salts & water retention.

6- Hepatic encephalopathy

DUO TO absorbed ammonia reach systemic circulation in increased amount through spontaneous porto-systemic collaterals.

C/P - Pre-coma

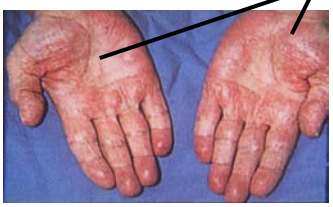
Hypersomnia, inverted sleep rhythm, apathy, micturation or defection in unsuitable places, childishness ….etc .

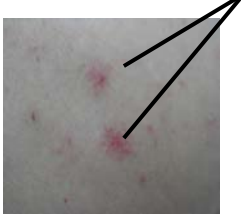
- Coma

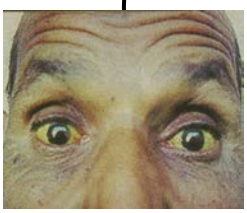
7- Liver cell failure (L.C.F)

MANIFESTED BY

- Low grade fever, fetor hepaticus, ascites & **jaundice**
- Gynaecomastia in male, atrophy of female breast & skin manifestations as **palmar erythema** & **spider naevi** .







- Bleeding tendency, anemia … etc

D.D " **Other causes of upper G.I.T hemorrhage** "

I- General causes

Hypertension, purpura or hypoprothrombinaemia.

II- Local causes

- OESOPHAGEAL CAUSES :
  - ① Oesophageal varices.
  - ② Oesophageal carcinoma.
  - ③ Oesophagitis (reflux)
- GASTRO-DUODENAL CAUSES :
  - ① Chronic peptic ulcer
  - ② Gastric carcinoma.
  - ③ Acute gastric erosions & acute peptic ulcer

In Egypt **The commonest 3 causes are** ↯

① Oesophageal varices

② Chronic peptic ulcer .

③ Acute gastric erosion.

	HEMATEMESIS	HAEMOPTSIS
HISTORY	G.I.T troubles.	Chest troubles.
PRECEDED BY	Vomiting.	Cough.
FOLLOWED BY	Melena.	Blood stained sputum.
THE BLOOD	Dark red, acidic with food particles.	Bright red, alkaline with frothy sputum.

	BLEEDING ULCER	BLEEDING VARICES
HISTORY	Dyspepsia relieved by antacids.	Liver cirrhosis & hepatosplenomegaly
ENDOSCOPY	**+ve** ulcer.	**+ve** varices.
PORTAL PRESSURE	Normal pressure	High pressure
SENGESTAKEN TUBE	No effect.	Control bleeding

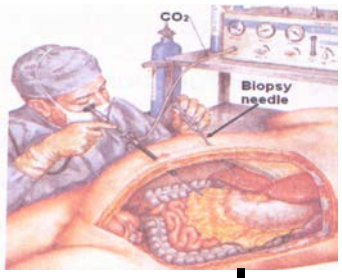
INVESTIGATIONS

1- Assessment of liver functions which will reveal

1- **Low** albumin level & **high** bilirubin level

2- SGOT & SG PT usually **high**

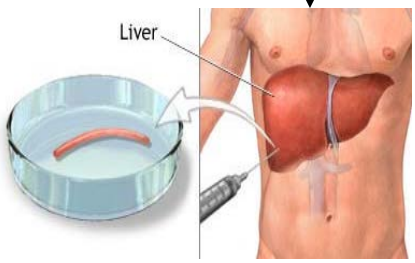
3- Prothrombin time is **prolonged**
& concentration is **low**



2- Assessment of liver state

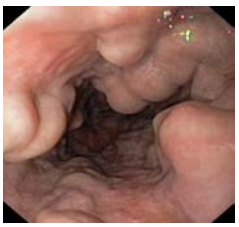
1- Immunological test for hepatitis markers.

2- Liver biopsy : The only, sure method after assessment of prothrombin time & concentration.



3- Abdominal U/S :
It shows (a) Cirrhotic liver
(b) ± Splenomegaly.
(c) Presence of ascites.

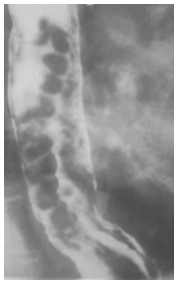
3- Detection of oesophageal varices & rectal polyps

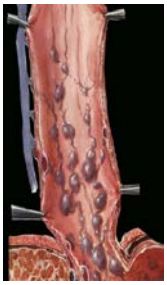


1- Fiber-optic upper endoscopy

Endoscopic grading of reflux :

- ***Grade I :*** Elevated normal mucosa
- ***Grade II :*** Elevated normal mucosa with elevated tortuous veins
- ***Grade III :*** No mucosa
- ***Grade IV :*** Impending rupture e.g. cherry-red spots





2- Barium swallow :
It shows varices as multiple, smooth & rounded filing defect
i.e. **Grape like appearance**. ⟶

3- Barium enema :
It may reveal presence of bilharzial polyps in sigmoid colon.

4- Detection of hypersplenism

1- Blood picture :

- It shows **pancytopenia**
  (anemia, leucopenia & thrombocytopenia)

2- Bone marrow examination :

- It shows **hypercellularity**.

3- Radio-active isotopic studies :

Using patients R.B.Cs tagged with (C_r^{51}) will reveal diminished half life of RBCs & increased radioactivity over spleen,
i.e. **↑↑ spleen / liver radioactivity index > 2 : 1 =** Hypersplenism

5- Visualization of portal system

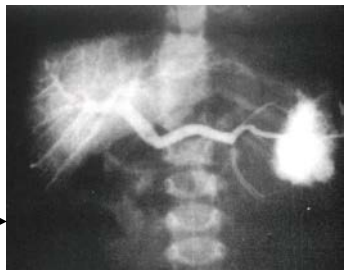
1- Ultrasonography

2- CT scan.

3- Digital subtraction angiography (D.S.A)

4- Splenoportography :

A dye can be injected into the spleen to demonstrate the anatomy of portal circulation & the site of obstruction →



6- Estimation of portal pressure

- Duplex U/S : (Recent, accurate & non invasive)
  to measure the amount & direction of blood flow in the portal vein.

7- Child's Pugh classification

- It gives good idea about the **functions of the liver**

	1	2	3
1- Serum bilirubin (mg %).	**1 - 2**	**2 - 3**	**> 3**
2- Serum albumin (gm %)	**> 3.5**	**3.5 - 3**	**< 3**
3- Ascites	**-ve**	Easily controlled	Poorly controlled
4- Nutritional status.	Excellent	Good	Poor
5- Neurological disorders.	None	Minimal	Coma

1 = **1** point, 2 = **2** points 3 = **3** points

The results

- CHILD A = **5 - 7** points = **Suitable** for surgery.
- CHILD B = **8 - 11** points = **Marginal** suitable for surgery.
- CHILD C = **12 - 15** points = **Unsuitable** for surgery.

N.B.: Modified Child's classification

1- Pugh's modification :

Prothrombin time is used instead of the nutritional status

so 1 = **12 -16** sec.

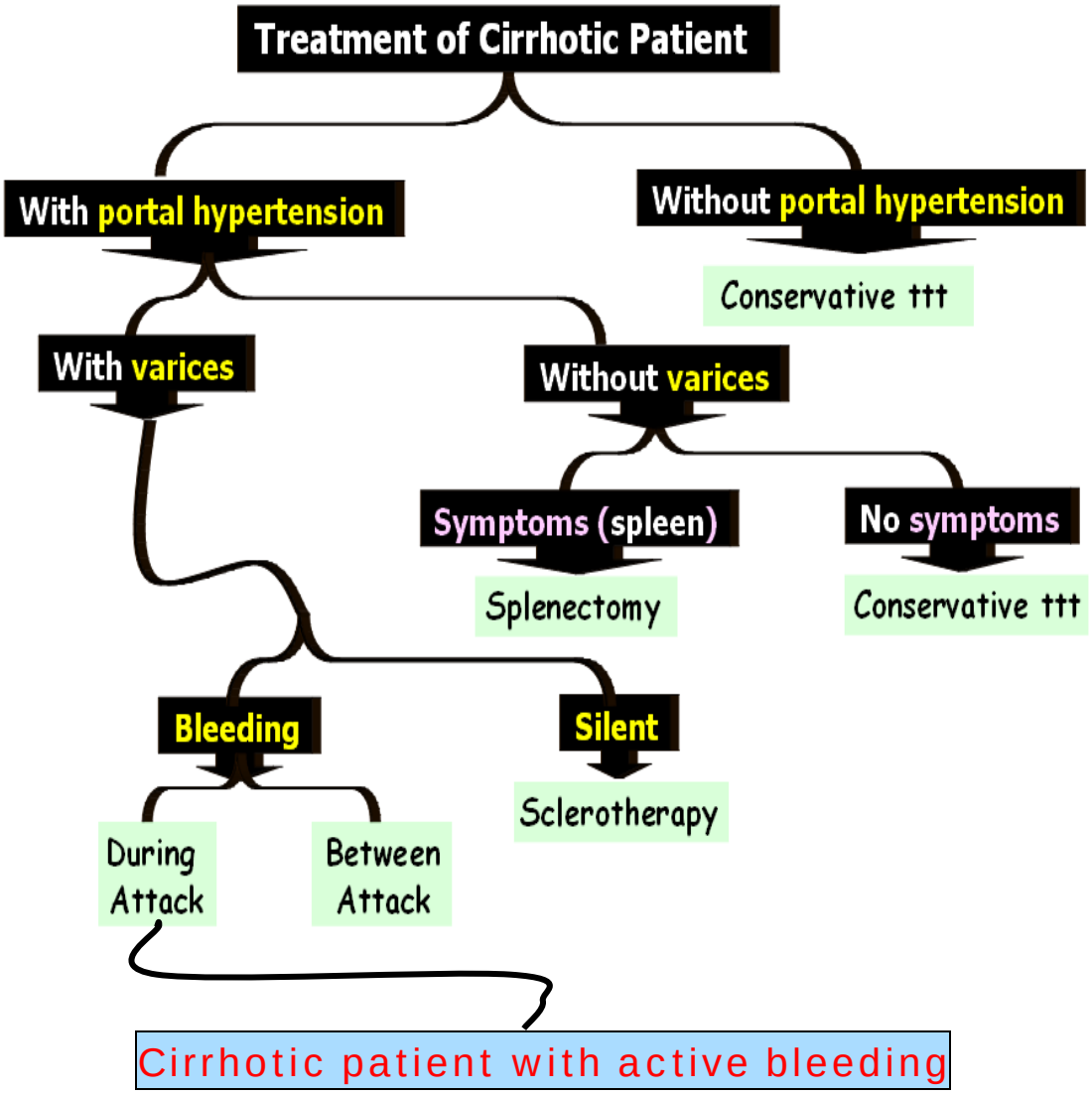
2 = **16 -18** sec .

3 = **> 18** sec.

2- Hobb's modification :

Prothrombin concentration is used instead of the prothrombin time

TREATMENT



1- Restoration of blood volume

- **Fresh blood transfusion** is the most important single factor.
  N.B.: Morphia is contraindicated due to liver affection

2- Prevention of hepatic encephalopathy

- Blood in the intestine will be fermented to ammonia.
  so these blood must be evacuated from G.I.T these occur by ⇩

1- STRONG LAXATIVE as magnesium sulphate.

2- STOMACH WASH must be done hourly

3- REPEATED ENEMA or colonic lavage.

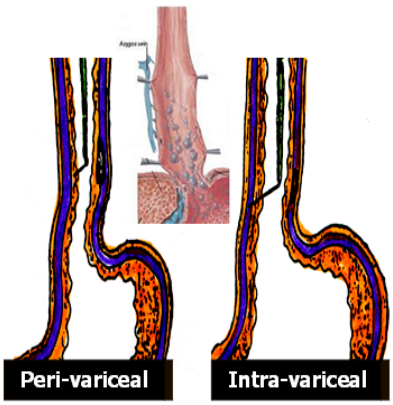
4- ORAL LACTULOSE : It is a disaccharide sugar
which is fermented by the intestinal flora to lactic acid.
The later will combine with ammonia & also it has a mild laxative effect.

5- NEOMYCIN : 0.5 gm/4 hours to reduce bacterial flora.

3- Measures to stop bleeding

1- I.V VITAMIN K.

2- ENDOSCOPIC INJECTION SCLEROTHERAPY :



Methods

- **Intra-variceal : 5** ml ***ethanolamine oleate***
  is inject to produce thrombosis
- **Peri-variceal : 0.5** ml ***aethoxysclerol***
  is injected to produce fibrosis

⇨ Both methods may be combined.

⇨ Injections must be repeated until the varices are obliterated.

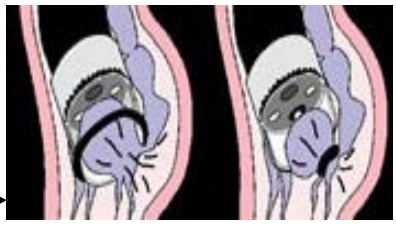
Side effects

① Retrosternal discomfort for few days

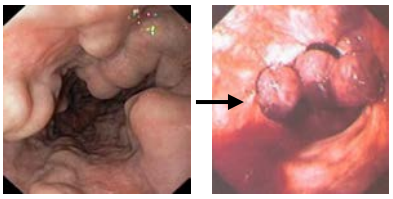
② Fever.

③ Oesophageal ulceration or strictures

④ Oesophageal perforation & mediastinitis



N.B.: ENDOSCOPIC BAND LIGATION →
- **The idea** is to encircle each varix by a tight band



3- DRUGS **as**

1- Vasopressin

DOSE **0.2** unit/kg body weight dissolved in **200** ml of 5 % dextrose and given over **20** min.

ACTION Vasoconstriction of splanchnic arterioles
→ ↓ Portal venous pressure.

SIDE EFFECTS
① Colicky abdominal pain
② Diarrhea.
③ Coronary spasm → anginal pain .

CONTRAINDICATED with elderly or cardiac patient

2- Glypressin

DOSE **2** mg / **6** hours I.V maximum for 24 hours.

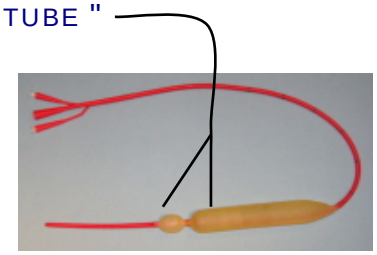
ACTION Same action of vasopressin but it controls variceal bleeding better than vasopressin

3- Somatostatin

ACTION Same action of vasopressin with strong action

DISADVANTAGE Very expensive

4- BALLOON TAMPONADE " SENGESTAKEN TUBE "

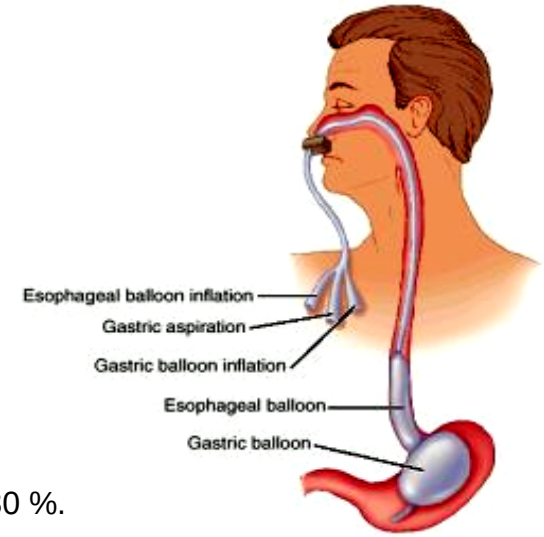


Method

The gastric balloon is inflated by **200 ml of air** & pulled upwards to press the gastric fundus

If bleeding not stopped

The oesophageal balloon as well is inflated but pressure should not exceeds **40 mmHg**



It is used as

A temporary measure before sclerotherapy or surgery.

Disadvantages

① Discomfort to the patient.

② The patient cannot swallow his saliva ➔ Chest infection

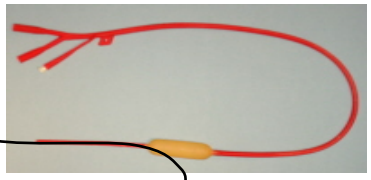
③ Oesophageal ulceration

④ Recurrent bleeding in 60 – 80 %.

⑤ Ulceration of the nostril

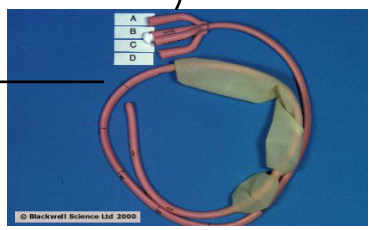
N.B 1. LINTON TUBE

Single gastric balloon.



2. MINNESOTA TUBE **(4 TUBES)**

The 4th tube is used to aspirate the oesophagus around the oesophageal balloon.



5- INFRA-RED PHOTOCOAGULATION :

The idea

Infra-red photocoagulation can give a tissue temperature of **100c** which produces an area of coagulation necrosis. The dead tissues separate after 10-14 days

6- EMERGENCY SURGERY

Indicated if all the previous measures fail to stop the bleeding

IF
- CHILD A, CHILD B → **Splenectomy + Porto-azygos disconnection**
- CHILD C → **Endoscopic stapling** alone can be performed

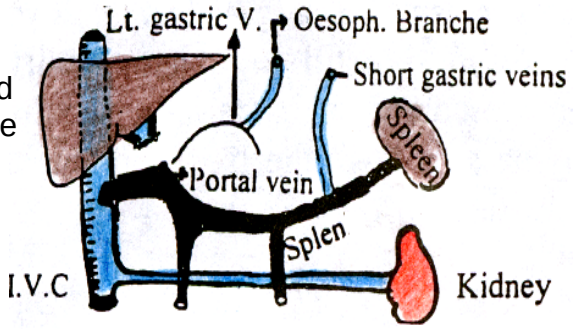
Surgical operations

SHUNT OPERATIONS	PORTO-AZYGOS DISCONNECTION
1- Total 2- Selective	(**Non shunt operations**)
The idea of these operations is to lower the portal pressure by shunting the portal blood away from the liver. so long as varices will collapse & bleeding stops	**The purpose** is to disconnect the collaterals between the portal & systemic circulations at the lower end of the oesophagus & the fundus of stomach

I- SHUNT OPERATIONS

1. Total shunt operations

A. Porto- caval operation



TECHNIQUE

The distal end of portal vein is divided & transfixed then anastomosed to the anterior surface of I.V.C.

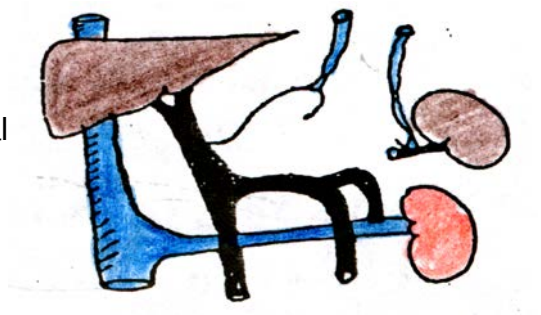
ADVANTAGE

lowering the portal pressure.

DISADVANTAGES

① Accelerate hepatic encephalopathy i.e passage of ammonia to brain.
② Accelerate liver failure i.e deprives the liver of the portal blood flow

B. Proximal spleno-renal shunt



TECHNIQUE

Splenectomy is done then the proximal end of splenic vein is anastomosed to left renal vein

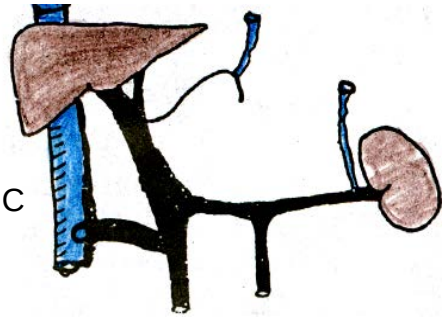
ADVANTAGE

lowering the portal pressure.

DISADVANTAGES

The risk of encephalopathy is still present.

C. Meso-caval shunt



TECHNIQUE

Insertion of a graft (synthetic vein) between the superior mesenteric vein & I.V.C

DISADVANTAGES

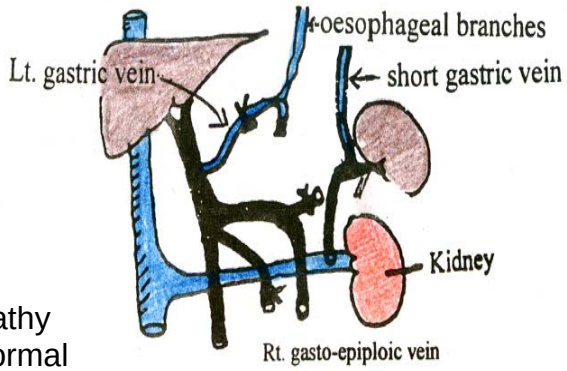
The incidence of thrombosis is high.

2. Selective shunt operation

Warren shunt

TECHNIQUE

The distal end of splenic vein is anastomosed to left renal vein
i.e. **distal spleno-renal shunt**
+ ligation of left gastric vein
+ right gastric vein



ADVANTAGE

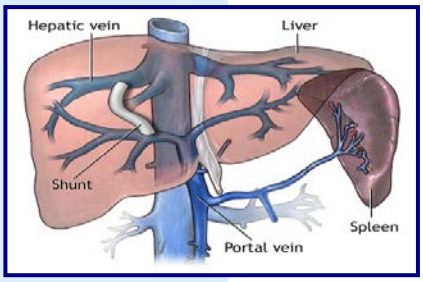
The incidence of risk of encephalopathy is low & the liver functions remain normal

DISADVANTAGES

After few years it turn into total shunt due to the presence of connections between the splenic vein & the pancreatic vein i.e. **pancreatic siphon**.
So to do proper selective shunt, the whole splenic vein should be disconnected from pancreas, which is called **modified Warren shunt**

N.B.: TIPSS (**T**rans-jugular **I**ntra-hepatic **P**orto-**s**ystemic **S**hunt)

- **This is done** using **self expanding stent** which introduced through jugular vein to reach the hepatic veins then manipulated to enter the branch of the portal vein.
- **It is indicated** with severe liver cirrhosis before liver transplantation

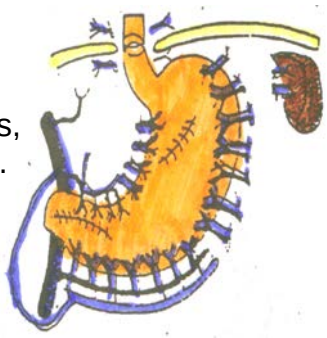


II- PORTO-AZYGOS DISCONNECTION

A. Hassab- Khairy operation

TECHNIQUE

- **Splenectomy +** ligation of Rt. & Lt. gastric vessels, short gastric vessels & Lt. gastro-epiploic vessels.
  + ligation of all vessels at lower 5 -10 cm of the oesophagus.
- **Leaving only** the Rt. gastroepiploic vessels



ADVANTAGES

- Stop of hematemesis with no risk of encephalopathy.

B. Esophageal transection

- Interruption of submucosal venous plexus which is still intact after "Hassab Khairy" operation.

III- LIVER TRANSPLANTATION

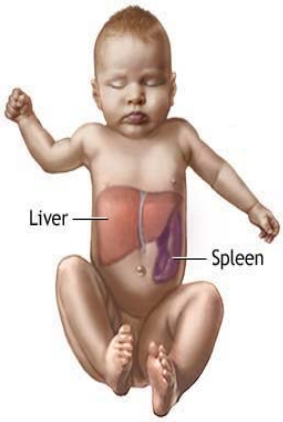
Indicated with end stage liver disease.

The Spleen

THE SPLEEN

EMBRYOLOGY

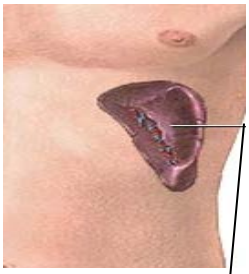
- The spleen begins in the 5th week intra-uterine when small collections of mesenchymal cells fuse to form the spleen.
- **The mesenchymal cells that fails** to fuse become accessory spleens.
  - Sites of accessory spleens :
    ① Hilum of the spleen (50 %).
    ② Splenic vessels & tail of the pancreas (30 %).
    ③ Splenic ligaments & mesocolon.
  - If accessory spleens are left after splenectomy : hyperplasia & recurrence of the disorder for which the spleen was removed.



SURGICAL ANATOMY

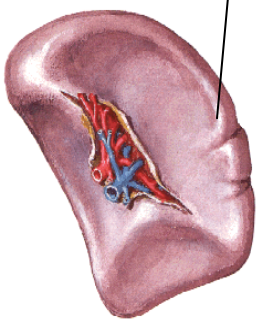
POSITION

Lt. hypochondrium, its long axis is in the line of **10th** rib.



SHAPE

It has
- 2 ends : ① Medial end.
  ② Lateral end.
- 2 borders : ① Superior border with (**notch**)
  ② Inferior border
- 2 surfaces : ① Diaphragmatic (convex).
  ② Visceral (carries impressions for stomach, renal & pancreatic).



SIZE

1 x 3 & **5 inches**

WEIGHT

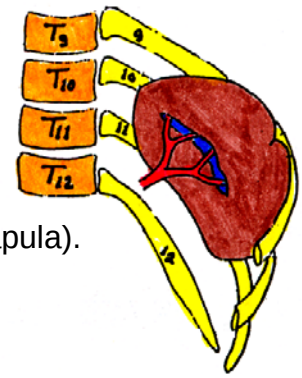
(**80 - 300 grams**)

SURFACE ANATOMY

Draw **2** transverse lines with **9**, **11** ribs

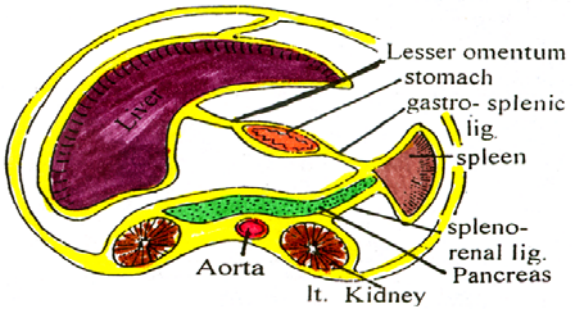
& draw **2** vertical lines mid-axillary line

& scapular line (from inferior angle of scapula).



PERITONEAL COVERINGS

- The spleen is **completely covered with peritoneum** .
- **The related ligament**



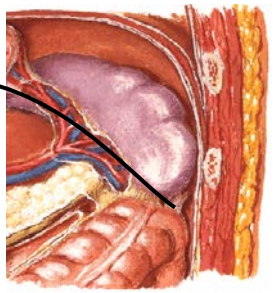
1- Lieno-renal ligament

- **Between** spleen & Lt. kidney.
- **Contains** splenic vessels & tail of pancreas.

2- Gastro-splenic ligament

- **Between** spleen & & stomach.
- **Contains** short gastric vessels

3- Phrenico-colic ligament

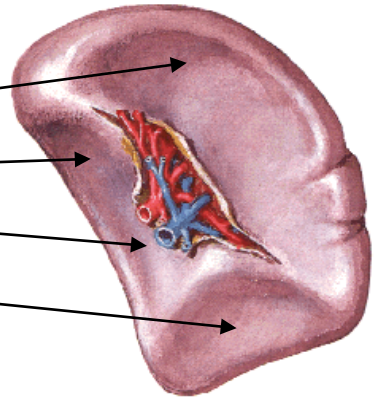


- **Between** diaphragm & splenic flexure of colon,
- **It is the main support** of the spleen, so it directs to Rt. side when it is enlarged.

RELATIONS

A- Diaphragmatic surface Related to the diaphragm.

B- Visceral surface **4** impressions & hilum



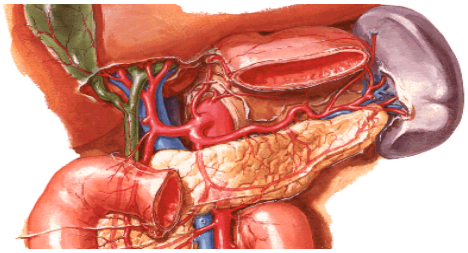
I- 4 Visceral surfaces

1- **Gastric** impression.
2- **Renal** impression.
3- **Pancreatic** impression.
4- **Colic** impression.

II- Hilum

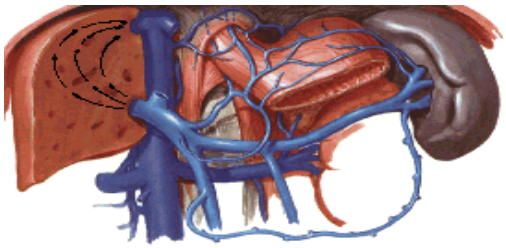
It is a depression contains splenic, vessels & tail of pancreas.

ARTERIAL SUPPLY Splenic artery



- Arises from celiac trunk. It passes along the upper border of pancreas.
- At hilum it divided into segmental branches.
- So partial splenectomy may be done.

VENOUS SUPPLY Splenic vein



- It receives inferior mesenteric vein at neck of pancreas then unites with superior mesenteric vein to form portal vein.

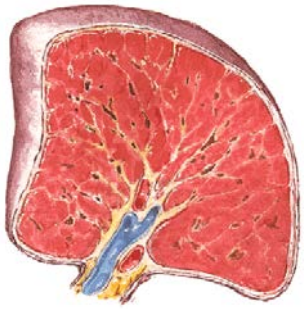
SURGICAL IMPORTANCE

(1) Segmental resection of spleen is possible as segmental blood supply

(2) Huge spleen can grow towards the left iliac fossa because of phrenico-colic ligament

PHYSIOLOGY OF SPLEEN

FUNCTIONS OF SPLEEN



1- Fetal erythropoiesis

During the **5th** to **8th** month of fetal life the spleen shares in the production of both red & white cells

2- Sequestration of old blood cells

Filtration of old & abnormal R.B.Cs.

3- Defensive functions

① Reticuloendothelial macrophages of the spleen are capable of destroying foreign substances by the process of phagocytosis

② The spleen plays an important role in proliferation of T-lymphocytes

③ The spleen helps in the production of various antibodies as IgM

CONGENITAL ANOMALIES

1- Agenesis : Absence of the spleen (**asplenia**) very rare

2- Accessory spleens : **See embryology**

3- Polysplenia : The normal spleen is divided into 2 or more parts

4- Wondering spleen : (**Splenic ectopia**)

This anomaly is caused by laxity of its ligaments. Torsion of the pedicle is the most frequent complication.

HYPERSPLENISM

- It is a case of splenomegaly

+ pancytopenia (anemia, leucopenia & thrombocytopenia) due to over activity of spleen

+ Active bone marrow

1RY HYPERSPLENISM

- This is the **idiopathic** type of the disease.
- The aetiology is **unknown**
- **Splenectomy** helps haematologic improvement in all patients

2RY HYPERSPLENISM

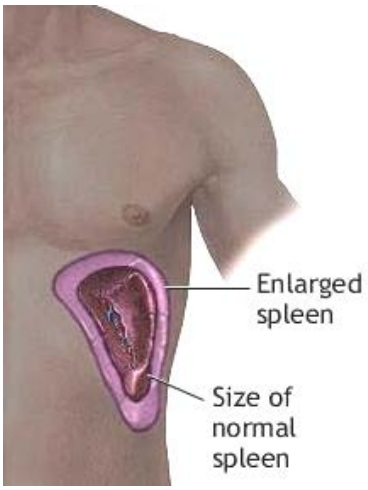
- It is a **congestion** of the vascular spaces of the spleen.
- This is due to **portal hypertension**
- **Splenectomy** is required for severe cases

  This may be combined with shunt surgery or porto-azygos disconnection to decompress the oesophageal varices.

SPLENOMEGALY

The normal spleen cannot be palpated clinically **unless it is enlarged 3 times** its normal size

CAUSES OF SPLENOMEGALLY



1. Congenital

e.g. cyst of spleen.

2. Infectious diseases

① **Bacterial :** T.B., $,Typhoid & para-typhoid.
② **Viral :** I.M.N.
③ **Parasitic :** Schistosomiasis & hydatid cyst.
④ **Protozoal :** Malaria & kala azar .

3. Blood diseases

① Hemolytic anemia
② Leukemia.
③ Thrombocytopenia.
④ Polycythemia rubra vera.

4. Metabolic diseases

① Gaucher's disease.
② Prophyria.
③ Rickets.
④ Amyloidosis.

5. Tumors

① Hemangioma.
② Lymphangioma.
③ Malignant Lymphoma.
④ Fibrosarcoma.

6. Circulatory diseases

e.g. Portal hypertension

7. Collagen diseases

e.g. Felty's syndrome & Still's disease.

CAUSES OF HUGE SPLEEN

1- Bilharziasis.
2- Kala azar.
3- Chronic malaria.
4- Chronic myeloid leukemia
5- Thalassaemia major.
6- Polycythemia rubra vera..

CAUSES OF TENDER SPLEEN

1- **T**yphoid.
2- **I**nfective endoconditis
3- **B**rucellosis.
4- **S**epticemia.

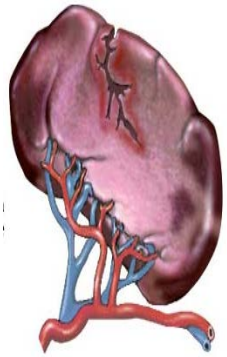
RUPTURE SPLEEN

PREDISPOSING FACTORS

A- Splenic enlargement which makes it more liable to trauma.

B- Diseases of the spleen e.g. Malaria which makes it soft.

EXCITING CAUSE "Trauma" which may be ⇩



A- Closed trauma

① **Direct trauma :**
Blunt trauma e.g. car accident.

② **Indirect trauma :**
Fracture ribs.

③ **Spontaneous rupture :**
Rare with pathological **spleen**.

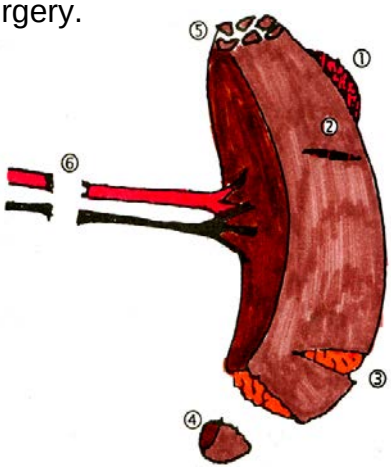
B- Opened trauma

① **Gunshot** wounds.

② **Punctured** due to stabbing.

③ **Operative trauma** e.g. during gastric surgery.

PATHOLOGY



Types of rupture spleen may be ⇩

1- **Subcapsular** hematoma.

2- **Superficial** tear or tears.

3- **Deep** tear or tears.

4- **Avulsion** of a pole of the spleen.

5- Complete **pulping** of the spleen.

6- **Injury** of vascular pedicle

COMPLICATIONS

① **Hemorrhage** which may be internal or external.

② **Splenic cyst** may follow a peri-**splenic** hematoma.

③ **Associated** other abdominal or thoracic injuries.

CLINICAL PICTURE

A- History of trauma

To upper abdomen (Lt. hypochondrium) or lower chest followed by an abdominal pain ± shock.

B- Examination

There are **3 clinical types** of rupture spleen ⇩
(**Fatal**, **classical** & **delayed** types)

1- Fatal type

Sever hemorrhage with sudden death due to tearing of splenic vessels or complete avulsion of splenic pedicle.

2- Classical type It passes in **3 stages** ⇩

1. STAGE OF SHOCK

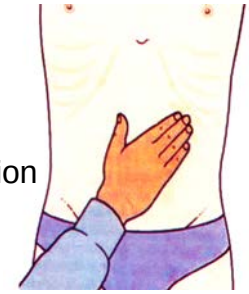
i.e. Hypovolemic shock

① Tachycardia & tachypnea (air hunger)
② Hypotension & hypothermia.
③ Pale cold skin & oliguria.

2. STAGE OF RECOVERY FROM SHOCK

i.e. Lucid interval

due to temporary arrest of bleeding from hypotension.



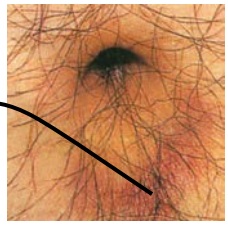
3. STAGE OF INTERNAL HEMORRHAGE

i.e. Signs of rupture spleen

due to rise again of the blood pressure after resuscitation

A- Tenderness & rigidity over Lt. hypochondrium

B- Mass in Lt hypochondrium i.e. perisplenic hematoma



C- Cullen's sign :

Brownish or bluish discoloration around the umbilicus

D- Balance's sign :

Shifting dullness on Rt. side from the free blood in the peritoneum & fixed dullness on the Lt. side due to blood clots & retro-peritoneal hematoma.

E- Kehr's sign : Referred pain to Lt. shoulder from diaphragmatic irritation especially with **trendlenburg position**.

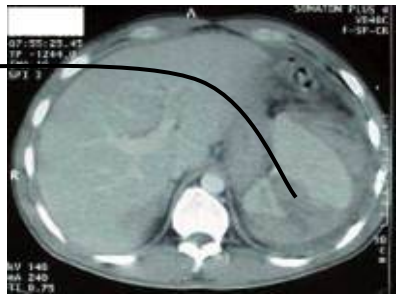
3- Delayed type

- The initial shock is followed by a long lucid interval up to **15** days, after which the patient presents with features of internal hemorrhage.
- **This delay is due to** ⇩
  - ① Formation of **subcapsular hematoma** which may be ruptured later.
  - ② The **greater omentum** seals the splenic tear then retracts later on.
  - ③ **Blood clots** block the tear then it is digested by enzymes from the injured pancreas.

INVESTIGATIONS

1. Sonar & CT scan (**abdomen**)

- Show pathological type of rupture spleen.

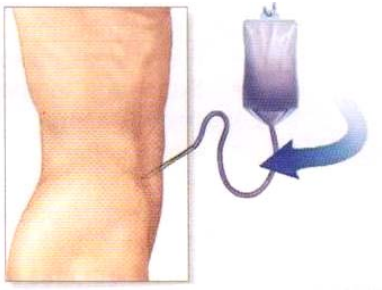


2. Plain X-ray (**upper abdomen**) shows ⇩

① Obliteration of psoas muscle shadow.
② Obliteration of splenic outlines.
③ Indentation of gastric air bubble.
④ Elevation of Lt. side (copula) of diaphragm.
⑤ Fracture one or more of lower ribs.
⑥ **±** Evidence of cause e.g. bullet.

3. Diagnostic peritoneal lavage "D.P.L."

- Done by inserting an intra-peritoneal catheter at umbilicus under local anesthesia & infusing **500 cc** saline in the peritoneal cavity. if saline returns bloody = intra-peritoneal bleeding.



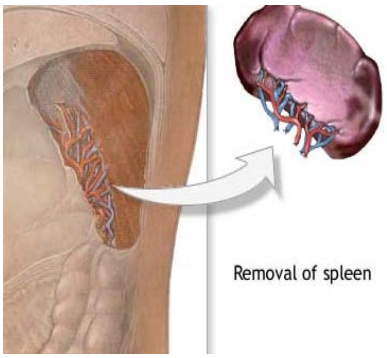
TREATMENT

A. Anti-shock measures

- Remember **ABCDE**
- Blood transfusion, warmth, morphia ……etc.

B. Immediate laparotomy & splenectomy

or splenic preservation especially in children as it plays an important role in immune mechanism especially against pneumococci.



THE PRESERVATION THROUGH

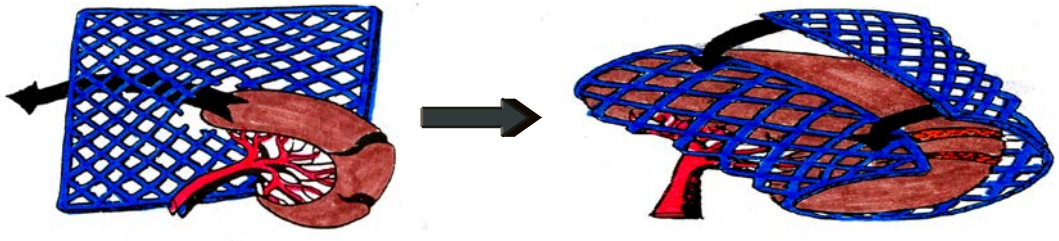
① **Sutures** of small lacerations or tears i.e. splenorrhaphy,

② **Partial splenectomy** if avulsed one pole.

③ **Auto-transplantation** of splenic fragments Which is wrapped by omentum. This may help if pulped.

④ **Therapeutic embolization** through the splenic artery by gel foam

⑤ **Splenic mesh wrap :** Placing the injured spleen in the center of the mesh & sewing both ends of the mesh together to tamponade the bleeding.



N.B.: Pneumococcal anti-toxin (Pneumovax)
Should be given in young child until 18 years of age after splenectomy

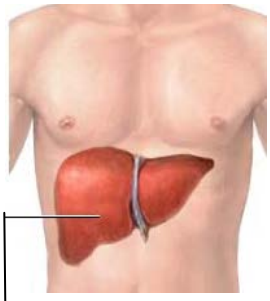
N.B

COMPLICATIONS OF SPLENECTOMY

- **Post-splenectomy fever :** due to collapse of basal lobe of left lung, chest infection, D.V.T. or subphrenic abscess.
- **O.P.S.I : O**verwhelming **P**ost **S**plenectomy **I**nfection with children.
- **Acute gastric dilatation** & **paralytic ileus**.
- **Burst Abdomen :** if the pancreas is injured → liberation of proteolytic enzyme.

The Liver

I- ANATOMY OF THE LIVER



➢ The largest organ in the body (**1200 - 1800** gram).

➢ It occupies the whole right hypochondrium & parts of epigastrium and left hypochondrium.

LOBES OF THE LIVER

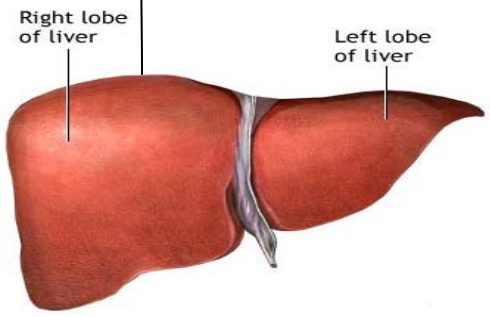
A. Anatomical lobes

● The liver is divided into small Lt. lobe & very large Rt. lobe.

● The line of division corresponds to

① **Falciform ligament :** on superior & anterior surfaces.

② **Round ligament :** on inferior surface.



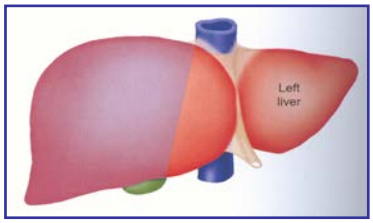
B. Surgical lobes

● The liver is divided into **2** equal **surgical lobes** by inter-lober fissure which extends from ⇘

① **Anteriorly :** the gall bladder fossa:

② **Posteriorly :** (I.V.C).

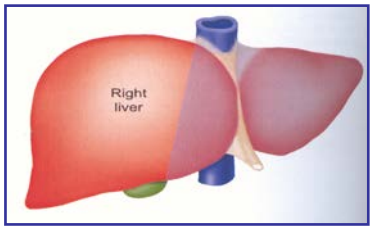
● Then divided into **4** sectors & **8** segments



A- Left lobe divided by falciform ligament into ⇘

① **Lateral** sector.

② **Medial** sector.

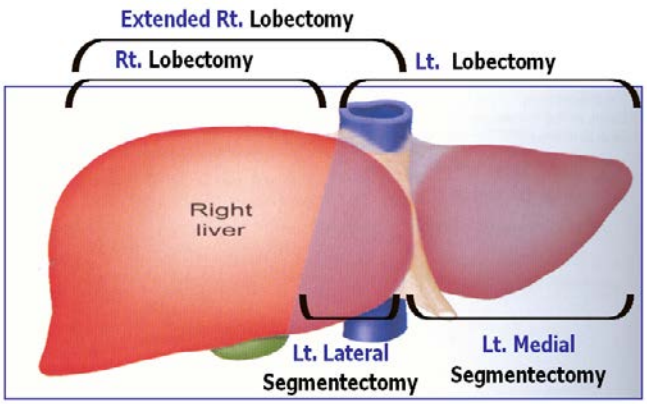


B- Right lobe divided by coronal plane into ⇘

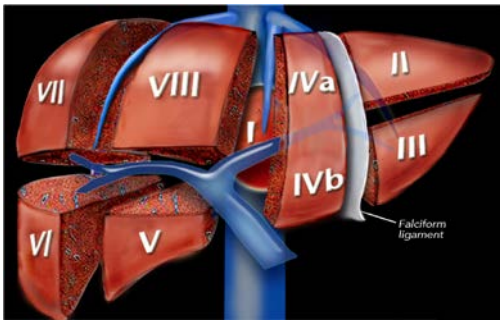
① **Anterior** sector.

② **Posterior** sector.

➢ So, hepatic resection may be done as follows :



Surgical importance



① Rt. lobectomy. ② Extended Rt. lobectomy. ③ Lt. lobectomy.

④ Lt. lateral segmentectomy ⑤ Lt. medial segmentectomy.

RELATIONS OF THE LIVER

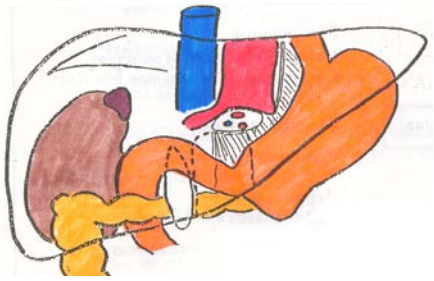
A. Rt. lateral surface

Related to the diaphragm.

B. Antero-superior surface

Related to the diaphragm & abdominal wall.

C. Posterior surface (Rt. to Lt.)



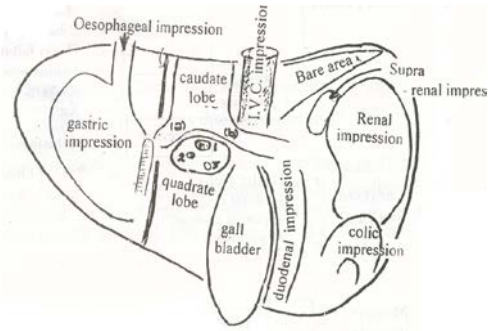
① **Bare area** of the liver.

② **Groove** for IVC.

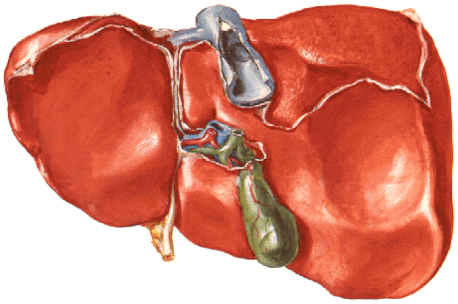
③ **Caudate lobe** (Segment **I**)
with **2 processes :**
a. **Papillary** process.
b. **Caudate** process.

④ **Fissure** for ligamentum venosum.

⑤ **Oesophageal** impression.



D. Inferior surface (Lt. to Rt.)



① **Gastric** impression.

② **Fissure** for ligamentum teres.

③ **Quadrate** lobe.

④ **Gall bladder** fossa.

⑤ **Duodenal** impression.

⑥ **Colic** impression.

⑦ **Renal** impression.

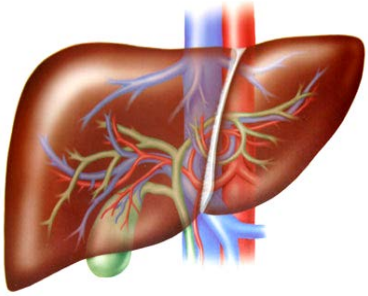
⑧ **Supra-renal** impression.

⑨ **Porta-hepatis** .

PERITONEAL COVERINGS

- The liver is completely covered with peritoneum **except** bare areas at upper part of post-surface, G.B fossa, groove for IVC, porta-hepatis.
- **PERITONEAL LIGAMENT CONNECTIONS ARE**
  ① ***Falciform ligament***.
  ② ***Ligamentum teres*** (obliterated umbilical vein).
  ③ ***Ligamentum venosum*** (obliterated ducts venosus).

BLOOD SUPPLY **1.5 L/min**.



- **Portal vein :** carrying venous blood from G.I.T & supplies **2/3** of total blood supply.
- **Hepatic artery :** supplies of **1/3** total blood supply.

VENOUS DRAINAGE

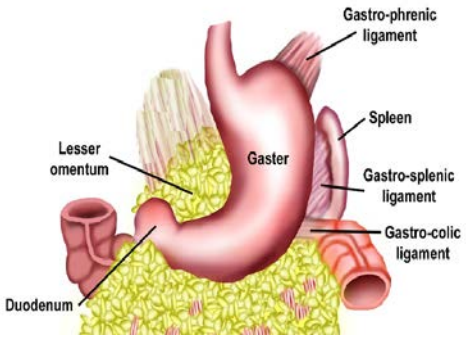
- **3 hepatic veins into I.V.C**.

LYMPHATIC DRAINAGE

- Liver drains into L.Ns at porta-hepatic & then into coeliac L.Ns.

II- ANATOMY OF THE LESSER OMENTUM

STRUCTURES & RELATIONS



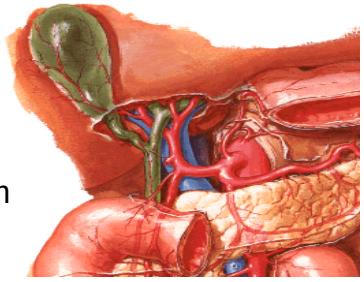
- THE LESSER OMENTUM is formed of a fold of peritoneum that has a **free** right border,

- **The free border** of the lesser omentum contains 3 important tubular structures & called Hepatico-duodenal ligament

1- The common bile duct
in an anterior plane and to the right.

2- The hepatic artery
in an anterior plane but to the left.

3- The portal vein
posterior to the other 2 structures. .

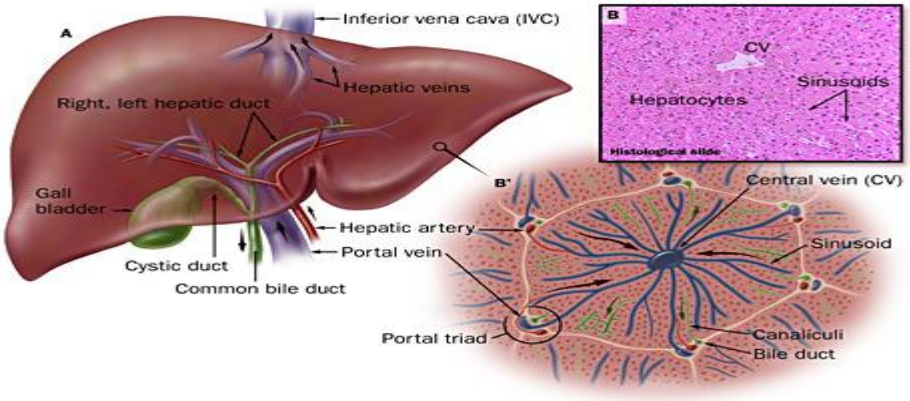


- THE LESSER SAC is bounded :
  - **Anteriorly** by free border of lesser omentum
  - **Superiorly** by the caudate lobe
  - **Posteriorly** by the IVC
  - **Inferiorly** by the 1st. part of the duodenum.

SURGICAL IMPORTANCE

PRINGLE'S MANEUVER which is application of a vascular clamp to free border of lesser omentum or holding it between 2 fingers, to occlude the hepatic artery & portal vein for **20** minutes during management of liver trauma or cholecystectomy

PHYSIOLOGY OF LIVER



1- Formation & secretion of bile
Bile salts, cholesterol, and phospholipids are formed in the hepatocytes

2- The liver is the site of metabolism
For carbohydrate, protein, and fat metabolism.

3- Metabolism role with drugs & hormones
e.g. Oestrogen.

4- Removal of ammonia from the portal circulation.

5- Storage of glycogen, vitamin B_{12}, vitamin A, iron, & copper.

HEPATOMEGALY

1. Congenital
e.g. Polycystic liver.

2. Infectious diseases
① **Bacterial :** Typhoid
② **Viral :** I.M.N.
③ **Parasitic :** Schistosomiasis & hydatid cyst.

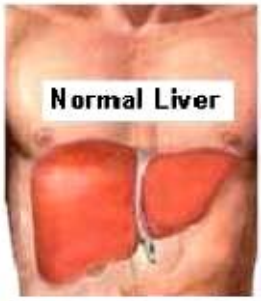
3. Blood diseases
e.g. Megaloblastic anemia

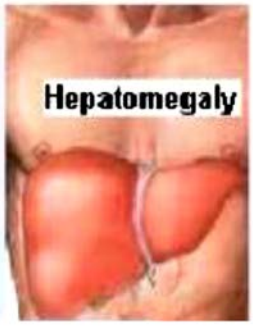
4. Metabolic diseases
e.g. Amyloidosis

5. Tumors
① Hemangioma.
②Malignant Lymphoma.

6. Circulatory diseases
e.g. Portal hypertension

7. Collagen diseases
e.g. Felty's syndrome & Still's disease.



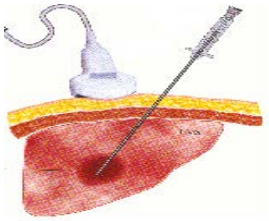


STUDIES OF THE LIVER

(A) Liver function tests

Biochemical tests provide simple way of monitoring liver function

(B) Imaging of the liver



1. Ultrasonography is the first choice imaging investigation .

2. CT scan& MRI visualize the parenchyma and adjacent tissues with great clarity.

3. Arteriography It is still useful in the diagnosis of liver tumors

4. Isotope scanning It has been nearly replaced by CT scan.

(C) Liver biopsy

Indications

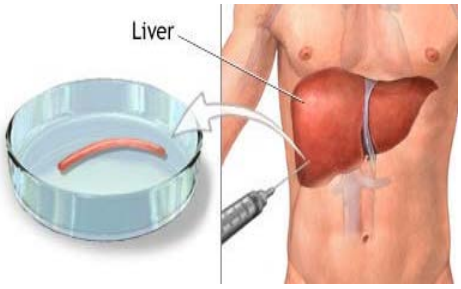
1. Assessment of chronic liver disease
as cirrhosis and chronic active hepatitis.

2. Investigation of **acute hepatic dysfunction**.

3. Target biopsy of focal lesions
that are suspected to be primary or secondary

4. Investigations of systemic diseases
as amyloidosis and sarcoidosis

5. Liver transplant assessment.
A preoperative histological diagnosis is essential



I- LIVER TRAUMA

PREDISPOSING FACTORS

A- Liver enlargement which makes it more liable to trauma.

B- Diseases of the liver e.g. congested **liver**.

