

Gastroduodenal Surgical Diseases and Gastric Cancers

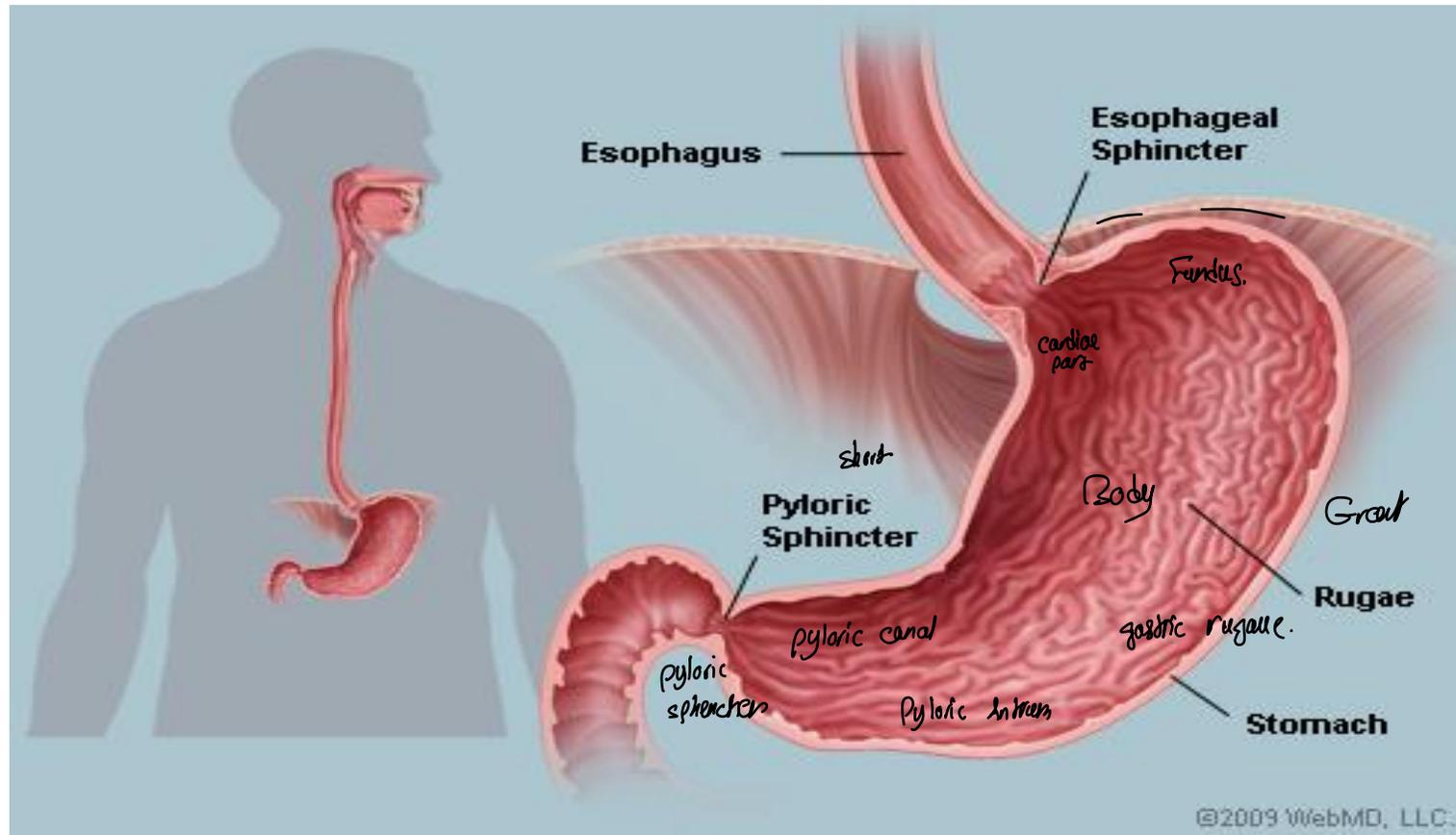


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Outlines

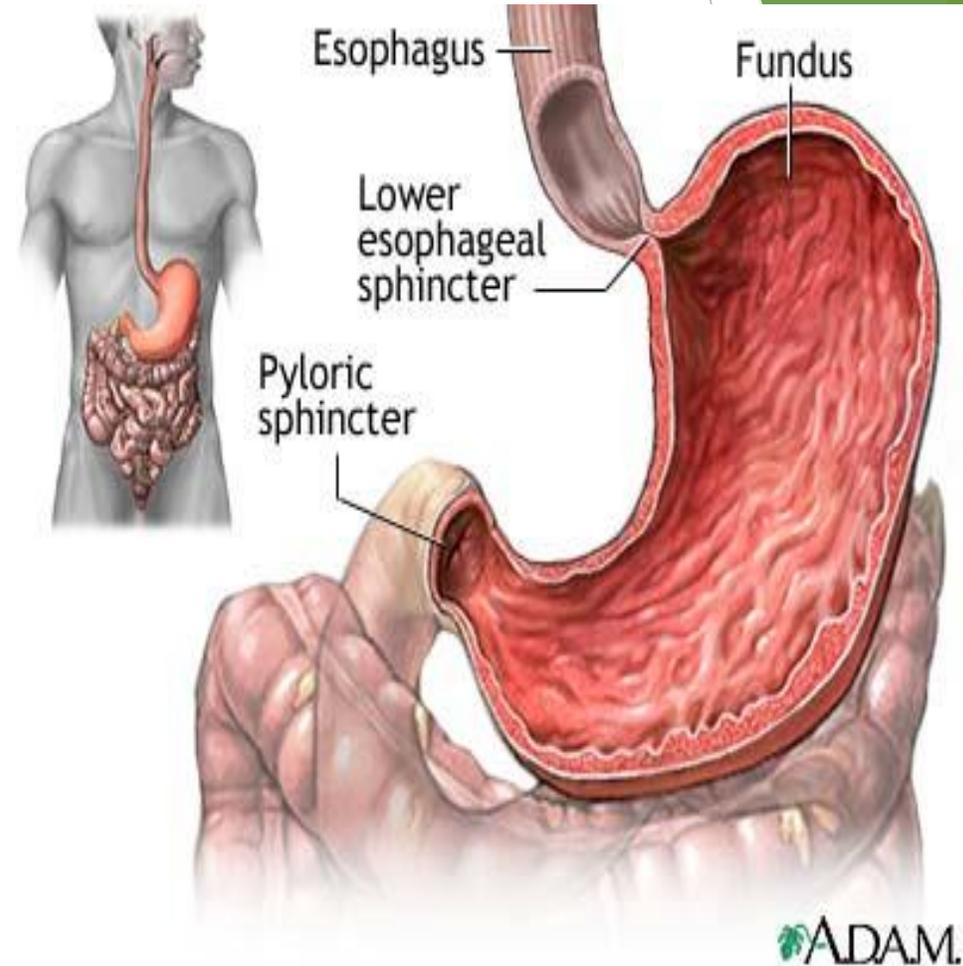
- ▶ Surgical anatomy of the stomach.
- ▶ H.PYLORI infection
- ▶ Peptic ulcer disease
- ▶ Post gastrectomy syndromes
- ▶ Gastric adenocarcinoma
- ▶ Gastric lymphoma
- ▶ Gastrointestinal stromal tumors (GIST)

