GASTROENTEROLOGY

• INDEX •

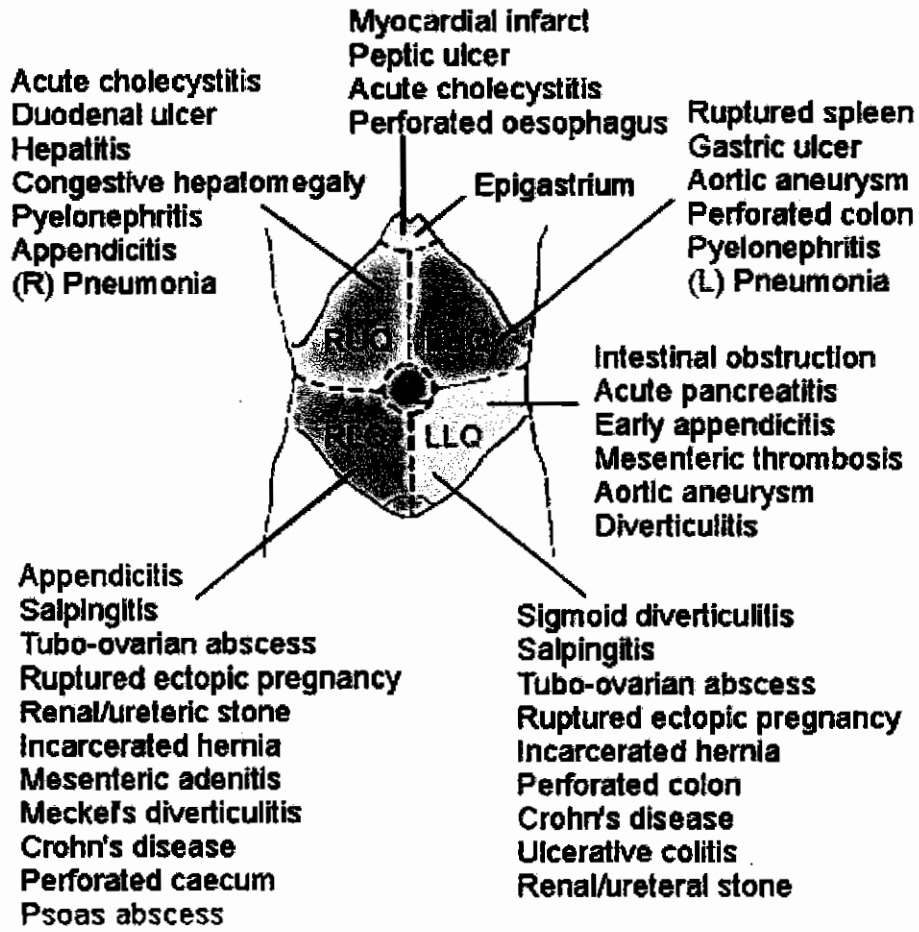
GALL BLADDER

LIVER

SMALL INTESTINE

COLON / RECTUM / ANUS

Acute Abdomen



Assessment

Initial impression/observation

- Does the patient look ill, septic or shocked?
- Are they lying still (think peritonitis), or rolling around in agony (think intestinal, biliary or renal colic)?
- Assess and manage Airway, Breathing and Circulation as a priority.
- In an emergency department setting: if there are signs that the patient is shocked or acutely unwell, assess quickly but carefully and arrange any early investigations.

- In a community setting: make arrangements for rapid transfer to hospital for further assessment.

History

This should cover the following points:

- Demographic details, occupation, recent travel, history of recent abdominal trauma.
- Pain: (SOCRATES)
- Associated symptoms:
    - Vomiting and the nature of vomitus (undigested food or bile suggests upper GI pathology or obstruction; faeculent vomiting suggests lower GI obstruction).
    - Haematemesis or melaena.
    - Stool/urine colour.
    - New lumps in the abdominal region/groins.
    - Eating and drinking - including when the patient's last meal occurred.
    - Bowels - including presence of diarrhoea, constipation and ability to pass flatus.
    - Fainting, dizziness or palpitations.
    - Fever/rigors.
    - Rash or itching.
    - Urinary symptoms.
    - Recent weight loss.
- Past medical and surgical history/medication.
- Gynaecological and obstetric history:
    - Contraception (including intrauterine contraceptive device (IUCD) use).
    - Last menstrual period.
    - History of sexually transmitted infections/pelvic inflammatory disease.
    - Previous gynaecological or tubal surgery.
    - Previous ectopic pregnancy.
    - Vaginal bleeding.
    - Drug history and allergies - including any complementary medication.

Examination[1]

- Pulse, temperature and blood pressure.
- Assess respiratory rate and pattern. Patients with peritonitis may take shallow, rapid breaths to reduce pain.
- If there is altered consciousness, check Glasgow Coma Scale (GCS) or AVPU (**A** lert, **V** oice response, **P** ain response, **U** nconscious) scale.
- Inspection:
    - Look for evidence of anaemia/jaundice.
    - Look for visible peristalsis or abdominal distension.

  - Look for signs of bruising around the umbilicus (Cullen's sign - this can be present in haemorrhagic pancreatitis and ectopic pregnancy) or flanks (Grey Turner's sign - this can be present in retroperitoneal haematoma).
  - Assess whether the patient is dehydrated (skin turgor/dry mucous membranes).
- Auscultation:
  - Auscultate the abdomen in all four quadrants.
  - Absent bowel sounds suggest paralytic ileus, generalised peritonitis or intestinal obstruction. High-pitched and tinkling bowel sounds suggest subacute intestinal obstruction.
  - Intestinal obstruction can also present with normal bowel sounds.
  - If there is reason to suspect aortic aneurysm, listen carefully for abdominal and iliac bruits.
- Percussion:
  - Percuss the abdomen to assess whether swelling/distension might be due to bowel gas or ascites.
  - Patients who display tenderness to percussion are likely to have generalised peritonitis and this should act as a red flag for serious pathology.
  - Assess for shifting dullness and fluid thrill.
  - Percussion can also be used to determine the size of an abdominal mass/extent of organomegaly.
- Palpation:
  - Palpate the abdomen gently, then more deeply, starting away from the pain and moving towards it.
  - Feel for masses, tenderness, involuntary guarding and organomegaly (including the bladder).
  - Test for rebound tenderness.
  - Examine the groins for evidence of herniae.
  - Always examine the scrotum in men as pain may be referred from unrecognised testicular pathology.
  - Check supraclavicular and groin lymph nodes.
- Further examination:
  - Perform rectal or pelvic examination as needed, with an appropriate chaperone in attendance.
  - Check lower limb pulses if there could be an abdominal aortic aneurysm.
  - Dipstick urine and send for culture if appropriate.
  - In a woman of childbearing age, assume that she is pregnant until proven otherwise - perform a pregnancy test.
  - Examine any other system that might be relevant, eg respiratory, cardiovascular.

Prehospital/emergency department care of suspected acute abdomen

- Keep patient nil by mouth.
- Apply oxygen as appropriate.
- Intravenous (IV) fluids: set up immediately if the patient is shocked and the equipment is available. Send blood for group and save/crossmatch and other blood tests as appropriate.
- Consider passing a nasogastric (NG) tube if there is severe vomiting, signs of intestinal obstruction or the patient is extremely unwell and there is danger of aspiration.
- Analgesia: the previous practice was to withhold analgesia until surgical review, but a surgical abdomen is very painful and is likely only to be adequately relieved by parenteral opiates, eg morphine. One recent review showed that opiate administration may alter physical examination findings, but these changes result in no significant increase in management errors.[6] Another study showed that morphine safely provides analgesia without impairing diagnostic accuracy.[7] A Cochrane review also supported the use of analgesia before assessment by a surgeon.[8]
- Antiemetic: avoid using this as a symptomatic treatment without considering a diagnosis in a community setting.
- Antibiotics: if systemic sepsis, or peritonitis, or severe urinary tract infection (UTI) is suspected. IV cephalosporin plus metronidazole are commonly used in acutely unwell patients in whom peritonitis is suspected.
- Arrange urgent surgical/gynaecological review as appropriate.
- Arrange investigations such as ECG if a medical cause is likely.
- Admit: if surgery is considered likely, if the patient is unable to tolerate oral fluids, for pain control, if a medical cause is possible or if IV antibiotics are required.

Investigation

- This is mainly relevant to patients being assessed in emergency departments or secondary care.
- With the exception of a urinary pregnancy test and urine dipstick, there are few tests that are useful in the community assessment of the patient with acute abdominal pain.
- On the whole, if you are concerned enough to be ordering blood tests or imaging, the patient should be referred to secondary care.
- The following tests are often used but can be nonspecific and must be interpreted in the clinical context and with appropriate medical/surgical expertise:
    - Blood tests: FBC, U&Es, LFTs, amylase, glucose, clotting, and occasionally Ca2+, arterial blood gas (ABG) (pancreatitis), calcium.
    - 'Group and save' or crossmatch.
    - Blood cultures.

- Pregnancy test in women of childbearing age.
- Urinalysis
- Radiology - abdominal X-ray (AXR) (supine), chest X-ray (CXR) (erect looking for gas under the diaphragm), intravenous pyelogram (IVP), CT scan, ultrasound scan, as appropriate.
- Consider ECG and cardiac enzymes.
- Peritoneal lavage if there is a history of abdominal trauma.

APPENDIX

Source: Washington Recall

It's an immunological organ that secretes **IgA** (However, it's not an essential organ & can be removed w/out immunocompromise)

EMBRYOLOGY

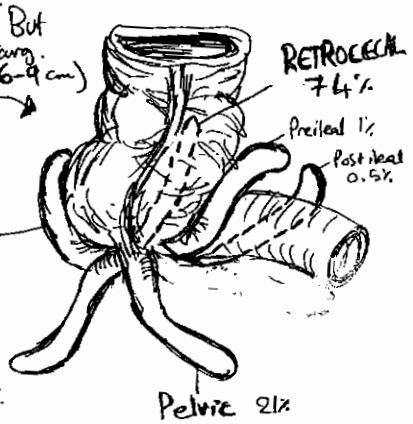
It begins to bud off from the cecum at around the 6th wk of embryological development.

* The base of appendix remains in a **fixed** position w/ respect to the **cecum**, whereas the **tip** can end up in various positions.

ANATOMY

- Length can range from 2 to 30 cm! (But avrg. 6-9 cm)

* Anatomic variation in the position of appendix:



Appendicial Luminal Capacity is 1 ml

m.c site — **Retrocecal** (~75%)

2nd m.c site — Pelvic (21%)

NOTE: To locate the appendix; locate the cecum, then follow the three **taenia coli** until they converge at the BASE of appendix.

Mesoappendix: is the mesentry that suspends the appendix from the terminal ileum. (contains the appendicular art.)

BLOOD SUPPLY

- By appendicular artery (A branch of ileocolic art. which is a branch of SMA)

ACUTE APPENDICITIS — It's inflammation of the appendix caused by OBSTRUCTION of the appendiceal lumen ⇒ closed loop ⇒ inflammation

INCIDENCE

life-time incidence is 7% of Population.

* Avrg. Age: (20-30) yrs

Most common Pathogens:
- E.coli
- Bacteroids Fragilis

CAUSES

- **FECALITH** (m.c) — 40%
- Hypertrophy of lymphoid tissue.
- Tumor (ex. Carcinoid)
- Vegetable / Fruit Seeds!
- Intestinal Parasites / worms.
- Inspissated barium from Prev. X-Ray.

* Acute appendicitis is usually misdiagnosed in ♀ & elderly
* RARE in extreme of age (& if it happens → Life-threatening!)

PATHOPHYSIOLOGY

▸ OBSTRUCTION → Distention (↑ Intraluminal Pressure) → Venous congestion →
↳ Impaired blood supply. → Bacterial accumulation →
↳ Inflammation → Necrosis & Perforation!

CLINICAL FEATURES

Sx: • In order •

- **PAIN** :- Diffuse (Periumbilical area) ↳ referred Pain.
  - Intermittent & crampy.
- Nausea / vomiting (AFTER Pain) — due to ① neural stimulation & ② Presence of ileus.
- Anorexia
- Pain migrates to RLQ (constant & intense Pain) — usually < 24 hrs (Due to Peritoneal irritation.)

Signs:

- Usually NL V/S
- Signs of Peritoneal irritation:
  - Guarding / muscle spasm
  - Rebound tenderness
  - Obturator & Psoas signs
  - Low-grade fever (High grade if PERFORATION occurs)
  - RLQ hyperesthesia.

Tenderness maximally at McBurney's Point.

Obturator Sign
Pain upon internal rotation of the leg @ the hip & Knee flexed (seen in Pts @ PELVIC appendix!)

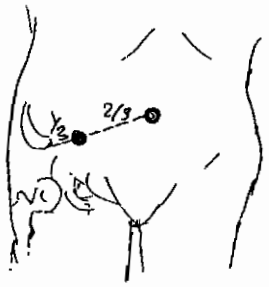
Psoas Sign
Pain elicited by extending the hip @ Knee extended on by flexing the HP against resistance (seen in retrocecal app.

Rovsing's Sign
Palpation / rebound Pressure of LLQ resulting in Pain in RLQ (seen in appendicitis)

Valentino's Sign
RLQ Pain / Peritonitis from succus draining down to RLQ from a Perforated gastric or duodenal ulcer.

McBurney's Point:

Point 1/3 from the ant. sup. iliac spine to the umbilicus (Often the Point of maximal tenderness)



INVX

LABS

- **CBC** : ↑WBC (>10,000 in >90%) — most often w a "left shift"
- **U/A**: To rule out UTI (If +ve you can't r/o appendicitis)
  - * You may have Abnormal U/A w appendicitis ⇒ Pyuria & mild hematuria are common in appendicitis. w Pelvic inflammation → resulting in inflammation of the ureter.
- Also do β-hCG / BUN / Cr / electrolytes

IMAGING

- **X-Ray** — to r/o other Pathologies:-

  CXR — to r/o Pneumonia & free air

  AXR — (non-specific) Calcified fecalith Present in about 5% of cases only!

  * FINDINGS on AXR:
    - — Fecalith (N 5% of cases)
    - — Sentinal loops.
    - — **Scoliosis** away from the right (due to pain)
    - — mass effect (Abscess)
    - — Loss of Psoas Shadow
    - — Loss of Preperitoneal fat stripe
    - — Free air (if Perforated) → Rare!

- **CT** — FINDINGS:
  - Periappendiceal fat Stranding.
  - Appendiceal diameter >6 mm
  - Periappendiceal fluid
  - Fecalith.
- **MRI** — in Pregnant ♀
- **Graded compression sonography (US)** — the most sensitive

ALVERADO SCORE

Used to assess the Probability

- Mnemonic: **MANTRELS**
  - M: Migration of Pain to RLQ (1)
  - A: Anorexia (1)
  - N: N, V (1)
  - ** T: Tenderness in RLQ (2)
  - R: Rebound pain (1)
  - ** E: Elevated temp. (1)
  - L: Leukocytosis (2) >10,000
  - S: Shift to the Lft (1)

2 Points for T, & E
1 Point for all others

SCORE
- ≤4 – unlikely
- 5–6 – Possible
- 7–8 – Probable
- 9–10 – v Probable (3)

~~tt~~

- **PREOP.**
  - Rehydration w/ IV Fluids (Ringer Lactate)
  - Preop. Antibiotics w/ anaerobic coverage
    NOTE appendix is considered part of the colon.
    ex. Cefoxitin / Cefotetan / CIPro / Flagyl

- **OP.**
  - If NON-perforated → Prompt appendectomy (to prevent perforation)
    24 hrs of abx (Anaerobic coverage)
    Discharge home usually on Postop. day #1
  - If Perforated (Rupture) → IV Fluid resuscitation & prompt appendectomy
    * All Pus is drained! w/ Postop. abx (Broad-spectrum) for 3–7 days
    * Wound is left open in most cases of Perforation after closing the fascia. (Heals by 2ry intention / delayed 1ry closure)

    RUPTURE: 25% occur after 24 hrs
    75% occur after 48 hrs!
  - If appendiceal abscess → Percutaneous drainage of abscess
    Abx
    Elective appendectomy ~6 wks later (Interval appendectomy).

- NOTE
  - If NL appendix is found upon exploration → Take it out! (even in crohns.)
  - Dx of ruptured appendix: Fever (>39°), ↑WBC, Rebound Tenderness
    U/S: Periappendiceal fluid collection
  - Appendectomy is the m.c.c of EMERGENT abdominal surgery.
  - Rocky-davis incision: Straight across, transverse — sometimes used.
  - Open vs. Laparoscopic.
    * Open is more cost-effective & time-saving, ↓ Pain ↓ risk of wound infxn better anesthesia
    - CI of laparoscopy:
      - Extensive adhesions
      - Severe Portal HTN
      - Coagulopathies
      - 1st trimester of Pregnancy.

COMPLICATIONS of Appendicitis

- Pelvic abscess / Liver abscess
- Perforation & Peritonitis
- Portal Pylethrombophlebitis (v. rare)
- Gangrene.

"Mittelschmerz"
Pelvic Pain due to ovulation

In Crohn's, you remove the appendix UNLESS the base is involved!

COMPLICATIONS of Appendectomy

- **Small Bowel obst.** (↑×4 more if there's Perforation!)
- **Enterocutaneous Fistula.**
- * **Wound infxn.** (m.c)
- ↑ incidence of Rt inguinal hernia
- Stump abscess
- Infertility ∵ Perforation in ♀.

Pts at risk of dying from acute appendicitis :=
EXTREMES of AGE!
due to uncontrolled sepsis!

Mortality rate 1%

DDx

Acute Abdomen: Meckel's diverticulum // PUD / Crohn's // Urological causes // Gastroenteritis

Acute Mesentric Adenitis (in children) — m.c organism is "Yersinia enterolytica"

Gyne Causes (in ♀) — PID / Ruptured ovarian cyst / Ectopic Pregnancy / Ruptured graafian follicle.

** Appendicitis in Pregnancy

Incidence : 1/1500

CP : **RUQ** Pain is the usual Presentation

— Risk of fatal loss — 4%.
— Risk of Preterm labor — 7%.

* It's the m.c Procedure done during Pregnancy!
* The only abnormal finding is Lt shift.

LAYERS CUT DURING SURGERY

3 Ss
- Skin
- Subcut. Fat
- Scarpa's fascia

3 muscles
- Ext. oblique
- Int. oblique
- Transversus abd.

- Transversalis fascia
- Preperitoneal fat
- Peritoneum.

McBurney's vs. Rocky Davis Incision

*** ▶ McBurney's — Angled down (oblique)
+++ ▶ Rocky Davis — Straight across (transverse)

◉ During surgery, electrocautery is used to AVOID **mucocele**.

TUMORS OF THE APPENDIX

CARCINOID — the m.c < 5% Malignant.

If < 1.5cm — Appendectomy
> 1.5 cm — Rt hemicolectomy

DDx

- Carcinoid
- Adenocarcinoma
- Malignant mucoid adenocarcinoma. — risk of seeding during surgery! (Pseudomyxoma Peritoni)

Thanks Gluck
The End.

ESOPHAGUS

ANATOMY

- 25 cm-long muscular tube (40 cm from the teeth to LES)
- Begins at the pharynx (lower border of C6) until reaching the cardia of the stomach.

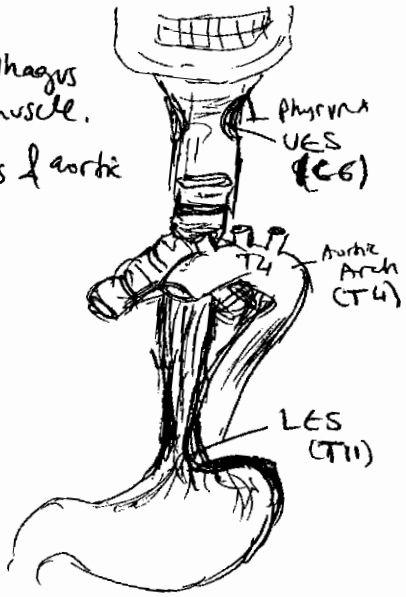
  - Sup. 1/3 → Striated muscle ONLY
  - Middle 1/3 → BOTH striated & smooth muscle.
  - Inf. 1/3 → Smooth muscle ONLY.

• THE AREAS OF NARROWING

① At the beginning of the esophagus — caused by cricopharyngeus muscle.

② Where the Lt mainstem bronchus & aortic arch cross.

③ At the hiatus of diaphragm.



• 2 SPHINCTERS

- Upper Esoph. Sphincter (UES)
- Lower Esiph. Sphincter (LES)

PHYSIOLOGY

Types of Peristalsis:

- **PRIMARY**: Esophageal Peristalsis accompanying Swallowing
- **SECONDARY**: Initiated by the esophageal musculature when the pharyngeal phase to clear the esophagus of any substance Left behind from Ivy Peristalsis.

PHASES OF SWALLOWING

- Oral-Phase (1 sec.) — voluntary
- Pharyngeal Phase (<1 sec.)
- Esophageal Phase (8-20 sec.)] Involuntary

BLOOD SUPPLY

Proximal 1/3 → Inf. thyroid art. & Ant. intercostals.
Middle 1/3 → Esophageal art. & Bronchial art.
Distal 1/3 → Lt gastric art. & Lt inf. Phrenic art.

* The vagus n. runs in the esophagus.

The esophagus is at risk of perforation due to absence of Serosa!

All GIT has serosa EXCEPT esophagus & rectum

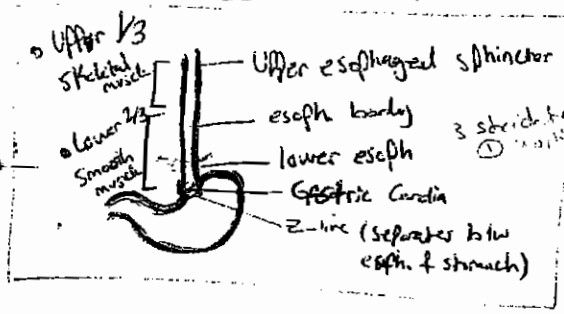
Diseases of the

ESOPHAGUS

Source: Medstudy Dessier Kaplan + Surgical additions 85

Intro

- **Anatomy**



* lining epithelium of the esophagus is Squamous epithelium. (while the Stomach is columnar)

- **Function** → Conduction canal through which the food Pass.
  - Transfers food by Peristalsis.

Anti reflux mechanisms :-

- Lower esopho. sphincter (LES)
- Crura of diaphragm (located above LES)
- Cardiac Angle (Angle of His)
- Peristalsis movement.
- Saliva !

SYMPTOMS OF ESOPH. DISEASES

- DYSPHAGIA (most imp.)
  - Difficulty on swallowing
  - duration
  - Solid / liquid
  - onset
  - Intermitted / Progressive.
  - W/Weight loss.
- Odynophagia
  - Pain during swallowing
- Heart burn
- Regurgitation
- Atypical Chest Pain.

Dysphagia & odynophagia often result in wt loss.

DIAGNOSTIC TOOLS

- **Hx** (V. IMPORTANT)
- P/E (minimal info.)
- Barium swallow : it's usually the 1st test performed in workup of dysphagia (Best initial test)
- Endoscopy : after barium swallow (if needed)
- Esophageal manometry (done ONLY if dysphagia persists w/ -ve barium & -ve endoscopy)
- 24hr esoph. monitoring

Barium Swallow is done BEFORE endoscopy

Why?

- Risk of perforation w/ diverticular or high grade obst
- Info. from barium might be enough
- barium might give a general idea of type & severity.

Diseases

ACHALASIA

- Of unknown etiology idiopathic
- Loss of intraluminal inh. neurons leading to ↑ tone of LES (failure of relaxation)

- **Findings:**
  - NO organized Peristalsis
  - LES has ↑ pressure
  - LES does NOT relax w/ swallowing
- **Clinical features:**
  - Dysphagia for solids AND liquids (pt eats slowly & drinks lots of water) simultaneously.
  - Long-standing sx (usually yrs)
  - Younger age group than CA (But not age/gender Predilection)
  - Not related to smoking or alcohol
  - Regurgitation of undigested food, esp. at night (BUT not bad smell)
    - BUT NO reflux
    - difficulty in retaining } infer in hx
- **Dx** - Hx & P/E
  - Barium swallow (best initial test) → Bird's-beak appearance.
  - Endoscopy w/ bx → to confirm dx & r/o CA at Lower esoph. junction
  - Esophageal manometry (the definitive dx) → it will show lack of Peristalsis & non-relax LES. (to confirm dx)

CXR may show air-fluid level in the dilated esoph * Not v. useful } evidence of medical

• **Complications**
  - Aspiration Pneumonia
  - Wt loss

• **tt** (Aim: to open the LES)

** – Pneumodilatation (BEST initial therapy) (Effective in 85% of Pts!)
  • (3-4 cm diameter balloon is inflated in the LES) → Produce higher Pressure than the used in strictures.
  • 5% risk of Perforation

Other options
- BOTOX – effective in 65% of Pts
  BUT requires repeat theraphy within (6-12) m.
- Surgical ~~myomectomy~~ myotomy
  Risk of GERD! "Heller" myotomy → circular muscle layer of LES is incised.
- CCB & (not effective). – temporary & Partial relief.
  nitrates

Psuadochalasia (2ry) CAUSES:
- CA (m.c.) esp. in elderly (adenocarcinoma of proximal stomach)
- Lymphoma
- Chaga's d.
- Eosinophilic gastroenteritis
- Neurodegenerative d.

** We differentiate it from achalasia by BIOPSY.

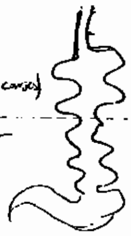
DIFFUSE ESOPHAGEAL SPASM (DES) & Nut-cracker

Idiopathic abnormalities of the neural processes of the esoph. sphincter function ... there is no ... problem ... in ... body

• **Clinical Features**
  • Dysphagia for BOTH solids & liquids – Intermittent
  • Chest PAIN (atypical) ← NOT odynophagia (Pain not related to swallowing)
    (may mimic MD)
    ↳ Intermittent
    ↳ NOT related to exertion or eating
    ↳ ↑ c cold liquids

DES: non-Peristaltic Cxn c high-amplitude
Nut-cracker: Peristaltic Cxn c high amplitude (normal)
BUT BOTH share the Sx, dx & tt.
↓
Generally both diseases are the same (the only diff. is manometric studies)

• **Dx**
  - Hx & P/E • ECG (to r/o cardiac causes)
  - Barium Swallow (best initial test) – Shows "corkscrew" app but may appear NL



  - Manometry (most accurate test)
    ↳ High intensity, disorganized cxns. & intermittent.
  * Endoscopy not helpful.

THINK of DES as irritable bowel of the esoph.

Occult reflux can be responsible for esph. spasm.

• **tt**
  * Reassure the pt
  1st line → CCB (ex. Diltiazem or Nifedipine) & Nidrates
  2nd line: Isosorbide or Sildenafil
  3rd line of tt: Botox injection.

LOWER ESOPHAGEAL RING (Schatzki Ring)

- Lower esoph. ring (usually at squamo-columnar j.) ∃ time

Clinical Features

- DYSPHAGIA ← NOT ass. w Pain (NO odynophagia)
  - Intermittent NOT Progressive
  - ONLY solids esp. meat bolus

On barium swallow the ring should be ≤ 13 mm in diameter to cause sx.

- Almost always ass. w hiatal hernia.

Dx

- Barium swallow (Best initial test)
- Endoscopy

℞

Dilation by
- bougie method
- or through the scope hydrostatic balloon.

*Note: Pts are placed on PPI after dilation.

ESOPHAGEAL WEBS

i.e. → more Proximal, usually in the hypopharynx

Ex. Plummer-Vinson Webs

Clinical Features

- DYSPHAGIA (like rings)
  - No ass. Pain
  - Intermittent NOT Progressive
  - ONLY solids

esoph. Web: is a thin fold of tissue covered w sq. epithelium that protrudes into the lumen.

Plummer-Vinson Synd.
- Upper esoph. web
w. Iron-def anemia

*Rare, usually in Postmenopausal ♀
*Slight ↑ risk of CA

Koilonychia (spoon shaped nails)

premalignant SCC

Dx

- Barium Swallow (Best initial test)
- Endoscopy.

℞

- Plummer Vinson synd. may respond to ℞ of IDA.
- Dilation (like rings)

ESOPHAGIAL STRICTURE → Narrowing of the esophagus

Clinical Presentation

DYSPHAGIA — constant (NOT Intermittent)
BUT Progressive! (Slowly)
Solids THEN liquids

CAUSES

- long hx of incompletely treated reflux
- Prolonged NGT Placement
- Lye (bleaching agent) ingestion decades ago ⇒ i.e. corrosive esophagitis (Alkali is worse than acidic)

Dx
Barium swallow

Tt
Dilation

ESOPHAGIAL CA *Discussed in GI CA summary

- Dysphagia — constant & Progressive
  - RAPID growing (unlike stricture)
  - Solids THEN liquids.

- Dx BIOPSY
  *Details in GI CA summary

- Tt 5FU + RTX
  Surgery

SCLERODERMA ESOPHAGUS (Systemic sclerosis)

- 85% of pts w systemic sclerosis have esophageal disorder.
- It's the m.c CT disease involving the esoph.

Findings

- LES is wide open w no tone/pressure.
- Weak/Absent esophageal cons. (due to atrophy & fibrosis of smooth ms)

Biopsy will show atrophy & fibrosis of smooth muscles

Clinical features

- Dysphagia — Progressive, No pain, ?only solids
- Reflux

Dx → clear pic (usually no need for bx)
- Barium swallow & endoscopy

Tt
Treat the reflux (PPI)
F/U after 2-3 months

ZENKER'S DIVERTICULUM

- Outpouching of the upper esophagus (Posterior pharyngeal constrictor m@ at the back of the pharynx)
  - *due to weakness of muscles.

Clinical Features

- Dysphagia — transfer dysphagia (difficulty initiating swallowing)
  - Solids only
- BAD smell (halitosis)
- Food regurgitation (of food eaten several days earlier)

*Usually in elderly (weakness of ms)

Dx

Barium Swallow

⊘ Endoscopy & NG placement are CI (due to risk of perforation)

tt

Surgical resection

ESOPHAGITIS

- general term referring to either infxn or inflam. of the esophagus.
  - PAINFUL (Odynophagia), but ONLY on swallowing (unlike DES)

PILL-INDUCED ESOPHAGITIS

- Inflammation due to direct effect of contact btw the mucosa & pill.
  - usually in pts who ingest pills w/o water.

Ex. of pills
- NSAID
- KCl
- Iron sulfate
- Vit C
- Doxycycline
- Alendronate (oral m/cn) → Bisphosphonate
- Risedronate

Dx

Based mainly on **hx** — hx of taking pills (w/ small amount of water)
- **PAIN** ONLY on swallowing (odyno

tt

- by simply swallowing pills in the upright position & drinking enough water (to flush it into stomach)

INFECTIOUS ESOPHAGITIS - odynophagia

- Opportunistic infxns usually occur in → immunocompromised (pts w/ HIV & DM)
  - ↳ CD4 < 200
  - ↳ can occur in pts on steroids.

ex. **Candida** → you may see oral thrush
HSV
CMV

Dx & ttt

Give **Fluconazole** (empirical ttt)

- If improved → Continue ttt (until the offending organism is gone) or CD4 improved > 200
- If no improvement → Do endoscopy w/ bx

MALLORY WEISS TEAR SYNDROME

- Tear at gastro-esophageal junction
- leads to **hematemesis** (Preceded by Vomiting or retching)
- NOT a cause of dysphagia
- RARELY, it causes severe bleeding.

Dx

- hx

upper Endoscopy (direct visualization)

ttt

No need, resolve spontaneously

BARRET'S ESOPHAGUS

Sq. → Colm.

lower part of the esoph.

- Its a change in cell type from esoph. **squamous** into → specialized intestinal **metaplasia** (columnar) w/ goblet & epith. cells.

CAUSE: Chronic GERD

(BUT many pts lack GERD sx)

↑Risk of CA (Adenocarcinoma ONLY) not SCC
↑ by x30 than N/L

Dx only by **Endoscopy**

ttt PPI + F/U

If present do F/U by endoscopic surveillance (4 quadrant bx) every 2cm if no dys. every 1cm if dysp

F/U (2011 ACG guidlines)

- NO dysplasia : 3-5 yrs
- Low-grade dysplasia : 6-12 m.
- High-grade dysplasia : 3 m. (if ablation not done)

ttt of choice

ablation therapy (esp. for high grade)

RF
- GERD
- Smoking

EOSINOPHILIC (ALLERGIC) ESOPHAGITIS

↳ Immune-mediated chronic inflam. d.

* Pathogenesis
  involves IL-5

* Usually in ♂ bdw. (20-40)ys
* Strong ass. w allergies — IgE is ↑ in 20% of Ptt.
  ↳ usually ass. w Peripheral eosinophilia

Dx

- Endoscopy: "classic" finding is **scalloped aff.** w ridges & rings.
- w Biopsy → to confirm dx
  ↳ dense eosinophilic infiltration in the **mid-esophagus**
  (GERD might have ↑ eosinophils BUT only in the DISTAL esoph.)

~~tx~~ → difficult!

- Fluticasone or budisonide. (Cortisone)
- PPI maybe helpful.

* GERD in next summary

Gut Glands
The Gut

Source: MedStudy Kaplan 93

DYSPHAGIA

Hx taking / OSCE

Dys- means difficulty

→ It's difficulty in swallowing.