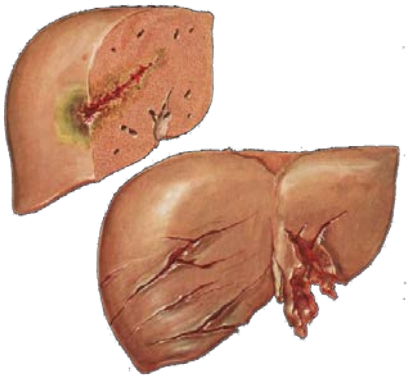
EXCITING CAUSE "Trauma" which may be ⇩

A- Closed trauma

① **Direct trauma :**
Blunt trauma e.g. car accident.

② **Indirect trauma :**
Fracture ribs.

③ **Spontaneous rupture :**
Rare with pathological **liver**.

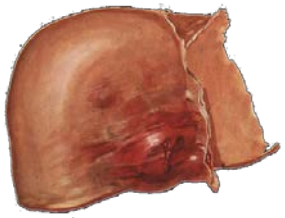


B- Opened trauma

① **Gunshot** wounds.

② **Punctured** due to stabbing.

③ **Iatrogenic** e.g. percutaneous liver biopsy.



PATHOLOGY

Types of rupture liver may be ⇩

1- **Subcapsular** hematoma.

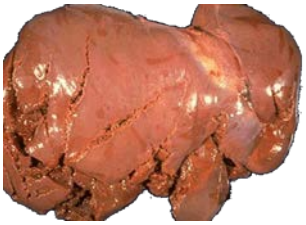
2- **Superficial** tear or tears.

3- **Deep** tear or tears.

4- **Avulsion** of a pole of the liver.

5- Complete **pulping** of the liver.

6- **Injury** of vascular pedicle



7- **Avulsion of extra-hepatic biliary passage**

COMPLICATIONS

① **Hemorrhage** which may be internal or external.

② **Liver cyst** may follow a **liver** hematoma.

③ **Associated** other abdominal or thoracic injuries.

④ Torn bile duct leading to "**Hemobilia**"

CLINICAL PICTURE

A- History of trauma

To upper abdomen (Rt. hypochondrium) or lower chest followed by an abdominal pain ± shock.

B- Examination

May reveals signs of internal Hge & shock
+ signs of external trauma as bruises

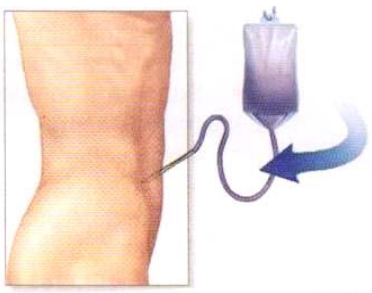
INVESTIGATIONS

1. Sonar & CT scan (**abdomen**)

- Show pathological type of rupture **liver**

2. Diagnostic peritoneal lavage "D.P.L."

- Done by inserting an intra-peritoneal catheter at umbilicus under local anesthesia & infusing **500 cc** saline in the peritoneal cavity. if saline returns bloody = intra-peritoneal bleeding.



TREATMENT

A. Anti-shock measures

- Remember **ABCDE**
- Blood transfusion, warmth, morphia ……etc.

B. Immediate laparotomy "The priority is to arrest bleeding"

SO We control the liver hemorrhage by " **Pringle's maneuver** " which is application of a vascular clamp to the free border of lesser omentum or holding it between 2 fingers, to occlude the hepatic artery & portal vein for **20** minutes.

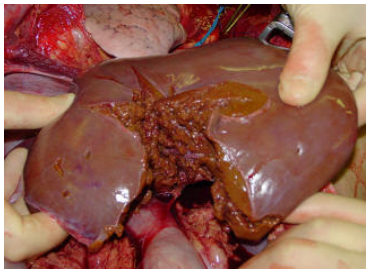
Then THERE ARE 3 POSSIBILITIES

⇨ If the tear is accessible,

- It is repaired by deeply placed mattress sutures supported by a pad of peritoneum.

⇨ If the tear is inaccessible,

- The incision should be extended into the chest along the **8th** inter-costal space.
- The aim is to arrest bleeding, to remove the necrotic devitalized tissue, to preserve as much viable tissue as possible & to do external drainage.



⇨ If there is extensive hematoma or ruptured liver tissue

- We must do "**hepatic resection**" then drainage of peritoneal cavity to avoid peritonitis then prophylactic A.B are prescribed.

PROGNOSIS

The mortality Rate = **15 - 20 %.**

II- LIVER INFECTIONS

1- Amoebic hepatitis & abscess.

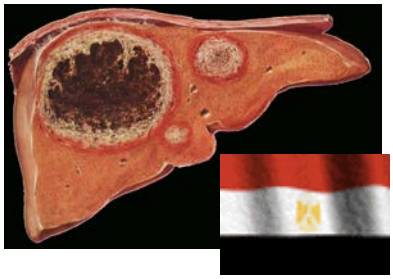
2- Hydatid disease of the liver

3- Pyogenic liver abscess.

4- Hepatic schistosomiasis.

1- Amoebic hepatitis & abscess

INCIDENCE



- **Common** in **endemic** area " **Egypt** "
- **Age :** Common with young or adult
- **Sex :** Male **>** female.

AETIOLOGY

Organism **Entamoeba histolytica**

Source of infection

Amoebic liver abscess may follow any stage of colonic amoebiasis

Route of infection

Ingestion of cysts within improper washed vegetables, then in the intestine, the cyst changed to be "**Trophozoites**" that induce colitis.

PATHOLOGY

Pathogenesis

- The trophozoites invade the portal blood from the colon.
- The **Rt**. lobe of the liver is **more affected than** the **Lt**. lobe as portal blood from **Rt**. side of colon (site of amoebic colitis) drains to the Rt. lobe of the liver.
- The parasite starts the process of liquefactive necrosis which result in the formation of an abscess.

Pathology of amoebic cyst

NUMBER Single (**70 %**) & multiple (**30 %**)

SITE In the postero-superior segment of the Rt. lobe of liver as the segmental branch of Rt. portal vein (to this segment) in direct continuity with Rt. portal vein.

SHAPE **The wall** is shaggy & containing entamoeba as they live in healthy not dead tissues.

The content is "**anchovy sauce**" or "**chocolate pus**" which is brownish necrotic material of destructed liver substance, leucocytes & RBCs, but never entamoeba.

CLINICAL PICTURE

Type of patient

20 - 40 years & Male > female.

General examination

- Low grade fever (**38°- 38.5°**)
- Patient usually pale & looks toxic (**earthy look**)
- Anorexia, nausia & weight loss.

Local examination

- Upper Rt. abdominal pain.
- Tender liver.
- **The patient may have chest signs :**
  - ① Impaired movement at Rt. side of chest.
  - ② Pleural effusion on Rt. side + lung crepitations

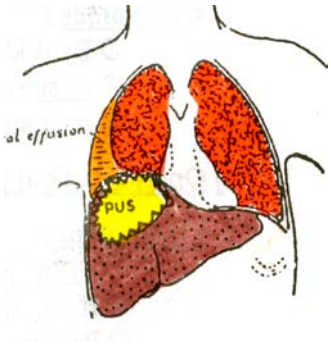
COMPLICATIONS

1- 2ry Bacterial infection ➔ Pyogenic liver abscess.

2- Calcification

3- Rupture

[a] Upwards :
- ① Subphrenic abscess.
- ② Pleura (empyema).
- ③ Lung (amoebic lung abscess)
- ④ Pericardium (pericarditis)



[b] Downwards :
- ① Stomach & duodenum.
- ② Colon.
- ③ Peritoneum (peritonitis)

[c] Outwards : Rarely below costal margin.

4- Destruction of liver cells ➔ Liver cell failure

D.D Other causes of tender liver as
- ① **Amoebic** liver abscess.
- ② Malignant liver.
- ③ Pyogenic liver abscess.
- ④ Portal pyemia.
- ⑤ Congestive liver.
- ⑥ Viral hepatitis.

INVESTIGATIONS

A- laboratory investigations

1- Stool analysis
for entamoeba histolytica.

2- Serological test
e.g. complement fixation test (C.F.T.)

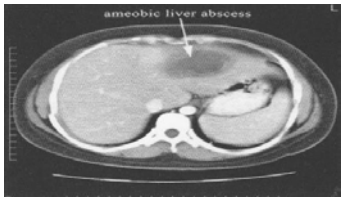
3- Therapeutic test
good response to metronidazole can help in diagnosis.

B- Radiological investigations

1- Plain X-ray (**chest**)

Shows ① Elevated fixed Rt. copula of diaphragm.
② Obliterated costo-phrenic angle if empyema
③ Basal lung collapse if present.

2- Imaging by U/S & CT scan :
Which can detect number, site, shape & size of the cysts.



3- MRI : The most accurate.

TREATMENT

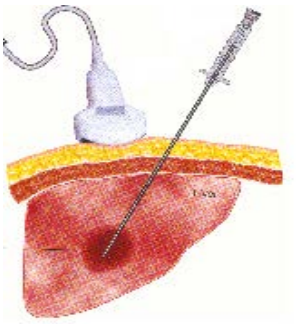
1- Medical treatment

AMOEBICIDAL DRUGS which are highly successful as

Metronidazole (**flagyl**) **750** mg t.d.s, for **7 -10** days

or Tenidazole (**fasigyn**) **2** gm orally after lunch for **3** days.

2- Aspiration [**U/S guided percutaneous aspiration**]



INDICATIONS

① Lack of response to **3** days flagyl treatment.
② Very large abscess or **2**^{ry} infection.
③ Very toxic patient.

TECHNIQUE

① By wide bore long needle under local anesthesia.
② At site of local evidence of underlying abscess.
③ Aspiration can be repeated.

3- Open surgical drainage

INDICATIONS

① Difficult aspiration because of **multilocular** cyst.
② If associated with **peritonitis**
③ Presence of **thick** pus.

TECHNIQUE

Through the bed of **12**th rib posteriorly.
As most of amoebic abscesses present at postero-superior segment of Rt. Lobe of Liver.

.....

2- Hydatid disease of the liver

INCIDENCE

It is commonly seen in **Iraqi**, **Yemen** & **Libya**.

AETIOLOGY

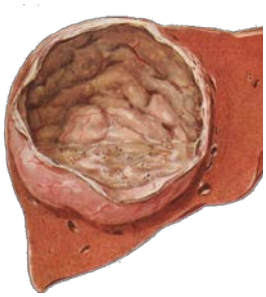
Organism Tape worm called **Echinococcus granulosus**.

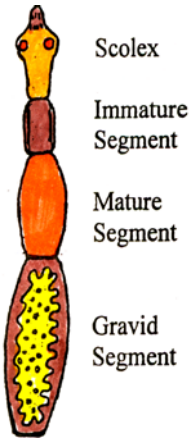
Definitive host

[**Dogs**] where adult worms live.

Intermediate host

[**Cattle** & **man**]

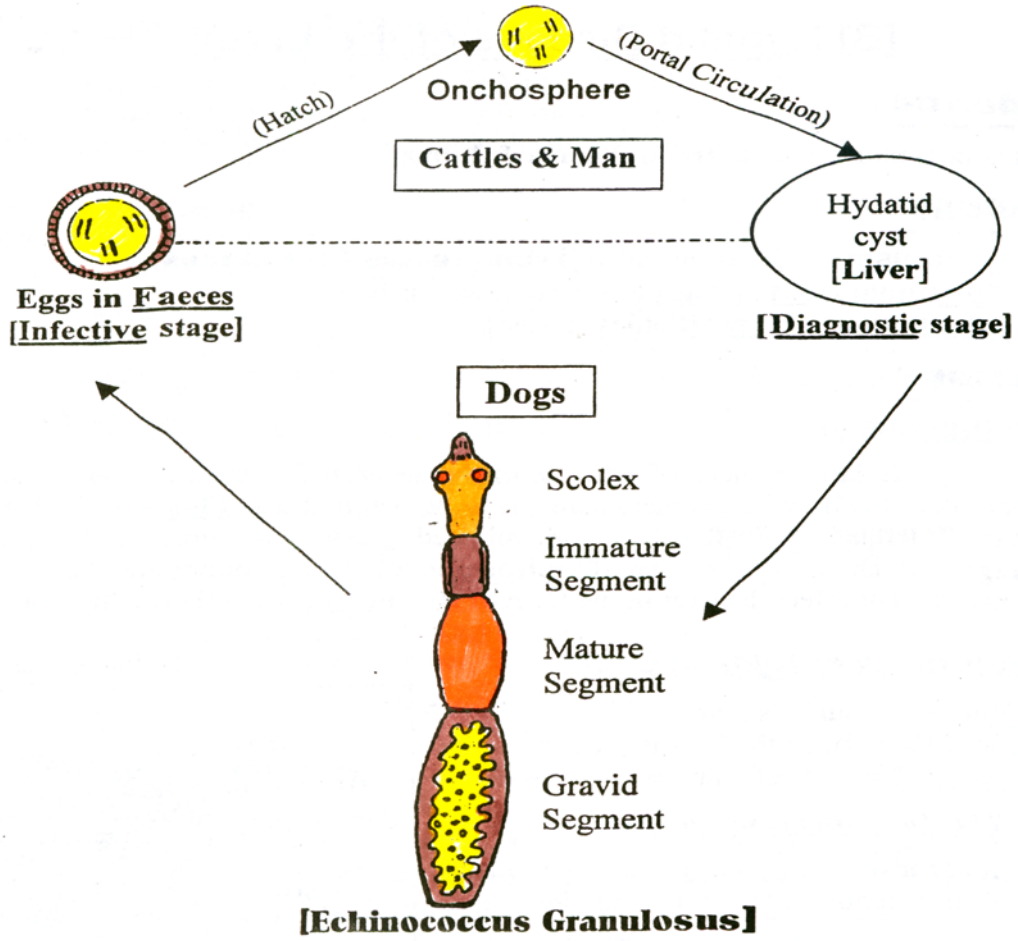




PATHOLOGY

Pathogenesis

The last segment of adult worm is separated to pass in stool of **dogs** which contaminate grass & vegetables. Then the **cattle** & **man** eating contaminated grass with eggs which hatch to give [**Onchosphere**] which penetrate portal circulation & affect the liver in the form of **hydatid cyst**

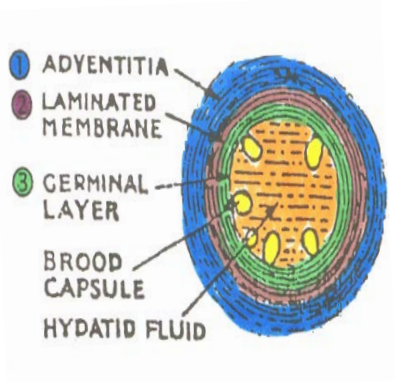


Pathology of hydatid cyst

NUMBER Usually single

SITE Rt. Lobe of the liver

SHAPE Consists of **3 layers**



1- THE ADVENTITIA (PSEUDO-CYST)

It is consists of fibrous tissue which result from reaction of liver with parasite.

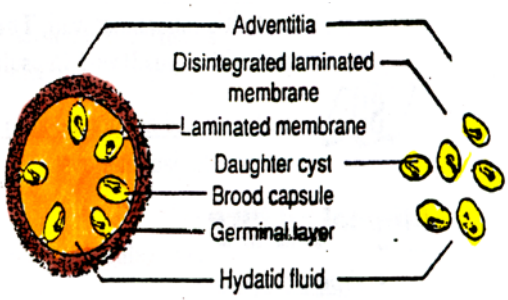
2- THE LAMINATED MEMBRANE (ECTO-CYST)

It is formed of parasite itself.

3- THE GERMINAL LAYER (ENDO-CYST)

It is the only living part of hydatid cyst, it secretes the hydatid fluid. **Brood capsules** are developed from this layer. then **Scolices** (head of future worms) are developed within this Brood capsules.

SO **if the laminated membrane** is damaged, **the Brood capsules** becomes free & grow internally into daughter cyst.



CLINICAL PICTURE

Type of patient

Usually male from Iraq, Yemen & Libya.

General examination

Asymptomatic or loss of weight inspite of good appetite.

Local examination

- **Intra-abdominal mass** related to Rt. lobe of the liver which is tense, cystic, painless & dull on percussion.
- **Hydatid thrill :** Thrill is felt on percussion due to vibration of daughter cyst against each other on hydatid fluid.

COMPLICATIONS

1- 2ry Bacterial infection ➔ Pyogenic liver abscess.

2- Calcification

3- Rupture

[a] Upwards :

e.g. Chest cavity leads to empyema.

[b] Downwards :

① Peritoneum (peritonitis) or dissemination & anaphylactic reaction, which **endanger the patients life**.

② Biliary tracts ➔ Obstructive jaundice

INVESTIGATIONS

A- laboratory investigations

1- Blood picture :

Shows high eosinophilia.

2- Serological test

e.g. complement fixation test (C.F.T.) & **ELISA**

3- Casoni's test

Intra-dermal injection of **0.2 cc** hydatid fluid, followed after **24** hours by edema & redness at site of injection.

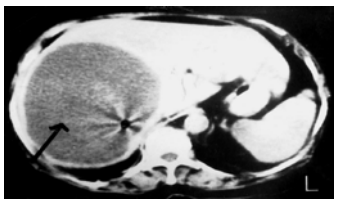
B- Radiological investigations

1- Plain X-ray (**chest**)

Shows obliterated costo-phrenic angle if empyema

2- Imaging by U/S & CT scan :

Which can detect number, site, shape & size of the cysts.



3- MRI : The most accurate.

TREATMENT

1- Surgical treatment [EXCISION] is the standard treatment.

PRE-OPERATIVE

Estimation of the number & sites of cysts is needed because of missed cyst after excision may leads to an anaphylactic reaction

PRECAUTIONS

The liver cyst (or cysts) is surrounded by **dark green towels** that are moisted with a scolicidal agent as **betadine** or **hypertonic saline**.

TECHNIQUE

① The cyst is aspirated 1st & hypertonic saline is injected
② The overlying liver substance & the adventitia are excised
③ The cavity is filled with a pad of peritoneum.

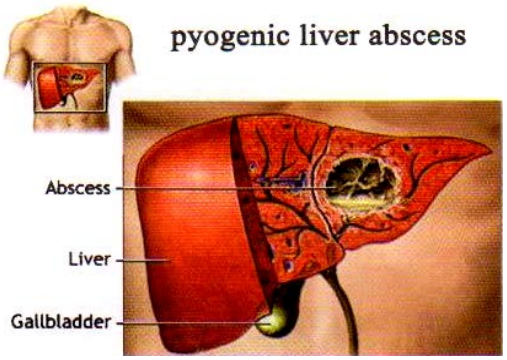
Other surgical options

- CYSTECTOMY suitable for pedunculated cyst.
- PARTIAL HEPATECTOMY suitable for localized multiple cysts

B- Medical treatment (MEBENDAZOLE) **400 - 600** mg t.d.s for **6** months.
It is not a good substitute for surgery but indicated only with, unfit patient or recurrent cases or inaccessible cysts.

3- Pyogenic liver abscess

AETIOLOGY



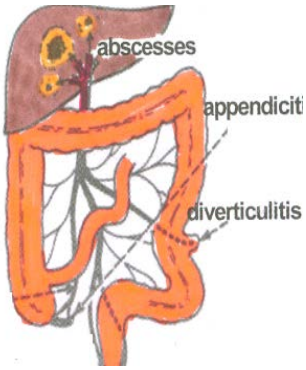
A- Predisposing factors

1- DECREASED IMMUNITY
e.g. DM, leukemia ... .etc.

2- 2RY INFECTION
e.g. - Infected **hematoma** of liver.
- Infected **amoebic** abscess.
- Infected **hydatid** abscess.

B- Source of infection

1- BILIARY TRACT (**Cholangitic abscess**)
due to biliary obstruction & infection by E-coli & gram – ve bacilli.



2- PORTAL VEIN (**Pyemic abscess**)
e.g. suppurative appendicitis, diverticulitis …etc.

3- HEPATIC ARTERY (**Hematogenous abscess**)

CLINICAL PICTURE

General Severe toxemia, fever & rigors.

Local Pain & tenderness at Rt. hypochondrium

D.D **Other causes of tender liver as** ⇘

① Amoebic liver abscess.
② Malignant liver.
③ **Pyogenic** liver abscess.
④ Portal pyemia.
⑤ Congestive liver.
⑥ Viral hepatitis

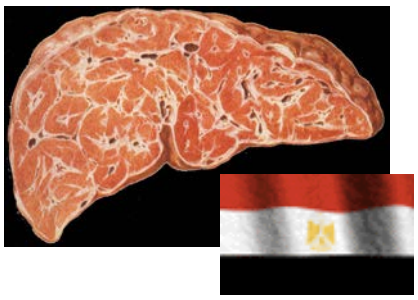
INVESTIGATIONS **U/S** & **CT scan** are diagnostic.

TREATMENT

① U/S guided percutaneous aspiration or open surgical drainage
② Under cover of strong antibiotics.

4- Hepatic schistosomiasis

INCIDENCE



- **Common** in **endemic** area " **Egypt** "
- **Age :** Common with young or adult
- **Sex :** Male **>** female.

AETIOLOGY

Schistosoma **mansoni** & less commonly schistosoma hematobium

PATHOGENESIS

Large number of ova are laid in terminal radicals of mesenteric vessels ➔ portal vein ➔ portal tracts ➔ induce inflammation ➔ Bilharzial granuloma ➔ Excessive **peri-portal fibrosis**.

PATHOLOGY **3 types**

1. Fine types

- It follows deposition of **few** number of ova in **small** portal Tracts.
- **Macroscopically :** The surface of the liver shows **fine** nodules.
- **Microscopically : Fine** fibrous tissue surrounds **small** portal tracts with **few** number of ova.

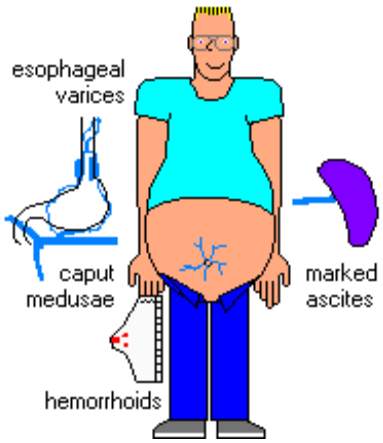
2. Coarse type

- It follow deposition of **large** number of ova in **large** portal tracts.
- **Macroscopically :** The surface of the liver shows **coarse** nodules.
- **Microscopically : Coarse** fibrous tissue surrounds **large** portal tracts with **large** number of ova.

3. Mixed type The commonest.

C/P& COMPLICATIONS

Portal hypertension, oesophageal varices, splenomegaly .... etc.



STAGES

1- HEPATOMEGALY.
2- HEPATOSPLENOMEGALY.
3- SPLENOMEGALY + SHRUNKEN LIVER.
4- AS (3) + ASCITES
5- AS (4) + L.C.F

TREATMENT

A- Anti-bilharzial drugs
as **oxamniquine** or **praziquantel**.

B- Treatment of complications

III- LIVER TUMORS

A- BENIGN TUOMERS

Liver cell adenoma (LCA)

Well-circumscribed adenoma that is arising within the liver.

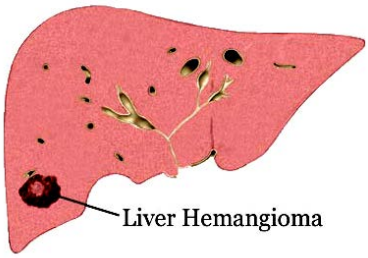
Aetiology **only in women**.
Cessation of pill intake can result in tumor regression.

Clinical features
The tumor is commonly **asymptomatic**,
and is discovered incidentally

Investigations
- **Imaging by** ultrasound, CT scan, and/or MRI.
- **Liver function tests** and **a-fetoprotein are within normal** levels.

Treatment
- **Liver resection** is indicated as to the possibility of malignancy

Haemangioma

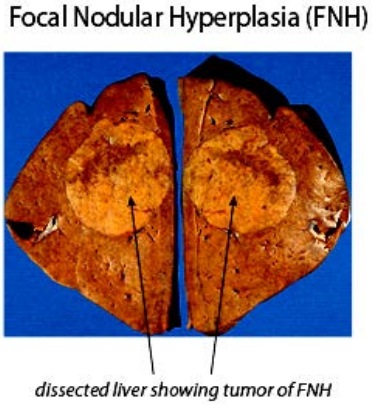


Pathology
They are of the cavernous type and may attain a large size.

Investigations
- **Imaging by** ultrasound, CT scan, and/or MRI.
- **Liver function tests**

Treatment
- **Liver resection** is indicated as to the possibility of malignancy

Focal nodular hyperplasia (FNH)



dissected liver showing tumor of FNH

Aetiology
Contraceptive pills stimulate the development of FNH.

Investigations
- **Imaging by** ultrasound, CT scan, and/or MRI.
- **Liver function tests**

Treatment
- **NO Treatment** & **Contraceptive pills should be discontinued**.

DD of a focal hepatic lesion

1- BENIGN LESIONS
2- HEPATOCELLULAR HEPATOMA
3- METASTASIS
4- CYST **as parasitc or non parasitic**

B- MALIGNANT TUOMERS

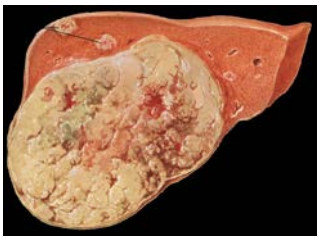
- Malignant tumors

1- Primary tumors

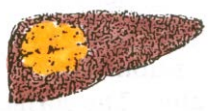
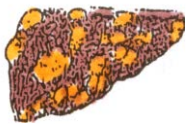
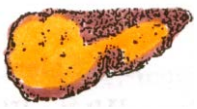
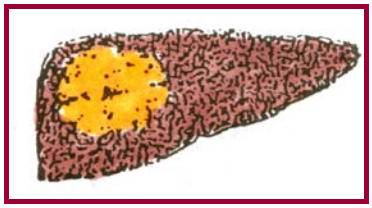
Hepatoma & **Cholangiocarcinoma**

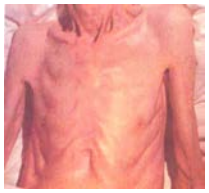
2- Secondary tumors

Metastasis.



1- Primary malignant tumors

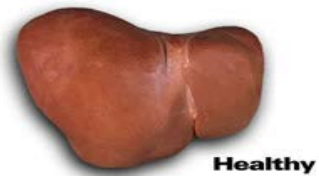
	HEPATO-CELLULAR	CHOLANGIOCARCINOMA
INCIDENCE	**80 %**	**20 %**
	Male > Female & **> 50** years.	
PREDISPOSING FACTORS	① **Infection :** e.g. Hepatitis B & C virus. ② **Liver Cirrhosis.** ③ **Aflatoxins** in food which formed by fungus called **Aspirgillus flavus** due to poor storage of grains	① **Congenital :** Cystic liver fibrosis. ② **Choledochal** cyst. ③ **Clonorchis sinesis** infestation. ④ **Sclerosing cholangitis** ⑤ **Hemochromatosis**.
PATHOLOGY **N/E**	Massive : Forming a localized mass  Nodular : i.e. multiple nodules scattered all over the liver  Diffuse : i.e. which can infiltrate the liver 	1. Solitary mass  2. Multiple nodules with golden yellow appearance and shows area of Hge and Necrosis.
M/E	**Malignant hepatocytes** with high vascularity	**Adenocarcinoma** from epithelial lining duct.
SPREAD	① **Direct :** To liver substance then to the surroundings ② **Lymphatic :** L.Ns at porta-hepatis & coeliac L.Ns. ③ **Blood :** Portal vein and I.V.C thrombosis & rarely lung. ④ **Peritoneal nodules** leads to malignant ascites.	

	HEPATO-CELLULAR	CHOLANGIOCARCINOMA
C/P **Symptoms**	① The common presentation is **deterioration** of health. ② Cachexia, **athenia** & metastasis. ③ Obstructive jaundice. 	
Signs	① Rt. hypochondrial or epigastric mass. ② Slight to moderate jaundice with low grade fever. ③ Malignant ascites in **40 %** of cases.	
D.D	**Other causes of tender liver as** ↓ ① Amoebic liver abscess. ② **Malignant** liver. ③ Pyogenic liver abscess. ④ Portal pyemia. ⑤ Congestive liver. ⑥ Viral hepatitis	
INVESTIGATION **Laboratory**	① **Liver function tests :** ↑ Alkaline phosphatase & ↑ serum bilirubin. ② **Alpha feto-protein** as a tumor marker.	
Radiological	① **CT scan**, **U/S** & **MRI** (the most diagnostic) ② **Hepatic Angiography.:** Shows tumor circulation & vascular hepatic anatomy before resection.	
Instrumental	**Biopsy :** controversial for high risk cirrhotic patient for bleeding	**ERCP :** diagnostic & therapeutic e.g. putting internal stent

TREATMENT

A- operable

Hepatic resection is the only hope for cure

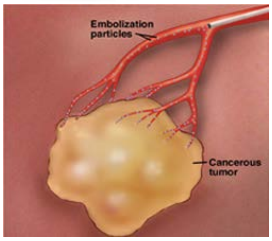


N.B.: Cirrhotic patient is contraindicated to surgery because of ↓
① Bleeding tendency.
② Poor function of reserved part of liver
③ ↓ Capacity of liver cell for regeneration.

B- Inoperable

1- Systemic chemotherapy

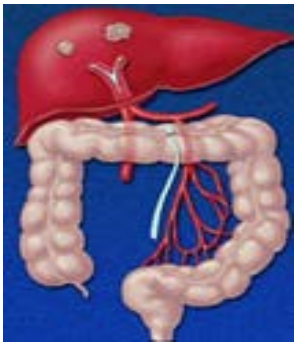
3- Trans- arterial Chemo-embolization (**TACE**)
(**double benefit**) as ischemia of tumor & chemotherapy



4- Percutaneous ethanol injection
leading to tumor necrosis.

2- Liver metastasis

INCIDENCE **20** times **> 1ry** tumor.



ROUTES

1- PORTAL CIRCULATION from G.I.T carcinomas.

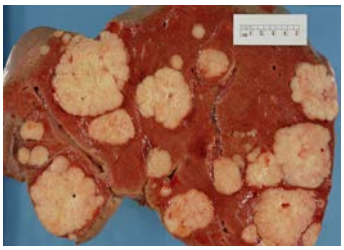
2- HEPATIC ARTERY from breast, lung, kidney….etc.

3- LYMPHATICS

4- DIRECT from gall bladder carcinoma.

PATHOLOGY

Adenocarcinoma



- They are multiple, white, umblicated nodules because of central necrosis
- They occur at same time of 1ry tumor (**Synchronous**) or months later after resection (**Metasynchronous**)

CLINICAL PICTURE

Picture of 1ry tumor +

Weight loss, fatigue, hepatomegaly, ascites & jaundice.

INVESTIGATIONS

A- laboratory investigations

- Blood picture :

Shows ↑ Serum bilirubin & alkaline phosphatase

B- Radiological investigations

- Imaging by U/S & CT scan :

More accurate

TREATMENT

A- Liver metastasis usually means inoperable

for chemotherapy or embolization via the hepatic artery

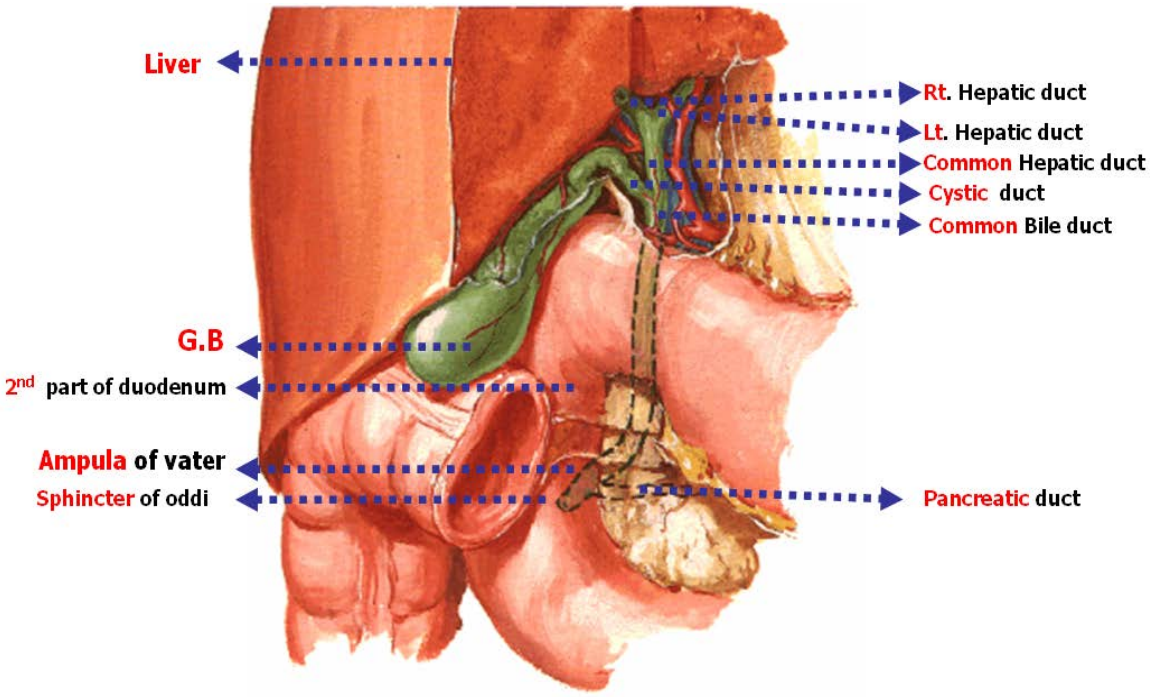
B- Liver resection as (**colorectal carcinoma**) may be curable

in few cases as solitary nodule & no extra-hepatic metastasis or multiple but confined to one lobe.

The Biliary system

THE BILIARY SYSTEM

SURGICAL ANATOMY

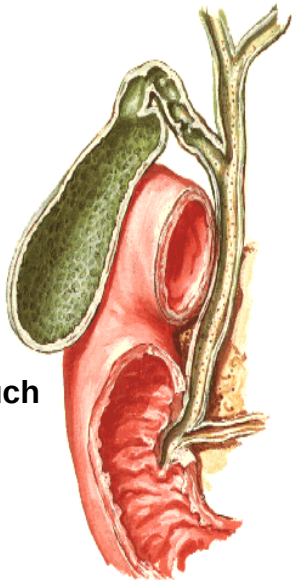


The Biliary system **= Gall bladder** + **extra-hepatic biliary tracts**

The Gall bladder

- It is a pear-shaped hollow organ lying on the visceral surface of the liver, measured about (**7.5** -**12.5** cm) long with normal capacity **50** ml but it may be distended.

MORPHOLOGY It is divided into



1- Fundus Closed end of the gall bladder & completely covered with peritoneum.

2- Body Lies in contact with 1st part of duodenum & occupies the gall bladder fossa of the liver & partially covered with peritoneum.

3- Infandibulm It is angulated posterior part of the body which when dilate is called **Hartmann's pouch**

4- Neck "**S**" shaped curve, its mucosa are arranged in crescentic folds to form spiral valve of **HEISTER**.

5- Cystic duct Starts after neck, its length about (**2.5** cm), its mucosa contains crypts of **Luschka**

N.B.: Relations of fundus of G.B.:
Anteriorly : anterior abdominal wall.
Posteriorly : Transverse colon.

The Extra-hepatic biliary tracts

1- Rt. hepatic duct

It is formed by union of **ant**. & **post**. segmental hepatic ducts.

2- Lt. hepatic duct

It is formed by union of **med**. & **lat**. segmental hepatic ducts.

3- Common hepatic duct

It is formed by union of **Rt**. & **Lt**. hepatic ducts at porta-hepatis.

4- Common bile duct

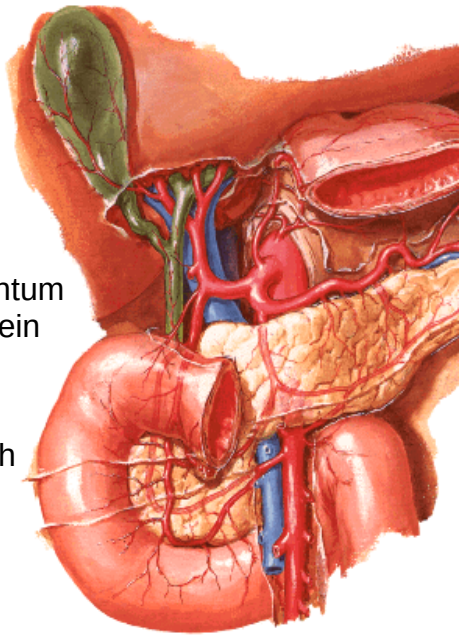
BEGINS By union of cystic duct & common hepatic duct.

ENDS At **ampula of vater** with pancreatic duct in the medial aspect of the 2nd part of duodenum.

LENGTH **5 -10 cm**

DIAMETER **8 mm**

PARTS (**4 parts**)

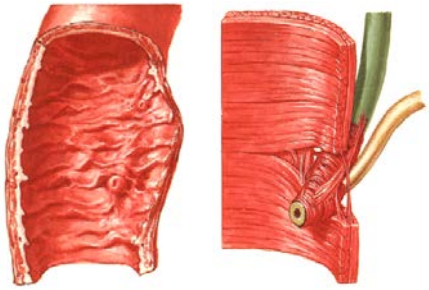


1- Supra-duodenal part (2.5 cm)
It runs in the free border of lesser omentum with hepatic artery to the **left** & portal vein **posteriorly**.

2- Retro-duodenal part (2.5 cm)
It runs behind **1st** part of duodenum with gastro-duodenal artery to the **left** and portal vein **posteriorly**.

3- Infra-duodenal part
i.e. Intra-pancreatic part.

4- Intra-duodenal part (2.5 cm)
It join the pancreatic duct to form ampula of vater which opens in the middle part of **2nd** part of duodenum, surrounded by sphincter of **oddi**.



BLOOD SUPPLY OF BILIARY SYSTEM

Arterial supply

1- G.B.: **Cystic artery** from Rt. hepatic artery.
2- Biliary tracts : **Cystic artery** & **gastro-duodenal artery** from common hepatic artery.

Surgical importance

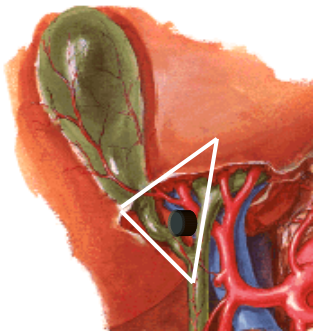
N.B: Blood supply of bile ducts runs longitudinally at **3** & **9** o'clock position. So avoid exploration at this positions

Venous drainage

Cystic vein end at Rt. branch of portal vein.

LYMPHATIC DRAINAGE

Cystic L.N of Lund at junction of cystic duct & common hepatic duct ➔ L.Ns in porta-hepatis ➔ coeliac L.Ns.



NERVE SUPPLY OF BILIARY SYSTEM

1- Parasympathetic

- **Hepato-biliary branch** from ant. vagus nerve.
- **It increases** the motor activity of G.B.

2- Sympathetic

- **coeliac plexus**
- **It is responsible about sensation** of G.B.

N.B. : Calot's triangle :

- **Bounded by** the liver, the common hepatic duct & the cystic duct.
- **It contains** the cystic artery, Lund lymph node at the junction of cystic & common hepatic duct

PHYSIOLOGY OF GALL BLADDER

FUNCTIONS OF G.B

1- Stores & concentrates bile

2- Mucus secretions

To protect the mucosa from the lytic action of bile.

3- Contracts & empties its contents

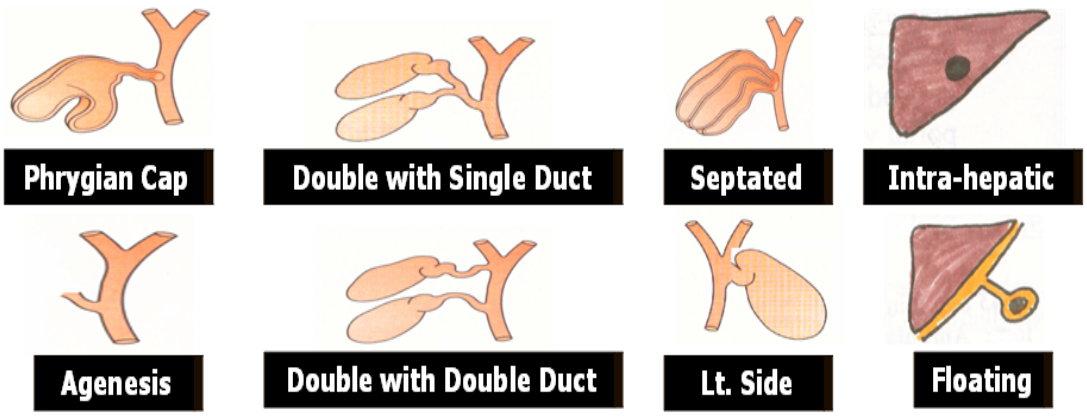
When bile is needed for digestion.

COMPOSITION OF BILE

① Water
② Electrolytes
③ Cholesterol & phospholipids
④ Bile salts
⑤ Bile pigments (the yellow colour of bile due to presence of bilirubin)

CONGENITAL ANOMALIES

I- Anomalies of gall bladder



II- Anomalies of bile ducts

1. Congenital biliary atresia

DEFINITION Failure of canalization of biliary ducts.

C/P Obstructive jaundice at birth.

PATHOLOGY Extra-hepatic or intra-hepatic .

TREATMENT (**only surgical**)

If extra-hepatic type Hepatico-jejunostomy (**kasoni operation**)

If intra-hepatic type Liver transplantation.

2. Choledochal cyst

DEFINITION Fusiform dilatation of C.B.D

C/P ① Obstructive jaundice.
② Rt. hypochondrial mass.
③ Cholangitis may occur.

TREATMENT (**only surgical**)

The cyst should be **excised** & **choledchojejunostomy** is performed

3. Cystic duct anomalies

Low insertion of cystic duct

Sessile G.B

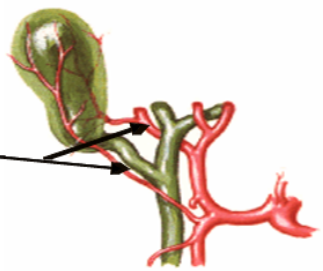
III- Anomalies of arterial supply

1. Anomalies of cystic artery

Accessory cystic artery

e.g Double cystic artery

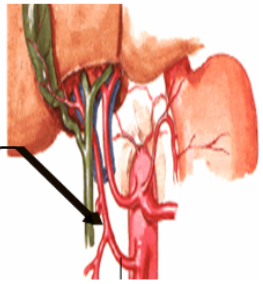
Ant. From Gatroduodenal a. **Post. From Rt. Hepatic a.**



2. Anomalies of Rt. Hepatic Artery

Accessory Rt. hepatic artery

may arise from the superior mesenteric artery

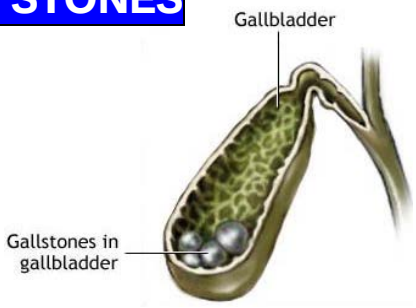


Abnormal course of Rt. hepatic artery

It may be tortuous, passing in front of bile duct ,

It is called " Caterpillar turn ". It may be legated instead of the cystic artery

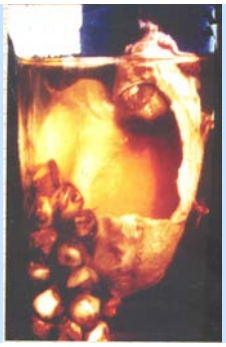
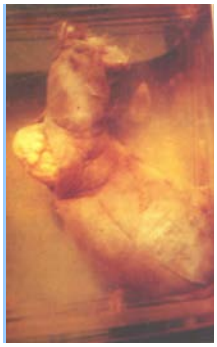
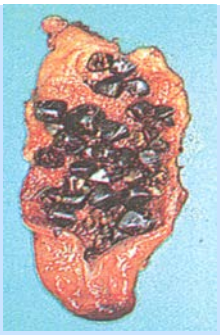
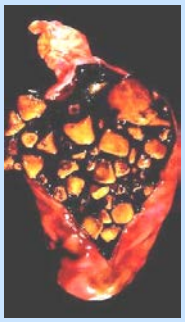
1- GALL STONES



INCIDENCE

- Common in **F**atty, **F**ilthy, **F**ertile,
- Age : **F**orty - **F**ifty years
- Sex : **F**emale > male

CLASSIFICATION

	CHOLESTEROL STONES		PIGMENT STONES	
	MIXED	PURE	BLACK	BROWN
				
INCIDENCE	**90 %**	**7 %**	**3 %**	
COMPOSITION	Cholesterol **Ca** bilirubinat **Ca** palmitat	**Pure** cholesterol	**Ca** bilirubinat	**Ca** bilirubinat **Ca** palmitat cholesterol
NUMBER	Multiple	**Single**	Multiple	Multiple
SIZE	< 2.5 cm	**> 2.5 cm**	< 2.5 cm	< 2.5 cm
COLOUR	Yellowish	Yellowish	**Black**	**Brown**
X-RAY	Radio-opaque	**Radiolucent**	Radio-opaque	Radio-opaque

AETIOLOGY

A. Causes of cholesterol stones

1-Disturbed bile salts : cholesterol ratio

A certain ratio between **bile salts : cholesterol = 25 : 1**
So any lowering of this ratio can lead to ***lithogenic bile***

1- Reduced bile salts : as in ⇘

① **Liver diseases** i.e. diminished synthesis of bile salts.

② **Excess oestrogen** which ↓ the concentration of bile salts.

③ **Malabsorption of bile salts** as in small gut resection.

2- Increased cholesterol synthesis :
e.g. Obesity or high dietary fat.

2- Stasis

This may occur in the following conditions :

① **Pregnancy** & **contraceptive pills :**
Progesterone causes relaxation & impairment of the gall bladder empting.

② **D.M** & **obesity**.

③ **Following truncal vagotomy**
i.e. denervation of gall bladder.

④ **Long term parenteral nutrition :**
because of prolonged decreasing of oral intake
→ decreasing of cholecystokinin release which in turn impair the gall bladder motility.

B. Causes of pigment stones

1- Hemolytic anemia

2- Liver cirrhosis

Most probably due to ↓↓ secretion of bile acids leading to ↓↓ the solubility of any unconjugated bilirubin.

3- Infection

Some strains of **E. coli** leads to formation of unconjugated bilirubin

N.B.: All unconjugated bilirubin precipitated with calcium to form of Ca bilirubinate

CLINICAL PICTURE

Type of patient

Fatty, **F**ilthy, **F**ertile, **F**emale & **F**orty - **F**ifty years.

N.B . Exceptions to this rule is too frequent.

Symptoms

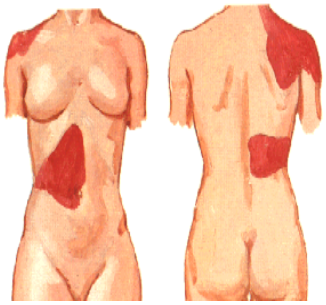
A. Silent gall stones

Discovered accidentally by X-ray or U/S.

B. Symptomatic:

1- RECURRENT ATTACKS OF BILIARY COLICS :

- **Site :** Rt. hypochondrium.
- **Character :** Sever abdominal colics.
- **Radiated to** Rt. Shoulder & inferior angle of Rt. scapula.
- **Precipitated by** fatty meals.
- **Relived by** anti-spasmodics.



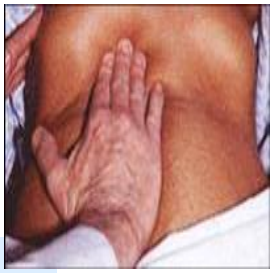
2- BILIARY DYSPEPSIA :

Nausia, fatty dyspepsia, distension, flatulence & eructation.

3- REFLEX SYMPTOMS :

i.e. Reflex vagal stimulation
leading to ↓ the coronary blood flow leading to retro-sternal pain diagnosed as anginal pain.

Signs



A- Tenderness in Rt. hypochondrium

B- +ve Murphy's sign

The gall bladder area is palpated while the patient is asked to take a deep breath, the patient will catch her breath.

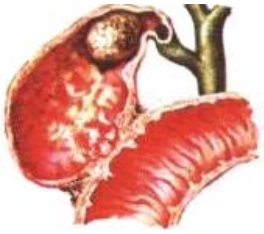
N.B. (I) SAINT'S TRIADE :

- Hiatus hernia.
- Diverticulosis coli.
- Chronic calcular cholecystitis.

(II) WILKIE'S TRIADE :

- Chronic peptic ulcer.
- Chronic appendicitis.
- Chronic calcular cholecystitis

COMPLICATIONS



I- Obstructive complications

1- Obstruction of the cystic duct

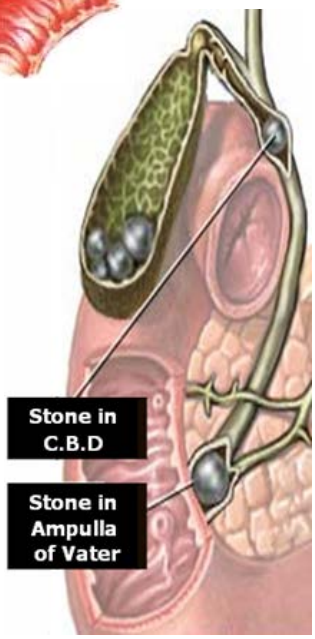
Leads to mucocele or empyema of the gall bladder.

2- Obstruction of the common bile duct

- Leads to calcular obstructive jaundice

3- Obstruction of ampula of vater

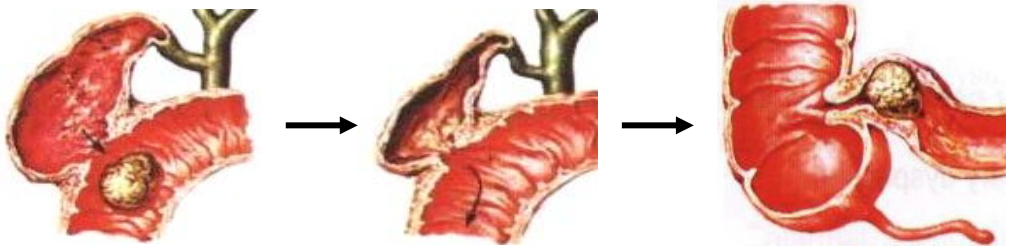
- May precipitates acute pancreatitis due to regurgitation of bile into the pancreatic duct.



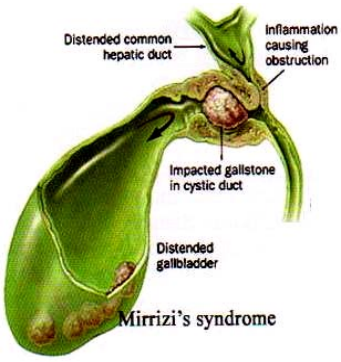
4- Obstruction of terminal ileum

i.e. GALL STONE ILEUS (**V. rare**)

- **It is a large stone** impacted in the Hartmann's pouch ➔ pressure necrosis so fistulised with the lumen of the duodenum.
- **It causes complete** obstruction in the terminal ileum by fecal accumulation around it.
- **Finally** it is impacted in terminal ileum 2 feet from Ileo caecal valve.
- **Treatment :** Milked upwards to a healthy part & removed by enterotomy or resection of affected segment if impacted.



5- Mirrizi's syndrome (rare)



- It is a partial obstruction of common hepatic duct (CHD) due to stone in the Hartmann's pouch. eventually the stone erodes into (CHD) forming single cavity & results in obstructive jaundice.
- So at operation CHD may be mistaken for cystic duct & legated.

II- Infective complications

1- Acute calcular cholecystitis.
2- Chronic calcular cholecystitis.
3- Ascending cholangitis & liver abscess.

III- Ulcerative complications

1- Chronic peptic ulcer from reflex pylorospasm & gastric stasis.
2- Internal biliary fistula with stomach, duodenum …….etc.
3- External biliary fistula with skin.

IV- Neoplastic complications

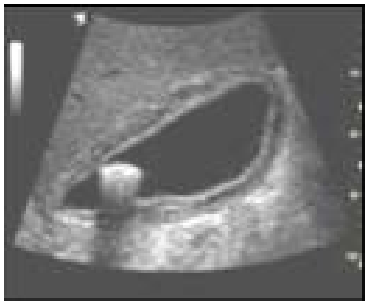
< 1 % carcinoma of gall bladder.

INVESTIGATIONS

I- Routine laboratory tests

- Liver function tests

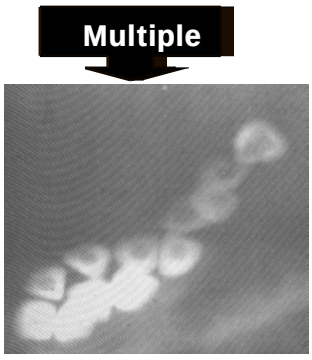
II- Abdominal U/S (95 %) accuracy

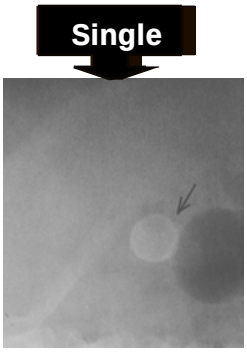


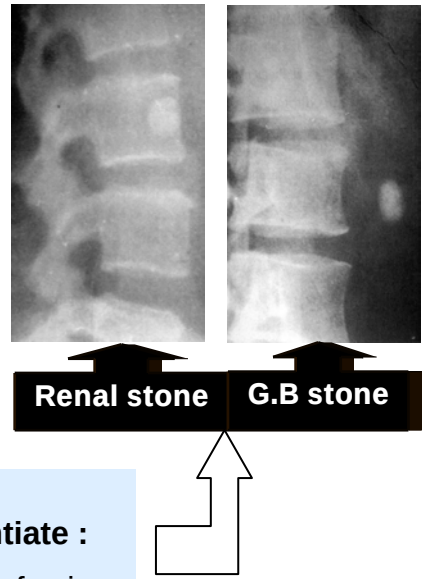
- It can detect **gall stone**
- It can detect thickness of gall bladder wall in chronic cholecystitis.
- It can detect dilated bile ducts in obstructive jaundice & dilated gall bladder

III- Plain x-ray (**Limited value**)

- It can detect **gall stone**







N .B : In case of single stone

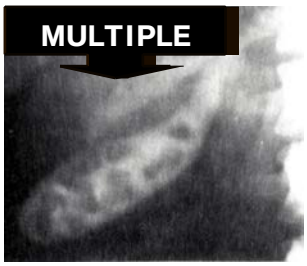
Lat. view must be done to differentiate :

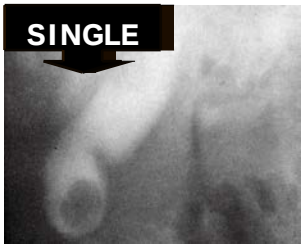
- **The gall stone** which present in front of spine
- **Rt. renal stone** which present upon spine

IV- Oral cholecystography

(**Rarely performed & now replaced by U/S**)

METHOD The patient fasts for **12 hours** before the examination and swallows **6 tablets** of **telepaque** which is secreted by the liver in the bile & passes via cystic duct to be concentrated in the gall bladder





N.B: Failure of gall bladder to opacify may be due to ☟

① Vomiting.
② Failure of absorption.
③ Inability of liver to secrete the dye
④ Cystic duct obstruction.

