# Gastrointestinal Surgery

## Esophagus: (retroperitoneal)

* Note: naturally esophagus is always collapsed & opens only when we swallow food.
* normal eoso. pH: 6-7 (alkaline).

**Physiology:** function = swallowing.

1. UES: cricopharyngeus muscle. (upper esophageal sphincter) → it's a true sphincter cuz it's formed by a muscle (skeletal).
2. Body: peristalsis
3. LES: not a true sphincter. 3-5 cm length. Tone: 10-30 mmHg (it's always contracted = Tonic contraction).
   ↳ pressure formed from contraction.

**Anatomy:**

1. Length: 20-25cm (10 inches)
2. Blood supply: upper 1/3: inferior thyroid, middle 1/3: aorta and bronchial, lower 1/3: left gastric and inferior phrenic.
3. Nerve supply: Vagus nerve: excitatory.
4. Upper 1/3 is skeletal, distal 2/3 is smooth muscle (involuntary).

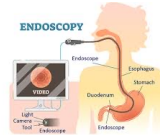
* Dysphagia: difficulty swallowing

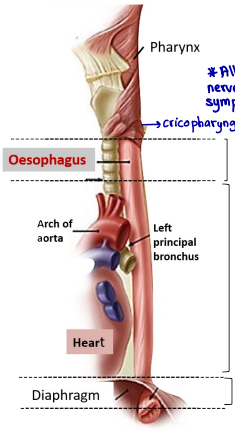
5. Mechanical/obstructive: to solids initially then progresses to liquids. DDx: (1) Cancer, (2) strictures, (3) diverticula, (4) webs, (5) rings, (6) esophagitis, (7) foreign body.
6. Motility/functional: both solids and liquids. DDx: (1) achalasia (no peristalsis, unrelaxed LES), (2) DES (synchronous non-peristaltic contraction, normal LES), (3) scleroderma (no peristalsis and low tone in LES).
   (1) Cuz tone↓

**Imaging of esophagus:** 'Bum'

1. Barium swallow: contrast and X-ray to see lumen anatomy (1) and some function (2). If suspected perforation: **use water-soluble contrast.** Never use if risk of aspiration.
   ↳ if can't use Barium.
   cuz Barium can leak to other anatomies of the body.

2. Upper endoscopy: see anatomy, take biopsies, interventions, EUS.?
3. Manometry: diagnose motility disorders. (physiology).


* All parts are vagal nerve supplied (parasympathetic).
→ cricopharyngeous muscle (UES).

Cervical part
The only one with skeletal muscle. (voluntary). inferior thyroid.

Thoracic part
smooth muscle (involuntary). thoracic aorta + bronchial arteries.

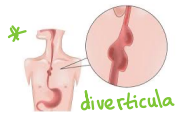
Abdominal part
smooth muscle
left gastric + phrenic.

## Dysphagia

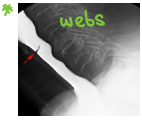
**mechanical/obstructive:** anatomical malfunction.
↓
In solids only (initially) but later progresses to liquids.
ex: cancer,

**dysmotility/dysfunctional:** physiological malfunction.
↓
In solids & liquids.
ex: spasm.

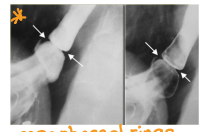
* Stricture = fibrosis in lower 3rd due to reflux esophigitis.



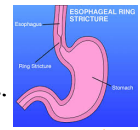
* diverticula.




* webs



* esophageal rings.




* esophigitis can distrupt tissue & cause rings & webs.

GERD: (opposite of dysphagia). gastroesophageal reflux disease.
① ②

- Excess reflux or loss of anti-reflux mechanisms. (causes).

*basically when stomach acid reaches eosophagous. (it's normal to have Gerds to a certain level, however when symptoms & irritation starts; it becomes a disease).

Anti-reflux mechanisms (organized according to imp).

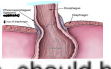
1- LES (normally always toned & relaxes intermittenly)
·(if over-relaxed-insuffeciency) ① tone <6 mmHg ./② length <2cm

2- Hiatal crura (phrenic muscle).

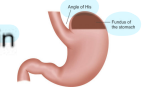
3- Intra-abdominal portion ideally >2cm

* intra-abdominal part of eos. =
● → +ve p that closes it.
● → -ve p that opens it.
* if ● is shorter than ● then ↑ risk of Gerds


4- Phreno-esophageal membrane: lax in hiatal hernia



5- Angle of His: angle between esophagus and fundus, should be acute. Underdeveloped in infants.



Clinical:

- Symptoms: heartburn (① substernal), regurgitation (② water brash), acidic taste in mouth (③), dysphagia (④), Respiratory (⑤) (reflux reaches larynx).
hoarseness (① dry), cough (②), wheeze (③), and aspiration (④). Increased with certain foods (①), lying flat (②), and stress (③).
Relieved with sitting upright (①), acid reducing agents (②): PPI, anti-H2, and antacids.

* in some cases → nausea & Vomitting.

* patient can come to you with pharyngitis/laryngitis due to reflux (لوز + لحة صوت) he thinks he has recurrent flues + chronic dry cough.

- Signs: minimal. Can see dental erosions.
Physical exam

Investigations: (we use them when history has something atypical/ a red flag that can suggest cancer (bleeding/wei. loss ...)). but normally we diagnose without them.

① Barium swallow: hiatal hernia? (always used as 1st test in eosophageal disease).
↳don't

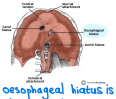
② Endoscopy: erosions, strictures, hiatal hernia, Barret (salmon-colored mucosa, do biopsy) (premalignant).
↳ for Gerds complications

③ 24-hour pH monitoring: confirm GERD. Positive if >6% of time (1.5 hours) has pH <4 Stop H2 blockers 3 days prior and PPIs 1 week prior. diag nos.
↳ of eoso.

④ - Manometry: if abnormal peristalsis, may do partial wrap instead of Nissen. **If no peristalsis, reconsider surgery for GERD.** LES abnormal if: <6 mmHg tone, length <2cm and intra-abdominal part <1 cm. (it can diagnose motility disorders).
(not diagnostic of gerds). but can diag. motility disorder).

⑤ Gastric emptying: nuclear medicine to rule out gastroparesis (N/V, bloating, early satiety). ↳nausea/vomitting. diagno stic

⑥ Impedance monitoring: can detect non-acidic reflux which cause similar damage. diagno stic



oesophageal hiatus is the area where eosophagus leaves thorax through diaphram, eosophagus there is supported by the phrenic muscle (hiatal crural). if it loses its support from that area, it will cause = hiatal hernia.

● foods that trigger it:
spicy foods + fatty foods + chocolate + coffee + smoking
● foods that relieve it:
- Salt crackers

* Barium swallow doesn't indicate Gerds it shows us it's complications ex: strictures, /hiatal hernia. or shows causes: Scleroderma..

* The only problem with 24Hr pH moni. is that it can only monitor acids (Sometimes reflux is alkaline/neutral). Impedance testing can moniter all PHS).

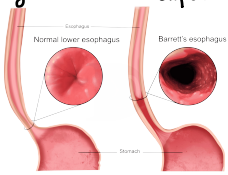
* gastric emptying → you eat a little meal that has a radioactive material, & we follow it to see how much time it takes for it to be digested.

* gastroparesis → weakness in stomach muscles leading to slow digestion & some similar symptoms to gerds.

**Barrett's esophagus:** (salmon coloured mucosa) = Adenocarcinoma.

- Intestinal metaplasia. Do serial endoscopy and biopsy.
- Seen in 10% of GERD. 30-40x risk of cancer if with high-grade dysplasia.
- GERD therapy can prevent Barret, but do not reverse it if already happened.
- Treat with ablation radiofrequency or cryotherapy. (① ② (freezing)) / endoscopic mucosal resection.