CAUSES & Sx of Dysphagia

Disease	Main Problem	Symptoms	Sx Precipitated by... (MedStudy)
Schatzki Rings & webs	Anatomic	Intermittent	ONLY solids
Stricture	Anatomic	Slowly Progressive	Solids, then liquids
CA	Anatomic	rapidly Progressive	Solids, then liquids
Achalasia	Motility / neurogenic	Long-Standing	Solids & liquids (simultaneously)
DES	Motility / Neurogenic	Intermittent	Solids & liquids (esp. cold)
Systemic Sclerosis	Various.	Slowly Progressive.	Solids & liquids
Neurological Disorders	Neurogenic	Various	liquids THEN solids

DDx

- Rings & webs
- Stricture
- CA
- Achalasia
- DES
- Scleroderma esoph.
- Zinker's diverticulum
- esophagitis
- Neurologic dystrophy (ex. Stroke, Parkinsonism, bulbar palsy, Pseudobulbar)

History Taking

Dysphagia
- Duration (long standing?)
- Onset (Rapid / gradual)
- Intermittent? / Progressive?
- Solids, liquids or BOTH? & order (solids then liquids? liquids then solids?)
- At what level does the food appear to stick?

Ass. Sx: Odynophagia (Pain on swallowing) — only on swallowing? (esophagitis) or not related? (DES)
- Site?
- Chest Pain.
- Wt loss? appetite?
- Regurgitation? undigested food? & bad smell? when?
- Heart burn?
- Lump in throat (globus)? Neck bulge (Pouch)?
- other GI Sx

Ask Resp. Sx: SOB, Stridor, Cough (when? nocturnal?), wheeze.

PMHx

- Previous dysphagia / Reflux / or known ulcer disease.
- Stroke / Neurological dz (bulbar palsy, MG)
- HIV

Drug hx

NSAIDs, Steroid inhalers

*Taking pills without water? (Pill-induced esophagitis)

FHx

CA

Social hx

Smoking, alcohol, diet.

Approach after hx taking:

START HERE!

If pt is prev. healthy w new-onset dysphagia →

Odynophagia Present? YES → Endoscopy (THINK esophagitis UNLESS pill-induced (unusual))

NO ↓

Sx of neurodysfxn – cough/gagging? YES → Modified Ba swallow

NO ↓

Ba Swallow

- LE ring → endoscopy
- Stricture → endoscopy
- Corkscrew esoph → Reassurance & medical tx
- Features of achalasia → endoscopy & manometry
- NL → consider endoscopy

Yash Gloria
The Exel

Gastroesophageal Reflux GERD

Source: Dossier Kaplan Medstudy 95

* Common disease.

CAUSES — loss of protective mech.

① **Loss of LES tone &/or Peristalsis**
ex. Smoking / Alcohol / Caffeine / Peppermint / & Chocolate ← Anticholinergic effects
CCB & nitrates.

② **↑ Gastric vol.**
ex. Stasis of food in diabetic pt. (delay emptying)
Pyloric Stenosis.

③ **Hiatal Hernia**

④ **↑ Gastric Pressure**
ex. Ascites, Pregnancy (due to Progesterone also)

SX *Not necessarily symptomatic!

- Sore throat / heartburn
- Bad, metal-like taste in the mouth.
- Hoarseness / Vocal cord dysfxn.
- Cough (usually nocturnal)
- Wheezing
- Epigastric / substernal pain.

Most non-cardiac chest pain (70%) are caused by GERD!

GERD may exacerbate asthma.

COMPLICATIONS

- Esophageal ulcers
- Stricture
- Bleeding
- Barrett Esophagus.
- ~~Exacerbate asthma.~~

Alarm Signals

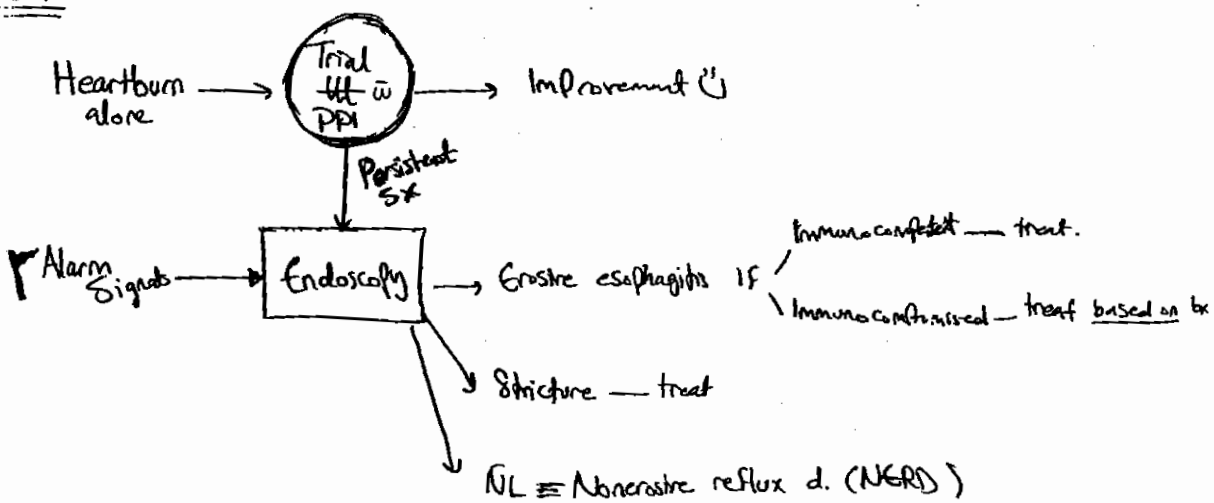
- Nausea/emesis
- Dysphagia / odynophagia
- wt loss / Anorexia / Anemia / Blood in stool
- Abnormal P/E
- FHx of PUD
- Failure to respond to PPI (8 wks)
- long duration of freq. sx (esp. in o↗ >45 yo)

DX

- Trial of PPI (In the ABSENCE of alarm Sx) ← Best initial step.
- Endoscopy (if trial of PPI failed / or Alarm sx present / or suspected Barrett's)
- 24hr PH monitoring (the most accurate test)

NL endoscopy does NOT r/o GERD, if normal it's called (NERD) ①

Dx



TREATMENT

* **Mild /or Intermittent**
  - Life-Style modification
    - Raise head on bed
    - wt loss
    - small meals / not fatty / at least 3 hrs before sleep. not sweet
    - Stop smoking, Avoid alcohol
  - Antacids (Prn)
  - H_2 blockers

* **Moderate/or Progressive**
  - Life Style modification
  - **PPI**

* If failed medical tt → Surgery (Nissen fundoplication)
  (in 5% of pts)

  S/E of surgery: Bloating
  Dysphagia
  Inability to belch.

Pts w GERD-related cough / hoarseness need longer tt — higher doses for sympt. improvement.

Good Luck
The End

ESOPHAGEAL HIATAL HERNIA

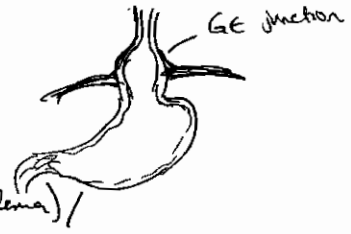
Source : Recall

TYPES

TYPE I ≡ Sliding
TYPE II ≡ Paraesophageal

SLIDING HIATAL HERNIA (TYPE I) — m.c

- **DEFINITION** : **BOTH** the Stomach & GE junction herniate into the thorax via esophageal hiatus
- **INCIDENCE** : > 90% of all hiatal hernia



- **SX** : mostly asx
  - BUT can cause **REFLUX** /
  - dysphagia (from inflammatory edema) /
  - esophagitis /
  - & Pulmonary Problems (2ry to aspiration).
- **Dx**
  - UGI series
  - Manometry
  - Endoscopy ± bx (for esophagitis)
- **COMPLICATIONS**
  - Reflux → Esophagitis → Barrett's Esophagus → CA & stricture formation
  - Aspiration Pneumonia
  - UGI bleeding (from esophageal ulcerations)
- **tt**

85% — MEDICAL tt ± antacids / H_2 blockers / PPI & also head elevation / small freq. meals / No food BEFORE sleep

15% — SURGICAL tt — IF failure of medical tt (Lap. Nissen fundoplication)

PARAESOPHAGEAL HIATAL HERNIA (TYPE II)

- **DEFINITION**: Herniation of all or part of the stomach through the esophageal hiatus into the thorax **without** displacement of GE junction.

- **INCIDENCE** < 5% of all hiatal hernias (Rare!)

- **Sx** (— Due to mechanical obstruction)
  - — Dysphagia
  - — Stasis gastric ulcer.
  - — Strangulation.

* Many are asx & **NOT** ass. w reflux (bcz of relatively NL position of the GE junction)

- **COMPLICATIONS**
  - . Hmg
  - . Obstruction
  - . Incarceration & Strangulation!

- ~~tt~~

SURGICAL (bcz of frequency & severity of complications)

NOTES

- **TYPE III** Hiatal Hernia:
  - Combined Type I & II
- **TYPE IV** Hiatal hernia:
  - Organ (eg. Colon/Spleen) + Stomach in Chest cavity!

The End
Good Luck

SURGERIES OF GERD

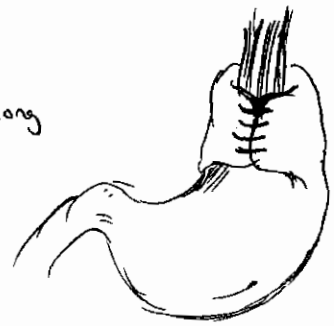
Indx for surgery in GERD

- Failure of medical tt
- Respiratory Problems like Pneumonia — resulting from reflux & aspiration of gastric mucosa.
- Severe esophageal injury (Ulcers / Hmg / Stricture / ± Barrett's)

SURGICAL OPTIONS

▸ Lap. Nissen

360° Fundoplication – 2cm long
(**Lap**aroscopically)



It works in 85% of cases

Mechanism of Nissen:
- Improves LES
- ↑ LES. tone
- Elongating LES ~3cm
- Returning LES into abd. cavity

o Postop. complications:
1. Gas-bloat Syndrome (Inability to burp/vomit)
2. Stricture
3. Dysphagia
4. Spleen injury requiring splenectomy
5. Esophageal Perforation
6. Pneumothorax

FUNDOPLICATION
360° → Nissen
240-270° → Belsey
200° → Toupet

▸ Toupet

INCOMPLETE Post. wrap (**200°**)
(Laparoscopic)

▸ Belsey mark IV

240-270° Fundoplication
(through **thoracic** approach)

▸ Hill

Arcuate ligament Repair (close large esophageal hiatus) & gastropexy to diaphragm (suture stomach to diaphragm)

Yash Gupta
The Med.

ESOPHAGEAL CA

- Mostly MALIGNANT ☹ only 1% benign!

TYPES

- **ADENOCARCINOMA** : — mc in USA
  - ↑ in incidence from Barrett's Esophagus
  - Distal 1/3
- **SQUAMOUS CELL CA (SCC)** — m.c worldwild.
  - Proximal 2/3
  - Usually environmental / Smoking & Alcohol (Synergetic effect!)

RF

- ♂ & Age > 50 yrs (Avg. 60-70), Black mm. (It's more in China --)
- Smoking
- Alcohol
  ] have synergetic effect (NOT additive)

- Esophageal disorders : - GERD / Barrett's Esophagus
  - Achalasia
  - Damage from caustic ingestion / stricture.
- Hx of RTX to the mediastinum.

CP

- Hx of dysphagia (It's the earliest sx BUT if it occurs then the disease has been for > 6 m.)
  - First for solids then for both solids & liquids
- Wt loss / weakness. / Chest Pain

* By the time we diagnose it, most of Pts are already in the advanced Stage!

Dysphagia does not usually develop until > 60% of esophageal lumen is obstructed!

ADENOCARCINOMA is ass. w:
- Barrett's Esophagus
- Ectopic gastric
- Tumors of cardia mucosa
- Esophageal gland

Dx

- History (Dysphagia)
- Barium Swallow
- Endoscopy w biopsy (for confirmation)
- CT & Endoscopic U/S (for staging)

* Full metastatic workup (CXR / Bone scan / CT / LFTs)

Premalignant disease
- Barrett's Esophagus
- Plummer Vinson Synd

DDx
- Leiomyoma
- Metz
- Lymphoma
- Benign Stricture
  - Achalasia
  - DES
  - GERD

Rx

If small & localized → Surgical resection

If large or Metz → Combination of CTX + RTX prior to surgery

CTX used:
- Cisplatin
- 5-FU

* Palliative care
  - Stenting
  - Palliative resection / Bypass
  - Laser therapy
  - Phototherapy

Px

5-yr survival is ~5%

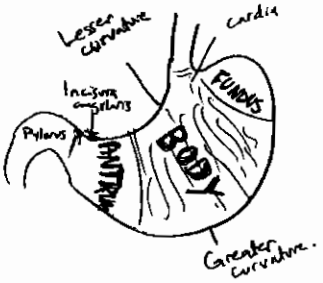
Yale Gluttis
The End

STOMACH

ANATOMY

- Cardia
- Fundus
- Body
- Antrum
- Pylorus
- Greater & lesser omentum.

*The space behind the stomach is the Lesser sac
*The pancreas lies behind the stomach.



The Opening into the lesser sac is the Foramen of Winslow.

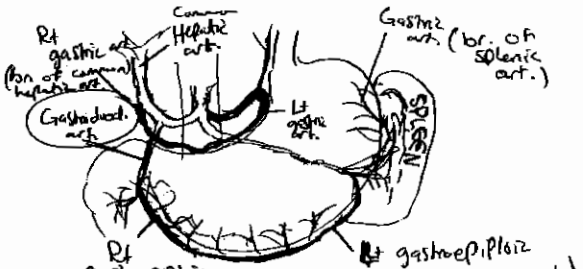
The folds of gastric mucosa are called → Rugae

BLOOD SUPPLY

- Lt gastric artery
- Rt gastric artery
] Lesser curvature

- Lt gastroepiploic art.
- Rt gastroepiploic art.
] Greater curvature

- Short gastrics. (from spleen)



VENOUS DRAINAGE

- Lt & Rt gastric v. → Portal v.
- Lt gastroepiploic v. → splenic v.
- Rt gastroepiploic v. → SMV

INNERVATION

Mnemonic
"LARP:"
Lt → Ant. wall
Rt → Post. wall
(Remember it as)

- Ant. gastric wall by Left vagus n. (gives branch to liver)
- Post. gastric wall by Rt vagus n. (gives celiac branch).
- Gastroduodenal pain is sensed via symp. afferents from $T_5 - T_{10}$

STOMACH CELLS

(Located in the BODY of the stomach) ← Parietal cell → Sxn of HCl, Intrinsic factor

- Chief cells → Sxn of Pepsinogen

(Fundus) ← Mucus neck cells → Sxn of Bicarbonate (HCO_3^{-2}), Mucus

(In the antrum) ← G-cells → Sxn of Gastrin

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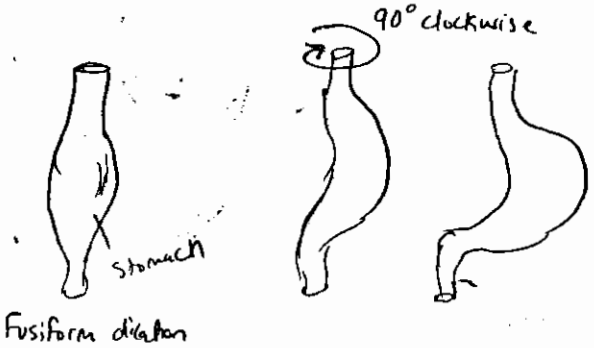
EMBRYOLOGY

It grows as a cylinder.

— there is a ventral & dorsal part

— It's suspended ant. → by ant. mesentery
Post. → by dorsal mesentery.

— It develops as a fusiform dilation BUT the dorsal part grows MORE rapidly than the ventral part
→ forming the greater curvature.

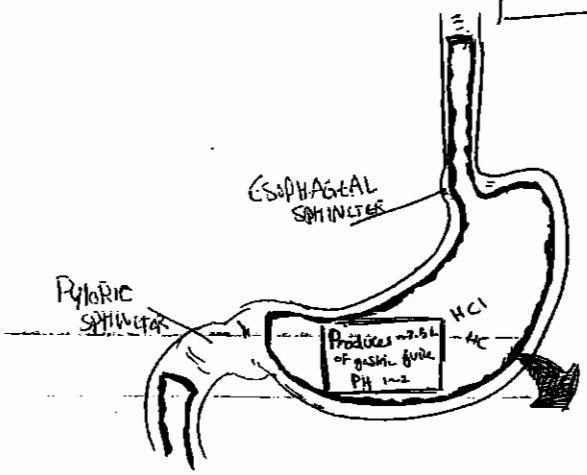


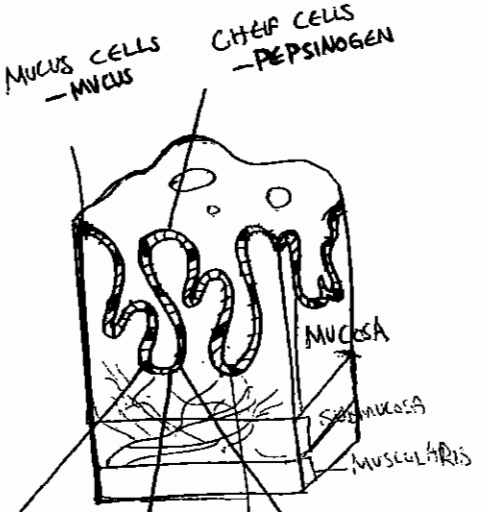
Dorsal part → greater curvature
Ventral part → lesser curvature.

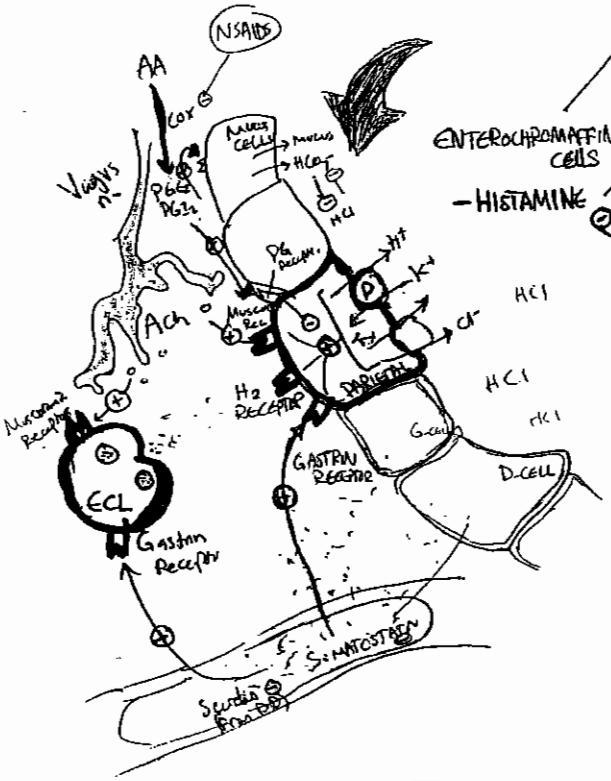
m.c congenital anomaly of the stomach is: Hypertrophic Pyloric Stenosis.

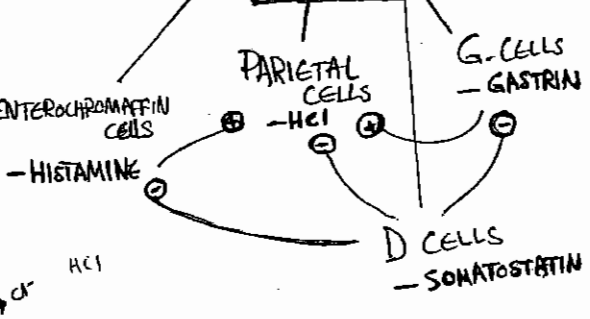
Study Guide
The Med.

Gastric Acid Physiology

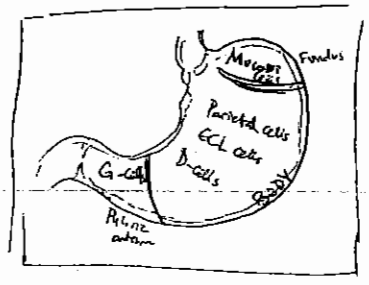








*Also see figure (1-3) Medstudy Page (9)



Notes:

* ↑PH → G-cells → Gastrin
  (From breakdown of food)
  (Like Ach, Hist.)
  Parietal cells → HCl
  ECL cells → Hist.

* Proton Pump is the final common Pathway → that's why PPIs are the strongest anti-gastric-acid drugs

Gastrin — from G-cells (Pyloric antrum)
- Dominant mediator of postprandial gastric acid production
- ⊕ BOTH Parietal cells & ECL cells.

* Stretching of the stomach ⊕→ Gastrin

Hist. — from ECL cells (body of stomach)
- ⊕ Parietal cells through H_2-receptors
- Mainly due to gastrin stimulation

Ach — from Vagus n.
- Direct neurocrine effect on Parietal cells.

↓PH will ↓ gastrin by ⊕ D cells to produce somatostatin & ⊕ DD to produce secretin.

Somatostatin & Secretin → Inhibitory
- from D-cells (body)
- from DD
- ↓ BOTH ↓ gastrin production
- ⊕ by ↓PH

- Stomach PH < 3 ⊕→ D-cells → Somatostatin ⊖→ G-cells (↓ gastrin)
- ↓DD PH ⊕→ Secretin ⊖→ G-cells (↓ gastrin)
  - ⊕→ Pancreas.

Good Luck
The End

Epigastric Pain (Dyspepsia)

Dyspepsia: non-specific term that refers to recurrent upper abd. Pain or discomfort. It includes: epigastric fullness / burning belching / bloating & heartburn.

CAUSES

- Non-ulcerative dyspepsia (n.c.c) — idiopathic / endoscopy is -ve — >60%
- GERD
- PUD (DU / GU)
- Gastritis
- CA <1% — least common

also
- Drug intolerance
- Food intolerance
- Chronic Pancreatitis / Pancreatic CA.
- Biliary colic
- IBS

Most dyspepsias are fxnal or due to medications.

ALARM Signs

Anemia (IDA)
Loss of wt
Anorexia
Recent onset of Progressive >a
Melena / hematemesis
Swallowing difficulty (Dysphagia)

PEPTIC ULCER DISEASE (PUD)

Source: Washington Dossier

It represents a spectrum of disease characterized by ulceration of the **Stomach** or **Proximal duodenum** — due to imbalance btw. acid sxn & mucosal defense mechanism.

- Common disease.
- Incidence ↓ due to — eradication of H. Pylori
  - more sanitation
  - Beter medical ~~tt~~
  - Improvement in quality of life.
  - more precations in the use of NSAIDs/ Aspirin.

[redacted]

DUODENAL : At the antral-Pylorus junction

GASTRIC : 5 categories

- Type I — lesser curvature (near angularis incisura) (60-70%) — ass. w/ ↓ mucosal Protection
- Type II — Lesser curvature & duodenal (DU+GU) (15%) — ass w/ ↑ acid sxn.
- Type III — Prepyloric (20%) — ass. w/ ↑ acid sxn
- Type IV — Proximal Stomach / cardia (near GE junction) — ass. w/ ↓ mucosal Protection
- Type V — Anywhere in stomach — medication-induced

[redacted] 4 etiologic factors responsible for the MAJORITY of PUDs.

m.c.c — ① **HELICOBACTER PYLORI** (H. Pylori)

- It causes BOTH **duodenal** ulcer (90%) & Gastric Ulcer (70-80%)
- It's infxn that Produces **Chronic antral gastritis**

* If eradicated → recurrence rate is very LOW
so if ulcer recurred — THINK of NSAID-induced PUD

2nd m.c — ② **NSAIDs**

- It cause BOTH duodenal (x8) & gastric (x40!!!) ulcers
- Due to ↓ PG Production
- DOSE-DEPENDANT relationship.
- It does not recur when NSAIDs are discontinued.

? ③ **SMOKING** (some say that smoking doesn't cause ulcer BUT delay healing of Preexistic ulcer.)

④ **ACID HYPERSECRETION**: Majority of Pts have **duodenal** ulcers (1)
ex. ZES

[redacted]

If uncomplicated — usually asx

If symptomatic :- Burning / gnawing intermittent epigastric pain (dyspepsia)
— relieved by food (if duodenal)
— exacerbated w/ food (if gastric)

± N, V & mild wt loss (esp. if gastric) / UGI bleeding
or: presents w/ complications!

[redacted]

- GERD
- Biliary colic
- Inflammatory & neoplastic Pancreatic d.
- Gastric neoplasms.

Remember the ALARM Sx:

- **A**nemia (IDA)
- **L**oss of wt
- **A**norexia
- **R**ecent onset of Progression
- **M**elena / hematemesis
- Swallowing difficulty (Dysphagia)

[redacted]

- Bleeding
- Perforation
- Obstruction.

[redacted]

- ENDOSCOPY (better) or barium contrast studies.

**Once the dx of PUD is confirmed, further testing is performed:

▸ H. Pylori infxn. tests
- Noninvasive ⇒ Serologic antibody test / Urea breath test / Fecal Antigen test
- Invasive ⇒ Biopsy (gold Standard) — most specific & sensitive also r/o CA
  & Urease test (CLO test)

▸ Fasting serum GASTRIN levels

Indx : If No hx of NSAIDs intake & -ve H-Pylori tests
or pts who have recurrent ulcer despite medical tx
or pts w/ multiple ulcers / unusual sites (2nd/3rd Part of DD / SI)
or complicated PUD
⇒ THINK of ZES (Zollinger Ellison Synd.)

▸ Endoscopic bx of gastric ulcers — to r/o CA

MEDICAL ttt

H. Pylori eradication

By triple therapy (1 PPI + 2 Antibiotics)
for 10-14 days

↳ Common regimen is "O-CLAM"
- **O**meprazole (PPI)
- **CL** arithromycin (Abx)
- **A**moxicillin (Abx)

- 90% effective in eradicating H.Pylori! ☺
- Antisecretory ttt is then continued.

NSAID – associated PUD

- STOP the drug
- Initiate antisecretory ttt

* If the NSAID must be continued → PPI is most effective in facilitating ulcer healing.

SMOKING cessation

- this facilitates ulcer healing

F/U endoscopy

- to ensure healing.

Sucralfate for mucosal Protection is not frequently used.

SURGICAL ttt

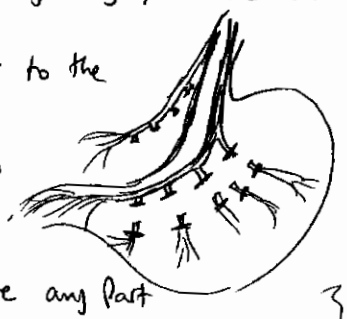
- RARELY done for UNcomplicated PUD
- Most common Indx → complicated PUD.

TYPES of surgeries

Highly Selective Vagotomy (HSV)

AKA: Proximal gastric vagotomy / Parietal cell vagotomy

- Transection of vagal fibers to the body of the stomach
WOUT interruption of fibers to the Pylorus



Advantages:
- we don't remove any part of the stomach
- we don't interfere w the process of emptying (No need for Pyloroplasty)

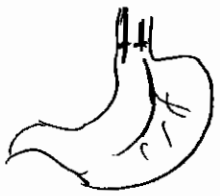
} less morbidity & mortality

Disadv: ↑Recurrence rate (15%) after 10yrs

- **Selective Vagotomy**

  We cut the nerve supply to the WHOLE stomach EXCEPT the hepatobiliary & celiac branches.

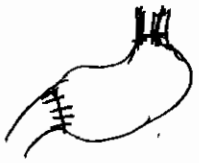
- **Bilateral Truncal Vagotomy ± Drainage**



  The vagus is cut
  So the stomach does NOT evacuate & there is some impairment in motility of the rest of the GIT
  ⇒ stomach must be drained by gastrojejunostomy or Pyloroplasty.
  (bcz Pylorus does NOT relax)

- **Truncal Vagotomy & Antrectomy**

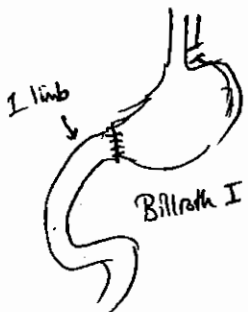
  - Transection of the vagus n. trunks
  ⇒ Then the distal 40% of the stomach is removed & anastomosed ± the jejunum.

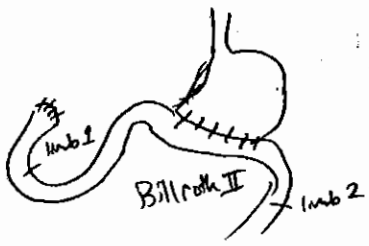


- **Subtotal (Distal) gastrectomy**

  — The duodenum is closed & the stomach is anastomosed to the stomach.

  TYPES: Billroth I & II





Remember 1 limb → Billroth (I)
2 limbs → Billroth II

COMPLICATED PUD

- PUD complicated by Hmg / Perforation / or obstruction
- In most cases, it's an indx for surgery in PUD.

BLEEDING PUD

- It's the leading cause of death dve to PUD
- 5-10% mortality
- the m.c.c of UGI bleeding (above lig. of Trietz)

CP

Vomiting ground-coffee blood or fresh blood.
Melena (Black tarry stool) or blood per rectum.

Mgt

- Aggressive resuscitation & correction of any coagulopathy
- Then endoscopy ± electrocautery w/wout epinephrine → Spontaneous cessation of bleeding occur in 70% of pts

***FINDINGS w endoscopy indicating ↑risk of rebleeding**

- LARGE sized ulcer
- visible vessels on a non-bleeding ulcer.
- Visible CLOT

⇒ Surgical intervention (if failure of medical tx)
(Vagotomy & drainage or vagotomy & Antrectomy)

The m.c site of bleeding in duodenal ulcer is POSTERIOR WALL
— typically eroding the **gastroduodenal Art.**

• PERFORATED PUD

↳ MOST common site is ANTERIOR wall

C/P

- Sudden onset of severe abd. Pain
  (Maybe less dramatic in elderly / hospitalized / or immunocompromised Pts)
- Fever / tachycardia
  Abd. wall rigidity (guarding) — due to peritonitis.

Labs

↑WBC

Imaging

on X-Ray — Air under diaphragm (in 80-85% of cases)

Mgt

- Aggressive Fluid resuscitation
- Analgesia
- Broad-Spectrum abx (for bact. peritonitis & sepsis)

After this → the pt is sent to OR.

Surgeries options

- Patch
- Patch + HSV (Highly selective vagotomy)
- Patch + Vagotomy & Antrectomy.

Remember!

Anterior ulcers Perforate & Post. ulcers bleed.

• GASTRIC OUTLET OBSTRUCTION (GOO)

It happens after the healing of a circumferential ulcer & fibrosis by scar tissue

or due to edema & spasm.

C/P

- Recurrent vomiting of Poorly digested food
- Dehydration
- Hypochloremic hypokalemic met. alkalosis.

On PE

- dilated full stomach (Central distention)
  Visible peristaltic waves & heard on auscultation
  +ve succesion splash.

*NGT will expel a muddy fluid in large quantities.

The wall of the stomach might become HYPERTROPHIED due to continuous Peristalsis.

DDx: Antral tumor obstruction

(6)

Mgt

. Insert NGT
. Start IV hydration
. Electrolyte correction
. Antisecretory meds.
*When stable → do endoscopy (to r/o antral CA & confirm Gastric outlet obst.)

If refractory to medical tt or the cause is scarring not edam:

Surgical options:

HSV + duodenoplasty
Vagotomy & Antrectomy

• INTRACTABILITY / NONHEALING ULCER

CAUSES
. RULE OUT CA!
. Look for persistent H. Pylori
. Non-compliant pt.
. Use of NSAIDs
. Motility disorder.
. ZES.

Yash Ghosh
The Great

GASTRIC SYNDROMES

POSTGASTRECTOMY SYNDROMES
- Dumping Syad.
- Blind loop Syad.
- Afferend loop syad.

- All are now rare. Why? bcz < 5% of PUD cases warrent surgery

* Dumping Syndrome
- Consists of postprandial vasomotor Sx: Palpitations, Sweating, lightheadedness
- TYPES:
  - Early — 30 min after eating, unknown etiology
  - Late — ≥90 min after eating, due to hypoglycemia
- ttt (Same for BOTH type)
  - Restrict sweets & lactose-containing food
  - Encourage frequent small meals

* Blind Loop Syndrome
- **Bacterial Overgrowth** in a loop
- Usually in pts w prev. gastrectomy or Billroth II gastrojejunostomy
- C/P Fat & B12 malabsorption.
- Labs Low xylose absorption test (≠ S.I mucosal Problems ≡ transport Problem)

* Afferent Loop Syndrome
- With **gastrojejunostomy** (* btw. Stomach & jejunum)
- "Afferent loop" = the portion that was **bypassed**
- C/P: Abdominal Bloating & Pain (20 min – 1 hr after eating)
  Vomiting often relieves Sx [Emesis is often Bile-stained]
- Etiology
  might be due to incompletely draining efferent loop which fills w biliary & Pancreatic Sxn).

GASTROPARESIS IN DM — Due to neuropathy

- Highly VARIABLE gastric emptying is seen in diabetics, may be: Slow / NL / or fast!

BUT long-term DM tend to develop SLOW gastric emptying (Gastroparesis)
(In Type I > II)

- BG >200 mg/dL, Results in:
  - ↓ Antral motility
  - ↑ Delayed gastric emptying.
  - May have DIRECT -ve long-term effect on Gastric emptying
- Conversely, Gastroparesis itself → ↑ BG! (due to delay of insulinemic & glycemic resp to CHO)

∴ So it's a **Vicious Cycle** (to cut this off & at least minimize it: tight glu control)

C/P - N, V
Early satiety
Predisposition for bezoars.

WORK-UP
- Requires Ruling Out OBSTRUCTION first
- Then, dx confirmed by Radioisotope-labeled solid meal.