TREATMENT

A. Silent gall stones

- IF YOUNG OR FIT :

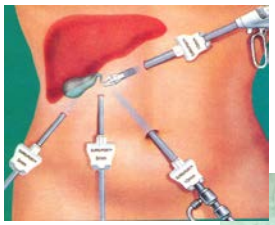
Cholecystectomy is the choice to avoid future complications

- IF OLD OR UNFIT:

Leave the patient for **follow up**.

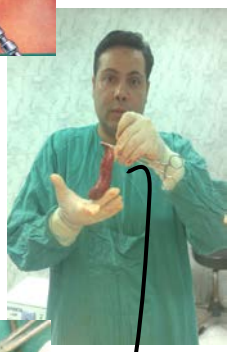
B- Symptomatic stones

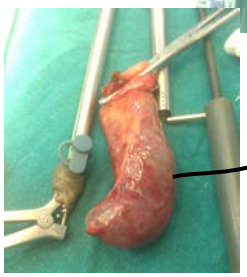
The following options are available ☟



- OPEN CHOLECYSTECTOMY .

- LAPAROSCOPIC CHOLECYSTECTOMY :





- **The advantages :**
  - ① Less post-operative pain.
  - ② Short post-operative hospital stay
  - ③ Early return to work.
  - ④ Better cosmetic result.

- **The contra-indications :**
  - ① Pregnancy as no space for pneumo-peritoneum.
  - ② Marked obesity as it is difficult to induce the ports.
  - ③ Bleeding tendency.
  - ④ Liver cirrhosis.
  - ⑤ Empyema of gall bladder
  - ⑥ Carcinoma of gall bladder.

- **The drawback :**
  - ① Shock, infection & pulmonary complications .
  - ② 1ry Hge : from bleeding vessels .
  - ③ Injury of Important structures as gall bladder, duodenum

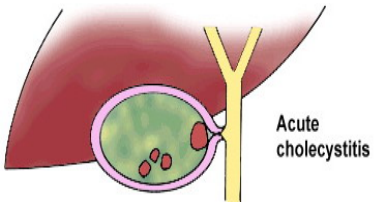
N .B : A wise surgeon **Should convert the procedures to open surgery**

2- ACUTE CHOLECYSTITIS

A- Acute calcular cholecystitis

DEFINITION

It is acute inflammation of gall bladder 2^{ry} to obstruction of cystic duct or Hartmann's pouch by a stone.

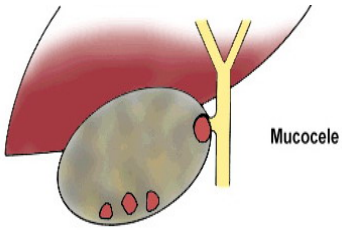


INCIDENCE 98 %

PATHOLOGY

As a result of obstruction the bile salts lead to chemical irritation of the gall bladder wall

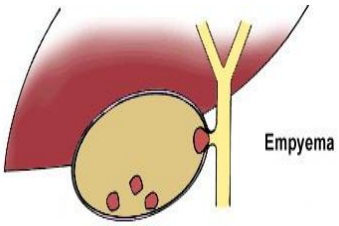
SO THERE ARE 2 POSSIBILITIES :



If the bile remains sterile

The gall bladder becomes distended by mucous & a **mucocele** of gall bladder develops.

But if 2^{ry} infection occur by gram -ve bacilli e.g. E.coli. The gall bladder becomes distended by pus & a **pyocele** (**empyema**) of gall bladder develops, leading to ⇩



1- THE WALL : Congested, oedematous & thick.

2- THE MUCOSA : Shows area of ulceration.

3- THE CONTENT : Becomes purulent.

4- THE OMENTUM & VISCERA :

e.g. Duodenum, colon... etc. are glued to gall bladder by adhesions which are 1st **fibrinous**, but later **fibrous**.

FATE

In most of cases

The stone dislodges & the inflammation is gradually subsided, leaving chronically inflamed gall bladder.

But less commonly

The inflammation is more severe with progressive distension leading to thrombosis of the blood vessels ➔ perforation of gall bladder with either localized peritonitis or less commonly generalized peritonitis (**0.5 %**) only.

N.B.(1) The commonest sites of perforation are ⇩

- **The fundus :** which is the far point from blood supply
- **The neck :** because of pressure necrosis of impacted stone.

N.B. (2) The perforation is rarely followed by generalized peritonitis because of surrounding adhesions resulting in localized peri-cholecystitic abscess which may be resolve or points

- **Externally** causing external biliary fistula.
- **Internally** causing internal biliary fistula.

N.B. (3) Acute emphysematous cholecystitis :

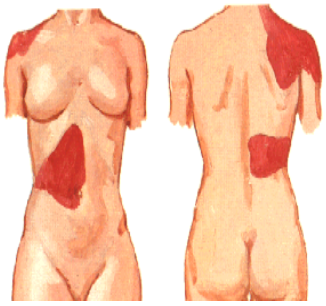
- It is a special case occur with **diabetic** patients
- It due to infection with **gas** forming organism e.g. **Clostridia**
- This infection is highly virulent & accompanied by gas formation ➔ **early gangrene** of the gall bladder.

CLINICAL PICTURE

History of biliary colics.

Symptoms **A** or **B**

A. Picture of simple obstruction (MUCOCELE)



- **Site :** Rt. hypochondrium.
- **Character :** Sever abdominal colics.
- **Radiated to** Rt. Shoulder & inferior angle of Rt. scapula.
- **Precipitated by** fatty meals.
- **Relived by** anti-spasmodics.

B. Picture of inflammation (PYOCELE)

- **Site : Localized** at right hypochondrium.
- **Character :** Throbbing pain
- **Associated with** hectic fever, rigors & sweating

Sings

A. General signs

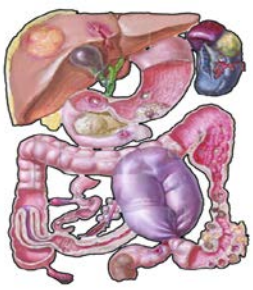
- Temperature : Increased i.e. **hyperpyrexia**.
- Heart rate : Increased i.e. **tachycardia**.
- **Jaundice :** Because of ⇩
  ① **Mirrizi's** syndrome.
  ② Associated stone in CBD.
  ③ Associated cholangitis.

B. Local signs

- Tenderness & rigidity at Rt. hypochondrium.
- **G.B is not felt** because of adhesions, tenderness & rigidity
- BOA'S SIGN
  Area of hyperesthesia between **9th** & **11th ribs** posteriorly on the Rt. side.

D.D "**Other causes of acute abdomen**" as ⇩

① Acute perforated peptic ulcer.
② **Acute cholecystitis.**
③ Acute pancreatitis.
④ Acute sub-hepatic appendicitis
⑤ High intestinal obstruction.



INVESTIGATIONS

A. Laboratory investigations

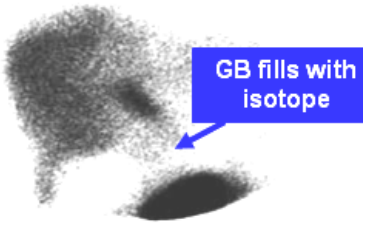
1- Blood picture ↑↑ Total leucocytic count.

2- Liver function test Usually normal.

B. Radiological investigations

1- Abdominal U/S which reveal

① Presence of gall stones.
② Distended gall bladder.
③ Thickened wall of gall bladder.

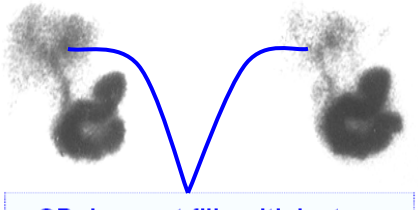


2- HID A scan **H**ydroxy **I**midazole **D**iacetic **A**cid

NOT commonly performed

THE TECHNIQUE

- **Tcm^{99}** is administrated I.V.
  Then secreted by the liver.
  Then excreted along the biliary pathway



THE RESULTS

- IF the CBD is visualized while the gall bladder is not seen, this is diagnostic for acute calcular cholecystitis due to cystic duct obstruction.

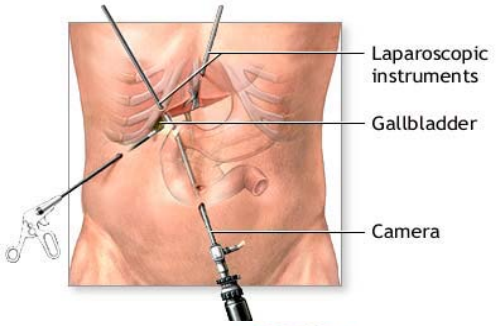
GB does not fills with isotope because its neck is obstructed, likely by a stone. The diagnosis is acute cholecystitis

TREATMENT

A. Early cholecystectomy

INDICATED IF

① The diagnosis is **sure**.
② The patient is **fit**.
③ The surgeon is **competent**.

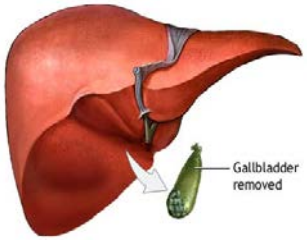


THE ADVANTAGES

① Early surgery (within 2-3 days) is not difficult as adhesions are fibrinous & not fibrous.

② Avoid possible complications.

③ One hospital admission with early return to work.



B. Conservative treatment followed by surgery i.e. 6 weeks interval

THIS POLICY IS TO TREAT THE PATIENT AS FOLLOWING

① Naso-gastric tube & I.V fluid.
② Antibiotics 3rd generation e.g. **Cephalosporins**.
③ Analgesics e.g. **Pethidine**.

Then follow up for pulse, temp, degree of pain and area of tenderness & rigidity.

But if deterioration occur for the patient, **surgical intervention must be carried out**.

B- Acute non calcular cholecystitis

DEFINITION

It is a serious condition of acute Inflammation of gall bladder **not related** to stone formation.

INCIDENCE

Rare about **2 %** only.

PATHOLOGY **Unclear**

But may be due to change in composition of bile or ischemia of the gall bladder,

So it may occur with ⇩

① Patient suffering from major burn or major trauma.

② Patient with certain infections such as brucellosis & typhoid.

CLINICAL PICTURE

As **acute calcular cholecystitis.**

But usually the diagnosis is delayed because it is not suspected.

INVESTIGATION

Abdominal ultrasound is diagnostic

TREATMENT

Urgent cholecystectomy.

3- CHRONIC CHOLECYSTITIS

A- Chronic calcular cholecystitis

DEFINITION

It is a chronic inflammation of gall bladder most probably as a fate of acute calcular cholecystitis when the stone is dislodged

PATHOLOGY.

A- Early changes of the gall bladder

- **Opacity** of gall bladder wall.
- **Fibrosis** of liver around the gall bladder bed so the bladder doesn't empty completely.
- **Enlargement** of cystic L.Ns.

B. In late cases

- **Calcification** of gall bladder wall.

CLINICAL PICTURE

INVESTIGATIONS

TREATMENT

SEE GALL STONES

B- Chronic non calcular cholecystitis

DEFINITION

It is a case of chronically biliary dyspepsia **not related** to stone formation.

PATHOLOGY

(**3 Pathological condition**)

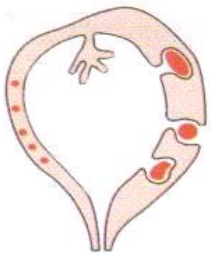
1- Infection e.g. Typhoid gall bladder.

2- Biliary dyskinesia

Due to **achalasia** of sphincter of oddi leading to marked dilatation of gall bladder,
i.e. Sphincters doesn't relax when G.B. contracts.

N.B. : Cholecystectomy will aggravates the case

3- Cholesterosis (**Strawberry** gall bladder) ⟶



Due to an error of cholesterol metabolism which causes deposition of cholesterol crystals in the mucosa.

TREATMENT **Only conservative**

① **Avoid** fatty diet & heavy bulky meal.

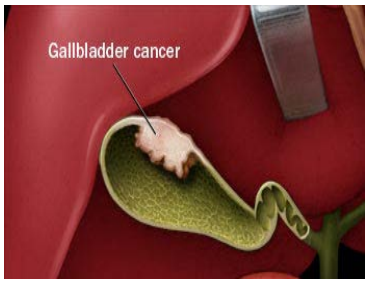
② **Antibiotics** & biliary **antiseptics**.

③ **Antispasmodics** especially for biliary dyskinesia.

4- CARCINOMA OF THE GALL BLADDER

INCIDENCE

- **Age :** > **50** years
- **Sex :** Female **>** male



PREDISPOSING FACTORS

- **Gall stones** due to chronic irritation.
- **Porcelain G.B** (calcified wall of the G.B.).
- Gall bladder **polyps**.

PATHOLOGY

- Adenocarcinoma (**90 %**).
- Squamous cell carcinoma (**10 %**).

SPREAD

- **Direct :** Liver & bile passages.
- **Blood :** Liver, lung, bone & rarely brain.
- **Lymphatics :** Cystic L.Ns (**Lund**) & porta hepatis L.Ns.

CLINICAL PICTURE

1- PATHOLOGICAL GROUP :

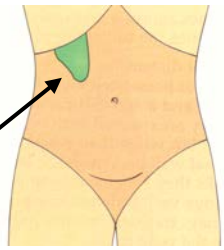
Discovered by the pathologist, after cholecystectomy.

2- OBSTRUCTIVE GROUP :

Acute cholecystitis, obstructive jaundice.

3- MASS GROUP :

Mass in the Rt. hypochondrium.



INVESTIGATION

1- ABDOMINAL ULTRASOUND (U/S)

Shows an mass inside the gall bladder (accurate **> 80 %**)

2- MAGNETIC RESONANCE IMAGING (MRI)

3- NEEDLE BIOPSY guided by U/S.

TREATMENT

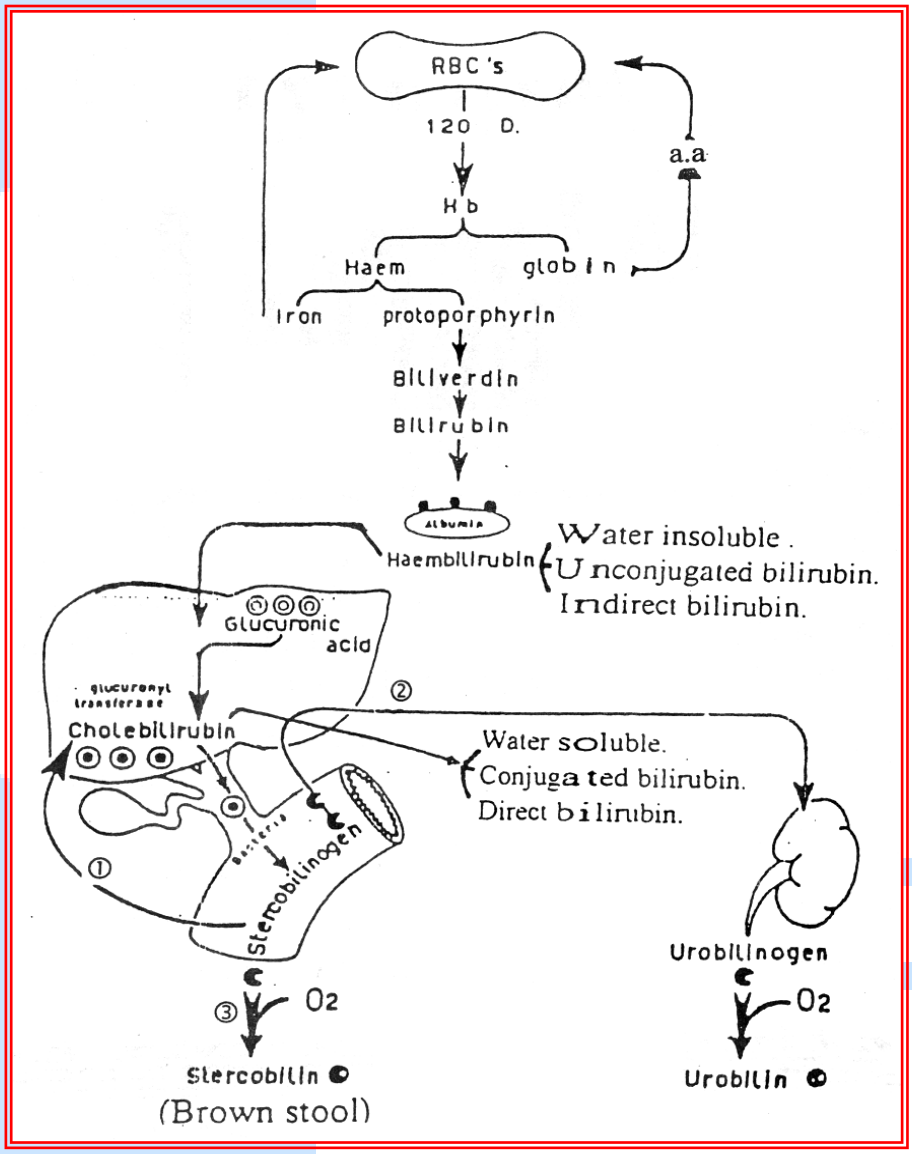
A- Operable cases

- **Cholecystectomy** with extended right hepatic resection.
- **If discovered during operation of cholecystectomy :**
  Cholecystectomy
  + resection of underlying liver tissue
  + any accessible lymph node.

B- Inoperable cases

- Relieving of the jaundice by a **stent**

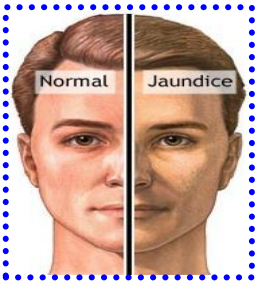
Jaundice


RBC 's
120 D.
a.a
Hb
Haem
globin
Iron
protoporphyrin
Biliverdin
Bilirubin
Albumin
Haembilirubin
Water insoluble .
Unconjugated bilirubin.
Indirect bilirubin.
Glucuronic acid
glucuronyl transferase
Cholebilirubin
Water soluble.
Conjugated bilirubin.
Direct bilirubin.
Bacteria
Stercobilinogen
Urobilinogen
O2
O2
Stercobilin
Urobilin
(Brown stool)

JAUNDICE

DEFINITION

Jaundice is the yellowish discoloration of the tissues & body fluid
Except (CSF, brain, tears, saliva & milk)
due to excess bilirubin in blood.



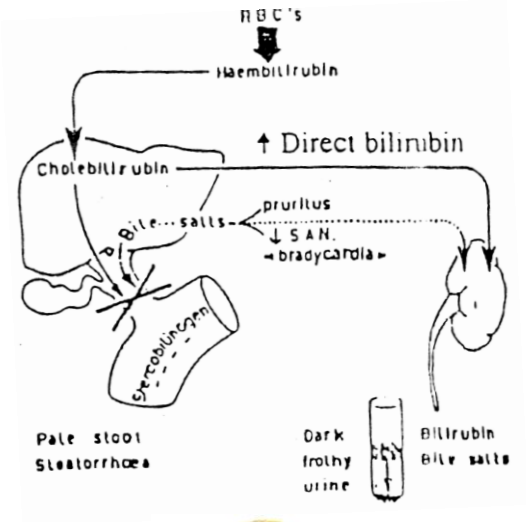
N.B.: Normal level of serum bilirubin **< 1 mg %**

Jaundice occurs if serum level of bilirubin **> 3 mg %**
So if serum level of bilirubin **1-3 mg %** it is called sub-icteric (chemical) jaundice.

OBSTRUCTIVE JAUNDICE

PATHOGENESIS

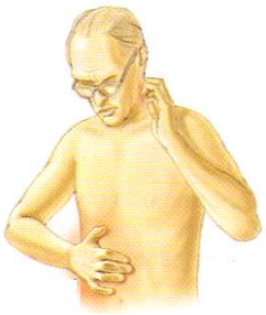
Because of the obstruction the direct bilirubin is prevented from reaching to intestine



So **1- No stercobilinogen**.
This will lead to **pale** (clay) stool.

2- The direct bilirubin
Will regurgitate to the blood & excreted to the urine leading to **dark** urine i.e. Tea like urine.

3- The bile salts will ↑↑ in blood & ↓↓ in intestine.



⇨ **↑↑ bile salts in blood leads to**
① Bradycardia
② Itching (pruritis) of skin
due to irritation of nerve endings.
③ Frothy urine.

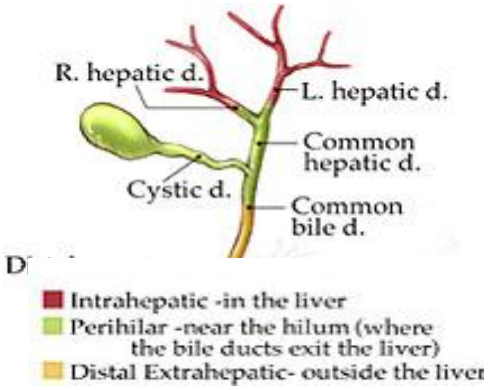
⇨ **↓↓ bile salts in intestine leads to**
① Defective absorption of fat & fat soluble vit. (**A**.**D**.**E** & **k**) : leads to steatorrhea & bleeding tendency

N.B.: ***I.V vit. K*** will improve the bleeding tendency

② Defective bacteriostatic function : leads to increasing the fermentation ➔ offensive bulky stool.
③ Defective its motility effect : leads to constipation, abdominal distension & anorexia.

N.B. : Absence of bile salts from intestine ➔ ↑ Absorption of bacterial endotoxins ➔ renal vasoconstriction ➔ acute renal failure i.e. **Hepato-renal failure**.

AETIOLOGY



A- Intra-hepatic obstruction

Liver tumors & liver cirrhosis

B- Extra-hepatic obstruction

A- Lumen

① **Stone** (choledecholithiasis)
② **Blood** e.g. hemobilia
③ **Parasite** e.g. ascaris or fasciola.

B- Wall (Strictures of the biliary tract)

① **Congenital biliary atresia** (Discussed before)

② **Traumatic strictures** This usually follows cholecystectomy

- **It may be due to :**
  1. Complete legation of the common hepatic or common bile duct.
  2. Devitalization of the duct due to rough dissection.

- **Operative correction** is the only treatment but is technically difficult.

③ **Inflammatory stricture** (**Sclerosing cholangitis**)

- **Aetiology :** This is a condition of **unknown** aetiology

- **Treatment :**These patients are candidates for **liver transplantation**.

④ **Neoplastic stricture** (**Cholangiocarcinoma**)

Risk factors

① **Choledochal** cyst
② **Clonorchis sinesis**infestation.
③ **Sclerosing cholangitis**
④ **Hemochromatosis**.

Treatment

- **Operable cases** at the lower end of CBD

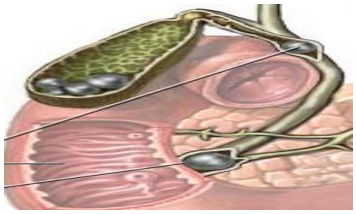
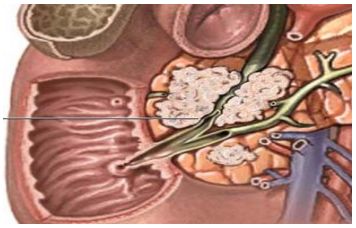
  are treated by pancreatico-duodenectomy (**Whipple's operation**).

- **Inoperable cases** are treated by **cholecysto-jejunostomy**

C- Outside

① L.Ns metastasis at porta-hepatis
② **Cancer head pancreas**
③ Peri-ampullary carcinoma of 2nd part of duodenum.

The common 2 causes

Calcular obstructive jaundice	Malignant obstructive jaundice
	

PATHOLOGY

EFFECTS ON BILE DUCT :

- **Proximally to obstruction :**
  - The bile duct is dilated i.e. retention of bile occur.
- **The content :**
  - 1st thick **biliary mud** due to stagnation, infection & pus
  - Then **liver damage** will occur because of back pressure
  - **So** the bile duct contains mucous (**white bile**) which is a serious bad sign of liver failure.
- **Distally to obstruction :**
  - The bile duct may be stenosed

EFFECTS ON LIVER :

- Dilated hepatic bile ductules (**hydrohepatosis**).
- If infection occurs = **suppurative cholangitis**.
- Finally, acute progressive **liver damage** occur.

EFFECTS ON GALL BLADDER :

According to Courvoisier's law

IN CALCULAR O.J	IN MALIGNANT O.J
The gall bladder is contracted & non palpable (**80 %**) due previous Cholecystitis. But in (**20 %**) the gall bladder is dilated & healthy due to ⇩ ① metabolic stone ② stone in the cystic duct causing mucocele of G.B or empyema if infected.	The gall bladder is dilated & healthy in (**98 %**) But in (**2 %**) gall bladder doesn't dilated due to ⇩ ① Associated cholecystitis ② The L.Ns from cancer head pancreas at porta hepatis ③ Previous cholecystectomy

C/P	Calcular obstructive jaundice	Malignant obstructive jaundice
Personal H.		
♦ **Age :**	• **Middle** age.	• **Old** age.
♦ **Sex :**	• Usually **female**.	• Usually **male**.
Complaint :	Yellowish discoloration or symptoms of the disease.	
Present H.		
♦ **O.C.D**	• Sudden **onset** with intermittent **course** & variable **duration**	• Gradual **onset** with progressive **course** & short **duration**.
♦ **Pain**	• Mild degree. • Colicky in Rt. hypochondrium • Radiated to Rt. shoulder • ↑↑ by fatty meals. • ↓↓ by antispasmodics.	• Severe degree • Boring • Referred to back • ↑↑ By lying down. • ↓↓ By leaning forwards.
♦ **Fever**	• **Charcot's triad** (pain, jaundice & fever with rigors) • **Reynold's pentad** as above + mental confusion & shock.	———
♦ **Wt. loss & Metastasis**	**- Ve**	**+ Ve**
	See pathogenesis (discuss **)**	
♦ **Stool**	• **Pale**, bulky, offensive & steatorrhea.	
♦ **Urine**	• **Dark** (tea like) & frothy.	
♦ **Pruritis**	• Present & leads to **scratching marks**.	
♦ **Bleeding tendency**	• Presents from the orifices & **improved by I.V vit K**	
Past H.	• Biliary dyspepsia	———
General Ex.		
♦ **Temp.**	• Hyperpyrexia (**Charcot's**)	• Normal.
♦ **Pulse**	• Bradycardia.	• Bradycardia.
♦ **Looks**	• Alert with cholangitis	• **Ill** with cachexia.
♦ **Built**	• Obese	• Underbuilt.
Local Ex.		
♦ **Liver**	• Enlarged tender liver.	• Enlarged tender liver.
♦ **G.B**	• **Courvoisier's law.**	• **Courvoisier's law.**
♦ **Spleen**	• **No**	• **No**
♦ **Ascites**	• **No**	• **±** as sign of malignancy.

INVESTIGATIONS (6 Problems)

1- To prove jaundice

Estimate serum bilirubin which is ⇩

- Normal **< 1mg** %
- Sub clinical **1-3 mg** %
- Clinical **> 3 mg** %

2- To prove that is obstructive or not

1- Stool analysis :

Clay, bulky & offensive.

2- Urine analysis :

Dark (tea like) & frothy.

3- Blood analysis :

↑↑ bilirubin mainly direct

4- Alkaline phosphatase : [N = **3** - **13** king armstrong unit]

This enzyme increased mainly with obstructive jaundice.

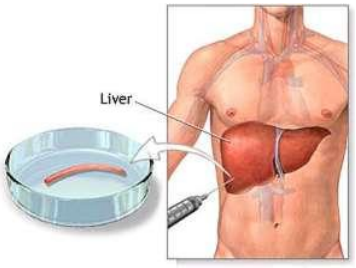
3- To exclude other types of jaundice

1- Blood examination:

To exclude hemolytic jaundice.

2- Liver biopsy

To exclude hepato-cellular jaundice.



4- To assess its effect on coagulation

Through prothrombin time & concentration which are ⇩

- **Normal** with hemolytic jaundice.
- **Prolonged** time & **diminished** concentration with both obstructive & hepatocellular jaundice.

So TO DIFFERENTIATE BETWEEN THE 2 CONDITIONS :

⇨ **GIVE I.V VIT. K (FEW DAYS)**

- ♦ IF improved prothrombin parameter
  = Obstructive jaundice
- ♦ IF not improved prothrombin parameter
  = Hepato-cellular jaundice.

5- To assess its effect on liver function

1- SGOT & SGPT : [N = **12** - **40** unit/ml]

Which is **increased** with obstructive jaundice

2- SERUM ALBUMIN : [N = **3.5** – **5** gm %].

Which is **decreased** with obstructive jaundice.

6- To prove the cause of obstructive jaundice (**Stone** or **malignancy**)

1- Abdominal U/S

It will show

① Dilatation of intra-hepatic radicals.
② Dilatation of CBD & presence of **stone**.
③ A **mass** of head of pancreas may be detected.

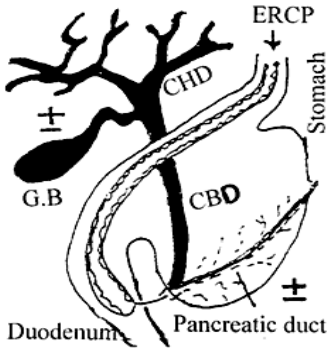
2- Abdominal CT scan

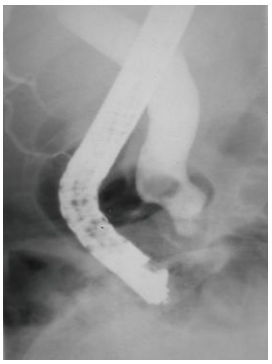
Valuable to detect pancreatic **mass** or metastatic **L.Ns**.

3- E.R.C.P

[**E**ndoscopic **R**etrograde **C**holangio-**P**ancreatography]

- **Both** CBD & pancreatic ducts are visualized.
  so the stones appear as a **filling** defect
- **Also** can detect any lesion of ampula & biopsy is taken.





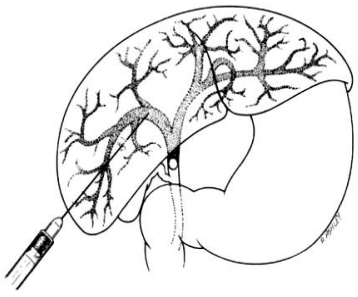
4- M.R.C.P (Recently used)

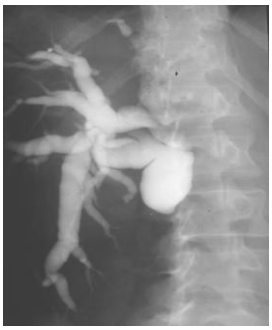
[**M**agnetic **R**esonance **C**holangio-**P**ancreatography]

5- PTC

[**P**ercutaneous **T**rans-hepatic **C**holangiography]

- **It is done** if the obstruction is high up in the hepatic ducts.
- **It ends by PTD** [**P**ercutaneous **T**rans-hepatic **D**rainage]
  if the serum bilirubin **> 20 mg** % to drain bile.

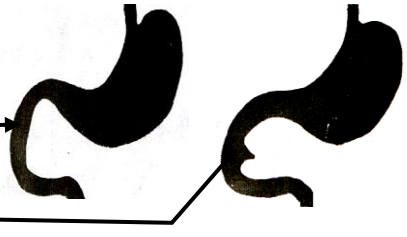




6- Barium meal (Duodenography)

A- Widening of duodenal curve
i.e. cancer head pancreas

B- Inverted 3 shaped
i.e. peri ampullary carcinoma



TREATMENT

A- Pre-operative management

1- CORRECT CLOTTING DYSFUNCTION :

① I.V **Vit. K**

② Fresh **blood** transfusion.

2- PROTECT AGAINST LIVER CELL FAILURE (L.C.F)

① High intake of **glucose** to increase liver glycogen.

② **Lactulose** & liver **tonics**.

③ **Antibiotics** e.g. Cephalosporins to prevent cholangitis.

3- PROTECT AGAINST RENAL FAILURE :

① Adequate **hydration** by I.V fluid.

② I.V **mannitol** to induce diuresis.

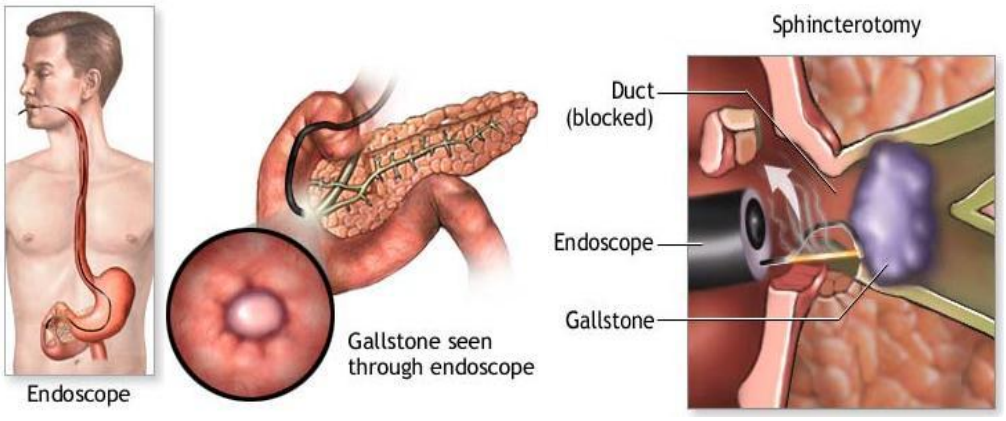
③ **Oral bile salts** to decrease liability of endotoxemia
i.e. to ↓ Hepato-renal failure.

B- Operative procedures

Treatment of calcular obstructive jaundice

(I) Endoscopic extraction of stones By (**ERCP**)

If ERCP is available & followed by cholecystectomy



TECHNIQUE (ENDOSCOPIC SPHINCTEROTOMY)

1- Diathermy is used to do **endoscopic sphincterotomy**.

2- Stone(s) can be extracted by " **Dormia basket** " from CBD

3- A large stone can be crushed through " **mechanical lithotripsy** " by a special crushing wire basket.

COMPLICATED BY

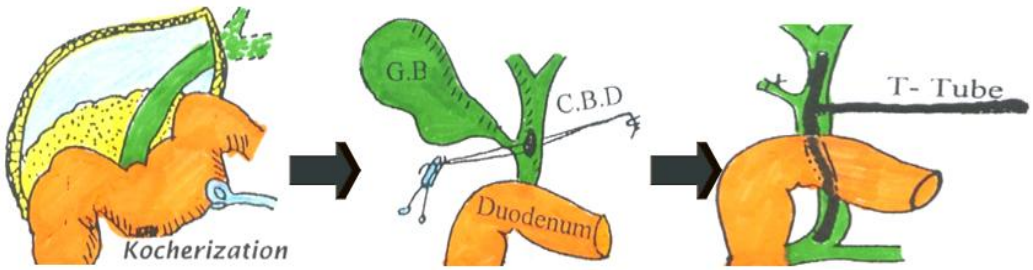
1- **Bleeding** (hemobilia) **2** - **9** %.

2- **Acute cholangitis** **1 – 3** %.

3- **Acute pancreatitis** **1 – 4** %

(II) Cholecystectomy & Choledocho-lithotomy If ERCP is NOT available

STEPS



1- The 1st step is to mobilize the duodenum from posterior abdominal wall (**Kocherization**) of duodenum to expose the retro-duodenal portion of C.B.D

2- 2 stay sutures are taken in the wall of the supra- duodenal portion of C.B.D.

3- A vertical incision (**2** cm long) is made in between the 2 stay sutures.

4- Stone forceps is introduced into the C.B.D to remove the stone, then check that there is no stenosis by passing **Backe's dilator**

5- Some surgeons insert a choledochoscope to check that there are no retained stones.

6- T-tube is inserted in (CBD) which is closed around the tube, the long limb of the tube is brought outside the patient.

7- T-tube cholangiogram can be performed after closure of CBD to check absence of filing defect, i.e. completion T-tube cholangiography.

8- Cholecystectomy is then performed

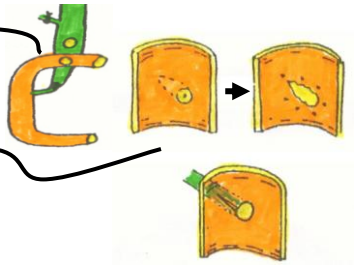
In some cases : An additional procedures have to be performed

I. Choledocho-duodenostomy

- **Indicated if** stone impacted at lower end of CBD.

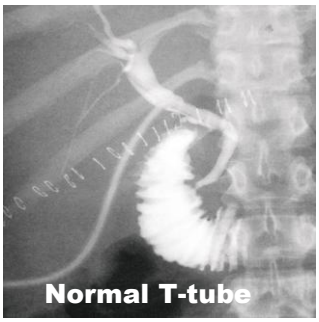
II. Sphincterotomy or Sphincteroplasty

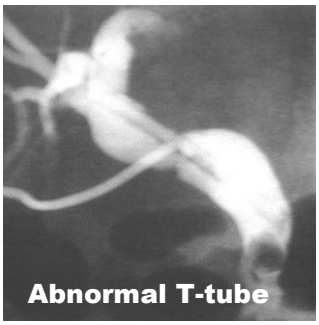
- **Indicated if** stone impacted at sphincter of oddi.



POST-OPERATIVE

10 days post-operative another **T-tube cholangiogram** to ensure that no residual stones before removal of the tube





N.B : Management of missed stones

Irrigation by saline, if failed administration of " **Dormia basket** " if failed re-exploration " surgically "

Treatment of malignant obstructive jaundice

TREATMENT OF CANCER HEAD PANCREAS

Operable

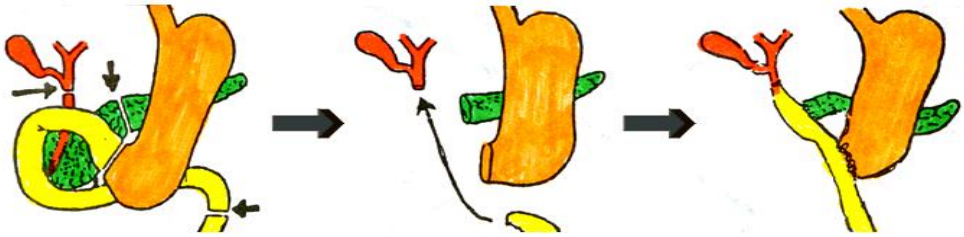
PANCREATICO-DUODENECTOMY (WHIPPLE OPERATION)

i.e. Removal of duodenum, lower part of CBD & head of pancreas.
Then restore the continuity by **the following anastomoses**

① The common bile duct to jejunum.

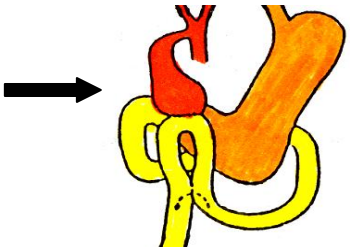
② The pancreatic duct to the jejunum.

③ The stomach to the jejunum .

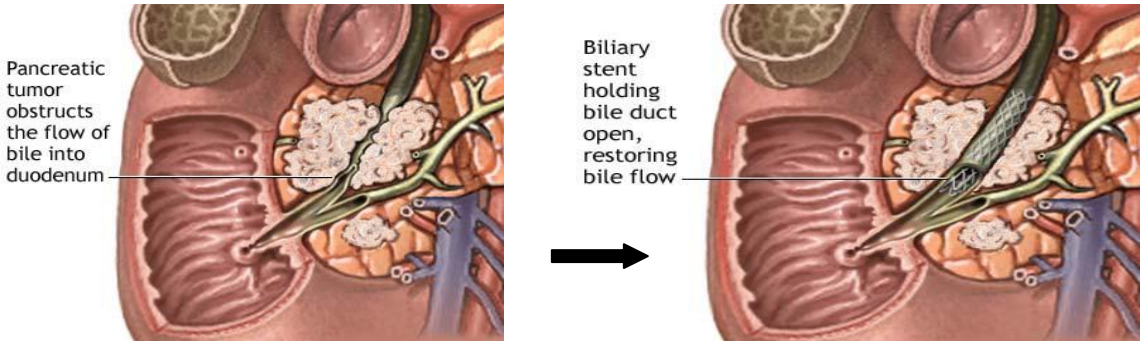


Inoperable **Either (A) or (B)**

(A) CHOLECYSTO-JEJUNOSTOMY WITH JEJUNO-JEJUNOSTOMY



(B) INTERNAL BILIARY STENT

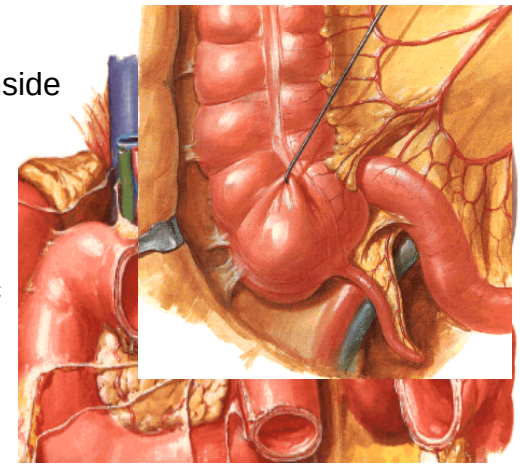


The Pancreas

THE PANCREAS

SURGICAL ANATOMY

STRUCTURES & RELATIONS



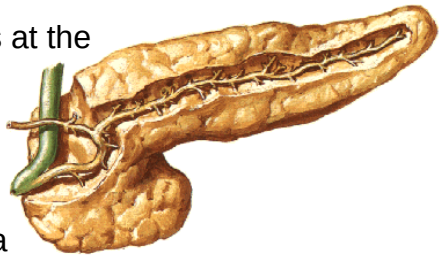
1- HEAD : The CBD is partially embedded inside it, so cancer head → obstructive jaundice.

2- NECK : The beginning of the portal vein.

3- BODY : Splenic vein lie behind it & splenic artery runs on its upper border.

4- TAIL related to the hilum of the spleen, it may be injured during splenectomy.

5- PANCREATIC DUCTS :



a. The main duct of (**Wirsung**) begins at the tail & ends by joining the common bile duct, at the ampula of vater.

b. 2 Accessory ducts of (**Santorini**) may join the main duct or may open in the minor duodenal papilla which lies 2 cm cranial & slightly anterior to the major papilla.

ARTERIAL SUPPLY

1- Mainly from pancreatic branches of splenic artery.

2-Superior & inferior pancreatico-duodenal arteries.

VENOUS DRALNAGE

Into the corresponding veins → portal vein.

SURGICAL IMPORTANCE

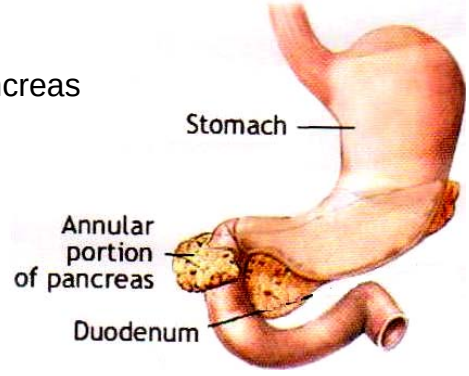
1- The CBD is embedded inside the head so cancer head → obstructive jaundice.

2- The splenic vein is behind pancreas, so legation of vein is difficult with modified Warren operation

3- Tear of tail of pancreas during splenectomy → burst abdomen

CONGENITAL ANOMALY

Annular pancreas



- It is due to abnormal fusion of ventral & dorsal pancreas to form a caller of tissue surrounding the 2nd part duodenum & may compress it.
- It presents with repeated bile stained vomiting if below the ampula.
- **Plain X-ray :** Double-bubble sign,
- **Treatment :** Duodeno-jejunostomy

I- PANCREATITIS

A- Acute pancreatitis

DEFINITION

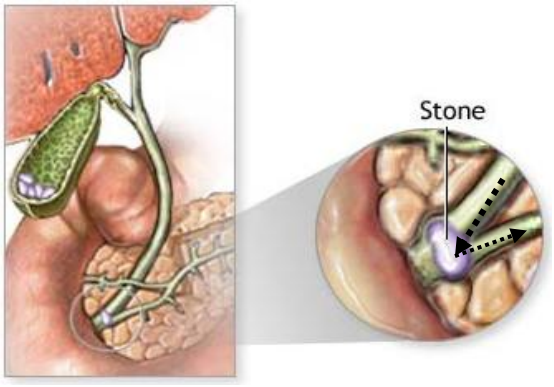
It is a serious condition that leads to death in **10 %** of cases.

AETIOLOGY

[**Unknown**] but the following theories are accepted ⇩

- 50 % BILE DUCT STONE :

  The stone is impacted at the ampula of vater ➔ obstruction of pancreatic duct ➔ regurge of bile into the pancreatic duct ➔ activation of the pancreatic enzymes(**Trypsinogen**) ➔ Auto-digestion of the pancreas.



- 20 % EXCESS ALCOHOL INTAKE.

- 20 % IDIOPATHIC.

- 5 % TRAUMATIC : Either accidental operative, ERCP …etc

- RARE CAUSES : e.g. Viral infection (mumps) or hyperparathyroidism

PATHOLOGY

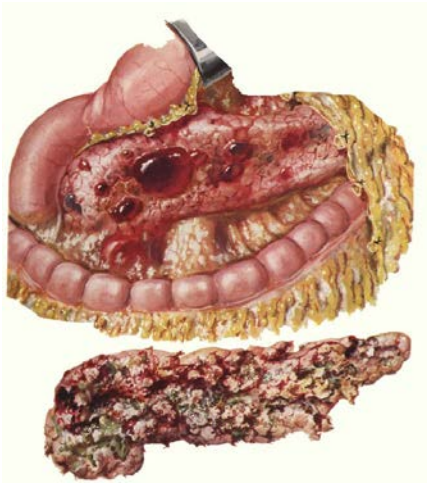
A- The pancreas

THE SEVERITY OF INFLAMMATION RANGES FROM ⇩

① MILD OEDEMA
i.e. Acute **oedematous** pancreatitis.

② HEMORRHAGE
i.e. Acute **hemorrhagic** pancreatitis.

③ SEVERE NECROSIS
i.e. Acute **necrotizing** pancreatitis.

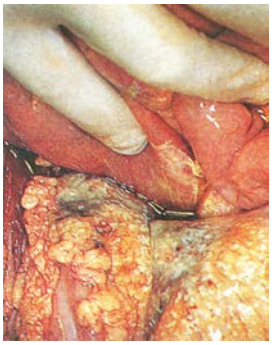


B- The peritoneal cavity

IT SHOWS THE FOLLOWINGS ⇩

① BLOOD stained exudate.

② FAT NECROSIS of greater omentum & mesentery. which present in the form of yellowish white areas like wax. if inflamed pancreas liberates lipase enzyme ➔ splits fat into fatty acids & glycerol. Then the fatty acids combine with calcium ➔ calcium soap.



③ PROTEIN BREAKDOWN PRODUCTS
e.g. Vaso-active kinines will be absorbed leading to generalized vasodilatation with hypotension.

N.B. The sources of protein :
1- Loss of plasma proteins into the peritoneum .
2- ↑ breakdown by the action of trypsinogen

COMPLICATIONS

A- General complications

1- HYPOVOLAEMIC SHOCK from loss of plasma proteins into the peritoneum & severe hypotension from circulating kinines.

2- MULTI-ORGAN FAILURE due to severe loss of blood with acute hemorrhagic pancreatitis as ⇩

(a) **ARDS** (**A**dult **R**espiratory **D**istress **S**yndrome).

(b) **ARF** (**A**cute **R**enal **F**ailure).

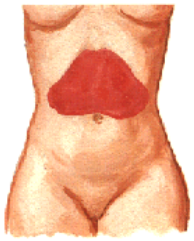
3- PARALYTIC ILEUS + FLUID & ELECTROLYTE IMBALANCE.

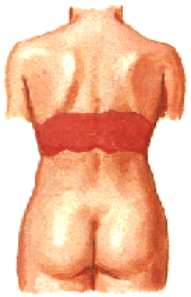
B. Local complications

1- PANCREATIC PSEUDOCYST (**discussed later**)

2- PANCREATIC ABSCESS

CLINICAL PICTURE





Type of patient Male **=** female & **30** - **50** years.

Symptoms

① History of the cause (**discuss**)

② Severe upper abdominal agonizing pain radiating to back.

③ Vomiting & hiccough are prominent features.

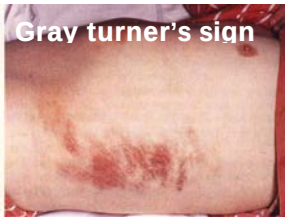
Signs

A- GENERAL EXAMINATION :

① Hypovolemic shock with fever.

② Faint jaundice may be seen in the 2nd day 2ry to oedema of pancreas.

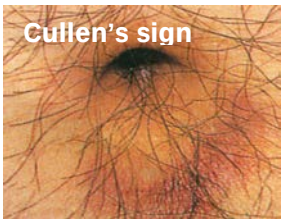
③ The pain relieves by leaning forwards



B- LOCAL EXAMINATION :

① Mild tenderness & rigidity at epigastric region.

② Bluish discoloration of skin in the loins (**Gray turner's sign**) & around umbilicus (**Cullen's sign**) due to trickling of bloody exudates in retro-peritoneal space.



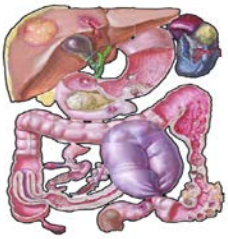
③ **Picture of complications as** ⇩

1- Abdominal distension due to paralytic ileus.

2- Palpable pulsating mass due to pseudo-cyst.

D.D "**Other causes of acute abdomen**" as ⇩

① Acute perforated peptic ulcer.
② Acute cholecystitis.
③ **Acute pancreatitis.**
④ High intestinal obstruction.



INVESTIGATIONS

A- Laboratory

1- SERUM AMYLASE IS elevated > **1000** IU/dL (Normal = **100** - **300** I.U./dL)

N.B. : ↑ Serum amylase not specific
- **Because elevated also with** ⇩
  ① Perforated peptic ulcer.
  ② Acute cholecystitis.
  ③ Myocardial infarction.
- **But** all these causes **never exceed 500** IU/dL

2- SERUM LIPASE is elevated (more specific than amylase).

3- BLOOD ELECTROLYTES & ARTERIAL BLOOD GASES.

4- ECG & CARDIAC ENZYMES
as creatine phosphokinase (**C**.**P**.**K**) to exclude myocardial infarction,

B- Radiological

1- PLAIN X-RAY : Shows ⇩
- Dilated short segment of small intestine " **sentinel loop** ".
- **Distended** ascending colon & **collapse** of the descending colon " **Colon cut off sign** "

2- ABDOMINAL U/S : Shows gall stones & enlarged pancreas.

3- CT SCAN : The most accurate.

ASSESSMENT OF SEVERITY **Ranson's criteria** **High risk mortality :**

Age > 55 years **White cell count** > 16000 **Blood glucose** > 200 mg/dl

Serum LDH > 350 IU/L **Serum AST** > 250 IU/L

TREATMENT [**Mainly conservative**]

A- Conservative treatment

SIMPLY REMEMBERED as the "R" regimen

1- R**elief** of pain by pethidine.

2- R**eplacement** of fluid & electrolytes loss.

3- R**est** of pancreas & bowel by no oral intake + Ryle's tube.

4- R**espiratory** support by oxygen mask & mechanical ventilator.

5- R**esistance** of infection by A.B.

6- R**emoval of obstructing cause** by ERCP

7- R**eassessment** after improvement by ERCP

B- Surgical treatment

ESPECIALLY WITH DOUBTFUL DIAGNOSIS :

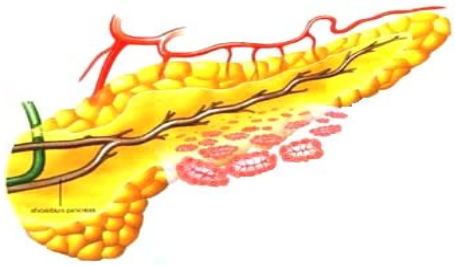
1- Through exploration, peritoneal lavage, removal of necrotic tissues & finally closure over a drain,

or 2- Drainage of pancreatic abscess or pseudocyst.

B- Chronic pancreatitis

INCIDENCE

- **Age :** > **50** years
- **Sex :** Male **>** female



AETIOLOGY

- Chronic **alcoholism**
- Less commonly stone, trauma ... etc.

PATHOLOGY

Pancreatic duct

It shows multiple strictures with consequent proximal dilatation.

Pancreatic acini

It shows slowly progressive destruction with increasing fibrosis & later on **calcification** with loss of its endocrinal & exocrinal function.

CLINICAL PICTURE

① PAIN : Epigastric & radiating to back.

② MALABSORPTION with steatorrhea & loss of weight.

③ DIABETES MELLITUS.

INVESTIGATIONS

A- Laboratory

① **Serum amylase** may be elevated.

② **Stool analysis** shows steatorrhea.

③ **Glucose tolerance test**.

B- Radiological

① **Plain x-ray** shows pancreatic calcifications.

② **Abdominal U/S** & **CT scan :** The most accurate.

③ **ERCP** shows multiple strictures of pancreatic duct with proximal dilatation

TREATMENT

A- Conservative treatment

① **Control of pain** by analgesics.

② **Correction of malabsorption** by pancreatic enzyme tablets with meals.

③ **Control of D.M** by insulin.

B. Surgical treatment (If persist uncontrolled pain)

1- Resection of part of pancreas

or 2- Drainage of dilated obstructed pancreatic duct, e.g. Pancreatico-jejunostomy

II- PANCREATIC CYSTS

I- True cyst (20 %)

TYPES

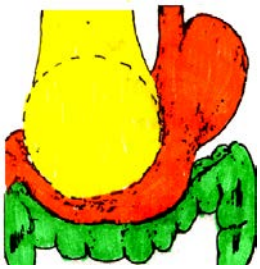
- **Acinar :** e.g. retention cyst, congenital cyst & cystadenoma.
- **Inter-acinar** e.g. dermoid cyst & hydatid cyst.

TREATMENT

1- Anastomosed to stomach i.e. **cysto-gastrostomy**

II- Pseudo-cyst (80 %)

DEFINITION



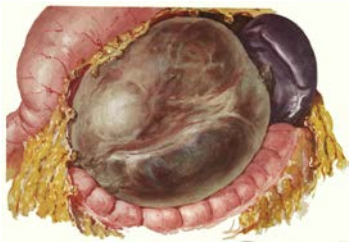
This is a collection of pancreatic secretions & inflammatory exudate within a lining of inflammatory tissue not epithelium. So it is called **" pseudo cyst "** in lesser sac

N.B.: The commonest site is behind & above the stomach.