*Barrettes: (premalignant) metaplasia of cells lining ↓ esophagus. (caused by chronic acid exposure).




**Treatment of GERD:**

① Lifestyle modification: weight loss (①). Avoid chocolate (②), caffeine (③), smoking (④), avoid food before bedtime (⑤). Elevate head of bed (⑥).

② PPI (severe) and H2 blockers (mild): reduce symptoms by reducing acidity, do not stop reflux itself. (prophylaxis not treatment)

**Surgical therapy:**

① Indications: failure of medial (①), hiatal hernia (②), complications (stricture, recurrent aspiration) (③).

- Surgery mainly reduces GI symptoms.

② Complications of surgery: Dysphagia (①) and failure (②) are MC. Wrap migration (if onto stomach: slipped Nissen).
↳ from making LES too tight.

③ Nissen fundoplication: laparoscopic. Complete wrap (360 degrees) of fundus onto distal esophagus. → ppl with normal peristalsis.

④ Toupet fundoplication: 270-degree posterior wrap of fundus. Used if motility issues.

⑤ Dor fundoplication: 180-degree anterior wrap. Used if motility issues.

] people with abnormal peristalsis (but present)

⑥ Hill repair: attach stomach and esophagus to median arcuate ligament.

*no surgery for ppl with no peristalsis.

treatment

**lifestyle**
- avoid trigger foods.
- raise head of bed.
- avoid food before sleep.
- weight loss (most imp).

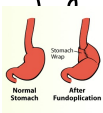
**meds**
- PPIs: most effec. (but we use them with severe cases only cuz they have side effects!).
- H2 blockers: 1st line for mild Gerds.

**surgery**
- if meds failed.
- complications: strictures & recurrent aspiration Pneumonia & hiatal hernia.
- *aim: to tighten LES. (m/c complication of surgery → dysphagia).
- *we do partial surgery for PS with ↓ peristalsis.
- *No surgery for PS with scleroderma or achalasia.

**Surgery**

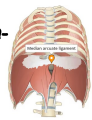
**(fundoplication).**
(we wrap the fundus of the stomach around the esophagus LES, to support it).

- **complete (Nissen) (360°)**
  {fundus wraped 360° (fully) around eoso.}
  
- **Partial** (if ↓ peristalsis).
  - **toupet (270°)**
    
  - **Dor (180°)**
    




**Hill repair**

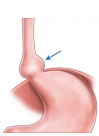
attach stomach & eoso. to medial arcuate ligament.

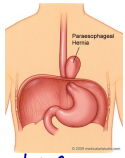


# Hiatal hernia:

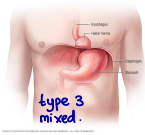
Types: *happens due to laxity of phrenoesophageal membrane.

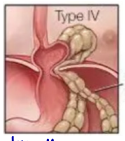
1. I: Sliding GEJ in chest, fundus in abdomen. 20% of people have it, mostly asymptomatic. (it increases risk of Gerds).
2. II: Para-esophageal (next.) GEJ in abdomen, fundus in chest. (less common than 1.)
3. III: Both GEJ and fundus in chest. (mixed)
4. IV: III plus another organ in chest (usually colon) (complex)


type 1 (sliding)



type 2


type 3 mixed.



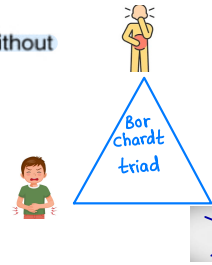
type 4. (complex).

Most commonly presents with reflux.

## Complications:

1. Bleeding: MC of type II and III. (cuz eoso is squeezed).
2. Cameron ulcer: ischemia from hiatal ring. (in mucosa.)
3. Obstruction (squeezed).
4. Gastric volvulus/incarceration: Borchardt triad: severe epigastric pain (1), retching (2) without vomiting, inability to place NG (3). (nasogastric tube.)

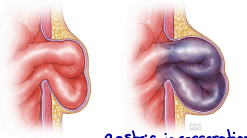
Type 2:
↑risk of complications (ex. dysphagia & obstruction + ischemia + ulcer + ischemia (mc) of eoso. cuz it's squeezed by stomach). gastric vulvulus to stomach.



Because area of hernia is swelled.

## Imaging:

1. CXR: AF level in chest (air fluid level). (it indicates presence of fluid in chest = due to eoso. perforation, pneumothorax, pleural effusion....).
2. Barium swallow: confirms and tells type.
3. Manometry: to rule out motility disease.
4. Endoscopy: can show complications and other abnormalities.


gastric incarceration (part of intestine is trapped in sac of hernia).

## Treatment:

*mostly no treatment.

### Surgical:

1. Emergency in volvulus (1) and obstruction (2)
2. Elective in most type II, III and IV. (patient gets to choose).
3. Add anti-reflux procedure: Nissen usually, do partial wrap if motility disorder. (if type 1/Gerds).

*when diagnosing it we don't depend much on symptoms because they vary "it's an anatomical disease".

- Type 1: symptoms of Gerds.
- T2: dysphagia & eoso. bleeding.
- others: shortness of breath/chest pain.

## Esophageal perforation:

- More likely to happen due to lack of serosa. (other organs have serosa) = mesothelium = last outer layer (found in visceral organs)

- MCC is iatrogenic (50%) EGD (esp If rigid) mostly at UES. Other cause: ① Pill-induced (tetracycline, KCl, ② NSAID, ③ iron, bisphosphonate), and infectious esophagitis. ↗ malignancy.

- Boerhaave syndrome: post-emetic perforation. Distal third with longitudinal mucosal tear (Mallory-Weiss). (severe vomitting mainly due to alcoholism). (boerhave is a complication of mallory mall. is a tear, boer is a perforation in tear)

- Upper 1/3 limited spread of infection in neck. Lower 2/3 more spread due to negative chest pressure > infectious mediastinitis. Mid-esophageal perforation is associated with right pleural effusion, lower esophageal with left effusion. (cuz mid is closer to right side) بنطمج شوي لليمين وهو نازل من الرقبة.

(presentation).
- Signs: ① Altered vital signs ↳ ↑HR ↗ ↓Bp, ② subcutaneous emphysema ↳ air under skin in neck (air escaped from eoso). felt with palpation., ③ Hamman's sign ↳ crunching sound with heart beat due to pneumomediastinum. ↳ if air leaked to media, ④ pleural effusion (typically left) ↳ when fluids of eoso leak through mediastinum to lungs pleura. (↓ breath sounds & dullness on percussion).
+ fever + pain & dysphagia + vomitting.

Imaging:

① - Do PA/Lateral CXR (↳ post-anterio.) and upright abdomen (↳ supine ↳ sitting) pneumomediastinum, subcutaneous emphysema, left pleural effusion.

② ① Water-soluble esophagram (↳ similar to Barium swallow but $H_2O$) if unstable. ② CT with water-soluble contrast if stable. (to confirm diagnosis).

Treatment:

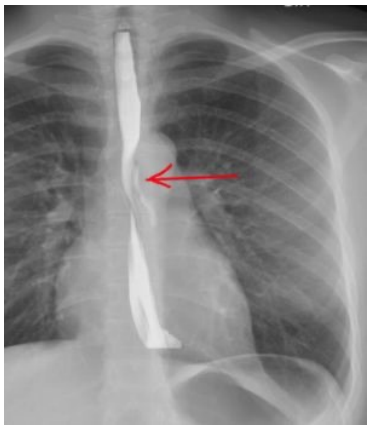
① - Non-surgical: if stable and contained peroration. ① NPO (↳ No food (Nill per os)) + ② NGT (↳ nasogastric tube.) + ③ IV Abx (↳ iv broad spectrum antibiotics). Follow-up CT/esophagram after 1 week (it'll resolve alone).