Tx
- Good hydration
- Low-fat diet
- Tight control of BG
- ↓ Metoclopramide (for long-term use)
  - ↳ CAUTION: It might cause PERMANENT Extrapyramidal S/E.
  - (Alternative) → IV Erythromycin — ⊕ gastric motility (similar in structure to motilin)
    - Less useful for long-term Tx.
    - Can be used in acute setting when Gastric motilin is inhibited (short-term) by severe stress.

CAUSES of Gastroparesis

1. Diabetic NEUROPATHY
2. Autonomic Dysfxn — Amyloid neuropathy
3. Infiltrative Process of S.M — Scleroderma/Amyloidosis
4. Antecedant viral infxn — Norovirus / Rotavirus
5. CNS disorder (Stress / MS / Parkinson's / Tumor / cord injury)
6. Post vagotomy
7. 1/3 → 1/2 of cases are IDIOPATHIC

The End

Source: Lecture
Lesh Manual
119

Bariatric Surgery

- Obesity is the 2nd MC cause of Preventable Death!
  → It is a disease w/ many commorbidities.

* What is a comorbidity?
  - A comorbidity is a condition that resolves with the Rx of the disease (ex. obesity)

* What comorbidities occur with obesity?
  - OSA - sleep apnea is the MC comorbidity of obesity & 70% - 85% of cases can be cured w/ Bariatric surgery!
    (OSA involves the deposition of fat in the neck & upper airway — causing sleep apnea & hypoventilation syndrome)

- Other Comorbidities

GI: - GERD (due to ↑ abdominal pressure), Constipation, Colon Ca

♡: HTN (62% cured), DM (77% cured), Heart failure, Hyperlipidemia.

🦴: Osteoarthritis, Disc prolapse.

♀: PCOS, Pseudotumor cerebri, Urge/stress incontinence, Depression!

Resp (+) obesity ↓ All Vital Capacities! (ex. Tidal Volume)

* ∝ ∞

Notes
- Once a patient reaches Morbid obesity (M.O) → the medical (conservative) failure rate is about (100%)!
- Bariatric surgery is the most effective, sustainable method of wt loss, with a failure rate of ~10%

BMI Scale

(1) Normal : 18.5 - 24.9
(2) Overweight: 24.9 - 29.9
(3) GI obese : 30 - 34.9
(4) GII severe obesity : 35 - 40
(5) GIII Morbid obesity : 40+
(4) Super obese : 50+
(5) Super Super obese : 60+

(+) comorbid

INDICATIONS

(1) G III (M.O) & up (BMI 40+)
(2) G II (+) comorbidities
(3) Severe comorbidities
(4) Social & Psychological implications

Shift for (Asian Population) (less BMI) → (1) BMI 32 (+) comorbidities
(2) BMI 37 & up

* Age is no longer a factor for surgery. If fit for surgery → proceed!!

Methods of Weight Reduction

- Conservative
  - Life Style Modification
- Surgical
  - Maintains wt loss

* Points to Ask for a Surgical Candidate *

(1) Sweet-eater? Salty foods?
(2) Family history? (of obesity)
(3) Maximum wt reached?
(4) Minimum wt reached?
(5) Trials of conservative Rx? Diet? Exercise?
(6) Sx of Comorbidities?
(+) Motivation

Types of Bariatric Surgery

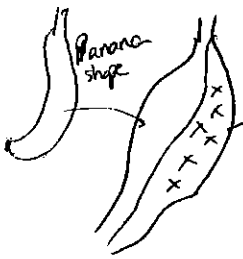
(1) Restrictive : VBGT, LAGB, LSG
(2) Malabsorptive : Pure malabsorptive procedures are no longer done!
(3) Combined : Fobi, BPD DS

Note.

- Failure of Surgery is if the Excess Wt Loss < 25 kg!
  & Surgeries success rated according to EWL (excess wt loss!)

(C) LSG (Laparoscip Sleeve Gastrectomy

A.k.A → Banana Stomach.



→ 85% of Stomach Reseeted!

- This used to be done as Bridge Procedure for Bilio Pancreatic Procedure. (A Bridge Procedure can be done pre-op / before the 'real' surgery to lose wt before & make the 'real' surgery easier!)

Pros ☺

- Good EWL [80% is lost!]
- A Re-Sleeve Procedure can be done or a Bypass if the original surgery does not have Satisfactory Results!

Cons ☹

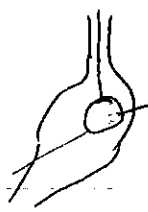
- Not enough Long-Term Results
- Morbidity is the same as Bypass
- Fever is an OMNIOUS SIGN! ↳could mean a LEAK - which is hard to manage
- Stenosis may be a complication
- Nutritional complications due to ↓ intake (but less than malabsorptive)

* * *

NOTE!

- Another Bridging Procedure:

- Ballon → placed endoscopically → Not considered Bariatric Surgery

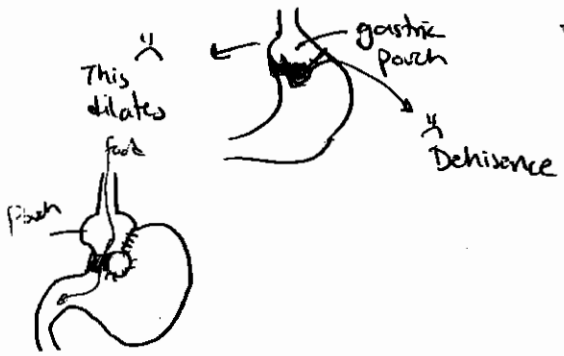


→ inflate w/ saline or gas for 6 (few) months for them to lose wt pre-op. before 'real surgery'

- Very obese pts are difficult to operate on, so this can be done as a bridging procedure to lose wt pre-op.

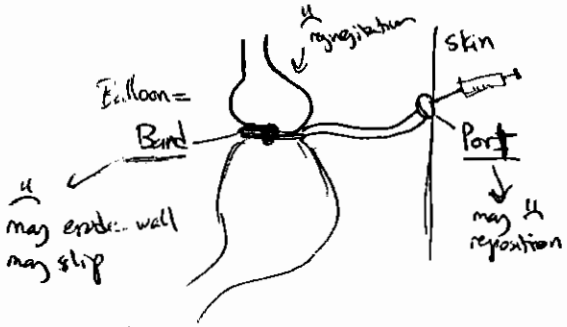
① RESTRICTIVE

Ⓐ VBGT (Vertical Banded Gastro Plasty)



* **NOT** Done anymore
* ↑ Failure rate
  ↳ Dehisence + dilatation of gastric pouch

Ⓑ LAGB (Band) Lap. Adjustable Gastric Band



→ Placing a silicone band with an inflatable balloon around the prox. stomach at angle of His.

- Laparoscopic procedure, placing a band.
- It is reversible (no removal of stomach) & adjustable, because there is a subQ port that can be injected with Saline solution to inflate the balloon more (if not losing weight) or saline can be suctioned to deflate the balloon if too much vomiting.

Pros ☺

→ No resection of stomach, less dangerous (No leaky peritonitis)

→ Good for Solid eaters (Rice bread), because they need time to pass.

→ Reversible, Adjustable.

→ Can be used for Borderline BMI (Relative C/I in BMI >50)
CC/I in Hernia or Reflux are you can ↑ pressure)

Cons ☹

→ Not good for sweet eaters (dissolves and is absorbed!)

→ Band may erode through the wall

→ Band Slippage – is an EMERGENCY!

→ Port may reposition (so fix it into the fascia!)

→ S/E: Reflux, Regurgitation, Vomiting, Esophageal dysmotility

→ Overall, Not many short term complications BUT it is the LEAST Effective in terms of EWL !! (lose 50-60% EWL) (3)

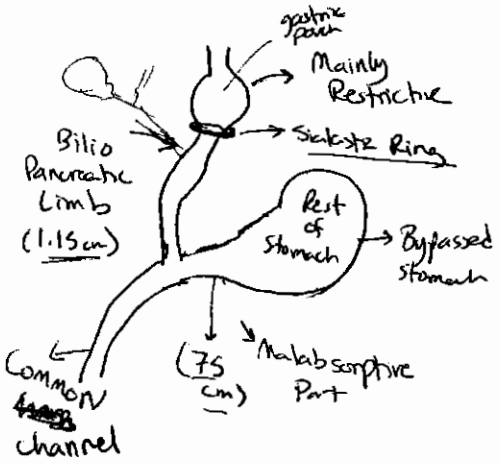
② MalABSORPTIVE

- Pure procedures not done

③ COMBINED

Ⓐ Fobi : = Banded Gastric Bypass

→ Combined malabsoptive + restrictive, But MORE RESTRICTIVE!



→ Small Pouch (Restrictive) with 2 m of Malabsorptive bowel.

→ ALL these procedures have failure →

- Dilatation of Pouch
- Vitamins for Life ! (Malabsoptive)

Dilated part → Jejunum may dilate like a new stomach

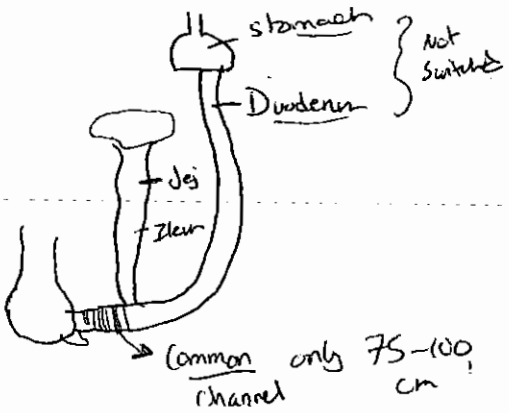
1.15 cm
+ 75 cm = 2 m Total Malabsorptive, Rest is Common

Ⓑ BPD DS (BilioPancreatic Diversion ± Duodenal Switch)

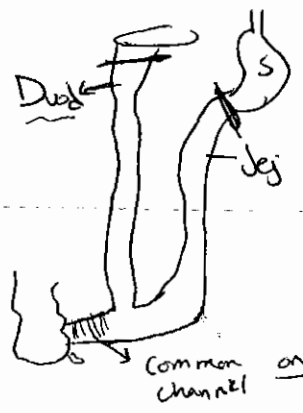
→ This is combined but MORE MALABSOPPTIVE!
→ Severe vitamin deficiey, ↑ Morbidity / S/E
→ Less commonly done !!

S/E
- Anemia
- Malnutrition
- Dumping syndrome
- Marginal Ulcers

BPD w/out duodenal switch



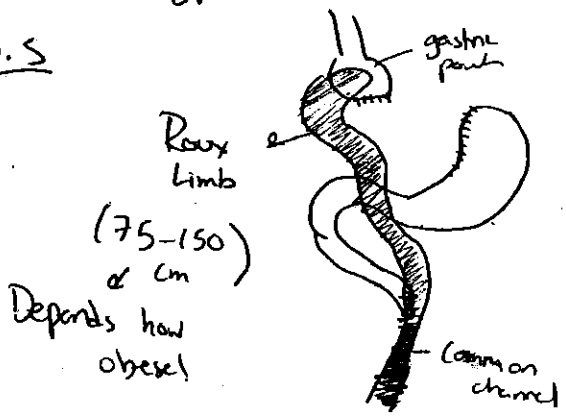
BPD w/ Duodenal Switch



Duodenum + Jejum switched!

© RYGB (Roux-en-Y- gastric Bypass)

- Most popular surgery in U.S
- 70% EWL
- Combined Procedure



IMPT ☺

RYGB (VS.) Fobi

→ Fobi is a modification of RYGB, but rather than staples, it uses a **silastic ring** around the distal end of the pouch (to stimulate the pylore valve & prevent stretching of dilatation of the bowel under the pouch)

Bushra Takhi

GASTRIC CA

Source : Washington Recall Dossier

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TYPES

- ADENOCARCINOMA (m.c, 95%)
- GIST (GastroIntestinal Stromal Tumors)
- Lymphoma
- Carcinoid (RARE!)

Gastric CA is the disease of elderly >60 yrs

- [redacted]
  - 2nd m.c CA worldwide (more in Japan)
  - Incidence : ↓ dramatically (due to the eradication of H. Pylori)
  - Originating from mucus-producing cells

RF

- Diet — Smoked meat / ↑Nitrates / ↓fruits & vegetables / Smoking
- ♂ / ↓ Socioeconomic status / Blacks > whites / Blood group type A
- FHx
- H. Pylori infxn.
- Atrophic gastritis / Adenomatous gastric Polyps.
- Prev. Partial gastrectomy. (>10 yrs)
- Menetrier disease (Large stomach folds from epithelial cell hyperplasia)

gAstric = type A

Recent studies showed that NEITHER alcohol nor Ulcers cause Gastric CA

BUT Gastric CA can present w an ulcer.

Protective factors:
- Aspirin
- Vit. C
- Diet: ↑ vegetables & fruits.

SUBTYPES

- **DIFFUSE TYPE** (70%) — Arise from lamina propria (No glands)
  - (In PROXIMAL stomach) — esp. the **Cardia** → BUT can affect any part of the stomach
  - Worse Px than intestinal type
  - Ass. w invasive growth pattern w rapid submucosal spread.
  - Occurs in younger pts, ♀
  - Metz is more common in this type.
- **INTESTINAL TYPE** (30%) — Arise from gastric mucosa.
  - In DISTAL stomach
  - Ass. w **H. pylori** & other environmental factor.
  - ↳ this will lead to Chronic gastritis → Intestinal metaplasia → dysplasia
  - Well-formed glandular structures.

S&S – generally involves NON SPECIFIC S&S

most Pts present at late stage.

(SX) Remember sx as "**WEAPON**"

- **W**t loss (m.c sx at presentation)
- **E**mesis
- **A**norexia & early satiety.
- **P**ain / epigastric discomfort (m.c early sx)
- **O**bstruction
- **N**ausea

Dysphagia is ass. ω Proximal CA NOT distal

Gastric outlet obst. is ass. ω distal CA NOT Proximal.

Signs of

- Anemia
- Coffee-ground emesis / melena / heme occult
- Epigastric mass (in advanced d.) - Lt axillary adenopathy

CLASSIC Physical findings that represent metastatic & incurable d.s

1. **Virchow's Node** : Enlarged Lt Supraclavicular nodes.
2. **Sister Mary Joseph's Node**: Infiltration of the umbilicus
3. **Blumer's Shelf** : Fullness in the pelvic cul-de-sac (Solid Peritoneal deposit ant. to the rectum forming a "shelf" Palpated on PR)
4. **Krukenberg's Tumor** : Enlarged ovaries on pelvic examination. (metz to ovaries)
5. Hepatosplenomegaly ω ascites & jaundice.
6. Cachexia
7. **Irish's Node**: Lt axillary adenopathy from gastric CA metz.

DX

Barium meal (double contrast)
or **Endoscopy** (The method of choice!)
ω **bx**

Tumor Markers

- CEA
- AFP

Useless!

Screening ω endoscopy or contrast studies recommended ONLY in high risk Pts :

- Pts > 20 yrs Post Partial gastrectomy
- Pts ω Pernicious Anemia / atrophic gastritis.
- Immigrants from endemic areas.

Staging TNM classification

By CT or endoscopic U/S & PET, laparoscopy for metz

DDx

- Adenocarcinoma
- leiomyoma
- leiomyosarcoma
- lymphoma
- carcinoid.
- ectopic Pancreatic tissue
- gastrinoma.
- Benign gastric ulcer.
- Polyp.

Histologic Morphologies

Ulcerative (75%) / Polypoid (10%) / Scirrhous (10%) / Superf. (5%)

Routes of Metz

Hematogenous & lymphatically.

* PET is useful in detecting nodal & distal metz. Not apparent on CT.
* Laparoscopy (surgical staging!) is used to r/o peritoneal implants & to evaluate for liver metz.

NON-operative CA
- Distant Metz (ex. liver metz)
- Peritoneal implants

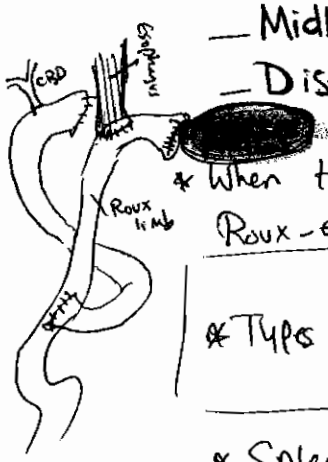
⇒ 10-15% of pts are inoperable.

Remember!
- Proximal gastric CA are more common than distal.
- Cancers are more common on Lesser curvature than greater curvature. "less is more"

P53 genetic alteration is seen in >50% of Pts w/ gastric CA.

* SURGICAL resection w/ wide margins (>5 cm) checked by frozen section & L.N DISSECTION

If the tumor is:
- **Proximal** → Do Total gastrectomy
- **Midbody** → Do Total gastrectomy
- **Distal** → Do distal subtotal gastrectomy (Removal of 75% of stomach)



* When total gastrectomy is performed, Roux-en-Y limb is sewn to esophagus.

* Types of anastomosis: Billroth II (NOT I) or Roux-en-Y

Billroth II
- Truncal vagotomy
- Antrectomy
- Gastrojejunostomy

* Splenectomy is done when tumor directly invades the spleen / splenic hilum / or splenic hilar adenopathy.

* Extended L.N dissection:
  - D1 → Perigastric LN
  - D2 → Splenic art. L.N / hepatic art. LN / Ant. mesocolon LN / Ant. Pancreas LN / & crural LN

* Adjuvent tt: Postop. CTX & RTX (done in stage II & III)

25% of Pts are alive 5 yrs after dx in U.S
BUT 50% of pts in Japan are alive at 5 yrs. Why?! bcz in Japan they have aggressive SCREENING & capturing early CA.

Linitis Plastica: AKA 'leather bottle'
When the entire stomach is involved & looks thickened (10% of cancers)

- [redacted] (Gastrointestinal Stromal Tumor)
  was Prev known as: Leiomyosarcoma.
- [redacted]: CAJAL (interstitial cells of Cajal)
- **Sites**: GI tract! from esophagus → rectum
  - m.c. site is STOMACH (60%)
  - other sites: - S.I (30%)
    - DD (5%)
    - Rectum (3%)
    - Colon (2%)
    - esophagus (1%)

Tumor marker
C-KIT
(CD117 antigen)

- **Sx**: - GI bleeding / occult bleeding
  - Abd. Pain
  - Abd. mass
  - Nausea
  - distention
- **Dx**:
  CT, endoscopy, colonoscopy
  for metz → do PET scan
- **Px**:
  - Local spread, distant metz
  - Poor long-term Px
- **tt**:
  Resection w -ve margins ± CTX (Imatinib)
  ↳ tyrosine kinase inhibitor. "Gleevec"
  - NO need for L.N dissection.

[redacted] <5% of gastric CA

- The stomach is the m.c involved organ in ~~extra~~nodal lymphoma
- Usually B-cell, non-Hodgkin lymphoma
- m.c site → DISTAL stomach. (cuz it has more lymphatics)
- ↑ risk w/ H. Pylori infx

POOR Prognostic factors:
- Involvement of lesser curv.
- large tumor size
- Advanced stage

S&S nonspecific (as in adenocarcinoma)

Tt IF MALT (low grade) → treat H. Pylori (by abx)
IF MALT (high grade) or non-MALT → RTX/CTX ± surgical resection

*Resection is reserved for Pts w/ Bleeding or Perforation.

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GI LYMPHOMA

→ HL
↘ NHL → Nodal
↘ Extra Nodal

} General classification of lymphoma

NHL

① Nodal - 70% (From LN basins)

② Extranodal - 30% → GI → MC extranodal (50%)
↘ Others (50%

Primary GI Lymphoma

→ NO lymphadenopathy otherwise
→ N bone marrow ⇊
→ N Peripheral Blood Smear
→ Disease confined to a certain affected viscus
→ Absence of hepatic or splenic involvement unless direct extention of 1o Tumor

GI

* MC → Gastric Lymphoma
2nd - SI, Colon, others

Sx similar to Gastric Adenocarcinoma

Gastric lymphoma

- MC site of G.I Lymphoma
- MC in distal stomach (antral)
- Pts > 50 years old
- 6th/7th decade
- ♂
- HIV is a RF

C/P

- Asx
- Abdominal Pain (MC)
- Anemia due to occult blood loss
- Late dx ⇊

S.I Lymphoma

- #2 MC
- Bimodal distribution
- Presentation may depend on site of obstruction, and may present as an emergency.

Types of GI Lymphoma

MC ① Diffuse Large B-Cell
② MALT
③ BURKITT
④ FOLLICULAR
⑤ MANTLE
⑥ ENTEROPATHY T-Cell

× × ○

① Diffuse Large B-Cell

- MC
- Stomach, Ileocecal

Bcl-2, Bcl-6

② MALT

- Associated w/ H.Pylori (90% gastritis from H.Pylori)
- Multifocal, Distal (Antrum), Lymphoepithelial lesions

MALT has the best Prognosis!

③ Burkitts

- EBV (RF)
- Starry sky histology
- Younger pts
- C-MYC gene (oncogene)
- Aggressive
- Cardia, Body, Terminal Ileum

④ Follicular

- Duodenum

⑤ Mantle Cell

- Polyposis in Small Bowel
- Tends to compress rather than infiltrate.

⑥ Enteropathy T-cell

- Celiac disease (RF)
- Jejunum & Ileum
- Circumferential Ulceration
- Eosinophils in histology

Staging

WHO < Low Grade / High Grade

TNM – No consensus
- Can use TNM for gastric cancer

Dx

- Endoscopy
± H. Pylori Test for MALT

Intestinal Lymphoma Dx:
- Emergency in 30-50% cases
- 90% dx intra-op
- ± Endoscopy

Staging

- CT chest/abdomen/pelvis
- BM biopsy
- Biopsy of enlarged peripheral LN

Rx

① Gastric – Conservative, Medical .. Mainly → Eradication of H. Pylori

② Intestinal – Surgery

MALT → ① Low-grade → Antibiotics for H. Pylori eradication
Remission rates 70–100%.
If Fail – chemoradiation or surgery

② High grade or NON-MALT → chemoradiation alone

Surgery Indx: ① Fail Chemoradiation
② Emergency – hmg or perforation

To Sum Up,

- Gastric Lymphoma
  - Most Common
  - Better Prognosis (& MALT is the best)

Rx

- Low-Grade MALT → H. Pylori eradication
- High-Grade MALT → Chemo radiation } → failure → Surgery → Resection!
- NON-MALT → Chemo radiation } → failure → Surgery → Resection!
  (Diffuse B cell, Burkitt, etc...)

* Surgery also for emergency cases

- Intestinal lymphoma
  - Many present as emergency cases, ex. obstruction Hmg
  - Need Surgical Resection

Bushra Tbakhi

Source
Dossier
Uath Manual

GIST

Source
- Wash Manual
- Recall

135

INTRO • Gastrointestinal Stromal Tumors (GIST) are only 3% of gastric Ca and arise from mesenchymal components of the gastric wall.

*MC Site → Stomach, then S.I
Other sites → esophagus, rectum, colon.

• Age 60 yrs, ♂ > ♀.

Cell of Origin
- Interstitial cells of Cajal

C/P
- They can grow very largely extraluminally before being symptomatic
- May have vague abdominal pain, discomfort 2° to mass
- May → GI hmg due to necrosis of overlying mucosa.
- Other GI Sx: Nausea, Distention

Dx • Endoscopy w/ FNA bx

Staging - Staging → CT of abdomen/pelvic
CXR
± PET scan for Distant Mets

Grade
- Tumor Size
- Histologic Freq of Mitoses

TUMOR MARKER - CD 117 (c-kit) → MOST GISTs express C-kit §
↳ Transmembrane Tyrosine kinase Receptor
So: CTx → Tyrosine kinase Receptor (-)

PROGNOSIS → Depends on Spread

Poor Prognosis :
- Tumor > 5cm
- MF > 5/10 HPF
- Male
- Necrosis
- Age > 40
- Local Recurrence
- Aneuploidy.

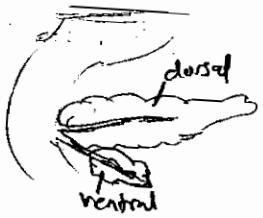
GLEEVEC
Imatinib Mesylate
C-kit receptor inhibitor (-)
for
- Mets w/ GIST
- reccurent GIST

Rx
- Lap Resection with 2cm -ve margin (grossly normal gastric wall)
- NO LN dissection needed! LN mets rare
- ± Chemotherapy (But NOT radio sensitive)
↳ usually dont respond !!

Z Bushra Thathi

PANCREAS

EMBRYOLOGY



- During the 4th wk of gestation, Pancreas begins development from duodenal endoderm
- 2 Buds form (which rotate & fuse by 8th wk):
  - **VENTRAL Bud** ⇒ Uncinate Process & Part of the head
  - **DORSAL Bud** ⇒ Remaining part of the head / neck / body / & tail

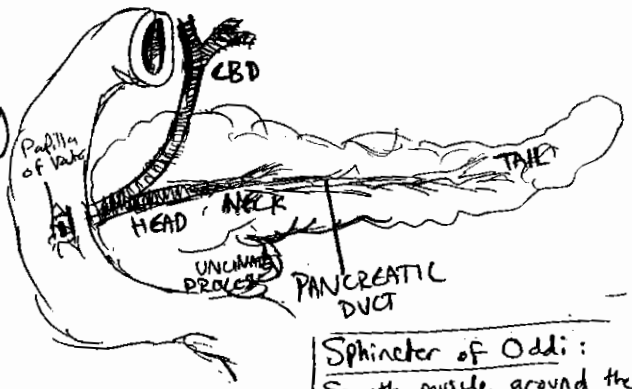
* The ventral bud rotates w/ duodenum & then, migrates Post. to fuse w/ the dorsal Part.
* The ventral duct will take over & open into duodenum.
* The dorsal duct may persist & opens into duodenum BUT it usually disappears.

ANATOMY

Site: Retroperitoneal (Post. to stomach / transverse mesocolon / & lesser omentum) at the Level of **L2**

Structures
- Head
- Neck (Portion ant. to SMV)
- Uncinate process
- Tail ("tickles the spleen)

Ducts
- Wirsung duct
- Santorini duct (Small)



Sphincter of Oddi: Smooth muscle around the ampulla of Vater.

BLOOD SUPPLY

Supply the head:
- Celiac Trunk → Gastroduodenal
  - → Ant. Sup. Pancreaticoduodenal art.
  - → Post. Sup. Pancreaticoduodenal art.
- SMA
  - Ant. Inf. Pancreaticoduodenal art.
  - Post. Inf. Pancreaticoduodenal art.

Supplies the neck / body / & tail:
- Splenic Art. → Dorsal Pancreatic art.

INNERVATION
- (Symp.) Pain sensation by Celiac Plexus
- (Parasymp.) for glands / ducts by br. of vagus n.

TYPES OF PANCREATIC CELLS

95% exocrine
2% endocrine
rest is extracell. matrix & vessels/ducts

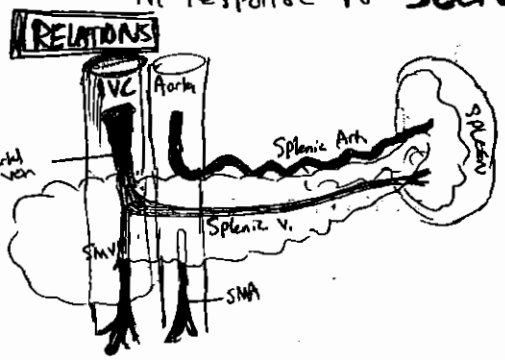
- **ENDOCRINE CELLS** (Islets of Langerhans) 2%
  - α-cells Secretes **Glucagon**
    - Fxn: Promotes the conversion of hepatic glycogen. ↑ glu level
  - β-cells Secretes **Insulin** (also secretes C-Peptide)
    - Fxn: Promotes glu transport into cells ↓glu level.
  - D-cells Secretes **Somatostatin**
    - Fxn: ⊖ release of GI hormones / gastric acid / electrolytes 5-I
  - PP cells Secretes Polypeptides / Vasoactive Intestinal Peptide (VIP)

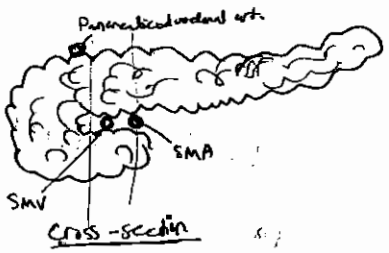
- **EXOCRINE CELLS** (Acinar cells) 95% & Centroacinar.

Acinar cells
Secrete enzymes — Trypsin, Chemotrypsin, Amylase, Lipase, Carboxypeptidase
} Secreted as inactive granules until they're activated by **enterokinase** in the duodenum.

Centroacinar & ductal cells
- Secrete water & electrolytes (ex. Na^+, K^+, HCO_3^-, Cl^-) in response to **Secretin** stimulation.

Secretin is the most potent endogenous stimulant of bicarbonate SRN.





Gabe Ghaith
The End.

ACUTE PANCREATITIS

Hrs the inflammation of the pancreas.

CAUSES

Remember it as "I GET SMASHED"

- **I**diopathic
- **G**all stones
- **E**thanol (Alcohol)
- **T**rauma
- **S**teroids
- **M**umps
- **A**utoimmune (ex. PAN)
- **S**corpion bite (RARE!)
- **H**yperlipidemia / Hypercalcemia
- **E**RCP (5-20%)
- **D**rugs (diuretics / isoniazid (INH))

DDx

- Biliary colic / Cholecystitis
- Gastritis / PUD
- Perforated viscus
- SBO
- Mesentric ischemia / infarction.
- Inf. MI / Pneumonia
- Ruptured AAA

Sx

- Sudden onset, Persistent deep epigastric pain
- Radiation to the back.
- Pain is ↑ when supine, ↓ on leaning forward.
- Severe N, V & Fever.

Signs

- Epigastric tenderness
- Diffuse abdominal tenderness
- ↓ Bowel sounds (Adynamic Ileus)
- Fever
- Dehydration / Shock (due to fluid sequestration)
- Signs of Hemorrhagic Pancreatitis (ONLY if severe):
  - **Umbilical** hemoperitoneum (Cullen's Sign)
  - **Flank** hemoperitoneum (Turner Sign)
  - **Fox's** sign (Bluish discoloration of inguinal lig.)

Dx Mainly based on HISTORY

- **LABS**
  - CBC (↑WBC : 10,000 - 30,000)
  - ↑Amylase (more sensitive) / ↑ Lipase (more specific)
  - LFT (If ALK PHOS > x3 ↑ :) — THINK biliary stones
    AST > ALT — THINK alcohol cause.
  - You should also order:
    - Calcium
    - Chemistry
    - Serum lipids.

- **AXR**
  - may show gallstones. (only 10% are visible)
  - "Sentinal Loop": Air-filled small bowel in LUQ — m.c sign of pancreatitis on AXR
  - "Colon cutoff": Abrupt ending of transverse colon.

- **RUQ U/S**
  - Reveals stones
    Phlegmon (collection of Pus/fluid)

- **CT** It's Prognostic
  Used to evaluate necrotizing Pancreatitis.

Assessing SEVERITY

- **SKIN FINDINGS**
  - m.c finding is erythema of flanks
  - Cullen's sign / Turner Sign / Fox's sign.

- **CT criteria for severity** —
  - (A) NL
  - (B) enlargement
  - (C) Peripancreatic inflammation
  - (D) Single fluid collection
  - (E) Multiple " Collection

(2)

SEVERITY SCORING SYSTEM

Ranson's Criteria (NOT specific/sensitive)

evaluate w/in 24 hrs "GA LAW"

- Glu >200 mg/dL
- Age >55
- LDH >350 U/L
- AST >250 U/L
- WBC >16,000

w/in 48 hrs "C HOBBS"

- Ca <8 mg/dL
- Hct ↓ <10%
- O_2 Arterial PO_2 <60 mmHg
- Base deficit >4 meq/L
- BUN ↑ >5 mg/dL
- Sequestered fluid >6 L

4 / 5 / 6 ↓

⇒ MORTALITY RISK

0-2	1%
3-4	16%
5-6	40%
7-8	100%

APACHE II (Good specificity & sensitivity)

*Needs a calculator!

If ≥8 → Severe!

BISAP Advantage: Can be done on bedside! No need for a calculator as APACHE II!

- BUN >25
- Impaired Mental Status
- SIRS
- Age >60
- Pleural Effusion

Tx w/ supportive tx — 90% resolve spontaneously!

- NPO
- IV hydration
- Analgesia
- Broad spectrum Abx (Imipenem) — ONLY if established infxn

* For gallstone Pancreatitis → do ERCP (diagnostic & therapeutic)

COMPLICATIONS

EARLY

- Shock / Renal Failure.
- Pancreatic ascites / Pleural effusion
- ARDS / Sepsis
- CoagulopathY / DIC
- Severe HYPOcalcemia (Why? due to fat saponification — fat necrosis binds to calcium)
- Splenic/mesenteric/portal v. rupture or thrombosis

LATE

- Necrosis
- Infxn
- Abscess
- Fistula
- Hmg
- Pseudocyst
- Diabetes.

Splenic vein thrombosis is a complication of BOTH acute & Chronic Pancreatitis.

Dx

Based on Ranson's Criteria.

The End.

CHRONIC PANCREATITIS

Persistent inflammation of Pancreas w IRREVERSIBLE histologic changes, recurrent abdominal pain & loss of exocrine & endocrine fxn

CAUSES

- **Alcohol** >10 yrs (60–70%) — m.c.c in developed countries
- Idiopathic (30%)
- **Obstructive**: Pancreas division / sphincter of odd; dysfxn / mass
- **Metabolic**: Malnutrition / Hyperlipidemia / hyperparathyroidism
- Familial · Trauma · Iatrogenic · Gallstones.