AETIOLOGY

As a result from 1- acute pancreatitis after **2** - **3** weeks .

2- Pancreatic trauma



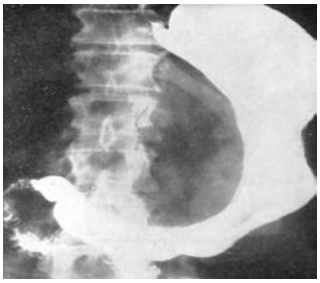
COMPLICATIONS

Hge, rupture, abscess **but never malignancy**.

CLINICAL PICTURE

Cystic epigastric swelling above umbilicus & shows transmitted pulsation from the aorta which disappears in knee- elbow position

INVESTIGATIONS



A- Barium meal

shows displacement & compression on stomach .

B- CT scan & U/S (The best)

TREATMENT

(Most of cyst resolve spontaneously)

1- Anastomosed to stomach i.e. **cysto-gastrostomy**

2- If related to tail of pancreas treated by **distal pancreatectomy**

III- PANCREATIC NEOPLASMS

Endocrinal	Exocrinal
1- **Alpha** cells ➔ Glucogonoma.	**1- Benign :** Adenoma
2- **Beta** cells ➔ Insulinoma.	**2- Malignant :**
3- **Gamma** cells ➔ Somatostatinoma	(a) Adenocarcinoma.
4- **Non beta** cells ➔ Gastrinoma	(b) Cysta-adenocarcinoma

1- Insulinoma

DEFINITION

Endocrinal pancreatic tumor of **beta** cells secreting **insulin**.

CLINICAL PICTURE (**Whipple's triad**)

① An attack of hypoglycemia occurs on fasting
② Sweating, dizziness, blurring of vision & hunger sensation
③ The symptoms are relived by glucose.

TREATMENT

Enucleation of the tumor or **distal pancreatectomy**.

2- Gastrinoma

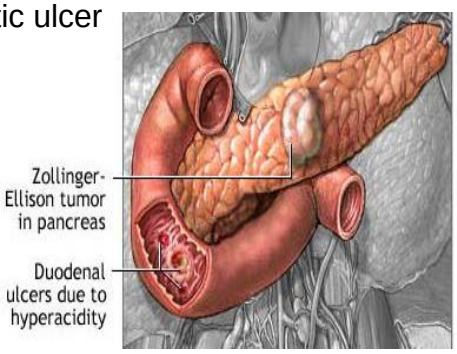
Zollinger Ellison syndrome

DEFINITION

Endocrinal pancreatic tumor of non **beta** cells secreting **gastrin**

CLINICAL PICTURE

Hypergastrinemia → ↑ HCL → intractable peptic ulcer & diarrhea.



INVESTIGATIONS

① **Gastric function test**
to estimate ↑ acid secretions.
② **Radio-immune assay**
to estimate ↑ serum gastrin.
③ **CT scan (abdomen)**
to localize the tumor.

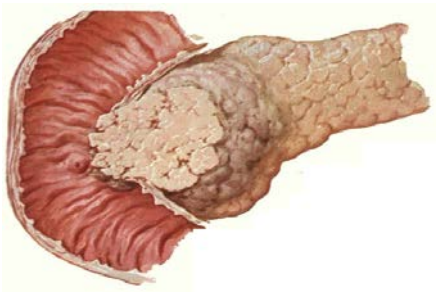
TREATMENT

Total gastrectomy (to abolish acid secretions)
+ **resection** of pancreatic tumor
+ trials by **omeprazole** "less effective"

3- Carcinoma of pancreas

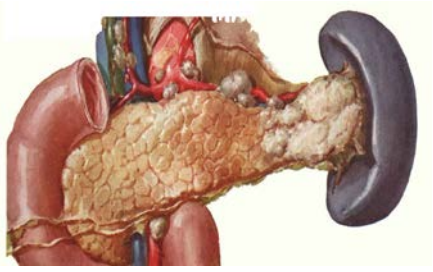
INCIDENCE

- It represents **3** % of all malignancies of the body
- **Age : 55** - **70** years
- **Sex :** Male **>** female



PREDISPOSING FACTORS

- Smoking & high (protein & fat) in diet.
- **Pancreatic calcinosis** from chronic pancreatitis.



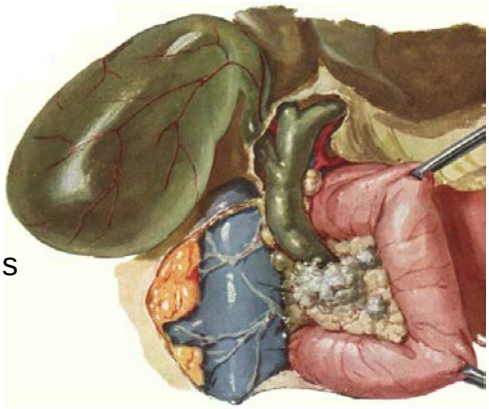
PATHOLOGY

Site - **Head** 60 % - **Body** & **tail** 40 %

N/E - Mass with central degeneration & necrosis.

M/E - **Adenocarcinoma**, (common)
& **cyst-adenocarcinoma** (less common)

SPREAD



Direct To CBD, duodenum, pyloric antrum, transverse mesocolon.....etc.

Lymphatic To regional L.Ns then to porta-hepatis then celiac L.Ns then para-aortic L.Ns then thoracic duct then Lt. supra-clavicular L.Ns (**Virchow's gland**).

Blood To the liver mainly.

Transperitoneal Malignant ascites (10%)

CLINICAL PICTURE

Type of patient (**> 50** years & Male > female)

Symptoms

1- Cancer head of pancreas :

- Progressive obstructive jaundice (**discuss**)

2- Cancer body & tail of pancreas :

- Dull pain at epigastric & radiate to the back, because of infiltration of celiac plexus.
- Athenia, anorexia, anemia & weight loss

Signs

1- Cancer head of pancreas :

- The patient is jaundiced & the gall bladder is distended according to **Courvoisier law**. (**discuss**)

2- Cancer body & tail of pancreas :

- Enlarged firm liver & rarely epigastric mass.
- Migrating thrombophlebitis (**Trousseau's sign**) of superficial veins 2^{ry} to increasing the coagulability factors

DIFFERENTIAL DIAGNOSIS

- Calcular obstructive jaundice (**discuss**)

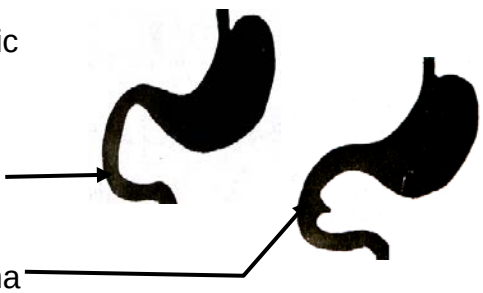
INVESTIGATIONS

A- Laboratory **Same as** obstructive jaundice (**discuss**)

B- Radiological

1- U/S & CT SCAN : The most diagnostic

2- BARIUM MEAL (DUODENOGRAPHY)



A- Widening of duodenal curve
i.e. cancer head pancreas

B- Inverted 3 shaped
i.e. peri ampullary carcinoma

3- ERCP :

⇨ **Diagnostic :** Shows dilated duct with localized narrowing.

⇨ **Therapeutic :**Through putting a **stent**

TREATMENT

A- Pre-operative management **Same as** obstructive jaundice (**discuss**)

B- Operative procedures

Operable

1- CANCER HEAD OF PANCREAS

PANCREATICO-DUODENECTOMY (**WHIPPLE OPERATION**)

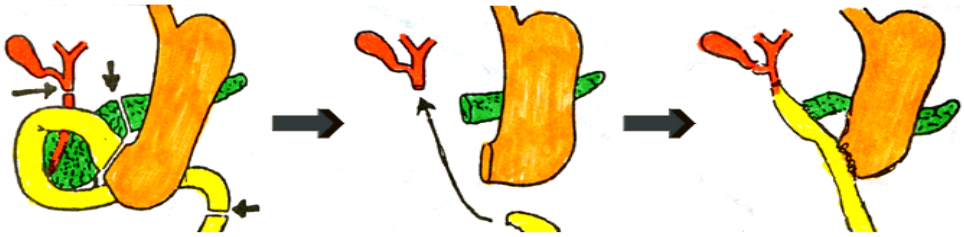
i.e. Removal of duodenum, lower part of CBD & head of pancreas.

Then restore the continuity by **the following anastomoses**

① The common bile duct to jejunum.

② The pancreatic duct to the jejunum.

③ The stomach to the jejunum .



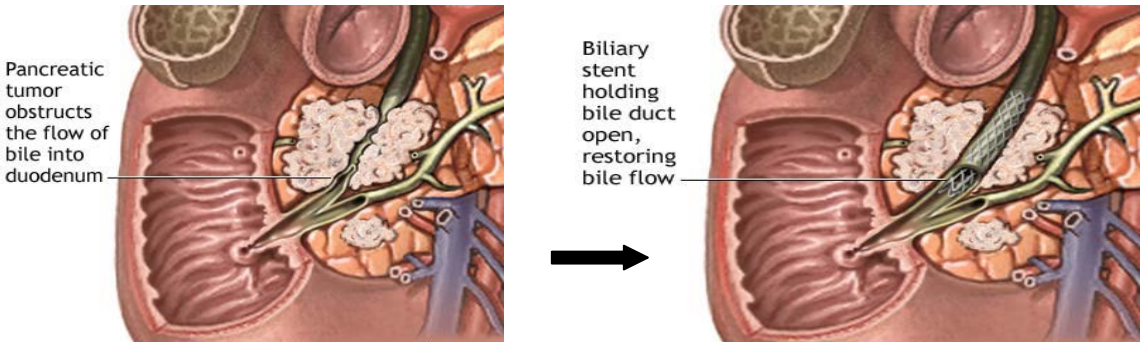
2- CANCER BODY & TAIL OF PANCREAS

SUBTOTAL PANCREATECTOMY leaving head only

Inoperable **Either** (A) **or** (B)

(A) CHOLECYSTO-JEJUNOSTOMY WITH JEJUNO-JEJUNOSTOMY

(B) INTERNAL BILIARY STENT



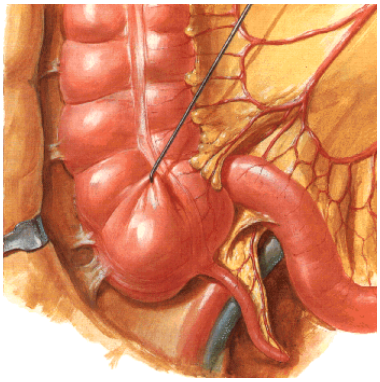
PROGNOSIS

5 year survival rate.

The Appendix

THE APPENDIX

SURGICAL ANATOMY



SITE Rt. iliac fossa

SHAPE Narrow worm like tube

LENGTH Variable from **2 - 5** inches (Average **10** cm)

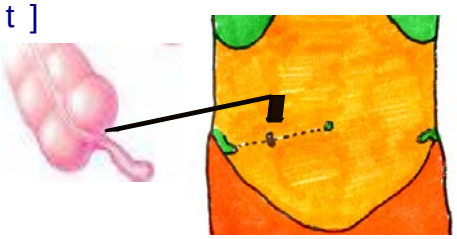
ORIGIN (**Base**)

It arises from the posteromedial aspect of the caecum, 1 inch below the Ileo-caecal junction.

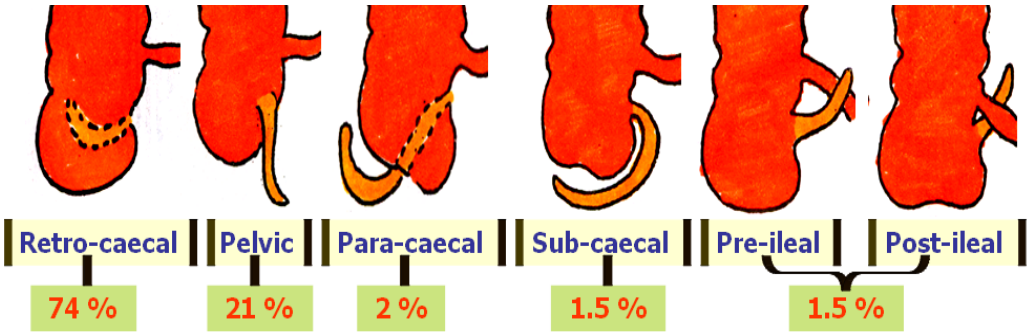
SURFACE ANATOMY

It is represented by a point [McBurney's point]

At the junction of **medial 2/3** with **lateral 1/3** of a line drawn from umbilicus & Rt. anterior superior iliac spine (A.S.I.S)

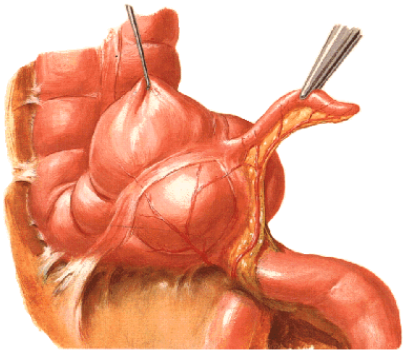


POSITIONS (**Variable**)



PERITONEAL COVERING (**Meso-appendix**)

It is completely covered by the peritoneal triangular fold. Sometimes the meso-appendix is absent & the appendix lies beneath the serosa of the caecum.



BLOOD SUPPLY

Arterial **Appendicular artery** from ileo-colic artery which is a branch of superior mesenteric artery

Venous **Appendicular vein** to ileo-colic vein to superior mesenteric vein to portal vein.

SURGICAL IMPORTANCE

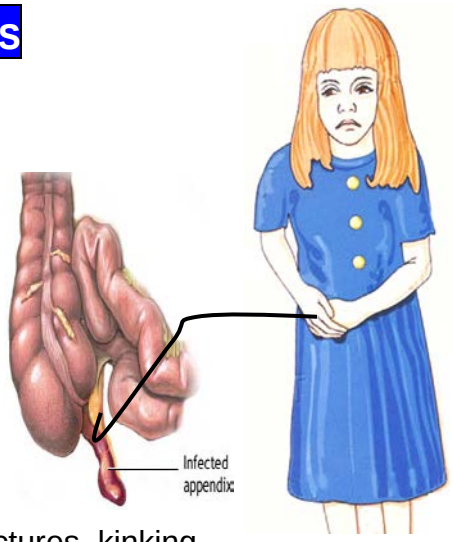
(1) Gangrene occur at tip, because it is far from blood supply

(2) Appendicitis can cause portal pyemia

(3) **Mc Burney's point** represent the base of appendix

(4) During appendicectomy we follow taenia coli as a guide to the appendix

ACUTE APPENDICITIS



INCIDENCE

The commonest cause of acute abdomen.

PREDISPOSING FACTORS

Age **RARE** in infants because of short appendix & wide base.

COMMON in children up to (**20** - **30**) years because of narrow base.

RARE AGAIN in elderly because of senile appendicular atrophy.

Obstructed appendix

May occur with Fecolith , strictures, kinking, ascaris, fruit seeds or any foreign bodies

PATHOLOGY

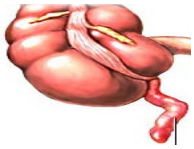
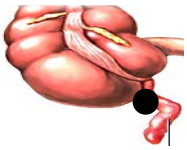
Organism E. coli & streptococcal faecalis.

Route of spread Direct from lumen & rarely blood.

Pathological stages

Acute catarrhal → acute suppurative → acute gangrenous

PATHOLOGICAL TYPES

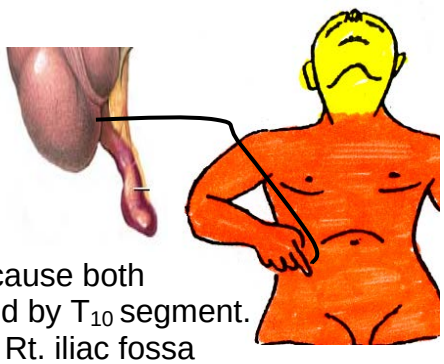
NON OBSTRUCTIVE TYPE	OBSTRUCTIVE TYPE
	
• **Slowly** developing inflammation. • **Less** common (**1/3** cases). • **Less** serious because of free drainage. • FATE - Infection starts in the **mucosa** then either **spontaneous** resolution with **slow** spread of infection with **little** incidence of suppuration & perforation. • RESULTING PERITONITIS is **localized** as perforation occurs very slowly **So** giving chance for localization	• **Rapidly** developing inflammation. • **More** common (**2/3** cases). • **More** serious as pus collects under tension i.e. empyema. • FATE - Infection starts in the **mucosa** then **no** resolution but **rapid** spread of infection to all with **high** incidence of suppuration & perforation. • RESULTING PERITONITIS is **generalized** as perforation occurs very rapidly **So no**. chance for localization.

N.B.: As the inflammatory process is very slow. The body defense has time to wall the inflamed appendix by adhesions with intestine & omentum

forming ⇨ **APPENDICULAR MASS** within 3 - 5 days. then any perforation

forming ⇨ **APPENDICULAR ABSCESS**

CLINICAL PICTURE



Types of patient Usually 20 - 30 years.

Symptoms

1- PAIN :

- **Onset :** Acute.
- **Site :** - Start at para-umbilical region because both umbilicus & appendix are supplied by T_{10} segment.
  - Later on it will be localized in the Rt. iliac fossa
- **Characters :**
  - At the onset, it is colicky if obstructive type or dull aching if non obstructive type i.e. **visceral pain**.
  - Later on, it becomes stabbing due to spread of inflammation to peritoneal coverings i.e. **somatic pain**.
  - This pain is aggravated by movement & cough.

2- ANOREXIA & NAUSEA are nearly always present.

VOMITING is always preceded by the pain.

3- CONSTIPATION may present. it may be the cause of obstruction.

Signs

GENERAL SIGNS

Fever (37.2 - 37.8) but if high fever it means complications

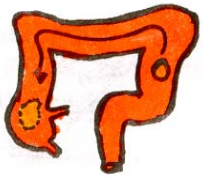
N.B.: Tachycardia is corresponding to fever

LOCAL SIGNS

- If the patient is asked to cough, the pain becomes sharp and well localized to site of appendix (**cough tenderness**)
- **Localized tenderness** and **rebound tenderness** over the Rt. iliac fossa, usually but not always, over McBurney's point

N.B.: P/R and P/V examination are essential to be done to differentiate gynecological problems from pelvic appendicitis.

- ROVSING'S SIGN (**Crossed tenderness**) pressure on Lt. iliac fossa causes pain in Rt. iliac fossa due to displacement of gas inside the colon to caecum & inflamed appendix distending them.

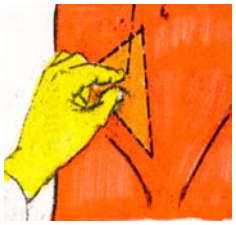


N.B.: SHERREN'S TRIANGLE of hyperesthesia :

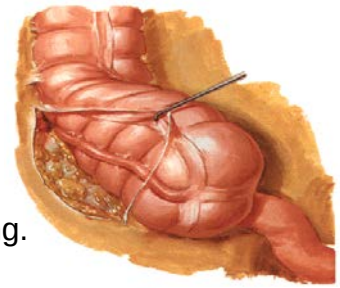
It is a Δ bounded by

- Symphysis pubis.
- Umbilicus.
- Rt. ASIS.

It disappears after perforation.

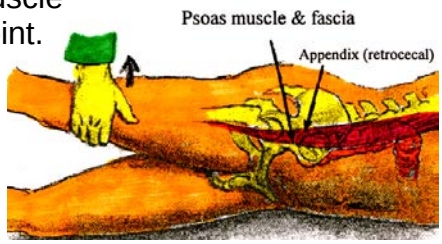


ATYPICAL CLINICAL FEATURES



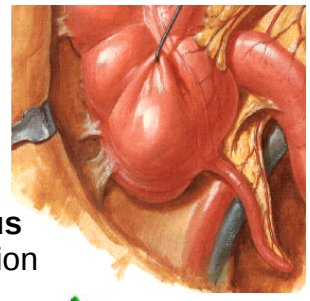
1- Long retro-caecal appendix

- **Tenderness** is felt in the Rt. loin.
- **IF** the appendix touches the **ureter**
  ➔ Pain simulating ureteric colic which will be misleading.
- **IF** the appendix comes in contact with **psoas** muscle
  ➔ Psoas spasm, this will lead to flexion of hip joint.



SO **Hyperextension** of the hip leads to abdominal pain.
which is called **PSOAS SIGN**

2- Pelvic Appendix



- **Tenderness** is felt only by P/V or P/R.
- **IF** the appendix touch the **rectum** or **bladder**
  ➔ Irritation which leads to tensmus or dysuria both will be misleading.
- **IF** the appendix comes in contact with **Obturator internus** muscle ➔ Obturator internus spasm, this will lead to flexion & external rotation of the Rt. hip joint.

SO **Flexion** & **internal rotation** of Rt. hip leads to abdominal pain
which is called **OBTURATOR SIGN**

3- Sub-hepatic appendix

- The tenderness is at a higher level than McBurney's point.

4- Appendicitis with infants & young children

- **usually serious due to**
  ① Difficult to be examined.
  ② Greater omentum is small & so localization is very difficult.
  ③ There is prominent vomiting so wrongly diagnosed as gastro-enteritis.

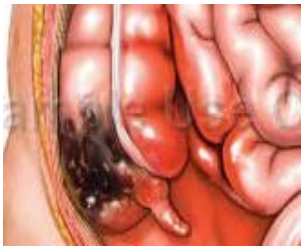
5- Appendicitis with elderly

- **usually serious due to**
  ① Difficult to be examined because of little tenderness & rigidity
  ② Atherosclerosis which leads to early thrombosis & gangrene
  ③ Natural weakness of Immune system.

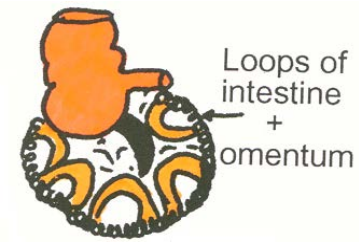
6- Appendicitis with pregnancy

- **usually serious due to**
  ① The site of pain is displaced upwards as pregnancy progresses.
  ② The localization by omentum is less efficient.
  ③ The condition is often misdiagnosed as pyelitis.

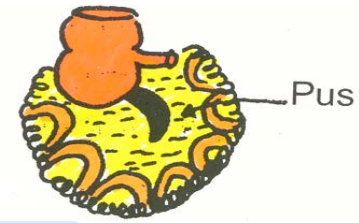
CLINICAL PICTURE OF COMPLICATIONS



1. PICTURE OF PERITONITIS :
   - **Inspection** : Restricted movement with respiration.
   - **Palpation** : Generalized rigidity & tenderness.
   - **Percussion** : Shifting dullness (if pus)
   - **Auscultation** : may be dead silent abdomen.
2. APPENDICULAR MASS :
   - History of **2** – **3** days.
   - Constitutional symptoms with fever **> 38°**C.
   - **Firm** tender mass in Rt. iliac fossa with overlying muscle rigidity.

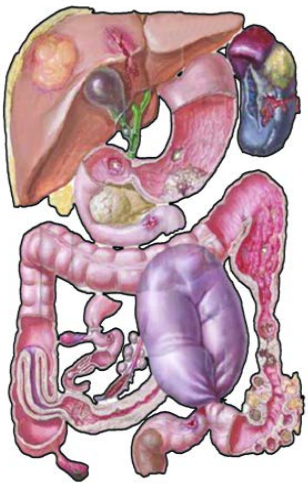


3. APPENDICULAR ABSCESS :
   - History of **few** days.
   - Constitutional symptoms with **hectic** fever
   - **Cystic** tender mass in Rt. iliac fossa with overlying muscle rigidity.



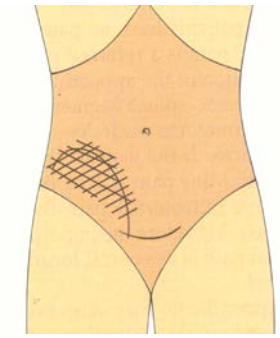
N.B.: IF the abscess is pelvic, It is best felt by P/R & P/V

DIFFERENTIAL DIAGNOSIS



I- Other causes of acute abdomen

1. GROUP I (**Thoracic problems**)
   e.g. Rt. sided pneumonia or Rt. basal pleurisy
2. GROUP II (**Upper abdominal problems**)
   e.g. Perforated peptic ulcer or Acute cholecystitis.
3. GROUP III (**Lower abdominal problems**)
   e.g. Intestinal obstruction, Meckel's diverticulitis , Crohn's disease or cancer caecum
4. GROUP IV (**Pelvic problems**)
   e.g. Ruptured ectopic pregnancy, tubal abortion , twisted ovarian cyst or pelvic Inflammatory disease
5. GROUP V (**Urological problems**)
   e.g. Ureteric colic, pyelonephritis.
6. GROUP VI (**Neurogical problems**)
   e.g. Herpes zoster



II- Other causes of mass in Rt. iliac fossa

① Appendicular abscess.
② Carcinoma of the caecum.
③ Crohn's disease of terminal ileum.
④ Hyperplastic ileo-caecal T.B .
⑤ Rt. iliac lymphadenitis.

INVESTIGATIONS

A. Laboratory investigations :

- **Total leucocytic count :** Increased up to (**10.000** - **16.000** U.L)
  However normal count doesn't rule out acute appendicitis.

- **Urine analysis :** The presence of (**R.B.Cs** or **pus cells**) raises the suspicion of a ureteric stone or urinary tract infection.

B. Abdominal ultrasound :

May help in diagnosis, but helpful in female with suspected gynecological problems.

C. Laparoscopy : Diagnostic & therapeutic laparoscopic surgery.

e.g. appendicitis or ovarian cyst can be managed laparoscopically.

TREATMENT

A. Non complicated acute appendicitis

i.e. **No** (peritonitis, appendicular mass or abscess)

The treatment is appendectomy either open or laparoscopic

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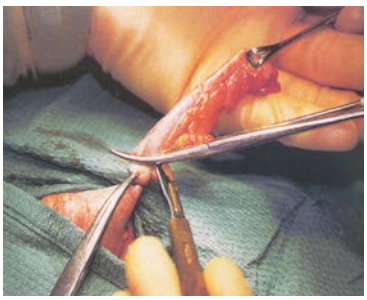
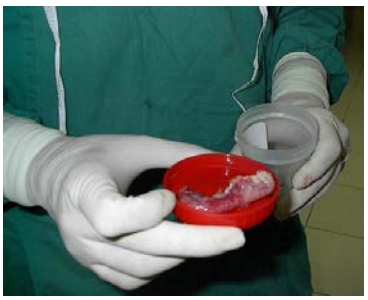




B. Complicated with peritonitis

Urgent surgery ; open appendectomy + peritoneal toilet (lavage)

C. Complicated with appendicular mass or abscess

APPENDICULAR MASS

Urgent appendectomy is **not** performed because ⇩

① The mass represents success of body to isolate the danger so it is better to be left undisturbed.

② Appendectomy is difficult with increased morbidity.

③ Appendectomy is carrying hazards of injuring the intestine.

SO **Initial conservative [Ochner – Sherren] treatment :**

① Rest in bed & Ryle's tube (no oral intake)

② I.V fluid & I.V antibiotics as a combination of ⇩
Ampicillin, **Aminoglycosides** & **Metronidazole**

③ **Good observation** for vital signs (Temp, pulse, ABP & RR), size of mass & degree of tenderness .

IN **80** – **90** % of cases on conservative ttt ➔ Resolution of the mass

THEN " **interval appendectomy** " is done **3 - 6** months later.

APPENDICULAR ABSCESS

The pus should be drained by open surgery or ultrasound percutaneous aspiration.

SO Open surgery :

① **If the abscess in the pelvis**
It will be drained through the rectum

② **If the abscess in the Rt. iliac fossa**
It will be drained through an incision of iliac muscle, then a rubber drain is left.

THEN " **interval appendectomy** " is done **3 - 6** months later.

CHRONIC APPENDICITIS

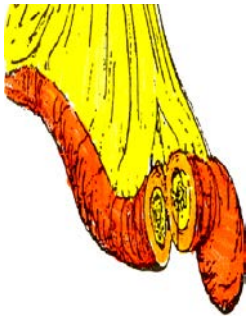
- **It is better** to called **recurrent subacute appendicitis**.
- **DD** from colitis or irritable bowel.syndrome
- **The treatment** is Appendicectomy

TUMORS OF THE APPENDIX

1. Carcinoid tumor

Argentaffinoma

PATHOLOGY



- Site : Mainly at distal **1/3** of the appendix.
- N/E : **Golden yellow** due to its lipid content.
- M/P : Arises in **Kulchitsky cells** in the depth of mucosal pits.
- Benign behavior but in minority of cases, the tumor invades & metastasizes to the liver.

TREATMENT

- If < 2 cm = Appendicectomy.
- If > 2 cm = Rt. hemicolectomy

2. Carcinoma

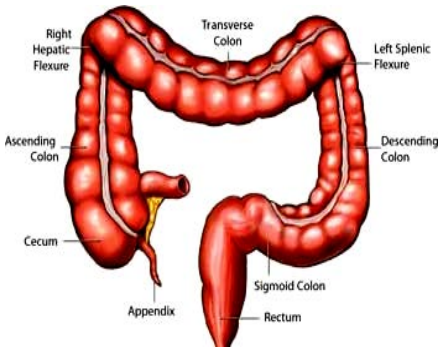
Adenocarcinoma

- **It may present as** acute appendicitis.
- **The tumor behaves** as carcinoma of the colon.
- **The treatment** is Rt. hemicolectomy.

Small & large intestine

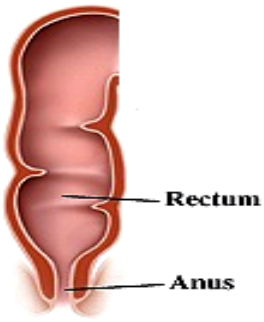
SMALL & LARGE INTESTINE

1- ANATOMY OF THE COLON



- **Begins** At the end of the ileum.
- **Ends** By the rectum.
- **Parts**
  - 1- Caecum
  - 2- Ascending colon
  - 3- Hepatic flexure
  - 4- Transverse colon
  - 5- Splenic flexure
  - 6- Descending colon
  - 7- Pelvic (Sigmoid) colon

2- ANATOMY OF THE RECTUM



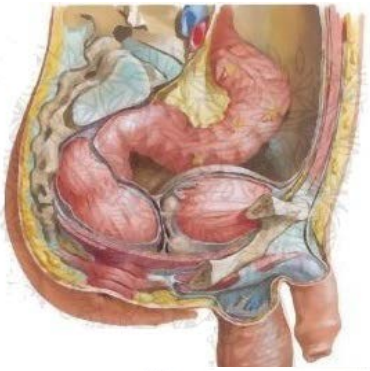
- **Begins** As the continuation of sigmoid colon
- **Ends** By the anal canal.
- **Length** **5** inches (**12.5** cm)
- **Flexures**

1- ANTERO-POSTERIOR FLEXURE It is **concave** anteriorly

2- LAT. FLEXURES It presents a **3 concavities,**

- the upper & lower are concave to the left while the middle to the right.

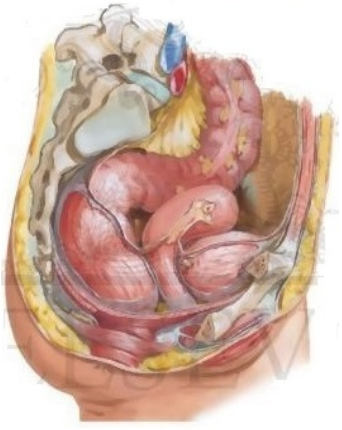
- **Peritoneal coverings**
  - **a. Upper 1/3 :** covered in front & sides.
  - **b. Middle 1/3 :** covered in front only.
  - **c. Lower 1/3 :** Not covered.



- **Relations**

1. POSTERIOR

Sacrum, coccyx, pyriformis, levator ani, sacral plexus, sympathetic chain, median sacral a., lat. sacral a. & sup. rectal vessels.



2. ANTERIOR

a. In males :

- Upper 2/3 : Loops of small intestine
- Lower 1/3 : base of urinary bladder, prostate, seminal vesicle, vas & ejaculatory ducts.

b. <u>In females:</u>

- Upper 2/3 : Loops of small intestine
- Lower 1/3: Vagina.

3. LATERAL

- Upper 2/3 : Loops of small intestine.
- Lower 1/3 : Levator ani.

✷ Surgical importance

(1) **Superior rectal veins** with 3 tributaries at 3, 7 & 11(site of mother piles)

(2) **Fascia of Denonvilliers** (between rectum & prostate)
delay the spread of cancer

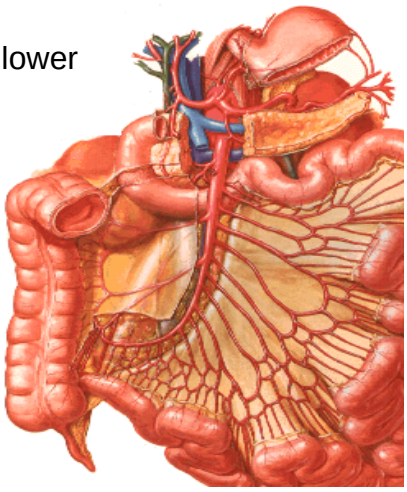
Arterial supply of colon & rectum

I. Arterial supply of the Colon

I- Superior mesenteric artery (The artery of midgut)

Origin

- **From** the front of abdominal aorta at the level of lower border (L_1)



Course & relations

- **At its origin** it lies behind the pancreas then crosses in front of uncinate process.
- **It enters** the root of mesentery, passes downwards & to the right, in front of **3rd** part of duodenum, I.V.C. and Rt. psoas major muscle.

End

- **In** Rt. Iliac fossa by anastomosing with ileal branches of ileo-colic artery.

Branches

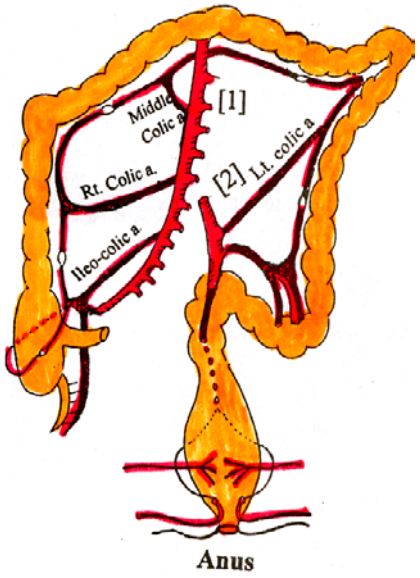
1- ILEO-COLIC ARTERY Which gives

① **Ileal** branches to anastomose with the end of superior mesenteric artery

② **Appendicular** branch to appendix

③ **Ant.** & **post, caecal** branches to caecum

④ **Ascending** branch to anastomose with descending branch from Rt. colic artery.



2- RT. COLIC ARTERY Which gives

① **Descending** branch which anastomose ascending branch of ileo-colic artery

② **Ascending** branch which anastomose Rt. branch of middle colic artery.

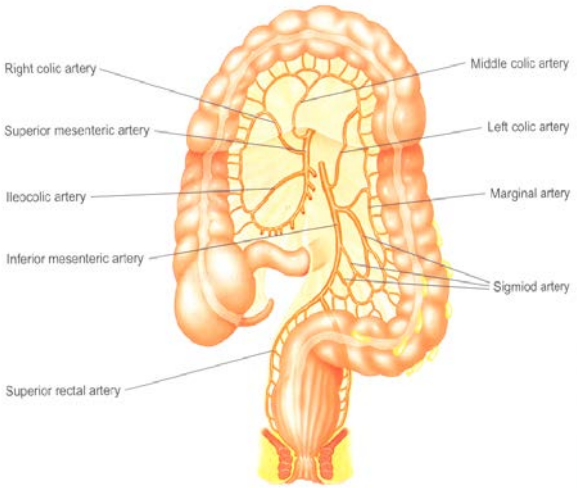
3- MIDDLE COLIC ARTERY Which gives

① **Rt. branch** which anastomose ascending branch of Rt. colic artery.

② **Lt. branch** which anastomose upper branch of Lt. colic artery.

II- Inferior mesenteric artery

(**The artery of hindgut**)



Origin

- **From** the front of abdominal aorta at the level of (L_3)

Course & relations

- **At its origin** it lies behind the **3rd** part of duodenum.
- **It runs** downwards & to the Lt.

End

- **By becoming** superior rectal artery

Branches

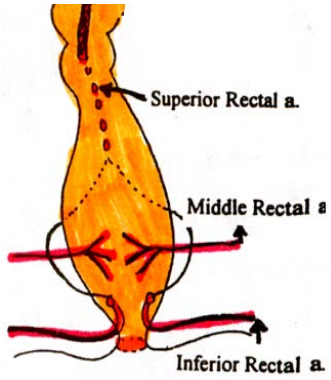
1- LT COLIC ARTERY Which gives

① **Upper branch** which anastomose Lt. branch of middle colic artery.

② **Lower branch** which anastomose to upper sigmoid arteries.

2- SIGMOID ARTERIES (**3** or **4**) arteries which supply sigmoid colon.

II. Arterial supply of the Rectum



I- Superior rectal artery

As continuation of **inferior mesenteric artery**.

II- Middle rectal artery

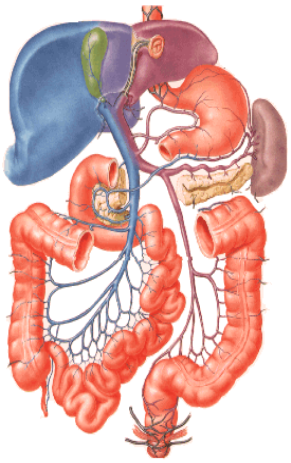
From **internal iliac artery**.

III- Inferior rectal artery

From **internal pudendal artery**.

2. Venous drainage

I. Venous drainage of the Colon



I- Superior mesenteric vein

Anastomose with **splenic vein** to form **portal vein**.

II- Inferior mesenteric vein

end with **splenic vein**

II. Venous drainage of the Rectum

I- Superior rectal vein

Corresponding to **inferior mesenteric artery** to **portal circulation**.

II- Middle & inferior rectal vein

Corresponding to **arteries** to **systemic circulation**.

3. Lymphatic drainage

See Colo-rectal carcinoma

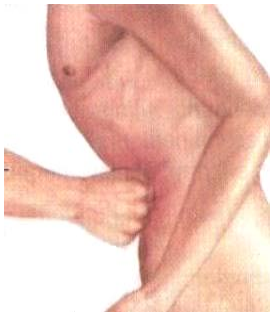
I- INTESTINAL TRAUMA

PREDISPOSING FACTORS

Pathological disease of intestine

EXCITING CAUSE

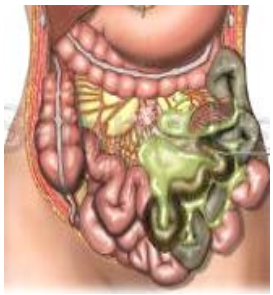
"**Trauma**" which may be ⇩



- Closed ① **Direct** trauma e.g. blunt trauma.
  ② **Indirect** trauma e.g. fracture pelvis
  ③ **Spontaneous** rupture e.g. diseases of intestine
- Opened ① Gunshot wounds.
  ② Punctured wounds due to stabbing .
  ③ Operative

PATHOLOGY

Colon surgery are **more dangerous than** those of the small intestine because of 1- The highly infective content of both aerobic & anaerobic organism
2- Constant gaseous distention .
3- Incomplete serous coat.



COMPLICATIONS (**Sequelae**)

- **Peritonitis :** Septic shock due to escape of intestinal contents into the peritoneum
- **Internal hemorrhage** i.e. hypovolemic shock
- **Paralytic ileus**

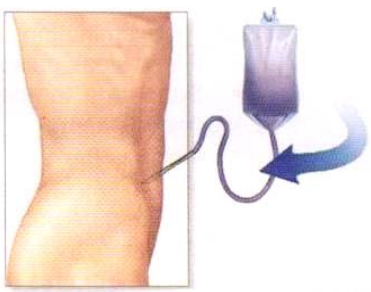
CLINICAL PICTURE

- History of trauma to abdomen followed by an abdominal pain + shock.
- Examination **:** May reveals signs of internal hemorrhage & shock + signs of external trauma as bruises.

INVESTIGATIONS

1. Sonar & CT scan (**abdomen**)
" - The most "**diagnostic**

2. Diagnostic peritoneal lavage "D.P.L."



- Done by inserting an intra-peritoneal catheter at umbilicus under local anesthesia & infusing **500 cc** saline in the peritoneal cavity. if saline returns bloody = intra-peritoneal bleeding.

TREATMENT

A. Anti-shock measures

- Remember **ABCDE**
- Blood transfusion, warmth, morphia ……etc.

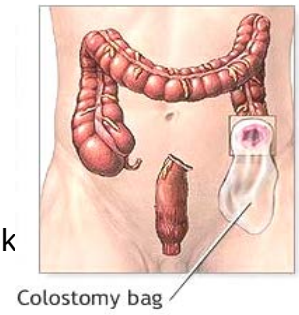
B. Immediate laparotomy "The priority is to arrest bleeding"

1- Small intestine & Rt. colon injuries

- **Sharp injuries :** Suturing in **2** layers.
- **Lacerated injuries :** Excise the edges & suturing.
- **Intestinal resection & 1^{ry} anastomosis :**
  ⇨ Indicated with ☟
  ① Ischemic or gangrenous segments.
  ② Extensively contused segments.
  ③ Multiple tears those are very close to each other.

2- Transverse & Lt. colon injuries

- The tear is act as a proximal colostomy with distal closure then preparation of colon & closure after **1** week i.e. **Hartmann's procedure** ⟶



PRINCIPLES OF COLON SURGERY

Colonic anastomosis is more liable to disruption, leakage and peritonitis more than small intestine because

1- The highly infective content of both aerobic & anaerobic organism
2- Constant gaseous distention .
3- Incomplete serous coat.

So Pre-operative preparation must be done

A Improving nutritional status of the patient

B Bowel preparation

Mechanical preparation

① Non residue diet **4** days before operation

② Enemas & mild laxatives **2** - **3** days before operation.

or ③ **Rapid preparation 1** day before operation by ☟
- Whole gut irrigation using 2 - 4 L/hour of saline passed via a nasogastric tube until the patient passes clear fluid per rectum.

Chemical preparation

① **Intestinal antiseptics** (neomycin + metronidazole) orally **2** days before operation.

② **At the time of anesthesia**
I.V Cephalosporins or aminoglycosides + metronidazole.

II- INTESTINAL FISTULAE

DEFINITION

A fistula is an abnormal communication between 2 surfaces.

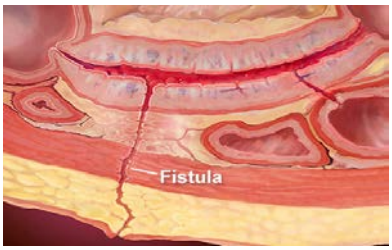
CLASSIFICATION

⇨ They classify into **Internal** (when connect to Hollow Viscera)
or **External** (when connect to skin)

⇨ Also External fistula classified into
Low output fistula that discharge < 500 ml/d.
or **High** output fistula that discharge > 500 ml/d.

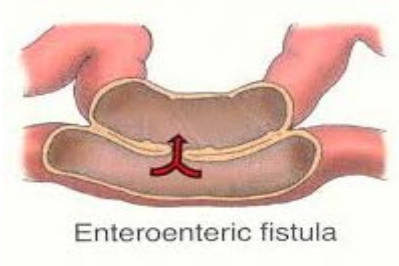
AETIOLOGY

After abdominal operations (80%)



1. Failure of anastomosis
   because of poor vascularity .
2. Anastomosis under tension .
3. Distal obstruction .

Other causes (20%)


Enteroenteric fistula

1. Congenital: patent vitello-intestinal duct .
2. Traumatic: abdominal injuries .
3. Inflammatory: Crohn's disease .
4. Neoplastic: Malignant tumors.

COMPLICATIONS

1. **Metabolic effects :**
   Malnutrition, dehydration, hypo-albuminaemia, acid-base, and electrolyte disturbances such as hyponatraemia and hypokalemia.
2. **Sepsis**
   Sepsis is the major problem
3. **Irritation and maceration of the skin**
   due to continuous flow of intestinal contents.

TREATMENT

1. Resuscitation & nutritional support :
   The priority is to save life of the dehydrated patient by I.V fluids.
2. Skin protection :
   Should be started early by a disposable collection bag.
3. Definitive treatment:

- **External fistula:**
  - **Continued conservative treatment** with nutritional support
  - **Surgical intervention** is indicated if No improvement.
- **Internal fistula:**
  - **Spontaneous** closure is rare to occur
    & many fistulae don't require correction
  - **Surgical intervention** is indicated if.nutritional problems.

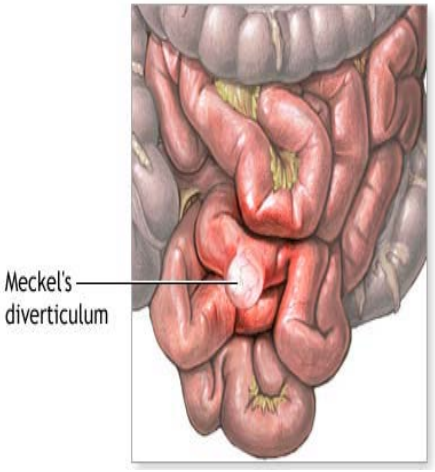
III- INTESTINAL DIVERTICULUM

Diverticulum is a blind pouch that is continuous with the lumen of a hollow viscus

1. Meckel's Diverticulum

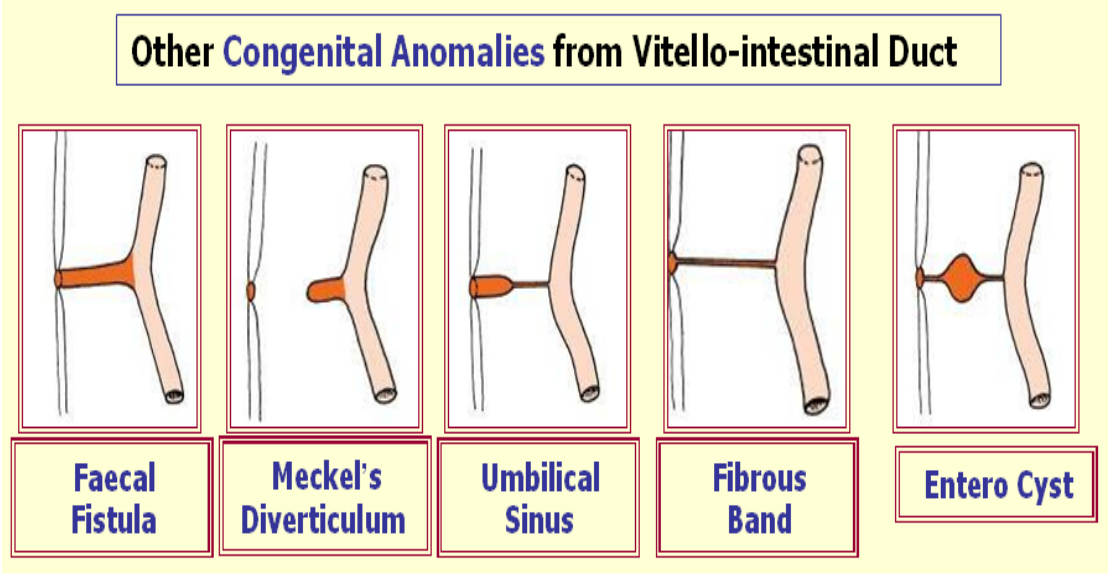
DEFINITION

Persistent patency of the proximal part of Vitello - intestinal duct of embryo.



N.B. Vitello -intestinal duct :

- It is a duct present in **4th** weeks joining the yolk sac & primitive gut then disappear **6th** week of intra-uterine life.
- **Obliteration starts** at umbilical end & so intestinal end is the last part.



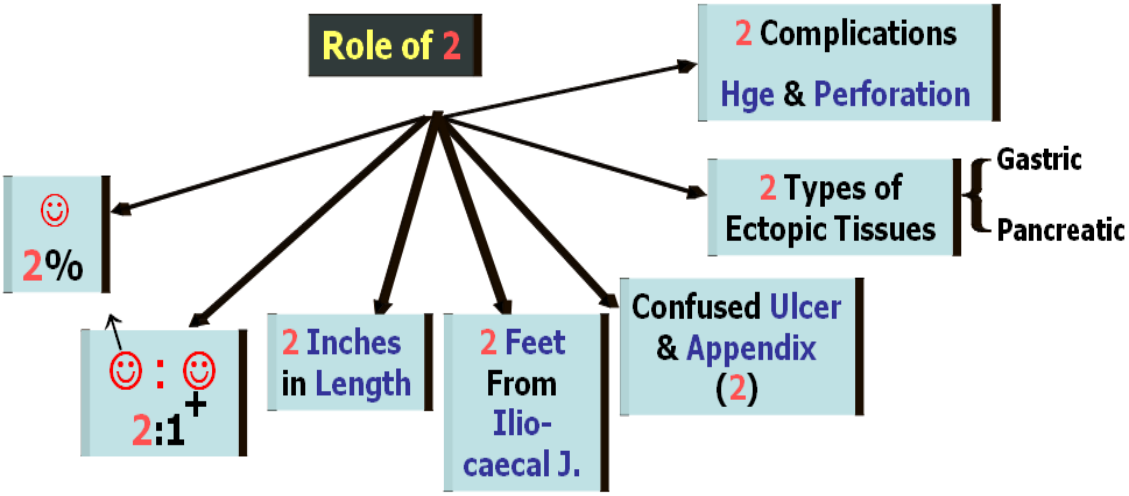
PATHOLOGY

- It is a **true** diverticulum i.e. consists of all layers of bowel wall.
- It projects from **Anti**- mesenteric borders.
- It may contain **2 ectopic tissues :** Gastric & pancreatic.
- It may give **2 major complications :** Inflammations & hemorrhage.

INCIDENCE

Rule of (2)

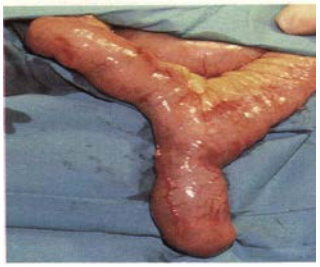
- It occur in **2 %** of human. & it is symptomatic in **2 %** of patient only.
- It is about **2 inches** in length.
- It project **2 feet** from ileo - caecal junction



CLINICAL PICTURE

It may be accidentally discovered at operation for another pathology, or may present by one of its complications.

COMPLICATIONS (Occur in about 4 % of cases)



1. Acute diverticulitis :

Simulating to acute appendicitis.

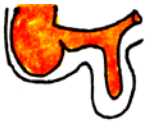
So clinically impossible to differentiate between the **2** conditions.

2. Bleeding per rectum :

From ectopic gastric epithelium i.e. peptic ulceration

3. Intestinal obstruction :

May be due to ⇩



① Inclusion as femoral hernia ➔ **Littre's hernia**

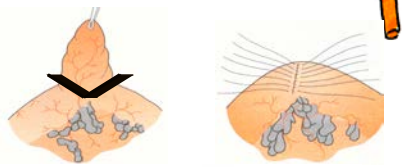
② Swollen inflamed base ➔ **Intussusception**

③ Band between apex of diverticulum & umbilicus ➔ **Volvulus**

INVESTIGATIONS

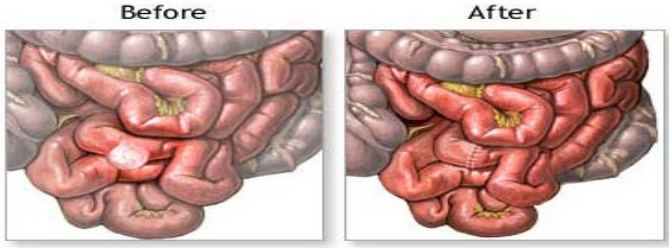
Tc^{99} can localize diverticulum containing gastric mucosa.

TREATMENT



A. Symptomatic cases.

⇨ **Resection** is indicated.



B. Accidentally discovered at laparotomy

⇨ **Resection** is indicated especially with children & young adults and in those with an attached band.

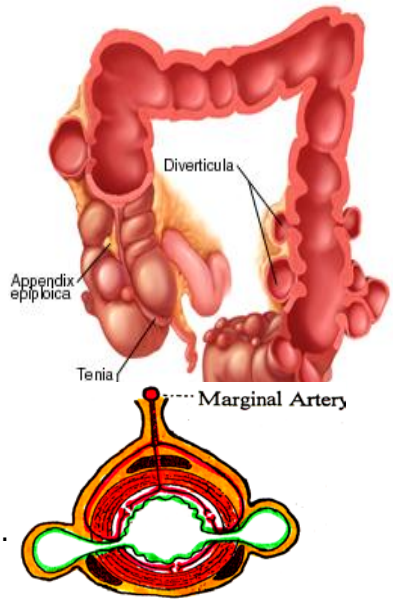
⇨ **In patients > 40 years :** It is better not to resect it & give the patient card that he has Meckel's diverticulum.

2. Diverticular disease of the colon

AETIOLOGY

Supposed to be lack of fibers in diet ➔ colonic constipation ➔ ↑ intra-luminal colonic pressure ➔ ↑ muscle spasm & segmentation of the colon.

SO The mucosa is pushed outwards through the defect in muscle coat.



PATHOLOGY

Nature It is a **pulsion diverticula** of the colonic mucosa through the circular muscle layer at the points of entery of blood vessels between the taenia coli.

Site ● The **sigmoid colon** is the commonest site affected but any area of colon may be involved.

● The rectum is **not** affected.

CLINICAL PICTURE

A. Uncomplicated diverticulosis coli = Irritable colon

① Distention & flatulence.

② Pain in lt. iliac fossa.

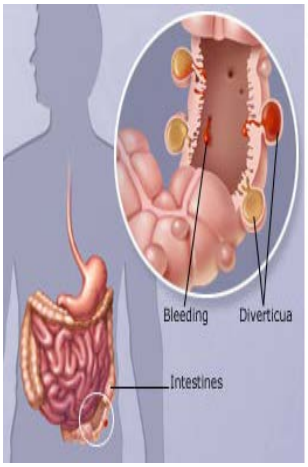
B. Complicated diverticulosis coli

1. Acute diverticulitis

MECHANISM

Obstruction of the mouth of one or more diverticula by fecal plug or inflammatory edema ➔ retention of contents ➔ infection ➔ diverticulitis.

C/P Simulate to acute appendicitis but on lt. side



N.B: **Perforation of acute diverticulitis** leads to either ⇩

① **Localized** peritonitis: i.e. peri- colic abscess.

② **Generalized** peritonitis with **50 %** mortality rate.

2. Chronic diverticulitis

Long history of recurrent attacks of pain with passage of **blood** per rectum. On palpation there is **tender mass** in the lt. iliac fossa. This mass has to be differentiated from carcinoma.