STOMACH ANATOMY

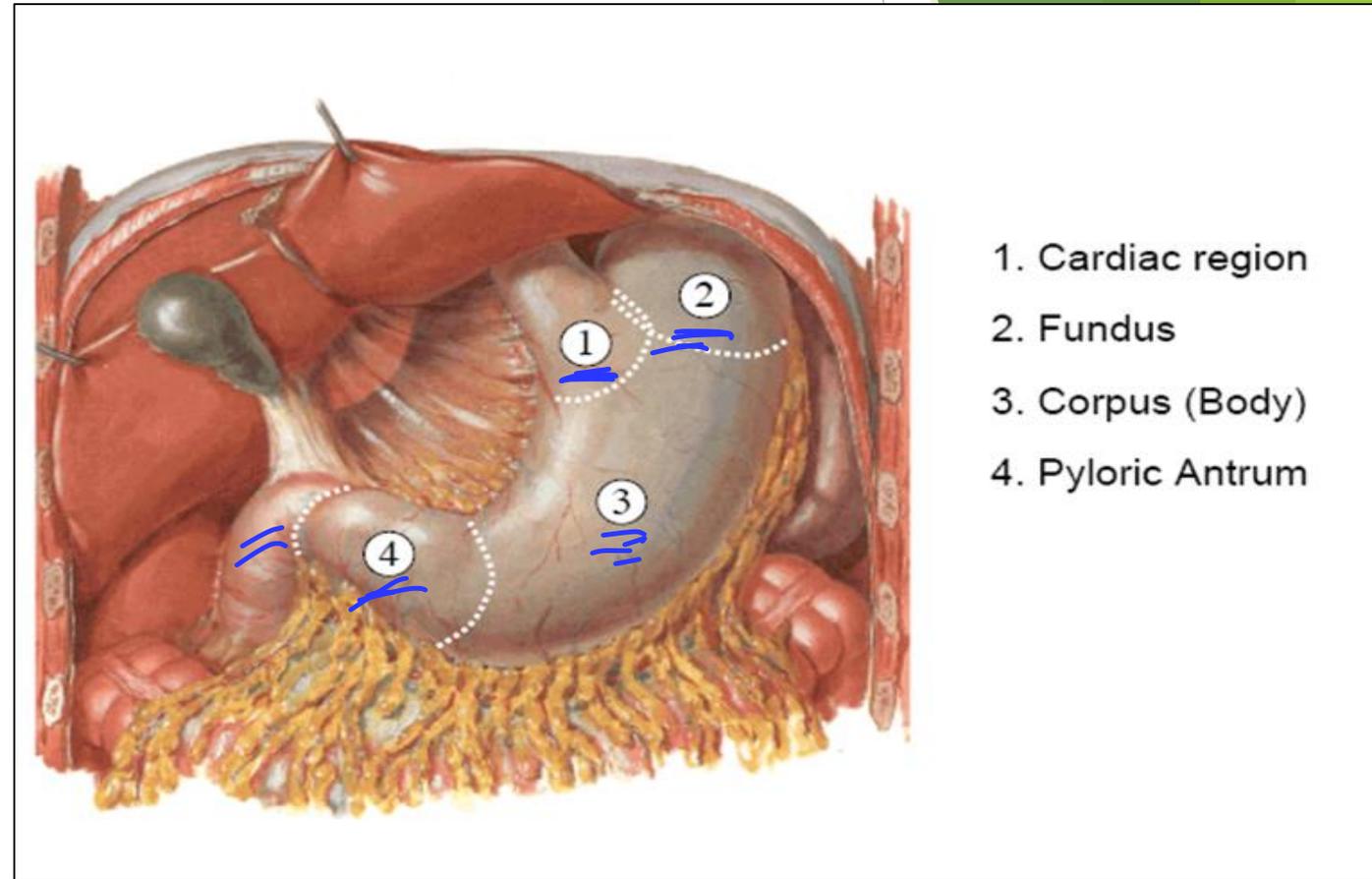
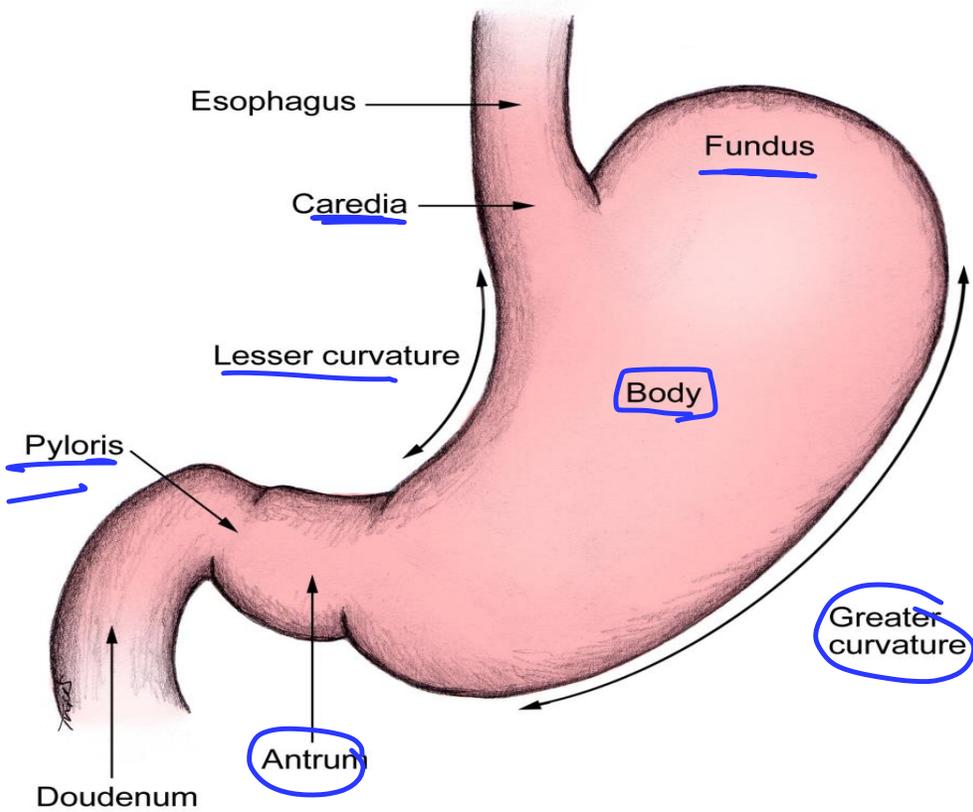


The stomach

- ▶ The stomach is a dilated part of the alimentary canal between the esophagus and the small intestine.
- ▶ It is a J-shaped muscular sac.
- ▶ located at level of T10 and L3 vertebral.
- ▶ Position of the stomach varies with body habitus



The Stomach



The stomach

- ▶ the **cardial notch**, which is the superior angle created when the esophagus enters the stomach.
- ▶ the **angular notch**, which is a bend on the lesser curvature.

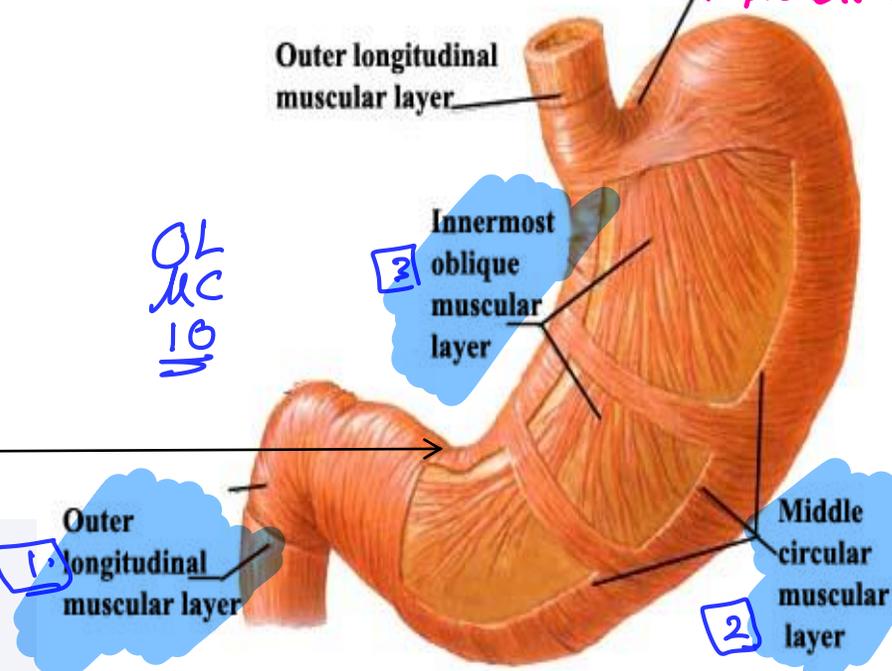
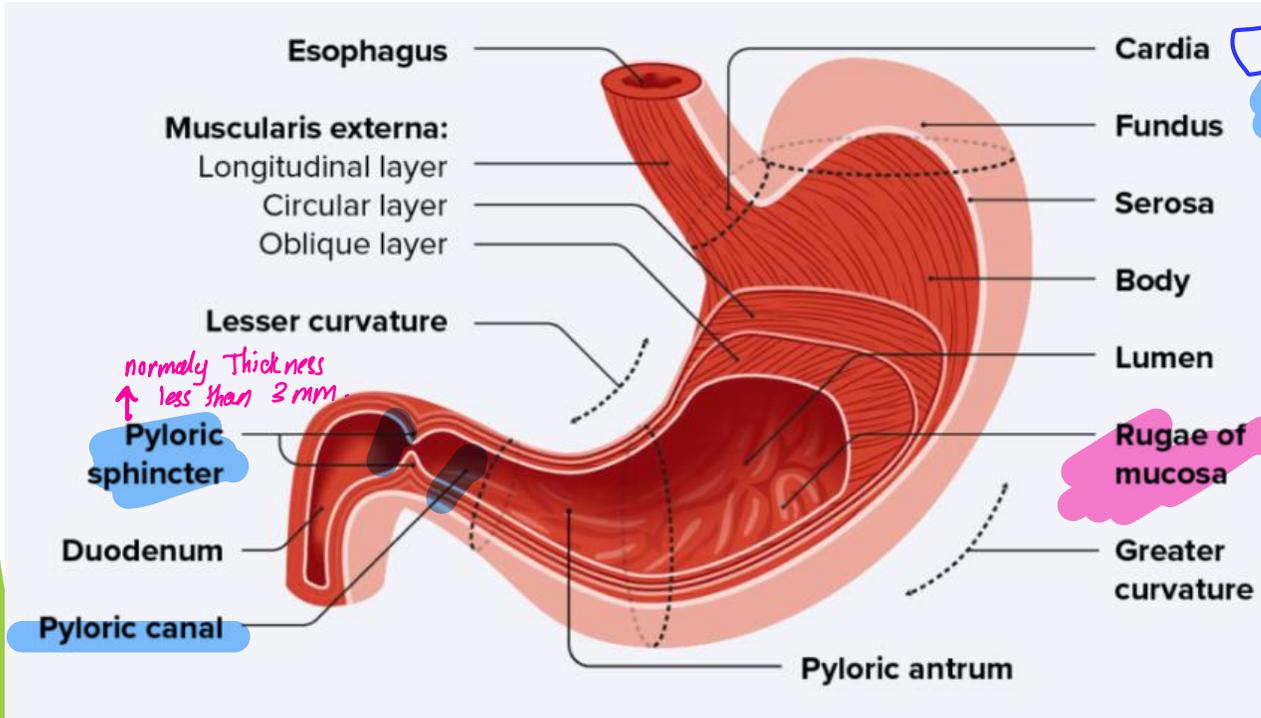
Cardiac notch of stomach
angle of H's to prevent Reflux