SUBTYPES

1. Chronic calcific Pancreatitis
2. Chronic obstructive Pancreatitis (5%)

Sx

Epigastric &/or back pain
Wt loss
Steatorrhea.

Pain Patterns in Chronic Pancreatitis

- Unrelenting pain
- Recurrent.

Sign

- Signs of EXOCRINE insufficiency: Steatorrhea (fat malabsorption from lipase insuff. — stool floats in water)
  Malnutrition
- Signs of ENDOCRINE insuff: Diabetes (glu intolerance)

Invx

LABS: Amylase / Lipase
72-hr fecal fat analysis.
Glu tolerance test

Why may amylase/Lipase be NL in a pt w Chronic Pancreatitis?

Bc ↓ of extensive Pancreatic tissue loss & "Burned-out Pancreas"

IMAGING

CT — has greatest sensitivity (80–85%) for gland enlargement / atrophy
- Calcifications
- masses / Pseudocysts

KUB — Calcification in the Pancreas
ERCP — ductal irregularities w dilation ↳ Pseudocysts & stenosis (Chain of Lakes)

Endoscopic U/S

V. sensitive & the BEST (BUT) needs very skilled doctor.

COMPLICATIONS

- A MAJOR complication is severe, Prolonged/refractory Pain.
- Insulin-dependant DM
- Steatorrhea & Malnutrition.
- Splenic vein thrombosis & Gastric varices.
- Biliary obstruction.
- Pancreatic Pseudocyst / Abscess
- Splenic art. aneurysm.
- Pancreatic CA (If >20yrs) ~2-4%

~~Tt~~

- STOP alcohol & avoid smoking / ↓ Dietary Fat & add MCT (Medium Chain Triglycerides)
- Pancreatic enzymes replacement & vit. (A, D, E, K)
- Analgesia.

Also insulin for type I DM

*If failure of medical tt: options

- Longitudinal Pancreaticojejunostomy (Pancreatic duct must be dilated)
- Distal Pancreaticojejunostomy.
- Near-total Pancreatectomy

Frey Procedure

* Longitudinal Pancreaticojejunostomy w/ core resection of Pancreatic head.

GALLSTONE PANCREATITIS

DEFINITION

Acute Pancreatitis from a gallstone in or passing through ampulla of Vater ((the exact mechanism is unknown))

Dx

Acute Pancreatitis + Cholelithiasis &/or Choledolithiasis
& NO OTHER cause of Pancreatitis (No hx of alcohol abuse)

Dx

U/S to look for gallstones
CT to looks at the Pancreas (IF sx are severe)

Tx

Conservative measure & early interval cholecystectomy.
& intraop. cholangiogram (IOC) 3-5 days (↓ inflam. resolves)

** Why EARLY interval cholecystectomy?
Bcz Pancreatitis will recur in ~ 38% of pts
Win 8 wks.

** The role of ERCP here is for: Cholangitis; Refractory Choledocholithiasis

Leah Griffith
The Grel.

PANCREATIC NECROSIS

Source : Recall 147

DEFINITION

Dead Pancreatic tissue, usually following acute Pancreatitis.

Dx:

- Abd. CT w Contrast
  (Dead Pancreatic tissue does NOT take up the IV contrast & is NOT enhanced on CT scan)
  ≡ i.e. it doesn't "light up"!

Rx

If Sterile → Medical Mgt

If suspicious of infxn → CT-guided FNA

If Toxic, hypotensive → Operative débridement.

PANCREATIC ABSCESS

DEFINITION

Infected Peripancreatic Purulent Fluid collection.

S&S:

- Fever
- Unresolving Pancreatitis
- Epigastric mass

Invx

LABS: +ve Gram Stain
+ve culture of bact.

PATHOGENS
G-ve (m.c)
E.coli / Klebsiella / Pseudomonas
G+ve
S. Aureus
Fungal Candida

IMAGING: Abd. CT w needle aspiration → send Gram's Stain/culture

Rx

Abx & Percutaneous drain Placement
or Operative debridement & Placement of drains

Yah Allah
The End.

HEMORRHAGIC PANCREATITIS

Source: Recall

Bleeding into the Parenchema & retroperitoneal structures w extensive Pancreatic NECROSIS.

SIGNS

- Abd. Pain/tenderness
- Shock /ARDS
- Cullen's Sign
- Grey Turner's Sign
- Fox's sign.

LABS

↑ Amylase / Lipase
↓ Hct
↓ Ca^{+2}

IMAGING

CT scan w IV contrast.

PANCREATIC TUMORS

EXOCRINE TUMORS

- **BENIGN:**
  - Mucinous cystadenoma (45%)
  - Serous cystadenoma (15%)
  - Intraductal Papillary mucinous adenoma (30%)
  - Mature cystic teratoma
- **BORDERLINE**
  - Mucinous cystic tumors w moderate dysplasia
  - Intraductal Papillary mucinous tumors w moderate dysplasia
  - Solid Pseudopapillary tumors
- **MALIGNANT**
  - Ductal adenocarcinoma
  - Serous/mucinous cystadenoca. (30%)
  - Intraductal mucinous Papillary tumor.

ENDOCRINE TUMORS

- Insulinoma
- Glucagonoma
- Somatostatinoma
- VIPoma
- Gastrinoma

* MAJORITY of Pancreatic tumors arise from **ductal system** & most of them are MALIGNANT!

* ONLY Exocrine tumors will be discussed here (Endocrine tumors in endocrine summary).

[redacted]

Cystadenoma

- Types: Serous / Mucinous — has Potential to be Malignant.
- Slow growing
- (+) surgical resection.

Mucinous = Malignant Potential

Solid & Papillary tumors

- Rare, mainly in ♀
- Grow to large size, have special histopathologic feature.
- (+) surgical resection.

[redacted]

PANCREATIC ADENOCARCINOMA

— MAJORITY arise from the ductal

RF

- ♂ (♂:♀ 2:1)
- Black (B:W 2:1)
- Smoking (↑ x3)
- Heavy alcohol use.
- Chronic Pancreatitis (>20 yrs) x20
- Diabete (Controversial!)
- ↑Age

PATHOLOGY
- K-ras oncogen mutation (90% of tumor

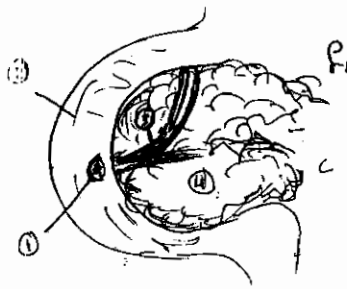
TYPES

- Duct cell Adenocarcinoma (>80%)
- Cystadenocarcinoma
- Acinar cell CA

CLASSIFICATION

- Periampullary Tumors
  - Head of Pancreas tumors 66%
  - Body & Tail tumors 33%

PERIAMPULLARY TUMORS



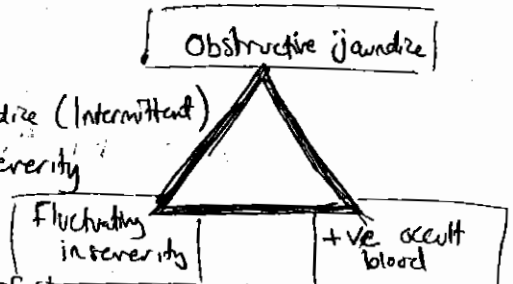
The tumors that arise AROUND the ampulla of Vater From 4 Main types:

1. Ampulla itself
2. Duodenum around ampulla
3. Terminal Part of bile duct near ampulla.
4. Head of Pancreas close to ampulla.

Why INTERMITTENT Jaundice?
due to central necrosis & then sloughing.

C/P

As a triad:
1. Obstructive Jaundice (Intermittent)
2. Fluctuating in severity
3. Stool +ve occult blood test.



Px

5-yr survival is about 5%
It's detected relatively early

tt Whipple Procedure (2)

HEAD OF PANCREAS CA

C/P
- **PAINLESS** jaundice (from obst. of CBD)
- **Wt loss**, Anorexia
- Abd. **Pain** radiating to the back (onset is after jaundice)
- Weakness
- **Pruritis** (from bile salts in skin)
- **Courvoisers Sign** (palpable, nontender, distended GB) — in 33%
- Acholic stools / Dark urine
- Diabetes.

The triad Sx
- Wt loss (90%)
- Pain (75%)
- Jaundice (70%)

* You may see Chronic Pancreatitis also due to obstruction of Pancreatic duct.

Px

50% of Pts have metz outside the liver at time of dx!

5-yr survival = 10%

BODY & TAIL PANCREATIC TUMORS

C/P

*Its very **UNLIKELY** for the Pt to present w jaundice except w liver metz.

- **Wt loss & Pain** (90%)
- **Migratory thrombophlebitis** (10%)
- Jaundice (<10%)
- N, V
- Fatigue.

* Usually Pt presents w back Pain due to invasion of neural ending!

Px

At the time of presentation, ALMOST ALL Pts have distant metz (Liver to Lung) → so Px is BAD!

INVX

LABS ↑direct Bilirubin, ↑Alk. Ph. (due to biliary obst.)
↑LFT
↑Pancreatic tumor markers (CA19-9, CEA)

IMAGING

- Abd. CT
- U/S (also endoscopic U/S w bx)
- ERCP. (to r/o Choledocholithiasis & cell brushings)

** CT & U/S guided FNA are the mvx of choice!
** For body & tail tumors — CT is quite adequate.

CA 19-9: Carbohydrate Antigen 19-9

CEA: CarcinoEmbryonic Antigen

STAGING *How? By < CT / endoscopic U/S (esp. if periampullary) / or diagnostic laparoscopy (esp. for peritoneal metz)

No nodes or metz:
- ▶ STAGE I — Tumor is limited to Pancreas; No nodes or metz
- ▶ STAGE II — Tumor extends into: bile duct / PeriPancreatic tissue / DD; No nodes or metz

▶ STAGE III — Same as stage II PLUS +ve nodes or Celiac/SMA invdv.

▶ STAGE IV (metz!)
- (A) Tumor extends to Stomach / Colon / Spleen / major vessels & Any nodal status & NO distant metz
- (B) DISTANT metz (regardless N, T)

Virchow's node & **Sister Mary Joseph nodule** are metz nodules found in BOTH Pancreatic & Gastric CA (metastatic)

ttt SURGICAL!

* If Head of Pancreas CA or Periampullary → Whipple Procedure
* If Body or tail → Distal resection (Distal "near total" Pancreatectomy)
* If UNRESECTABLE → Palliative ttt:
  - → Relieve biliary obst. → Stenting / operative bypass
  - → Relieve duodenal obst. → gastrojejunostomy (Bypass)
  - → Analgesia — Narcotics / Nerve blocks
* Palliative CTX or RTX can be used.

Whipple is the mainstay of ttt!

Rx

- 5-yr survival of unresectable CA is < 3%
  mean survival = (4 – 6) months
- Survival of pts who undergo successful resection is (12 – 19 months) in
  5-yr survival of 15–20%.

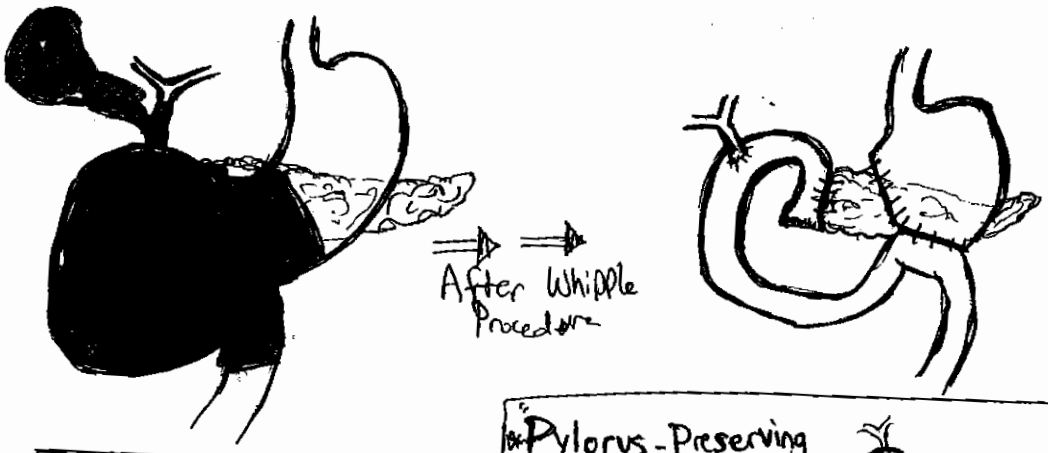
UNRESECTABLE CA

CAUSES
- Distant mets.
- Local invasion of major vessels (Portal vessels/ SMA, SMV).

Thanks Glitch
The End

WHIPPLE PROCEDURE

(: Pancreaticoduodenectomy)



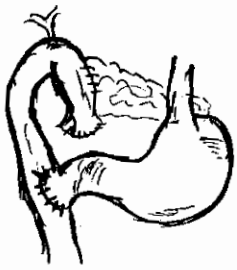
DEFINITION

- Cholecystectomy
- Truncal vagotomy
- Antrectomy
- Pancreaticoduodenectomy (Removal of the head of Pancreas + duodenum)
- Choledocho'jejunostomy (# of CBD to JJ)
- Gastrojejunostomy (# of stomach to JJ)

"Pylorus-Preserving Whipple"

NO antrectomy as conventional whipple

⇒ anastomose duodenum to jejunum



#: Anastomosis

INDICATIONS

- CA
  - CA of the head of Pancreas
  - CA of duodenum
  - CA of bile duct (distal Part)
  - CA of ampulla of Vater

  } Periampullary tumors

- Benign cases — sometimes
  - in case of Chronic Pancreatitis (refractory to medical tx)
  - Benign tumors of the head of Pancreas.

* Mortality rate ass. w Whipple is < 5%.

COMPLICATIONS

- **Anastomotic leak** (from the bile duct or Pancreatic anastomosis)
- Delayed gastric emptying (if antrectomy is performed)
- Pancreatic / biliary fistula
- Wound infxn.
- Postgastrectomy syndromes
- Sepsis
- Pancreatitis.

NOTES

* Why must the duodenum be removed if the head of Pancreas is resected?

  Bcz they share the SAME blood supply (Gastroduonal artery!)

The End.

PANCREATIC PSEUDOCYST

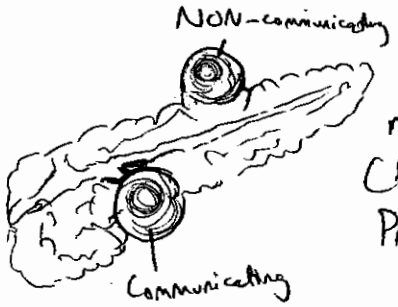
Source: Recall 157

DEFINITION

Encapsulated collection of Pancreatic fluid.

TYPES

1. Communicating
2. Non-communicating



m.c.c is CHRONIC ALCOHOLIC PANCREATITIS.

INCIDENCE

nl in 10' after alcoholic Pancreatitis.

"Pseudo" cyst | Wall is formed by inflammatory fibrosis **NOT** epithelial cell lining.

RF

Chronic Pancreatitis > Acute Pancreatitis

SYS

(Sx)
- Epigastric mass/Pain
- Emesis
- Mild fever
- Wt loss

(Signs)
- Palpable epigastric mass
  Tender epigastrium.
  Ileus.

DDX

Cystadenoma
Cystadenocarcinoma

* SUSPECT it in a pt w acute Pancreatitis fails to resolve Pain!

INVX

LABS: ↑Amylase/lipase, ↑Bilirubin, UBC (↑WBC) (IF there is obstruction)

IMAGING:
- U/S — Fluid-filled mass
- CT — Fluid-filled mass (good for showing multiple mass)
- ERCP — Radiopaque contrast material fills cyst IF communicating! So you differentiate it from non-communicating.

COMPLICATIONS

- Infxn & Fistula
- Bleeding into the cyst —tt→ Angiogram & embolization
- Pancreatic ascites
- Gastric outlet obst. (GOO)
- SBO
- Biliary obstruction.

tt If not resolved spontaneously w/in 6wks; Drainage of the cyst or Observation.

*You WAIT 6 wks for a Pseudocyst walls to "mature" or become firm enough to hold sutures & most will resolve in this period of time.

* ~50% resolves spontaneously.

• Indx of drainage:
- >5cm (b/c it has higher chance of complications & less likely to resolve spontaneously)
- Calcified cyst wall
- Thick cyst wall.

tt options

1) Percutaneous aspiration/drain
2) Operative drainage
3) Transpapillary stent via ERCP

During drainage, take **BIOPSY** of the cyst wall to RULE OUT cystic CA (Cystadenocarcinoma)

▶ Operative drainage (Pseudocyst must be communicating)
- If adherent to the stomach → Do cystogastrostomy (drain into the stomach)
- If adherent to the duodenum (DD) → Do cystoduodenostomy (drain into duodenum)
- If NOT adherent to stomach/DD → Roux-en-Y cystojejunostomy
- If it's in the tail of Pancreas → Resection of Pancreatic tail w Pseudocyst.

Dx

m.c.c of death → Massive Hmg. into the Pseudocyst.

The End

CONGENITAL ANOMALIES OF THE PANCREAS

Source: Recall Dossier.

[redacted] — the mc congenital anomaly of Pancreas.

Its failure of the 2 Pancreatic ducts to fuse; the normally small duct (Santorini)

(embryology: failure of fusion of dorsal & ventral Pancreas)

Note: If further narrowing of the minor Papilla occurs, (by an inflammation) → pt might present w obstructive form acute Pancreatitis!

So if young pt w NO hx of GBS or alcoholism THINK of Pancreatic dMsn.

[redacted]

* During rotation & migration of Pancreatic tissue, some tissue may be left around the duodenum.

— Although Congenital → 50% presents in adulthood!

C/P OBSTRUCTION!

tt

Bypass **NOT** resection

cuz resection is almost impossible & you'll end up by Pancreatic fistula: or Pancreatitis!

→ So bypass the obstructed segment by duodenojejunal bypass.

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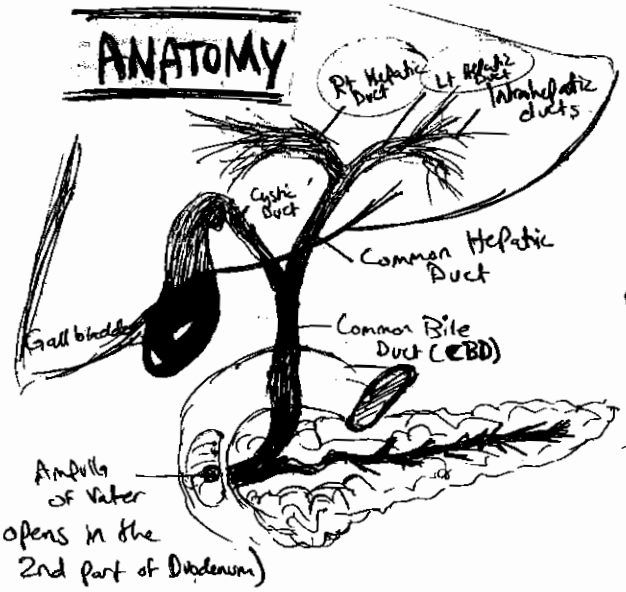
[redacted]

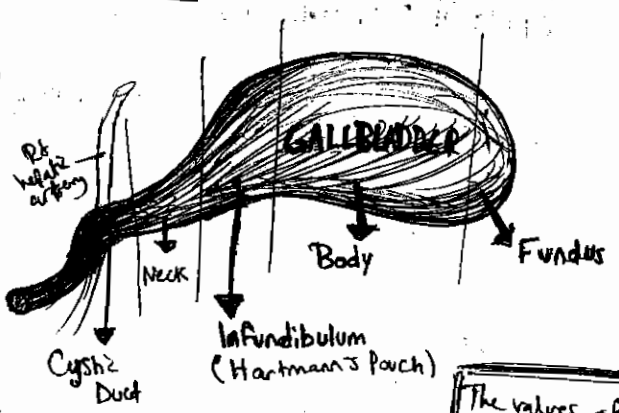
- Pancreatic tissue found usually in stomach / Intestine / duodenum

The End.
Yale Gynie

GALL BLADDER

ANATOMY



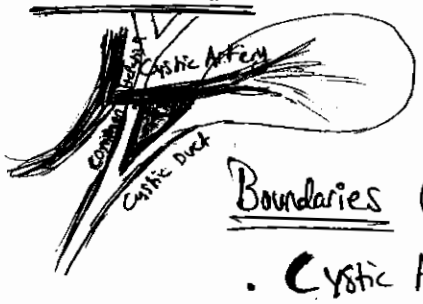


The valves of the gallbladder are called **Valves of Heister.**

Ducts of Luschka: Small ducts that drain bile directly into the gallbladder from the liver.

10% of ppl has accessory cystic artery.

CALLOT'S ▲



Boundaries (3 Cs)

- **C**ystic Artery
- **C**ommon Hepatic Duct
- **C**ystic Duct

Contents

Calot's Node

PHYSIOLOGY

FUNCTION:

- Storage of **Bile**
- Secreting bile in response to **CCK** (Cholecystokinin) + Vagal response.

Fxns of CCK

- GB emptying
- Opening of ampulla of Vater
- Slowing gastric emptying.
- Pancreatic acinar cell growth & release of exocrine products

(Fxn)

BILE is = Cholesterol + Lecithin + Bile acid + Bilirubin

- Cholesterol: Mainly Produced by liver
- Bile acid: Emulsifies fat

NOTES

- Bile is Secreted to intestine & reabsorbed mainly in **Terminal ileum**
- CCK is secreted from **duodenal mucosal cells** (2nd part of DD) as response to **fat mainly** & also to amino acids / Proteins / HCl
  CCK is ⊖ by Trypsin & Chemotrypsis (from **Pancreas**)
- **Enterohepatic Circulation**: Circulation of bile acids from liver → gut & back to the liver.

(1)

PATHOPHYSIOLOGY

- When bilirubin (Total) is **>2.5** → Pt starts to get jaundiced.
- The anatomic location where one FIRST finds evidence of jaundice is UNDER the TONGUE.

*The source of Alk. Phosphatase is bile duct epithelium so expect Alk. Phosphatase to be ↑ in bile duct obst.

S+S of obstructive jaundice

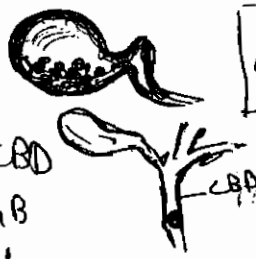
- Jaundice
- Dark urine
- Clay-colored stools (acholic stools)
- Pruritis (itching)
- Loss of appetite
- Nausea

imp.

The CAUSE of itching in obst. jaundice:
* **Bile salts** in the dermis (NOT bilirubin)

DEFINITIONS

Mucocele: sterile collection of sxns



- **Cholelithiasis**: Gallstones in GB
- **Choledocholithiasis**: Gallstones in CBD
- **Cholecystitis**: Inflammation of GB
- **Cholangitis**: Infxn of biliary tract.
- **Cholangiocarcinoma**: Adenocarcinoma of bile ducts.
- **Klatskin's Tumor**: Cholangiocarcinoma of bile duct at the junction of the Rt & Lt hepatic duct

- **Biloma**: Intraperitoneal bile fluid collection.
- **Choledochojejunostomy**: Anastomosis btw. CBD & jejunum
- **Hepaticojejunostomy**: Anastomosis of hepatic ducts or CBD to JJ
- **Biliary Colic**: Pain from gallstones (usually from stone at cystic duct)
  - The pain is located in the RUQ / epigastrium or Rt subscapular region of the back
  - Lasts min-hrs But eventually goes away
  - It often Postprandial (esp. after fatty food).
- **Hydrops GB**: Complete obstn of the cystic duct by Gallstones w/ filling of GB w/ fluid from GB mucosa.

DIAGNOSTIC STUDIES

* The initial diagnostic study of choice for evaluation of Biliary tract / GB / Cholelithiasis is **U/S** !

Other diagnostic studies:

- **ERCP** : **E**ndoscopic **R**etrograde **C**holangio**P**ancreatography.
- **PTC** : Percutaneous Transhepatic **C**holangiogram.
- **IOC** : Intra**O**p. Cholangiogram (Done laparoscopically or open to r/o Choledocholithiasis)

Index for IOC
- Jaundice
- Hyperbilirubinemia
- Gallstone Pancreatitis
- ↑ ALK. Ph.
- Choledocholithiasis
- Define of anatomy if needed.

HIDA/PRIDA scan: Radioisotope study (using technetium-agm) (Isotope concentrated in liver & secreted into bile; it will demonstrate **Cholecystitis**, **Bile Leak** or **CBD** obstruction)

* Plain X-Ray detects only 10-15% of gallstones.

BILIARY SURGERY

- Cholecystectomy : Removal of the GB (open / or laparoscop.)
- "**Lap Chole**" ≡ LAParoscopic CHOLEcystectomy.
- Kocher Incision ≡ Rt subcostal incision.
- Sphincterotomy : Cut through the sphincter of Oddi to allow passage of gallstones from CBD (most often done @ ERCP)

  AKA : Papillotomy

NOTES

* ttt of **Postop.** BILOMA after lapChole:
  1) Percutaneous drain bile collection
  2) ERCP w/ Placement of biliary stent past leak (usually cystic duct remnant leak)
* ttt of major **CBD** injury after lapChole:
  ⇒ Choledochojejunostomy.

The End.

GALL BLADDER STONES (CHOLELITHIASIS)

Source: Surgical Recall
Dossier
First Aid - Surgery

Incidence: ~10% of U.S Population will develop GBS

RISK FACTORS

- **Common RF** ("Big 4"): 4 F's
  - Female ×2
  - Fat (×3 ↑ ⚖ obesity; ↑ ⚖ fatty diet)
  - Forty
  - Fertile (multiparity) (↑ ⚖ OCPs)
- **Less common RF**
  - Advanced age
  - Infxn
  - Bile stasis
  - Cirrhosis
  - IBD
  - Chronic hemolysis (→ Pigmented GBS)
  - OCPs, Somatostatin ↓↓
  - TPN
  - Hyperlipidemia
  - Obesity
  - Rapid wt loss
  - Bypass surgery / Terminal ileum resection
  - Vagotomy
  - Native Americans

TYPES

• MIXED – 80%

- Content: Cholesterol content (50–80%)
- Various shapes & sizes
- Usually small, multiple stones of faceted surface.
- Radiolucent

• PURE CHOLESTEROL – 10%

- Content: Cholesterol (100%)
- Pale yellow
- Usually large & solitary.
- Radiolucent.

• PIGMENTED – 10%

Cholesterol content < 20%

➤➤ Black Stones

- (Causes): – Hemolysis
  – Cirrhosis
- (Content): MAINLY Ca-bilirubinate.
- Homogenous, brittle
- Small multiple stones
- Radiopaque (75%)

➤➤ Brown Stones

- (Cause): After biliary infxn (m.c is Klebsiella)
- (Content): MAINLY Ca-Palmitate
- Small, multiple stones, soft stones
- Radiolucent.

★ Hypercholesterolemia is NOT a RF for GBS formation — BUT hyperlipidemia is.

Ileal resection is one of the risk factors of GB stones
Why? due to loss of entero-hepatic circulation.

PATHOGENESIS

* **Major organic solutes in the bile:**
  - Bilirubin
  - Bile salts
  - Phospholids
  - Cholesterol

* There way to keep cholesterol from precipitation is the complexes (micelles & vesicles)

Cholesterol
- Produced primarily by the LIVER w/ little contribution from dietary sources.
- Insoluble in water, also in bile.

↑ Cholesterol
↓ Bile salts / Phospholipids
→ Cholesterol precipitation → Precipitation of crystals & stone formation

PATHOGENESIS of Cholesterol Gs

- **Supersaturation** of bile w/ cholesterol: (most imp.)
  - ↑ Sxn of cholesterol
  - ↓ bile salts & lecithin.
- Nucleation:
  - Formation of solid crystals from bile saturated w/ cholesterol.
  - **Nidus** (Ca Bilirubinate) is another mechanism of
  - Promoters of nucleation (mucus glycoproteins) are imp. RF.
- Growth
  - Individual growth of each crystal.
  - Promoters (Ca^{+2} & mucus glycoproteins) act as a framework for crystal formation.

PATHOGENESIS of Pigment Gs

BLACK stones
- ↑ level of unconjugated bilirubin → Precipitate w/ Calcium.
- **NOT** ass. w/ infected bile.
- Almost exclusively in the Gall bladder.

BROWN Stones
- Typically found in the BILE DUCT as a **Primary** stone
- 2ry to bact. infxn (having the enzyme — **Glucoronidase**)
  Causing → Hydrolysis of soluble conjugated bilirubin → unconj. ↓ Precipitates w/ Calcium
- Another bact. enzyme (**Phospholipase**) — which hydrolyzes the lecithin → Palmitate

S&S

80% of pts are asymptomatic!

If symptomatic:
- RUQ Pain that radiates to the back / epigastrium / L UQ
- Pain worse after eating (esp. fatty food)
- ± Nausea & vomiting. (why? body tries to prevent fatty food to reach duodenum)
- ↑ right sweats

*Biliary colic (NOT really "colic") — monomer. — Sx usually last for hrs.

- NO jaundice.

Boas' Sign
Referred Rt subscapular pain of biliary colic.

COMPLICATIONS

1. Acute Cholecystitis
2. Chronic Cholecystitis
3. Choledocholelithiasis (CBD stones)
4. Gallstone Pancreatitis.
5. Gallstone ileus.
6. Cholangitis
7. GB CA.
8. Oriental Cholangiohepatitis — often incidental

DDx of Biliary Pain
- Cholelithiasis
- Acute cholecystitis
- Liver d.
- PUD
- Renal colic
- GERD
- IBS
- Inf. wall MI
- Rt Lower lobe Pneumonia

DDx for Night sweats
- Biliary Pain
- MI
- TB
- CA
- Congestive hepatosplenomegaly

DDx for Pain radiating to Back
- Cholelithiasis
- Acute Cholecystitis
- Pancreatitis
- Penetrating PUD (Not Perforation)
- Ruptured aneurysm
- Disk Prolapse.

Dx

Hx, P/E

U/S (detects stones in >98% of cases)
- FINDINGS: Acoustic shadow

U/S detects
Gall bladder stones in >98% of cases
While in Choledocholithiasis it detects ONLY 33%
So it's NOT a very good study for Choledocholithiasis.

Abd. X-Ray detects only 15% of gallstones!

~~Tt~~

- If symptomatic / complicated → **Cholecystectomy** — lap chole
  - (If medical tt): Ursodeoxycholic acid (Dx stopped → Recurrence!)
- If asymptomatic → No tt EXCEPT:
  - Porcelain gallbladder (due to risk of CA)
  - Pediatric Pt (relative indx)
  - Sickle-Cell disease
  - ImmunoSuppression
  - DM
  - others: ♀ Predicting Pregnancy
  - Incidental finding intraop.
  - GB Polyp (due to risk of CA)

For pain mgt, give Pethidine NOT morphine cuz morphine contracts the sphincter of Oddi.

COMPLICATIONS of lap chole:
- CBD injury
- Rt hepatic duct/art. injury
- Cystic duct leak
- Biloma (Collection of bile.
- Bowel injury.

Smile Guys, The End. (3)

ACUTE CHOLECYSTITIS

- CALCULOUS CHOLECYSTITIS
- ACALCULOUS CHOLECYSTITIS
- EMPHYSEMATOUS CHOLECYSTITIS
- XANTHOGRANULOMATOUS CHOLYCYSTITIS

PATHOGENESIS

Obstruction of cystic duct ⟶ Inflammation of GB

⟹ so Pain is CONTINUOUS / (>3 hrs)

Obstruction of cystic duct (that leads to inflam.)
- ~95% from calculi
- ~5% from acalculous obst

RF

Gallstones

S&S

- More severe & continuous Pain than cholelithiasis
  UNRELENTING RUQ Pain or tenderness
- **FEVER**
- N, V, Anorexia.
- Murphy's Sign **+ve** (Inspiration arrest during deep palpation of RUQ)
- Painful Palpable GB in 33%
- Mild jaundice (if severe — THINK of CBD stone!)
- Rt subscapular Pain / Epigastric discomfort (referred Pain)

INVX

LABS: CBC — ↑WBC (BUT could be NL)
↑Alk. phosphatase, ↑LFT, ↑Total bilirubin
Slight ↑ amylase.

IMAGING: **U/S** (The diagnostic test of choice)
HIDA scan (The most accurate)
CT scan (less sensitive)

FINDINGS on U/S
- Thickened GB wall >3mm
- Pericholecystic fluid
- Distended GB
- Gall stone present / Cystic duct stone
- Sonographic murphey's sign

(1)

~~Mx~~

- ADMIT the Pt
- IV Fluids
- IV Abx (Piperacillin / Tazobactam)

The gall bladder specimen is opened in the operating room. Why?
Looking for GB CA & anatomy.

▲ If mild → Early Cholecystectomy (within 24-48 hrs)

▲ If moderate → Early vs. delayed (after 6 w.) Cholecystectomy

* Recent studies showed that early is BETTER regardless the degree.

▲ If severe → Percutaneous Cholecystostomy.

(for pt have severe medical illnesses / or very elderly / or can't tolerate GA

COMPLICATIONS

- Empyema
- Abscess formation
- Perforation
- Gangrene
- Cholecystenteric fistula formation
- Choledocholithiasis
- Gallstone ileus.
- Mirrizzi Syndrome

According to **Tokyo Guidlines**:

MILD (Grade I): mild inflammation
No organ dysfun.

MODERATE (Grade 2): Leukocytosis
Palpable tender mass
Duration > 72 hr
Marked inflam.

SEVERE (Grade 3): MOF
Hypotension
Resp. failure
Renal failure
Mental status Δ

Mirizzi's Synd.: The presence of CBD stone, cholangitis or obstruction of CBD caused by EXTERNAL compression from a stone impacted in an inflamed Hartman's Pouch.