② - Surgical: ① NPO + ② NGT + ③ IV Abx. ④ Remove dead tissue, use ⑤ drainage (2-3 tubes), establish *if leakage. feeding. If ① delayed presentation or ② malignancy may do partial/complete esophagectomy (↳ laparoscopic). May use endoscopic stents if cannot use surgery.

*Note: if the perforation was in the cervical portion; it's usually contained & not fatal (cuz neck is a small compressed area) however if it was in the thoracic area it's fatal because of ↑ pressure (+) in that area → mediaststanitis → death.

*in any body part perforation → sudden ↑HR & ↓Bp (due to body's response to either bleeding / spread of infection) (can lead to sepsis & death).

*Endoscopy is contraindicated in diagnosis, but allowed in treatment.



PA CXR.

**Mallory-Weiss tear:** (it's a tear not a perforation).

cause: ① ②

- Repeated vomiting and high gastric pressure > longitudinal mucosal laceration at GEJ > UGIB (upper GI bleeding)

- **Risk factors:** alcoholism ① (causes vomitting (m/c)), NSAIDs ② (causes vomitting), portal hypertension ③ (weakness of wall), hiatal hernia ④ (causes ↑ pres → vomitting), cough ⑤, Bulemia ⑥ & Anorexia ⑦. (gastroeso. junction.)

**Treatment:** as in any UGIB: (ABCs) (airway, breathing & circulation.)

- ① NPO (x food), ② get IV access, ③ type and crossmatch PRBCs (packed) and ④ transfuse if needed, ⑤ lavage stomach (NG) (in many p's bleeding stops spontaneously). (↑ nasogastric tube empties stomach content.)

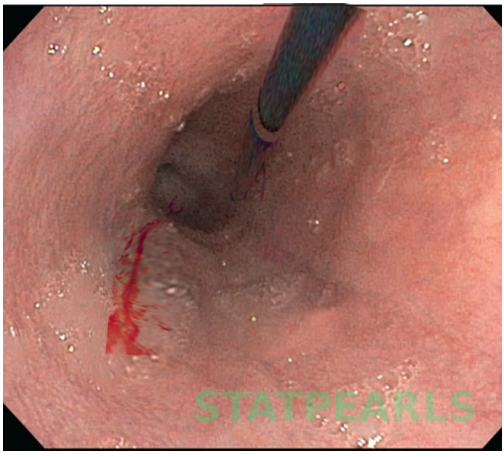
After initial stabalization

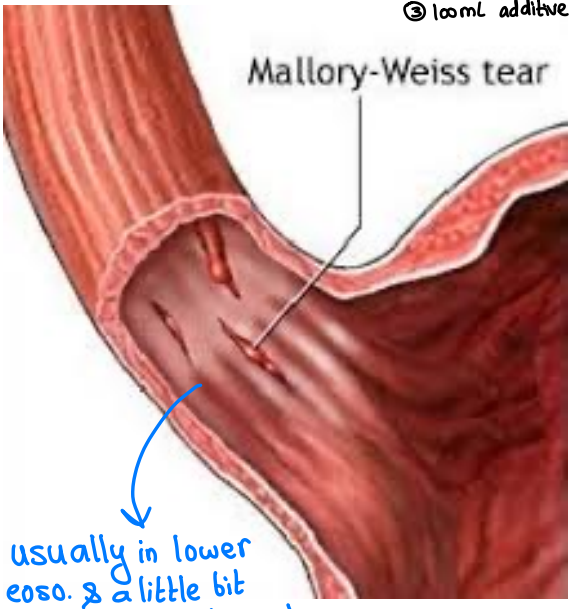
① **Endoscopy** operate if visible vessel with adherent clot or active bleeding. Options: epinephrine (vassopressin), heat coagulation, electric coagulation, clipping, band ligation, sclerotherapy. (if failed) (↑ signs that locate bleeding vessel.)

② **Angiography** inject vasopressin or embolize vessel. if failed to stop bleeding

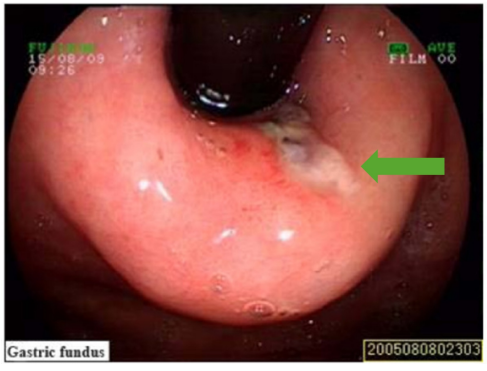
③ **Surgery** if endoscopy and angiography fail or needing >6 units of PRBCs. (big) within 24 hrs.

1 unit PRBCs = 330ml
① 200ml RBCs.
② 30ml plasma.
③ 100ml additive.




usually in lower eoso. & a little bit extending to stomach.




- presentation: ① hematemesis ② /melena ③ w/out ↓Bp if sudden. ④ Anemia (if happened slowely). (same as any ↑ GI bleedings' symptoms).

**Achalasia:** (a motility disorder)

- **Etiology:** mostly idiopathic (autoimmune, infectious, congenital?), Chagas disease (less common). (infection with trypanosoma cruzi parasite).
  (motility nurons in eoso are dysfunctional).

**Pathophysiology:**

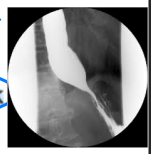
1. Aperistalsis (neural disruption)
2. Failure to relax LES (inflammation, fibrosis, and loss NO releasing neurons).

**Presentation:**

- Progressive motility dysphagia (but worse with solids) +- ① heartburn, ② regurgitation, and ③ weight loss. (dysphagia is gradual).
  (liquids+solids).

*severe food retention in eoso. can force LES to open suddenly & then close this can cause 2ndary gerds & food regurgitation.

**Imaging:**



1. Barium swallow: distal narrowing (LES) and proximal dilation (aperistalsis): Bird's beak deformity.
2. Endoscopy: to rule primary or surrounding malignancy (pseudo-achalasia). → same presentation of achalasia but cause is cancer.
3. Manometry: gold-standard: aperistalsis + incomplete LES relaxation. Tone >30 mmHg.
   (if tone was very high >120mmHg = Nut cracker eosophagus.

**Treatment:**

- Non-surgical: ① Botox (temporary response). (to expand LES.)
  ↳ needed every 6 months. this may cause fibrosis due to continuous injections.
  ↳ cuz wall is stiff.
  ② Balloon dilation (Endoscopy). (risk of recurrence, less effect if young). + risk of rupture)
- ② Surgical: Myotomy: esp in young. May need to add anti-reflux (partial wrap) to reduce secondary GERD. (fundoplication). (myotomy → cut parts of smooth muscle in LES).
  - Heller Myotomy cut longitudinally then do Dor or Toupet fundoplication.

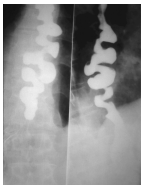
## motility disorders

| Achalasia | scleroderma | diffuse E spasm. |
|---|---|---|
| aperistalsis | aperestalsis | non-peristaltic synchronous contractions |
| failure of relaxation of LES. | ↓ LES tone | normal LES. |
| Dysphagia. | mimic Gerds | chest pain |
| Gold standard for all = manometry | | |
| myotomy of LES (removal). | Anti reflux drugs, no other treatment. (fundoplication can't be done). | smooth muscle relants. (Ca⁺ channel Blo. Nitrates ....). if sever: long myotomy. |

* scleroderma has Gerds symptoms + systemic symptoms like raynauds/other organ involvement.