*OL
MC
IO*

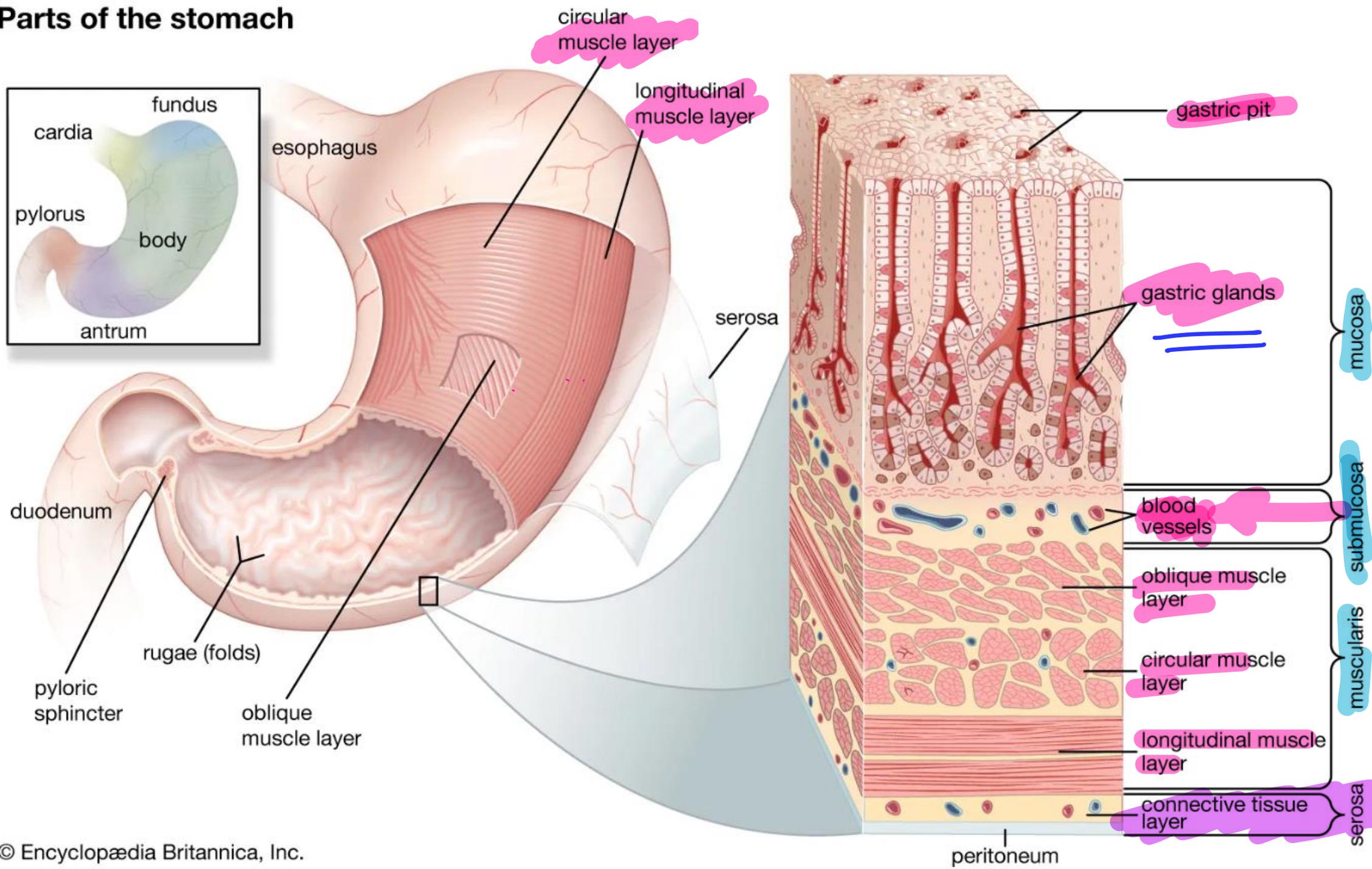
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3

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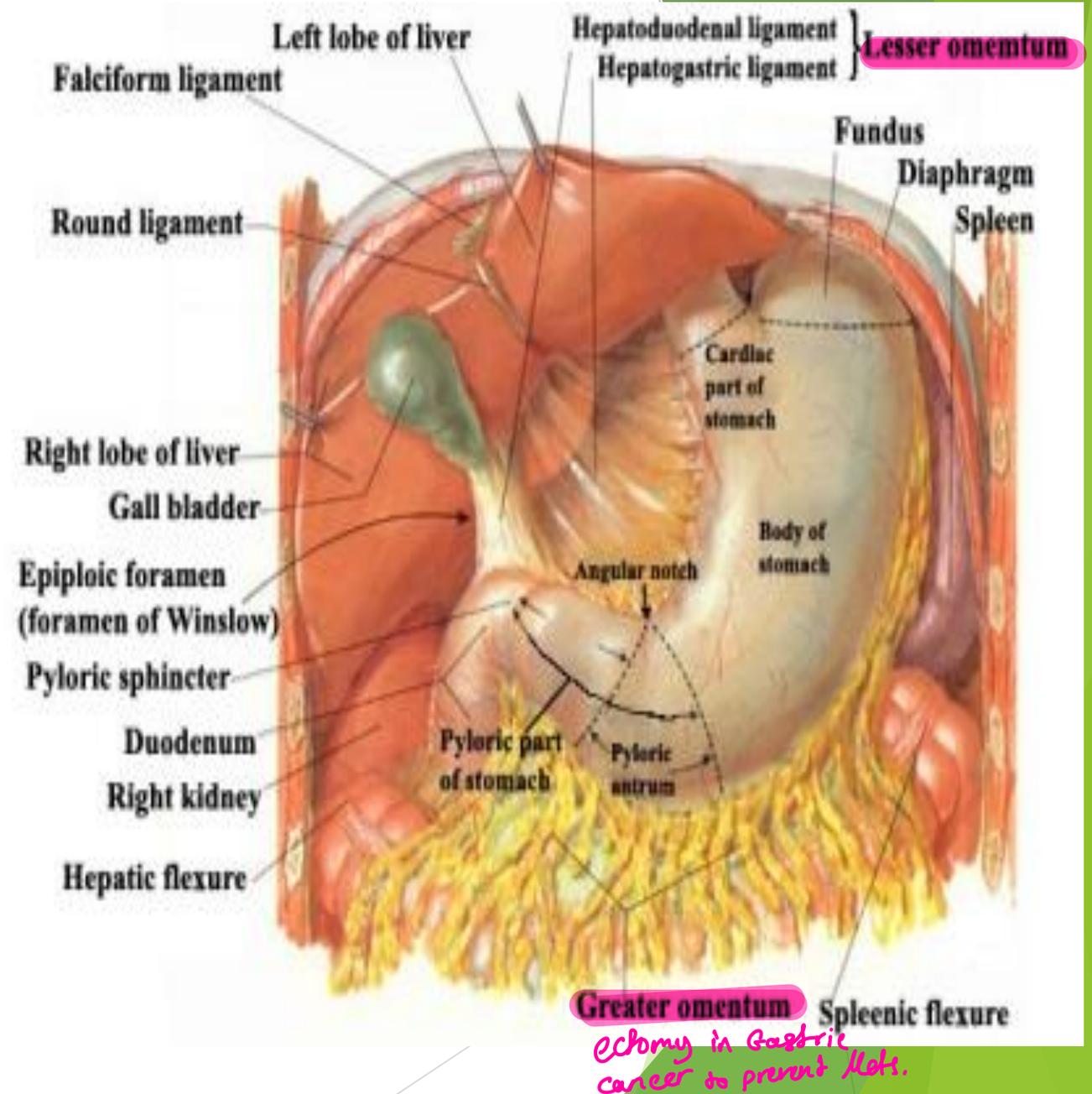