NOTES

* Difference btw. acute cholecystitis & biliary colic:-
  - Biliary colic has temporary Pain, while acute cholecystitis has Pain that does NOT resolve
  - Cholecystitis has also ↑WBC, fever & signs of acute inflam. on U/S.

ACALCULOUS CHOLECYSTITIS → mortality rate is HIGH! 30%

↳ Think of it if deterioration occurs in ICU pt.

DEFINITION

Acute cholecystitis **wout** evidence of stones.

PATHOGENESIS

- Itrs believed to result from **sludge** & GB disease & biliary stasis (2ry to absence of CCK stimulation → ↓ can of GB)

Pathophysiology (Theories)
- Sludge
- Thickening of mucosa
- Ischemia (ds in ICU Pt)

RF

- Prolonged fasting
- TPN
- Trauma
- Multiple transfusions
- Dehydration
- Prolonged Postop. setting or ICU Pts (critically ill) (esp. w/ hx of hypotension)
- Sepsis or multiple organ dysfxn.
- Burns

INVX

- **LABS**: Leukocytosis (↑WBC) / Abnormal LFT / Pancreatic.
- **IMAGING**: **U/S**
  HIDA scan (FINDING: Non filling of GB) — most accurate.
  CT (has the same sensitivity as U/S!)

~~Tx~~

If STABLE → Cholecystectomy

If UNSTABLE → Cholecystostomy tube — placed percutaneously to decompress the GB **then** do Cholecystectomy.

Limitations to U/S
- Overlying bowel gas
- Concomitant abd. wounds / dressing.

XANTHOGRANULOMATOUS CHOLYCYSTITIS

RARE inflammatory di. of GB, a foreign body giant cell reaction leading to → formation of Xanthoma cells.

EMPHYSEMATOUS CHOLCYSTITIS

- Gas forming bacteria (E. coli)
- usually in DM & ♂ & elderly → High mortality & morbidity rate ∴
- often results in perforation of GB

● NOTE

☺ If Gas present in biliary tree → THINK of fistula

BUT IF in GB wall → THINK of emphysematous!

Remember!
- In acute cholecystitis → PALPABLE **PAINFUL** GB
- In GB cancer → PALPABLE **PAINLESS** GB.

Ends Gloriously
The End.

CHOLEDOCAL CYST

DEFINITION

- Its congenital dilation of biliary tree

* Usually in CBD

* ♀ x3

60% of cases present before the age of (10) yrs

C/P

- Jaundice
- RUQ Pain
- Palpable mass

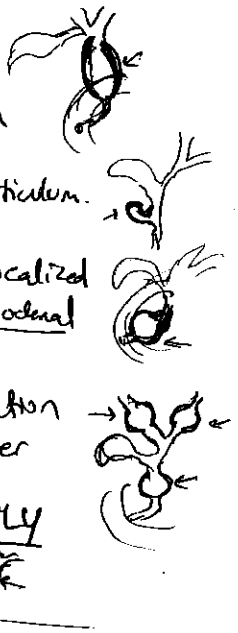
Dx

- Hx & P/E
- U/S or CT

COMPLICATIONS

- Choledocholithiasis
- **Cholangitis.**
- Portal HTN
- **Cholangiocarcinoma** – 30%
  - ↳ usually in 4th decade of life.

TYPES



- I — Fusiform/diffuse dilatation
- II — Isolated sacular diverticulum.
- III — Choledochocele/cyst (localized dilation w/in intraduodenal Part of CBD)
- IV — Multiple cystic dilatation inside AND outside liver
- V — Single/multiple lesions ONLY INTRAHepatic.
  AKA: Caroli's Disease

* 75% of cases are type I !

Tt

According to the type

I, IV → Hepatojejunostomy

~~II~~ II → cyst excision.

III → Cyst unroofing + sphincteroplasty

V → Hemihepatectomy.

Complete excision of the cyst is important due to risk of Cholangiocarcinoma!

The End

Enah Ghanem

CHOLEDOCHOLITHIASIS

DEFINITION

Stones in the CBD

(It's mostly GB stones pass through cystic duct into CBD — usually brown (pigmented))

INCIDENCE

found in 5-15% of acute calculous cholecystitis
1-2% of acalculous cholecystitis.

CAUSES of CBD OBSTRUCTION: (SINGE")

- **S**tricture
  - **I**atrogenic (ERCP/PTC/biliary stent placement)
- **N**eoplasm
- **G**allstones (choledocholithiasis)
- **E**xtrinsic compression (ex. Pancreatic Pseudocyst/Pancreatitis)

SxS

- Epigastric / RUQ Pain
- Jaundice (if bilirubin >2.5)
- Pruritis
- Icterus.

Dx

- LABS: ↑Alk. Ph., ↑LFT & total bilirubin & direct B.
- **ERCP** (The gold standard for dx of CBD stones) — Also therapeutic!
- PTC (Percutaneous Transhepatic Cholangiography)

Mgt

- **ERCP**: Involves endoscopic sphincterotomy w/ retrieval of the CBD stones w/ a basket — 85-90% successful! :)
- If ERCP fails → CBD is opened **SURGICALLY** & stones removed. (**T-tube** is placed, so bile can drain externally)
  - removed 2-3 wks later on an outpt basis.
- Other ~~tt~~ option is Lapchole & Intraop. Cholangiogram (IOC)
  - & blind Passage of balloon catheter / Stone basket.

Yash Gohil
The End.

CHOLANGITIS

ACUTE (ASCENDING) CHOLANGITIS

- Biliary infxn of the biliary tract from obstruction (either Partial/or Complete) — Potentially life-threatening!

CAUSES (Any cause of obstruction)

"SINGE"
- **S**tricture (usually Post-op.)
- **I**atrogenic (ERCP/PTC)/Biliary Stent
- **N**eoplasm (usually ampullary Ca)
- **G**allstones (Choledocholithiasis) — m.c
- **E**xtrinsic compression (Pancreatic pseudocyst/Pancreatitis)

SIS

- **Charcot's triad** (50-70%) (In ascending Cholangitis (Non-suppurative)):
  - Fever/Chills
  - RUQ Pain
  - Jaundice
- **Reynold's Pentad** (In suppurative Cholangitis (more in elderly)): Charcot's triad PLUS
  - Altered mental status
  - Shock

Suppurative Cholangitis is Severe infxn w sepsis (Pus under Pressure)

PATHOGENS

- Usually BACTERIAL!
  - E coli
  - Klebsiella
  - Pseudomonas
  - Enterobacter (Proteus/Serratia)
  - } m.c G-ve
  - Enterococci — m.c G+ve.
- LESS common:
  - Anaerobes (B. Fragilis)
  - Fungi (Candida) — least common.

INVX

- LABS: ↑WBC ↑Alk. Phosphatase, Abnormal LFT, ↑bilirubin
- U/S — Should be the initial study
  - FINDINGS: Dilation of CBD & intrahepatic ducts along w/ gallstones
    Thickened, edematous gallbladder wall.
- ERCP / PTC — Provides "Definitive" dx — can also be therapeutic
  (Percut. Transhepatic Cholangiogram)
- Bile cultures

[redacted]

If nonsuppurative — IV Fluids & IV abx
(w/ definitive ~~tx~~ later) → Lapchole ± ERCP

If suppurative — IV Fluids & IV abx
GB Decompression by — ERCP w/ sphincterotomy
or PTC w/ catheter drainage
or Laparotomy w/ T-tube placement

PRIMARY

SCLEROSING CHOLANGITIS (PSC)

- Autoimmune Progressive fibrous obliteration of the bile duct
  (Multiple inflammatory fibrous thickening of bile duct walls resulting in biliary strictures)

* Natural hx: Progressive obstruction Possibly leading to cirrhosis & Liver failure!
  10% will develop Cholangiocarcinoma.

CAUSE: idiopathic BUT Probably autoimmune.

MAJOR RF: IBD! (5%) 60% of UC pts develop PSC

- In young & middle aged ♂

* Liver Biopsy — shows Periductal concentric fibrosis around the macroscopic bile duct.

* The hepatic duct bifurcation is most severely involved.

CP

- Usually asx for yrs! (up to 15 yrs)

If symptomatic;

RUQ pain / Jaundice / Hepatosplenomegaly
Itching (Pruritis)
Dark urine
Clay-colored stools
Malaise
Wt loss

COMPLICATIONS

- Cirrhosis
- Obstructive jaundice
- Cholangiocarcinoma (10%)
- Cholangitis.

Dx

LABS: ↑Alk. Phosphatase, +ve PANCA (80%),

ERCP → shows "beads on a string" — characteristic
or PTC (Diffuse irregular narrowing of the entire biliary tree + annular stricture)

~~Mx~~

- Endoscopic balloon dilations (for strictures) & Stent after dilation
- **LIVER TRANSPLANT** (the definitive!) esp. if primarily intrahepatic disease / cirrhosis.

* If primarily extrahepatic ducts are involved → do hepatoenetric Anastomosis & resection of extrahepatic ducts due to risk of Cholangiocarcinoma.

Notes

Close F/U is Imp. due to risk of CA

↑Ca 19-9 Suggests Carcinoma

Px

10-12 yrs

ORIENTAL CHOLANGIOHEPATITIS

Lt hepatic duct is more affected than the Rt.

AKA: Recurrent Pyogenic Cholangitis

Its infestation w PARASITES (Ascaris) that causes bacterial overgrowth / stasis / & brown stone formation
- Its one of the complications of cholithiasis.
- More common in far east

C/P: Multiple intrahepatic & extrahepatic biliary ductal stones / strictures & hepatic bact. infxns.

Mgt: Palliation of biliary stricture (by stents) & biliary drainage.

Gabe Ghattas
The Gut

GALL STONE ILEUS

DEFINITION

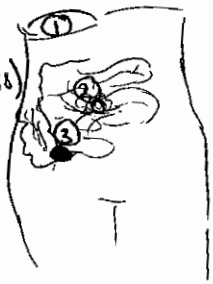
Small bowel obstruction from a large gallstone (>2.5 cm) that has eroded through the gall bladder into the duodenum/small bowel.

CLASSIC SITE OF OBSTRUCTION → Just Proximal to ileocecal valve

(But may cause obstruction in duodenum & sigmoid colon)

CLASSIC FINDINGS

① Air in the hepatic ducts
② Small bowel obstruction (SBO) w air fluid levels.
③ Gallstone in ileocecal valve.



S&S

- Sx of SBO: distention / vomiting / hypovolemia / RUQ Pain.

RF

♀ >70 yrs

Gallstone ileus represents < 1% of cases of small bowel obstruction.

DIAGNOSTIC TESTS OF CHOICE

- Abdominal X-Ray: • Reveals radiopaque gall stone in the bowel
  • 40% of Pts show AIR in the biliary syst.
  • Small bowel distention.
  • Air-fluid levels 2ry to ileus.
- UGI
- Abdominal CT: reveals air in biliary tract
  SBO
  ± gallstone in lumen

182

~~tt~~

SURGICAL:

enterotomy ω removal of stone

± interval cholecystectomy (interval = delayed)

The End.

BILIARY SYSTEM TUMORS

GALLBLADDER CA

Malignant neoplasm arising in the gall bladder

Vast majority are **Adenocarcinoma (90%)**

RF
- Gallstones !
- Porcelain GB
- Cholecystenteric Fistula

SITE : m.c site is **Fundus** (60%)

♀ : ♂ 4 : 1

INCIDENCE
~1% of all GB specimens.
Rare!

Porcelain GB

CALCIFIED GB seen on abd. X-Ray — results from Chronic Cholelithiasis/ Cholecystitis ⇒ calcified scar tissue in GB wall.
⇒ So it's an indx for Cholecystectomy.

~50% of pts ⓦ Porcelain GB will have GB CA.

Sx most pts are asx until late

Biliary colic
Wt loss / Anorexia
— might present as acute cholecystitis.

Signs
- Jaundice (Why? From invasion of the common duct or compression by involved Pericholedochal L.N)
- RUQ mass
- Palpable GB (advanced d.)

Courvoisier's Sign

A Palpable, nontender GB often ass. ⓦ CA in the head of Pancreas or GB

DX
- U/S
- Abdominal CT
- ERCP

Route of spread : Contiguous spread to the liver is most common.

Px 5% 5-yr survival (mostly unresectable at dx! ☹)
But T_1 (5-yr survival is 95%)

Tt Depends on the extent of tumor involvement:

Tumor confined to GB Mucosa → Cholecystectomy
Muscularis/Serosa → Radical cholecystectomy*

"The MAIN complication of lapchole for gall bladder CA is Trocar site tumor implants (so if known preop. perform open cholecystectomy)

*Cholecystectomy + wedge resection of overlying liver
+ L.N dissection
± CTX / RTX

BILE DUCT TUMORS
- Benign
- Malignant (Cholangiocarcinoma)

BENIGN
- Adenomas (m.c)
- Rare
- Arise from ductal glandular epithelium.
- Polyploid.
- < 2 cm
- Most common site: - Ampulla (1st m.c site)
  - CBD (2nd m.c site)

C/P: Intermittent obstructive jaundice & RUQ Pain (so confused w Choledocholithiasis)

Tt Complete resection of the tumor w a margin
- High recurrence rate after simple curretage of polyp!

MALIGNANT - [CHOLANGIOCARCINOMA]

It's a malignancy of the extrahepatic or intrahepatic ducts.
- Primary bile duct CA

- **HISTOLOGY** Almost all are adenocarcinoma.
- **Avg. Age of dx** ~65 yrs ♀ = ♂
- **MOST COMMON SITE**: Proximal bile duct (At the junction of Lt & Rt hepatic duct)

Klatskin Tumor
Cholangiocarcinoma arises at the junction of the Rt & Lt hepatic ducts.

• RF

- Choledochal cysts
- Primary Sclerosing Cholangitis (PSC)
- Ulcerative Colitis (UC)
- Radiation exposure
- Toxin exposure
- Parasitic infxn

TYPES

- Intrahepatic 20%
- Upper extrahepatic 40% (Klatskin)
- Lower extrahepatic 40%

• Dx

- U/S — shows bile duct dilatation.
- CT scan
- ERCP/PTC ± bx/brushing for cytology
- MRCP

• Tx Depends on site:

If proximal → Resection ± Roux-en-Y hepaticojejunostomy ± Unilateral hepatic lobectomy.

If distal → Whipple Procedure.

• Px

Depends on: ① Location & extent
② Portal vein invasion
③ Hepatic lobar atrophy

5-yr survival rate is 15-20%

LIVER

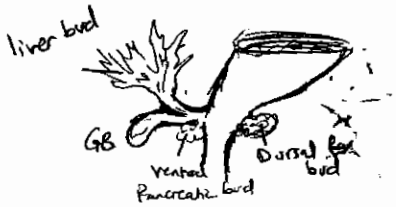
Sources: Reeall Dossier

EMBRYOLOGY

Starts as hepatic bud / hepatic diverticulum just proximal to the ampulla of Vater (at the same area, arises the pancreatic duct)

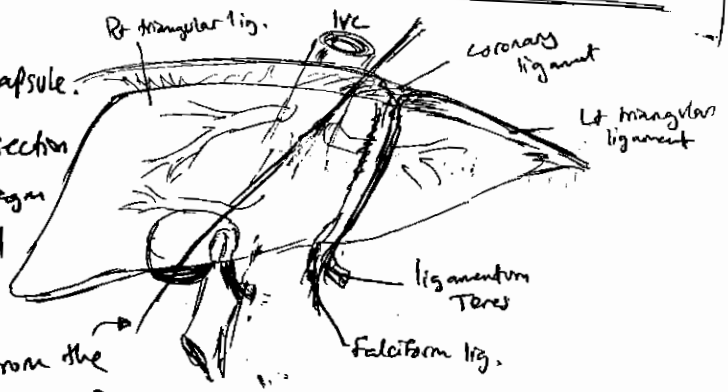
- So the hepatic duct arises at the ventral aspect & rotates 90° clockwise (that's why the liver is on the Rt side)

The diverticulum divides into
- Cranial Part → gives rise to → CBD / Rt & Lt hepatic duct / & the liver
- Caudal Part (smaller) → cystic duct / GB



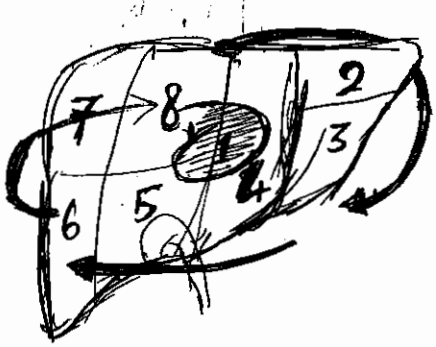
- The liver grows very rapidly, esp. during the 5th – 10th wks of gestation bcz the blood is produced from the liver & spleen in this period.
- The liver is relatively very huge, leaving no space in the peritoneal cavity → this will push the small bowel outside
  ⇒ So called "Herniation".

ANATOMY



- **Glisson's capsule** is the liver capsule.
- **"Bare" area of the liver**: Post. section of the liver against the diaphragm that is "bare" (wout peritoneal coverage.
- **Cantlie's Line**: Line drawn from the GB to a point just the Lt of the IVC (which transects the liver into Lt & Rt Lobes)
- **Falciform Lig.**: ligament goes from the anterior abd. wall to the liver (It contains Ligamentum teres — obliterated umbilical v.)
- **Coronary lig.**: Peritneal reflection on top of the liver than crowns ("coronary") the liver & attaches to the diaphragm.
- **Triangular lig.**: Rt & Lt lateral extents of the coronary lig. (which form triangles)

The Segments of the LIVER:

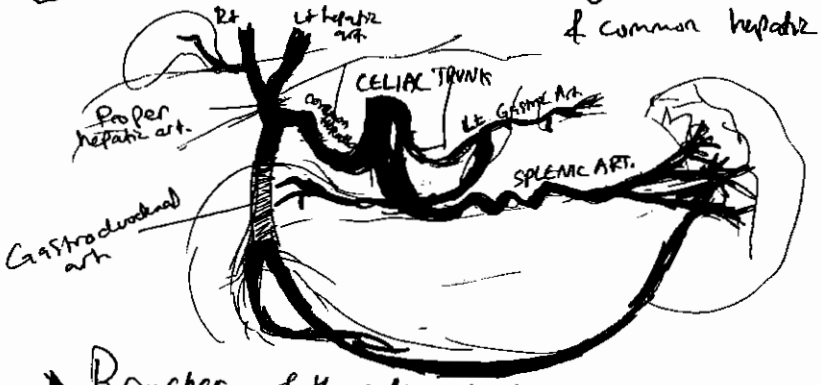


"Clockwise" starting from segment 1

"French system"

BLOOD SUPPLY

- Celiac trunk from aorta — giving off the Lt gastric / splenic / & common hepatic art.



- Branches of the celiac trunk:
  - Lt Gastric Art.
  - Splenic art.
  - Common hepatic art

VENOUS SUPPLY

Portal v. (formed from splenic v + SMV)

Hepatic venous drainage ⇒ via hepatic veins which drain into IVC
(3 veins: Lt, middle & Rt)

NOTES

- Sources provide O_2 to the liver are — Portal v. blood – 50%; Hepatic art. blood – 50%
- Sources in which the liver recieve blood — Portal syst. – 75%; Hepatic art. syst. 25%
- The max. amount of liver that can be resected while retaining adequate liver fan → >80% !! (If given adequate recovery time it will regenerate) (2)

Child's Classification (Child-Pugh)

Classification that estimates hepatic reserve in pts w hepatic failure & mortality.

- **LAB**: Bilirubin, Albumin
- **CLINICAL**: Encephalopathy, Ascites, PT

Mnemonic:
"A BEAP"
Ascites
Bilirubin
Encephalopathy
Albumin
PT

	Ascites	Bili	Enceph.	Albumin	PT
A	None	<2	None	>3.5	<1.7
B	mild	2-3	minimal	2.8-3.5	1.7-2.2
C	marked	>3	severe	<2.8	>2.2

If 5-6 → A
7-9 → B
10-15 → C

overall mortality
10%
30%
75%

MELD score

Model for End-stage Liver Disease
- used more than Child

measurements:
INR, T. Bilirubin, Serum Cr

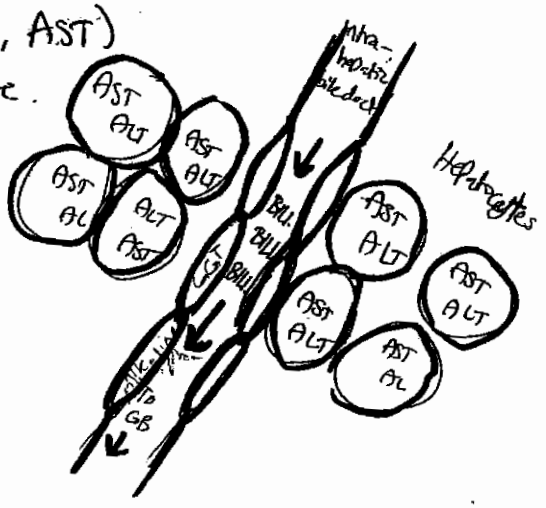
*Mortality in cirrhotic pts for
- NONemergent nontransplant surgery is:
  ↑ in mortality by 1% per 1 Point in MELD score until 20
  then 2% for each 1 Point
- EMERGENT nontransplant surgery:
  14% ↑ in mortality per 1-Point of MELD.

Thanks

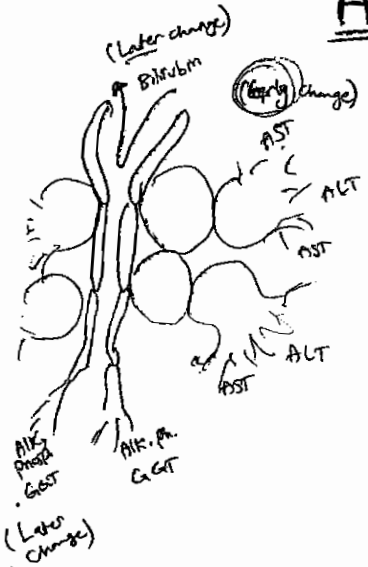
Liver Tests

Liver Fxn Tests (LFT)

Full work up PLUS
- Transaminases (ALT, AST)
- Alkaline phosphatase.
- PT/ INR
- Bilirubin
- Albumin
- GGT
- CBC



AST, ALT (Transaminases)



- They don't really ~~reflect~~ liver fxn BUT they're reflection of hepatocellular injury.
- ALT: more Liver-specific than AST
- AST also ↑ in MI, skeletal m. damage & hemolysis.

	AST	ALT	AST : ALT	MCV
Alcoholic liver d.	↑↑	↑	AST > ALT 3:1 (>2)	↑↑
Viral hepatitis	↑	↑↑	AST < ALT <1	↔
NAFLD	↑	↑↑	AST < ALT	↑ or ↔

Albumin

↓ in liver disease.

Alkaline Phosphatase

* Found in LIVER, BONE, GI & placenta.
* ↑ in cholestasis
* Varies w age & gender (higher in ♂ higher in children) x3 Lo correlates w bone growth.
↑ in pregnancy (produced by placenta) x2

GGT (Gamma Glutamyl Transpeptidase)

- Rises in parallel w Alk. Phosphatase from the liver.
- Should be checked in cases of ↑ alk. phosph. w NL bilirubin & transaminases.

If BOTH (Alk. phosph. & GGT) are ↑ → NEXT step, Abd. U/S to look for dilated bile ducts.

DDx for ↑GGT ALONE

Drugs: Barbiturates / Carbamazine / Phenytoin
Ethanol
Steroids
INH / Rifampicin.

PT

Detects the severity of hepatocellular injury
(the most sensitive test to test the severity)
Why? w/z it's ~~doesn't~~ not affected by mod. lower d. only severe are.

- PT & serum ammonia reflects metabolic func of liver cells.

Bilirubin

Direct (conjugated)
Indirect (Unconjugated) = (NO) bilirubinuria

Reflects clearance & excretion fxn

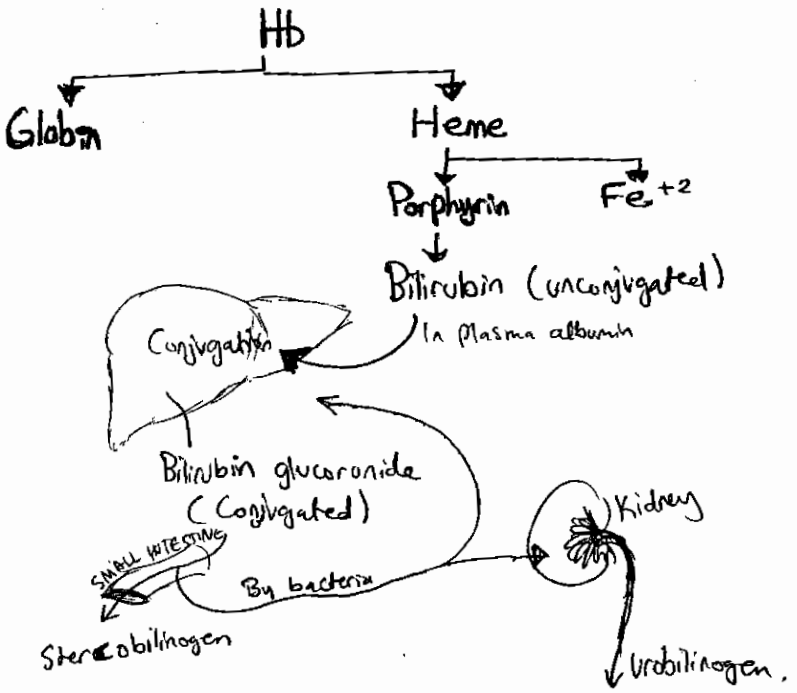
Bilirubinuria is an index of cholestasis

Thank God

JAUNDICE

Source: Recall Dossier 193

BILIRUBIN METABOLISM



DDx of INTERMITTENT Jaundice:

- Choledocholithiasis
- Ampullary tumors

Appearance of jaundice depends on:

- Type of bili. elevated
- Duration

CAUSES of ↑ Bilirubin

- Overproduction by reticuloendothelial system.
- Failure of hepatocyte uptake.
- Failure to conjugate or excrete.
- Obstruction of biliary excretion into intestine.

JAUNDICE: is yellowish discoloration of skin & mucus membranes.

* Detected clinically when bilirubin is > 2.5 - 3 g/dL

* Hyperbilirubenemia
  - Conjugated (Post hepatic) → Obstruction (surgical)
  - Unconjugated (Pre-hepatic & hepatic)
    - ↑ RBC breakdown
    - ↓ hepatic bilirubin conjugation

PREHEPATIC

Due to:
- excessive Production of bilirubin
- Ability of liver to conjugate is overcome
- ↑ Plasma unconjugated bilirubin.

↓ Conjugated bilirubin
↑ Unconjugated (indirect) bilirubin.

Labs indicating hemolysis:
- ↑ LDH.
- ↓ serum haptoglobin
- evidence of hemolysis on blood film.

DDx
- **Hemolysis** / Hematoma resorption / multiple blood tx
- Gilbert syndrome
- Crigler-Najjar Synd.

HEPATIC

- Defect in:
  - uptake
  - conjugation
  - Sxn

— It reflects liver dysfxn. (↑ Alk. phosph., ↑↑↑ AST/ALT)

DDx
- Viral hepatitis
- Medications (Erythromycin / INH / Phenytoin / Valproate / OCP)
- Alcohol abuse
- Cirrhosis.

Gilbert Syndrome

- It's the m.c
- Benign condition affecting upto 7% of the population.
- Its an inborn error in liver bilirubin uptake & **G**lucuronyl transferase resulting in hyperbilirubinemia.
  (THINK: **G**ilbert's = **G**lucuronyl)

*Affected ppl may have jaundice after stress / infxn.

⇒ So the cause of Unconjugated hyperbilirubinemia in Gilbert is both ↑ RBC production & ↓ hepatic conjugation.

POST-HEPATIC

↑ conjugated bilirubin
↑↑ Alk. Phosph. ↑ GGT
± ↑ AST, ALT

CAUSES : 2ry to biliary OBSTRUCTION (Post-hepatic / surgical jaundice)
. Hepatic jaundice (AKA: Non-obstructive/medical jaundice)

C/P : . Jaundice
• Pale-colored stool (due to absence of fecal bilirubin)
• Dark urine (↑ conjugated bilirubin)
• Itching.

Diagnostic test of choice is **U/S** !

CHOLESTATIC SYNDROME

Characteristics:
- Conjugated hyperbil (Dark urine / Pale stool / Pruritis)
- Chronic malabsorption of fat-soluble vits

● **DDx of PROXIMAL** bile duct obstruction

- Gallbladder stones
- GB cancer
- Hepatoma
- Cholangiocarcinoma
- Benign bile duct tumor
- PSC
- Parasites
- Metastatic tumor
- Lymphadenopathy

● **DDx of DISTAL** bile duct obstruction

- **Choledocholithiasis**
- Benign bile duct tumor
- Ampullary CA
- Pancreatic CA / Pancreatitis.
- Pseudocyst
- Lymphadenopathy / Lymphoma
- Postop. stricture
- Parasites

APPROACH

- By hx & PE
- Invx

Direct vs. Indirect

Indirect

Direct

↓

Hepatic vs. Posthepatic

ABSCESSES OF THE LIVER

Source Recall 197

LIVER ABSCESS: is a collection of Pus in the liver Parenchema

TYPES

- PYOGENIC (Bacterial)
- PARASITC (Amebic)
- FUNGAL

SITE

m.c is RE Lobe.

SOURCES

- **Direct** spread from biliary tract infxn.
- **Portal** spread from GI infxn (ex. Appendicitis / Diverticulitis)
- **Systemic** source (Bacteremia)
- **Liver Trauma** (ex. Liver gunshot wound)
- **Cryptogenic!** (Unknown source)

The 2 m.c types are — **Bact.** (m.c in USA)
— Amebic (m.c worldwide)

BACTERIAL LIVER ABSCESS

The m.c Pathogens: G-ve bact. — E.coli
— Klebsiella
— & Proteus.

CAUSES / SOURCES

- Cholangitis
- Diverticulitis
- Liver CA / metz

S&S

- Fever, Chills
- RUQ
- ↑WBC
- ↑LFT
- Jaundice
- Sepsis
- wt loss.

Indx of operative drainage :

- Multiple/loculated abscesses.
- multiple Percut. attempts have FAILED!

tt

- IV abx (Triple Abx w/ metronidazole)
- Percutaneous drainage w/ CT or U/S guidance

AMEBIC LIVER ABSCESS

PATHOGEN: Entamoeba Histolytica
(Typically reaches liver via Portal v. from intestinal amebiasis)

SPREAD: feco-oral transmission

RF:

- Pts from countries South of America – Mexican border
- Institutionalized Pts.
- Homosexual men
- Alcoholic Pts.

Note
Chills are much LESS common w/ amebic abscesses than Pyogenic!

S&S

- RUQ Pain
- Fever
- Hepatomegaly
- Diarrhea

SITE
m.c — Rt lobe

Dx

- Labs, U/S, CT

(LABS: Indirect hemagglutination titers for Entamoeba abx) ↑ in >95% of cases
- ↑LFT

~~tt~~

Metronidazole

If refractory to medical tt → Percut. drainage.

* Indx for percut. surgical drainage :

① Refractory to metronidazole
② Bacterial co-infxn
③ Peritoneal rupture.

* Possible complications of large Lt lobe liver amebic abscess :

▶ Erosion into the Pericardial sac (Potentially FATAL !!)

HYDATID LIVER CYST

– Dissussed later.

The God

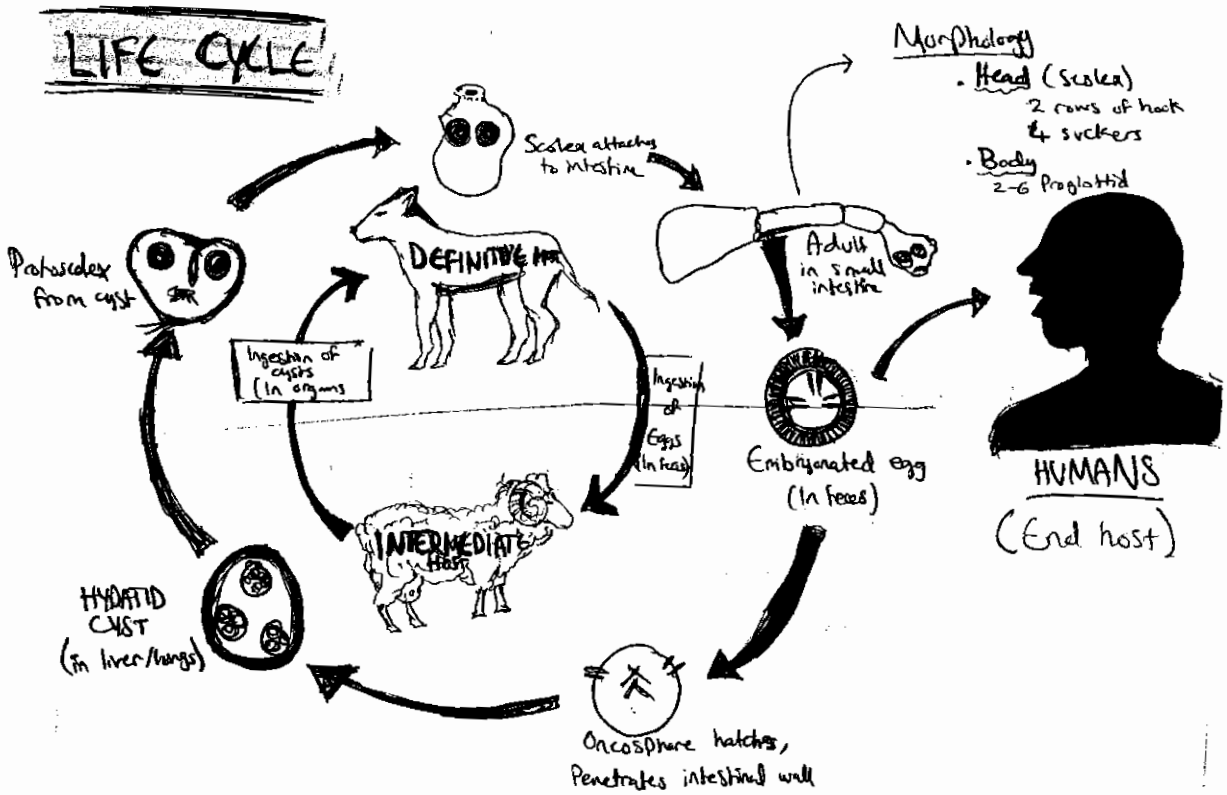
HYDATID DISEASE

Source: Dossen Recall 20'

AKA: Echinococcosis / Echinococcal Disease

- It's a parasitic disease of tapeworms — Echinococcus.
- It affects BOTH humans & other mammals e.g. sheep / dogs / rodents & horses.
- It affects humans in 2 FORMS (depending on the larval stage)
  - CYSTIC ECHINOCCOSIS (most common) — By Echinococcus Granulosus
  - ALVEOLAR ECHINOCCOSIS — By Echinococcus Multilocularis

LIFE CYCLE



* The MAJOR "intermediate host" is the sheep (also pigs / horses / camels)
* The MAJOR "definitive host" is the dog (also foxes / wolves, —)

— Infxn of the intermediate host occurs after ingestion of food contaminated w/ eggs containing embryos (oncospheres) Passed from feces of the definitive host.