* Achalasia may have similar symptoms to Gerds, but dysphagia is very severe (can't swallow water + has to tilt his head upwards & backwards to assist swallowing) + ↑weightloss.

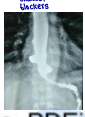
## Other motility disorders:



**Diffuse esophageal spasm:**

- Repeated simultaneous non-peristaltic contractions.

- MC presentation is substernal chest pain. Treat with smooth muscle relaxants: ① CCB (Calcium channel blockers), ② nitrate, ③ PDEi (phosphodiesterase inhibitor). May require long myotomy (if not relieved by meds).



**Nutcracker esophagus:** very high amplitude distal contractions >180 mmHG. (type of achalasia).

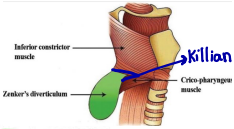
**LES hypertension**: normal peristalsis + high LES tone. Treat with CCB, Nitrate, PDEi +- myotomy.

**Scleroderma**: sclerosis of smooth muscle: reflux more than dysphagia. Low LES tone, deceased peristalsis, normal UES. No role for fundoplication.

* Note: scleroderma = systemic sclerosis its an autoimmune disorder in which tissue is replaced with thick dense connective tissue. (affects visceral organs + skin + vessels): its most visible feature is hard skin.

**Zenker's Diverticulum:** = pharyngeoesophal diverticulum.

- MC esophageal diverticulum. Typically, in older men.
- False diverticulum (pulsion/acquired). *eoso diverticula is usually due to ↑ pressure due to motility problem. (usually false).




- Killian triangle: between cricopharygneus and thyropharyngeus. Potentially due to high UES pressure. (above cricopharyngeous muscle)

upper eoso.

- ① Dysphagia, ② food regurgitation (↳ cuz of ↑ UES tonicity), ③ halitosis (↳ cuz food is stuck up), ④ aspiration, ⑤ audible swallowing. Can have neck swelling.

* physical exam insignificant. but if too large may show lat. neck mass.

- Compare with epiphrenic diverticula: distal third, due to high LES tone. (in LES).

**Imaging:**

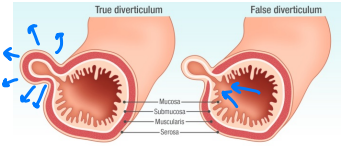
- Barium swallow: gold standard. Avoid Endoscopy.

**Treatment:** Surgical: ① diverticulectomy and ② diverticulopexy (له نثبت ما تبقى من الجدار.) + ③ myotomy of cricopharyngeus (↳ to avoid recurrence). *imp exam Q!

# Diverticula

(it's when a bulge forms in a wall of a visceral organ).

- Diverticula
  - **True**: bulge is caused by an external force, forcing all layers of wall out (Traction)
  - **false**: Bulge caused by a force inside lumen pushing only mucosa & submucosa out. (pulsion).



## Esophageal neoplasia → benign. / malignant.

Benign: MC is leiomyoma (smooth muscle) → appear as smooth filling defect | causing ↓ mechanical dysphagia.

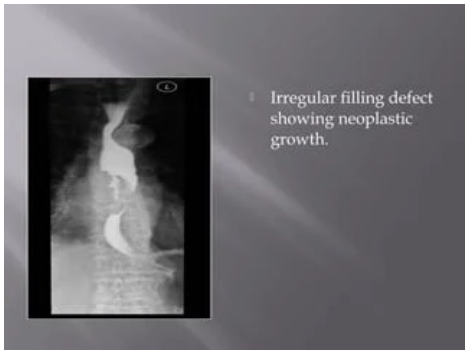
1. Epithelial: MC is esophageal cysts.
2. Non-epithelial: leiomyoma (1), fibroma (2), lipomyoma (3), etc.

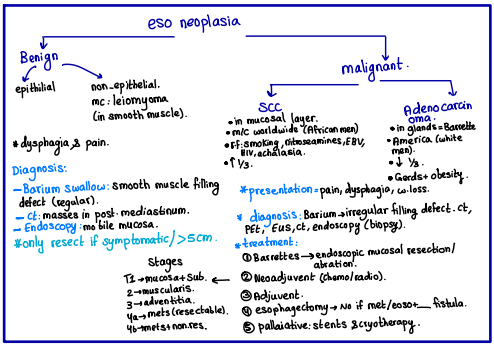
Presentation: dysphagia, pain

Imaging:

1. Barium swallow: smooth filling defect, well-demarcated, non-ulcerated.
2. CT scan: posterior mediastinal mass. (if tumor is in thoracic part).
3. Endoscopy: very mobile submucosal mass. Do not biopsy of intact mucosa.


Remove if symptomatic or larger than 5 cm.



*malignancy → men > women

*adenocarcinoma → cancer in glandular tissue lining an organ ex: mucosa.

*mucosal layer in eso. is made of squamous cells.

*so both adeno & scc are in mucosa, but one is in the glands & one in squamous cells.

Malignant: SCC (1) is MC worldwide adenocarcinoma (2) became MC in the US.
↳ Barrette.

- Usually locally advanced at presentation.

Risk factors:

types
1- SCC: alcohol, smoking, nitrosamines (in asian food), achalasia, HIV and EBV. More in African, more in men. (in ↑ 2/3).
2- Adenocarcinoma: Barret/chronic GERD and obesity. More in White, more in men. (in ↓ 1/3)

Presentation: dysphagia (progressive), pain, weight loss. (usually locally advanced in lymph nodes/regional invasion)

Imaging:

1. Barium: filling defect, irregular.
2. Endoscopy: biopsy, brush cytology for Dx
3. EUS: for depth and nodes. (endoscopic ultrasound).
4. CT: for mets (metastasis).
5. PET: for occult mets. (+ve emission tomography).
↳ unknown 1° origin metastasis.

| SCC | Adenocarcinoma |
|---|---|
| in mucosal layer | mucosal layer |
| squamous cells | mucosal glands. |
| African males | white males |
| mc worldwide. | mc america. |
| upper 2/3 of eso. | lower 1/3 of eso |
| Alcohol, smoking, achalasia HIV, EBV & nitrosamines. | Gerds, obesity |

Staging: T1 (mucosa and submucosa), T2 (muscularis), T3 (adventitia), T4a (pleaura/pericardium/diaphragm: resectable), T4b (aorta, vertebra, trachea: unresectable).

Treatment:

1- Barrett's: ablation or endoscopic mucosal resection (EMR).

2- Surgery: esophagectomy, contraindicated if mets, LN not in field of excision, bronchesophageal fistula. Look out for leak.
↳ No point in opening his body & removing organ if cancer has already metastasized.

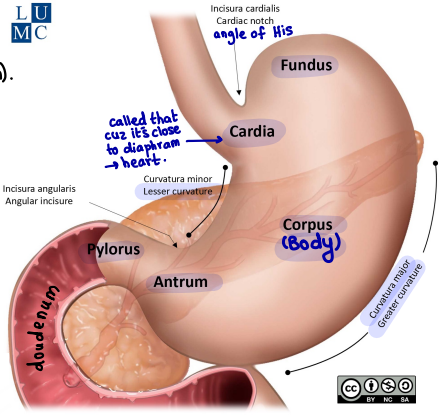
3- Use neoadjuvant chemo-radio if locally advanced (high T or high N) to make resection more complete.
↳ to ↓ size of tumor so it becomes easily resectable.
no. of lymph nodes affected.

4- Adjuvant: chemo or radio → after surgery.

5- Palliative: stenting, laser or cryotherapy to reduce symptoms only.
↳ if we can't do excesion surgery we do palliative (things that help with symptoms).