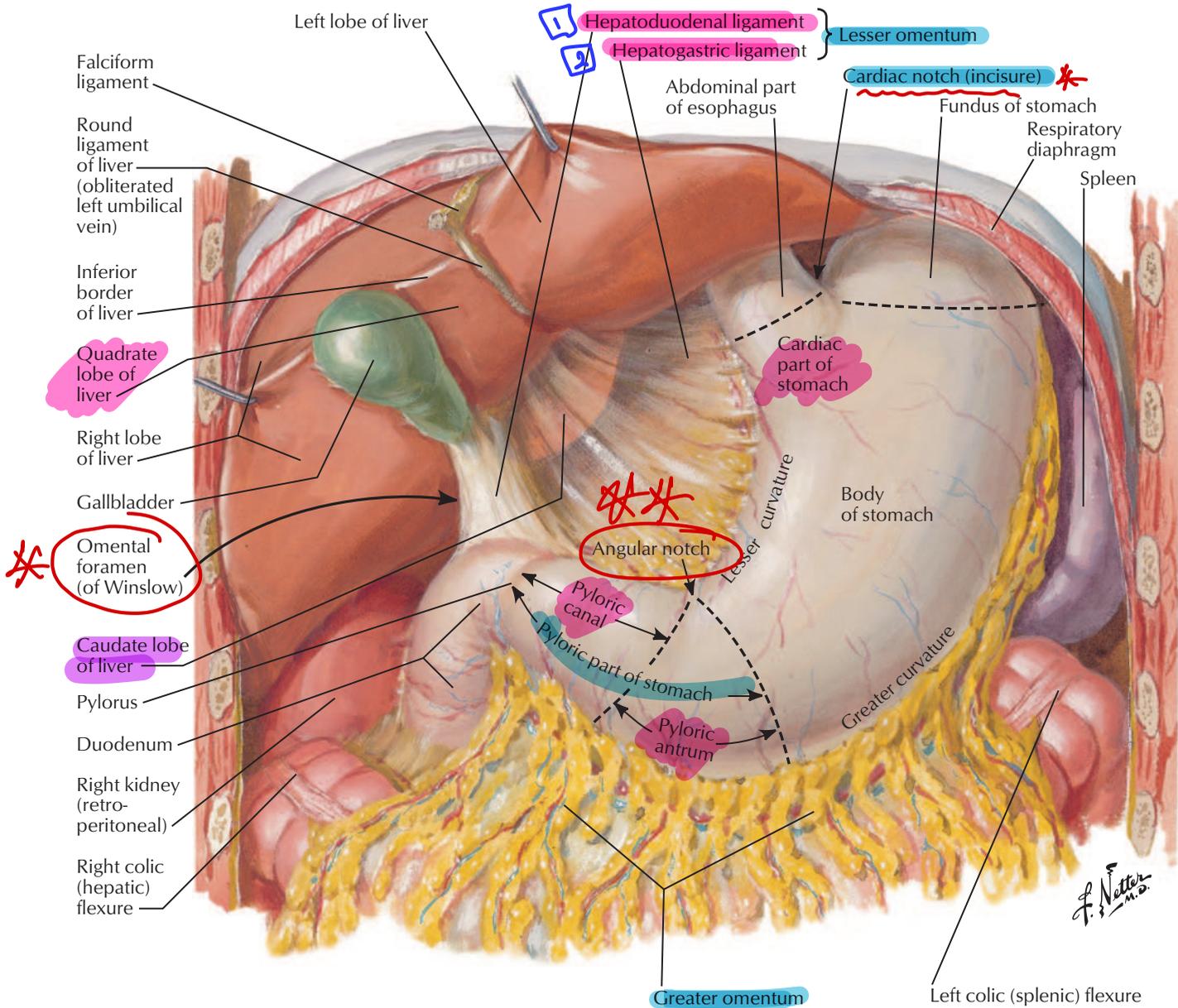
Parts of the stomach



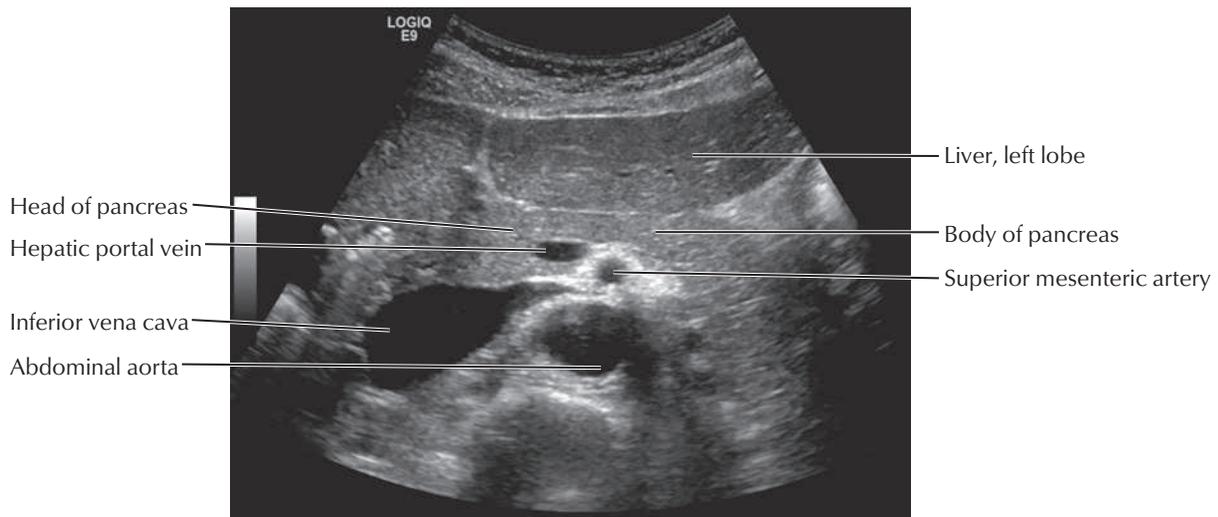
The stomach

- Layers of peritoneum attached to the stomach:
 - * **Lesser omentum**: attaches the liver to the lesser curvature.
→ removed in gastric Cancer.
 - * **Greater omentum**: attaches the greater curvature to the posterior body wall.



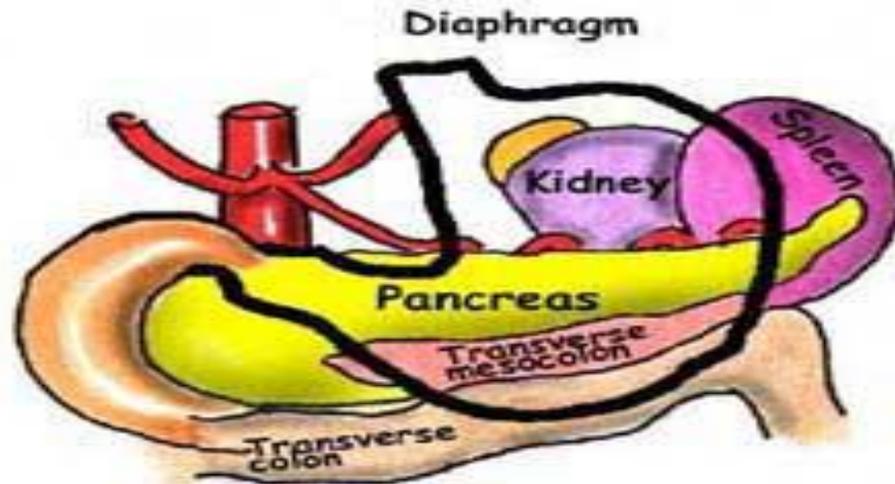


Transverse gray-scale ultrasound image of midabdomen



Stomach Anatomical Relation

STOMACH - RELATIONS



স্বাস্থ্য

ANTERIOR

- Abdominal wall
- Left costal margin
- Diaphragm
- Left lobe of liver

SUPERIOR

- Left dome of diaphragm

POSTERIOR

- Lesser sac
- Pancreas
- Transverse mesocolon
- Transverse colon
- Left kidney/suprarenal gland
- Spleen/splenic artery

Stomach Blood Supply

Celiac Trunk → Left Gastric. A
→ Splenic. A
→ Common Hepatic. A.

Arterial blood supply:

▶ 3 Branches

1 ▶ Left Gastric Artery

▶ Supplies the cardia of the stomach and distal esophagus

2 ▶ Splenic Artery

▶ Gives rise to 2 branches which help supply the greater curvature of the stomach

* ▶ Left Gastroepiploic

* ▶ Short Gastric Arteries

3 ▶ Common Hepatic or Proper Hepatic Artery

▶ 2 major branches

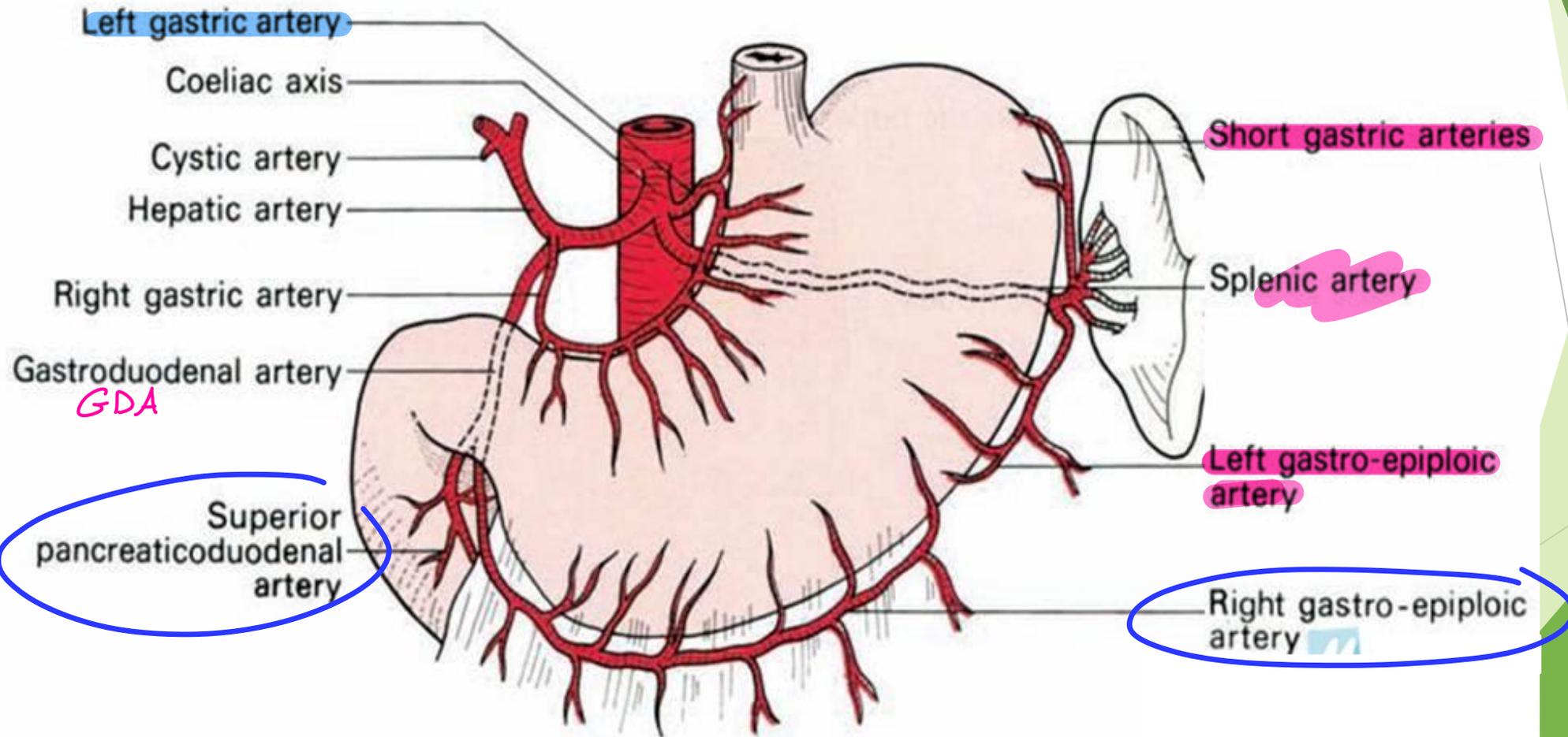
▶ Right Gastric- supplies a portion of the lesser curvature