— Humans are infected in 2 ways:-
1. Direct contact
2. Eating products contaminated by the feces of the definitive host.

DISTRIBUTION

- Endemic areas are: Mediterranean countries / Middle East / Asia Turkey / South america / New zealand Africa
- UNCOMMON in USA & most of central Europe

PATHOLOGY

- The eggs (oncospheres) Penetrates the wall of the intestine of the intermediate host & via the blood stream they first reach the LIVER!

  ⇒ The LIVER is affected in 60% , LUNGS in 30%

- Rt lobe of the liver is mostly affected in 80%
- In 1/3 of the cases the cysts are multiple.
- Single organ involvement in 90% of cases

HISTOLOGY

- The outer adventitial layer coming from the HOST is called Ectocyst or Pericyst
- The 2 inner layers coming from PARASITES — the most inner (germinal layer)] Endocyst
  — the outer (Laminated layer)]
  - The germinal layer Secretes the laminated layer which is mucopolysaccharide – Protein – lipid complex.
- The cavity of the cyst contains hydatid fluid – which is clear & similar to interstitial fluid.

NOTES

- The time required to become mature varies from 10 – 20 months
- Daughter cysts (degenerated or 2ry cyst) which have fragments of germinal layer — It may develop with the 1ry cyst or separately.

- **Calcification** of the cyst occurs usually after 5–10 yrs !! — commonly in liver cyst
- **COMPLETE CALCIFICATION** indicates **NONVIABLE** cyst
- Usually Protoscolics produced after 1 yr following the infxn.
- MOST of the cysts remain **UNCOMPLICATED** ! ☺

C/P

- RUQ Pain (m.c sx)
- Liver enlargement / Palpable mass (1–5 cm/yr ↑ in size)
- Jaundice & Pressure sx
- Suppuration

Sx usually when size is >10 cm

- Rupture may occur, into:
  - Biliary tree
  - Thorax
  - Peritoneum
  - Vascular structure
  - GIT

RF

- Travel
- Exposure to dogs/sheep/ & cattle (carriers)

Dx

- **Blood Tests:**
  - most cases have limited eosinophilia <15% or absent.
  - If biliary communication — ↑ LFT
- **Serology**
  - For detection of **Anti-echinococcus antibodies**
    - ✱ Antibody detection is more sensitive than Serum antigens (IHA/ELISA)
    - ✱ More informative in E. multilocularis than E. Granulosis.
    - ✱ PCR technology.

AIKA — Indirect hemagglutination test

- **Imaging**
  - Plain X-Ray
  - **US**
    - Simple vs. Parasitic cyst
    - Abscess / Neoplastic vs. Parasitic (when membranes inside)
    - "Hydatid sand sign" is diagnostic in most cases.
    - Active vs. Inactive "water lily sign"
    - Eggshell app. in CALCIFIED cyst.

Classification upon U/S findings (Gharbi Classification)

TYPE I: Cyst w/ Pure fluid collection.
TYPE II: Cysts w/ variable morphology, detached membrane/ split wall.
TYPE III: Multiple septa / daughter cysts
TYPE IV: High internal heterogenous echos.
TYPE V: Cyst w/ CALCIFIED, reflecting thick wall.

WHO Classification for:

- Final State of the Parasite: Active / Inactive / Transitional
- Size: Small (< 5 cm) / Medium (5-10 cm) / Large (>10 cm)

. CT

- Sensitivity reaches 100%!
- #, size, location & Presence of extrahepatic lesions
- More sensitive in detection of gas & minimal Calcifications.
- BUT U/S is more informative regarding wall cyst changes.

. MRI
. MRCP / ERCP

Tt options:

- **CTX**
- Percutaneous drainage
- Surgery (Open vs. Laparoscopic)
  - Radical
  - Conservative
- Open vs. Laparoscopic

CTX (used ALONE is NOT successful, should be combined w/ other Tt)

Dose
10-15 mg/Kg/d in 2 divided doses.

Failure rate
20-30%.

* Albendazole (ABZ) & ABZ sulfoxide (the active metabolite) are the most effective adjuvant CTX
  - Better GI absorption & better tissue distribution.
  - Reaches higher intracystic fluid conc.
  - Can be started Preop. (From none to 3 m. Preop.) & continued Post. op (4 wks Postop.)

— ABT alone can cure 10–30% of case f degeneration of the cyst in up to 92%. (So it should be combined w/ percut. drainage or surgery)

Indx of medical tt (CTX)

- Inoperable d. or unfit pt.
- Pts w/ MULTIPLE cysts in ≥2 organs
- Multiple small liver cysts / cysts DEEP in liver
- PERITONEAL cysts
- Pts following incomplete surgery or relapse.
- PREVENTION of 2ry spread of echinococcal infxn following Percut. rupture / aspiration of cyst.

• PERCUTANEUS DRAINAGE

Some studies show that percut. drainage in combination w/ CTX is safe & efficient / lower complications/ & better Postop. recovery.

BUT [in surgical Recall] — it's mentioned that you should NEVER do Percut. drainage dve to risk of ~~rupture~~ or leakage of into Peritoneal cavity & anaphylaxis

• SURGERY (The MAINSTAY of tt!)

Principles: Eradicates the Parasite in >90%
Avoids spillage
Obliterates the residual cavity.

Indx:
- Superficial cyst w/ risk of rupture
- Large cyst >10 cm w/ many daughter cysts
- Cystobiliary communication
- Mass effect to vital organs.
- Infected cysts.
- Any extrahepatic localized cyst.

Leakage of cyst contents into Peritoneum may be FATAL, cuz it causes Anaphylactic rxn.

* Surgical vs. Conservative
* Open vs. Laparoscopic.

Surgical options

- **RADICAL** approach
  - Cystectomy
  - Pericystectomy
  - Liver resection.

* LESS recurrence! ☺

VS

CONSERVATIVE approach

- External drainage.
- Wide roof excision
- Evacuation & sterilization of the cavity
- Capitonnage
- Marsupialization
- Partial cystopericystectomy
- Near total pericystectomy

* EASIER to perform & Less operative risk

BUT HIGHER recurrence rate! 10-30%

** Considerations during conservative Mgt:

In case of wide communications ⇒ do biliary bypass
sphincteroplasty
ERCP & endoscopic sphincterotomy
CBD exploration & T-tube insertion

- LAPAROSCOPIC vs. OPEN

⊘ CI of lap. approach:

- Cholangitis (due to communication)
- Liver cirrhosis
- cyst
  - Recurrent cyst
  - Complicated cyst ☺ rupture / infxn.
- location
  - Deep intraperitoneal cyst.
  - cyst in the post. segments.
  - cyst close to major vessels.
- > 3 cysts
- Thick calcified wall cyst.

Postop. complications

- Infxn of the residual cavity
- Intra-abd. abscesses
- Anaphylactic reactions.
- Spillage of Parasite material → 2iy echinococcosis
- Biliary fistulation
- Sclerosing Cholangitis.

*~~Postop~~

- During surgery → Toxic irrigation ☺ scolicidal agents. before cyst removal.

Scolicidal Agents

- Hypertonic Saline 10-20% (for 5-10 min)
- Peroxide solution 10%
- Chlorhexidine
- Cetrimide

The Girl
Yasmin Gabriella

TUMORS OF THE LIVER

Source : Surgical Recall

- Benign Liver Tumors
- Malignant Liver Tumors
- Metz

Overview:

- m.c liver CA is METZ! (it's way more common than liver tumors 20:1 !)
  - * Primary site is usually GIT
- m.c PRIMARY malignant liver tumor → Hepatocellular CA (Hepatoma) ≡ HCC
- m.c PRIMARY benign liver tumor → Hemangioma

Definitions

- Rt hepatic Lobectomy : Removal of Rt lobe of liver
  (i.e. Removal of ALL liver tissue to the Rt of Cantle's line)
- Lt hepatic Lobectomy : Removal of Lt lobe of liver
  (i.e. Removal of ALL liver tissue to the Lt of Cantle's line)
- Trisegmentectomy : Removal of all liver tissue to the Rt of the falciform ligament.

LIVER TUMORS

BENIGN

- Hemangioma
- Hepatocellular Adenoma
- Focal Nodular Hyperplasia (FNH)
- Hamartoma: white hard nodule made of liver cells

* Other Benign Liver masses :
  - Benign liver cyst
  - Bile duct hamartoma
  - Bile duct adenoma.

MALIGNANT

- Hepatocellular CA (Hepatoma) 8x
- Cholangiocarcinoma (when intrahepatic)
- Angiosarcoma (ass. w Chemical exposure)
- Hepatoblastoma (m.c in infants & children)
- ~~Hamartoma: white hard nodule made of liver cells~~

RF for angiosarcoma (Chemical exposures):
- Vinyl Chloride
- Arsenic
- Thorotrast contrast

METZ

m.c lymphatic site is GIT

BENIGN

[redacted]

- It's benign vascular tumor of the liver
- It's the m.c Primary benign liver tumor (up to 7% of population)

- **SS:**
  - RUQ Pain / mass
  - Bruits.

- **COMPLICATIONS:**
  - Pain
  - Congestive HF
  - Coagulopathy
  - Obstructive jaundice.
  - Gastric outlet obstruction.
  - Kasabach - Merritt synd. (see box)
  - Hmg (Rare!)

> **Kasabach - Merritt Synd.**
> - Hemangioma **AND** Thrombocytopenia & fibrinogenopenia.

- **DX:**
  - CT scan w IV contrast.
  - Tagged red blood scan.
  - MRI
  - U/S

- (NOTE) Biopsy should NOT be performed! due to risk of Hmg w bx.

- **Tx:**

  OBSERVATION (>90%)

- **Indx for resection**
  - Symptomatic
  - Hmg
  - cannot make dx.

[redacted]

- BENIGN liver tumor.

HISTOLOGY

DL hepatocytes WO bile ducts.

RF:

♀ : ♂ ratio
9 : 1

- ♀
- Birth control pills (THINK ABC : Adenoma Birth Control)
- Anabolic steroids
- Glycogen storage disease

Avg. Age: 30-35 yrs of age.

S&S:

- RUQ Pain / mass
- RUQ fullness
- Bleeding (rare)

COMPLICATIONS:

- Rupture w/ bleeding (33%)
- Necrosis
- Pain
- Risk of HCC.

Dx:

- CT
- US
  ± bx (r/o hemangioma w/ RBC-tagged scan!)

~~Tx~~

If Small → Stop Pills — it may regress
If not → Surgical resection is necessary.

If Large (>5cm) / Bleeding / Painful / or Rupture → Surgical resection!

[redacted] (FNH)

. BENIGN Liver tumor

HISTOLOGY: NL hepatocytes & **Bile ducts**
(while adenoma has NO bile ducts)

~~Avg Age~~: ~40 yrs

~~RF~~: ♀

Dx: - Nuclear Technetium-99 study
- U/S
- CT scan
- A-gram
- Biopsy

Birth control pills are ass. w/ FNH but NOT as clearly ass. as w/ adenoma.

Classic CT Findings
Liver mass w/ "**central scar**"!
(THINK: focal = central)

COMPLICATIONS

. **Pain**
* NO RISK OF CA ☺ (unlike adenoma)
. Hmg (v. rare)

~~Tx~~:

- Resection or **Embolization** — if pt is symptomatic

Otherwise, follow if dx is confirmed
also STOP birth control pills.

* Why does embolization work w/ FNH?
w/ 2 this tumor is usually fed by **one major** artery.

MALIGNANT

[redacted]-CA (HCC)

AKA: Hepatoma

- m.c malignant Primary liver tumor.

INCIDENCE: 80% of all Dg malignant tumors
High risk areas: Africa & Asia.

RF:

most imp.
- Hep.B
- Cirrhosis
- Aflatoxin (Fungi toxin of "Aspergillus Flavs")

- α_1 antitrypsin def.
- Hemochromatosis
- Liver fluke (Clonorchis sinesis)
- Anabolic steroids
- Polyvinyl chloride
- Glycogen Storage d. (type I)

> ~5% of pts w Cirrhosis will develop HCC!

SFS:

Classic Presentation: PAINFUL HEPATOMEGALY
- Dull RUQ Pain
- Hepatomegaly
- Abd. mass
- Wt loss.
- Paraneoplastic synd.
- Signs of Portal HTN
- Ascites
- Jaundice
- Fever
- Anemia
- Splenomegaly.

> Tumor marker for HCC is
> ↑ α-FetoProtein

InVx:

- Tumor marker (↑ α-Fetoprotein)
- U/S
- CT
- Angiography.

Dx:

M.C way to get a tissue dx is
Needle bx ω CT / US / or Laparoscopic guidance.

Tx:

- Surgical Resection (if possible)
  ex. Lobectomy
- Liver transplant. (see indx)

* If NOT candidate for surgery do:
  Percut. ethanol tumor injection/
  cryotherapy/
  & intra-arterial CTX

The M.C site of Metz is **LUNGS!**

Indx for Liver Transplant

- Cirrhosis & NO resection candidacy as well as NO distant L.N metz & NO vascular invasion.
- The tumor must be SINGLE & < 5 cm
  or have three nodules ω NONE > 3 cm.

Px:

If unresectable → Almost NONE survive! ☹
If Resectable → ~35% are alive at 5yrs

* Fibrolamellar Hepatoma is a subtype of HCC has the BEST Px! (in young adults)

Yours truly
The End.

HEMOBILIA

Source Read

DEFINITION

Blood draining via CBD into the duodenum

DX TRIAD!

- RUQ Pain
- Jaundice
- Guaiac +ve / UGI bleeding.

CAUSES

- Trauma w/ liver laceration
- Percut. transhepatic cholangiography (PTC)
- Tumors

Invx

- Endoscopy — Blood out of the ampulla of vater
- A-gram

Tx

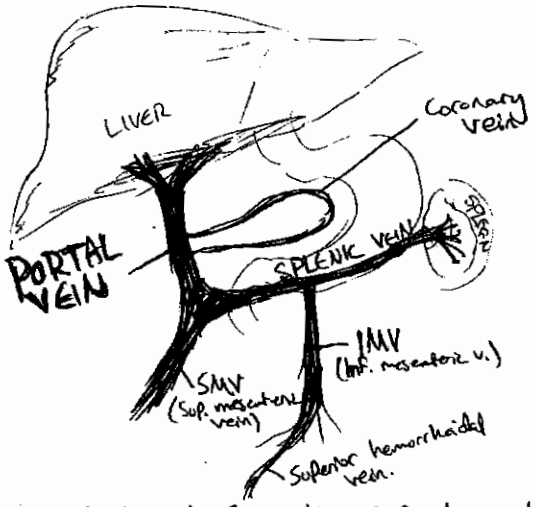
A-gram w/ embolization of the bleeding vessel.

The End

PORTAL HTN

Source: Reacall 215

* Portal Pressure > 10 mmHg



ÑL Portal Pressure is < 10 mmHg

* Drainage of blood from the Sup. hemorrhoid v. :
Sup. hemorrhoidal v. → IMV → Splenic V. → Portal v.

* Portal v. starts at the confluence of **Splenic Vein** & **SMV**.

- 6 Potential routes of Portal-Systemic **collateral** blood flow: (As seen in Portal HTN)
  1. **Umbilical** v.
  2. **Coronary** v. to esophageal venous plexus
  3. **Retroperitoneal** veins (Veins of Retzius)
  4. **Diaphragm** veins (Veins of Sappey)
  5. **Sup. hemorrhoidal** v. & then to the iliac v.
  6. **Splenic vein** to the short gastric v.

PATHOPHYSIOLOGY of Portal HTN is ↑ Portal Pressure resulting from RESISTANCE to Portal flow

CAUSES

Cirrhosis is the n.c.c of Portal HTN in U.S (90%)

PREHEPATIC: Portal v. thrombosis / Atresia of Portal v. / Splenic v. thrombosis

HEPATIC:
- **Cirrhosis** (Distortion of ÑL Parenchema by regenerating hepatic nodules)
- HCC
- Fibrosis

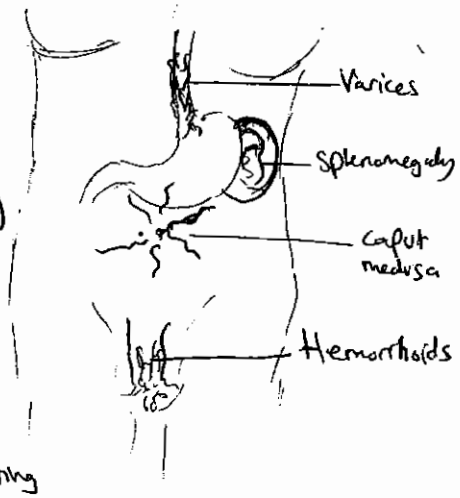
POST-HEPATIC: Budd-Chiari Synd. (Thrombosis of hepatic vn)
Tumors
~~Splenic v. thrombosis~~

* ~40% of pts w cirrhosis develop esophageal varices.
* 2/3 of pts w cirrhosis develop PORTAL HTN
* m.c Physical finding in pts w Portal hypertension is **SPLENOMEGALY**.

CLINICAL FINDINGS

most imp
- Esophageal Varices
- **Splenomegaly**
- Caput medusa (engorgement of Periumbilical veins)
- Hemorrhoids

also,
- Spider Angioma
- Palmar erythema
- Ascites
- Truncal obesity & Peripheral wasting
- Encephalopathy
- Asterexis (Liver Flap)
- Gynecomastia
- Jaundice.



* Esophageal Varices — coronary v. backing up into Azygous System.
* Caput Medusae — Umbilical v. (via falciform lig.) draining into the epigastric veins.
* Retroperitoneal varices — Small mesentric veins (veins of Retzius) draining retroperitoneally into lumber v.
* Hemorrhoids — Sup. hemorrhoidal v. (which drains into IMV) backing up into the middle & inf. hemorrhoidal veins.

* The most feared COMPLICATION of Portal HTN is BLEEDING from esophageal varices!

● Esophageal Varices: Engorgement of the esophageal venous plexuses 2ry to ↑ collateral blood flow from Portal System as a result of Portal HTN.

- Mortality Rate from acute esophageal variceal bleeding is 50%
- ~~tt~~ of variceal bleeding

Initially, 2 Large bore IV canula
IV Fluids
Foley catheter
Type & cross blood
Send Labs
Correct coagulopathy (Vit K, FFP)
± Intubation to protect from aspiration.

Endoscopy is diagnostic & therapeutic.

ENDOSCOPIC ~~tt~~ options:
— Emergent endoscopic sclerotherapy
— Endoscopic band ligation

MEDICAL tt options:
— Somatostatin (Octreotide)
— IV vasopressin (& nitroglycerin to avoid MI)
* to achieve vasoconstriction of the mesentric vessels
* If bleeding continues — consider balloon tamponade of the varices.

NOTES

● If sclerotherapy & conservative methods failed to stop the variceal bleeding / or bleeding recurs ∴ Mgt:
— Repeat sclerotherapy / Banding & treat conservatively
— TIPS
— Surgical shunt (selective / Partial)
— Liver transplantation

TIPS: Transjugular Intrahepatic Portosyst. Shunt.

TIPS Procedure:

Angiographic radiologist places a small tube stent intrahepatically btw. the hepatic v. & a branch of the Portal v. via a percutaneous jugular v. route.

SURGICAL Shunt:

- Partial Shunt: Shunt that directly decompresses the Portal v. but only Partially.

  - Selective shunt (Warren): Distal splenorenal shunt w/ ligation of the coronary v.
    — ass. w/ ↓incidence of encephalopathy.
    - CI: Ascites.

- M.C Perioperative cause of death following Shunt Procedure is HEPATIC FAILURE (2ry to ↓ blood flow)

* MAJOR Postop. MORBIDITY after shunt Procedure is ↑ Incidence of Hepatic ENCEPHALOPATHY bce of ↓ Portal blood flow to the liver & ↓ CLEARANCE of toxins/metabolites from the blood!

- What LAB value roughly correlates w/ degree of encephalopathy?
  Serum Ammonia Level
  (NOTE: It's thought to correlate w/ BUT NOT cause encephalopathy!)

- ttt of Hepatic encephalopathy:
  Lactulose PO w/wout Neomycin PO

Yale Gurley
The Great.

SMALL INTESTINE

(Duodenum/Jejunum/Ileum)

Source : Recall, 21st
First aid

EMBRYOLOGY

All of the S.I is derived from the **midgut** EXCEPT the proximal duodenum — which is derived from the foregut.

*The junction btw. the foregut & midgut is immediately distal to the opening of CBD

ANATOMY

- Total length : Avg. = 6m.
- **Ligament of Treitz** marks the end of the duodenum & the start of the jejunum.

Fxn of Small Intestine: Digestion & Absorption

DUODENUM

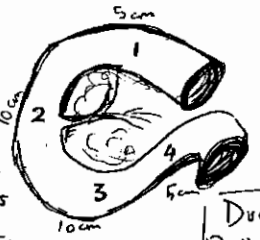
- Extends from the Pylorus to the duodenojejunal junction.
- **Parts**

① 1st Part (Superior) — 5cm — duodenal bulb (Site of most ulcers)

② 2nd Part (desc.) — 10cm — curves around the head of Pancreas

③ 3rd Part (Transverse) — 10cm — crosses ant. to the aorta & IVC & Post. to SMA & SMV

④ 4th Part (Asc.) — 5cm — ascends Past left side of aorta then curves ant. to meet the JJ (duodenojejunal junction) — suspended by the Ligament of Treitz



Duoden is retro-Peritoneal EXCEPT the 1st 2cm.

- **Blood Supply**

→ PROXIMAL Part (up to ampulla of vater) — Gastroduodenal art. (br. of Proper hepatic art.) Bifurcates into Ant. & Post. Sup. Pancreatic duodenal art.

→ DISTAL Part (beyond the ampulla of vater) — by Inf. Pancreaticoduodenal art. (br. of SMA) — ant. & Post.

Venous Drainage

- Ant. & Post. pancreaticoduodenal v. drain into → SMV (which joins the splenic v. to form Portal v.)
- Prepyloric v. of Mayo is landmark for pylorus.

• JEJUNUM & ILEUM

- No anatomic boundary btw. the two.
- Jejunum is the prox. (40% of S.I) — distal to lig. of Treitz
- Ileum is the distal (60% of S.I)
- Mesentery tethers the jejunum & ileum to Post. abd. wall.

* The diff. btw. Jejunum & Ileum

- Jejunum — long vasa recta
  large plicae circularis
  Thicker wall
- Ileum — Short vasa recta
  Smaller plicae circularis
  Thinner wall

Plicae Circulares
the circular folds of mucosa in S.I lumen
AKA: Valvulae Conniventes
Plicae: "folds"

Mnemonic
THINK: Ileum = **I**nferior vasa recta
Inf. plica circularis
Inf. wall thickness
} In comparison to the jejunum.

* The terminal ileum absorbs B12, fatty acids, bile salts

Blood Supply

Branches of SMA (which runs in the mesentry)
* The arteries loop to form arcades that give rise to Straight arteries — vasa recta.

Venous drainage

SMV

* LYMPHATICS

Bowel wall → mesentric nodes → lymphatic vessels → cisterna Chyli (retroperitoneal structure btw. aorta & IVC)
Lt Subclavian v. ← Thoracic duct ←

* INNERVATION

- Parasymp. — originate from vagus & celiac ganglia — ⊕ motility
- Symp. — originate from ganglion cells that reside at the base of SMA — ⊖ motility.
- Enteric Nervous Syst. — Meissner plexus

INTESTINAL OBSTRUCTION

Source : Dossier Recall 221

CLASSIFICATION

- Mechanical vs. Fxnal (AKA Pseudo-obstruction / Adynamic / Paralytic ileus)
- Complete vs. Incomplete
- Simple vs. complicated (Strangulated?)
- Small bowel vs. Large bowel obst. (Prox. vs. distal)
- Acute vs. Chronic
- Closed loop vs. Open loop
- Gangrenous vs. Nongangrenous

Trichobezoars
Is a hair bolus occuring in young ♀ w long hair w Psychiatric disorders.

CAUSES

- CAUSES of **Mechanical** obst. :
  - **INTRALUMINAL :**
    - Fecal Impaction
    - Foreign Bodies
    - Bezoars
    - Gall stones (usually due to fistula)
  - **INTRAMURAL :**
    - Stricture (as in Crohn's)
    - CA
    - Diverticulosis

*Strictures are due to:
- Ischemia
- Inflam. (Crohn's)
- RTX
- Surgical (Iatrogenic) Trauma

  - **EXTRAMURAL :**
    - Bands / Adhesions
    - Hernia (Internal / External)
    - Volvulus
    - Intussusception

- The **m.c.c** of **small intestinal obstruction in**
  developed countries → Adhesions
  developing " → Hernias
  (or worldwide)
- The **m.c.c** of **Colonic** obstruction :
  1. Colon CA (1st m.c.c)
  2. Volvulous (2nd m.c.c)
  3. Diverticulosis (3rd m.c.c)

The most Prominent Gas found in Bowel during obst. is **N_2** bcz it's absorbable Gas.

- CAUSES of **Fxnal** obst. :
  - Postop. (Normally resolves in 3-5 days)
  - Electrolytes abnormalities. (Hypokalemia)
  - Peritonitis / Sepsis / Shock
  - Drugs (Opiates / Anticholinergics)
  - Hemoperitoneum / Retroperitoneal hmg.
  - Spine injury.

*What tumor classically causes Small bowel obst. due to "mesentric fibrosis"?
Carcinoid tumor

S&S

- Colicky Pain → If Proximal, time btw. attacks is LESS than if it's distal.
- Vomiting → Proximal > Distal
- Constipation → Distal > Proximal
- Distention → Distal > Proximal
- Diarrhea → in certain cases like colon CA / GB obstruction

↑ Bowel sounds
Visible peristaltic waves

Signs of Strangulated bowel w/ SBO
- Fever / Severe, Continuous Pain
- Hematemesis
- Shock / Acidosis (? lactate) / Peritoneal Signs
- Gas in the bowel wall or Portal v.
- Abdom. free gas

Invx

- CBC, electrolytes
- Plain, Abdominal X-Ray: (FINDINGS)

Erect: multiple air-fluid level (non-specific)

Supine (more ind.) distended bowel

By supine abdominal X-Ray:
- Confirm dx
- Detect if Proximal or distal.
- Detect type of intestine involved.

* If you see plica circularis → this is jejunum
* Gas in intramural space → Infarction
* Gas under diaphragm → Perforation.

- CT
- Endoscopy

Presence of any gas in the small intestine is indicative for Intestinal obst.
UNLIKE colon, which normally contains gas.

Mgt

(Initial)
- NPO
- NGT
- IV Fluids
- Foley cath.

(Then,)

If Complete obst. → do Laparatomy & lysis of adhesions (LOA)

If incomplete → Initially, conservative tt w/ close observation + NGT decompression.

Complete vs. Incomplete

- Complete — usually NO Passage of BOTH stool & flatus. (Obstipation) ↑RISK of Strangulation → Necrosis
- Incomplete — some Passage of flatus.

How to diff.?
- By CT w/ oral contrast
- & small bowel follow-through.

The End
Sara Ghaith (2)

FISTULAS

Source: Washington 223 Recall

DEFINITION

It's an abnormal communication btw. 2 epithelialized organs.

CAUSES — that maintain Patency of fistula:

Mnemonic

Remember it as "HIS FRIEND"

- **H**igh-output Fistula
- **I**ntestinal destruction >50% of circumference
- **S**hort segment fistula <2.5 cm
- **F**oreign Body
- **R**TX
- **I**nfxn
- **E**pithelization
- **N**eoplasm
- **D**istal obstruction

CLASSIFICATION

- **External vs. Internal** (m.c)
  - External: to skin.
- **Proximal vs. Distal.**
  - Proximal — usually high output
    - ass. w/ dehydration / malnutrition / electrolyte disturbance
  - Distal — after ileum & colon
    - less S/E

LOW OUTPUT	<200
MODERATE OUTPUT	200 - 500
HIGH OUTPUT	> 500

SPECIFIC TYPES

- **ENTEROCUTANEOUS FISTULA** — from (GIT → skin)

  CAUSES:
  - Anastomotic leak
  - Trauma / Iatrogenic
  - Abscess / TB / Amebiasis (Infxn)
  - Crohn's disease.
  - Diverticulitis (cause of colovesical fistula)
  - Infxn / Inflammation.
  - Inadvertent suture into the bowel.
  - Vascular compromise.

Invx
- CT scan to r/o abscess / inflammation.
- Fistulogram.
- Endoscopy.

COMPLICATIONS
- High-output fistula
- Malnutrition
- Skin breakdown

Factors ↑ Rate of Closure:
- ↓ Output
- Long tract >2cm
- Small orifice <1cm

ttt
- NPO, TPN (Enteral is CI).
- Drain the abscess.

r/o & correct the underlying cause.

50% → resolve spontaneously · after 4-6 wks of sepsis & adequate nutrition support

50% → Need surgery (considered dirty surgery)

NOTES
- Long fistula heals faster

ttt options
- Resection & Primary anastomosis.
- Vacuum-assisted closure device.

COLONIC FISTULA

ex **Colovesical (n.c)** / Colocutaneous / colovaginal / coloenteric.

CP: Present w recurrent UTI

CAUSES:
- **Diverticulitis (n.c)**
- Foreign body
- Cancer
- IBD
- RTX

Cholecystenteric (GB w. duodenum connection) due to large gallstone erosion

Gastrocolic fistula, may cause penetrating ulcer.

DX: by Barium enema or cystoscopy.

ttt: Surgery
- Segmental resection & 1° anastomosis & Repair of the involved organ.

Complication
- malnutrition
- severe enteritis.

• PANCREATIC ENTERIC FISTULA

Decompression of a Pseudocyst or abscess into adjacent organ — usually done surgically

• EXTERNAL PANCREATIC FISTULA

(Drains exocrine syn to skin)

Somatostatin
↓ output of the fistula.

Tx: NPO, TPN, skin protection for treatable

Dx: ERCP

If refractory, resect the tail of the Pancreas if it's in the tail.

or Pancreaticojejunostomy (if fistula is in head)

• BLADDER FISTULA

TYPES:- Vesicoenteric

50% to sigmoid

Signs: Pneumaturia / fecaluria

- Vesicovaginal

Sign: urinary leak to vagina.

• FISTULA IN ANO

From rectum to anal skin.

CAUSES - Anal crypt infxn.
Perianal abscess ~ 3 days

S&S - Perianal drainage
- Perirectal abscess
- Diaper-rash
- Itching.

The End

SPLEEN

Source : Washington Recall 22nd

EMBRYOLOGY

- Develops from condensations of mesoderm in the dorsal mesogastrium,
  then rotation of the gut occurs & it become located in the upper quadrant of the abdomen.

ANATOMY

- Located in the LUQ (Lt hypochondrium) btw. 9th - 10th ribs.
- 12 cm long, 7 cm wide, 4 cm thick
- Wt: (100 - 150) g
- **Boundaries**

Sup. : Lt diaphragm leaf.
Inf. : Colon / splenic fl. / Phrenocolic lig.
Med. : Pancreas (tail) / Stomach
Lat. : Ribs
Ant. : Ribs / Stomach
Post. : Ribs

① Cardiodiaphragmatic recess of the Lt pleural cavity extend to the inf. border of a ↑↓ spleen.
② Splenic flexure of the colon.
③ Greater curvature of the stomach.
④ Lt Kidney
⑤ Tail of Pancreas ("Tickles" the spleen)

The spleen is completely intraperitoneal organ EXCEPT the hilum!.

- **Peritoneal Reflections :**
  - Splenocholic
  - Splenorenal
  - Gastrosplenic
  - Splenophrenic

10 - 20% of Population have accessory spleen

m.c site of accessory spleen in
- splenic hilum.
- gastrosplenic omentum
- along the tail of Pancreas
- Retroperitoneum

BLOOD SUPPLY

- Splenic Art. (Branch of the celiac trunk) → tortuous!
- Short gastric art. (From gastroepiploic art.)

VENOUS DRAINAGE

Splenic vein & Lt gastroepiploic v. ⇒ to Portal v.