# The stomach

## ① Anatomy: it's in the left upper quadrant. (intraperitoneal + connected to omentum).




### Arterial supply

- **lesser curvature** → L. & R. gastric arteries.
- **greater curvature** → L. & R. gastroepiploic arteries.
- **fundus** → short gastric.
- **Pylorus** → gastroduodenal artery.
- **others** → Post. gastric. / L. inf. phrenic

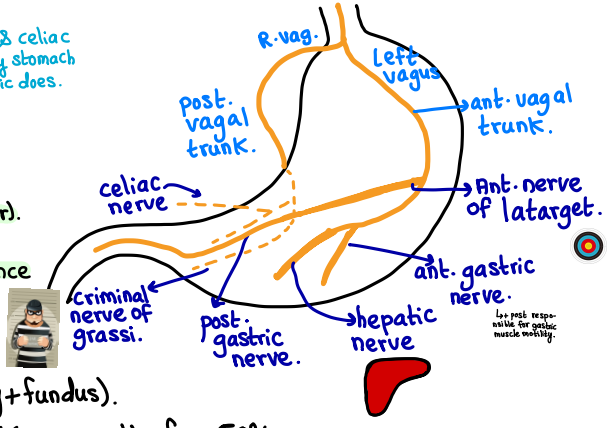
Left gastric vein (also known as coronary vein) communicates with azygous and is site of esophageal varices in portal hypertension. (dilated esoso. veins). ↳ it communicates with portal & esophageal veins.

Lymphatics: spread of cancer to esophagus is more common than to duodenum.


* Note: arterial supply of stomach comes from around the curvatures.

* Left vagus nerve goes ant. → anterior vagal trunk, Right vagus, goes to the back → post. vagal trunk.

* hepatic & celiac don't supply stomach, only gastric does.




* Ant. nerve of latarjet → Antrum & pylorus (sensory) (it allows opening of pyloris → gastric emptying). if it's cut; food will stay in stomach (obstruction).
* Criminal nerve of grassi → supplies body of the stomach (motor). (it supplies parietal cells, so if it's cut → ↓ acidity).
- Criminal is usually left in vagotomy causing ulcer recurrence

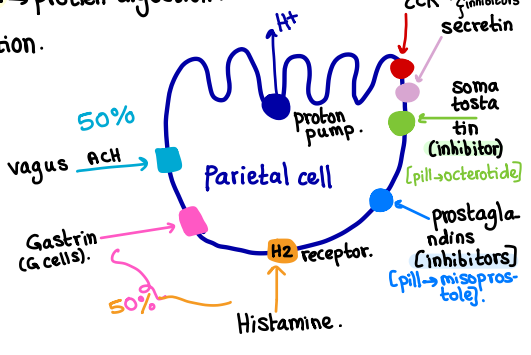
## ② function:

* to secrete acids. (by parietal cells in body + fundus).
* parietal cells are stimulated by vagus nerve (it's responsible for 50% of acidity. (vagus ACh → Parietal).
* parietal cells also secrete intrinisc factor for B12 absorption.
* Cheif cells → in body make pepsinogen → with acidity → pepsin → protien digestion.
* mucus cells → in distal stomach → $HCO_3$ & mucus for protection.

### Parietal cells

**Stimulated by**
1. Vagus nerve (50%).
2. Histamin on $H_2$ receptor.
3. Gastrin. (from G-cells).

**Inhibited by**
1. Somatostatin.
2. Prostaglandin.
3. CCK.
4. secretin.




* Note: NSAIDS ↓ prostaglandins → ↑ acidity.

# Peptic ulcer disease

- Ulcer: a mucosal defect that reaches submucosa. (if it was only in mucosa → erosion) it can be in doudenum or stomach.
- m/c location → post. wall of upper part of doudenum.
- peak age: 55-65.

*remember: gastroduodenal art. passes behind post. wall of 1st part of doud., so doudenal ulcers have ↑ tendancy of bleeding.

•m/c cause of UGIB is doudenal ulcers.

• **pathophysiology:**

① ↑acidity = due to: ↑vagal stimuli/↑gastrin/↓prostaglandin.

② ↓ protection against acidity → ↓mucous production/direct damage to mucosa/ischemia

→ causes

**mc**

① 80% H pylori

② 12% NSAIDS.

**other risk factors.**

① Gastrinoma. ② ZES. ③ Smoking. ④ Alcohol. ⑤ ↑icp. ⑥ Burns. ⑦ stress ulcers (multiple, superficial infundus from ischemia).

*if acidity ↑ it'll usually cause doudenal ulcer & ↓protection → gastric

**Types:** (of gastric ulcers) · (ulcers above antrum → loss of protection, below → ↑acidity).

Type I: Gastric (70-80% of which): lesser curvature towards antrum. Due to loss of protection. Blood type A.

*gastric ulcers' mc site is 'incisora angularis' in the lesser curvature between body & pylorus. (it's a weak spot → ↑ risk of perforation).

Type II: Gastric and duodenal: lesser curvature and duodenum. Excess acid. Blood type O.

Type III: Gastric (2nd MC in stomach): antral (prepyloric). Due to excess acid. Blood type O.

→ antrum usually has ↓ protection than other parts of stomach thats why ulcers in it form due to ↑ acidity.

Type IV: Gastric (<10%): lesser curvature close to GEJ. Due to loss of protection. Blood type O.

Type V: Diffuse. NSAIDs. (any ulcer in any part of stomach if it was caused by NSAIDS).

# H pylori

• H.pylori → G-ve spiral bacteria, it produces enzyme = Urease which transforms urea to ammonia (alkaline) H. usualy lives in antrum causing an infection called antral gastritis → lowers stomach's protection → ulcers + Ammonia stimulates gastrin → ↑acidity

*H.pylori is ↑in ↓socioeconomic class (cuz of ↓ food hygiene)

*↑risk with age (peak 55-65 yrs).

Presentation:

① peptic ulcer. ② dyspepsia ③ nausea & vomitting. ④ early statiety.

⑤ abdominal pain → epigastric & either radiates to the left or back.

food relieves pain if doudenal ulcer (cuz it ↓ acidity) & increases pain if gastric).

*dyspepsia: Belching, early statiety, Nausea, heartburn, abdominal pain, uncomfortable fullness, bloating.

*Note: H.pylori can cause gastritis → inflammation → distrupt stomach lining → distrupts nerves responsible for gastric muscles → slow down digestion.

*food ↑ gastric ulcer pain mechanically + cuz it ↑ acid production, but as food reaches doudenum it will have buffered the acid → decreases doudenal ulcer pain. (also food increases Hcl secretion by parietal cells in the stomach & Alkaline secretion in doudenum from pancrease).

# Diagnosing H.pylori:.

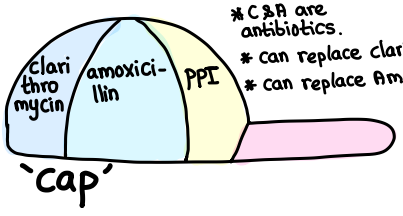
① physical exam: insignificant.

② Tests

- **Urea breath test**
- **Endoscopy** (invasive)
  - Done for ppl with ↑ risk of cancer ex: old pts.
  - take biopsy from antrum to:
    ① See bacteria from Gemsia or culture.
    ② locate ulcer if present.
    ③ rule out other diseases.
- **fecal antigen test.**
- **serology**
  - look for H.pylori antigens. (Igm/IGg).
  - setback: test can still be +ve in treated pts.

## ③ H.pylori treatment:

### [1] Triple therapy for 2 weeks.




* C & A are antibiotics.
* can replace clarithromycin with tetracycline.
* can replace Amoxicillin with metronidazole.

### [2] Gold standard: Quadriple therapy:

- Triple + Bismoth (antibacterial).
- used when triple doesn't work.

* Note: antibiotic → not specific for a bacteria. antibacterial → specific.