▶ Gastroduodenal artery

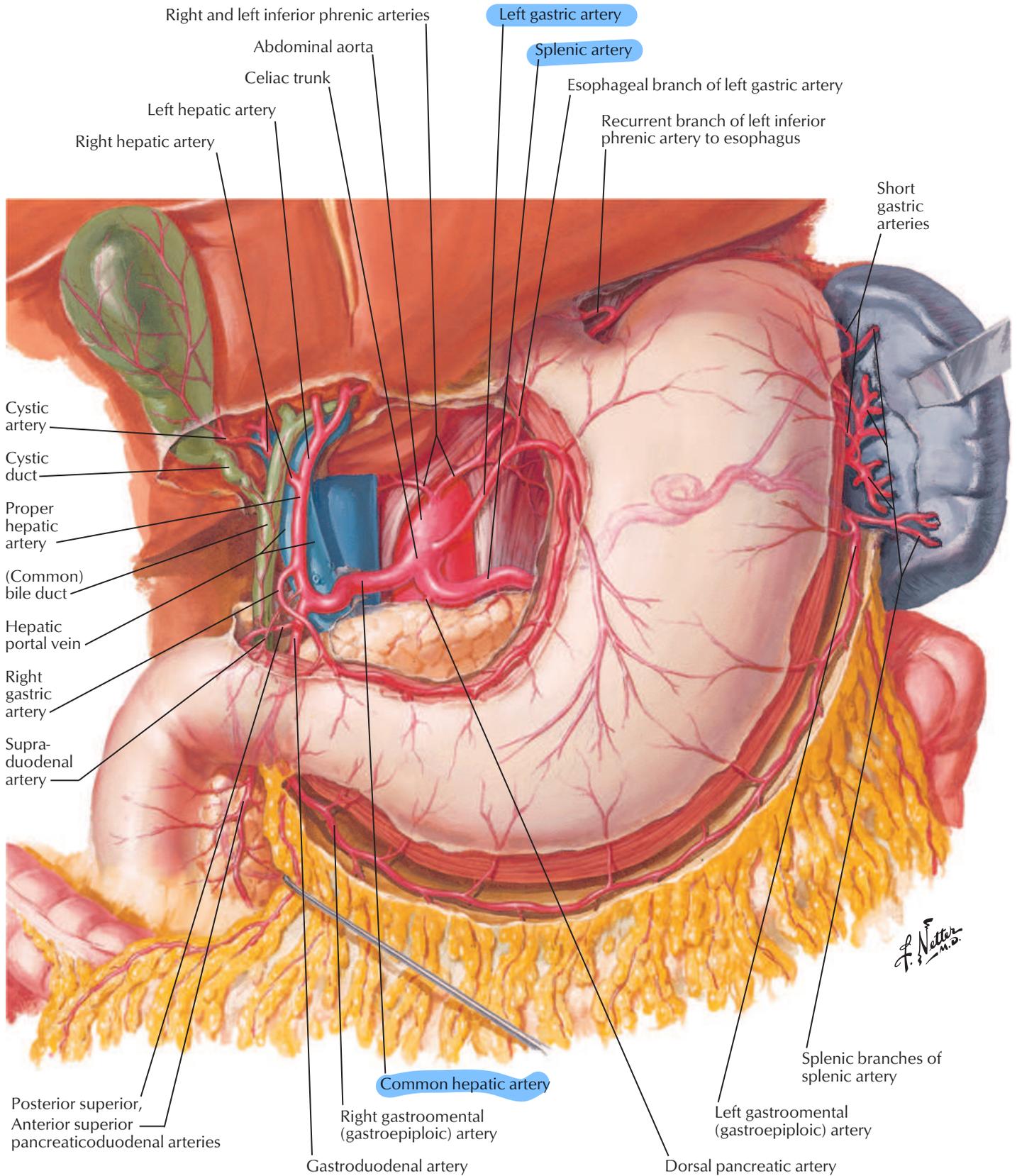
-Gives rise to Right Gastroepiploic artery