3. Bleeding

The proximity of marginal artery to the diverticulum may predispose to bleeding which is **bright red** & **massive**.

COMPLICATED BY FISTULA FORMATION Which may be ⇩

1- External :

Colo- cutaneous after drainage of peri- colic abscess.

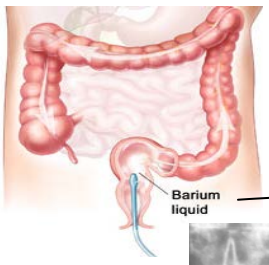
2- Internal :

Colo- vesical or colo- vaginal or colo-enteric fistula.

DD From other causes of bleeding per rectum

The commonest **3** causes are

① Anal conditions
② Diverticulosis coli
③ Ulcerative colitis

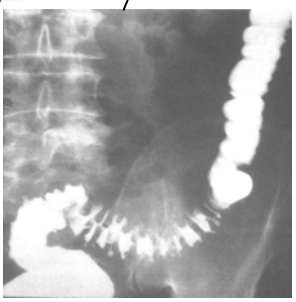


INVESTIGATIONS

A- Barium enema

- **In prediverticular stage :** A **saw tooth** appearance of colon is present.

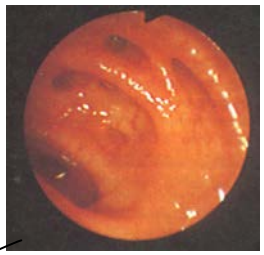
Then fully developed diverticulae will be visualized.



N.B.: Barium enema is contraindicated with acute diverticulitis because it predisposes to perforation.

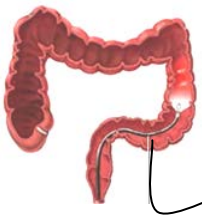
B- CT scan

- The best investigation in acute diverticulitis



C- Sigmoidoscopy

- It shows the mouth of diverticulae, also it has an important value to diagnose any concomitant lesion.



D. Mesenteric angiography

- It consider the most useful investigation to locate the site of bleeding.

TREATMENT

Uncomplicated diverticulitis

High fiber in diet, laxative & antispasmodics

Complicated diverticulitis

1. Acute diverticulitis : Treated conservatively on the same principles as appendicular mass
2. **Acute diverticulitis with** peri-colic abscess : Incision & drainage.
3. **Acute diverticulitis with** generalized peritonitis : **Urgent laparotomy** & resect the perforated colon by **Hartman's procedure** then peritoneal toilet & drainage

N.B.: Hartman's procedure

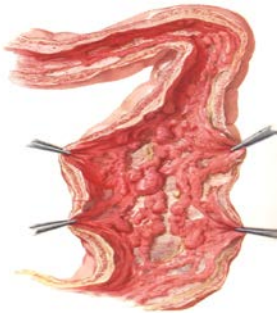
1st stage : Resection of sigmoid colon then proximal colostomy & distal closure.

2nd stage : Restoration of the continuity by anastomosis again

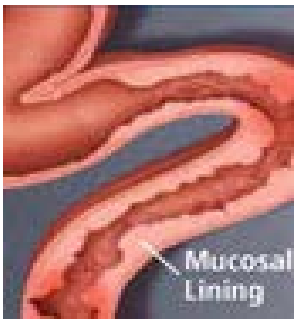
4. Chronic diverticulitis : **Colectomy** after adequate preparation.
4. Bleeding : Resuscitation (usually the bleeding stops) If failed **colectomy** after adequate preparation.

IV- INFLAMMATORY BOWEL DISEASE

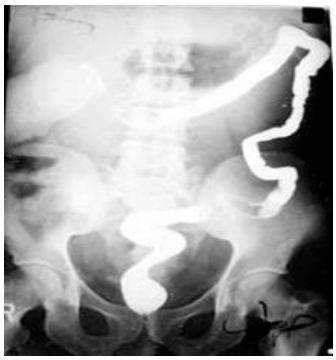
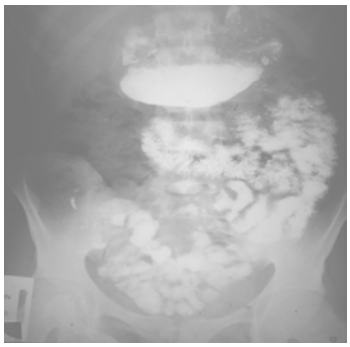
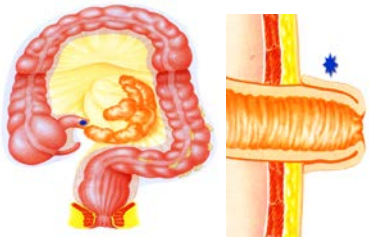
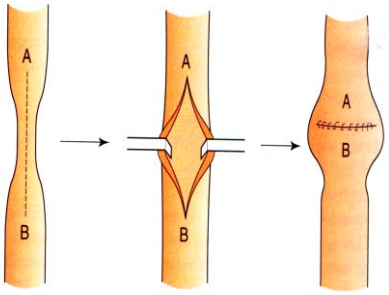
1. Ulcerative colitis (Procto-colitis)



2. Crohn's disease (Regional enteritis)

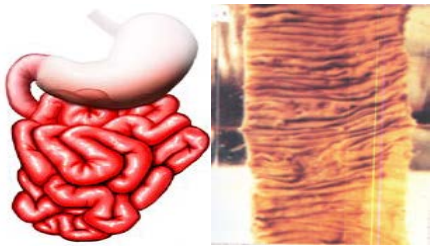


	1. Ulcerative colitis (Procto-colitis)	2. Crohn's disease (Regional enteritis)
DEFINITION	**Non** specific **ulceration**	**Non** specific **granulomatous** inflammation
AETIOLOGY	**Unknown.** may be immunologic or genetic or environmental.	**Unknown,** may be hereditary focal ischemia
PATHOLOGY	Ulceration affect mucosa & submucosa of colon & rectum **(procto-colitis)** or rectum alone **(proctitis).**	Inflammation affect all layers of G.I.T. mainly intestine **(distal ileum)**
C/P		
- Age	• **3rd** - **4th** decades.	• **2nd** - **4th** decades.
- Sex	• Female **>** Male.	• Female **=** Male.
- Presentation	• Watery diarrhea mixed with (blood, pus & mucus) with tenesmus	• Diarrhea with **mass** at Rt. iliac fossa.
	• Weight loss & dehydration	• Weight loss & dehydration
COMPLICATIONS		
- Intestinal	① Toxic megacolon (**fatal**)	① Mal-absorption (**extensive**)
	② Hemorrhage.	② Abscess & fistula to another bowel or skin.
	③ **Cancer colon :** If pancolitis **>10** years.	③ Peri-anal abscess, anal fistula & fissure.
	④ Stricture formation ➔ intestinal obstruction.	④ Stricture formation ➔ intestinal obstruction.
- Extra-intestinal	**Arthritis, cholangitis, liver cirrhosis ,** **skin lesion & finger clubbing.**	

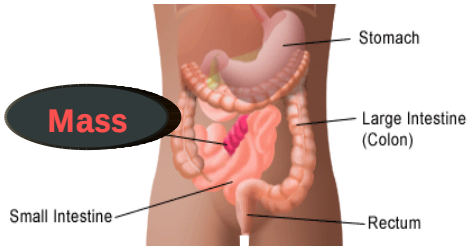
	1. Ulcerative colitis	2. Crohn's disease
	(Procto-colitis)	**(Regional enteritis)**
		
INVESTIGATIONS	• **Barium enema :** Shows colonic shortening & loss of haustrations " **Pipe stem** "	• **Barium meal and follow through** shows segmental areas of stricture & narrowing at terminal ileum " **String sign of kantor** "
TREATMENT		
A- Medical	① Correction of anemia & hypoproteinemia. ② **Corticosteroids** (systemic or by enema) ③ **Antibiotics** (acute disease) sulphasalazene or flagyl ④ **Antispasmodics** for pain	
B- Surgical	① Total proto-colectomy + terminal ileostomy.  ② Total colectomy + ileo-rectal anastomoses with regular endoscopic follow up of the rectum. ③ Total colectomy + rectal mucosectomy + ileo-anal anastomoses.	① Localized resection of the affected loop. ② **If strictures occur :** we will do stricturoplasty i.e. incise longitudinally & closed transversely. 

V- TUBERCULOSIS OF THE INTESTINE

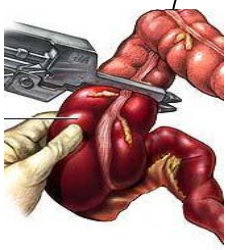
1. Ulcerative T.B (Enteritis)



2. Hyperplasic (Ileo-caecal T.B)



	1. Ulcerative T.B (Enteritis)	2. Hyperplasic (Ileo-caecal T.B)
AETIOLOGY	**2ry** to pulmonary T.B	**1ry** due to ingestion of infected milk
PATHOLOGY	Multiple ulcers in terminal ileum. The long axis of the T.B ulcers lies **transversely**.	Marked thickening of the intestinal wall mainly the **ileo-caecal** region.
C/P	• Attacks of diarrhea, weight loss & abdominal pain. • Intestinal obstruction from stricture.	• Attacks of diarrhea, weight loss & abdominal pain. • **Mass** in Rt. iliac fossa
COMPLICATIONS	① Intestinal obstruction ② Perforation (rare).	① Intestinal obstruction ② Abscess & fistula (rare)
INVESTIGATIONS	CBP, ESR, tuberculin test, chest x-ray & sputum analysis	
TREATMENT	① Anti-T.B. drugs ② Resection & anastomoses if stricture occur.	① Anti-T.B. drugs. ② **Rt. hemi-colectomy** if obstructed lesions occur.



VI- TYPHOID & PARATYPHOID

SURGICAL COMPLICATIONS

1. **Paralytic Ileus** (The commonest)
2. **Intestinal hemorrhage**.
3. **Perforation of typhoid ulcer**
4. **Cholecystitis** (non calcular)
5. **Phlebitis :** especially common iliac vein.
6. **Genito-urinary** e.g. cystitis.
7. **Arthritis & osteomyelitis**.

VII- INTESTINAL BILHARZIASIS

Bilharzial colitis

INCIDENCE

- **Common** in **endemic** area " **Egypt** "
- **Age :** Common with young or adult
- **Sex :** Male **>** female.

AETIOLOGY

Schistosoma **mansoni** & less commonly schistosoma hematobium

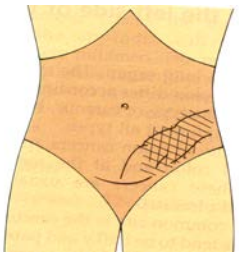
PATHOLOGY

- Site : Sigmoid colon & rectum.
- chronic inflammatory cells surround the deposited ova leads to formation of Bilharzial granuloma.
- Types : **2 Forms**

1. SUBMUCOUS FORM

- **The mucosa** shows polyps & ulcers.
- **The submucosa** shows localized granuloma.
- The polyps & ulcers arranged in circular transverse manner due to circular arrangement of veins of colon .

2. DIFFUSE FORM (All layers of colon)



- The **Mucosa** shows ulcers & polyps.
- The **Submucosa** shows fibrosis.
- The **musculosa** & **serosa** are fibrosed to form " Bilharzial peri-colic mass "

CLINICAL PICTURE

Type of patient (young or adult & male > female)

Symptoms

① Weakness, pallor & easy fatigability.
② Diarrhea, tenesmus & passage of blood and mucus in stool
③ **Mass** in lt. iliac fossa if peri-colic mass.

Signs

1- General signs :
- Anemia, clubbing fingers & hepato-splenomegaly

2- Local signs :
- Tender & thickened sigmoid colon.
- Hard nodular **mass** in lt. iliac fossa if peri-colic mass.
- **P/R** detects multiple rectal polyps.

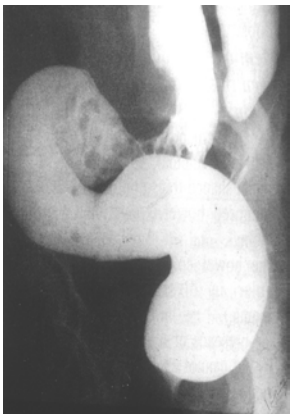
COMPLICATIONS **Never pre-cancerous**

Anemia, hemorrhage, rectal prolapse & intestinal obstruction by bilharzial polyps.

INVESTIGATIONS

A- Laboratory investigations

- **Stool analysis :** shows blood, pus, and probably Bilharzial ova.
- **Blood picture :** may show anemia.



B- Barium enema

- It shows multiple, rounded filling defect of variable sized and localized to sigmoid colon ± rectum.

C- Sigmoidoscopy

- It shows mucosal polyps →

TREATMENT

1- Anti-bilharzial drugs
as **oxamniquine**
or **praziquantel**.

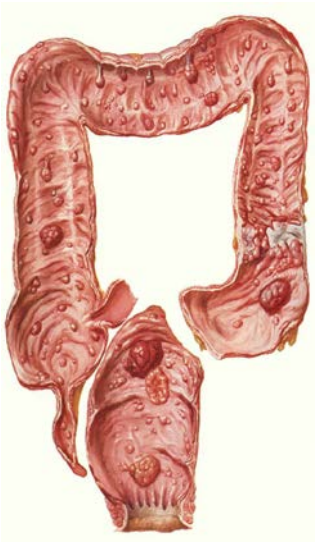
2- Polyps are removed by **sigmoidoscopic polypectomy**

N.B: Excision of affected sigmoid is **rarely** needed

.....

VIII- COLO-RECTAL TUMORS

Polyps of the colon & rectum



A. Epithelial

1- Hamartomatous polyps

Solitary **Juvenile** polyp.

Multiple e.g. Multiple **Juvenile** polyps
& **Peutz-Jegher's** polyps

2- Inflammatory polyps

No **solitary polyp**.

Multiple e.g. **Bilharzial** polyps
& **Ulcerative colitis** (pseudo-polyps).

3- Neoplastic polyps

Solitary Adenoma or carcinoma.

Multiple e.g. **Familial polyposis coli** (**F.P.C**)
& **Gardener's syndrome**.

4- Unclassified polyps

Solitary Solitary hyperplasic polyp.

Multiple Multiple hyperplasic polyps.

B. Mesenchymal

As **lipoma**, **leiomyoma** or **hemangioma**.

1. Benign Tumors

A. Familial polyposis coli (F. P.C)

AETIOLOGY It is an autosomal dominant disease.

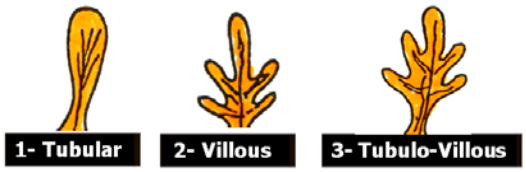
INCIDENCE

- Runs in **family**
- **Age :** Between **10** -**15** years (**never** since birth)
- **Sex :** Male **>** female.

PATHOLOGY

- Site : Multiple polyps at colon & rectum
- N/E : Sessile or pedunculated at least **100** in number

3 Types ➪
- Tubular
- Villous .
- Tubulo-villous.



N.B : F.P.C considered pre-cancerous.
So if untreated the carcinoma developed **100 %** at **5th** decades.

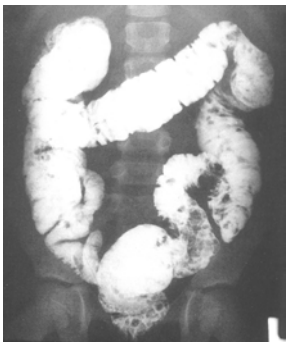
CLINICAL PICTURE

Symptoms

Lower abdominal pain, diarrhea & bleeding per rectum.

Signs

① Tender colon.
② **P/R** ➔ Rectal polyps.



INVESTIGATIONS

A- Barium enema

- It shows multiple, rounded & same sized defects

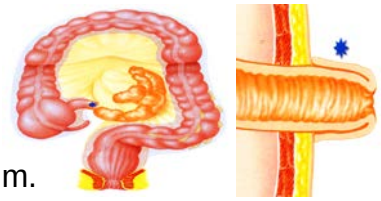
B- Sigmoidoscopy & biopsy

TREATMENT

1- The affected persons : Many options as ➪

① Total proto-colectomy + terminal ileostomy.

② Total colectomy + ileo-rectal anastomoses with regular endoscopic follow up of the rectum.

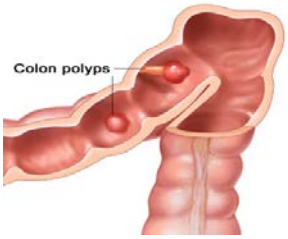


③ Total colectomy + rectal mucosectomy + ileo-anal anastomoses.

2- The family members :

Regular colonoscopic follow up.

B. Gardeners syndrome

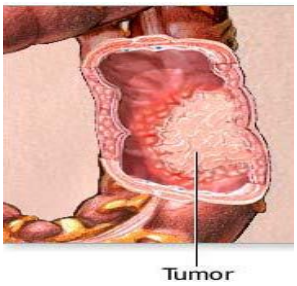


- It is a **rare variant** of familial polyposis coli.
- It can turn into malignant.
- It is associated with extra-colonic lesions include ➪ **Osteoma** of skull & mandible, **sebaceous cysts** and **desmoid tumors**.

2. Malignant Tumors

Carcinoma of the colon & rectum

INCIDENCE

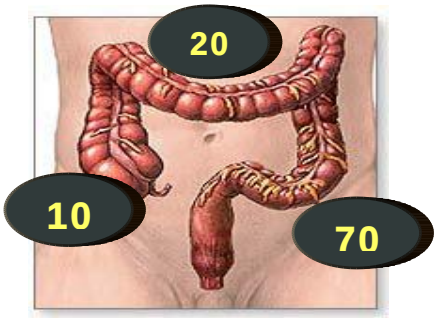


- The commonest **lower** G.I.T carcinoma
- **Age : 50** - **70** years
- **Sex :** Cancer caecum common with **female**.
  & cancer rectum common with **male**.

PREDISPOSING FACTORS

- Chronic irritations as high fat with low fibers diet
- Pre-cancerous lesions as ⇩
  ① Benign adenoma particular by Villous .
  ② F.P.C. & gardener's syndrome
  ③ Ulcerative colitis (If pancolitis **>** 10 years)
  ④ After uretro-sigmoid anastomosis.

PATHOLOGY

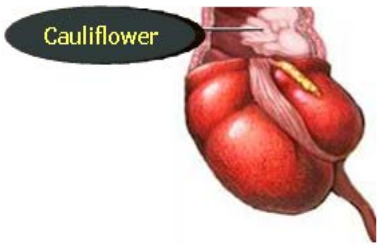


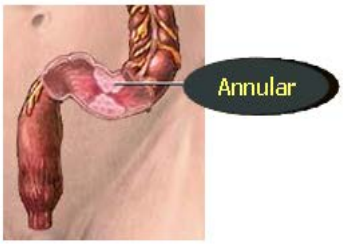
Site

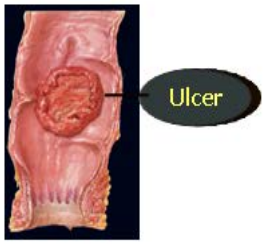
- **70 %** in sigmoid colon & rectum
- **10 %** in caecum
- **20 %** in rest of colon.

NIE picture

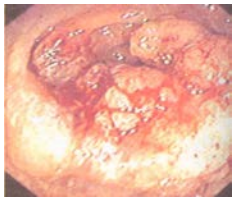
- **Cauliflower** mass mostly in the caecum
- **Annular scirrhous** mostly in the sigmoid colon
- **Malignant ulcer** mostly in the rectum.







Microscopic picture



- **Adenocarcinoma** that arises from columnar epithelium.

N.B : **Colloidal carcinoma** occurs in younger patients,
and has a **poor** prognosis.

SPREAD

Direct Both in (circumferential & longitudinal) direction
then Infiltrate the surrounding e.g. Small intestine, liver ... etc

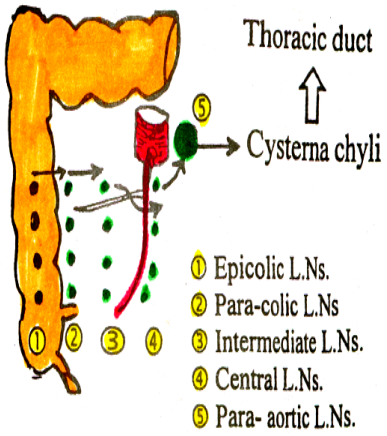
- The strong fascia of **Denonviller** lying in front of the rectum retards the spread of cancer rectum to the urinary bladder

Lymphatic spread

- **LYMPHATIC SPREAD TO THE COLON**

1st epicolic L.Ns
(overlying wall of colon)
Then to para-colic L.Ns
(along the side wall of colon)
Then to intermediate L.Ns
(along branches of meso-colic arteries)
Then to central L.Ns
(along the superior & inferior mesenteric arteries)
Then to the para-aortic L.Ns

Then to cysterna chyli & thoracic duct



- **LYMPHATIC SPREAD TO THE RECTUM**

- Upper $^1/_2$: Para-rectal L.Ns & superior rectal L.Ns then to inferior mesenteric L.Ns then to para-aortic L.Ns → Thoracic duct.

- Lower $^1/_2$: Middle & inferior rectal L.Ns then to internal iliac L.Ns then to para-aortic L.Ns → Thoracic duct.

Blood spread

Mainly through portal vein to liver & rarely to the lung

Transperitoneal spread

Leads to peritoneal nodules & ascites.

STAGING

1- T.N.M STAGING

T = Tumor	N = Nodes	M = Metastases
T_1 = invades into **submucosa** T_2= invades into **musculosa** T_3 = invades into **serosa** T_4= invades into **another organ**	N_0 = **No** nodes are involved N_1 = **1 - 2** nodes are involved N_2 = **3 or more** nodes are involved	M_0 = **No** metastases. M_1 = distant metastases.

2- DUKE'S STAGING

A =	The growth is **limited** to the bowel wall
B =	The growth extends **outside** the bowel wall **but NO L.Ns**
C =	**There are L.Ns** **C_1=** Para-rectal or Para-colic L.Ns **C_2=** Superior or Inferior mesenteric L.Ns
D =	**Distant metastasis**

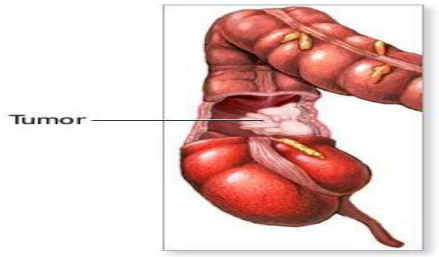
CLINICAL PICTURE

A- General examination

- **Picture of metastasis** : e.g. jaundice
- **Abdominal examination** for ascites or mass

B- Local examination

1. RT. COLON CANCER

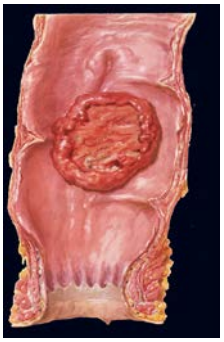


1. LT. COLON CANCER

• **Pain** at Rt. iliac fossa due to hyperperistaltic waves to get rid of tumor leading to **diarrhea**	• **Irritation** by hard impacted stools causing excessive mucus secretions leading to spurious (false) **diarrhea**
• **Mass :** which is hard, irregular, fixed & ill defined at Rt. iliac fossa (for DD)	• **Mass :** Not present **except** if there is faecal impaction above the tumor
• **Rarely** intestinal obstruction as ⇩ ① The **wider** the lumen ② The stool is still **liquid** ③ Carcinoma is **not** stenotic	• **Commonly** intestinal obstruction as ⇩ ① The **narrower** the lumen ② The stool is more **solid** ③ Carcinoma is **more** stenotic
• **The patient may present** by ⇩ Weakness, loss of weight & **3 A_s** **A**naemia , **A**norexia, **A**sthenia	• **The patient may present** by ⇩ Change in bowel habits as progressive constipation with short duration doesn't respond to any purgatives
• **Bleeding per rectum** So leading to severe microcytic anemia.	• **Bleeding per rectum** As hard stools may injure the mucosa lining the stenotic lesion.

3. RECTAL CANCER often present with ⇩

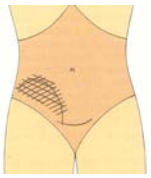
- Bleeding per-rectum which is usually slight i.e. blood streaked stool.
- Tenesmus (sense of painful incomplete evacuation) and passage of mucus.
- P/R Examination :
  - ① palpation of lesions in **90 %** of cases
  - ② Detection of **2^{ry} piles**.
  - ③ Detection of **blood at tip of examiner's finger**.



DIFFERENTIAL DIAGNOSIS

Mass in RT iliac fossa

1. Rt cancer colon
2. Appendicular mass .
3. Iliac lymphadenitis
4. Ileo-caecal T.B
5. Crohn's disease
6. Psoas abscess



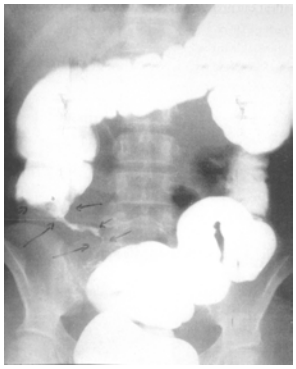
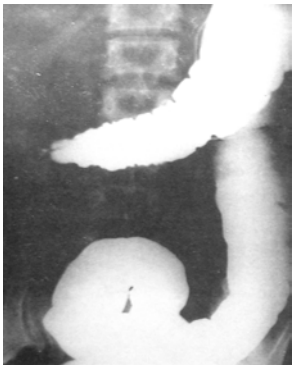
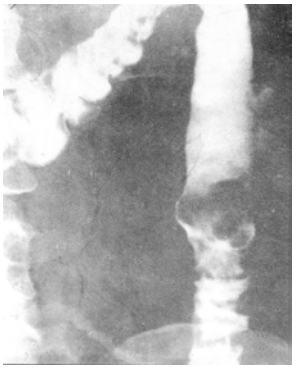
INVESTIGATIONS

A- Laboratory

Blood picture for **microcytic anemia**.

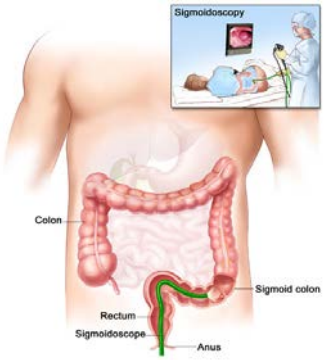
B- Barium enema

Irregular filling defect & (Apple core appearance on Lt. side)

C- Sigmoidoscopy & Colonoscopy

① It is essential in all patients with altered bowel habits or rectal bleeding.
② Mandatory in patient **> 40** years with piles.
③ **Biopsy** is obtained from suspicious lesions.



D- Metastatic work-up

e.g. liver function test, U/S or C.T scan for liver metastasis & chest x-ray for lung metastasis.

E. Tumor markers

Through **Carcino-embryonic antigen** (**C**.**E**.**A**) which is prognostic only. So replaced now by (**CA 19-9**) which is prognostic rather than diagnostic.

TREATMENT

TREATMENT OF CANCER COLON

I- OPERABLE

A. Tumors of the Caecum & ascending colon

Rt. Hemi-colectomy is done by ➪

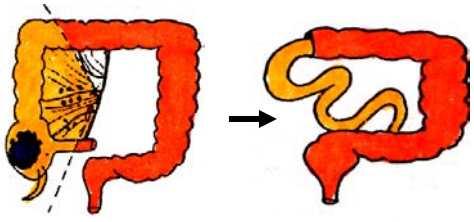
- Removal of draining L.Ns & ligation with division of **ileo-colic** & **Rt. colic** vessels at their origin from the superior mesenteric vessels

So The Extent of devascularized resected gut is :

① last **10** inch of terminal Ileum, caecum & appendix.

② Ascending colon, hepatic flexure & proximal **1/3** of transverse colon

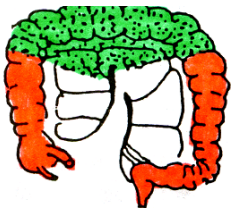
- Removal of peritoneum of post, abdominal wall between the resected colon & sup. mesenteric vessels as it contains lymph vessels and L.Ns



Finally Restoration of continuity by ileo-transverse anastomosis end to end

NB : **Tumors of hepatic flexure**. We do extended Rt. hemicolectomy
i.e. Rt. hemicolectomy + removal of Rt. **2/3** of transverse colon

B. Tumors of transverse colon



Transverse colectomy is done by ☟

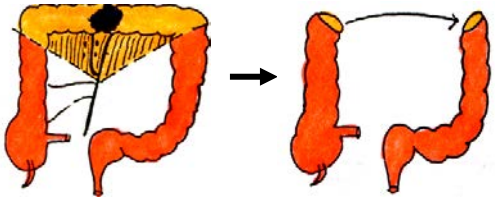
- Removal of draining L.Ns & ligation with division of **middle colic** vessel at their origin from the superior mesenteric vessels

So The Extent of devascularized resected gut is :

① Transverse colon & mesocolon

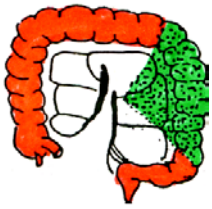
② 2 flexure (hepatic & splenic)

- Removal of greater omentum



Finally Restoration of continuity by end to end anastomosis

C. Tumors of descending colon



Lt. Hemi-colectomy is done by ☟

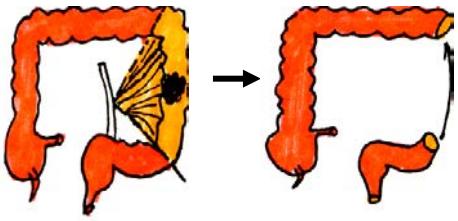
- Removal of draining L.Ns & ligation with division of **Lt. colic** vessels at their origin from the inferior mesenteric vessels

So The Extent of devascularized resected gut is :

① The Lt. **1/3** of transverse colon

② The splenic flexure & descending colon

- Removal of a wedge of peritoneum of post, abdominal wall



Finally Restoration of continuity by end to end anastomosis

NB : **Tumors of splenic flexure**. We do extended Lt. hemicolectomy
I.e. Lt. hemicolectomy + removal of Lt. **2/3** of transverse colon

D. Tumor of sigmoid colon

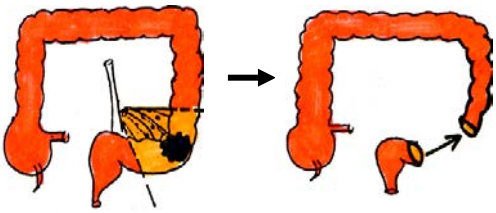
Sigmoid (Pelvic) colectomy is done by ☟

- Removal of draining L.Ns & ligation with division of **sigmoid** vessel at their origin from the inferior mesenteric vessels

So The Extent of devascularized resected gut is :

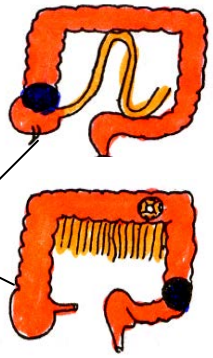
① Sigmoid colon

② Sigmoid mesocolon.



Finally Restoration of continuity by end to end anastomosis

II- INOPERABLE



- If resectable tumors :
  - palliative resection
- If irresectable tumor :
  - **Rt. colon :** side to side ileo-transverse anastomosis.
  - **Lt. colon :** proximal colostomy in transverse colon

N.B.: **Chemotherapy** [5 Fluro-uracil]
& **Radiotherapy** may be useful

TREATMENT OF CANCER RECTUM

I- OPERABLE

A. Tumors at the upper $^{1}/_{3}$ of rectum

Anterior resection (**without colostomy**) = **Dixon**

- Removal of draining L.Ns & ligation with division of inferior mesenteric vessels. Lt. colic vessel is spared

So The Extent of devascularized resected gut is :

① The sigmoid colon .

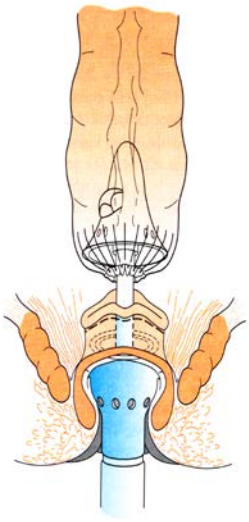
② The **upper half** of rectum

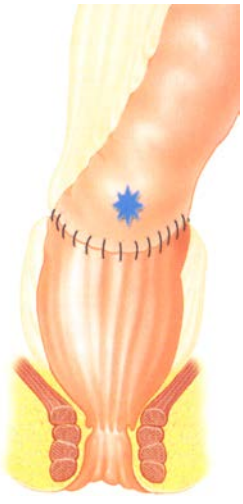
③ The 2 lateral rectal ligaments

- With preservation of sphincters & levator ani.

So this operation is called
Sphincter saving anterior resection

Finally Restoration of continuity by end to end anastomosis of upper sigmoid & lower rectal stump deep in pelvis.





N.B.: The **staplers** facilitate this anastomosis

B. Tumors at the lower $^{2}/_{3}$ of rectum

Abdomino-perineal resection (**with colostomy**) = **Miles**

- Removal of draining L.Ns & ligation with division of inferior mesenteric vessels. Lt. colic vessel is spared

So The Extent of devascularized resected gut is :

① The sigmoid colon .

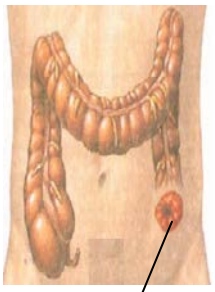
② The **whole** rectum

③ The 2 lateral rectal ligaments

④ The whole anal canal .

⑤ The sphincters & levator ani.

⑥ Peritoneum of pelvic floor & wide ellipse of peri-anal skin .

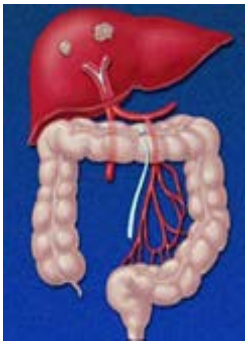


Finally It is ended by **terminal colostomy** at Lt. iliac fossa.

N.B: It is called **abdomino-perineal resection** because part of operation is done through the abdomen & the other part is done through the perineum

II- INOPERABLE

- If resectable tumors :
  - palliative resection
- If irresectable tumor :
  - Chemotherapy & radiotherapy



N.B: **Hepatic metastasis**

Usually it means inoperable

but with colo-rectal tumors it may be operable

If ① Solitary nodule with no extra-hepatic metastasis

② Multiple but confined to one lobe

IX- SMALL INTESTINAL TUMORS

A. Benign tumors

1- Adenoma

2- Submucous lipoma

3- Leiomyoma

B. Malignant tumors

1- Adenocarcinoma

2- Non Hodgkin's lymphoma

3- Carcinoid tumor

X- RECTAL PROLAPSE

DEFINITIONS

Protrusion of rectum through the anus

AETIOLOGY

A. Partial prolapse

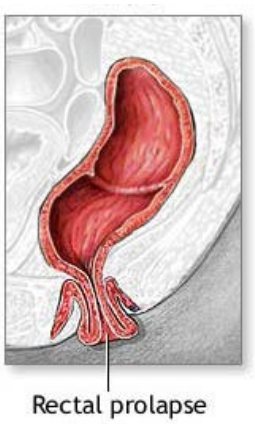
(**Prolapse of mucosa only of rectum**)

- Common in children due to

① Loss of curve of sacrum so rectum is a vertical tube.
② Loss of weight so loss of ischeo-rectal & para- rectal fat
③ Chronic straining at defecation with prolonged diarrhea.

- But may occur with Adult due to

① Advanced cases of hemorrhoids .
② Atony of sphincters (mainly with elder)
③ Chronic straining due to urethral stricture.


Rectal prolapse

B. Complete prolapse

(**Prolapse of whole rectal wall**)

- Common in elderly particularly female

In Egypt, Bilharzial proctocolitis ➔ tenesmus ➔ straining at defecation

CLINICAL PICTURE

Symptoms

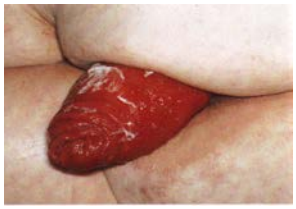
① **Something protruding** from the anus at defection .

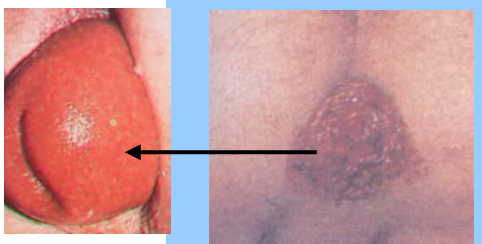
② **Picture of complications**
as irreducibility, bleeding, fecal incontinence, ulceration & pruritis.

Signs

① **Inspection :** prolapse is seen in squatting or lat. position with straining

② **Palpation :** for thickness of prolapse & tone of sphincters.





	Partial prolapse	Complete prolapse
Incidence	**Common**	**Less** common
Age	More in **children**	More in **adult**
Length of prolapse	**< 5** cm	**> 5** cm
Mucosal corrugation	Absent	Present
Thickness of prolapse	Mucosa only	Whole rectal muscle

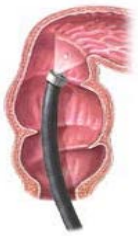
DIFFERENTIAL DIAGNOSIS

1- Prolapsing hemorrhoids
2- Prolapsing polyp
3- Prolapsing intussusception

INVESTIGATION

① Ano-rectal **manometer** & **EMC** of rectal sphincters

② **Sigmoidoscopy** or **barium enema**
to exclude other causes as Bilharziasis



TREATMENT

Prolapse in children

- Surgery is **rarely** indicated as the condition resolves spontaneously.
- Conservative treatment mainly through ⇩
  - ① **Correct** the cause of straining & improve nutritional status.
  - ② **Manual reduction** by mother after defecation followed by strapping of buttocks.
  - ③ **Submucous injection** of 5 % phenol in almond oil.

Prolapse in Adult

A. PARTIAL PROLAPSE

- **Submucous injection** of 5 % phenol in almond oil.
  or Excision in a fashion as hemorrhoidectomy.

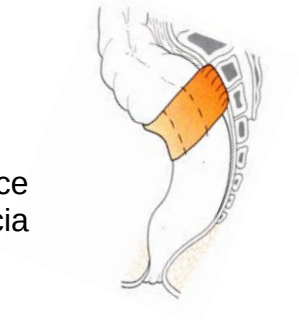
B. COMPLETE PROLAPSE

- Surgical treatment **:**

Various surgical procedures include ⇩

1- Rectopexy [Charles Well's Operation]

- Through an abdominal approach
- The rectum is mobilized and pulled upwards. then a piece of prolene mesh is attached by sutures to pre-sacral fascia and then it is wrapped around back & sides of rectum. So fibrosis leads to fixation



2- Excision of the redundant rectum

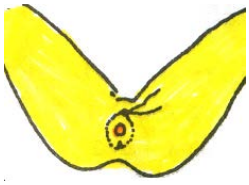
- Through an abdominal or a perineal approach

3- Delorme's operation

- Through a transanal approach
- The excess rectal mucosa above the dentate line is circumferentially excised.

4- Thiersch's operation (peri-anal circulage)

- The prolapse is reduced, and a stainless steel wire is used subcutaneously around the anus.
- This operation is simplest but it provides the **poorest** results.

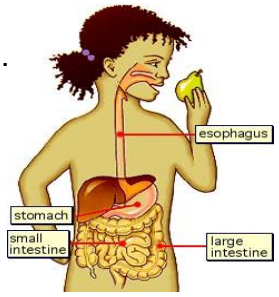


Intestinal obstruction

INTESTINAL OBSTRUCTION

DEFINITION

Arrest of downward propulsion of intestinal contents.



CLASSIFICATIONS

There are 3 different types of classifications ⇩

1. According to pathological nature of the cause

1- SIMPLE OBSTRUCTION

- It is due to **mechanical occlusion** of the gut lumen **without** interference with its blood supply.

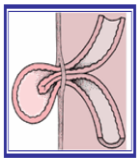
2- STRANGULATION

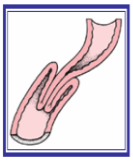
- **Pure** as in **mesenteric vascular occlusion** with patent lumen.
- **Mixed** (both lumen & vessels of intestinal loops are blocked)

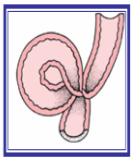
as in ① **Strangulated hernia**

② **Intussusception** .

③ **Volvulus**


Strangulated hernia


Intussception


Volvulus

3- NEUROGENIC

- It is due to paralysis of peristaltic activity of the gut ➔ **Paralytic ileus**. i.e. loss of propulsive power of the bowel.

2. According to the level of obstruction

1- HIGH SMALL bowel obstruction

2- LOW SMALL bowel obstruction

3- LARGE bowel obstruction.

3. According to the onset & the course of the cause

1- ACUTE OBSTRUCTION clinical course is **rapid**.

2- CHRONIC OBSTRUCTION clinical course is **slow**.

3- ACUTE ON TOP OF CHRONIC OBSTRUCTION

- As in fecal impaction on top of colonic tumors .

Also **It is classified according to** (**dynamic state**)

1- DYNAMIC (**Increased peristalsis**)

- It is due to mechanical occlusion of lumen

2- A DYNAMIC (**No peristalsis**)

- It is due to ⇩

(1) **Paralytic ileus**

(2) **Vascular** i.e. **m**esenteric **v**ascular **o**cclusion.

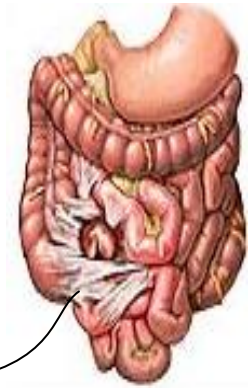
I- SIMPLE OBSTRUCTION

ADHESIVE INTESTINAL OBSTRUCTION

POST OPERATIVE INTESTINAL OBSTRUCTION

DEFINITION

It is a **mechanical occlusion** of the gut lumen **without** interference with its blood supply



AETIOLOGY

A- In the lumen

As fecal impaction, gall stones, F.B, tumors ……..etc.

B- In the wall

As atresia or stricture (inflammatory or neoplastic)

C- Outside the wall

As adhesions

or **internal herniation**

- **Portion of the small intestine** passes into one of retroperitoneal fossa or into a congenital defect in the mesenteries.
- **The potential openings are**
  1. Foramen of **winslow** or **obturator** foramen.
  2. Hole in the mesentery of intestine or transverse mesocolon.
  3. Defect in the broad ligament of the uterus.

N.B.: Causes of intestinal obstruction according to age

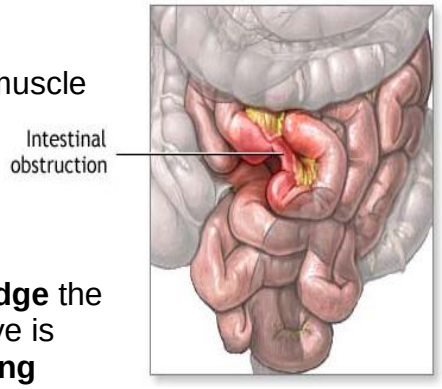
1. **Newborn :** mechonium ileus & congenital megacolon
2. **Children :** strangulated hernia & Intussusception .
3. **Adult :** strangulated hernia & tumors.
4. **Old age :** strangulated hernia, tumors & volvulus sigmoid..

PATHOLOGY

Proximal segment

- Hyperperistaltic wave :

Sudden obstruction → stretch of the muscle of proximal segment → ↑ contraction i.e. hyperperistalesis to dislodge the obstructing agent **= colic & pain**

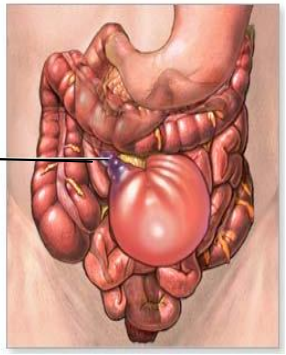


- Anti-peristaltic wave :

Failure of proximal segment to dislodge the obstructing agent → Anti-peristaltic wave is initiated i.e. Anti-physiological **= vomiting**

- Stage of dilatation :

The proximal segment start to fatigue then dilates ➔ stagnation which invites infection & fermentation **= distension**.



N.B : The sources of gas production:

1. The Bacterial fermentation (**10 %**)
2. Swallowed atmospheric air (**68 %**)
3. Diffusion from blood into bowel lumen (**22 %**)

Then severe continuous distention leads to occlusion of veins ➔ edema of the wall of lumen ➔ more distention leading to occlusion of arterioles ➔ ischemia & gangrene of the bowel ➔ perforation of the gut ➔ **peritonitis**.

Distal segment

Always empty & collapsed **= absolute constipation**

CLINICAL PICTURE

Symptoms

1. **Pain :** [The **1st** & earliest symptom].
   - **Onset :** sudden
   - **Site :** - **above** umbilicus **=** high small intestine.
     - **around** & **below** umbilicus **=** low small intestine
     - **peripheral** & **lower** abdomen **=** large intestine.
   - **Character :** colicky in nature.

2. **Vomiting :**
   - **At 1st** gastric juice (**whitish** mucoid)
   - **Then** jejunal contents (**greenish** bile stained)
   - **Finally** intestinal contents (**brownish** faecal)

3. **Distention :**
   - Which is marked in colonic obstruction

4. **Absolute constipation :**
   - Which is failure to pass flatus or stools in spite of desire

Signs

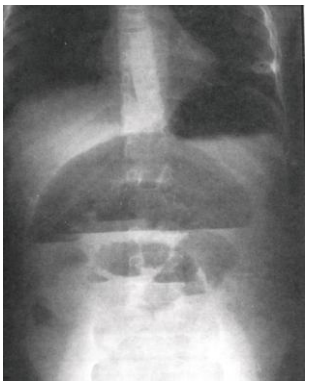
1. **General signs :**
   - **Signs of dehydration :**
     Inelastic dry skin, sunken eye, dry tongue, oliguria....etc
   - **Signs of hypovolemic shock :**
     ① Tachycardia & tachypnea (air hunger)
     ② Hypotension & hypothermia.
     ③ Pale cold skin & oliguria.

2. **Local signs :**
   - **Inspection :** distended abdomen with visible peristalsis.
   - **Palpation :** tender abdomen & a mass may be felt if tumor is present.
   - **Percussion :** hyper-resonance.
   - **Auscultation :** early loud intestinal sound followed by dead silent

INVESTIGATIONS

1- Laboratory "Serum electrolytes" for Na^+ & K^+

2- Plain x-ray (**Abdomen**)

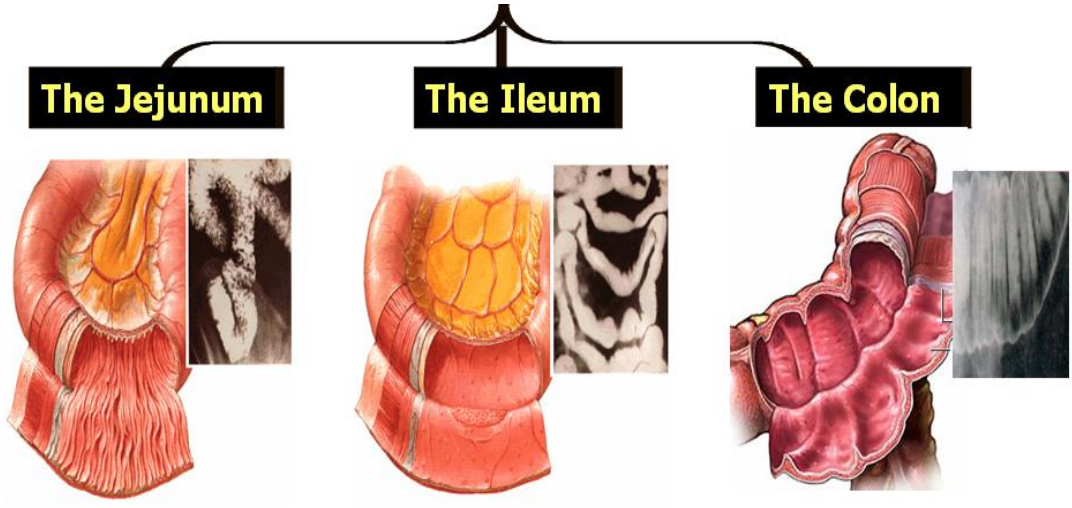


1- Erect position :

- **It shows** multiple fluid levels (**> 3** fluid levels)
- **It may be** arranged in a "**step-ladder**" pattern.
- **Normally, 3 fluid levels may be present :**
  - ① At the stomach.
  - ② At duodeno-jejunal flexure.
  - ③ Ileo-caecal region.

2- Supine position :

- **It shows the pattern of the mucosa** of the dilated part as follows



THE JEJUNUM

- **Regular** mucosal folds **valvulae conniventes** (concertina like) is seen complete with regular spaces in between.

THE ILEUM

- **Featureless** with **no** mucosal pattern

THE COLON

- **Irregular** mucosal folds incomplete folds & irregular spaces in between.

3- Abdominal U/S

- May reveal distended bowel loops.

4- Barium enema

- May reveal the cause if colonic obstruction.

5- Double enema test

- **1st enema** to washout the distal segment and wait for **1 hour**.
- **2nd enema** (If obstruction) It will comes out clear (**no stools**)

TREATMENT [Urgent surgery after resuscitation]

A- Immediate resuscitation

- **Ryle's tube** for suction + no oral intake.
- **I.V fluids** to correct electrolyte imbalance
- **I.V blood & Ringer's lactate** to correct hypovolaemic.
- **I.V broad spectrum A.B**. to guard against peritonitis.

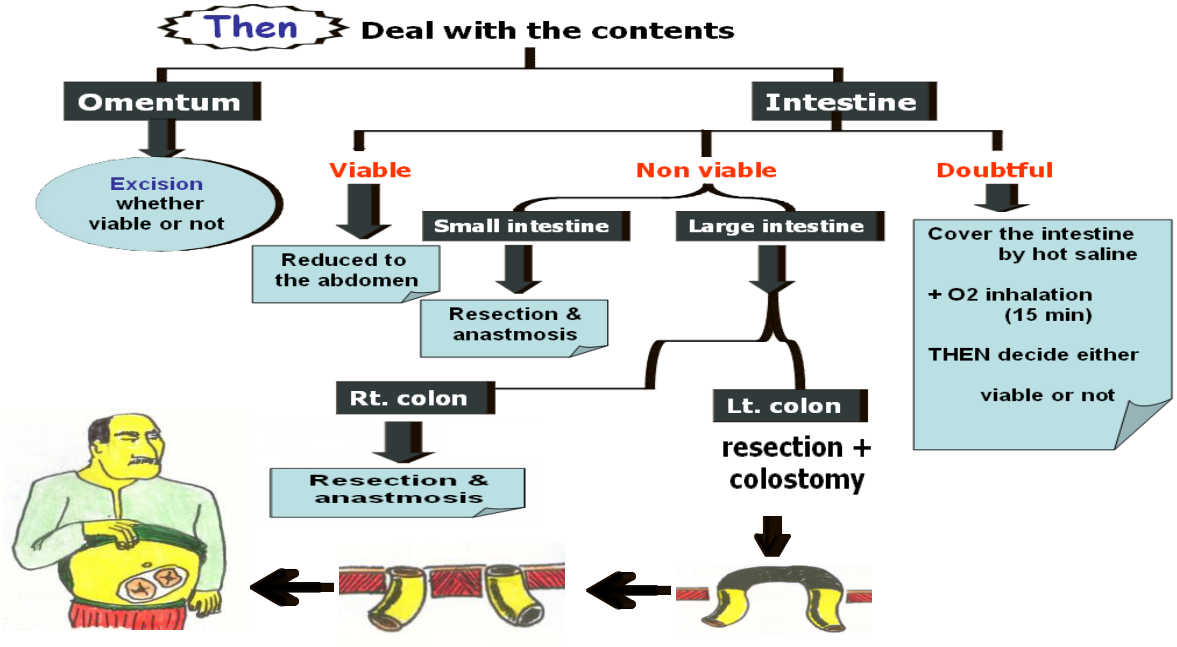
B- Immediate operation Exploratory laparotomy

- **Inspect the caecum**
  - If ***distended*** → colonic obstruction.
  - If ***collapsed*** → small bowel obstruction
- **Intestinal decompression** by retrograde milking or introduction of a large tube sucker through a small enterotomy.
- **Remove** the cause of obstruction
- **The contents** are pulled out & examined, viable or not

	Viable intestine	Non-viable intestine
• Intestinal color • Peritoneal luster • Mesenteric arteries	• Pink or dark red • Present. • Pulsating	• Grey or black. • Absent. • Non pulsating
• By pinching • Consistency • If injured	• Contracts • Firm • Bleeding occur	• No response • Floppy • No bleeding

D- Intraoperative tests (To detect viability)

1. **Doppler ultrasound**
2. **Fluroscein test :** Inject 1 gm fluroscein I.V then inspect the bowel under U.V. rays. If the bowel has good blood supply it will flurescence
3. **Oximeter**



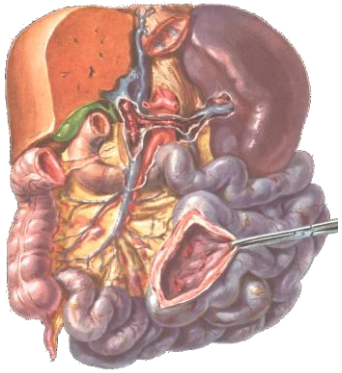
II- STRANGULATION

1. Pure strangulation

Mesenteric vascular occlusion (ischemic colitis)

DEFINITION

- It is a sudden occlusion of major mesenteric vessels with patent lumen.



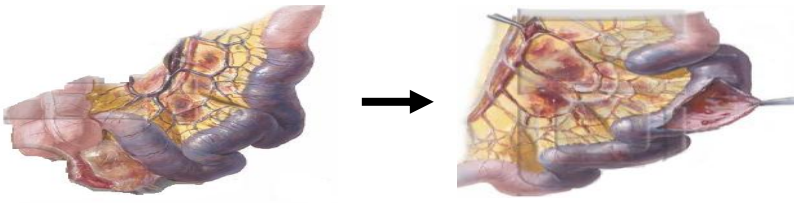
AETIOLOGY

① Mesenteric **arterial** embolism.
② Mesenteric **arterial** thrombosis
③ Mesenteric **venous** thrombosis.

PATHOLOGY

ischemic damage

- **Mucosa** sloughs within **3 hours** & bleeds in the lumen.
- **The whole** thickness of intestinal wall is affected within **6 hours** & exudes serosanginous fluid in the peritoneum.



- **The intestine** becomes gangrenous & perforates ➔ **peritonitis**.

Reperfusion damage

Return of blood flow (either spontaneously or by surgery) ➔ release of oxygen free radicals from ischemic bowel.

CLINICAL PICTURE

General "**Hypovolemic shock**"

① Tachycardia & tachypnea (air hunger)
② Hypotension & hypothermia.
③ Pale cold skin & oliguria.