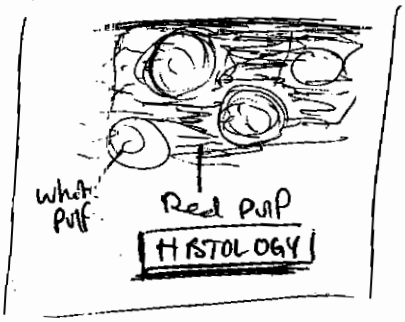
LYMPHATIC DRAINAGE : nodes at hilum → retropancreatic LN → celiac LN (1)

PHYSIOLOGY

Splenic parenchema consists of (surrounded by serosa & collagenous capsule)

RED PULP: 80% — Highly vascular (RBCs)
- Composed of splenic cords

WHITE PULP: 20%
- Periarterial lymphoid sheaths (T-cells)
- Lymphoid nodules (B-cells)
- Marginal zones.



FUNCTIONS

1. **FILTRATION** of RBCs (NOT storage) — By RED PULP.
2. **STORAGE of Platelets** (33% of total body plts are stored in the spleen)
3. **IMMUNITY:** — By WHITE PULP.
   - Produces **tufsin & properdin (opsins)**
   - Produces antibodies (esp. **IgM**)
   - Site of phagocytosis.

NOTES

- Splenomegaly Vs. Hypersplenism

  Splenomegaly: Enlarged spleen (Part of hypersplenism)

  Hypersplenism:
  - Hyperfunctioning spleen.
  - Documented loss of blood elements (WBC / Hct / Plts)
  - Large spleen! (Splenomegaly)
  - Hyperactive BM (trying to keep up w/ loss of blood elements)

- Pts w/ UC develop HYPOSPLENISM.

Mnemonic
- Remember opsins as:

  "**Ops!** it's **PRO**fessionally **TUF** spleen"

  Ops = opsins : **PRO** = Properdin **TUF** = Tufsin

INDICATIONS OF SPLENECTOMY

- Types of splenectomy
  - Laparoscopic
  - Open.

① TRAUMA

② THROMBOCYTOPENIA

- **Idiopathic Thrombocytopenic Purpura (ITP):**
  - Autoimmune
  - m.c indx for **elective** splenectomy
  - Autoantibodies produced against a platelet glycoprotein (IgG)
  - ttt
    - 1st line → Corticosteroids
    - 2nd line → Surgery (If refractory to steroids)
  - 90% of children w acute ITP will resolve spontaneously w/in (6–12) months!

Mnemonic

I's in the ITP:

- **I**mmune etiology (IgG antiplatelet)
- **I**mmunosuppressive ttt (steroids)
- **I**mmunoglobulin
- **I**mprovement w/ splenectomy (75% have improved plt counts after splenectomy).

- **Thrombotic Thrombocytopenic Purpura (TTP)**
  - RARE!
  - Diagnostic **Pentad**: Mnemonic "FAT RN"
    - **F**ever
    - **A**nemia
    - **T**hrombocytopenia
    - **R**enal dysfxn
    - **N**eurologic dysfxn
  - ttt
    - 1st line → Plasmapheresis (plasma exchange)
    - If failed → splenectomy. (last resort!)

NOTE

~~Transfusing~~ plts in TTP is thought to "fuel the fire!" & exacerbate consumption of plts & clotting factors.
→ Resulting in more thrombi in the microvasculature
i.e. Plasmapheresis is the ttt of choice!
NOT transfusion.

③ ANEMIAS

- Hereditary spherocytosis (m.c AD defect in spectrin – RBC membrane Protein)
- Medullary fibrosis w/ myeloid metaplasia
- Autoimmune hemolytic Anemia
- Sickle cell Anemia
- Thalassemia. (ex. β-thalassemia major, AKA Cooley's)

④ MALIGNANCIES

- Hodgkin's Staging not conclusive by CT (not used nowadays)
- Splenic tumors (ly / metz / locally invasive)
- Hypersplenism caused by other neoplasms / non-Hodgkin

⑤ Miscellaneous Indx

- Variceal bleeding ẅ splenic vein thrombosis
- Gaucher's disease (Storage disease)
- Splenic Abscess / cyst / Pseudocyst (refractory) — Rare!
- Splenic artery aneurysm.
- Hypersplenism
- Felty's Synd. (Autoimmune Neutropenia)
- Palliation of ~~cirrhosis~~ splenism

NOTES

- Pts ẅ sickle cell have autosplenism.
- G6PD def. is NOT an indx for splenectomy.
- The M.C indx for splenectomy are:
  - → Trauma
  - → ITP (refractory to steroids)

Splenectomy

After surgery:

- Complete response if platelet count ↑ 100×10^9/L
- Partial response if Platelet count ↑ 30×10^9/L

Abnormal Labs Postop
- ↓WBC >50% above baseline
- marked thrombocytosis
- RBC smear is abnormal

Platelets ↑ AFTER splenectomy. Why?
Bcz spleen stores Plts (33%)

- If failed to ↑, LOOK for **accessory** spleen tissue.

Preop. consideration:

- **Vaccination** — For encapsulated bacteria:- (2 wks preop.)
  - Pneumococcus
  - H. Influenza type B
  - Meningococcus.
- **Transfusion consideration**
  - esp. Pt ẅ hematological d.
  - do cross-match 24 hrs prior to surgery.
  - ⇒ If pt has SEVERE thrombocytopenia, Plts should be available Preop.
- **Imaging**: US, CT — to determine spleen size.
- **Embolization of spleen preop.** (To ↓ size)

▶ Splenectomy:

Open vs. Laparoscopy

⊘ CI of LAPAROSCOPIC splenectomy:

ABSOLUTE : . Portal HTN
. Splenic trauma & unstable pt
. Massive splenomegaly (>30 cm length!)

RELATIVE : . Morbid obesity
. Splenic vein thrombosis
. Moderate splenomegaly (>20-25 cm length)

The m.c index for splenectomy:
- TRAUMA
- ITP

Open splenectomy Incisions: — midline (Preferred)
— Lt subcostal Lt incision.

Note
* If suspected injury to Pancreatic tail → Put a drain.

ABNORMAL lab tests Post-splenectomy:
on Peripheral smear,
- Pappenheimer bodies
- Howell-Jolly bodies
- Heinz bodies

COMPLICATIONS OF SPLENECTOMY

INTRA-OP

- Hmg (due to hilar distention or capsular tear) or injury to a vessel.
- Pancreatic injury (esp. the tail)
- Bowel injury / colon / Stomach.
- Diaphragmatic injury.
- Pancreatic gastric dilatation

EARLY

Thrombocytosis will cause Splenic / Portal V. thrombosis.

- Pulmonary Atelectasis / Pneumonia / Pleural effusion
- Subphrenic abscess (Tt Percutaneous drainage & IV abx)
- Wound Problems (Hematoma / Seroma / wound infxn)
- Thrombocytosis (if > 1 million → give Aspirin)
- Ileus

LATE

- **O**verwhelming **P**ost**S**plenectomy **S**epsis (OPSS)
  - ↑ susceptibility to fulminant bacteremia, meningitis, or Pneumonia b/c of loss of splenic fxn.
  - Incidence < 1% in adults.
  - >30% mortality rate.
  - C/P – Fever / lethargy
    – Common cold / sorethroat / URTI followed by confusion
    – Shock & coma w/ death ensuing w/in 24 hrs in 50% of pts.
  - Organisms Encapsulated bact. (Strep. Pneumonia / H. influenza / N. meningitis.
  - Occurs usually w/in 2 yrs post op.
  - (#) : Give daily Prophylactic Abx.
    Vaccinations.

PF
– Young (3 yrs)
– Immunocompr.

m.c.c of isolated gastric varices is Splenic V. thrombosis

m.c.c of splenic V. thrombosis is PANCREATITIS!

- Splenosis :
  Disseminated intraabdominal splenic tissue occurs after splenic rupture

- Delayed splenic rupture:
  Subcapsular hematoma or pseudoaneurysm rupture after blunt trauma 2 wks after.

  Presents as Shock / Abd. Pain

  Sx & Signs : LUQ Pain (like any splenic injury)
  - Kehr's Sign (Lt shoulder Pain from diaphragmatic irritation)
  - Ballance's Sign (LUQ dullness to Percussion)
  - Seagesser's Sign (Phrenic n. compression causing neck tenderness)
  - Hemoperitoneum.

SPLENIC ARTERY ANEURYSM

RF (for rupture)
- Pregnancy (3rd trimester)
- >2cm
- Symptomatic

AXR shows eggshell calcification.

Splenorrhaphy

"Splenic salvage operation"
≡ Wrapping visceral mesh, aid topical hemostatic agents or partial splenectomy then ⇒ sutures

SPLENIC TRAUMA

- Usually BLUNT trauma.
- one of the m.c.c of splenectomy

Conditions ass. w rupture of spleen:
- Mononucleosis!
- Malaria
- Blunt LUQ trauma
- Splenic abscess

C/P LUQ Pain

Signs
- signs of Peritoneal irritation
- External signs of injury
- Kehers sign
- Ballance's Sign. & Seagesser's sign
- Hemoperitonium
- Shock
- Lt-sided lower rib fracture.

Dx
* If unstable → DPL or FAST exam.
* If stable → CT

tt
* If Stable → Non-operative tt (w an isolated splenic injury w/o hilar involvement or complete rupture)
* If unstable → DPL/FAST laparotomy w splenorrhaphy/splenectomy
* Embolization in selected pts.

TUMORS OF SPLEEN

BENIGN: Hemangioma / lymphangioma
Hamartoma
Iry cyst / echinococcal cyst.

MALIGNANT: - lymphoma / myeloproliferative disorder.
- Metz

SPLENIC ABSCESS

CAUSES
- Sepsis seeding
- Infxn from adjacent structures
- Trauma
- Hematoma
- IV drug use.

SAS
- Fever / chills.
- LUQ tenderness & guarding.
- Spleen may / may not be palpable.

Dx
- U/S
- CT

tt
Splenectomy for most cases.
If large / solitary juxtacapsular abscess → Percut. drainage.

Complications
- Spontaneous rupture.
- Peritonitis
- Sepsis.

Yash Gavit
The Great

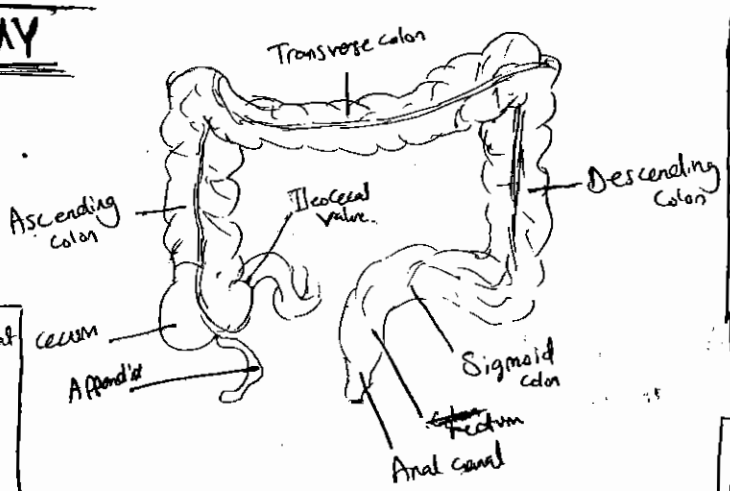
COLON, RECTUM & ANUS

EMBRYOLOGY

Origin: Embryonic **midgut** (Ascending colon & mid transverse colon)
Embryonic **hindgut** (rest of colon, & desc. colon & Proximal anus)
Ectoderm (distal anus)

* The dentate line marks the transition btw. hindgut & ectoderm.

ANATOMY



Bowel Anatomy

In development of **midgut** loop rotates 270° counterclockwise around the axis of SMA.

*Developmental anomalies include malrotation or failure of Rt colon to elongate.

Parts of GIT that do NOT have a serosa:
- Esophagus
- Middle & distal rectum.

Ileocecal valve fxn:
⊖reflux of bowel contents from cecum back to the ileum.

• COLON

- Extends from the ileocecal valve to the rectum: Rt colon, Transverse colon, Left colon, Sigmoid colon
- Cecum is the widest, The colon progressively narrows distally.
- The colon has taenia coli, haustra & appendices epiploicae (fat appendages that hang off antimesenteric side of colon)

Taenia Coli
3 distinct bands of longitudinal muscle **converge** at the appendix & spread out to form a longitudinal muscle layer at the proximal rectum.

*Retroperitoneal structures: Asc. colon / Desc. colon / Post. hepatic // splenic

*Intraperitoneal Structures: Cecum / Transverse colon // Sigmoid Colon.

• RECTUM

Pelvic Floor
= Levator ani
- Puborectalis
- Pubococcygeus
- Iliococcygeus

Innervated by S4

- 12-15 cm in length.
- It has distinct Peritoneal coverage.
- **Fascia**: ① **Waldeyer's Fascia**: Rectosacral fascia that extends from S4 vertebral body to rectum.
② **Denonvillier's Fascia**: Ant. in lower ⅓ of rectum. (Condensed part)

• ANUS 4-5 cm

- It runs from pelvic diaphragm to anal verge. (junction of anoderm & perianal skin)

- **Dentate line**: A mucocutaneous line that separates Proximal, pleated mucosa from distal, smooth anoderm.

ANATOMICAL Anal canal
From anal verge to dentate line (one embryonic & anatomical structure)
SURGICAL Anal canal
from anal verge to anorectal ring 4-5 cm.

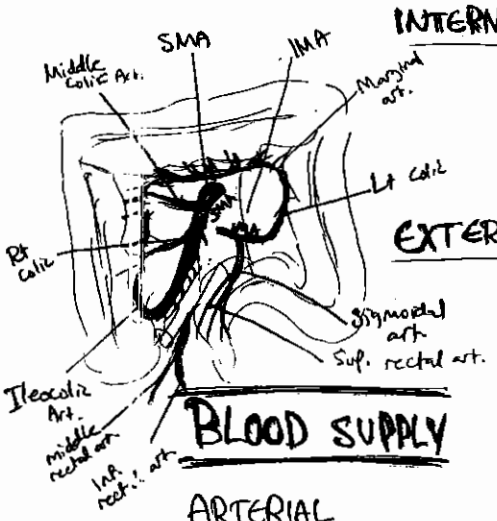
- Anal mucosa: Prox. to dentate line — lined by ~~St~~. columnar epithelium
  Distal to dentate line (Anoderm) — lined by Squamous epithelium & lacks glands & hair

- Columns of Morgagni: 12-14 columns of pleated mucosa sup. to the dentate line, separated by crypts.
  * Perianal glands discharge their secretions at the base of the columns.

- **Anal sphincter:**

INTERNAL ⇒ consists of specialized rectal ~~muscle~~ smooth muscle (from inner circular layer)
- Involuntary
- Contracted at rest
- Responsible for 80% of resting pressure.

EXTERNAL ⇒ Consists of 3 loops of voluntary striated muscle
- a continuation of puborectalis muscle.
- Responsible for 20% of resting pressure & 100% of voluntary pressure!



BLOOD SUPPLY

BLOOD SUPPLY
Midgut → SMA
Hindgut → IMA
Distal anus via Int. Pudendal art. br.

ARTERIAL

- **Superior mesenteric Art. (SMA)**: Supplies **cecum / ascending colon / & Prox. 2/3 of transverse.**
  By ileocolic / Rt colic / middle colic art.

- **Inferior mesentric Art. (IMA)**: Supplies distal 2/3 of transverse / Sigmoid / Sup. rectum
  By ~~middle rectal~~ Lt colic / Sigmoidal art. / Sup. rectal art.

- **Internal iliac art.**: Supplies the middle & distal rectum
  By middle rectal & Inf. rectal. (br. of int. Pudendal art)

- **Internal pudendal art**: Supplies the anus (br. of int. iliac art.)

The SPLENIC FLEXURE represents a "watershed" area btw areas supplied by SMA & IMA. * This area is particularly susceptible to ischemic injury as seen in ischemic colitis

VENOUS

Sup. mesentric vein (SMV): Drains the cecum, asc. & transverse colon before joining the splenic vein.

Inf. mesentric vein (IMV). Drains the desc. colon / Sigmoid / & Rectum before joining the splenic v.

Internal iliac vein: Drains the middle & distal rectum.
Middle rectal v. (A branch of int. iliac v.): drains upper anus.
Inferior rectal v. (A branch of internal Pudendal v.) drains lower anus.
Hemorrhoidal complexes: 3 complexes in the anus that drain into Sup. rectal v. & one ext. complex that drains into the Pudendal v.

LYMPHATIC DRAINAGE

- It usually follows the arterial supply

Anal canal **above** dentate line drains to Inf. mesentric nodes or to internal iliac nodes
BUT lower anal canal drains to → inguinal nodes

INNERVATION

- Derives primarily from autonomic nervous syst.
  - Symp. n. → ⊖ Peristalsis
  - Parasymp. n. → ⊕ Peristalsis.

HISTOLOGY

From inner lumen → outer lumen

COLON: Submucosa / inner circular m. layer / outer longitudinal m. (taenia coli)

RECTUM: Mucosa / Submucosa / inner circular m. / outer longitudinal

ANUS: Anoderm

MICROBIOLOGY

- Colon Sterile at birth, Normal flora established shortly thereafter.
- Normal Flora: 99% **Anaerobic** (Predominantly Bacteroides fragilis)
  1% **Aerobic** (Predominantly E. coli)

Constipation: Ability to pass flatus BUT inability to pass stool
Obstipation: Inability to pass BOTH flatus & stool.

PHYSIOLOGY

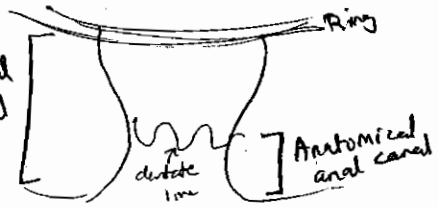
3 Physiologic fxns:
1. Absorption of water & electrolytes from stool
2. Storage of feces.
3. Motility.

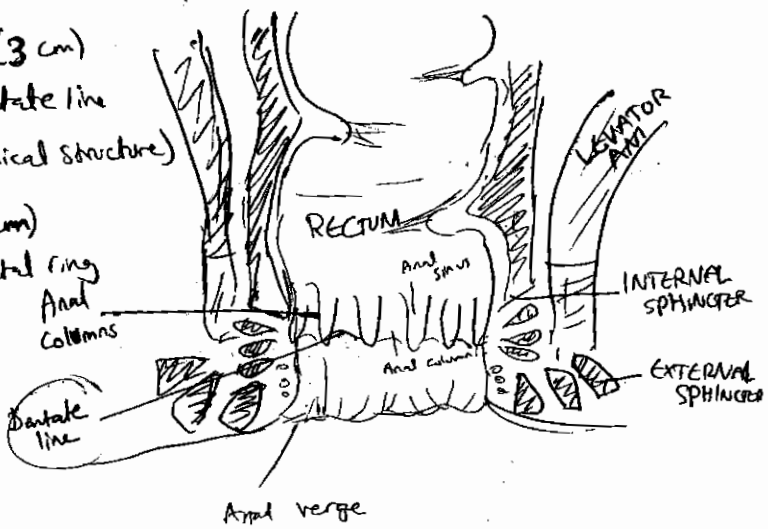
The End
Mark Griffith

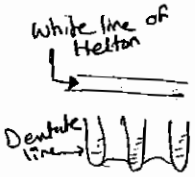
ANORECTUM & ITS DISEASES

ANUS

- ANATOMICAL Anal Canal: (3 cm)
  From anal verge → dentate line
  (One embryonic fanatomical structure)
- SURGICAL Anal Canal: (5 cm)
  From anal verge → Anorectal ring







DENTATE LINE

Formed by series of cusps — It contains spaces called crypts into which open the ducts of the mucus secreting anal glands.

*** It's considered a watershed area bcz it **SEPARATES** 2 embryonic structures that differ in their sensation, nerve supply & even their color!

Notes!

* The anoderm below the pectinate line is a SPECIALIZED form of skin that is DEVOID of skin appendages.

* The transitional area (AKA: Cloacogenic area / White line of Hetton) is the ACTUAL mucocutaneous junction NOT the dentate line — it's **1cm** ABOVE dentate line.
  ↳ This area can be lined by columnar cells, squamous or any type of epithelium.

COLUMNS OF MORGAGNI

Condensation ABOVE dentate line of no special importance.

ANAL GLANDS

- 8-12 in no.
- They lay in the **INTERSPHINCTERIC PLATE** & their ducts pass into the **crypts.**
- MOST of them are located in the **anterior** part of the anus.

ANAL SPHINCTERS

▶ 3-loop theory for the EXTERNAL sphincter.

1. **Subcutaneous** Part — has no attachment
2. **Superficial** Part — Attached to the coccyx
3. **Deep** Part — Attached to the Pubis

▶ The INTERNAL sphincter is a **continuation** for the **CIRCULAR** muscles of the rectum — so it's a smooth muscle.

NOTE: The longitudinal muscles continue as the conjoint tendon that is inserted to the perineal skin

RECTUM

- 12-15 cm
- Divided into: Upper 1/3, Middle 1/3, Lower 1/3

- **PERITONEUM:** Upper third — covers the ant. & lat. sides
  Middle 1/3 — covers ONLY the ant. part
  Lower 1/3 — NO Peritoneum Coverage. (extraperitoneal)
  the fascia in front of it is → **Denovilliers fascia**

- **FASCIA**
  - Denovilliers Fascia — forms the ant. part of lower 1/3 of rectum
  - Waldeyrs fascia (Rectosacral fascia) — condensation of Presacral fascia in the lower part of sacrum (S4)
  - Lat. Ligaments — From rectum to the sides of Pelvis. they contain the middle rectal vessels

- **ANORECTAL RING**
  - Formed of: Deep part of internal sphincter, Deep external sphincter, Puborectalis m.
  - Imp. in continence mech. it maintains a Right angle/acute (2)

If Ptosis ↓ Rectal Prolapse

PERIANAL SPACES

- Perianal space proper
- Ischiorectal fossa
- Intersphincteric space
- Supralevator space.

ARTERIAL SUPPLY OF RECTUM & ANUS

RECTUM — **Portosystemic** — Sup. rectal art. (from IMA) — Portal
Middle & inf. rectal art. (from int. iliac) — Systemic

ANUS — ONLY **Systemic** — inf. rectal art.

HEMORRHOIDAL PLEXUSES

Internal — contains highly oxygenated blood
External

LYMPHATIC DRAINAGE

* Perirectal lymphatics — drain into → **mesentric nodes** (m.c)
  → Internal iliac L.N
* Anal area — drains into → superf. inguinal L.N

NERVE SUPPLY

- Sphincters
  - Internal — By symp. (L1-L3) & parasymp. (S2-S4) (Hypogastric Plexus)
  - External — By (internal) Pudendal nerve. (S2-S4)
- Anus — By internal Pudenal n. (S2-S4) — Sensory & motor

* Below dentate line → sensitive to pain (so anal fissure is painful!)
* Above dentate line → Not sensitive to pain

Hypogastric Plexus (= Presacral Plexus)
- located in front of the Promontory of sacrum
- Contains BOTH symp. & parasymp.
- Innervates int. sph. & GUT.

NOTES

- Internal sph. is a smooth m. → involuntary & has tonic ctn, it does **NOT** fatigue, its fxn modified by symp. & parasymp.
- External sph. is a skeletal m. → voluntary & has somatic supply, it **fatigues** easily & under higher control (cortical)

DEFECATION — Physiology

Rectal distention
↓
⊖ of internal sphincter &
⊕ of defecation center

Cortical Facilitation (When it's appropriate to defecate)
↓
Cxn of levator ani & abd. muscles
↓
Defecation

Cortical Inhibition (When it's inappropriate to defecate)
↓
Cxn of external sphincter & Puborectalis sling
↓
Receptive relaxation of rectum & disappearance of urge.

NOTE

- **Receptive Relaxation:** It allows ↑ volume **wout** ↑ in Pressure (So when urge comes & no defecation occurs → dilation of rectum → urge will disappear)

ANORECTAL DISEASES

- Hemorrhoidal Disease
- Anal Fissures
- Anorectal Abscess
- Anorectal Fistula
- Pilonidal disease
- Perianal suppuration.

ANAL INCONTINENCE

TYPES

- MAJOR: Solid feces — defect in **external** sphincter
- MINOR: Gas / liquid stools / night soiling — defect in **internal** sphincter

The End.

HEMORRHOIDAL DISEASE

Source : Recall Dossier

* Hemorrhoids are NL structures that play a minor role in continence.

* When hemorrhoids enlarge / prolapse / or bleed, then become hemorrhoidal disease.

* 40% of pts with hemorrhoids will have Sx.

TYPES of Hemorrhoids

- INTERNAL — Above dentate line
- EXTERNAL — Below dentate line.

It's a RECURRENT Disease !

DEFINITION

It's a degenerative disease of the connective tissue.

PATHOPHYSIOLOGY

1. Problem in the venous channels / Engorgement of the venous plexuses
2. Redundant mucosa
3. Lax matrix

⇒ So it's a disease of 3 components !

Hemorrhoids are NOT ONLY vessels but also mucosa that become redundant & loose matrix due to separation from underlying tissue & vessels that contain blood.

RISK FACTORS

- Constipation / Straining
- Pregnancy
- ↑ Pelvic Pressure (Ascites/Tumor)
- Portal HTN

NOTES

① Bleeding is NOT spontaneous, it's traumatic - related to defecation !

② Hemorrhoidal d. should be Painless UNLESS complicated by inflammation or thrombosis

INCIDENCE

♀ = ♂

S & S

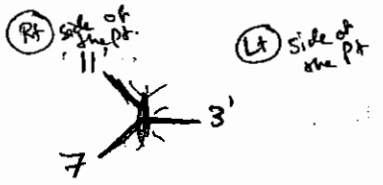
- BLEEDING (usually fresh blood) - Major Sx after defecation
- Anal mass / Prolapse
- Itching
- Pain

ALWAYS rule out colon CA in lower GI bleeding & hemorrhoids

→ this is 2ry hemorrhoids (if it's due to colon CA)

SITES — When examined in the Lt lat. Position

- Rt ant. (11 o'clock)
- Rt Post. (7 o'clock)
- Lt lat. (3 o'clock)



* Not always Pts are presented like this.

CLASSIFICATION — According to their site in lithotomy Position

GRADE I (1st degree) — NOT Prolapsed

GRADE II (2nd degree) — Prolapsed ⓦ defecation BUT returns spontaneously

GRADE III (3rd degree) — Prolapsed ⓦ defecation & reduced ONLY manually (do NOT return spontaneously)

GRADE IV (4th degree) — Prolapsed & IRREDUCIBLE!

* Soiling can occur esp: if hemorrhoids are always outside & some minimal incontinence can occur esp. mucous. → which leads to Pruritis bec it makes the area wet.
  — so one of the common presentations is Pruritis Ani.

COMPLICATIONS

- Thrombosis
- Ulceration
- Infxn.

Dx

- Hx
- P/E
- Anoscopy / Proctoscopy / Sigmoidoscopy.

DDx

- Anal CA or any CA
- Polyp
- Anal melanoma
- Anything that causes masses.

tt
- conservative
- outpt Procedures] Grade I/II
- Surgery — Grade III/IV

CONSERVATIVE

Indx: 1st & 2nd degree hemorrhoids

- High-fiber diet / Bulk-forming agents & laxatives.
  (to ↓ shearing & trauma to mucosa → ↓Bleeding)
- Topical Hygiene
- Sitz baths (warmth relaxes muscles)

OUTPATIENT PROCEDURES — If refractory to medical tt

- **Rubber band ligation** (usually anesthetic is NOT necessary for internal hemorrhoids).
- Injection sclerotherapy
- Cryotherapy
- Infrared Photocoagulation

SURGERY

- Anal dilation (Not used normally)
  — was previously done on complicated hemorrhoids
- Hemorrhoidectomy:

*AVOID removing too much skin causing anal fibrosis & stenosis.

→ Closed vs. Open
- Closed: "Closes" the mucosa w/ sutures after hemorrhoid tissue removal.
- Open: leaves "mucosa" open".

→ Complications
- Exsanguination — Bleeding may pool proximally in lumen of colon w/out any signs of external bleeding.
- Pelvic infxn — may be extensive & Potentially fatal!
- Incontinence ← Injury to sphincter complex.
- Anal stricture

→ CI for hemorrhoidectomy:
Crohn's disease

The End. (3)

ANAL FISSURE

DEFINITION

Tear or fissure in the anal epithelium. (Anoderm)

PATHOPHYSIOLOGY

Hypertonic (Hyperactive) Internal sphincter. is the usual Primary Pathology, aided by other mechanisms: trauma of hard stool (constipation)

⇒ This will go into vicious cycle: Pain → spasm → constipation.

CAUSES

- Hard stool Passage (Constipation)
- Hyperactive sphincter.
- Disease process (ex. Crohn's disease)

* Anal fissure is located at the **anoderm**, so it's VERY PAINFUL condition & this Pain is due to Stimulation by feces.

ACUTE fissure is a **tear**
CHRONIC fissure is an **ulcer**
*The time needed for an acute ulcer to become chronic is ~1month

↳ Signs of chronicity:
① Sentinal pile
② Hypertrophic anal Papilla

SITE

- **Posterior** fissure.

* Ant. fissures are more commonly seen in ♀ **BUT** still they are less common than Post. ones.

* **LATERAL** fissures are usually seen in Pts w Chron's d. / UC / TB

S&S

Anal Fissure is ALMOST ALWAYS related to defecation.

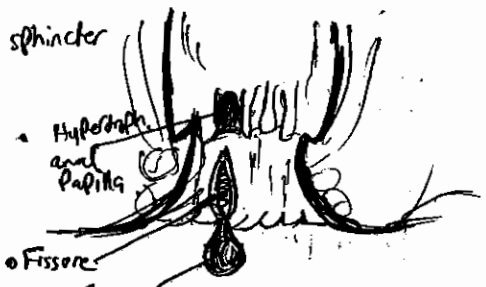
Sx
- **PAIN** in the anus
- Painful bowel movement.
- Rectal bleeding — usually minimal. — appears as **streaks** in acute phase

Signs
- Blood on toilet tissue after bowel movement.
- Sentinal tag.
- Tear in the anal skin.
- PAINFUL PR exam.
- Sentinal Pile / Hypertrophic Papilla

Constipation is related to anal fissure bcz the Pt will be afraid to go to the toilet.

Anal fissure triad for a Chronic fissure:
① Fissure / Hypertonic sphincter
② Sentinal pile
③ Hypertrophied anal Papilla

Chronic ≡ > 1 month



* Chronic fissure is a cause of submucosal fistula which is NOT cryptogenic — & this fistula is NOT treated w/ fistulotomy & sphincterotomy.

● Diseases that must be considered w/ a CHRONIC anal fissure:
- Crohn's disease / UC
- Anal CA
- STD
- AIDS

~~tt~~ / Conservative
Surgical

NEVER treat anal fissure in IBD pt surgically!

- **CONSERVATIVE**
  - High-fiber diet
  - Stool softners & laxatives
  - Local analgesia
  - Sitz baths
  - Anal hygiene.

RULE of 90% for anal fissures
90% occur Post.
90% heal w/ medical ~~tt~~ alone.
90% of pts who undergo LIS heal successfully.

- **SURGERY**

Indx: Chronic fissure refractory to conservative ~~tt~~.

≡ Lateral internal sphincterotomy (LIS) — cut the internal sphincter to release it from spasm.

± Excision of the piles (if present)

Sinha Ghanta
The End.

PERIANAL SUPPURATION (ABSCESS, FISTULA)

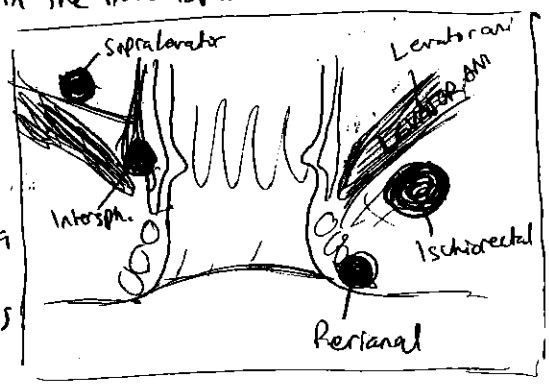
Source: Dossier Recall

ANORECTAL ABSCESS

TYPES
- Perianal
- Ischiorectal
- Intersphincteric
- Supralevator

DEFINITION

Obstruction of anal **crypts** w/ resultant bacterial overgrowth & abscess formation w/in the INTERSPHINCTERIC space.



RISK FACTORS

- Constipation / Diarrhea / IBD
- Immunocompromise
- Hx of recent surgery / trauma
- Hx of colorectal CA
- Hx of prev. anorectal abscess

S&S

- **Rectal Pain**: often of sudden onset / throbbing / continuous / ↑ w/ defecation or coughing
- Drainage of Pus.
- Fever / Chills
- Malaise
- Leukocytosis
- Tender perianal swelling w/ signs of inflammation.

If pt is severely diabetic, horrible **necrotizing** soft tissue infxn may follow; WATCH him closely!

Indx of PostOp. IV abx for drainage:
- ~~Recurrence~~
- Cellulitis
- DM
- Immunosuppression
- Heart valve abnormality
- Sepsis
- Leukocytosis

Tx

Surgical **drainage**

Complications

- May extend upward (will become supra-levator abscess)
- Fistula.

NOTE: 50% of pts w/ abscess will develop a fistula in/ano during the 6 m. after surgery!

ANORECTAL FISTULA

DEFINITION

Anal fistula, from rectum to perianal skin.