-helps supply greater curvature in conjunction with Left Gastroepiploic Artery

Blood supply of the Stomach

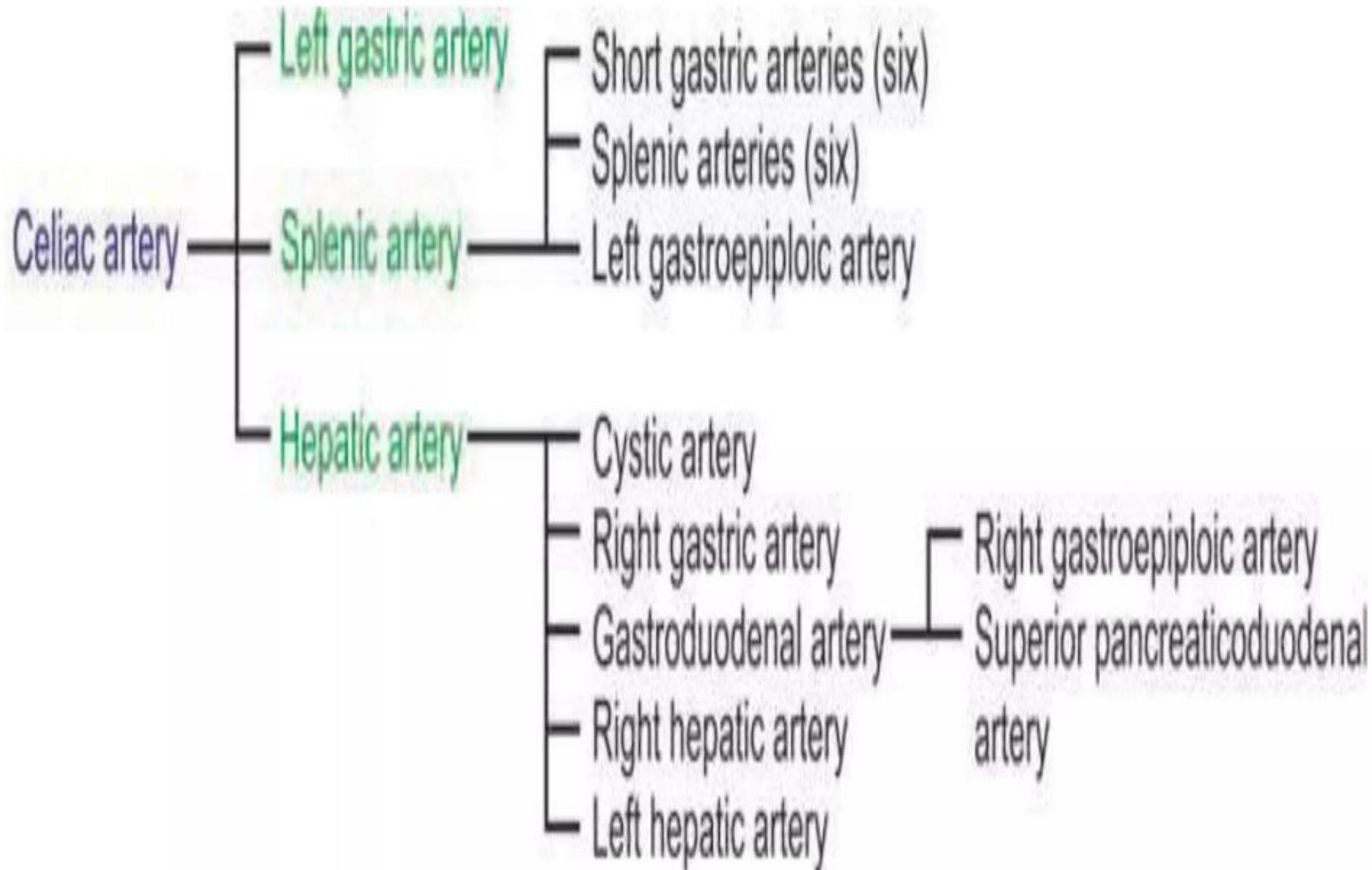


Arteries of Stomach, Liver, and Spleen



F. Netter M.D.

Arterial Supply Of Stomach

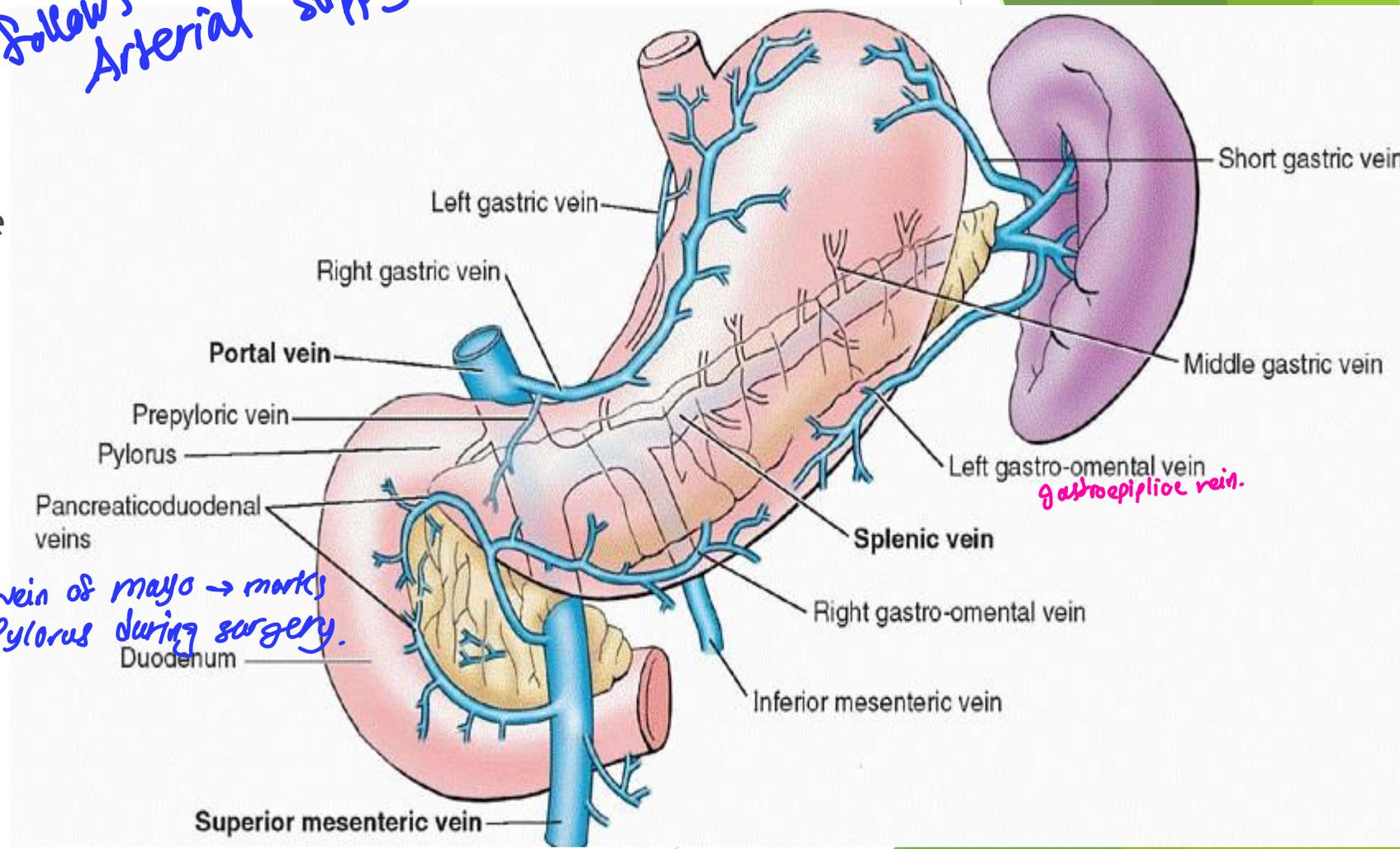


Stomach Venous Drainage

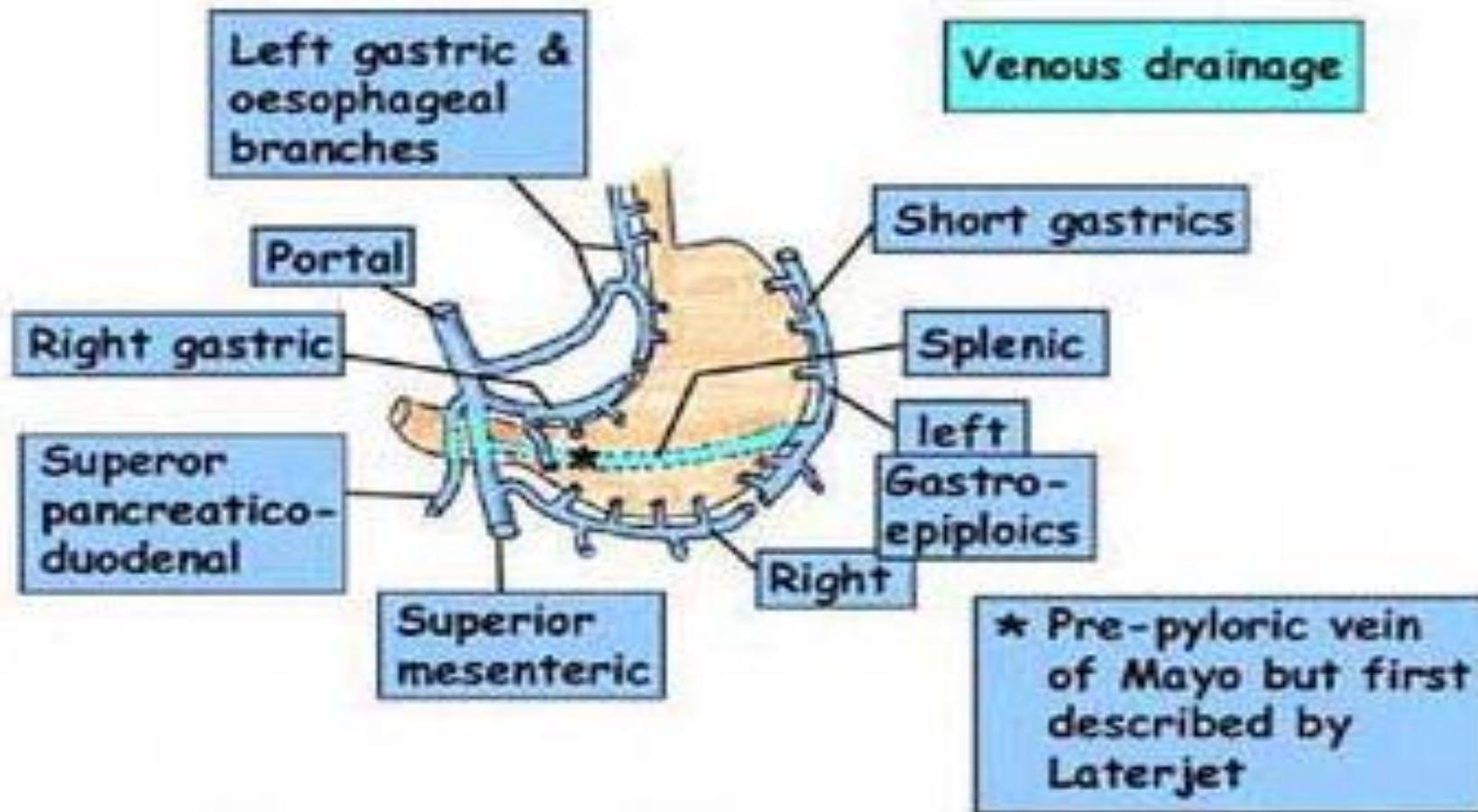
- ▶ Venous Drainage
 - ▶ Parallel to arterial supply
- ▶ Rt & Lt gastric veins drain to the portal
- ▶ Rt gastroepiploic drains to the SMV
- ▶ Lt gastroepiploic drains to the splenic
- ▶ Short gastric V. into splenic

Follows Arterial supply.