Local

Severe acute abdominal **pain**, **vomiting** & **bleeding** per rectum.

TREATMENT **[Urgent surgery after resuscitation]**

A- Immediate resuscitation

- **As before** + blood transfusion.

B- Immediate operation

- **Exploratory laparotomy**
  then resect the gangrenous intestine and restore blood flow of the viable ones by **endarterectomy** or **bypass** for thrombosis
- **A 2nd look operation after 24 hours** is advised for patients who receive a 1ry anastomosis

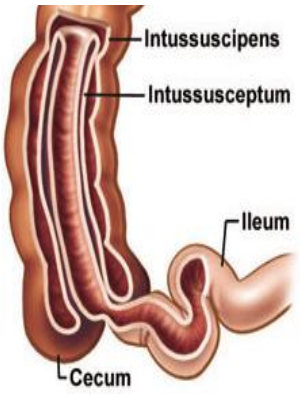
2. Mixed strangulation

A. Strangulated hernia

See (HERNIA)

B. Intussusception

DEFINITION



It is invagination of segment of the bowel (**intussusceptum**) into the lumen of the adjacent one (**intussuscepient**)

ANATOMICAL TYPES

1- Ileo- ileal : Ileum is invaginated into the ileum.

2- Ileo- colic : Ileo- ileal advancing till its apex enters the colon.

3- Colo- colic : Colon is invaginated into the colon

4- Retrograde : as jejunogastric intussusception after gastrojejunostomy .

CLINICAL TYPE

1- Infantile type ; Common, usually ileo-caecal type.

2- Adult type ; Rare, usually due to organic lesion at apex as carcinoma

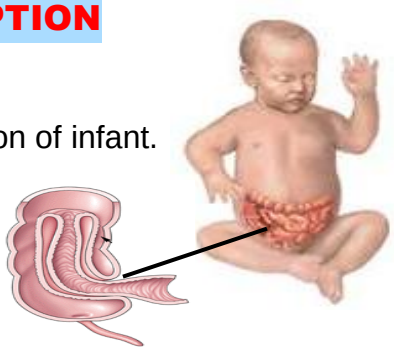
PATHOGENESIS

Partial obstruction of gut lumen by any cause will initiate the hyperperistalitic activity of proximal segment to overcome this obstruction.

But if hyperperistalasis is too much, it will push the wall of the gut of one loop to another.

N.B. : Complete obstruction never leads to intussusception as the lumen is occluded i.e. **no space** for passage of a loop

INFANTILE INTUSSUSCEPTION



INCIDENCE

- The most common cause of intestinal obstruction of infant.
- **Age: 1st** 2 years of life
- **Sex:** Male > female

AETIOLOGY

A- Idiopathic (**95 %**) but may be due to ☟

1- Repeated gastroenteritis → swollen lymphoid follicle in the terminal ileum which act as source of partial obstruction.

2- In infant the caecum & colon are mobile. so they telescope easily over the terminal ileum.

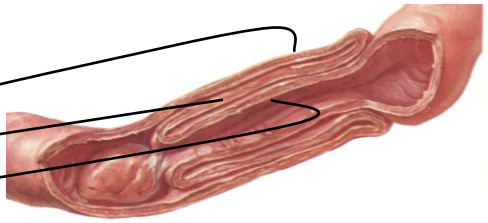
3- In infant the ileo-caecal valve is bulky & projects into the caecum

B- Secondary (**5 %**)

There is an evident cause e.g. **Meckel's diverticulum**

PATHOLOGY

- **The ileo-caecal valve is invaginated into the caecum** & may travel along the ascending, transverse, descending colon & may protrude through the rectum & anus.
- **An intussusception consists of**
  ① The ensheathing or outer layer.
  ② The returning or middle layer
  ③ The entering or inner layer



So The entering & the returning layers called **intussusceptum** & they meet at the **apex**

The ensheathing layer called **intussuscepien** & It joins the returning layer at the **neck**

- **The mesentery** (containing blood vessels) may be compressed between the entering & the returning layers leading to ischemia & gangrene.

CLINICAL PICTURE

Any infant having colicky abdominal pain with passage of bloody mucus per rectum should be suspected of having intussusception until prove otherwise

Type of patient [**3 -12** month & Male **>** female]

Symptoms

① Attacks of **colicky pain** denoted by screaming or drawing the legs up to the abdomen.
② **Vomiting**.
③ **Passage of bloody mucus** like (**red current jelly**) per rectum.

Signs

① **Distention** is late with **empty Rt. iliac fossa = sign de Dance**.
② **A sausage shaped mass** any where in the abdomen except Rt. iliac fossa
③ **P/R Examination** reveals bloody mucus & sometimes the head of the intussusception can be felt.

DD From gastro-enteritis or feeding problems

INVESTIGATIONS

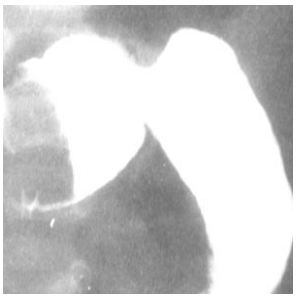
1- Laboratory "Serum electrolytes" for Na^+ & K^+

2- Barium enema

Claw sign :

- **It shows** sudden arrest of the dye at a cylindrical filling defect

Barium enema

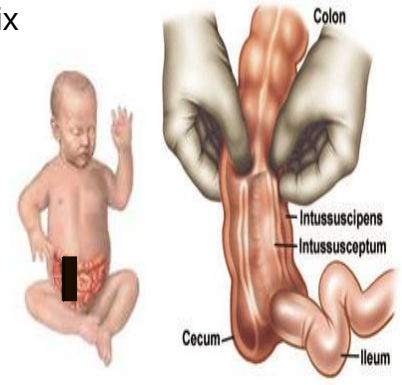


TREATMENT

- **Resuscitation** with correction of fluid & electrolyte
- **Reduction of intussusception** either by ⇩

A- Hydrostatic reduction (only with early cases)

- Barium is running through rectum under pressure & followed up under screen until complete reduction occurs.
- Evident by the appearance of caecum, appendix & terminal ileum.



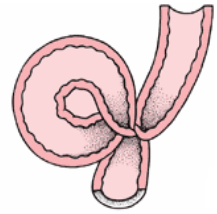
B- Operative reduction

- It is still the standard treatment of all cases, through a Rt. lower trans-rectal incision
- The thumb, index & middle fingers are inserted into the abdomen and the intussusception is reduced by squeezing the apex in a proximal direction.

N.B: Don't pull on invaginated segment to avoid intestinal tear

If The reduction fails or gangrenous loop occur, we do resection with end to end anastomosis.

C. Volvulus



DEFINITION

Twisting of loop of gut around the axis of its own mesentery

PATHOGENESIS

For a twist to occur, the gut should be in the form of a **loop** with a mesentery (**Omega loop = 2 long** limbs, **narrow** base & **wide** apex)

SIGMOID VOLVULUS

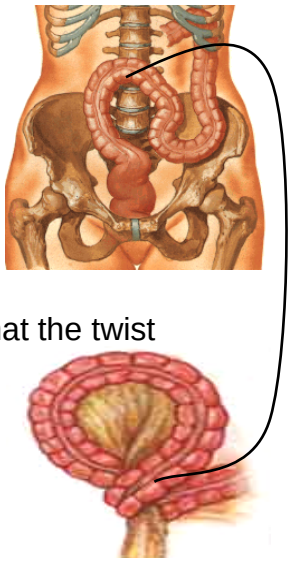
PREDISPOSING FACTORS

Chronic constipation which leading to ⇩

1- Over distension of the colon

2- Abnormal elongation of meso-colon.

3- Adhesions at the apex of the sigmoid loop facilitate its twisting.



PATHOLOGY

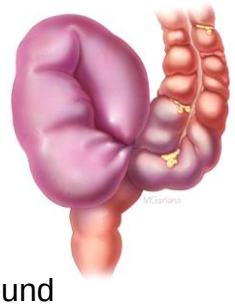
- The upper loop usually falls in front of the lower. so that the twist always in anti-clock wise direction.

N.B.: Closed Loop obstruction leading to ⇩

① Huge distension of the sigmoid colon
② The colon above is distended
③ The rectum below is collapsed & empty.

- Any interference with the blood supply occur if it rotates **> 1.5** turn → ischemia & gangrene → perforation & peritonitis.

CLINICAL PICTURE



Type of patient Elderly **constipated** male.

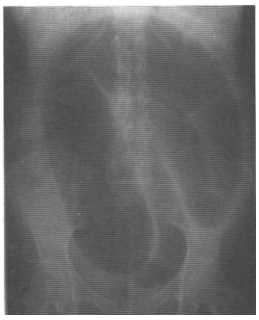
Symptoms

① Sudden onset of severe abdominal **pain** & **distension**.
② Absolute **constipation** is " early **"**
③ **Vomiting** " late "

Signs

① **P/R examination** shows empty rectum, blood may be found on tip of examining finger.
② **Neglected cases** may show picture of peritonitis.

INVESTIGATIONS



1- Laboratory "Serum electrolytes" for Na^+ & K^+

2- Plain x-ray

- **It shows** huge gas-filled sigmoid loop that may look like the **inner tube of car tire** (**Omega loop**)

TREATMENT

A- Conservative treatment

INDICATED WITH

Early cases with no evidence of gangrene.

METHOD

Rectal tube is passed through a sigmoidoscope to untwist the sigmoid loop. success will lead to gush of gas & fluid stools.

Finally the tube is left on place and the patient is prepared for later elective resection of the long sigmoid to prevent recurrence

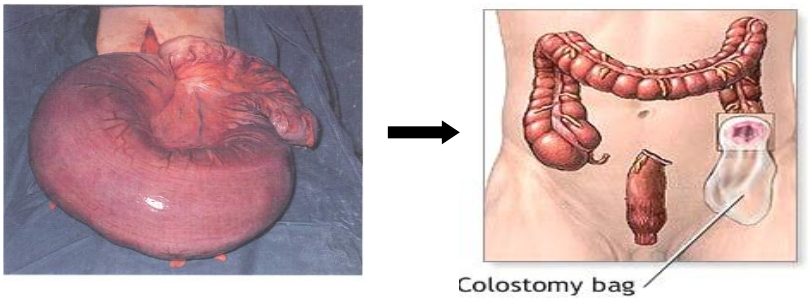
B- Emergency surgical treatment

INDICTED WITH

Late cases or failure of conservative treatment

METHOD

Deflate the sigmoid by untwisting the volvulus
then if gangrenous ➔ resection is done, the proximal end is brought out to the skin as terminal colostomy & the distal end is sutured (**Hartmann's procedures**)



III- PARALYTIC ILEUS

DEFINITION

- It is a paralysis of peristaltic activity of the gut.
  i.e. loss of propulsive power of the bowel.

AETIOLOGY

① **Reflex inhibition** of intestinal motility following abdominal operation or spine fracture. It may be due to sympathetic overtone.

② **Toxic inhibition** of intestinal motility as peritonitis

③ **Metabolic abnormalities** as **↓ K** & **↓Na** ……..etc.

④ **Drugs** overdose as anticholenergics.

CLINICAL PICTURE

- Abdominal distension, absolute constipation & vomiting.
- The patient **doesn't** have colicky abdominal pain (**silent abdomen**).

INVESTIGATIONS

1- Laboratory "Serum electrolytes" for Na^+ & K^+

2- Plain x-ray - **It shows** multiple gas fluid levels

PREVENTION

- Pre-operative : Correction of biochemical disturbances.

- Operative : Gentle handling of the intestine.

- Post-operative : Naso-gastric tube to decompress the bowel until return of intestinal sound or passage of flatus.

TREATMENT

- Gastro-intestinal suction until intestinal sounds return.

- I.V fluid therapy especially K^+ & Na^+.

- Drugs : Para-sympathomimetics e.g. Prostigmine may be helpful.

.....

DD OF DIFFERENT TYPES OF I.O

	Simple Intestinal obstruction	Mesenteric vascular occlusion	Infantile Intussusception	Volvulus Sigmoid	Paralytic Ileus
Colics	Present	Present	Present	Present	Absent
Vomiting	Present	Present	Present	Present (Late vomiting)	Present
Distention	Present	Absent	Absent	Present	Present
Absolute constipation	Present	Absent Bleeding per rectum	Absent Bleeding & mucus per rectum	Present (Early constipation)	Present

IV- NEONATAL INTESTINAL OBSTRUCTION

AETIOLOGY

1- Lumen :

① Meconium ileus.
② Meconium plug syndrome.

2- Wall :

① Duodenal atresia.
② Annular pancreas (**see pancreas**)
③ Congenital megacolon
④ Imperforated anus

3- Outside the wall :

① Volvulus neonatorum.
② Irreducible congenital hernia (**see hernia**)

1- Meconium ileus

DEFINITION

It is a defect of mucous secretion of pancreas "**Mucoviscidosis**".

PATHOLOGY

The terminal ileum is obstructed by thick meconium.

CLINICAL PICTURE (2**V**, 2**C** & 2**D**)

① 2**V**s **= V**omiting & **V**isible peristalsis.
② 2**C**s **= C**olics & **C**onstipation (absolute)
③ 2**D**s **= D**istension & **D**ehydration

INVESTIGATION **Gastrographin enema**

TREATMENT **Ileostomy :** May be needed & may be life saving.

2- Meconium plug syndrome

DEFINITION

The left colon is obstructed by a thick plug of meconium,

TREATMENT

During the 1st 24 hours, after rectal examination or saline washout, a large grayish plug of mucous followed by sticky meconium in a large amount are passed.

3- Duodenal atresia

DEFINITION

It is imperforation of the duodenum i.e. atresia.

PATHOLOGY

It is usually affect the 2nd part of duodenum, it may be partial or complete

CLINICAL PICTURE

(2**V**, 2**C** & 2**D**)

INVESTIGATION **Plain X-ray**

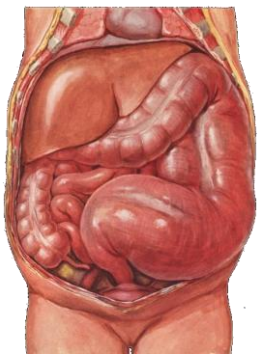
It shows distension of the stomach & proximal duodenum i.e. **Double bubble sign**

TREATMENT **Duodeno-jejunostomy** or **gastro-jejunostomy**

4- Congenital megacolon

Hirschsprung's disease

(AGANGLIONIC MEGACOLON)



DEFINITION

It is dilatation & hypertrophy of colon,
in absence of mechanical obstruction.

AETIOLOGY [**unknown**]

It is due to failure of migration of ganglionic cells
of the para-sympathetic plexus into the distal bowel

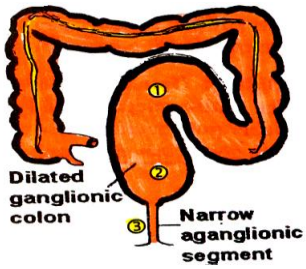
PATHOGENESIS

The Aganglionic segment lacks the normal peristaltic activity
leading to obstruction ➔ progressive dilatation of proximal colon.

PATHOLOGY

- **It is mainly** affect the rectum & anal canal.

N.B.: Sigmoid & whole colon is rare



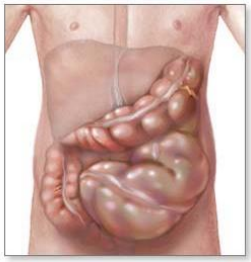
It divided into 3 segments

① **Proximal** dilated hypertrophied (colon).
② **Transitional** zone (sigmoid).
③ **Spastic aganglionic** segment (rectum).

CLINICAL PICTURE

Any newborn presenting with delayed passage of meconium for 24 hours
considered as having **Hirschsprung's disease** until proved otherwise

Symptoms



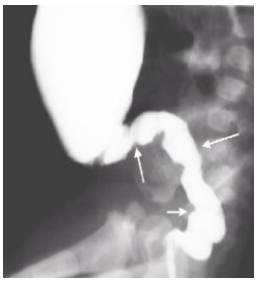
- Progressive constipation
Motion every few days & only after insertion
of a suppository or doing an enema.

- Progressive distention.

Signs

- Visible peristalsis to terminate in the Lt. iliac fossa.
- Fecal mass felt in the Lt. iliac fossa.
- P/R Examination shows empty rectum and when the finger
is removed, it is followed by a sudden gush of gases or stool.

INVESTIGATIONS



1- Barium enema
It will show **3 segments** without preparation to
avoid masking the changes of colonic caliber

2- Rectal biopsy
To show **absence** of ganglionic cell in submucosa

TREATMENT

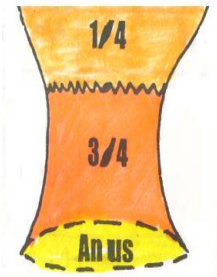
- The aim of treatment is to do complete excision of the
A ganglionic segment followed by a colo-rectal anastomosis

5- Imperforated anus

Ano-rectal malformation

Embryology

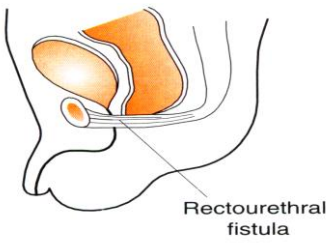
- The upper **1/4** of anal canal is derived from the distal part of hindgut
- The lower **3/4** of anal canal is derived from the proctodaeum which is invagination of the ectoderm.
- The 2 separated developed parts meet at the **dentate** or **pectinate** line.

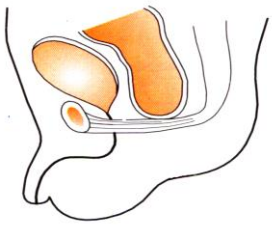


TYPES

1- High anomalies (Male > female)

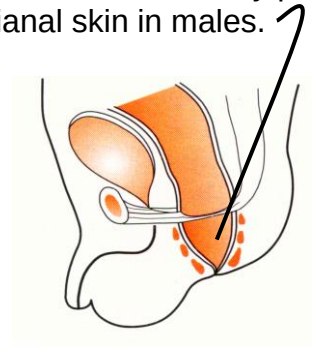
- Where the rectum to pass **above** the level of pelvic floor.
- It is either shows a blind end or more commonly there is a fistulous communication with urinary bladder or posterior urethra in male or to the vagina in females.

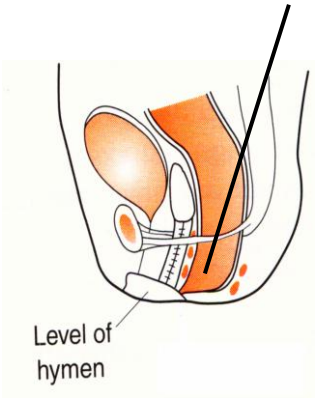




2- Low anomalies (Female > Male)

- Where the rectum has passed **through** the level of pelvic floor.
- It is usually opens into an **ectopic site** anterior to the normal anus position.
- A fistulous connection may pass to the vestibule in females or to the perianal skin in males.





CLINICAL PICTURE

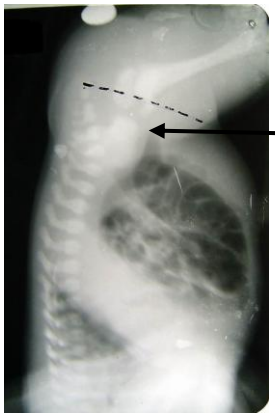
The diagnosis is easy, provided that the anus has been inspected as essential part of routine examination of any newborn.

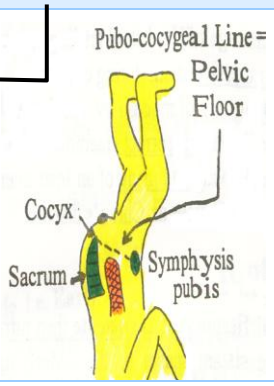
N.B.: IF mechonium present on the tip of penis this means high anomaly with recto-urethral fistula

INVESTIGATIONS

X-ray " Invertogram "

- **24** hours after birth with the newborn held upside down with a radio-opaque marker on the anus e.g. **coin**
- **Then A line** is drown from the symphysis pubis to the coccyx



HIGH ANOMALY	LOW ANOMALY
	

- IF the gas shadow in the rectum is seen **above** the pubo-coccygeal line the anomaly is **low** . but if **below** the line the anomaly is **high** .

TREATMENT

1- High anomalies

- Treated by **1st stage** temporary colostomy,
  2nd stage abdomino-ano- rectal pull through to be followed by **3rd stage** closure of colostomy.

2- low anomalies

- **Local perineal operation** gives excellent results.

6- Volvulus neonatorum

DEFINITION

It is a **volvulus** in the midgut.

AETIOLOGY

Arrested rotation of mid gut predispose to volvulus

PATHOLOGY

The floating caecum, together with small intestine, which has a narrow attachment, revolves in a **clockwise** direction.

CLINICAL PICTURE

(2**V**, 2**C** & 2**D**)

TREATMENT **Immediate surgery**

- The entire midgut should be rotated in **Anti- clockwise** direction.
- **Non-viable gut should be resected**
  (up to **70 %** of small intestine may be removed)

The Peritoneum & mesentery

THE PERITONEUM & THE MESENTERY

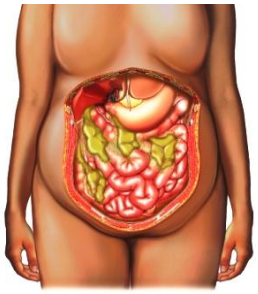
I- THE PERITONEUM

1- Acute septic peritonitis

DEFINITION

Acute inflammation of peritoneal cavity by pyogenic organisms.

AETIOLOGY



A- The organism

Pyogenic organisms such as E. coli, aerobic, anaerobic streptococci & bacteroid ….etc.

B- Rout of entery

1- LOCAL SPREAD :

- **Infected** organs e.g. appendicitis or cholecystitis.
- **Leaking** organs e.g. perforated peptic ulcer or rupture gut.

2- DIRECT ENTERY : as operative or traumatic wounds.

3- BLOOD SPREAD (**rare**) e.g. septicemia or pyaemia.

4- PRIMARY PERITONITIS :

- **Definition :** Peritonitis with **no** apparent intra-peritoneal pathology.
- **Incidence :** Usually affects female children.
- **Organisms :** Streptococci & pneumococci.
- **Pathogenesis :** The infection reach the peritoneal cavity through the Fallopian tubes.

FATE

The fate depend on the virulence of organism on one side & the efficiency of treatment & the body resistance on other side.

SO the end result may be ↓

A. RESOLUTION If good resistance & proper treatment.

B. LOCALIZATION (Abscess formation)

- **Around 1^{ry} focus** e.g. appendicular abscess.
  or **Away** in one of the anatomical peritoneal compartments,
  e.g. iliac abscess, pelvic abscess or sub-phrenic abscess.

C. FLARING UP (Generalized peritonitis) if ↓

① **High** virulence with **low** body resistance.
② **Sudden** perforation of hollow viscus (no time for localization)
③ **Spread** of infection through stimulation of peristalsis e.g. eating.
④ **Immunosuppression** as in D.M.
⑤ **Children** due to small greater omentum which not help in localization of infection.

CLINICAL PICTURE

Symptoms

A- Picture of the cause e.g. acute Appendicitis.

B- Picture of peritonitis

- **PAIN :** which is persistent, dull aching & increased with movement or coughing. The site of maximum pain at original lesion.

- **VOMITING**, **ABDOMINAL DISTENTION** then **ABSOLUTE CONSTIPATION** due to paralytic ileus.

Signs

A- General signs

- **VITAL SIGNS SHOW :** Tachycardia, high fever, hypotension & ↑ respiratory rate..

- **APPEARANCE :** Anxious look with sunken eyes & dry lips.

B- Local signs

- **Inspection** : Restricted movement with respiration.
- **Palpation** : Generalized rigidity & tenderness.
- **Percussion** : Shifting dullness (if pus)
- **Auscultation** : may be dead silent abdomen.

INVESTIGATIONS

A- Laboratory

BLOOD PICTURE shows leucocytosis .

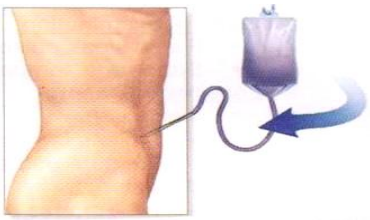
B- Radiological

1- X-RAY ABDOMEN : Demonstrates paralytic ileus & may determine the 1ry lesion e.g. air under diaphragm with perforated ulcer.

2- ABDOMINAL ULTRASOUND : Shows free fluid in the peritoneal cavity.

C- Peritoneal diagnostic aspiration

Which detect the nature of peritoneal fluid.



TREATMENT

A- Pre-operative preparation

① **Ryle's tube** suction to deflate the stomach.

② **I.V fluid** to correct the hypovolemia & electrolyte imbalance.

③ **I.V antibiotics** as a combination of ➪
Ampicillin, **Aminoglycosides** & **Metronidazole**

B- Exploratory laparotomy

① **The pus** is aspirated & the 1^{ry} lesion is dealt with.

② **Peritoneal toilet** (lavage) with large amounts of sterile saline.

③ Closure over peritoneal **drains**.

C- Post-operative care

Admission in an I.C.U

B- T.B peritonitis

AETIOLOGY

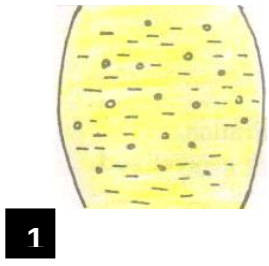
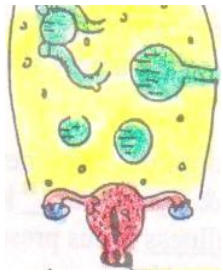
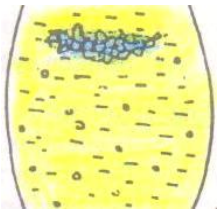
The disease is always 2ry to a T.B focus elsewhere that reaches the peritoneum through ⇘

① **DIRECT SPREAD** e.g. T.B. lymphadenitis & T.B salpingitis.

② **BLOOD SPREAD** e.g. pulmonary T.B.

③ **LYMPHATIC SPREAD** e.g. from bowel or pleura.

PATHOLOGY (**5 types**)

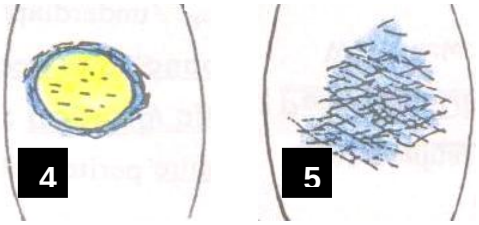
 1	 2	 3
1- Acute milliary type	**2- Casseous type**	**3- Ascitic type**
• The **peritoneum** is studed with T.B. • **Exudates** is straw in color	• The **peritoneum** is studed with T.B. • **Multiple** collection of casseous T.B. **(cold abscess** if pointed ➔ sinus or fistula. • **Mainly** due to T.B salpingitis.	• The **peritoneum** is studed with T.B. • **Ascitic fluid** in a large amount. • The greater omentum is rolled up ➔ sausage shape mass above the umbilicus.

4- Encysted type

It is a localized form of an ascitic type

5- Adhesive type (**Fibrous type**)

It is extensive adhesions with intestinal obstruction.



CLINICAL PICTURE

Type of patient

- **Age :** Children & young adults
- **Sex :** Both sexes.

Symptoms

- **T.B. toxemia** (Night sweat, night fever, loss of weight & loss of appetite).
- **Recurrent attacks** of abdominal pain, distention & vomiting.

Signs

- A **sausage shape** mass above the umbilicus may be felt.
- **Abdomen is felt** doughy with multiple palpable swelling which may be L.Ns,

INVESTIGATIONS

A- Laboratory

1- BLOOD PICTURE : ↑ ESR & lymphocytosis.

2- TUBERCULIN TEST : Highly **+ve** test.

B- Abdominal tapping

IF ASCITIC TYPE shows straw colored fluid rich in lymphocytes.

C- Laparoscopy The choice.

TREATMENT

A- General treatment

Anti-tuberculous drugs at least **1** year.

B. Local treatment

Surgery is indicated in a few cases
e.g. T.B fistula i.e. (casseous type)
or intestinal obstruction (i.e. adhesive type)

.....

C- Peritoneal abscess

INTRA-PERITONEAL ABSCESS : May occur as ⇩

1- Iliac abscess.
2- Pelvic abscess.
3- Subphrenic abscess.

1- Iliac abscess

DEFINITION

Collection of pus in **iliac fossa**.

AETIOLOGY

On the Rt. side

due to acute appendicitis or perforated D.U.

On the Lt. side

due to perforated diverticulitis.

On the both sides

due to tubulo-ovarian abscess or osteomyelitis of iliac bone.

CLINICAL PICTURE

C/P of cause +

Symptoms

① **General :** Toxemia (**F**ever, **h**eadache, **m**alaise & **a**norexia)

② **Local :** Pain at iliac fossa.

Signs

① Tense, cystic mass in **iliac fossa**.

② Tenderness at **iliac fossa**.

INVESTIGATIONS

TOTAL LEUCOCYTIC COUNT + U/S

TREATMENT

Treatment of the cause + **antibiotics** + **drainage** of abscess as ⇘

1- OPEN SURGERY DRAINAGE :
The abscess should be drained through an **extra-peritoneal** muscle cutting incision.

2- PERCUTANEOUS : (**Nowadays**) it is possible to do **percutaneous** drainage of the abscess **guided by U/S** or **CT scan**.

2- Pelvic abscess

DEFINITION

Collection of pus in recto-vesical pouch or douglas pouch.

AETIOLOGY

① Acute appendicitis.

② Localization of resolving diffuse peritonitis.

③ Pelvic inflammatory disease in females.

CLINICAL PICTURE

C/P of cause +

Symptoms

① **General :** Toxemia (**F**ever, **h**eadache, **m**alaise & **a**norexia)

② **Local :** - Deep pain in pelvis, perineum or supra-pubic.
- Diarrhea & passage of mucous in stools (The most characteristic) due to rectal irritation.

Signs

① Tense, cystic mass by **P/R** or **P/V**

② Tenderness by **P/R** or **P/V**.

INVESTIGATIONS

TOTAL LEUCOCYTIC COUNT + U/S

TREATMENT

Treatment of the cause + **antibiotics** + **drainage** of abscess as ⇘

1. IF THE ABSCESS IS POINTING IN RECTUM :
Trans-rectal drainage is recommended

2. IF IT IS POINTING IN VAGINA
The abscess is to be drained through the posterior fornix.

3. IF IT IS POINTING SUPRA-PUBICALLY :
Suprapubic extra-peritoneal drainage is done.

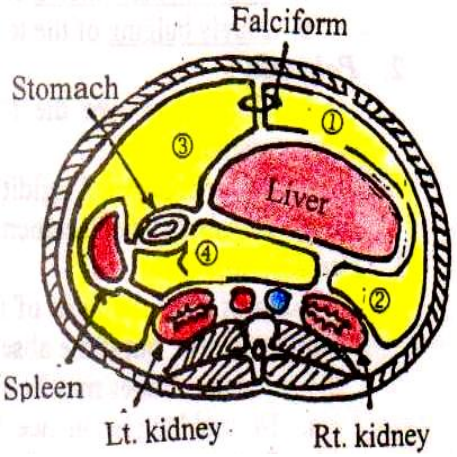
3- Subphrenic abscess

DEFINITION

Collection of pus under the diaphragm.

ANATOMY

- The subphrenic region is considered as a portion of the abdominal cavity.
- The region is **divided by liver into** ⇘
  - ① Rt. anterior intra peritoneal
  - ② Rt. posterior intra peritoneal.
  - ③ Lt. anterior intra peritoneal
  - ④ Lt posterior intra peritoneal :
  - ⑤ Rt. extra-peritoneal space (bare area)
  - ⑥ Rt. perinephric space
  - ⑦ Lt. perinephric space.



AETIOLOGY

I- 4 Intra peritoneal spaces

1- RT. ANTERIOR INTRA-PERITONEAL

- **It lies between** the Rt. lobe of the liver & diaphragm
- **Causes :** Perforated peptic ulcer, cholecystitis & liver abscess.

2- RT. POSTERIOR INTRA-PERITONEAL = Morison or Hepatorenal pouch

- **It lies between** the Rt. lobe of the liver & the Rt. Kidney .
- **Causes :** Perforated peptic ulcer, cholecystitis &. acute appendicitis.

3- LT. ANTERIOR INTRA-PERITONEAL

- **It lies between** the Lt. lobe of the liver & diaphragm
- **Causes :** Post-splenectomy or gastrectomy operations .

4- LT. POSTERIOR INTRA-PERITONEAL (The lesser sac)

- **It lies between** the stomach, the pancreas & Lt. kidney
- **Causes :** Acute pancreatitis & perforated posterior gastric ulcer

II- 3 Extra peritoneal spaces

5- RT. EXTRA-PERITONEAL SPACE

- **It lies behind** the bare area of the liver
- **Causes :** Acute retrocaecal appendicitis, liver abscess, & pyelonephritis.

6 & 7- THE 2 PERINEPHRIC SPACES

- **Causes :** Renal infection

CLINICAL PICTURE

Subphrenic abscess should be suspected if there is evidence of toxemia with **no** apparent cause after upper abdominal operations or pathology

" PUS SOMEWHERE, PUS NO WHERE ELSE, PUS UNDER THE DIAPHRAGM "

Symptoms

C/P of cause +

① **General :** Toxemia (**F**ever, **h**eadache, **m**alaise & **a**norexia)

② **Local :** - Pain at epigastrium or referred to tip of shoulder.
- Persistent hiccough.

Signs

1- Inspection

- Impaired movements of the chest on the affected side.
- Rarely bulging of the lower ribs or upper abdomen.

2- Palpation

- Tenderness over the lower ribs or just below the costal margin.
- Upper abdominal rigidity or swelling.
- Downward displacement of the liver.

3- Percussion

- Dullness over the base of the lung due to pleural effusion is often present, but if the abscess contains gas,
  4 percussion zones may be elicited ⇩
  ① **Resonance** of the lung.
  ② **Dullness** of pleural effusion.
  ③ **Resonance** of gas in the abscess.
  ④ **Dullness** of the liver.

4- Auscultation

- Diminished air entry with basal crepitations on the affected side.

INVESTIGATIONS

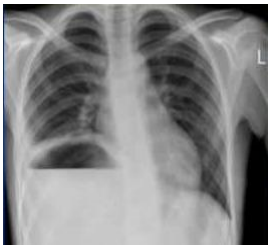
1- TOTAL LEUCOCYTIC COUNT + U/S

2. PLAIN CHEST X-RAY :

① Thickened, elevated & fixed diaphragm (**tented** diaphragm).

② Obliteration of costo-phrenic space by a minima! pleural effusion may be seen.

③ Gas under the diaphragm is sometimes seen when the cause is a perforated viscus, or when there is infection with gas forming organisms.



TREATMENT

Treatment of the cause + **antibiotics** + **drainage** of abscess as ⇩

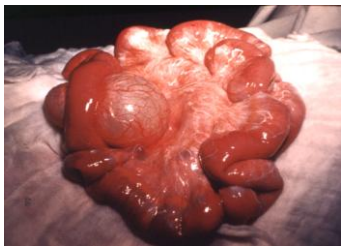
1- IF A SWELLING CAN BE DETECTED :
incision over the site of maximum tenderness.

2- IF NO SWELLING IS APPARENT :
a- Anterior subcostal approach
or **b- Posterior approach**
with resection of the last rib.

3- ASPIRATION GUIDED BY U/S may be done.

II- THE MESENTERY

A- Mesenteric cyst



DEFINITION

It is fluid collection between the layers of the mesentery.

CLASSIFICATION

I. True cyst

1- CHYLOLYMPHATIC : (The commonest variety)

- It is a retention cyst due to obstructed **lymphatic** drainage.
- Distension of sequestrated mesenteric **lymphatics**
- It contains lymph (**chyle**).

2- ENTEROGENOUS :

- It is a cyst between the 2 layers of the **intestinal** mesentery.
- Distension of sequestrated part of the **small** intestine
- It contains **mucous** fluid.

3- TERATOMATOUS DERMOID CYST.

II. False cyst

1- BLOOD CYST (hematoma).

2- T.B cold abscess.

CLINICAL PICTURE

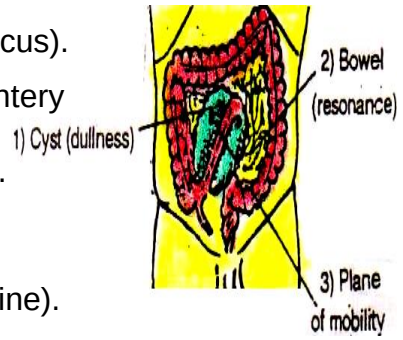
Mesenteric cyst is characterized clinically by a triad of signs

TILLAUX TRIAD

① **A central abdominal swelling** (near the umbilicus).

② **Moves across but not along** the root of mesentery
(the root of mesentery extends from the left hypochondrium to the right iliac fossa).

③ **The mass is dull on percussion**
with an overlying band of resonance
(corresponding to the related loop of intestine).



INVESTIGATIONS

1- ABDOMINAL U/S.

2- ABDOMINAL CT SCAN.

TREATMENT

1- Chylolymphatic cyst

Excision of the cyst, where it has a separate blood supply (not related to the loop of intestine).

2- Enterogenous cyst

Excision with related intestine (resection & anastomosis) where its blood supply from the same blood supply of related loop.

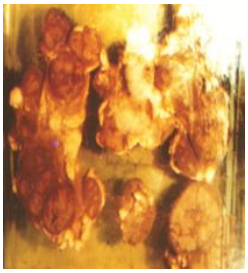
B- Mesenteric lymphadenitis

Tuberculous mesenteric lymphadenitis

Tabes mesenterica

AETIOLOGY

- It is a 1^{ry} tuberculosis of mesenteric lymph nodes usually affecting children after ingestion of contaminated milk



N.B.: 1^{ry} sites of T.B are ⇩

1. Lung
2. Cervical lymph nodes.
3. Mesenteric lymph nodes.
4. Skin.

FATE & COMPLICATIONS

- Usually spontaneous healing occurs
  + calcification of lymph nodes
- Some cases persist
  which are liable for **the following complications :**
  ① T.B peritonitis.
  ② Intestinal obstruction (adhesive obstruction).

CLINICAL PICTURE

Type of patient

- **Age :** Children & young adults
- **Sex :** Both sexes.

Symptoms

- **T.B. toxemia** (Night sweat, night fever, loss of weight & loss of appetite).
- **Recurrent attacks** of abdominal pain, distention & vomiting.

Signs

- **Tenderness** in the right iliac fossa
- **Abdomen** shows palpable irregular, firm lymph nodes.

INVESTIGATIONS

- PAIN X-RAY : may show **calcified** lymph nodes.

TREATMENT

Medical treatment

Antituberculosis drugs.

Surgical treatment

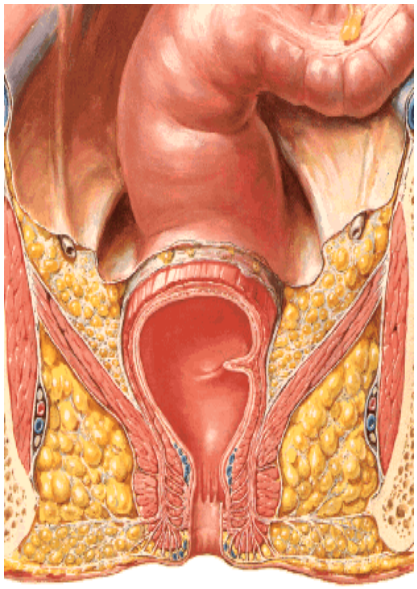
For complicated cases as **intestinal obstruction :** Division of adhesions.

Anal canal

ANAL CANAL

SURGICAL ANATOMY

Anal canal



Length **4 cm**.

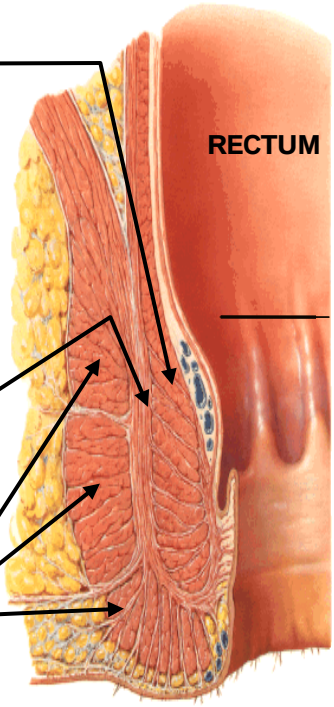
Begins At ano-rectal junction

Ends At the anal verge

A. Ano-rectal ring

- It is the junction between the rectum & the anal canal, it is formed by fused parts of ☟
  - ① **Pubo-rectalis** muscle (part of levator ani).
  - ② **External** sphincter
  - ③ **Internal** sphincter
  - ④ **Longitudinal** muscle.
- It can be felt clinically.

B. Anal canal musculature



1- The internal sphincter

- It is a continuation of the **circular** muscle coat of the rectum.
- It is an **involuntary** muscle)
- It is **2.5 cm** long and **2 - 5 mm** thick
- **Spasm of this muscle :** play a major part in anal fissure

2- The longitudinal muscle

- It is a continuation of the **long** muscle coat of the rectum.
- Its fibers fan-out through the external sphincter to be inserted into the anal & peri-anal skin

3- The external sphincter

- **It is subdivided into ;** 1- deep part
  2- superficial part
  3- subcutaneous part.
- It is a **voluntary** muscle.

✷ Surgical importance

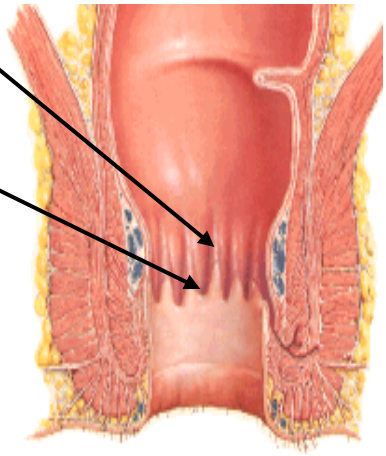
(1) **Internal sphincter** shows spasm in any painful anal condition (e.g. anal fissure) so we must to do internal sphincterotomy

(2) Division of deep part of external sphincter leads to incontinence. So must be avoided.

(3) Above dentate **NO** need for anesthesia

	Internal sphincter	External sphincter
• ***Type of muscle***	Smooth (involuntary)	Skeletal (voluntary)
• ***Innervations***	Autonomic	Somatic
• ***Role in continence***	Responsible for sustained contraction closing anal canal, but has **no role** in voluntary control of defecation.	Contracts voluntary only to inhibit defecation if surrounding conditions are unsuitable.
• ***Effect of injury***	**No** incontinence.	Leads to **incontinence**.

C. The mucosa of the anal canal



1- Anal columns (**Columns of Morgagni**)
- longitudinal folds end in anal valves.

2- Anal valves
-Transversely semi-lunar folds.

D. The dentate line (pectinate line)

- It corresponds to the columns of Morgagni,
- It is about **3 mm**
- It represents the site of fusion of

1- Proctodaeum
Which is invagination of the ectoderm

2- Post allontoic gut
Which is the gut endoderm

	Above dentate line	Below dentate line
- Embryology :	**En**doderm - hind gut	**Ec**toderm - proctodaeum
- Anatomy : **Lining :** **Arterial supply :** **Venous drainage :** **Lymphatic drainage :** **Nerve supply :**	 - Simple columnar. - Superior rectal artery. - Portal vein. - Pelvic & lumbar L.Ns - Autonomic.	 - Stratified Squamous. - Inferior rectal artery. - Systemic vein - Inguinal. L.Ns - Somatic.
- Physiology :	**Less** sensitive	**Excellent** sensation.
- Pathology : **Cancer :** **Piles :**	 - Adenocarcinoma. - **Internal** piles.	 - Squamous cell carcinoma. - **External** piles.

E. Arterial supply
Superior, middle & inferior rectal artery.

F. Venous supply
See (**Piles**)

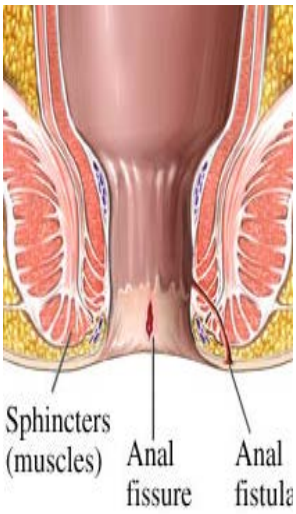
G. Lymphatic supply
See (**Cancer rectum**)

I- ANAL FISSURE

Fissure in ano

DEFINITION

- Elongated ulcer in the long axis (mid line) of the lower end of anal canal i.e. below the dentate line.



INCIDENCE

- Posterior fissure about **90 %**
  because less blood supply & maximum site of trauma by stool
- Anterior fissure 10 %

AETIOLOGY

① Passage of hard stool leading to over distention of anal canal.

② Excessive strain during child birth

③ Dragging of mucosa by a prolapsed pile.

④ Following incorrect hemorrhoidectomy.

⑤ Following bad using of an instrument e.g. speculum.

⑥ **Rare causes** as Crohn's disease.

PATHOLOGY

The anal fissure start as an **acute** then turn to be **chronic** due to ⇦

1- Persistent spasm of the underlying sphincter due to irritation of exposed nerve endings in floor of fissure

2- Continues contamination from passing stools.

3- Progressive Inflammation turn it from superficial ulcer into deep ulcer.

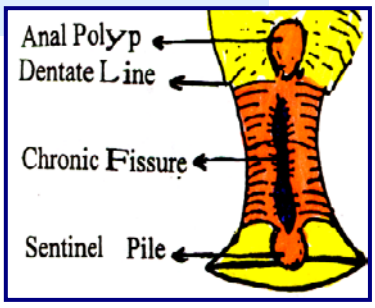
ACUTE ANAL FISSURE	CHRONIC ANAL FISSURE
- **Superficial** ulcer with little inflammation & edema.	- **Deep** ulcer with marked inflammation & edema.
- **Spastic** internal sphincter.	- **Fibrosed** internal sphincter.
- Mobile base **(no** fibrosis).	- Fixed base (**fibrosis**)
- No sentinel pile.	- **Sentinel pile**

N.B SENTINEL PILE

① It is raised (indurated) skin tag.

② It occurs due to edema.

③ It present just below the fissure

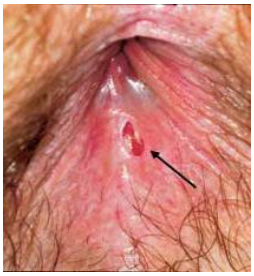


COMPLICATIONS

Abscess, fistula or perianal eczema with pruritis ani.

CLINICAL PICTURE

Symptoms



1- Pain during & after defecation

2- Discharge : slight serosanginous discharge or slight bright blood on stools.

3- Constipation

4- Reflex symptoms as burning micturation, dysmenorrhea ...etc.

Signs

1- Acute fissure is seen & not felt with severe spasm of anal sphincter .

2- Chronic fissure is seen & felt with sentinel pile below.

D.D From painful anal conditions :

① Anal fissure

② Prolapsed strangulated piles.

③ Pruritis ani.

④ Anal carcinoma

⑤ Perianal hematoma (external piles) .

⑥ Peri-anal suppuration.

TREATMENT

The aim is to obtain complete relaxation of internal sphincter.

A- Acute fissure (**Mainly conservative**)

1- Relieve constipation by liquid paraffin & dieting.

2- Local anesthetic ointment e.g. **Lignocain**

3- Warm baths after defecation for **15 minutes**.

B- Chronic fissure (**Mainly surgical**)

1- IF the fissure is (not) very chronic i.e. early

CLOSED LATERAL INTERNAL SPHINCTEROTOMY

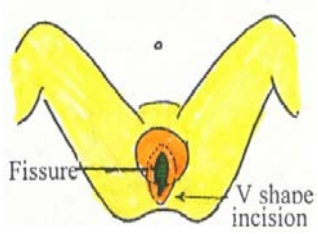
- This operation is very successful.
- The internal sphincter is divided at **3** O'clock position
- This leads to relief of spasm & allow healing.

2- IF the fissure is very chronic i.e. late

FISSURECTOMY

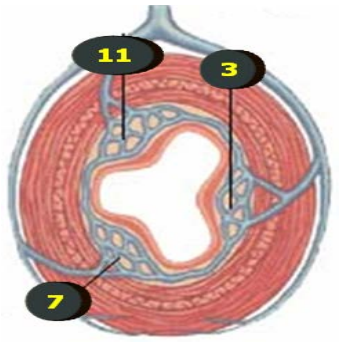
& POSTERIOR INTERNAL SPHINCTEROTOMY

Excision of the fissure, polyp & sentinel pile + division of internal sphincter posteriorly



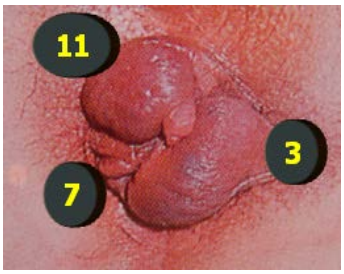
II- HEMORRHOIDS

Piles



Introduction

Normally, the terminal branches of the superior rectal vessels (portal) form a sort of vascular plexus with middle & inferior rectal vessels (systemic) beneath the epithelial lining of anal canal called "Anal cushions" which arranged at **3**,**7**,**11** o'clock positions



SO Any congestion, enlargement & prolapse of this cushions will constitute piles.

1. External piles = below dentate line & covered by skin.
2. Internal piles = above dentate line & covered by mucosa
3. Intero-external piles.

1- Internal piles

AETIOLOGY

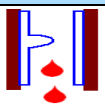
A. Primary piles predisposed by ⇘

- 1- Genetic : Congenital weak mesenchyme .
- 2- Sphincter relaxation due to loss of tone as in old age .
- 3- Anatomical factors :
  - ① The dependency with absence of valves of the tributaries of superior rectal vein.
  - ② The veins lay in lax submucosa & can be easily compressed by stool.
  - ③ The veins are constricted as they pass through the muscle coat.

B. Secondary piles

- 1- Pregnancy : ↑↑ Intra-abdominal pressure & laxing effect of the progesterone.
- 2- Pelvic tumors especially rectal carcinoma (It should be excluded in any patient **> 40** years with bleeding per rectum with evident piles .
- 3- Portal hypertension leading to ano-rectal varices.

PATHOLOGY 4 degrees



1ST DEGREE : The patient has only bleeding but no prolapse of piles. they only diagnosed by proctoscopy.

2ND DEGREE : The piles prolapse only during defecation, but they are spontaneously reduced at the end of the act.

3RD DEGREE : The piles prolapse during defection & the patient has to reduce it manually

4TH DEGREE : There is permanent prolapse of piles.

CLINICAL PICTURE

Symptoms

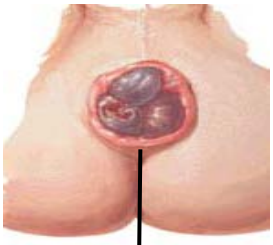
1- Bleeding per-rectum :

Bright red at 1st noticed at toilet paper after defecation, later on becomes profuse

2- Prolapse (swelling) & anal discharge

3- Pain is absent except if complicated

(The patient use the ward pain to mean "**I don't like it**")



Signs

1- Inspection :

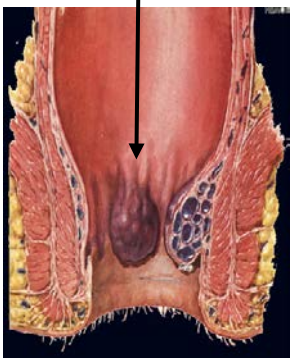
For perianal area for prolapse & skin tags.

2- Palpation :

Not reveal the presence of piles unless they are thrombosed. also to exclude rectal carcinoma

3- Proctoscopy :

will reveal the piles & sigmoidoscopy with old patient to exclude rectal carcinoma.



COMPLICATIONS

1- Bleeding per rectum :

usually mild, moderate or severe leading to anemia.

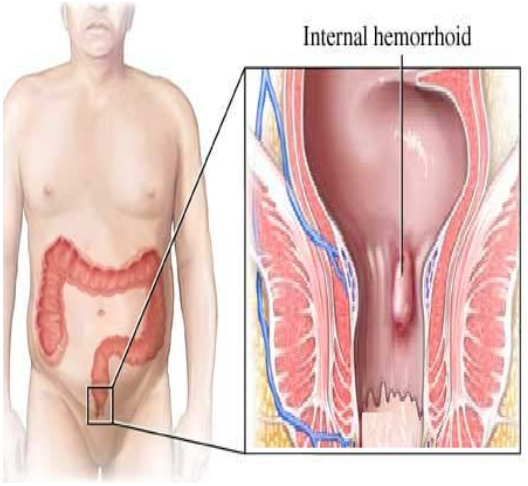
2- Prolapse of piles.

3- Strangulation of prolapsed piles.

4- Thrombosis or ulceration following stagnation.

5- Gangrene if tight strangulation.

6- Suppuration if infected piles leading to portal pyaemia



TREATMENT

⇨ **If primary piles**

1ST & 2ND DEGREE

① **Conservative,**
② **Injection sclerotherapy,**
③ **Rubber band ligation,**
④ **Cryosurgery,**
⑤ **Photocoagulation.**

3RD & 4TH DEGREE

Surgery is recommended. we do **hemorrhoidectomy**
= Trans-fixation excision operation

⇨ **If secondary piles**

Treatment of the cause.

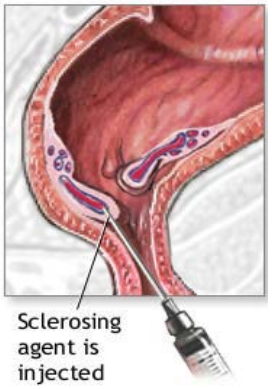
1. Conservative treatment

① Light non-irritant diet.
② Small doses of laxatives.
③ Venous decongestants as suppositories e.g. **Proctocedyl**

2. injection sclerotherapy

AIM Injection of irritant material produce thrombosis which stop the bleeding & prevent prolapse by fibrosis.

METHOD **5** – **20** % **Phenol** in almond oil injected above dentate line. about **3** – **5** cc are injected one each time is injected at one week interval.



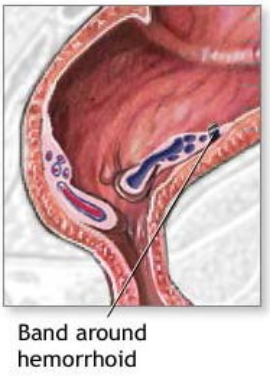
N.B: **No need for anesthesia**
because injection is above dentate line (Non sensitive area)

COMPLICATIONS

① Stricture of rectum due to excessive fibrosis.
② Submucous abscess.
③ Pain if injected too low or too deep in internal sphincter.