CAUSES

usually anal crypt / gland infxn (usually perianal abscess)

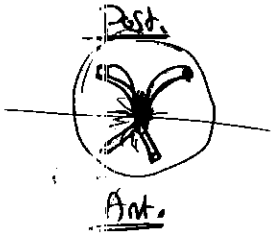
S&S

- Perianal drainage
- Perirectal abscess (recurrent)
- "Diaper rash" / Itching

Dx

- P/E
- Proctoscope

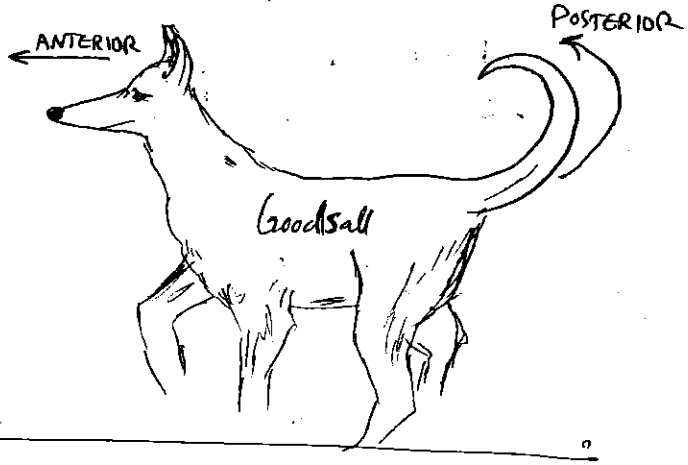
Goodsall's Rule



Fistulas originating **ANTERIOR** to a transverse line through the anus will course **STRAIGHT** ahead & exit ant. whereas those existing **POSTERIORLY** have a **CURVED** tract.

Remember it like this! (Surgical Recall)

THINK of a dog w 4 STRAIGHT nose (Ant.) & CURVED tail (Post.)



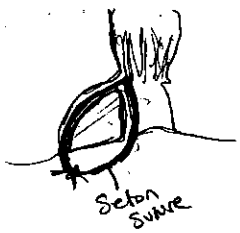
Mgt

Intraop.
To find the internal opening of anorectal fistula, we inject H_2O_2 (or methylene blue) in ext. opening — then we look for bubbles (or blue dye) coming out of it.

1. Define the anatomy
2. Marsupialization of fistula tract (i.e. fillet tract open)
3. Wound Care: routine sitz baths & dressing changes
4. Seton Placement if fistula is through the sphincter muscle.

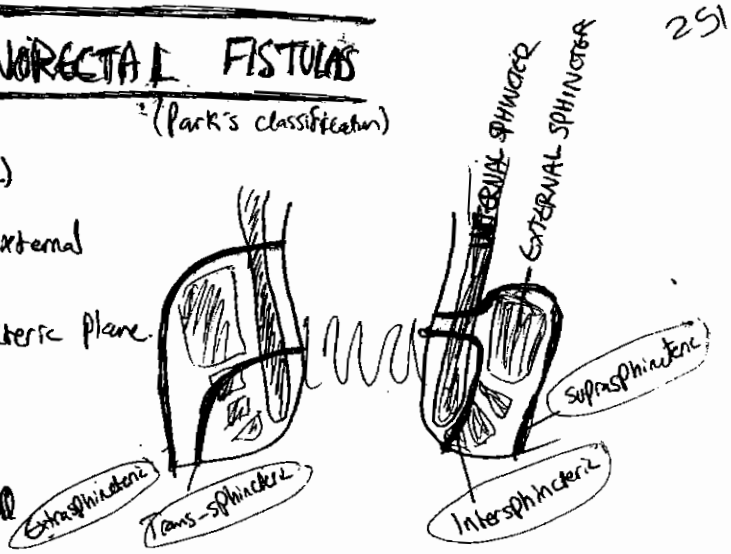
Sitz Bath
Sitting in a warm bath (usually done after bowel movement)

Seton: Thick suture placed through fistula tract to allow slow transection of sphincter muscle
⇒ Scar tissue formed will hold the sphincter muscle in place & allow for continence after transection.



CLASSIFICATION OF ANORECTAL FISTULAS

(Park's classification)



• INTERSPHINCTERIC (m.c)

- Does NOT cross the external sphincter.
- located in the intersphincteric plane.

• TRANS-SPHINCTERIC

- It crosses BOTH internal & external sphincters & it passes through the ischiorectal fossa to reach the skin of the buttock.

• SUPRA-SPHINCTERIC – v. rare

- usually iatrogenic
- difficult to distinguish from high-level trans-sphincteric (BUT Mgt is similar)

The primary track of fistula may have also secondary tracks arising from it.

• EXTRASPHINCTERIC

- Runs w/out specific relation to the sphincters & usually results from Pelvic d. or trauma.

The End
Nada Ghaith

OTHER ANORECTAL DISEASES

PILONIDAL DISEASE

DEFINITION

A cystic inflammatory process generally occuring at or near the cranial edge of the gluteal cleft.

*The term of Pilonidus is Latin;
'Pilo' ≡ Hair
'Nidus' ≡ origin
indicating that it's ass. w/ hair follicle.

Results from trauma to hair follicle → infxn.

INCIDENCE

m.c in ♂, young (20's-30's)

SYS

Either presents

ACUTELY as an abscess (Fluctuant mass)

or CHRONICALLY as a draining sinus w/ pain at the top of gluteal cleft.

Tt

Incision & drainage under local anesthesia w/ removal of involved hairs.

PERIANAL WARTS

DEFINITION

Warts around the anus / perineum

CAUSE

Condyloma acuminatum (HPV)

*THE MAJOR RISK is SCC (Squamous cell CA)

Tt

If small → Topical Podophyllin

If Large → Surgical resection or Laser ablation.

The End.

ANAL CANCER

* The m.c CA of anus is → **SCC** _80%

Anal CA
- Anal SKIN CA (Anal margin tumors)
- Anal Canal CA
  - Epidermoid
  - Malignant Melanoma

TYPES OF ANAL CA

- Squamous Cell CA (SCC) — 80%
- Cloacogenic (Transitional cell)
- Adenocarcinoma / Melanoma / Mucoepidermoid

INCIDENCE

Rare! 1% of colon CA incidence.

RF

- HPV / condyloma / Herpes.
- HIV
- Smoking
- Multiple sexual Partners / Anal intercourse
- Immunosuppression.
- Chronic inflamm. (Fistulae / Crohn's)

2 Carcinoma in situ of the Perianal Skin:-
1. **Bowen's disease**: Squamous cell CA in situ
2. **Paget's disease**: Adenocarcinoma in situ

↳ of BOTH: Wide local excision

SYS

- Anal BLEEDING
- Pain
- Mass
- Mucus Per rectum
- Pruritis

Margin CA: Anal verge out 5 cm onto Perianal skin.

Canal CA: Prox. to anal verge up to the border of int. sphincter.

* 25% of Pts w Anal CA are asymptomatic.

Dx

- Hx & PE
- Surgical **bx** w histopathologic evaluation.

Sites of Metz
- L.N
- Liver
- Lung
- Bone

Remember! Lymphatic drainage below dentate line is to inguinal L.N.

* Histology
  - Anal margin tumors (Anal skin): SCC / BCC / Bowen's / Paget's d.
  - Anal canal tumors: epidermoid (SCC or transitional cell CA) / Malignant Melanoma.
* Clinical Staging — hx / PE / Proctocolonoscopy / Abd. or pelvic CT or MRI / CXR / LFT / transanal U/S

⇒ Most pts w anal CA are diagnosed LATE! ∩ — often missed!

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Tt based on "NIGRO Protocol"

- If Anal **canal** epidermal CA → • CTX (5-FU & mitomycin C)
  • RTX
  • Postradiation therapy scar bx (6-8 wks post RTX)

(Px)

90% of pts have complete response

5-yr survival w NIGRO Protocol is 85%

* If local recurrence of anal CA after NIGRO Protocol, Repeat CTX & RTX or Salvage APR.

- If Anal **margin** CA — if Small (<5cm): Surgical excision w 1-cm margins
  Large (>5cm): CTX

- If Anal **Melanoma** — Wide local excision or APR (esp. if large) ± RTX / CTX (Postop.)

(Px)

5-yr survival < 10% ☹

NOTE

* In Anal canal tumors, Local excision is NOT an option! — CTX & RTX are often successful.
  * APR is done only if F/U bx indicates residual tumor.

In pts w anal melanoma, ONLY 1/3 of them have an **amelanotic** anal tumor (Pink in color)
⇒ Thus making dx difficult w/out Pathology!

Good luck
The End.

COLON CA

Risk Factors
- Age > 50
- Adenomatous polyps (current/past)
- IBD (UC/CD)
- Smoking
- Obesity
- Acromegaly
- BRCA1 mutation.

Hereditary & Environmental RF
- 1st degree relatives w/ colon CA or adenomatous polyps
- Familial Polyposis (FP) Synd.
- Diets (↑ in calories & fat) ↓ in
- Hereditary non-polyposis colon CA (HNPCC)

* Life-time risk of colon CA is 4% in avg.-risk persons.

Diagnostic Flags
- Wt loss / Anorexia
- Fever
- +ve Heme Stool
- Anemia
- Δ in bowel habits (esp. **nocturnal** stools)
- Onset of Sx after age 45.

Remember!
Endocarditis caused by Strep. Bovis or C. Septicum is often ass. w/ colon CA.
∴ So do GI workup in these Pts

Polyps
- Hyperplastic Polyps — NO ↑ risk of CA; NO features of dysplasia
- Tubular Adenomatous Polyps — ↑ Risk of CA
- Villous Adenomatous Polyps — ↑ Risk of CA

* Most GI CA arise from adenomas
* 25% of colorectal CA are located beyond the splenic flexure.

Aspirin & Colon CA
NSAIDs (COX II inhibitors)

- Low-dose Aspirin (81 mg) causes mild ↓ risk of recurrent ADENOMAS BUT NO. ↓ risk of Colon CA
- Full-dose Aspirin ↓ risk of colon CA

* * *

Protective effect of Aspirin is related to:
- Dose of ASA
- Freq. of use / wk
- Duration of use (yrs)

~30% of pt > 40 yrs old have adenomatous polyps BUT only 1% become malignant.

Adenomas w/ "Advanced" features: (likely to develop into CA)
- High-Grade dysplasia
- Villous histology
- Size > 1 cm

Guidlines for screening adenoma

FINDINGS	COLONOSCOPY
1 or 2 small tubular adenoma w/ low-grade dysplasia	Repeat colonoscopy 5-10 yrs after polypectomy
• 3-10 adenomas • or 1 adenoma >1 cm • or any villous feature • or High-grade dysplasia	Repeat colonoscopy in 3 yrs
>10 adenomas	Repeat in <3 yrs
Pts w/ sessile adenoma that are removed	Repeat in 2-6 mo (to verify complete removal)

• Hyperplastic Polyps, if < 1cm (except those w/ hyperplastic Polyposis s have the same F/U as no polyp (10 yr

• ONLY adenomatous polyps needs specific F/U (see table)

Malignant Potential vs. size

	<1cm	1-2cm	>2cm
Tubular	1%	10%	35%
Mixed (TV)	5%	10%	45%
Villous	10%	20%	55%

INHERITED COLON CA

Lynch I early onset + po
Lynch II same ↑ risk of other cancers

POLYPOSIS (Familial Polyposis Syndromes)

TYPES

Adenomatous

① **Familial Adenomatous Polyposis (FAP)**
- 100% risk of CA if not treated! 3rd
- Need proctocolectomy by age 20!
- * Hundreds of adenomas in colon, always involved
- * Tend to have duodenal polyps
- * ↑ risk of 2ry CA (Ampullary adenoma/CA)
- * Giant stomach tumors are common BUT benign

② **Gardner Synd.** — Variant of FAP w/ more extraintestinal benign growths
- * Also 100% risk of CA if untreated
- dermoid cysts, osteomas Bone lesions Osteomas + soft tissue tumors. + dental abnormalities

Hamartomas

③ **Peutz-Jeghers Synd.** ↑risk of CA (~50% risk by age 60)
- multiple hamartomatous Polyps. ↑ SI
- + melanotic Pigmentation on lips + buccal mucosa, freckles
- mc c/p is abd. Pain d/w to intussusception or bowel obst by large polyp.

④ **Juvenile Polyposis** — No malignant Potential.

NON-POLYPOSIS (Hereditary Non-Polyposis Colon Ca-HNPCC)

* Pts do NOT have Familial Polyposis

AKA: Lynch Syndrome.

(CRITERIA)
It's the occurance of colon CA in
at least 3 1st-degree relatives over
at least 2 generations, & w/
at least 1 person diagnosed <age 50
THINK 3, 2, 1!

* ♀ w/ HNPCC have ↑risk of Ovarian & endometrial CA. (also renal/ureteral stomach pancreas

* Start Screening at age 25

⑤ Turcot's syndrome
AR
polyps + Cerebellar med/all oblastoma, glioblastoma.

Screening for colon CA
- Fecal occult Blood Testing (FOBT)
- Colonoscopy (or flex. sigmoidoscopy + Barium enema) ↓ less desirable
- CEA

• In general, If low-risk pt ⟶ do multiple FOBT (/Year)

*Tests that detect adenomatous Polyps & CA
- Colonoscopy / 10 yrs
- or Flexible sigmoidoscopy / 5 yrs
- or Double contrast barium enema / 5 yrs
- CT colonography / 5 yrs

*Tests that detect CA
- Annual Fecal immunochemical test
- Annual guaiac-based Fecal occult blood
- Stool DNA test (interval uncertain)

⟹ If Polyp is benign, repeat every 3 yrs.

*Any +ve test (other than Colonoscopy) should be followed up by colonoscopy ∵ ↑ Rx of any polyp/ adenoma

FOBT Annually

+ve in about 2% (varies ∵ age; >5% after age 60)
↳ 2% of it have colon CA

*Poor screening test! But quick & cheap, also least invasive

-ve in up to 66% of pts ∵ colon CA
It can miss ⅓ of advanced colon CA!

** Full FOBT series: use 6 Hemoccult

Even If only one FOBT is +ve ⟶ do colonoscopy
(Alternative: -Flex. sigmoid + Air-contrast BE) But less desirable

COLONOSCOPY
- has the highest yield of finding Polyps & CA
- It's the screening procedure of choice!

CEA (CarcinoEmbryonic Antigen)
good only in checking for recurrence of CA

CEA also ↑ in smokers, pts ∵ benign biliary d., PSC or IBD.

* The "10 yr" Rule

Increased-risk Pts: Colonoscopy should be at age 40 yrs
or 10 yrs before age at which index case is diagnosed

ex. • Start at age 40 if a 1st relative was dx with an adenoma/ at age 52 yrs
• Start at age 20 if several 1st degree relatives had Colon CA at age 30.

Indx for Colonoscopy

- Occult blood
- Abnormal barium enema
- Adenomatous Poly
- FP synd. / HNPCC
- Hx of colon CA
- 1st degree relative with colon CA
- Unexplained IDA
- Gross lower GI bleeding (except if bright red in young Pt)
- IBD
- Strep. Bovis or C. septicum bacteremia
- 4-8 wks after new-onset diverticulitis (to r/o CA)
- Persistent diarrhea with -ve blood tests & not meeting the criteria for dx of IBS

Pattern of spread
- Direct: circumferentially bowel wall - abdomen.
- hematogenous: Portal system → liver mets; systemic → lungs
- lymphatic, transperitoneal & intraluminal

CT → for detecting liver metastasis.

STAGING

- TNM (Preferred)
- Duke's: A, B, C, D (cut from abdomen... / L.N / Distant)

STAGE I: T_1 (submucosa) or T_2 (muscularis), N_0, M_0

STAGE II (a/b/c): T_3 (to tissues), or T_4 (visceral peritoneum/invasive), N_0, M_0

STAGE III: T_1 — T_4 (any), N_1, M_0

STAGE IV: Any T, Any N, M_1

Metz: Always to liver first (via Portal circulation) BUT if it involves only the rectum, it will bypass Portal circulation (mets won't liver)

TREATMENT

* 1st option → Surgical resection (potentially curative)
  - Recurrence due to micrometz
* Adjuvant CTx (5-FU) effective only for stage III or locally advanced II
* RTX (Prior to surgery) is helpful for rectal lesion only.
* Hepatic resection ↑ survival with solitary liver metz.

The End
Saah Gilbert

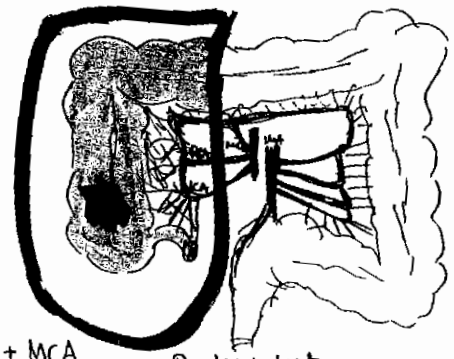
OPERATIVE MANAGEMENT OF COLORECTAL CA

Source : First Aid 261

RIGHT HEMICOLECTOMY

- **Resected Material**
  - Terminal ileum
  - Cecum
  - Asc. colon
  - Proximal transverse colon

  PLUS
  - Resection of RCA + ICA ± MCA
  - Removal of fat & L.N
- **Indx**
  - Rt colon CA
  - Cecum CA



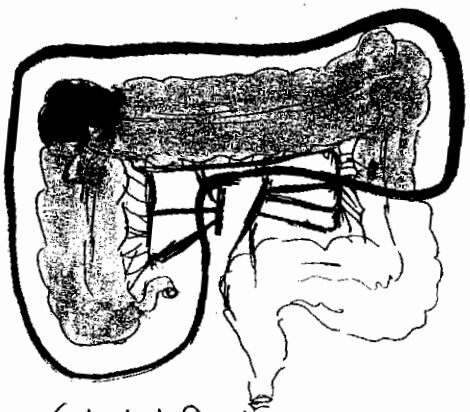
Rt Hemicolectomy

RCA : Rt Colic Art.
ICA : Iliocecal Art.
MCA : Middle Colic Art.
LCA : Lt Colic Art.

EXTENDED RIGHT HEMICOLECTOMY

- **Resected Material**

  Same as Rt hemicolectomy
  + Remainder of transverse colon & splenic flexure.
  - Resection of RCA + ICA + MCA
- **Indx**
  - Hepatic flexure CA
  - Transverse colon CA (Prox./Mid)

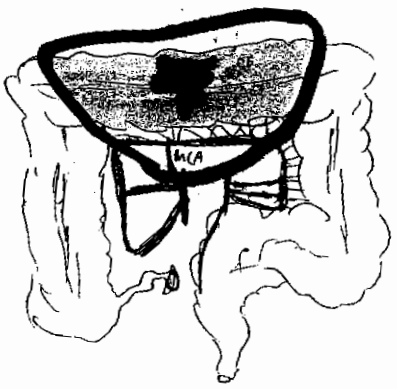


Extended Rt Hemicolectomy

TRANSVERSE COLECTOMY

- **Resected Material**

  Transverse colon
  + Middle colic art. (MCA)
- **Indx**
  - Transverse colon CA

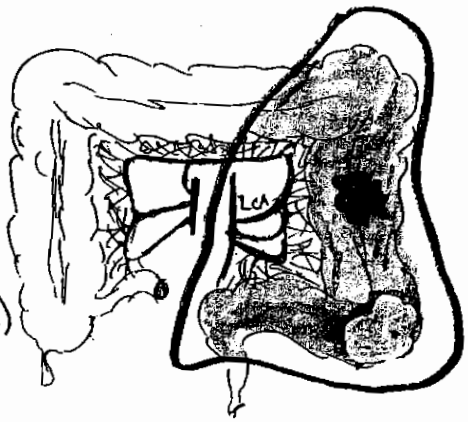


LEFT HEMICOLECTOMY

- Resected Material
  - Desc. colon
  - Left colic art. (LCA)

  PLUS

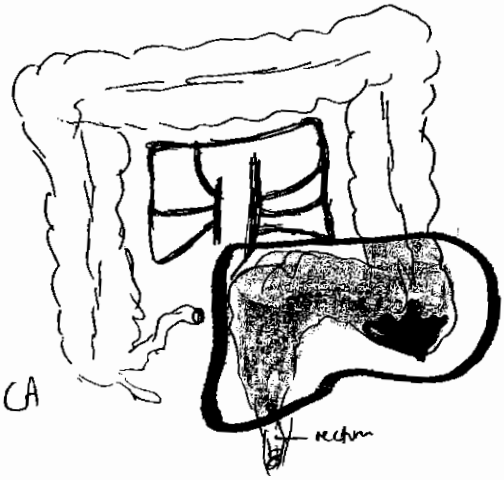
  Resection of L CA (Lt Colic Art.)



- Indx
  - Splenic flexure CA
  - Left Colon CA

SIGMOID COLECTOMY

- Resected Material
  - Sigmoid CA
  - Sigmoidal art.



- Indx
  - Sigmoid / RectoSigmoid CA

TOTAL COLECTOMY

- Removal of the entire colon **Wout** the rectum

PROCTOCOLECTOMY

Procto = Rectum

- Removal of the entire colon & Rectum.

SUBTOTAL COLECTOMY

- Removal of part of colon / all of the colon Wout complete resection of the rectum.

LOW ANTERIOR RESECTION (LAR)

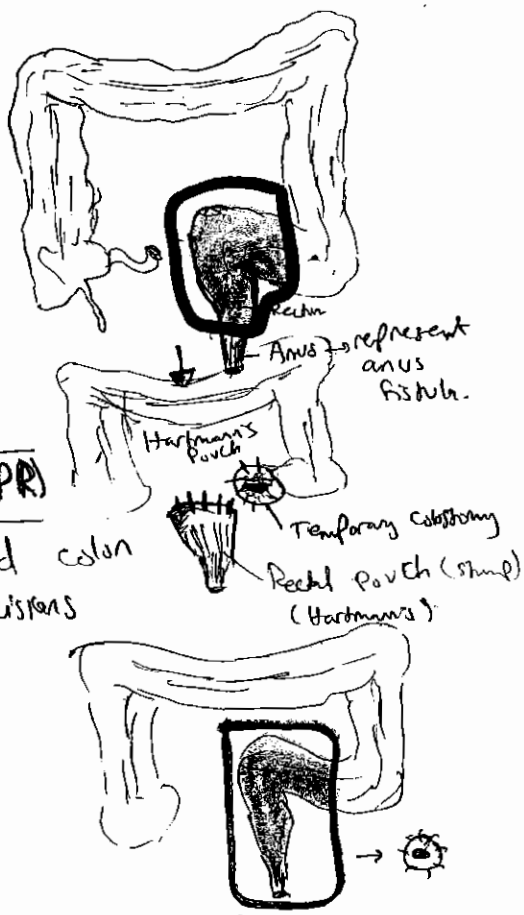
Resection of **LOW** rectal tumors through an **ANTERIOR** approach

Hartmann's Procedure
1. Proximal colostomy
2. Distal stapled-off colon/rectum that is left in peritoneal cavity

- **Indx**
  - **Proximal** Rectum CA
- **Criteria**
  - Tumors >4 cm from anal verge (w/ distal intramural spread <2cm)
  - Must be able to get 2-cm margin

*Includes total mesorectum excision

- **Complications**
  - Incontinence
  - Urinary dysfxn
  - Sexual dysfxn
  - Anastomotic leak (5-10%)
  - Stricture (5-20%)



ABDOMINAL-PERINEAL RESECTION (APR)

- Removal of the rectum & sigmoid colon through abdominal & perineal incisions (Pt is left w/ a colostomy)

- **Indx**
  - **Distal** Rectum CA
  - Anal CA

* Done in tumors **NOT** fitting criteria for LAR.
* The anus is closed.
* PERMANENT colostomy!! (due to removal of the **ANUS**.

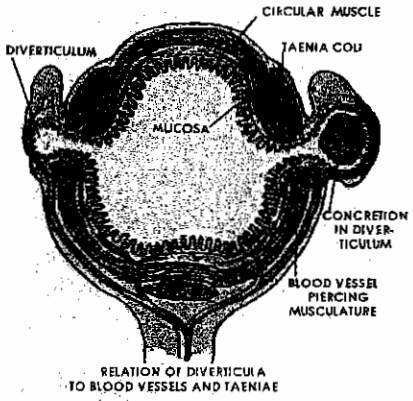
- **Complications**
  - Stenosis
  - Retraction or prolapse of ostomy
  - Perineal wound infxn.

The End

DIVERTICULAR DISEASES:

Introduction:

- Colonic Diverticula are **false diverticula** in which mucosa and submucosa protrude through the muscularis propria (not all the layers!)
- Diverticulosis is just the presence of outpouchings without inflammation, Diverticulitis is if they become inflamed.
- **Outpouchings** occur along the mesenteric aspect of the antimesenteric tenia where arterioles penetrate the muscularis
- The sigmoid colon is MC affected (due to decreased luminal diameter and increased luminal pressure)



Risk Factors:

- Low-fiber diet
- Elderly (incidence increases with age- 75% prevalence by age 80)
- Chronic constipation
- Family history

DIVERTICULOSIS:

- **MC** cause of lower GI bleeding
- **Sx:** Asymptomatic (80% of cases!), Bleeding, Diverticulitis and Complications
- Bleeding, and may be **massive** because the media of the perforating artery adjacent to the colonic diverticulum may become attenuated and eventually erode
- Bleeding is bright red and NOT associated with previous melena or chornic blood loss
- Bleeding most often from Left colon

Management of Diverticulosis:

- **If Asx**- a high-fiber diet is recommended
- **If bleeding,** although it may be massive, it is usually self-limited (80% spontaneously stop). Resuscitation with fluids.
- **Preform** colonoscopy 6 weeks after inflammation (but not during attack, due to risk of perforation) to rule out colon cancer as a cause of bleeding.

When is surgery indicated in diverticulosis?

- **Elective Resection** of the affected colonic segment
  - Pts with reccurent bleeding
  - Need for long term anticoagulation
  - Excessive blood loss cannot be tolerated
- **Urgent Resection** of the affected colonic segment
  - Active ongoing bleeding (>6 Units Packed RBCs/24 hours)

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DIVERTICULITIS: Infection or Perforation of a diverticulum

Pathophysiology: Obstruction of diverticulum by a fecalith leading to inflammation and microperforations, leading to fecal extravasation and subsequent peridiverticular and pericolic inflammation.

Epidemiology: Occurs in 10-25% of patients with diverticula (90% left-sided, 10% right-sided)

Presentation:

- **LLQ pain**, may radiate to suprapubic area, left groin or back, cramping or steady pain
- **Fever, Altered bowel habits (diarrhea), Urinary urgency** or dysuria, Nausea, vomiting
- **P/E:** varies with the severity of the disease, but the MC is LLQ tenderness. A mass may suggest abscess or phlegmon

Classification:

1. **Uncomplicated Diverticulitis:**
   - Only inflammation (LLQ pain), usually resolve without surgery
2. **Complicated Diverticulitis**
   - Diverticulitis with abscess, obstruction, diffuse peritonitis, fistulas.
   - **Hinchey classification** used to assess severity (mentioned below)

Diagnosis:

- **CT scan:** may find segmental colonic thickening, swollen edematous wall, focal extraluminal gas, helpful to dx abscess formation.
- **CBC:** high WBC
- **NOT indicated:** sigmoidoscopy (due to risk of perforation), contrast enema (risk of barium/fecal peritonitis)

DIVERTICULITIS MANAGEMENT depends on whether it is an uncomplicated or complicated attack, and whether it is a first attack or not.

First *uncomplicated* attack of diverticultis should be treated conservatively, while *complicated* attacks (**complications include: abscess, fistula, peritionitis, perforation, obstruction**) need operative management according to each complication.

Conservative Management:

1. Bowel rest
2. Clear liquids for 2-3 days then advance diet as tolerated
3. IV fluids
4. Antibiotics
    a. IV antibiotics that cover G –ve/Anaerobes for 3-5 days then switch to oral to complete 10-14 day course
    b. Either Monotherapy: Ticarcillin-Clauvalanate OR Piperacillin-Tazobactam OR Ampicillin-sulfabactam
    c. Or Rocephin (Ceftriaxone) + Flagyl (Metronidazole)
5. May Include Percutaneous drainage of abscess

- After successful conservative Rx of 1st episode, 1/3 have a 2nd attack, and 1/3 of those who have a 2nd attack have a 1/3 attack.

Surgery Indications:

1. After first or any COMPLICATED diverticulitis attack
2. After 2 or more episodes of uncomplicated
   *Mgmt is always individualized according to patient, these are general guidleines

To assess Severity, Degree of Peritoneal contamination (which determines pre-op antibiotics and appropriate intervention), the Hinchey Classification (and in 1999, Modified Hinchey Classification) was developed:

HINCHEY CLASSIFICATION:

1. Stage 1: pericolic or mesenteric abscess:
2. Stage 2a: distant abscess:
   Stage 2b: complex abscess and fistula:
3. Generalized purulent peritonitis
4. Generalized fecal peritionitis

Stages 1 and 2 can be treated conservatively during attack, with percutaneous drainage of abscess. After the attack has resolved, an elective laparoscopic resection of diseased segment with primary # and stoma. They can receive bowel prep pre-op.

Stage 3 and 4 need EMERGENT operative treatment usually done in 2 stages. First, Hartmann procedure (= resection of the diseased colon, an diverting end-colostomy, and creation of a rectal stump). This is followed by colostomy closure three months later

IN SUMMARY,

- **Mild Diverticulitis** treated as an outpatient, with a clear liquid diet and broad-spectrum oral antibiotics for 10 days
- **Severe diverticultis:** complete bowel rest, IV fluids, narcotic analgesics, broad-specreum IV antibitoics.
- **After episode:** a high-fiber low residue diet should be resumed. Fiber supplements and stool softeners should be given to avoid constipation.

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POSSIBLE COMPLICATIONS of DIVERTICULITIS:

DIVERTICULAR ABSCESS:

- Usually identified on CT Scan
- A percutaneous drain should be placed under radiologic guidance- which avoids immediate operative drainage, and allows time for the inflammatory phlegmon to be treated.
- Treat with IV antibiotics
- Thus, a one-stage procedure can be done ☺ (instead of 2 or 3 stage!)

GENERALIZED PERITONITIS:

- RARE, results if diverticular perforation leads to widespread fecal contamination.
- In most cases, **resection of the diseased segment** is possible, and a **Hartmann Procedure** is done. The colostomy later closed (2-stage procedure)

- Another option for a patient without significant fecal contamination: sigmoidectomy + colonic lavage +colorectal anastomosis +/- loop ileostomy

FISTULIZATION:

- Fistulas between colon and other organs may occur secondary to diverticultis

- **COlOVESICAL fistulas are the MC, and diverticulitis is the MCC of colovesical fistuals!**

- Colovaginal and colovesical fistulas usually occur in women who have previously undergone hystectomy
- Colocutaneous and coloenteric fistulas are uncommon

- **Colonoscopy** should be done **after 6 weeks** to rule out other causes of fistulas.

COLONIC VOLVULUS

Source: Recall 26th

DEFINITION

Twisting of colon on itself about its mesentery
⇒ Resulting in obstruction ± — if complete, vascular compromise ⇒ Potential necrosis, Perforation or Both.

TYPES

- **SIGMOID** Volvulus (m.c) — 75%
- Cecal volvulus — 25%
- Transverse volvulus. (RARE!)

SIGMOID VOLVULUS

Incidence 75% (THINK: **S**igmoid = **S**upra)

RF

- High fiber diet
- elongated colon.
- Chronic constipation
- Laxative abuse
- Pregnancy.
- hx of abd. surgery or distal colonic obst.

SxS

- Acute abd. Pain
- Progressive abd. distention
- Anorexia
- **Obstipation**
- Cramps
- N/V

AXR FINDINGS

Distended loop of Sigmoid colon
- Classic "Omega" sign / "Coffee-bean" sign
- loop aiming **toward** RUQ

Signs of Necrotic bowel in colonic volvulus:
- Free air
- Pneumatosis (Air in bowel wall)

Dx

- Sigmoidoscopy or radiographic exam. w/ gastrografin enema.
  - ↳ If sigmoidoscopy & plain films fail to confirm dx — "Bird's beak" is Pathognomonic seen on enema contrast study.

SIGNS of STRANGULATION

- Discolored / hemorrhagic mucosa on sigmoidoscopy
- Bloody fluid in rectum.
- Frank ulceration / necrosis at the point of twist.
- Peritoneal signs
- Fever / Hypotension / ↑WBC.

Tx:

Initially — (Non-operative)

If there's no Strangulation — Sigmoidoscopic reduction is successful in ~85% of cases. (enema will reduce only ~5%)

- Recurrence → ~40%!!

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EMERGENT surgery!

Rt colectomy w Irg anastomosis or ileostomy & mucus fistula (Irg # may be done in stable pt)

NOTE

* Pts w cecal volvulus require SURGICAL reduction while the vast majority of pts w Sigmoid volvulus undergo initial ENDOSCOPIC reduction of the twist.

* Transverse volvulus is VERY RARE.

* Gastric volvulus can occur.

The End.

Indx of SURGERY

- If strangulation is suspected
- Unsuccessful reduction

* Most pts undergo resection after successful non-operative reduction due to ↑recurrence rate (~50%)

CECAL VOLVULUS

Incidence ~25% (less than sigmoid)

CAUSE
- Idiopathic
- Poor fixation of the Rt colon
- hx of abd. surgery

S+S
- Acute onset of abd. & colicky Pain.
  - starting in RLQ & Progressing to a constant Pain
- Vomiting
- Obstipation.
- Abd. distention
- SBO

Dx
- AXR. — dilated ovoid colon w/ large air-fluid levels in RLQ "coffee-bean" sign w/ apex toward epigastrium/LUQ
  (Must r/o gastric dilation w/ NG aspiration)
- Water-soluble contrast study — if dx can not be made on AXR.