**vein of Mayo → marks Pylorus during surgery.*



Stomach Venous Drainage



Stomach Lymphatic Drainage

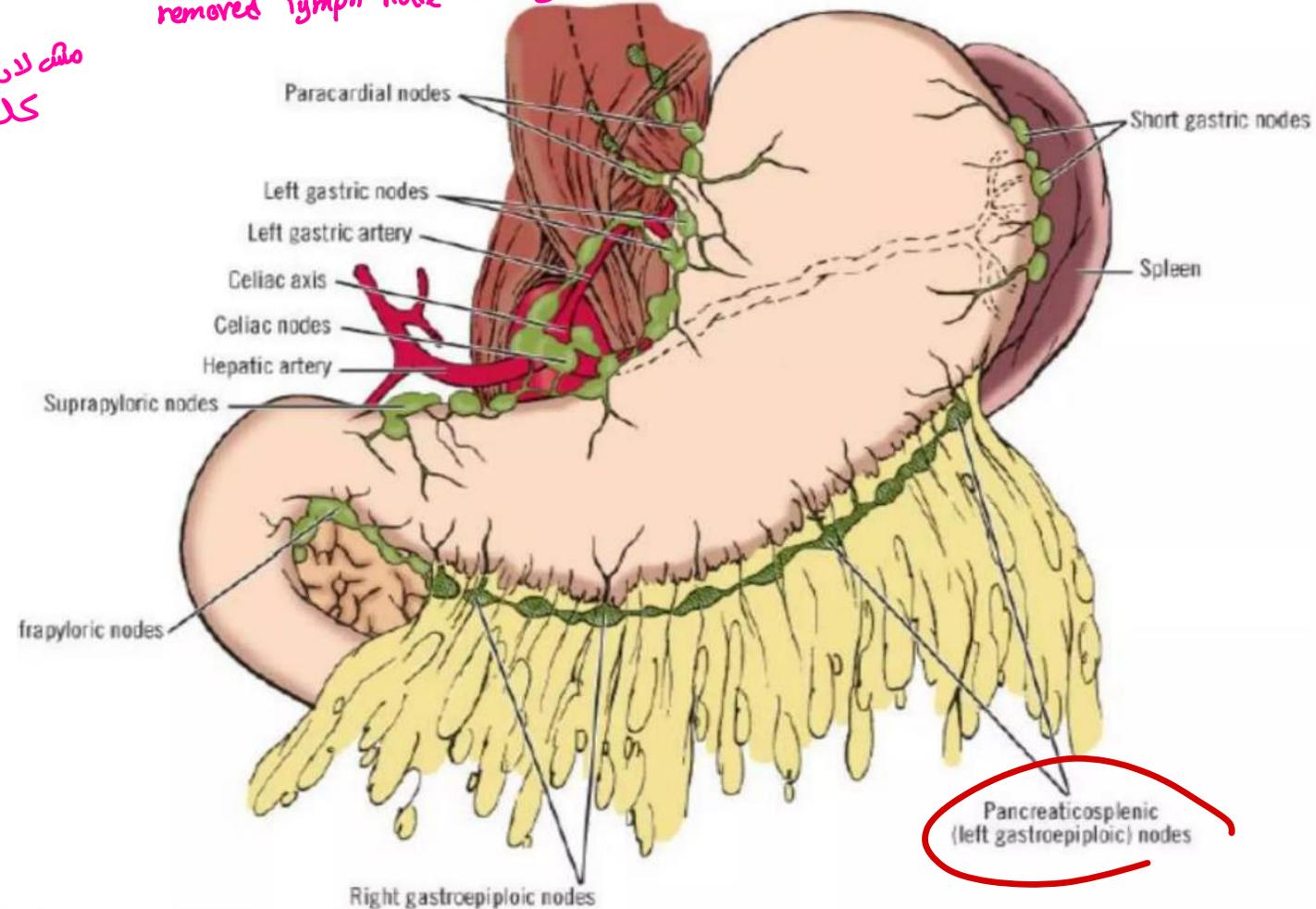
• Eight groups of lymph nodes of the stomach:

1. Paracardial nodes
2. Left gastric nodes at the left gastric artery
3. Celiac nodes at the celiac artery
4. Suprapyloric nodes
5. Infrapyloric nodes
6. Right gastroepiploic nodes at the pathway of the right gastroepiploic artery
7. Pancreaticosplenic nodes at the pathway of the left gastroepiploic artery
8. Upper greater curvature nodes at the short gastric vessels

8 lymph node stations.

منه لازم تحفظهم
كلهم

→ celiac + Hepatic + splenic.
*most cancer surgery mostly removed lymph node during surgery.



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Eight groups of lymph nodes of the stomach.

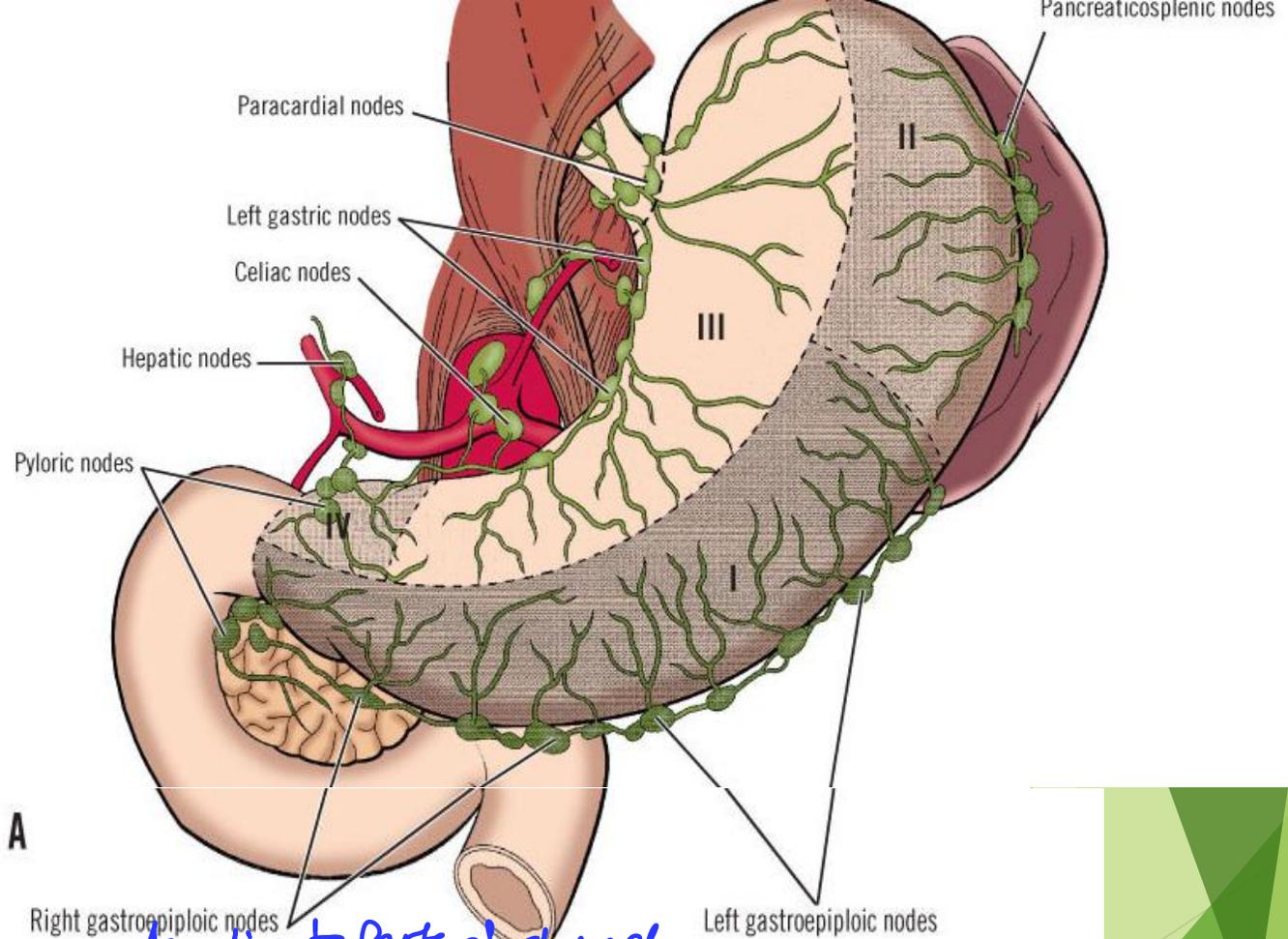
1. Left gastric lymph nodes: drains areas of both anterior and posterior gastric walls
2. Pancreaticosplenic nodes: drains gastric fundus and body
3. Right gastroepiploic nodes: drains the right half of the greater curvature, occasionally including the pylorus
4. Hepatic-pyloric-left gastric nodes: drain the pyloric part of the stomach