3. Rubber band ligation "Barron's"

THE IDEA is to place a tight elastic rubber band around the pedicle of the pillows leading to ischemia, necrosis & separation later on



4. Cryosurgery

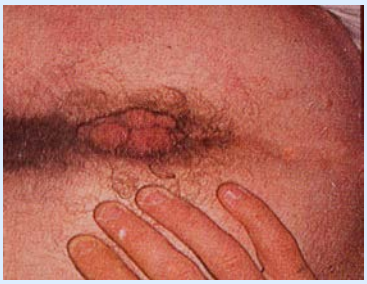
THE IDEA is to apply a liquid nitrogen (**- 196 °C**) causes coagulating necrosis of the piles which separates later. But the disadvantage is that patient has prolonged mucous discharge.

5. Photo-coagulation

THE IDEA is to give infra-red photo-coagulation at (**100 °C**) which cause coagulates necrosis.

N.B.: **Prolapsed strangulated piles**

It is **very dangerous** because act as source of portal pyaemia.



TREATMENT

A- If early diagnosed

Surgical intervention with strong antibiotics .

B- If delayed diagnosed

Conservative measures including rest in bed with foot of bed is raised to ↓ venous congestion, antibiotics, analgesics, laxatives, frequent warm baths & lead subacetate lotion to ↓ edema.

Some surgeons :

Perform **maximal anal dilatation under anesthesia** to relieve Sphincter spasm.

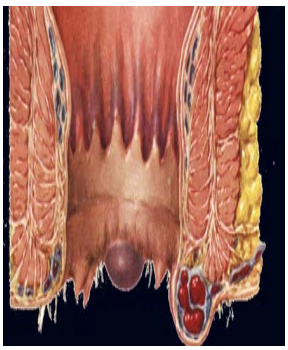
.....

2. External piles

Perianal hematoma

DEFINITION

It is a **small clot** occurring in the perianal subcutaneous connective tissue.



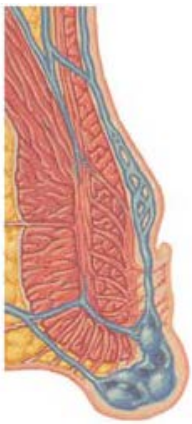
AETIOLOGY

It is due to back pressure on an anal venules as a result of straining at stool, coughing or lifting a heavy weight.

CLINICAL PICTURE

Symptoms

The condition appears suddenly & is very painful.



Signs

Tense, tender bluish swelling covered with smooth shining skin.

FATE

it may resolve or suppurate or fibrose or burst.

TREATMENT

Under local anesthesia,

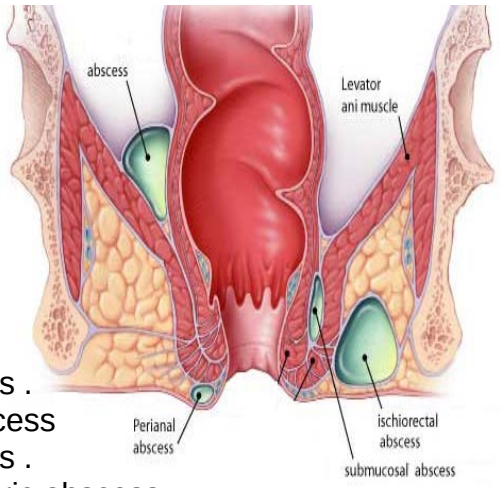
The hemorrhoid is **bisected** & the **2** halves are excised with a small portion of adjacent skin so as to leave a pear shaped wound, which is left open to granulate.

III- ANO-RECTAL ABSCESS

AETIOLOGY

This is either 1ry or 2ry ⇦

A- 1ry ano-rectal abscess



DUE TO infection of the anal glands or 2ry to infected skin.

PATHOLOGY

- Infection of these glands by gram -ve bacilli leads to formation of an inter-sphincteric abscess which may spread ⇦
  - ① **Downward** → Peri-anal abscess .
  - ② **Out-wards** → Ischio-rectal abscess
  - ③ **Inwards** → Submucous abscess .
  - ④ **Upwards** → High inter-sphincteric abscess

- **In the majority of these abscess**, there is an inner opening in the anal canal & drainage of the abscess usually followed by a fistula.

B- 2ry Ano-rectal abscess May developed in ⇦

1- Inflammatory bowel disease as in Crohn's disease
2- Specific infection as T.B .
3- Ano-rectal carcinoma.
4- Infection of peri-anal hematoma.

CLASSIFICATION

1- Peri-anal (**60 %**)

- The abscess lies subcutaneously adjacent to the anal orifice. the pain & toxic symptoms are **not** marked.

2- Ischio-rectal (**30 %**)

- The abscess shows large indurated, tender swelling filling the ischio-rectal space. toxic symptoms marked i.e. throbbing pain.

- If Involve the other side it will lead to (**horse shoe**) abscess.

3- Submucous (**5 %**)

- It lies in submucosa above dentate line. the patient has severe pain & fever but nothing is seen outside the anal verge.
- **P/R** reveals tender **boggy** swelling.

4- Pelvi-rectal (**5 %**)

- It is actually a pelvic abscess 2ry to appendicitis, salpingitis or diverticulitis.
- It is localized above the levator ani muscle.

TREATMENT (**Don't wait for fluctuation**)

Urgent surgical drainage under general anesethia

1& 2 Perianal or ischio-rectal abscesses **Cruciate** incision over the abscess

3- Submucous abscess **Deroofing** through the proctoscope.

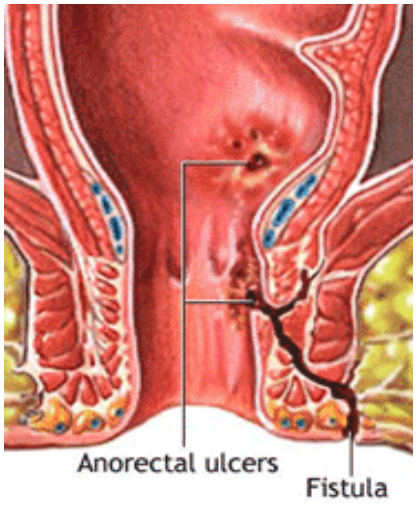
4- Pelvi-rectal abscess According to the cause.

IV- ANAL FISTULA

Fistula in ano

DEFINITION

Chronic granulating track opened between the skin of peri-anal region & the cavity of the anal canal or rectum.



AETIOLOGY

Neglected peria-anal abscess :

4 Factor responsible for chronically

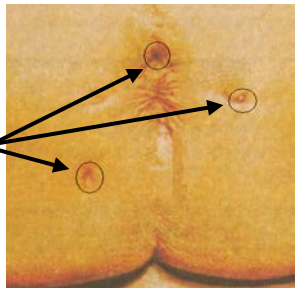
① **1st :** Anal gland act as reservoir for infection.

② **2nd :** presence of internal opening allows recurrent activation of infection.

③ **3rd :** Associated specific pathology such as Crohn's, T,B ……etc.

④ **4th :** Fecal material may, act as foreign body.

CLINICAL PICTURE



1- History of an abscess followed by purulent discharge.

2- Inspection : one or more external peri-anal opening

3- Palpation : Tender indurate cord under skin.

CLASSIFICATIONS

1- Old classification

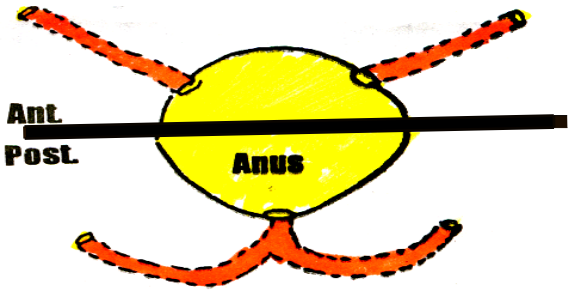
1- Low anal fistulae :

Have their internal opening **below** ano-rectal ring.

2- High anal fistulae :

Have their internal opening **above** ano-rectal ring.

2- Goodsall's rule



- **All fistulae** behind **the transverse anal line :**

Open by a common internal opening posteriorly in the middle line & **curved**.

- **All fistulae** in front **the transverse anal line :**

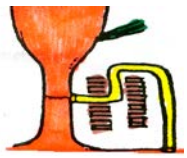
Has its own internal opening & the track is **straight**

3- Recent classification

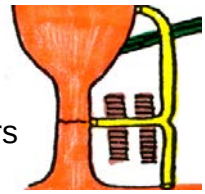
Almost all anal fistulae have their opening at the level of the dentate line of the anal canal communicating to an inter-sphincteric abscess cavity.

1- Inter-sphincteric fistula : (**70 %**)
It passes **between** internal & external sphincters.

2- Trans-sphincteric fistula : (**25 %**)
It passes **through** both internal & external sphincters.

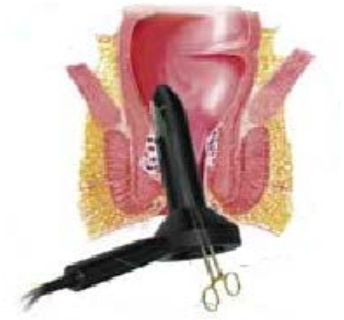


3- Supra-sphincteric fistula : (**4 %**)
It passes **upwards** along the inter-sphincteric plane **then laterally** to above the ano-rectal ring to enter the ischio-rectal fossa to the peri-anal skin.



4- Extra-sphincteric fistula : (**1 %**)
It passes **outside** the plane of external sphincter & They usually have an intermediate communicating track passing through the external & internal sphincters to the internal opening

INVESTIGATIONS

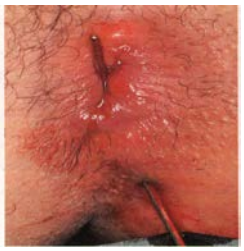


A- Proctoscopy
May show the internal opening of the fistula

B- Colonoscopy ± Barium enema :
May be needed to exclude an underlying specific cause.

TREATMENT

1& 2 Inter-sphincteric or trans-sphincteric fistulae



a- **Fistulotomy** (Opening the fistulous track).
Curettage or cautary of the track then left open to heal by granulation tissue

b- **Fistulectomy** (Excision of the fistulous track)
Not indicated to avoid the risk of sphincter injury.

3- Supra- sphincteric fistula 2 stages

a- **1st stage :** Deroof the lower part of the inter-sphincteric component.

b- **2nd stage :** Excision of the remaining track 2 weeks later

4- Extra-sphincteric fistula

a- Treatment of the underlying cause.

b- Proximal colostomy & staged fistulotomy

V- PILONIDAL SINUS

DEFINITION

Common minor condition of skin overlying sacrum. There is a S.C granulating cavity containing **hair** & connected to skin by midline opening.

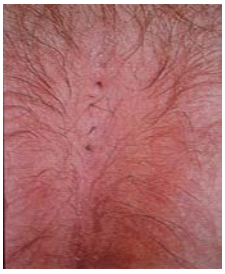
AETIOLOGY **2 Theories**

1- Congenital theory :

Infected dermoid cyst which present after puberty.

2- Acquired theory : (**More accepted**)

Loose hairs from head & back gravitate to the skin over the sacrum & coccyx



CLINICAL PICTURE

Type of patient

young adult males with dense & strong **hair**.

Symptoms

Patient may be asymptomatic or usually present with discharge

Signs

The discharges are seen sometimes loose **hair** comes out of them.

TREATMENT

1- Pilonidal abscess :

- Initially treated by incision & drainage of pus .

2- Pilonidal sinus : **Different options**

- **Opening the resulting wound** is allowed to heal by 2^{ry} intention .
- **Localized excision** of the cavity & side tracks. The wound may be left open to granulate or is closed by sutures

.

VI- FAECAL INCONTINENCE

DEFINITION

Faecal incontinence is defined as the inability to retain the rectal contents .

CAUSES

1. Damage to the anal sphincter :

a. Obstetrical trauma causing complete perineal tear.
b. Surgical trauma, e.g., during surgery for a high anal fistula.

2. Complete rectal prolapse

The prolapsing rectum stretches the anal sphincters damaging them.

3. Idiopathic fecal incontinence.

4. Neurological diseases :

a. Trauma or Tumours affecting the 2nd, 3rd & 4th sacral nerves.
b. Diabetic neuropathy.

ASSESSMENT

1. Manometry :
   To detect rectal function and their strength.
2. Trans-anal ultrasound :
   Can accurately localize the site of sphincteric damage .

TREATMENT

Conservative treatment

a. Constipating agents to thicken the stools.
b. Anal sphincters and pelvic floor exercises.
c. Evacuating the bowel completely,
   with a glycerin suppository in the morning.

Surgery

a. Repair of divided sphincters.
b. **Rectopexy** of those having complete Rectal Prolapse.

VII- ANAL CARCINOMA

PATHOLOGY

- Above dentate ➔ Adenocarcinoma.
- Below dentate ➔ Squamous cell carcinoma.

SPREAD

- **Direct** e.g. rectum
- **Blood :** Mainly to liver
- **Lymphatics :** - Above dentate ➔ Middle & inferior rectal L.Ns
  then to internal iliac L.Ns then to para-aortic L.Ns
  - Below dentate ➔ Inguinal L.Ns.

CLINICAL PICTURE

Symptoms

Anal pain with defecation & **Bleeding per rectum**

Signs

Indurated ulcerated **mass** is felt.

INVESTIGATION

BIOPSY is essential before treatment.

TREATMENT

1- Carcinoma of the anal verge

- **Wide local excision** with **2.5 cm** safety margin.

2- Carcinoma of the anal canal

- **Abdomino-perineal resection** with a terminal colostomy.

Paediatric surgery

I- GENERAL PRINCIPLES

TO BE CONSIDERDBEFORE & DURING PAEDIATRIC SURGERY

1. BODY FLUIDS & ELECTROLYTES

- **Neonates** → 60 ml/kg to be raised by 10 ml/kg daily up to 100 ml/kg.
- **Body weight < 10 kg** → 100 ml/kg.
- **Body weight > 10 kg** → 100 ml/kg for the 1st 10 kg + 50 ml for each additional kg of body weight.

2. BLOOD VOLUME

About 80 ml/kg in neonate
So blood transfusion should be carefully given by a general formula of 10 ml/kg or 5 ml/kg of packed RBCs.

3. RESPIRATORY SYSTEM

(The lungs matures rapidly after birth)
So The most energy consuming activity of the neonate is breathing.

II- TUMORS WITH CHILDREN

I- In order to different age

1- Neonatal period :

① Ovarian cyst lesion.
② Metastatic neuroblastoma
③ Sacro-coccygeal teratoma

2- In 1st 2 years of life :

① Neuroblastoma.
② Nephroblastoma
③ Hepatoblastoma

2- Between 2 - 6 years of life :

① Ovarian teratoma..
② Maignant lymphoma
③ Rabdomyo-sarcoma

II- In order to frequency

1- Acute lymphocytic laukaemia
2- Brain tumours
3- Neuroblastoma
4- Nephroblastoma (Wilm's tumor)
5- Hepatoblastoma
6- Rhabdomyosarcoma
7- Sacro-coccygeal teratoma

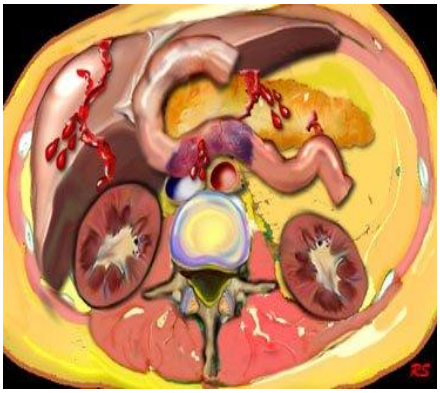
Review subjects

REVIEW SUBJECTS

I- ABDOMINAL INJURIES

EXCITING CAUSE "**Trauma**" which may be ⇩

A- Closed trauma



① **Direct trauma :**
Blunt trauma e.g. car accident.
② **Indirect trauma :**
Fracture ribs or pelvis
③ **Spontaneous rupture :**
Rare with pathological diseases

B- Opened trauma

① **Gunshot** wounds.

② **Punctured** due to stabbing.

③ **Iatrogenic**

PATHOLOGY Which may be ⇩

1- **Subcapsular** hematoma.

2- **Superficial** tear or tears.

3- **Deep** tear or tears.

4- **Avulsion** of a pole.

5- Complete **pulping**

6- **Injury** of vascular pedicle

CLINICAL PICTURE

A- History of trauma

To abdomen or lower chest followed by an abdominal pain ± shock.

B- Examination

May reveals signs of internal Hge & shock + signs of external trauma as bruises.

INVESTIGATIONS

1. Sonar & CT scan (**abdomen**)

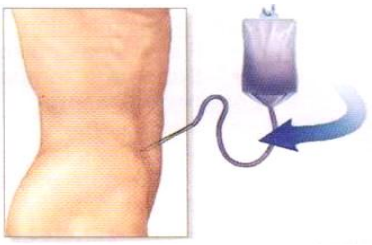
2. Diagnostic peritoneal lavage "**D.P.L.**"

INDICATIONS

1. **Suspicion of organ injury** with equivocal signs.
2. **Unreliable abdominal examination** because patient is unconscious, e.g., head trauma, or drug or alcohol intoxication.
3. **Unexplained hypotension** that may be caused by blood loss.

TECHNIQUE

- Done by inserting an intra-peritoneal catheter at umbilicus under local anesthesia & infusing **500 cc** saline in the peritoneal cavity. if saline returns bloody = intra-peritoneal bleeding.



CONTRAINDICATIONS

1. Pregnancy. 2. Liver cirrhosis. 3. Severe obesity.

TREATMENT

A. Anti-shock measures

- Remember **ABCDE**
- Blood transfusion, warmth, morphia ……etc.

B. Immediate laparotomy "**The priority is to arrest bleeding**"

ANESTHESIA **General** anesthesia .

POSITION **Supine** position

INCISION **Midline incision** from xiphisternum to symphysis pubis.

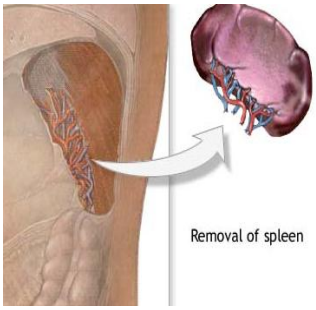
STEPS - **Exploration** of all abdominal viscera starting with solid organs, any escaping bowel contents denote hollow viscus injury.

- **Injured organs are dealt with as follows** ➮

1- Spleen :

Splenectomy or splenic preservation especially in children as it plays an important role in immune mechanism especially against pneumococci.

THE PRESERVATION THROUGH



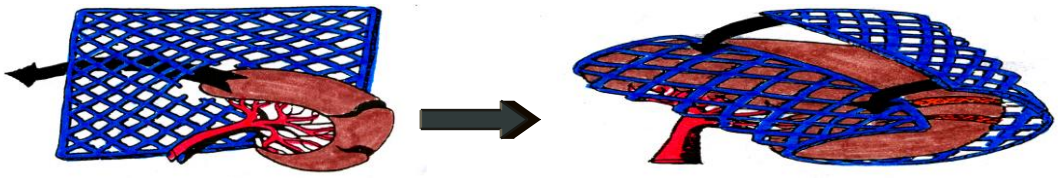
① **Sutures** of small lacerations or tears

② **Partial splenectomy** if avulsed one pole.

③ **Auto-transplantation** of splenic fragments Which is wrapped by omentum.

④ **Therapeutic embolization** through the splenic artery by gel foam ➔ splenic infarction

⑤ **Splenic mesh wrap :** Placing the injured spleen in the center of the mesh & sewing both ends of the mesh together to tamponade the bleeding.



N.B.: Pneumococcal anti-toxin (Pneumovax)
Should be given in young child up to 18 years of age after splenectomy

2- Liver :

We control the liver hemorrhage a combination of **temporary packing** of the bleeding area & application of the " **Pringle's maneuver** "

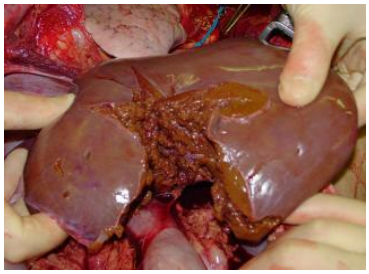
Then THERE ARE 3 POSSIBILITIES

⇨ **If the tear is accessible**,

- It is repaired by deeply placed mattress sutures supported by a pad of peritoneum.

⇨ **If the tear is inaccessible**,

- The incision should be extended into the chest along the **8th** inter-costal space.
- The aim is to arrest bleeding, to remove the necrotic devitalized tissue, to preserve as much viable tissue as possible & to do external drainage.



⇨ **If there is extensive hematoma or ruptured liver tissue**

- We must do "**hepatic resection**" then drainage of peritoneal cavity to avoid peritonitis then prophylactic A.B are prescribed.

3- Stomach :

Tear is sutured or the damaged part is excised & the defect is closed.

4- Duodenum :

Tear is sutured & **a catheter** can be inserted into the duodenum to prevent its narrowing.

5- Pancreas :

Tear is sutured or if the tail is injured, **distal pancreatectomy** is done.

6- Small & large intestine :

- **Small intestine & Rt. side of colon :** Resection & anastomosis
- **Lt. side of colon :** Colostomy

7- G.B & C.B.D :

- Cholecystectomy then tear is sutured to C.B.D on T-tube

8- Kidney :

- If avulsed pole ➔ partial nephrectomy.
- If avulsed pedicle ➔ Nephrectomy provided that the other one is good.

9- Urinary bladder :

Tear is sutured with supra-pubic tube to keep the bladder empty.

The abdomen is closed over drains

II- ACUTE ABDOMEN

DEFINITION

- It is a term applied to any abdominal pain requiring urgent relief.

CLASSIFICATIONS

I- According to the mode of presentation

1- Colics e.g. intestinal, appendicular & biliary .

2- Inflammation e.g. appendicitis, cholecystitis &pancreatitis,

3- Perforations e.g. perforated appendix & peptic ulcer

4- Intestinal obstruction e.g. simple & strangulation

II- According to the causes

1- Gastrointestinal causes :

- Acute perforated peptic ulcer.
- Acute cholecystitis.
- Acute pancreatitis.
- Acute appendicitis.
- Acute diverticulitis.
- Intestinal obstruction.
- Mesenteric vascular occlusion.

2- Urological causes :

- Calculi of urinary Tract.
- Inflammation of urinary tract.

3- Internal hemorrhage

4- Gynecological causes :

- Mid menstrual pain.
- Dysmenorrhea.
- Rupture ovarian cyst.
- Twisted ovarian cyst.
- Tubal abortion.
- Rupture ectopic pregnancy.
- Pelvic inflammatory disease (**PID**).

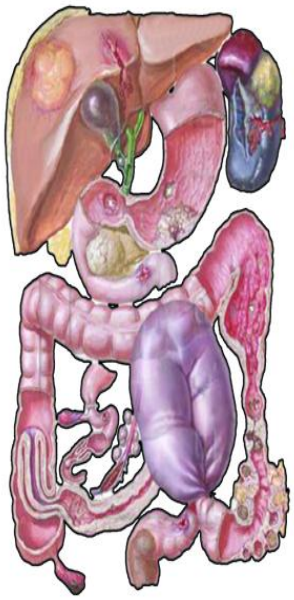
5- Other causes :

- Basal pneumonia & pleurisy.
- Myocardial infarction.
- Severe gastritis & duodenitis.
- Hepatitis.
- Diabetic ketoacidosis.
- Uremia.
- Hemolytic crisis.

III- According to site

1- Upper abdominal :

- Perforated peptic ulcer.
- Acute cholecystitis & biliary colic.
- Acute pancreatitis.
- Mesenteric vascular occlusion.
- Acute myocardial infarction.



2- Mid-abdominal :

- Intestinal obstruction.
- Mesenteric vascular occlusion.

3- Rt. lower abdomen :

- Acute appendicitis.
- Meckel's diverticulitis.
- Crohn's disease.
- Torsion undescended testis.
- Rt. ureteric colic

4- Lt. Lower abdomen :

- Colonic diverticulitis.
- Colitis
- Torsion undescended testis.
- Lt. ureteric colic

5- Pelvic :

- Pelvic inflammatory disease (**PID**).
- Complicated ovarian cyst.
- Pelvic appendicitis
- Rupture ectopic pregnancy.
- Proctitis
- Cystitis

6- Abdominal & back pain :

- Acute pancreatitis
- Rupture abdominal aortic aneurysm.
- Posterior duodenal ulcer penetrating pancreas.
- Renal & ureteric colic.

DD OF ACUTE ABDOMEN

A. History

1- Age :

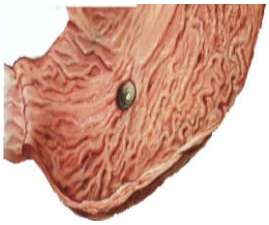
- **Neonate** e.g. Neonatal intestinal obstruction.
- **Child** e.g. Strangulated hernia.
- **Adult** e.g. Appendicitis, cholecystitis, peptic ulcer … etc.
- **Old age** e.g. Ischemic colitis … etc.

2- Sex :

If female think about ectopic pregnancy, rupture ovarian cyst,.. etc.

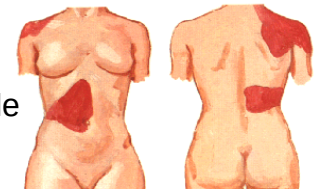
B- Examples

1- Acute perforated peptic ulcer :



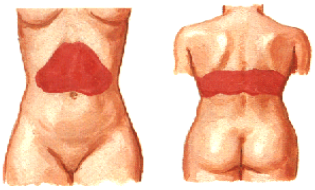
History of ulcer in (**80** %), sudden onset of severe agonizing pain in the epigastrium, followed with short period of (lucid Interval) followed by picture of peritonitis

2- Acute cholecystitis :



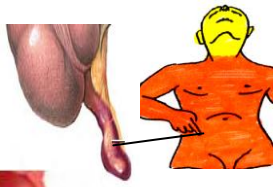
History of pain at Rt. hypochondrium associated with fever & radiated to Rt. shoulder & inferior angle of Rt. scapula, ↑↑ by fatty meals & ↓↓ by anti-spasmodics.

3- Acute pancreatitis :



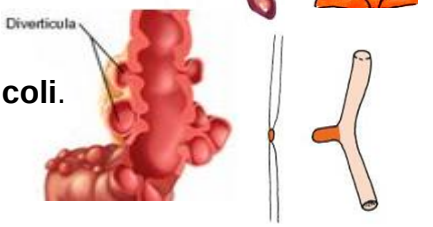
History of gall stones or alcohol drinks, with severe agonizing pain at epigastrium & radiated to back, ↑↑ by on flat position & ↓↓ by leaning forwards.

4- Acute appendicitis :



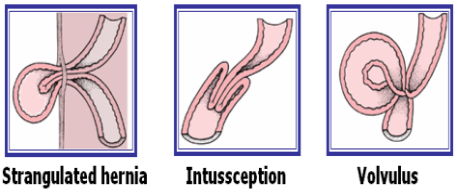
History of pain around umbilicus, shifted to Rt. iliac fossa with nausea or vomiting.

5- Acute diverticulitis :



History of colicky pain at sigmoid colon of old constipated patient i.e. **diverticulosis coli**. or history of colicky pain similar to acute appendicitis i.e. **Meckel's diverticulitis**.

6- Intestinal obstruction :



History of colicky pain, vomiting, distension & absolute constipation.

7- Rupture ovarian cyst :

History of severe lower abdominal pain tenderness & guarding with no toxemia.

8- Torsion of ovarian cyst :

History of severe lower lateral abdominal pain with palpable adnexal mass.

9- Pelvic inflammatory disease (PID) :

History of sever lower abdominal pain with high toxemia suspected with female in the reproductive period. IUD increases the possibility.

10- Rupture ectopic pregnancy :

History of sever lower abdominal pain with patient with high risk factors as salpingitis, tubal ligation, tubal repair or I.U.D.

C- General examination

- Dehydration.
- Shock.
- ±Toxemia

D- Local examination

(Inspection, palpation, percussion, auscultation, PR & PV)
especially for ↬

1- Murphy's sign = **Cholecystitis**

2- Tenderness & guarding = **Peritonitis**

3- Tender Mc burney's = **Appendicitis**

4- Pointing sign by asking the patient to point of maximum pain

E- Investigations

1- Laboratory :

- Leucocytes (inflammation)
- Urine analysis
- Serum amylase (pancreatitis)
- Serum electrolytes.

2- Radiological (plain x-rays)

- Examples ↬

- **Perforated peptic ulcer =** Air under diaphragm
- **Intestinal obstruction =** Multiple fluid levels
- **Urological causes =** Calculi is seen in **80 %** of cases.
- **Rupture spleen =** Loss of psoas shadow + fracture rib, obliterated splenic outline, Indentation of gastric air bubble.

3- Abdominal ultrasonography :

- Examples ↬

- **Acute cholecystitis =** Enlarged G.B with thick wall, ulcerated mucosa, contains thick bile & adhesions
- **Free fluid =** Internal hemorrhage or peritonitis
- **Acute pancreatitis**, **abscess** or **pseudo cyst**
- **Rupture liver** or **spleen**
- **Intestinal obstruction =** distended bowels with fluid and gas, free fluid if perforation occurs.
- **Ureteric colic =** it may show back pressure on the kidney, renal or ureteric stones
- **Gynecological conditions =** Ectopic pregnancy, ruptured ovarian cyst.

4- Abdominal CTscan :

- Examples ↬

- **Acute pancreatitis**
- **Retroperitoneal hematoma**.

III- HAEMATEMESIS

DEFINITION

Vomiting of blood which will be of dark red in colour, acidic in reaction, associated with food particles & may be preceded by vomiting & followed by melena .

CAUSES OF HEMATEMESIS

Usually due to lesion **above** duodeno-jejunal junction.

I- General causes

Hypertension, purpura or hypoprothrombinaemia.

II- Local causes

- **OESOPHAGEAL CAUSES :**
  - ① Oesophageal varices.
  - ② Oesophageal carcinoma.
  - ③ Oesophagitis (reflux)
- **GASTRO-DUODENAL CAUSES :**
  - ① Chronic peptic ulcer
  - ② Gastric carcinoma.
  - ③ Acute gastric erosions & acute peptic ulcer

In Egypt **The commonest 3 causes are** ↓

- ① Oesophageal varices
- ② Chronic peptic ulcer
- ③ Acute gastric erosion.

	HEMATEMESIS	HAEMOPTSIS
HISTORY	G.I.T troubles.	Chest troubles.
PRECEDED BY	Vomiting.	Cough.
FOLLOWED BY	Melena.	Blood stained sputum.
THE BLOOD	Dark red, acidic with food particles.	Bright red, alkaline with frothy sputum.

	BLEEDING ULCER	BLEEDING VARICES
HISTORY	Dyspepsia relieved by antacids.	Liver cirrhosis & hepatosplenomegaly
ENDOSCOPY	**+ve** ulcer.	**+ve** varices.
PORTAL PRESSURE	Normal pressure	High pressure
SENGESTAKEN TUBE	No effect.	Control bleeding

MANAGEMENT

Investigations & treatment for ↓

1- OESOPHAGEAL VARICES → Look for **portal hypertension**

2- CHRONIC PEPTIC ULCER → Look for **stomach**

MANAGEMENT OF ACUTE UPPER G.I BLEEDING

A- Estimation of severity of bleeding & resuscitation

⇨ **Endotracheal intubation** should be considered to avoid aspiration pneumonia. **Then** ⇩

① Insert **2 peripheral venous lines** & withdraw blood for cross-matching.

② A **central venous catheter** is needed for monitoring of severs cases.

③ Insert a **foley's catheter** for urine output

④ A **Naso-gastric tube** is inserted for all cases.

⑤ If the patient is irritable give, 5-10 mg **Morphia IV** to sedate him, but contraindicated in cases of liver insufficiency.

⑥ **Ringer's lactate** are started until blood available.

⑦ **Correct coagulopathies** by I.V vit. K.

B- Localization of the site & cause of bleeding

1- History :

- Previous attacks & their management.
- Hepatitis & schistosomiasis.
- Medications, particularly NSAIDS.
- Peptic ulcer symptoms.

2- Examination for splenomegaly, ascites & liver.

3- Laboratory tests :

- **Hg %** & **haematocrite** value.
- **Liver function tests** will be disturbed in patient with cirrhosis and oesophageal varices.
- **Kidney function tests** as blood urea & creatinine.

4- Fibreoptic endoscopy :

Endoscopy is the most important test. It should be performed as early as possible once the patient has been resuscitated. The procedure is done under a mild sedative as diazepam.

5- Barium radiography :

Rarely done because it is less accurate than endoscopy.

6- Angiography :

In difficult situation where radiology or endoscopy fails to diagnose the lesion.

C- Treatment of specific lesions

1- OESOPHAGEAL VARICES ➔ Look for **portal hypertension**

2- CHRONIC PEPTIC ULCER ➔ Look for **stomach**

IV- BLEEDING PER RECTUM

DEFINITION

Passage of blood per rectum may be ⇩

1- Occult bleeding per-rectum .
2- Melena.
3- Fresh bleeding per-rectum (**Hematocheizia**)

TYPES & AETIOLOGY

I- Occult bleeding per-rectum

- **Aetiology :** may be any cause of internal hemorrhage.

- **It means** passage of small microscopic quantity of blood in stools that cannot be detected by gross appearance.

- **It is diagnosed** chemically only as by "**Benizidine test**"

II- Melena

- **Aetiology :** All causes of **hematemesis +**
  ① Meckel's diverticulum
  ② Crohn's disease
  ③ Bleeding typhoid ulcer.

- **It means** black tarry stools from lesion **above** Ileo-caecal valve.

- **It must be to differentiated from** patient which ingest **iron**, **charcoal** or who treated by **Bismuth**

.

III- Fresh bleeding per-rectum

A- AETIOLOGY

I- General causes

Hypertension, purpura or hypoprothrombinaemia.

II- Local causes

- **ANAL CANAL CAUSES :**
  ① Piles.(The commonest cause)
  ② Anal fissure.
  ③ Anal Carcinoma

- **COLORECTAL CUASES :**
  ① **Congenital :** Familial polyposis coli.
  ② **Inflammatory :** Amoebic or Bacillary desentery.
  ③ Bilharzial colitis. ulcerative colitis.
  ④ **Vascular :** Ischemia colitis.
  ⑤ Diverticular disease of colon.
  ⑥ **Neoplastic :** Cancer colon or rectum.
  ⑦ **Angiomatous malformation** of colon.

DD OF FREASH BLEEDING PER RECTUM

A. History

1- Age :

- **Neonate** e.g. Juvenile polyps.
- **Child** e.g. Meckel's diverticulum.
- **Adult** e.g. Piles or anal fissure.
- **Old age** e.g. Malignancy.

2- Related to pain :

- **Bleeding + pain :**
  - ① Anal fissure
  - ② Anal carcinoma.
- **Bleeding + No pain :**
  - ① Blood **mixed** with stool = Cancer colon.
  - ② Blood **Streaked** on stool = Cancer rectum.
  - ③ Blood **after** defecation = Piles
  - ④ Blood & **mucous** = (ulcerative & bilharzial) Colitis
  - ⑤ Blood **alone** = Diverticulitis coli.
  - ⑥ **Melena** = Peptic ulceration.

B- Examples

1- Anal fissure :

Painful bleeding, bright red & pain lasting long after passing stool

2- Cancer Rt. colon :

Recent change in bowel habits (diarrhea) & blood mixed with stool. Athenia is a common presentation.

2- Cancer Lt. colon :

Recent change in bowel habits (progressive constipation not respond to purgative) & blood mixed with stool.

3- Cancer rectum :

Sense of incomplete defecation with blood streaked on stool
2^{ry} piles may be associated.

4- Piles :

Blood after defecation at toilet paper

5- Colitis :

Bilharzial or ulcerative

6- Diverticulosis coli :

Predisposed usually with constipation.

C- General examination

- Pallor.
- Shock.

D- Local examination

- **P/R** for (Piles, fissure, polyps)

E- Investigations

1- Laboratory :

- **Hg %** & **haematocrite** value.
- **Stool** analysis.

2- Radiology :

- **Abdominal U/S :**
  for liver, ascites, masses.
- **Trans-rectal U/S :**
  for small lesions

3- Instruments :

- **Endoscopies** (the most important investigations)
- **Proctoscopy**, **sigmoidoscopy** or **colonoscopy**

4- Exploration :

Laparoscopic or **open** are the last investigations after failure of all previous investigations.

TREATMENT OF FREASH BLEEDING PER RECTUM

In 90% of the cases

The conservative measures stop bleeding & elective surgical treatment according to the causes is carried out.

In 10% of the cases

The bleeding is massive or continued.
So one of the followings may be done ☜

1- Therapeutic colonoscopy :

- **Polypectomy**
- **Electrocoagulation**
- **Diathermy**
- **LASER**
- **Injection of vasopressors**
- **Embolization**.

2- Surgery :

- **Segmental resection** of the bleeding site if the source of bleeding is clearly identified.
- **Subtotal or total colectomy** if the source of bleeding is not clearly identified.

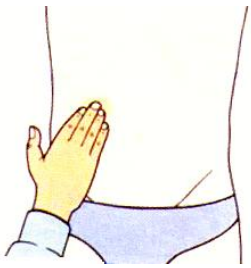
V- DD OF ABDOMINAL SWELLINGS

1- RIGHT HYPOCHONDRIAL SWELLINGS

A. Abdominal wall masses

e.g. Lipoma, abscess, fibrosarcoma of muscles ... etc

B. Intra-abdominal masses



1- Liver :

1. Localized, mass due to ⇩

① Amoebic liver abscess.
② Hydatid cyst.
③ 1^{ry} carcinoma e.g. hepatoma.
④ 2^{ry} deposits of liver.

2. Diffuse mass due to ⇩

① Bilharzial liver fibrosis (the commonest)
② Hepatitis.
③ Obstructive jaundice.
④ Congestive liver due to heart failure.

2- Gall bladder :

① Mucocele or pyocele (empyema)
② Carcinoma of gall bladder.

3- Rt. kidney & Rt. supra-renal gland :

① Hydronephrosis.
② Polycystic kidney.
③ Renal tumors.
④ Supra-renal mass (rare)

4- Colon : (**Hepatic flexure**) as carcinoma.

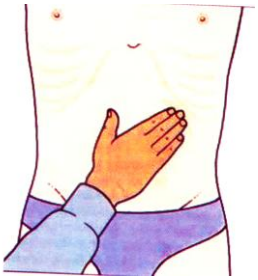
The commonest : Gall bladder mass & hepatomegaly.

2- LEFT HYPOCHONDRIAL SWELLINGS

A. Abdominal wall masses

e.g. Lipoma, abscess, fibrosarcoma of muscles ... etc

B. Intra-abdominal masses



1- Spleen : causes of splenomegaly (discuss)

2- Stomach : e.g. carcinoma.

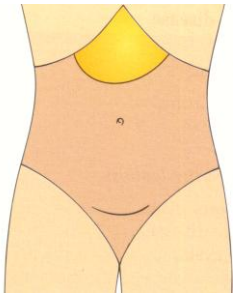
3- Lt. kidney & Lt. supra-renal gland :

① Hydronephrosis.
② Polycystic kidney.
③ Renal tumors.
④ Supra-renal mass (rare)

4- Colon : (**Hepatic flexure**) as carcinoma.

The commonest : Splenomegaly & renal mass.

3- EPIGASTRIC SWELLINGS



A. Abdominal wall masses

e.g. Lipoma, abscess, fibrosarcoma of muscles.

+ Epigastric hernia

B. Intra-abdominal masses

1- Stomach : Pyloric obstruction or cancer.

2- Lt. lobe liver : Tumor or cyst.

3- Transverse colon : Carcinoma or intussusception.

4- Pancreas : Cancer head pancreas or pseudo-cyst

5- Aorta : Aneurysm or para-aortic L.Ns.

4- LUMBAR SWELLINGS

A. Abdominal wall masses

e.g. Lipoma, abscess, fibrosarcoma of muscles.

+ Lumbar hernia

B. Intra-abdominal masses

1- Colon : Carcinoma

2- Kidney : Hydronephrosis, polycystic kidney, renal tumors.

3- Retro-peritoneal mass

5- UMBILICAL SWELLINGS

A. Abdominal wall masses:

e.g. Lipoma, abscess, fibrosarcoma of muscles.

+ Umbilical hernia, malignant nodules or desmoid tumor.

B. Intra-abdominal masses:

1- Transverse colon : Carcinoma or intussusception.

2- Mesenteric cyst .

3- Retro-peritoneal mass

6- SUPRA-PUBIC SWELLING

A. Abdominal wall masses:

e.g. Lipoma, abscess, fibrosarcoma of muscles.

+ Urachal cyst.

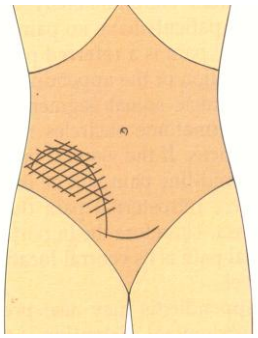
B. Intra-abdominal masses:

1- Urinary bladder : Cancer bladder or urinary retention .

2- Uterine : Pregnancy or fibroid .

3- Retro-peritoneal mass

7- RIGHT ILIAC FOSSA SWELLINGS



A. Abdominal wall masses

e.g. Lipoma, abscess, fibrosarcoma of muscles ... etc

B. Intra-abdominal masses

1- Appendix : Appendicular mass & abscess.

2- Caecum : Cancer caecum.

3- Ileo-caecal : Ileo-caecal T.B & crohn's disease

4- Iliac L.Ns : Acute iliac lymphadenitis.

5- Iliac artery : Aneurysm.

6- Iliacus muscle : Myosarcoma & fibrosarcoma.

7- Psoas abscess (rare)

8- Iliac bone : Tumors or osteomyelitis.

9- Kidney : Ectopic kidney or renal transplant.

10- Genital organs :

- **Male :** undescended testis
- **Female :** fibroid, ovarian cyst or tubal ectopic pregnancy.

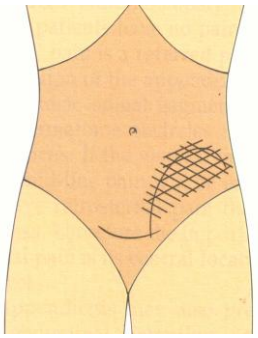
11- Retro-peritoneal mass

The commonest swellings are:

① Appendicular mass & abscess .

② Cancer caecum

8- LEFT ILIAC FOSSA SWELLINGS



A. Abdominal wall masses

e.g. Lipoma, abscess, fibrosarcoma of muscles.. etc

B. Intra-abdominal masses

1- Sigmoid colon : Bilharzial peri-colic mass, cancer sigmoid, pelvic diverticulitis.

2- Iliac L.Ns : Acute iliac lymphadenitis.

3- Iliac artery : Aneurysm.

4- Iliacus muscle : Myosarcoma & fibrosarcoma.

5- Iliac bone : Tumors or osteomyelitis.

6- Kidney : Ectopic kidney or renal transplant.

7- Genital organs :

- **Male :** undescended testis
- **Female :** fibroid, ovarian cyst or tubal ectopic pregnancy.

8- Retro-peritoneal mass

The commonest swellings are:

① Bilharzial peri-colic mass .

② Cancer sigmoid

VI- POSTOPERATIVE

COMPLICATIONS OF ABDOMINAL INCISIONS

1. **Hematoma.**
2. **Infections.**
3. **Desmoid tumor.**
4. **Incisional hernia.**
5. **Burst abdomen**

BURST ABDOMEN

➢ **Aetiology :**

Failure of abdominal wound to heal (see incisional hernia)

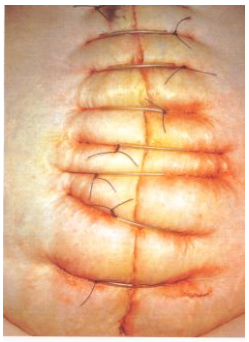
➢ **Pathology :** (at **6 - 8^{lh}** post-operative day)

- 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.

VII- POSTOPERATIVE

ABDOMINAL DISTENSION & VOMITING

1. **Acute gastric dilatation.**
2. **Paralytic ileus.**
3. **Intra-abdominal abscess,** e.g. sub-phrenic or pelvic.
4. **Peritonitis** due to a leaking anastomosis.
5. **Adhesive intestinal obstruction.**
6. **Medical causes** as uraemia or diabetic keto-acidosis.

VIII- POSTOPERATIVE

PYREXIA

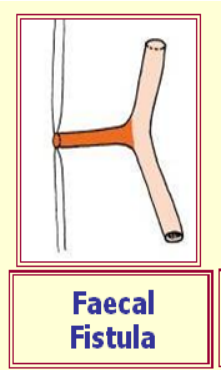
1. **In the first 24 hours :**
   May occur as a reaction to the stress of the operation.
2. **In the first few days :**
   The commonest cause of pyrexia is respiratory complications, usually atelectasis and pneumonia.
3. **Wound infection.**
4. **Deep vein thrombosis.**
5. **Thrombophlebitis** at the site of a peripheral cannula
6. **Urinary tract infection** especially in the presence of a catheter.
7. **Intra-abdominal abscess.**

IX- DISEASES OF THE UMBILICUS

1. Umbilical fistula

I- Fecal fistula :

- Congenital : Patent vitello-intestinal duct,
- Traumatic
- Inflammatory : T.B of small intestine
- Malignant : Carcinoma of the transverse colon ulcerating through the umbilicus.



II- Urinary fistula : Is either congenital from patent urachus or is rarely acquired.

III- Biliary fistula : Is due to perforation of an inflamed gall bladder.

2. Umbilical sinus

Discharging pus from umbilical infection.

3. Umbilical stone

4. Umbilical polyp

5. Umbilical granuloma

6. Umbilical hernia

7. Tumors of umbilicus

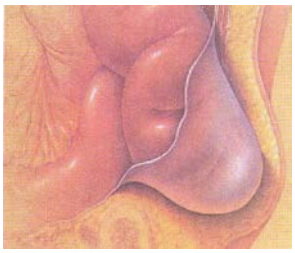
I- Squamous cell carcinoma.
II- Secondary nodules.

Hernia

HERNIA

DEFINITION

- **Hernia** is a protrusion of a viscus or part of a viscus usually within a peritoneal sac through a defect in the abdominal wall
- **Clinically ;** painless swelling characterized by ⇩
  - Reducible or gives history of reducibility
  - Gives expansile impulse on cough.
  - On the anatomical site of hernia defect



TYPES

1- Inguinal hernia

- **above** inguinal ligament **(above** groin crease**)**

2- Femoral hernia

- **below** inguinal ligament **(below** groin crease**)**

N.B.: [1] & [2] called **groin hernia**

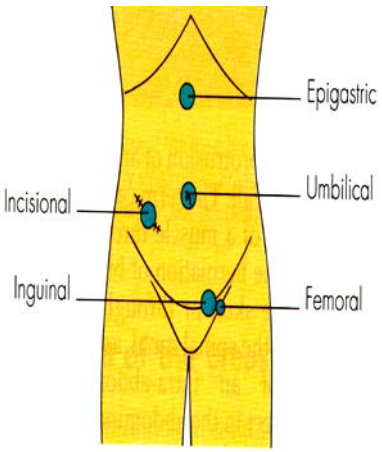
3- Umbilical hernia

- **midway** between xiphisternum & symphysis pubis

4- Epigastric hernia

- **away** from umbilicus at site of linea alba.

N.B : [3] *&* [4] called **abdominal hernia.**

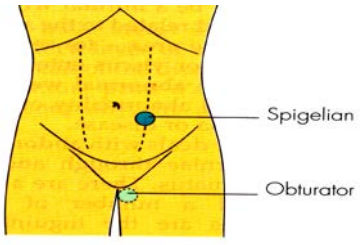


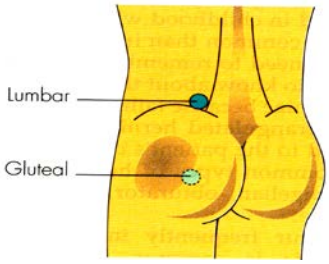
5- Incisional hernia

- **paralytic type :** due to injury of the nerve supplying the overlying muscles
- **defective type :** due to defect in the repair of abdominal incision.
  e.g. infection, using absorbable sutures.

6- Others

- **Rare sties of hernia as** ⇩





OBTURATOR HERNIA through obturator foramen.

LUMBER HERNIA through lumbar region.

GLUTEAL HERNIA through greater sciatic foramen.

SCIATIC HERNIA through lesser sciatic foramen.

SPIGELIAN HERNIA through spigelian fascia

AETIOLOGY

A- Congenital

due to presence of a **congenital performed sac.**

① Unobliterated processus vaginalis (**congenital O.I.H**).

② Unobliterated physiological umbilical hernia of the fetus (**exomphalos**)

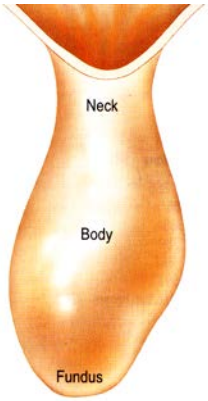
B- Acquired due to ⇩

1- ↑ INTRA ABDOMINAL PRESSURE

- **Chronic straining** due to chronic cough, constipation …etc.
- **Abdominal swelling** due to pregnancy, ascites or organomegaly.
- **Occupational** as porters.

2- WEAKNESS OF ABDOMINAL WALL

- **Obesity** because fats separate the muscle bundles
- **pregnancy** due to stretching of the abdominal wall.
- **Abdominal operations** i.e. abdominal scars.



COMPONENTS

A- Sac

- This is the peritoneal pouch which bulges out through The abdominal wall defect. It has a neck, body & fundus

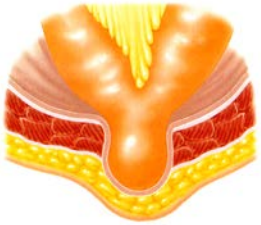
B- Contents

- It may be any abdominal viscus **except the pancreas**. (being retroperitoneal) the most common are ⇩

	ENTEROCELE	OMENTOCELE
• **Consistency** • **Reducibility**	• Soft • **1st part** difficult, because of gases & show **gurgling**	• Doughy • **Last part** difficult, because of adhesion of sac & omentum. & not show **gurgling**
• **Percussion** • **Palpation**	• Resonant • Lobulated surface	• Dull. • Smooth surface

Special contents

1. RICHTER'S HERNIA - a portion of circumference of intestine. →
   - it occurs with femoral hernia.

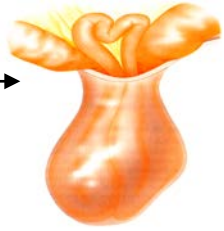


3. LITTRE'S HERNIA

   The content is **Meckel's diverticulum.**

2. MAYDL'S HERNIA

   It contains 2 loops of the bowel **(hernia in W)** →
   while the intermediate loop lies in peritoneal cavity.

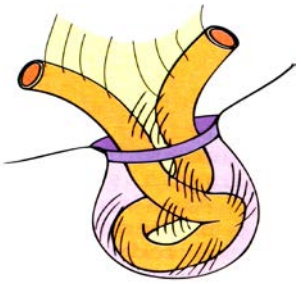


C- Coverings

- Structures stretched over the sac.

COMPLICATIONS OF HERNIA

1. Irreducibility



DEFINITION

Failure to return the contents into the abdomen.

AETIOLOGY

- Adhesions between the contents & the sac.
- Adhesions between the contents themselves.
- Protrusion of new content during strain.

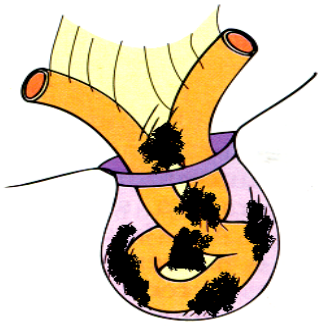
D.D

	Irreducible Hernia	Obstructed Hernia	Strangulated Hernia
• Acute obstruction	-	+	+
• Impulse on cough	+	+	-
• Tense & tender	-	-	+

TREATMENT

Irreducibility increases the risk of obstruction & strangulation.
this means early operation (must be according to type of hernia)
then cut of adhesions & reduction of hernia.

2. Obstruction



DEFINITION

Occlusion of intestinal lumen from outside (adhesions) or inside (fecal material) but the blood supply is still **unaffected**

AETIOLOGY

Usually 2ry to irreducibility.

CLINICAL PICTURE

Symptoms of acute intestinal obstruction
(colics, vomiting, distention & **absolute** constipation)

DD

	Irreducible Hernia	Obstructed Hernia	Strangulated Hernia
• Acute obstruction	-	+	+
• Impulse on cough	+	+	-
• Tense & tender	-	-	+

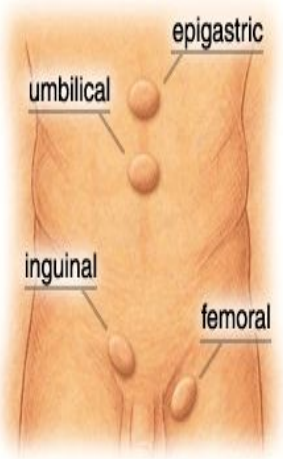
TREATMENT

Early surgery as DD between it & strangulation is very difficult.

3. Strangulation

DEFINITION

The hernia becomes strangulated when the blood supply of its content is **seriously impaired**.



INCIDENCE

[A] It varies according to the type of hernia

- Inguinal **2 – 4 %**
- Femoral **25 – 30 %**
- Para-umbilical **15 – 20 %**
- Incisional **3 – 5 %**

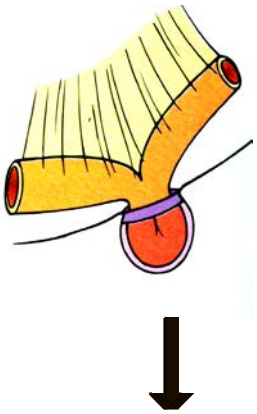
[B] Although the incidence is higher in femoral hernia yet strangulated inguinal hernia account for more than 50 % (as it is more common)

[C] Strangulation occur at any age & commoner after prolonged use of **truss**

PREDISPOSING FACTORS

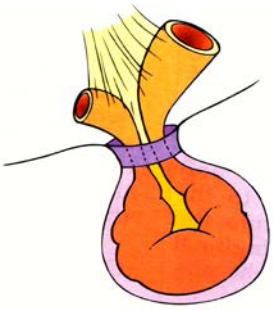
1. Irreducibility & obstruction.
2. Sudden expulsion of new contents following straining.
3. Repeated attempts at reduction producing oedema

PATHOLOGY



A- The constricting agents

- **Any resistant structures** outside the sac like ⇘
  - Superficial or deep ring with **inguinal** hernia
  - Sharp edge of lacunar ligament with **femoral** hernia
  - Defect in linea alba with **para-umbilical** hernia
- **Narrow neck** of the hernia sac.
- **Bands of adhesions** within the sac.



B- The contents

- Constricting agents will compress the veins in the intestinal mesentery (thin walled) **1st** → ↑ Venous pressure → oedema & congestion of intestinal loops. if the congestion is marked increased → hemorrhage in intestinal wall & lumen
- **Further** rise of pressure → impairment of arterial blood supply so the contents lose their vitality & may be undergo gangrene.
- **Finally**, gangrene occurs, it starts at ring of constriction then affects the anti-mesenteric border of intestine. if perforated → peritonitis.