* Repeat EGD after 6-8 weeks to check H.pylori, if still present → give triple again for 2 weeks then at 6-8 weeks EGD, if still there 2 weeks of triple again, then 6-8w EGd if still → surgery.

### ④ Antisecretory therapy: (given in addition to H.pylori treatment or for ppl who have ulcers but no H.pylori).

① Antacids: Mg and Al compounds. (many side effects).
② H2 blockers: safe and cheap.
③ PPI: irreversible, require acid environment. (most effective).
④ Sucralfate: creates a protective layer on ulcer. (protects stomach wall).
⑤ Bismuth: unclear, has anti-H pylori effect.
⑥ Prostaglandins: misoprostol, inhibits acid.

## • Other causes of ulcers & their treatment:

① NSAIDS: stop them.
② Alcohol / smoking → cessation.
③ stress ulcers → usually appear in fundus due to shock/ischemia.
④ Ticp → called cushing ulcer (due to vagus stimulation → ↑acid → ulcer).
⑤ Burn → called curling ulcer. (reduced blood flow to stomach).

## H pylori complications →

① acute gastritis.
② chronic antral gastritis (causes peptic ulcer).
③ chronic atrophic gastritis → increases risk of gastric cancer & gastric lymphoma.

## Complications and Surgery:

### Surgical techniques: (for peptic ulcers)

① Truncal vagotomy: cut all branches of vagus including hepatic & celiac.
② Selective: x cut hepatic & celiac.
③ Highly selective: only cut branches of vagus going to parietal cells.

① **Vagotomy** to cut around 50% of acid secretion. ① **Truncal** cut all vagal trunks (including hepatic and celiac branches), posterior trunk is commonly missed. ② **Highly selective vagotomy** (HSV, parietal cell vagotomy): preserves antrum, almost normal motility. Keeps the anterior nerve of Latarjet supplying antrum.

- Vagotomy leads to loss of gastric motility, needs **pyloroplasty** to allow emptying, may cause dumping syndrome. (truncal).

• Pyloroplasty: cutting pyloric sphincter to hide effect of cutting n. of latarget.
*dumping syndrome:
– rapid gastric emptying

② **Antrectomy**: remove ulcerated part ① and G cells ②. Two types of anastomosis: ① **Billroth I** stomach to duodenum. ② **Billroth II**: stomach to jejunum.
↳ if we had to remove doudenum.

① (less risk of complication).

*AXR = air under diaphram (air escapes a perforation). (happens only if gastric or ant. doudenal.
*omentum: part of peritonium

### Complications: (of ulcers).

رحمك الله اضطريتي تعملي عملية لإني بالعدة بدل تحسسي حساب (vagotomy).

① **Perforation**: MC indication for surgery in gastric ulcers. (m/c in gastric ulcers).

Diagnose: upright AXR showing free air. Perforation is ① gastric or ② anterior duodenal.

Treatment: ① conservative if stable patient and contained: NPO, NG, IVF, Abx (antibiotics), PPI. Surgery: do ① base of ulcer biopsy and do ② Graham patch (omental patch). If low-risk patient: may do highly selective vagotomy (parietal cell vagotomy). If patient already on PPI, consider truncal vagotomy + pyloroplasty or HSV or vagotomy and antrectomy.

② **Bleeding**: MC indication for surgery in duodenal ulcers. (typically, posterior duodenal ulcer, bleeding from GD artery).

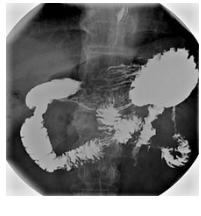
Treatment: get IV access and blood → Npo + Nasogastric tube → lavage. PPI IV. If >6 units in 24 hours of hypotensive this needs intervention. Endoscopy: look for active bleed, vessel, visible clot. Treat. Surgery to ligate GDA. If patient already on PPI, consider ① truncal vagotomy and pyloroplasty ② or HSV. If larger ulcer: vagotomy and antrectomy.

③ **Obstruction**: least common complication. Usually in chronic and GOO (gastric outlet obstruction).

Treatment: Diagnose with upper GI series ① or CT scan ②. NG decompression ① (lavage), NPO ②, IVF ③, start PPI ④. Can do ① endoscopic serial dilation. ② Surgery: antrectomy (Billroth II) + vagotomy.

④ **Recurrence**: ① incomplete surgery, ② ZES, ③ H pylori or ④ cancer.

*upper gi series same idea of eosophagogram (Barium), but for eoso+stomach +upper small intestines.



*symptoms of gastric obstruction:
– vomitting
– ↓ Bowel motion.
(diagnosis requires imaging).
↳ AXR / CT / UGI series.
(Treatment → NPO, NG, IVF (wait till it resolves) if not → Endoscopy (open it) if not surgery).

*Note: ulcers can cause obstruction through:
– inflammation → edema
– inflammation → scarring → strictures.
– inflammation → muscle spasms.

# Gastric neoplasia

## cancers (Gastric carcinoma)

### Adenocarcinoma (95%)

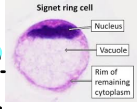
**intestinal (60%)**
- due to Environmental
- m/c with age +men
- antrum & pylorus.
- either looks like an ulcer (ulcerative type) / mass → exophytic.
- called intestinal cuz stomach lining cells look like intestinal cells.
- Hematogenous spread.

↳ Environmental risk factors:

- Risk factors: more in Japan/Korea, possibly due to high salt ① and high nitrate food ②. Low socioeconomic status ③. Family Hx ④. Smoking ⑤. Males ⑥. Blood groups A ⑦ and O ⑧. H pylori ⑨. Gastric ulcers (10% risk) ⑩. Chronic gastritis ⑪. Gastric surgery ⑫. Villous polyps ⑬. Adenomatous polyps >2cm.

*Note: ↑inflammation → Atrophic gastritis → intestinal metaplasia = intestinal gastric adenocarcinoma.

**Diffuse (30%)**
- heridtary.
- diffuse.
- young+women (m/c).
- worst
- poorly differentiated signet ring cells.
- spread:
  ① transmural (through stomach wall).
  ② lymphatic.



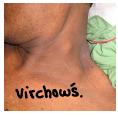

eventually causes thick walled stomach by fibrosis we call the wall: "linitis plastica".

*loss of E-Cadherin in stomach wall is linked to adenocarcinoma.

### Others

- lymphoma (5%)
- gastrinoma.
- Gist (gastrointestinal stromal tumor).
  ↳ ct, nerves, vessels, ducts.

*Note: gastric cancers are 40% antral.









### Presentation:

**History:** ① pain, ② anorexia, ③ weight loss, etc. (postprandial pain).

**Physical:** ① Virchow's node (left supraclavicular), ② Sister Mary Joseph's nodule (umbilical mets), ③ Irish's nodes (axillary LN), ④ acanthosis nigricans, ⑤ Leser-Trelat sign. +mets to uterus

**Imaging:** Do ① CT abdomen/chest. ② Endoscopy with US and biopsy: diagnose, and determine T and local N. May do staging laparoscopy.

**Stage:** must sample at least 15 nodes. T1 (mucosa and submucosa), T2 (muscularis propria), T3 (serosa), T4 (adjacent organs). N: LN count. M: distant mets.

T = tumor = size & extent.
N = Nodes = involvement.

| poorly differentiated (↑ grade) | well-differentiated (↓ grade) |
|---|---|
| act diff from normal cells. | act similar to normal cells. |
| spread & ÷ more | grow slowely. |
| more aggressive. | less aggressive |
| responds better to adjuvant therapy | doesn't respond to adjuvent. |

*mc organ for stomach cancer to spread in:
① liver ↑↑↑ ② peritonium ↑↑
③ lungs ↑ ④ bones ↑.