N.B.: Neglected cases will die from septic shock & dehydration

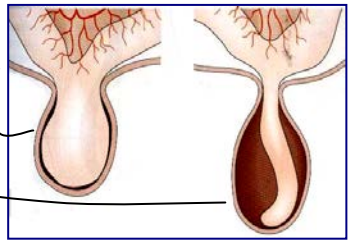
CLINICAL PICTURE

(A) General examination

- Manifestations of acute intestinal obstruction
  (colics, vomiting, distention & **absolute** constipation)
- Hypovolaemic shock & signs of dehydration
- If gangrene occurs → peritonitis → paralytic ileus i.e. no colics
  which is " **bad** sign " → finally septicemia → septic shock.

N.B.: Manifestations of (A.I.O) are **absent** with1. Strangulated **omentum**.

2. Strangulated **Richter's** hernia.
3. Strangulated **Littre's** hernia.



(B) Local examination

" **Cardinal signs** of strangulation "

1. Tense & tender
2. Irreducible
3. **No** impulse on cough.

DD

	Irreducible Hernia	Obstructed Hernia	Strangulated Hernia
• Acute obstruction	-	+	+
• Impulse on cough	+	+	-
• Tense & tender	-	-	+

TREATMENT

A- Immediate resuscitation

- **Ryle's tube** for suction + no oral intake.
- **I.V fluids** to correct electrolyte imbalance
- **I.V blood & Ringer's lactate** to correct hypovolaemia.
- **I.V broad spectrum A.B**. to guard against peritonitis.

B- Taxis

TECHNIQUE **(Better avoided)**

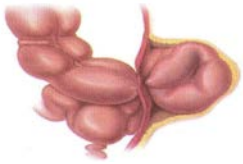
- **Reduction by manipulation,**
  this done within the 1st 6 hours after the onset

COMPLICATIONS

- **Shock**
- **Rupture of intestine**
- **Reduction en masse** i.e the sac & strangulated contents are reduced as one mass into the abdomen
- **Peritonitis** due to squeezing of harmful fluid of sac to the peritoneal cavity

C- Immediate operation

- **Incision** should be planned to expose the fundus of sac & open it to evacuate toxic fluid 1st.

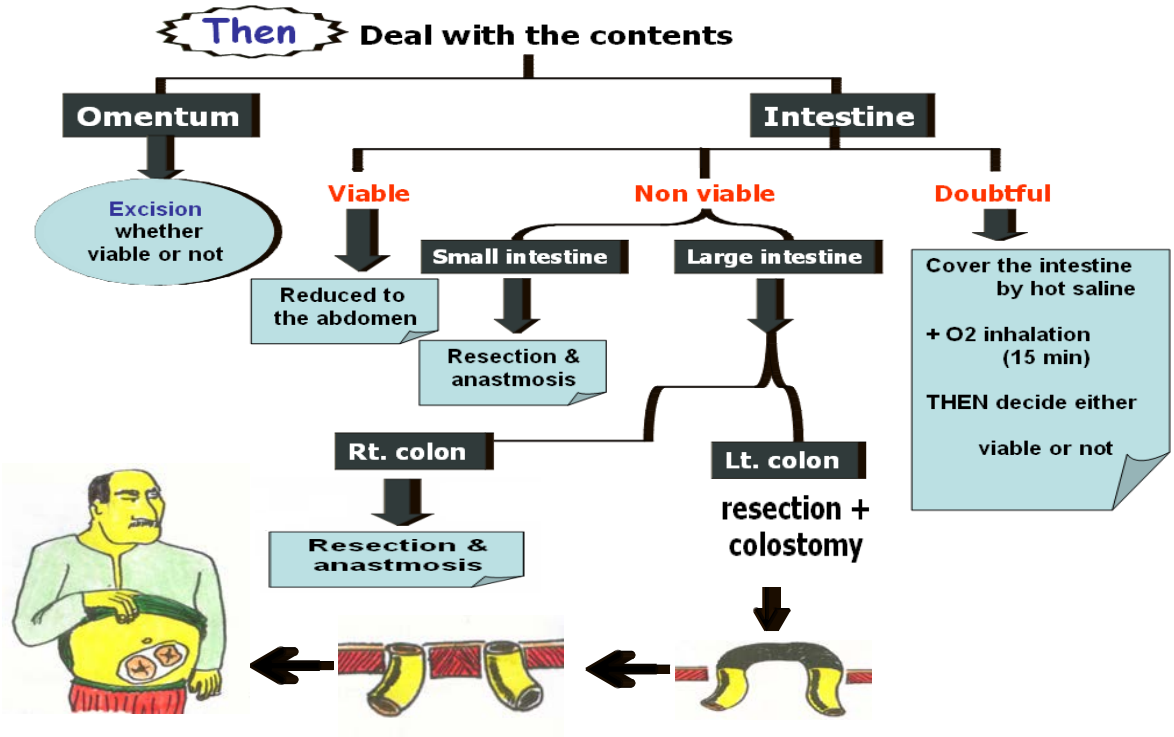


- **The constricting agents** should be divided over a grooved director or the left finger to avoid injury of intestine.
- **The contents** are pulled out & examined, viable or not

	Viable intestine	Non-viable intestine
• Intestinal color • Peritoneal luster • Mesenteric arteries	• Pink or dark red • Present. • Pulsating	• Grey or black. • Absent. • Non pulsating
• By pinching • Consistency • If injured	• Contracts • Firm • Bleeding occur	• No response • Floppy • No bleeding

D- Intraoperative tests (To detect viability)

1. **Doppler ultrasound**
2. **Fluroscein test :** Inject 1 gm fluroscein I.V then inspect the bowel under U.V. rays. If the bowel has good blood supply it will flurescence
3. **Oximeter**



- **N.B :** IN CASE OF LT. COLON
  We do resection & colostomy then preparation of colon then closure after one week.
- **Repair the hernia** defect by prolene sutures (non absorbable)
- **Subcutaneous drain** is usually used.

4. Inflammation

DEFINITION

It means inflammation of the contents.

AETIOLOGY

1. Rough manipulations.
2. Ill fitting truss → repeated rough friction.
3. Inflammed contents (appendix or meckle's diverticulum)

CLINICAL PICTURE

The hernia is **tender** but **not tense** and overlying skin is red & oedematous.

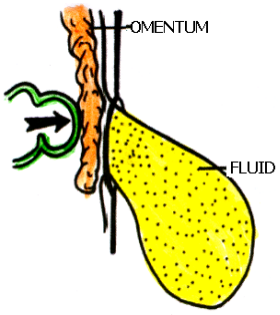
TREATMENT

Operation is essential as strangulation cannot be excluded.

5. Hydrocele of the hernia sac

AETIOLOGY

When narrow necked sacs becomes occluded by omentum or adhesions after reduction of its contents → collection of serous fluid in the sac.



CLINICAL PICTURE

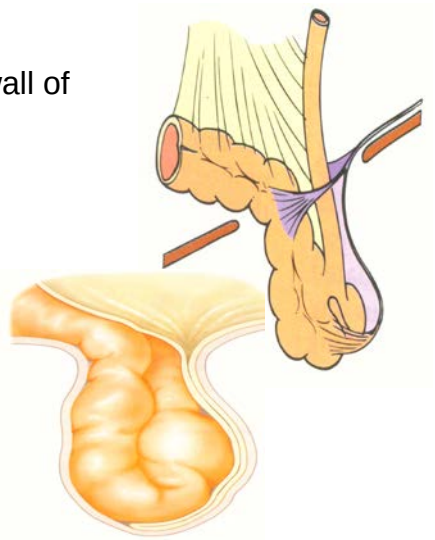
Cystic translucent swelling which is **tense** but **not tender**.

TREATMENT Excision

6. Torsion of the omentum

7. Rupture of the hernia sac

SLIDING HERNIA



DEFINITION

This is a hernia where a viscus form a part of the wall of the sac & not part of the contents.

INCIDENCE

1. Common with long standing hernia & old males
2. The commonest sliding organs are
   urinary bladder, caecum & sigmoid colon.

CLINICAL PICTURE

- **Old** male with **incomplete** reducible complete inguino-scrotal hernia.
- **Urinary symptoms** (double micturation) if sliding urinary bladder.

TREATMENT

- Do not try to dissect the sliding viscus from the sac as this may lead to devascularisation or injury of the viscus.
- Free the sliding sac & viscus well from the surrounding structures & push them back then good repair is done as hernioplasty.

SURGICAL ANATOMY OF THE INGUINAL REGION

Layers of abdominal wall

Skin

Superficial fascia

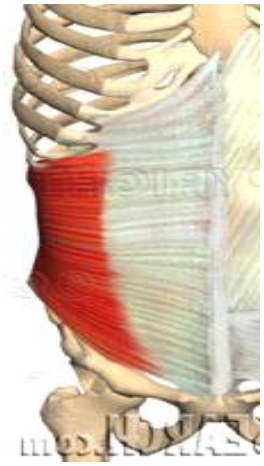
- **Superficial** fatty layer (**Camper's** fascia)
- **Deep** membranous layer (**Scarpa's** fascia)

3 Muscles

1. **External oblique muscle**

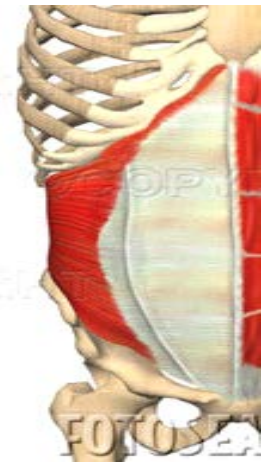
- **Origin :** from outer surface of lower 8 ribs.
- **Insertion :** Xiphoid process, linea alba, A.S.I.S, outer lip of iliac crest & pubic tubercle.
- **Surgical importance**
  - Fibers run downwards, forwards & medially
  - The lower part becomes aponeurotic & it's free border enfolded as inguinal ligament
  - **External** (**superficial**) **inguinal ring** is an opening of external oblique aponeurosis.

2. **Internal oblique muscle**



- **Origin :** from lateral **1/2** of upper surface of inguinal ligament.
- **Insertion :** lower 6 costal cartilage, xiphoid process & linea alba.
- **Surgical importance**
  - Fibers run upwards, forwards & medially
  - The lower fibers inserted as **conjoined tendon** with transversus abdominis muscle into the pubic crest & pectineal line.

3. **Transversus abdominis muscle**



- **Origin :** from lateral **1/3** of upper surface of inguinal ligament & lower 6 costal cartilage.
- **Insertion :** Xiphoid process & linea alba.
- **Surgical importance**
  It is inserted as **conjoined tendon** with internal oblique muscle.

Fascia transversalis

- Thin but strong fascial layer lies in front of the peritoneum
- **Internal** (**deep**) **inguinal ring** is an opening of fascia transversalis

.

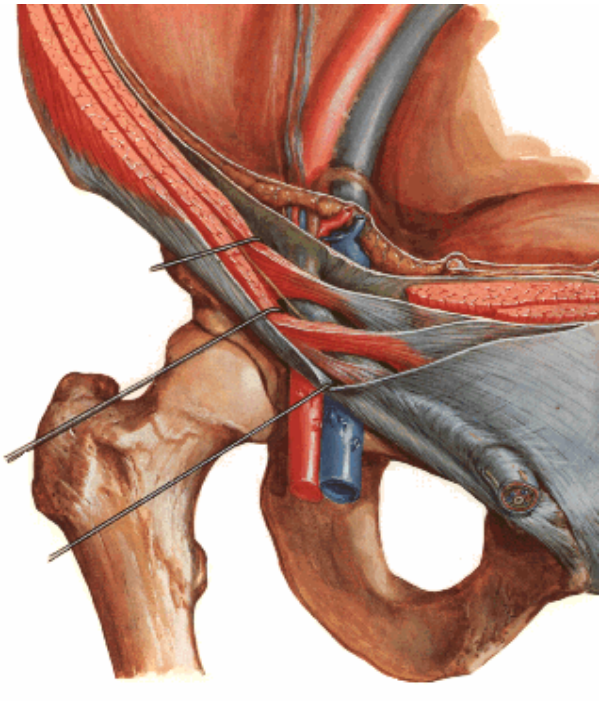
INGUINAL CANAL

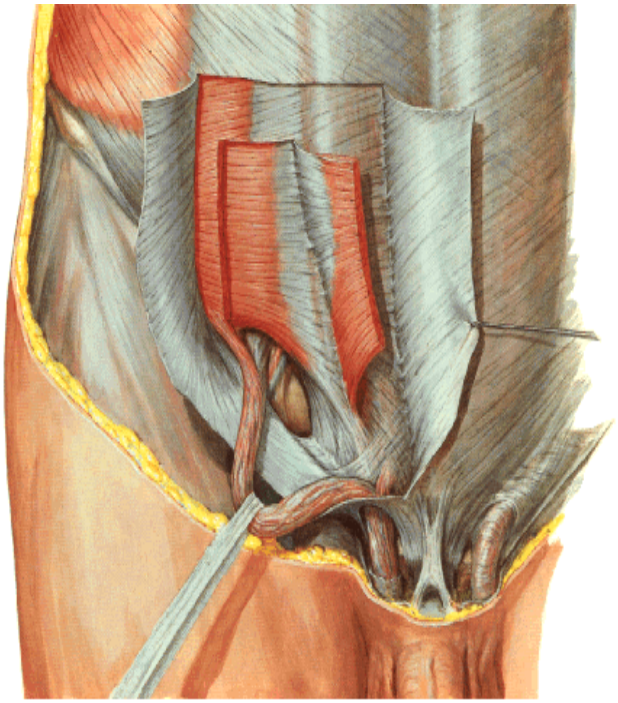
DEFINITION

- It is an oblique passage in the lower part of the anterior abdominal wall, through which the testis passes in order to leave the abdomen & reach the scrotum

LENGTH & SITE

- Its length about **1.5 inches** (**4 cm**) and passes downward, forward & medially from deep to superficial ring, it lies parallel to the medial half of the inguinal ligament





BOUNDARIES

Beginning

- **Deep inguinal ring :**
  - An opening of fascia transversalis
  - **1/2** inch above the **mid inguinal point**
    (point midway between A.S.I.S & symphysis pubis)
  - It is an **inlet** for spermatic cord.
  - The inferior epigastric vessels run **medial** to it.

N.B.: Mid-point of inguinal ligament
(point midway between A.S.I.S & the pubic tubercle)

End

- **Superficial inguinal ring :**
  - A triangular opening of external oblique aponeurosis
  - **1/2** inch above & medial to pubic tubercle.
  - It is an **exit** for spermatic cord & ilio-inguinal nerve.

Anterior wall

- External oblique aponeurosis.
- Lower parts of internal oblique muscle **laterally**.

Posterior wall

- Fascia transversalis.
- Conjoined tendon **medially**.

Floor

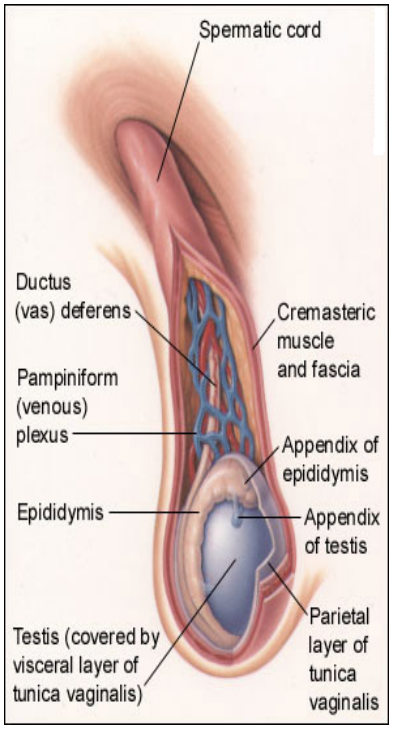
- **Upper grooved surface** of inguinal ligament

Roof

- **High arched** fibers of conjoined tendon

CONTENTS

- **Ilio-inguinal nerve**
  which pierces the posterior wall of the canal then passes through superficial inguinal ring.
- **Spermatic cord** if male
  or **round ligament** If female

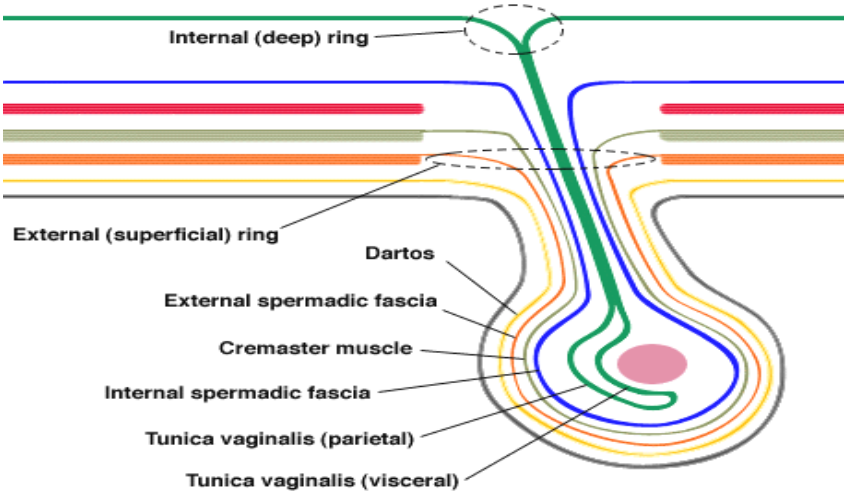


Contents of the spermatic cord

- **Vas** deferens.
- **Artery** of vas & testicular artery.
- . **Vein** of vas & pampiniform plexus.
- Autonomic **nerves**.
- Testicular **lymphatic**
- **Vestige** of processus vaginalis.
- Genital branch of genito-femoral nerve.

Coverings of the spermatic cord

- **External spermatic fascia** from external oblique aponeurosis.
- **Cremasteric muscle** from internal oblique muscle.
- **Internal spermatic fascia** from fascia transversalis.



Surgical importance

FACTORS PREVENT INGUINAL HERNIA

1. **Inguinal canal is oblique**

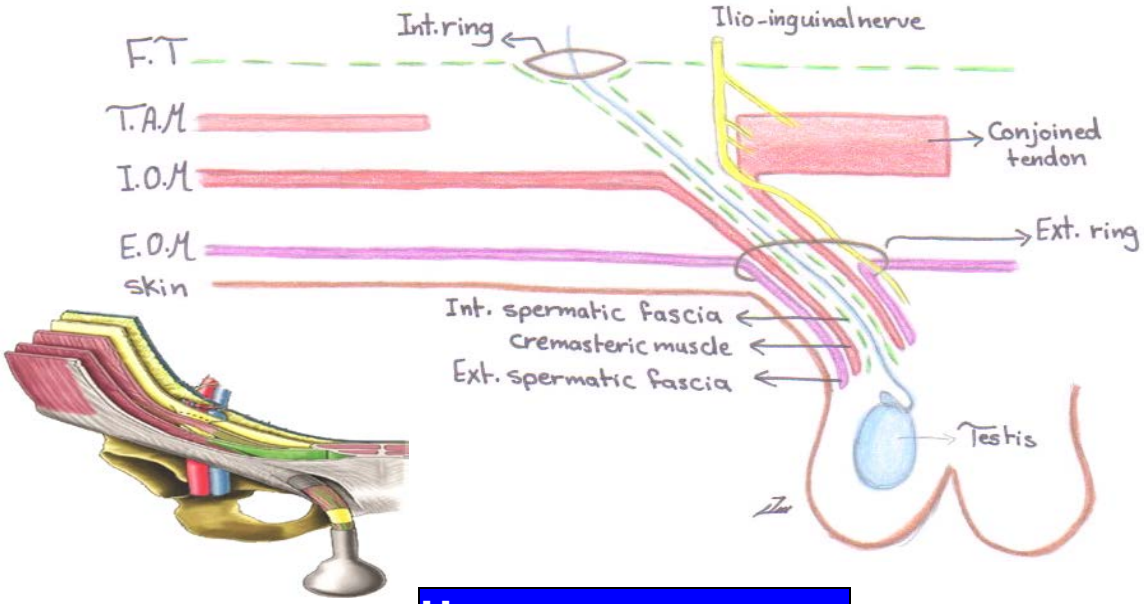
   So the internal ring & external ring not at same plane.

2. **During ↑ intra-abdominal pressure The followings occur ⇘**

   - Contraction of **anterior abdominal wall** so that the anterior & posterior wall of inguinal canal are approximated.
   - Contraction of **external oblique muscle** leads to narrowing of superficial ring.
   - Contraction of **cremasteric muscle** leads to elevation of scrotum so closure of superficial ring occur.

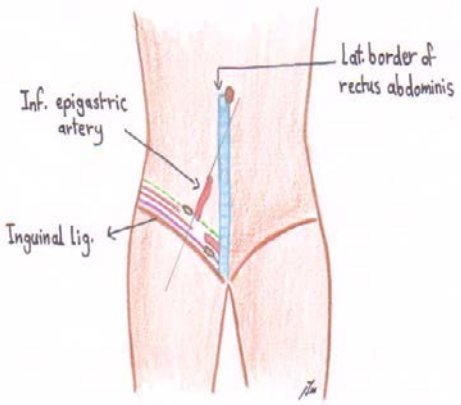
3. **Shutter mechanism**

   - Due to **triple** relation of the lower fibers of **internal oblique muscle** to the inguinal canal. In the (anterior, roof & posterior wall)
   - So that contraction of this muscle during strain leads to closure of inguinal canal around the spermatic cord.



HASSELBACH TRIANGLE

- Direct inguinal hernia protrude through this triangle



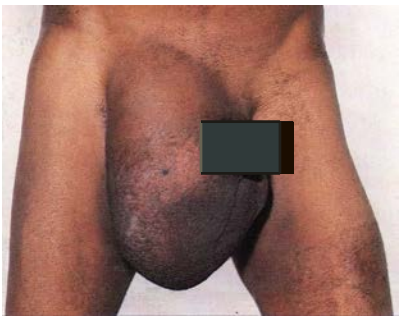
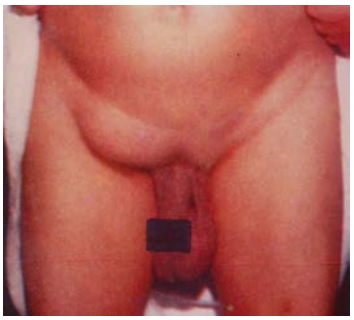
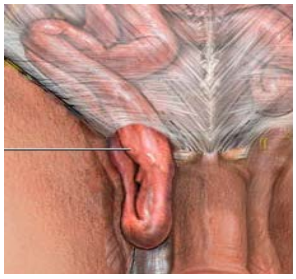
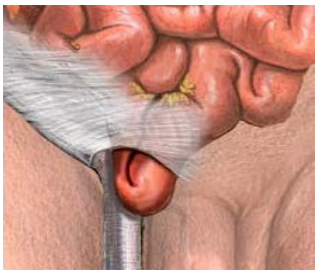
Site of Hasselbach triangle

medial part of posterior wall of inguinal canal

The Boundaries

- **Medially :** lateral border of rectus sheath.
- **Laterally :** inferior epigastric vessels.
- **Inferiorly :** inguinal ligament.

I- INGUINAL HERNIA

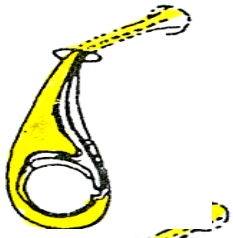
	Indirect (Oblique) Inguinal Hernia	Direct Inguinal Hernia
		
AETIOLOGY	• Congenital performed sac	• Paralysis of ilio-inguinal nerve during appendectomy
	• Acquired causes	
INCIDENCE	**80 %**	**20 %**
PATHOLOGY		
a. Defect	• **Deep ring** lateral to inferior epigastric artery	• **Hasselbach Δ** medial to inferior epigastric artery
b. Sac	• It presents **inside** the cord	• It presents **behind** the cord.
c. Content	Small intestine, omentum or both	
d. Coverings		
	IN INGUINAL REGION 1.Skin. 2. Superficial fascia 3. Ext. oblique apponeurosis 4. Cremasteric muscle. 5. Int. spermatic fascia. 6. Extra-peritoneal fat IN THE SCROTUM 1. Skin 2. Superficial fascia with dartos muscle. 3. Ext. spermatic fascia 4. Cremasteric muscles. 5. Int. spermatic fascia. 6. Extra-peritoneal fat	INGUINAL REGION 1.Skin. 2. Superficial fascia 3. Ext. oblique apponeurosis. 4. Conjoined Tendon. 5. Fascia transversalis. 6. Extra-peritoneal fat

CLINICAL PICTURE	Indirect (Oblique) Inguinal Hernia	Direct Inguinal Hernia
• **Age**	• Any age.	• Usually old age
• **Side**	• **Less** common bilateral	• **More** common bilateral
• **Shape.**	• Pyriform (oblong)	• Hemispherical (rounded)
• **Descent**	• Downwards, forwards & medially	• Forwards
• **Descent to scrotum**	• Can descend.	• Extremely rare.
• **Reduction.**	• Upward, backwards & laterally.	• Backwards
• **Internal ring test**	• Hernia **does not** protrude	• Hernia protrudes
• **External ring test**	• impulse at **tip** of little finger.	• impulse at **medial** side of little finger.
• **Complications**	• **More** common	• **Less** common.

SPECIAL TYPES

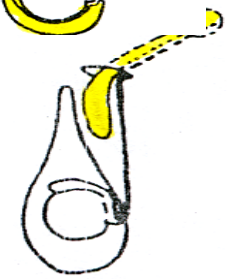
Indirect (oblique) inguinal hernia

1. Congenital hernia



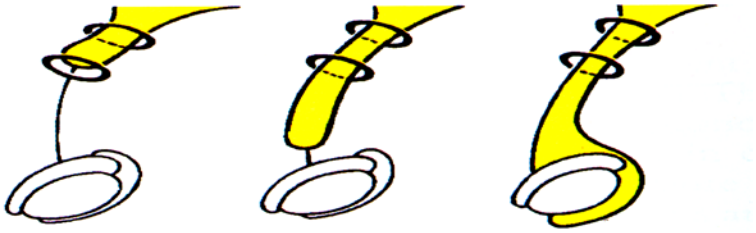
- due to persistence patency of processus vaginalis.
- it reaches down the scrotum from the start
- although congenital, it may appeared with adult life.

2. Infantile hernia (**operative finding only**)



- the tunica vaginalis extends upwards to the external ring e.g. hydrocele & another true hernia sac passes behind it i.e. **2** sacs.

3. Adult type **which may be**



BUBONOCELE TYPE

Hernia is limited to the inguinal canal & seen as bulge or mass at inguinal region

FUNICULAR TYPE

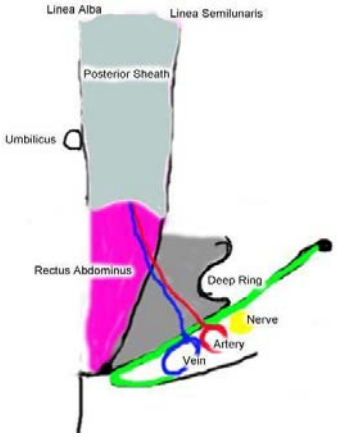
Hernia passes with cord & stops just above the epididymis

COMPLETE SCROTAL HERNIA

Hernia descends to the bottom of scrotum.

SPECIAL TYPES

Direct inguinal hernia



1. Lateral type

- Hernia bulges through the **lateral** part of Hasselbach's Δ (made by fascia transversalis only) & thus it has a very **wide** neck & it is **less** liable to complicate.
- Hernia **never** descends to the scrotum

2. Medial type

- Hernia bulges through the **medial** part of Hasselbach's (defect in conjoint tendon in front of fascia transversalis) & thus it has a very **narrow** neck & it is **more** liable to complicate.
- Hernia **may** descends to the scrotum; but never reach the bottom.

DD OF INGUINAL & INGUINO-SCROTAL SWELLINGS

Inguinal swellings

1. **Hernia :** Oblique inguinal hernia (bubonocele or funicular types) & direct inguinal hernia.
2. **Hydrocele :** Hydrocele of hernia sac.
3. **Testis :** Undescended, ectopic or retractile testicle.
4. **Cord :** Lipoma of the cord.

Inguino-scrotal swellings

1. **Hernia :** Oblique inguinal hernia (complete type)
2. **Hydrocele :** Hydrocele of hernia sac, congenital & infantile types
3. **Testis :** Retractile testicle.
4. **Cord :** Varicocele

MANAGEMENT OF INGUINAL HERNIA

Investigations

1- To detect underlying cause of ↑ I.A.P

As chest X-ray, abdominal U/S & trans-rectal U/S for S.E.P

2- To assess surgical fitness of the patient

As E.C.G, blood picture, blood sugar & kidney function tests

Treatment

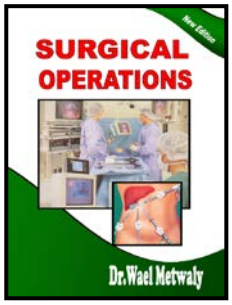
1. TRUSS

- **Indication :**
  If patient unfit for surgery.
- **Complications :**
  1. Infection.
  2. Adhesions → strangulation.
  3. Pressure atrophy of local muscle.

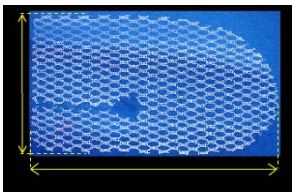
2. OPERATIONS **See next page**

CHOICE OF OPERATION

For more details see Operative notes



Indirect (oblique) inguinal hernia

Herniotomy	Herniorrhaphy	Hernioplasty
HERNIECTOMY		
• Removal of hernia sac after reduction of the contents	• **Herniotomy** + narrowing the defect & repair of post. wall of inguinal canal through one of the following methods 1- **Bassini** repair. 2- **Bloodgood** repair. 3- **Shouldice** repair.	• **Herniotomy** + repair the defect by synthetic material i.e **prolene mesh** 
INDICATED WITH ① infants ② children < 12 years ③ **small** hernial defect in adult with good musculature	INDICATED WITH **large** hernial defect in adult with good musculature	INDICATED WITH ① old patient with weak musculature ② very wide defect ③ recurrent hernias

Direct inguinal hernia

Management of direct hernias, differs from indirect hernias in

① The sac is medial to inferior epigastric vessels.

② The sac lies behind the cord & not within its covering.

③ The sac & defect are wide, so the sac is not excised & just invaginated by repair i.e. **herniorrhaphy** or **hernioplasty**.

RECURRENT INGUINAL HERNIA

AETIOLOGY

1. Leaving part of the original sac.
2. Missing a second sac at operation e.g. **Dual (pantaloon) hernia**.
3. Use of absorbable sutures in hernia repair
4. Postoperative hematoma or infection → weakness of the repair
5. Rapid return to hard work

TREATMENT

Correction of predisposing factors + **hernioplasty**

ANATOMY OF FEMORAL TRIANGLE

Scarapa's triangle

✷ **Site** It is a subfascial space occupying the front of the upper 1/3 of the thigh just below the inguinal ligament.

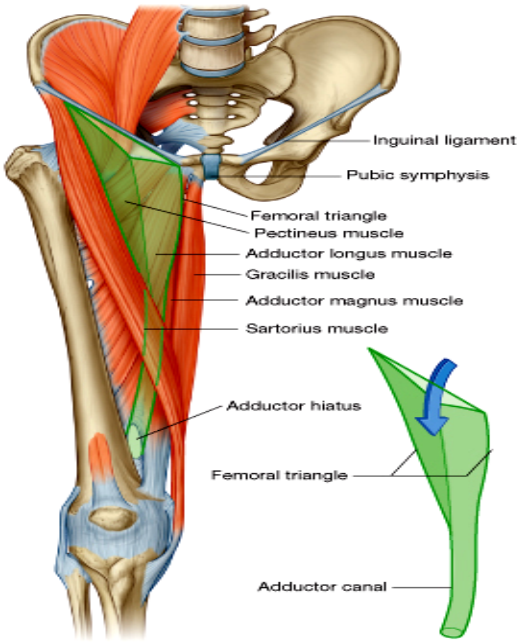
✷ **Boundaries**

Laterally : Medial border of Sartorius.

Medially : Medial border of adductor longus (so the muscle also shares in the floor)

Base : Inguinal ligament.

Apex : meeting of sartorius & adductor longus (beginning of Hunters canal)



✷ **Floor** **From medial to lateral**

Adductor longus, pectineus, psoas major & iliacus muscle.

✷ **Roof**

1. **Skin**.
2. **Superficial fascia**
3. In between there are :

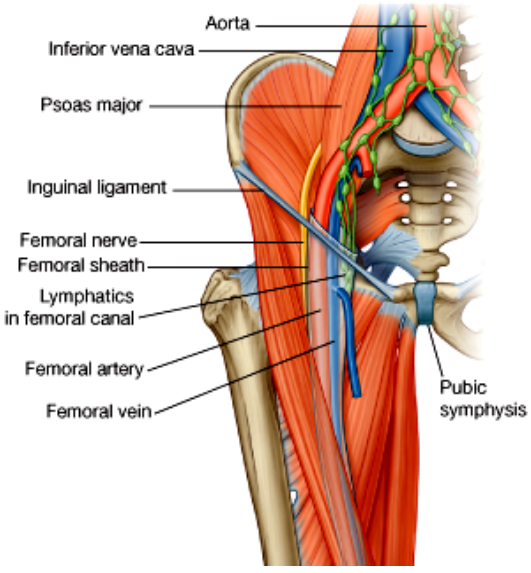
a- **Superficial branches of femoral artery** :
(superficial epigastric a. , superficial external pudendal a. & superficial circumflex iliac a.)

b- **The "T" shaped** superficial inguinal lymph nodes

c- **Great saphenous vein**

d- **The ilio-inguinal nerve**.

4. **Fascia containing the saphenous opening** (which is a defect in the fascia covered by cribriform fascia)



✷ **Contents**

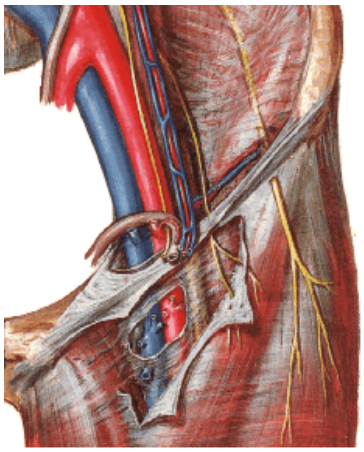
1. **Femoral artery & its branches :** profunda femoris artery & deep external pudendal artery.
2. **Femoral vein & its tributaries**.
3. **Femoral sheath**
4. **Femoral nerve** (outside the sheath),
5. **Deep inguinal L.Ns**.

✷ **Surgical importance**

Presence of femoral canal & their contents

SURGICAL ANATOMY

FEMORAL SHEATH



Anterior femoral sheath

Formed of **fascia transversalis**

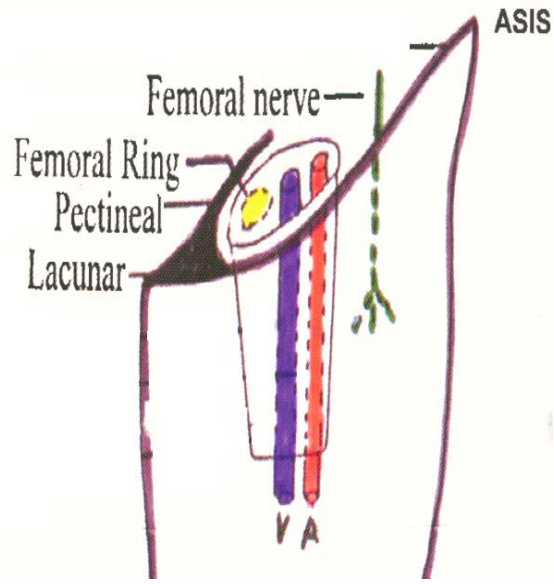
Posterior femoral sheath

Formed of **fascia iliacus**

- **The femoral sheath** is divided by **2** thin fibrous septum into **3** compartments.
- The most medial compartment of femoral sheath is called **femoral canal**.
- The intermediate compartment contains the femoral **vein** & the lateral compartment contains the femoral **artery**

N.B.: The femoral nerve lies outside the sheath

FEMORAL CANAL



Structure

- The most medial compartment of femoral sheath
- Cone shaped (**1/2** inch long)
- Its **mouth** (**femoral ring**)
- Its **apex** is formed of fusion of medial border of femoral sheath & septum between the femoral canal and the femoral vein.

Contents

- Fat, lymphatics & lymph node of **Cloquet**

Function

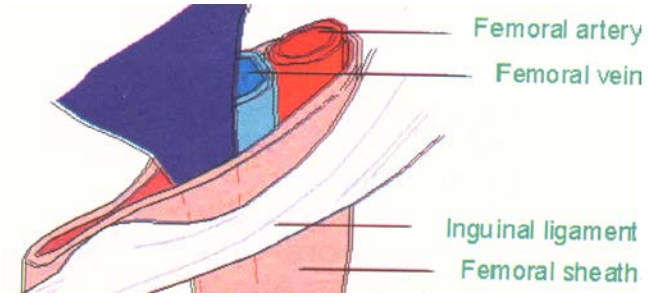
- Give space for expansion of femoral vein during ↑ venous return with lower limb exercise

FEMORAL RING

Mouth of the femoral canal

Boundaries

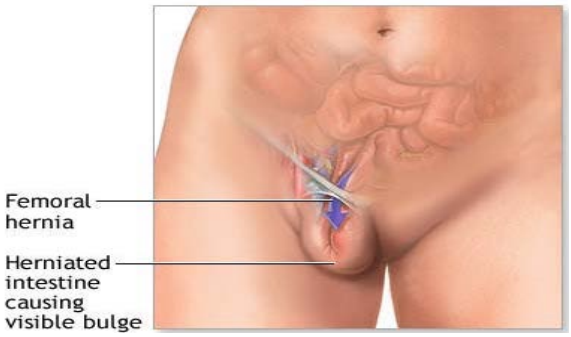
- **Anterior : Inguinal** ligament (**Poupart's** ligament)
- **Posterior : Cooper's** ligament (**Pectineal** ligament)
- **Medially :** (**Lacunar** ligament)
- **Laterally :** Femoral vein



II - FEMORAL HERNIA

DEFINITION

- Hernia which leaves the abdomen through the femoral ring into the femoral canal



AETIOLOGY

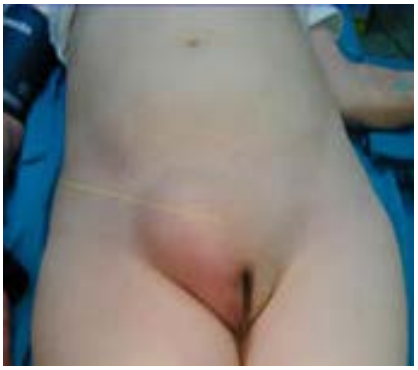
- Always acquired **never** congenital

PATHOLOGY

a. Defect : Femoral ring

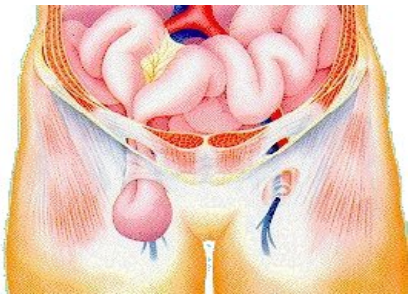
b. Sac : passes **downwards** in the femoral canal then **forwards** stretching the cribriform fascia of the saphenous opening then **upwards** & **laterally** towards A.S.I.S.

so femoral hernia is liable to be strangulated



c. Contents : Omentum, bowel or both.

d. Coverings : 1. Skin.
2. Superficial fascia
3. Stretched **cribriform fascia**
4. Anterior femoral sheath



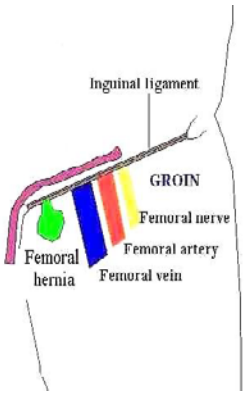
CLINICAL PICTURE

- **Hernia** is common in **females** due to ☟
  (1) The female pelvis being larger
  (2) Small sized femoral vein
  → wide femoral ring

- **Hernia** is characterized by ☟
  - Present below & lateral to the pubic tubercle.
  - Gives an expansile impulse on cough.
  - It can be reduced if early only

DIFFERENTIAL DIAGNOSIS

Reducible femoral hernia



REDUCIBLE INGUINAL HERNIA which characterized by ☟
Above & medial to pubic tubercle

FEMORAL ANEURYSM which characterized by ☟
Expansile pulsation & moves across but not along the course of artery.

PSOAS ABSCESS which characterized by Cross fluctuation

SAPHENA VARIX which characterized by ☟
Thrill on cough, completely disappear on lying down, venous hum on auscultation & apparent varicose vein.

Irreducible femoral hernia

IRREDUCIBLE INGUINAL HERNIA

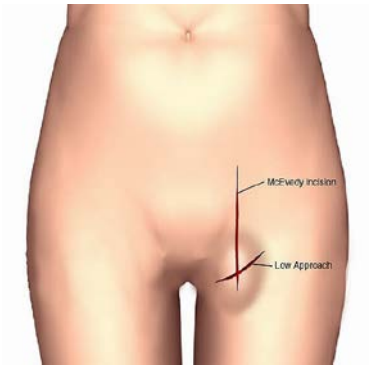
LIPOMA which characterized by

Soft, lobulated **s**urface , **s**lippery edge, **s**uperficial to muscles, **s**kin over show dimpling, **p**ainless & **p**seudo-fluctuant swelling

INGUINAL L.NS

ILIOPSOAS BURSA which characterized by

Associated osteoarthritis of hip joint



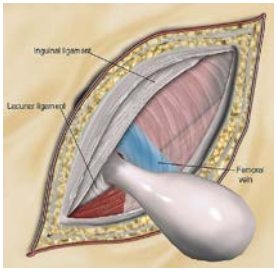
TREATMENT

Surgery is the only line of treatment

(**3 Approaches**)

1- Low approach

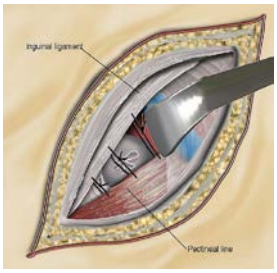
- This approach is **rarely done nowadays**, because of low transfixation of the sac which is therefore not completely excised.
- The incision **1/2** inch below & parallel to the inguinal ligament.
- **The repair :** by suturing the inguinal ligament (anterior border of femoral ring) to cooper's ligament (posterior border of femoral ring)
  i.e. **P**oupart's to **P**ectineal



2- High approach

(Lotheissen's operation)

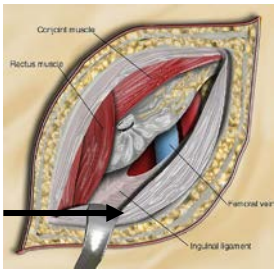
- The incision above & parallel to the medial **2/3** of the inguinal ligament
- The inguinal canal is opened & fascia transversalis is incised, the sac is opened & the content are reduced then the sac is transfixed & excised.
- **The repair :** as above



3- Preperitoneal approach

(Mc Evedy's operation)

- The incision is para-rectal at lateral border of lower part of rectus abdominis.
- The incision is deepened dividing fascia transversalis Till the peritoneum then the sac is transfixed & excised.
- **The repair :** as above

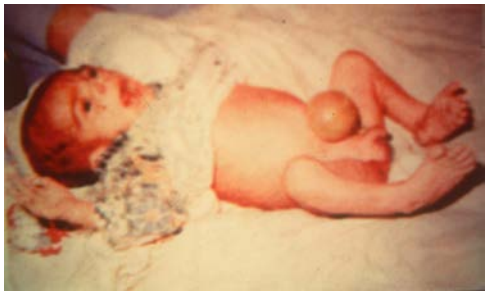
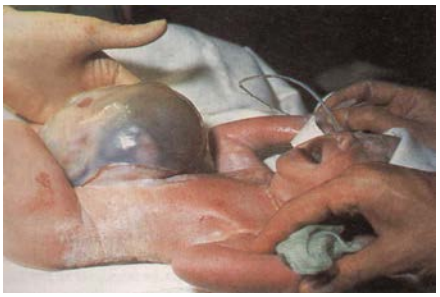
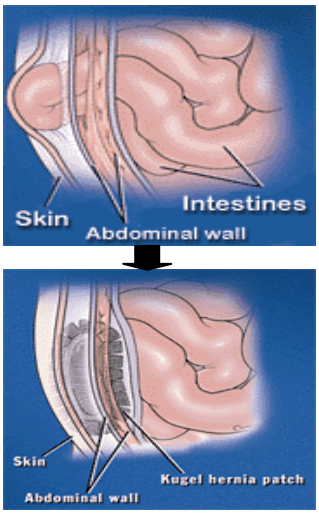


III- UMBILICAL HERNIA

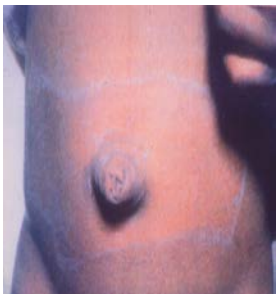
1. Congenital | **2. Infantile** | **3. Adult**

1. Congenital umbilical hernia

Exomphalos

	Exomphalos minor	Exomphalos major
		
PATHOLOGY **a. Defect**	• Small (**< 5 cm**) at the umbilicus	• Large (**> 5 cm**) at the center of abdomen.
b. Sac	• Peritoneum.	• Peritoneum
c. Contents	• Bowel.	• Bowel ± **liver**
d. Coverings	• Wharton's Jelly + layer of amniotic membrane.	• Layer of amniotic membrane
COMPLICATIONS	• During legation of an umbilical stump, a loop of intestine may be entangled in the ligature	• Rupture may occur with infection → peritonitis (**The cause of death**)
TREATMENT	• Content are reduced & returned to the abdomen then the sac is excised & the defect is repaired in layers 	**Urgent operation** **The problem :** No space in abdomen to accommodate the contents so the skin on either sides of the defect is undercut then flaps will be sutured together over the sac + release incision over the flanks. **Later on :** If infant survive, definitive repair is done.

2. Infantile umbilical hernia



AETIOLOGY

- Weakness of the umbilical scar from infection
- ↑ **I.A.P** from cough.

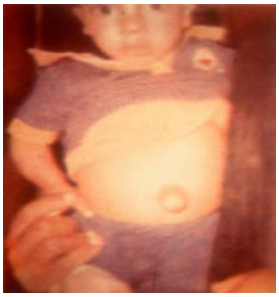
PATHOLOGY

a. Defect : Umbilical scar

b. Sac : Small, conical with **wide** neck

c. Contents : Omentum, bowel or both.

d. Coverings : 1. **Umbilical scar**.
2. Extra- peritoneal fat



CLINICAL PICTURE

- Patient shows umbilical protrusion on cough .
- Edges can be palpated as firm ring

COMPLICATIONS **Rare**

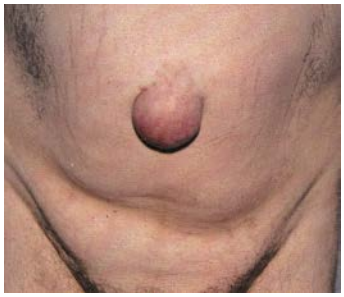
TREATMENT

Strapping better avoided as most of cases closed spontaneously within **2** years

Anatomical repair with prolene sutures if the defect more than **2** fingers or the hernia persist more than **2** years

3. Adult umbilical hernia

Para-umbilical hernia



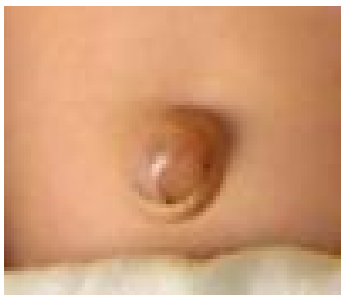
AETIOLOGY

- Middle aged female.
- Usually obese, multipara .
- Para- umbilical & **never** umbilical.

PATHOLOGY

a. Defect : Linea alba.

b. Sac : Small, crescentic with **narrow** neck



c. Contents : Omentum, bowel or both.

d. Coverings : 1. Skin.
2. Superficial fascia
3. Stretched **linea alba**.

CLINICAL PICTURE

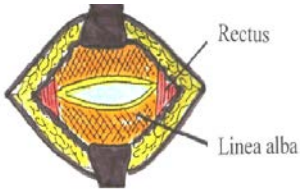
- Patient shows para-umbilical protrusion on cough.
- **DD** between **supra**- umbilical & **infra**- umbilical hernias by the (crescentic shape)

N.B.: Adult umbilical hernia (acquired)
usually seen as **everted umbilicus** with patient with ascites or ↑ **I.A.P**

COMPLICATIONS **Common**

TREATMENT

Mayo's repair



- Elliptical transverse incision is made over the hernia
- Sac is exposed & dissected down to the neck.
- Sac is opened at its neck because of adhesions at fundus.
- Contents are dealt with & the sac is transfixed & excised.
- **Repair :** the defect is closed by overlap of the upper flap over the lower flap of anterior rectus sheath.

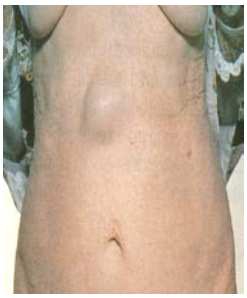
Hernioplasty

using prolene mesh .

IV. EPIGASTRIC HERNIA

AETIOLOGY

Usually acquired, It is formed as a direct result of sudden strain → tearing of the interlacing fibers of linea alba.



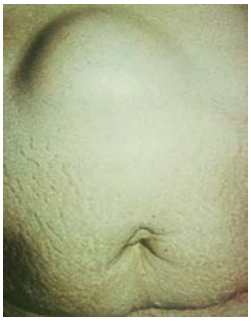
PATHOLOGY

a. Defect : Linea alba.

b. Sac : may be **2** types

1. FATTY HERNIA OF LINEA ALBA

It is a protrusion of extra-peritoneal fat only without a peritoneal sac



2. TRUE EPIGASTRIC HERNIA

It drags a pouch of peritoneum as a hernia sac

c. Contents : The sac is empty (because of narrow neck) or it contains a small portion of greater omentum

d. Coverings : 1. Skin.
2. Superficial fascia
3. Stretched **linea alba**.

CLINICAL PICTURE

- Patient shows a small irreducible protrusion simulating to lipoma .
- Severe epigastric pain with nausia & vomiting from friction of herniated omentum on the stomach (DD peptic ulcer or gall bladder disease)

TREATMENT

If the defect is small

Excision of the protruding extra peritoneal fat & the hernia sac

If the defect is large

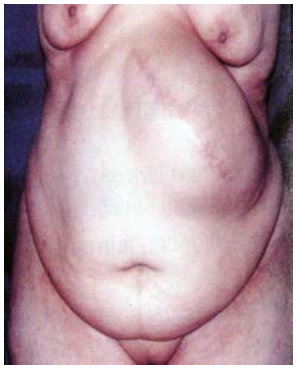
It is repaired by **Mayo's repair**.

V. INCISIONAL HERNIA

DEFINITION

Hernia developing after abdominal incision.

AETIOLOGY

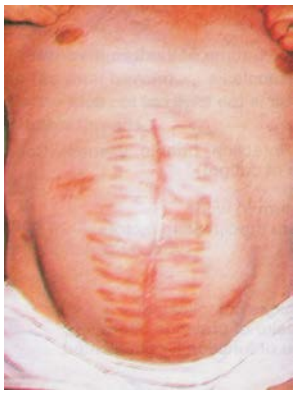


Pre- operative causes

- Obesity .
- Diabetes mellitus, cirrhosis, steroid therapy
- Anemia & hypoproteinemia
- Respiratory problems as chronic bronchitis

Operative causes

- Uses of **absorbable** sutures .
- **Rough** surgical technique .
- **Tight** stitches→ devitalized wound
- Insertion of a **drain** through the wound



Post- operative causes

- Persistent pre-operative causes
- Wound hematoma or infection.
- Rapid return to hard work

CLINICAL PICTURE

- Patient shows a bulge involve the surgical scar gives an expansile impulse on cough & ↑ steadily by time.

TREATMENT

Anatomical repair

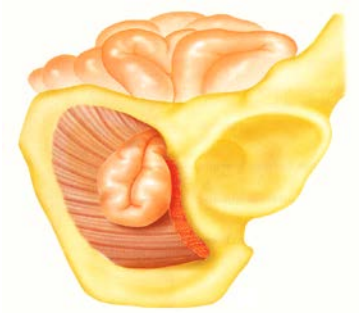
Repair the defect in layers according to site of incision.

Hernioplasty

using prolene mesh

VI. RARE HERNIAS

1. Obturator hernia

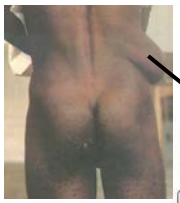


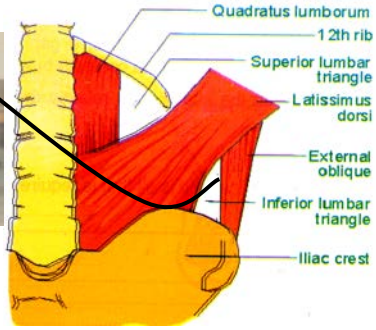
- The sac passes through the **obturator foramen** inside the pelvis so **no** external swelling so unlooked until strangulation occurs.

TREATMENT (Trans-abdominal approach)
contents are reduced, sac is excised & obturator canal obliterated by sutures.

2. Lumbar hernia

Petit's lumber triangle





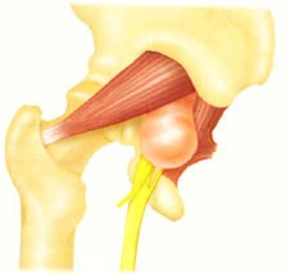
Boundaries of inferior lumber triangle

- **Below :** Iliac crest.
- **Laterally :** Ext. oblique muscle
- **Medially :** Latissmus dorsi.

TREATMENT

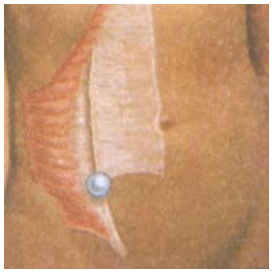
The hernia can be controlled by a **belt**, but if large **hernioplasty** is required.

3. Gluteal & Sciatic hernia



- **Gluteal hernia** → protrude through **greater** sciatic notch
- **Sciatic hernia** → protrude through **lesser** sciatic notch.

4. Spigelian hernia



- The sac of hernia passes through a defect in the **spigelian fascia**, the sac lies between the transversus abdominis muscle and internal oblique muscle or lie beneath the external oblique muscle
- The strangulation is very common.

TREATMENT

Excision of the sac & closure of the defect i.e. **herniorrhaphy**.

تحـــذير

هذا الكتاب مسجل ومحفوظ بدار الكتب والوثائق القومية

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