*staging laparascopy:
- it increases stage that's why it's imp.
- (you can estimate the stage of cancer for ex. 2 from other techniques, but when you do staging lapro- it might discover new metastasis & staff → increase stage to 3) (this happens in 25% of cases).

### Surgery:

- Distal tumors: ① radical subtotal gastrectomy + ② Billroth II: radical = first part of duodenum, hapatogastric lig and greater omentum. Surrounding LNs. (remove all surrounding tissues & lymph nodes)

- Proximal tumors: ① total gastrectomy + ② Roux-en-Y.

- Unresectable: if peritoneal mets, liver mets, LN remote (if we can't remove them; we do palliative txR).

*Note: in colon cancer if there was liver mets, it's still resectable, but if gastric it's not.

# Other neoplasia

## ① Gastric lymphoma (5%): (benign).

– 90% of cases are Maltoma = malt lymphoma = caused by Hpylori.

*If we have ↓ grade lymphoma we can treat it with H.pylori triple therapy.

*If high grade → chemo/radio

Note: it's a non-Hodgkin lymphoma.

## ② GIST:

gastrointestinal stromal lymphoma. (originated from intestital cell of Cajal)

* CKIT & CD34+ are markers of GIST (they're cell surface protiens that are overexpressed in GIST).

* imatinib (CKIT inhibitor) can be used for therapy.

## ③ Gastrinoma: (benign/malignant).

– 50% malignant, 50% multiple. classified as neuroendocrine tumors cuz they're similar in characteristics to pancreatic cancerous cells.

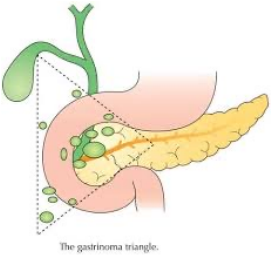
tumor of the G-cell that produces very ↑ levels of gastrin → parietal cell hypertrophy → ↑ gastric acidity → atypical peptic ulcer & ↑ secretory diarrhea.

- Atypical peptic ulcer: ① unusual loci: eosophagous, jejunum ② multiple ③ refractory to therapy. ④ ↑ complication.

*zollinger Ellison Syndrom (ZES): when p. has Gastrinoma + Atypical peptic ulcer.

*Gastrinoma can be familial: MEN 1 syndrome → 3ps: pituitary tumor, hyperparathyroidism (↑ca), pancreatic neuroendocrine tumor (Gastrinoma) (so if a p. has gastrinoma I check $Ca^{+}$ levels to see if it's Men 1 syndrome).

*gastrinoma is mainly a pancreatic tumor, but it can happen in any part of gastrinoma triangle (includes: doudenum, pancrease & pylorus & antrum). mlc doudenum.



The gastrinoma triangle.

- Diagnosis of Gastrinoma:

① Gastrin >1000. (normal 0-180 adults & 125 children).
② If secretin stimulates stomach acidity (it normally ↓ it).
③ Acidity test. pH <2. (normal empty stomach pH 1.5-3, here it has to be <2 + other symptoms, it's not diagnostic alone).
④ Imaging to locate tumor.

*Treatment is surgery, but if we can't we give meds to ↓ acidity like Somatostatin/ppis.

- Note: Somatostatin ↓ Diarrhea & acidity.

**Bariatrics:** (weightloss science).

- BMI: 25-29.9 is overweight, 30-39.9 is obese, 40-49.9 is morbid (18.5-24.9 normal). (super obesity >50).

- Comorbidities of obesity: endocrine: T2DM, hyperlipidemia, PCOS. Cardiac: CAD, HTN, venous stasis. Respiratory: OSA (obstructive sleep apnea). GI: GERD, FLD, hernias. Neurpsychiatric: depression, IIH (idiopathic intracranial HTN). Urinary: stress incontinence. Skeletal: OA (Osteoarthritis). Several cancers: endometrial, colorectal, RCC, etc.

Medical treatments:

- Less successful. (non-surgical).

- LSM: diet, exercise, etc.

- Medications: ① orlistat, ② phentermine-topiramate, ③ liraglutide, ④ bupropion-naltrexone, etc.
  orlistat ✓ stat

Surgeries
- restrictive: ↓ size of stomach. ↓ food intake.
- malabsorptive: ↓ absorption of food. (more effective).

**Bariatric surgery:**

- Criteria: morbid obesity: ① BMI >40 or ② >35 + comorbidity. Failed other approaches. (when do we do it?)

① Laparoscopic adjustable gastric band → stomach used. → Band.

- A band creates a proximal small pouch, restrictive. 30-40% weight loss, low mortality. (↳ of excess weight).

- More bands are removed now than placed.

*it ↑ risk of Gerds due to ↑ pressure in LES.

- Complications: ① slipped band, ② obstruction, ③ band erosion (↳ scar stomach), ④ adjusting device malfunction (↳ device connected outside to adjust band side), ⑤ pouch/esophageal dilation.

② Sleeve gastrectomy: (fundus & body are removed) + also laparoscopic.

- Resection of part of stomach, leaves antrum and pylorus intact, restrictive. 60-70% weight loss, low mortality. (80% of stomach is removed) * It's good because grelin receptors on stomach are removed with the resected part; so p doesn't feel that hungry.

- Complications: leak, stricture.

*we do a gastric sleeve, but we also remove duodenum (where bile for digestion is secreted) & we join stomach to ilium, so food isn't digestion untill it reaches terminal ilium (small amount only).

*Note: protien is farther digested in duodenum.

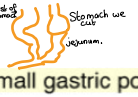
③ Bilopancreatic diversion and duodenal switch: (old malabsorptive procedure). Roux en y + sleeve

- Stomach resected as in sleave, duodenum diverted, duodeno-ileal anastomosis proximally, ileo-ileal anastomosis distally. Causing malabsorption. + restrictive.

- 70-90% excess weight loss, but higher mortality. Consider in high BMI >50.

- Complications: ① protein malnutrition, ② risk of biliary cirrhosis. (liver cirrhosis cuz bile goes back to liver).

⑤ Roux-en-Y gastric bypass: (Best).




- Alimentary limb: esophagus to small gastric pouch to gastro-jejunal anastomosis.
Biliopancreatic limb: rest of stomach to duodenum to jejuno-jejunal anastomosis. Causes both restriction and malabsorption.

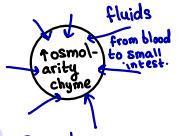
- 60-80% weight loss.

- Complications: ① internal hernias (from mesenteric defects), ② anastomotic strictures, ③ fistulas and ④ leaks. ⑤ Marginal ulcer. ⑥ PE. ⑦ Metabolic disorders.
  ↳ Pulmonary Embolism.
  ↳ cuz we made perforations in it when we cut small intestines.
  ↳ 2 Parts of intestine that join → narrowing in joining point.

## Complications of stomach surgery:

### ① Dumping syndrome:

① Early dumping (75%).




- Minutes after meal. Rapid gastric emptying of hyperosmolar chyme > osmolar fluid shift in small bowel > sympathetic stimulation + diarrhea.
  - ↓fluid in Blood = ↓Bp.
  - ↳ digested food in stomach.

- 75% of patients after surgery. More in BII than BI.

- Symptoms: after eating in a few mins → ↑HR, ↑RR, diarrhea
- *usually after ingesting ↑ sugary meals.
- * ↑HR, ↑RR, ↓Blood sugar + dizziness & blurry eyesight.
- * remember → insulin helps cells absorb glucose from blood.
- * hypoglycemia → sympathetic stimulation → ↑Epinephrine & nor. → inhibits insulin secretion. (this keeps sugar in blood so its easier to access by muscles) also symp. ↑gluconeogenisis in liver.

② Late dumping:

- Hours after meal. Hyperosmolar chyme causes increased insulin > hypoglycemia (insulin ↓ sugar in blood) > sympathetic ① stimulation + neuroglycopenic ② symptoms (dizziness, blurring of vision).

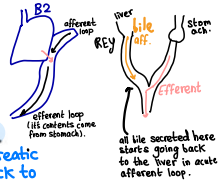
Diagnosis of dumping: UGI series.

Treatment

- Mostly resolve with conservative measures: ① diet (small meal, no liquids, less sugars), ② medical (somatostatin slows transit time and reduces diarrhea) (octerotide).
  - (Liquids → emptying faster).
- Surgery: convert Billroth to REY: reduce emptying by increasing size of jejunal pouch or slow transit with reversal of jejunal loop.

*Note: normal blood sugar level → before meal: 70-100.
→ 2hrs after meal less than 140.
(↑ after meals because sugar in carbs is absorbed to blood which stimulates insulin, but insulin takes time to work).

② **Afferent loop syndrome:**




- Incidence around 1%. Only in BII or REY: intestinal secretions into afferent loop that is obstructed lead to distention > bacterial stasis

① Acute: obstruction of bile flow: obstructive jaundice, ascending cholangitis, pancreatitis.
↳ biliary & pancreatic secretions go back to pancreas.

② Chronic: SIBO > loss of bile aids > steatorrhea, B12 deficiency.
Small intestinal bacterial overgrowth.

- Treatment: endoscopy or surgical (Both chronic & acute).

*In chronic → obstruction is slow so it doesn't go back to organs, but it causes stasis & bacterial overgrowth (SIBO). Bacteria causes: ① deconjugation of bile salts (fat malabsorption → steatorrhea).
② Eat ↓ B12 → difficiency + megaloblastic anemia.

③ **Alkaline reflux gastritis:** (same as dumping but the opposite way)

- 5% of patients with Billroth or pyloroplasty.

- Bile salt reflux to stomach causes damage, presents with bilious vomiting. green/yellow.

*Bile & pancreatic secretions secrete in doudenum, due to removal of pylorus.

- Treatment: ① metoclopramide (prokinetic antiemetic) and ② sucralfate. Surgery: convert to REY with large jejunum.
↳ it separates bile from stomach.

③ **Postvagotomy diarrhea:** vagus: ×mmc + ↑ bile when p eats.
mmc

- Excess postprandial migrating motor complexes (should be only in fasting), pushing bile salts to colon resulting in diarrhea.

- Treatment: ① octreotide (inhibits MMCs and diarrhea) + ② cholestyramine (bile salt binding resin).

**Metabolic derangements** (mlc in Bariatrics).

① Vitamin B12 deficiency due to loss of IF. → megaloblastic anemia.

② IDA (iron dificiency anemia). (iron absorption → doudenum).

③ ADEK vitamin deficiency (lipid soluble vitamins). (cuz they're absorbed in doudenum, also Bariatrics ↓ fat absorbed by small intestine (ex. ↓ pancreatic lipase, Bile emulsification...).

*migrating motor complexes (mmc):
wave of peristalsis that happens along Gi tract during fasting when we eat vagus inhibits mcc so digestion can start & it ↑ bile release.
when vagotomy → mcc stays on & at the same time bile is released when p. eats. (normally bile doesn't reach colon, but due to mcc it does → bile induced diarrhea).

*obstruction of bile flow → jaundice, because bile has bilirubin (a result of RBC death), in obstruction bilirubin (yellow) stays in blood.

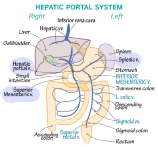
* Bile is made in liver & stored & secreted from gallbladder to doudenum.

* Bile components: 98% water + Bile salts & phospholipids (for fat emulsification) + Bilirubin + Electrolytes (to maintain its alkaline pH).

**The Small Intestines:** (midgut). (only upper ½ of doudenum → foregut → celiac artery).

**Anatomy:** (intraperitoneal but part of doudle is retra).

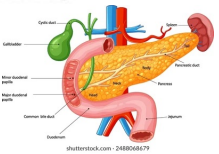
-Arterial supply ① upper half of duodenum: via celiac artery (foregut), ② distal half of duodenum, jejunum, ileum: via SMA (midgut). Veins same as arteries. SMV and splenic vein join behind neck of pancreas to form portal vein.
↳ doesn't drain SI.




-Innervation: ① vagal ② secretomotor. Sympathetic: ① vasoconstriction, ② reduced secretion and ③ motility, and sensory fibers.

- Layers: **mucosa**: epithelium (villous cells: absorption and digestion, microvilli increases surface area and contain brush border enzymes plus some receptors for absorption), in addition to crypt cells inside crypts of Lieberkuhn (regenerate epithelium), goblet cells (mucus secretion), Paneth cells (defense), enteroendocrine cells (secrete gastrin, secretin, CCK, somatostatin, motilin, and others). Lamina propria: loose CT with immune cells and layer of muscularis mucosae). Mucosa forms folds known as plicae circularis (folds of Kerckring, valvulae conniventes) that start at distal duodenum and diminish in distal ileum. **Submucosa:** fibroelastic CT for strength (important in anastomoses), contains Meissner plexus of ENS, and vascular supply. **Muscularis:** thick inner circular layer and thin outer longitudinal, innervated by Auerbach plexus. **Serosa:** visceral peritoneum. mucosa → submucosa → muscularis → serosa.

*Note: doudenum has the widest lumen.

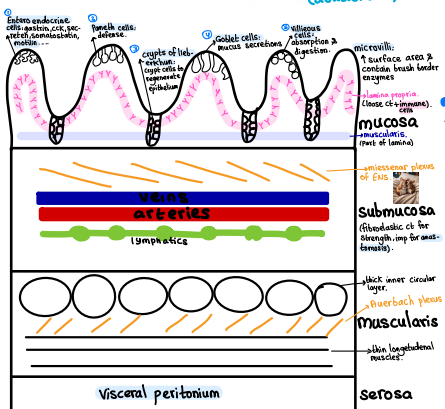




**Function:**

Vagus + hormones.

① Motility the ② pacemaker is in the duodenum. Peristalsis is stimulated by vagal cholinergic input and other ② neurohormones. Migrating motor complexes are waves of peristalsis during fasting that are mediated by motilin. mmc (prevents bacterial overgrowth + cleans int.).

② - Digestion and absorption: ① fat (mainly via ① pancreatic lipases with help from ② bile salts, absorbed as chylomicrons mainly via lymphatics). ① Protein (starts in stomach via ① acidity and pepsin, then in duodenum via ② pancreatic peptidases (need alkaline and enterokinase to be activated) → ex. trypsin. ② absorbed via portal vein). ③ Sugar (starts via saliva ① amylases, disaccharides digested via brush border ① enzymes, absorbed via portal vein). ④ Water and vitamins most water in small intestine. ① Calcium, ② iron and ③ folate mainly in proximal intestine (doudenum). ④ B12 through terminal ileum.
↳ it binds to IF in stomach then gets absorbed in ilium.




*Note: villi contain cells for digestion & absorption of food.

Meissener nervous system.
Aurbach nervous system.

*Note: in surgery it's ok to suture small intest. ine anastomosis parts, caz it's ↑ generable. (can't do that in colon).

imp + distal doud.
*in jeujenum & terminal ilium there are special folds of mucosa called plica circularis

*Note: bile does emulsification of fat (dissolve fat).

*fats absorbed by intestines go to lymphatics first not blood (chylomicrons = fat in lymph).

*in doudenum enzymes like trypsin & chymotrysin help digest protiens (they are released from pancrease), they are secreted inactive to avoid degrading pancreas, then get activated in doudenum: by alkaline environment & by Enterokinase (aka Entero peptidase) in villi of S.I.
*amaylases secreted by: saliva & pancrease.