Zone 1 : left and right gastroepiploic LNs

Zone II : Pancreaticosplenic LNs

Zone III : left gastric + celiac LNs

Zone IV : pyloric + hepatic LNs

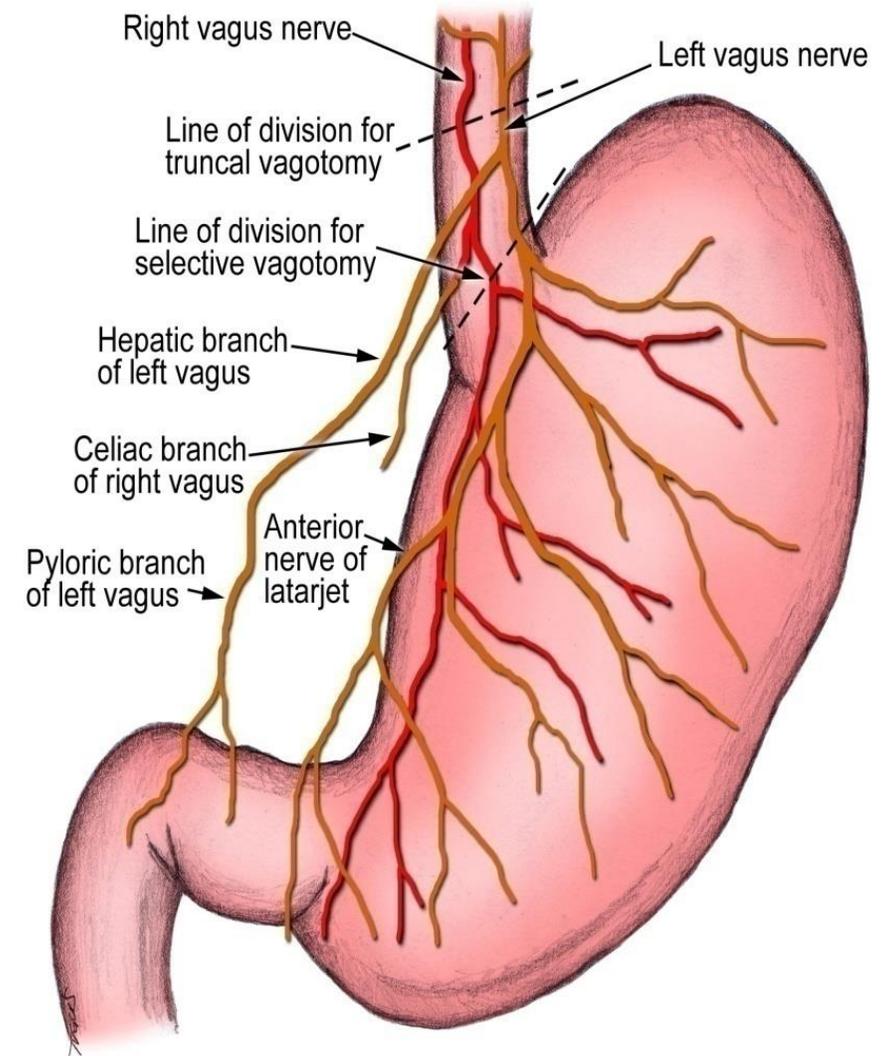


According to parts of stomach.

The 4 zones: I, Inferiorgastric; II, Splenic; III, Superior gastric IV, Hepatic.

Stomach Innervations

- **Parasympathetic innervation of Stomach-Vagus Nerve** *facilitate Gastric emptying.*
↳ Acid secretion.
- **80%** of fibers in vagal trunk is **afferent sensory** (info transmitting from stomach to CNS) **sensations of pain, fullness, and nausea** from the stomach.
- **20% motor fibers** for **gastric motility** and **relaxation of the pyloric sphincter** during gastric emptying.
- responsible for inducing **gastric acid secretion** also

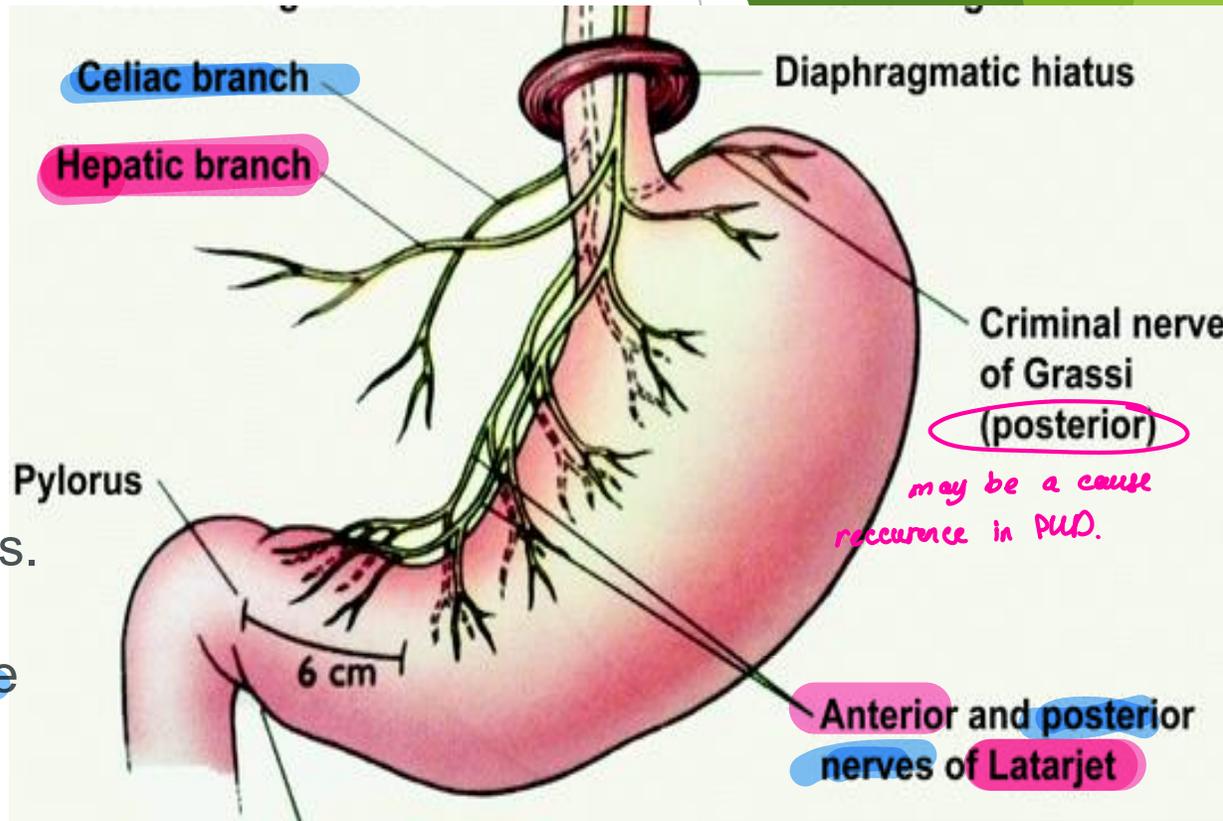


important anatomy
↓
Vagus trunk

responsible in Acid secretion.

**left become anterior
right become posterior.

- In the chest → Rt and Lt Vagal trunks
- At the level of the cardia, the left becomes anterior and the right becomes posterior
- The anterior vagal trunk divides into hepatic and anterior gastric (anterior nerve of Latarjet) branches.
- The posterior vagus divides into the posterior nerve of Latarjet and celiac branches.



- **criminal nerve of Grassi** is a proximal branch of the posterior vagal trunk as a possible cause of recurrent ulcers if left undivided during selective vagotomy.

usually from posterior

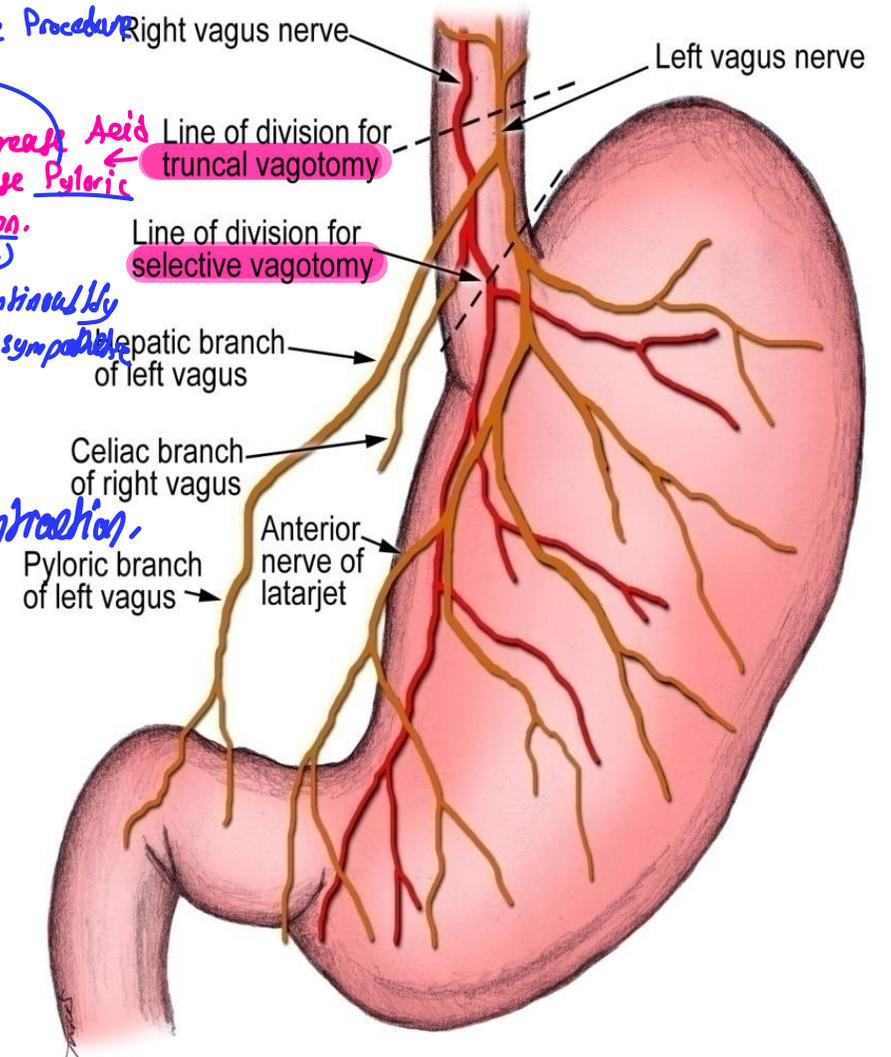
if forgotten to removed it leads to recurrent ulcer.

Levels of vagotomy

→ The most one that decrease Acid secretion. But there is a complication → Pyloric dysfunction.
 → Bcz we cut vagus before it give its branches to stomach.

so need drainage Procedure
 most one decrease Acid secretion & But cause Pyloric dysfunction.
 become continuously contracted by sympathetic upper hand
 → ass with drainage Procedure → Pylorus cut layer. to prevent continuous contraction.

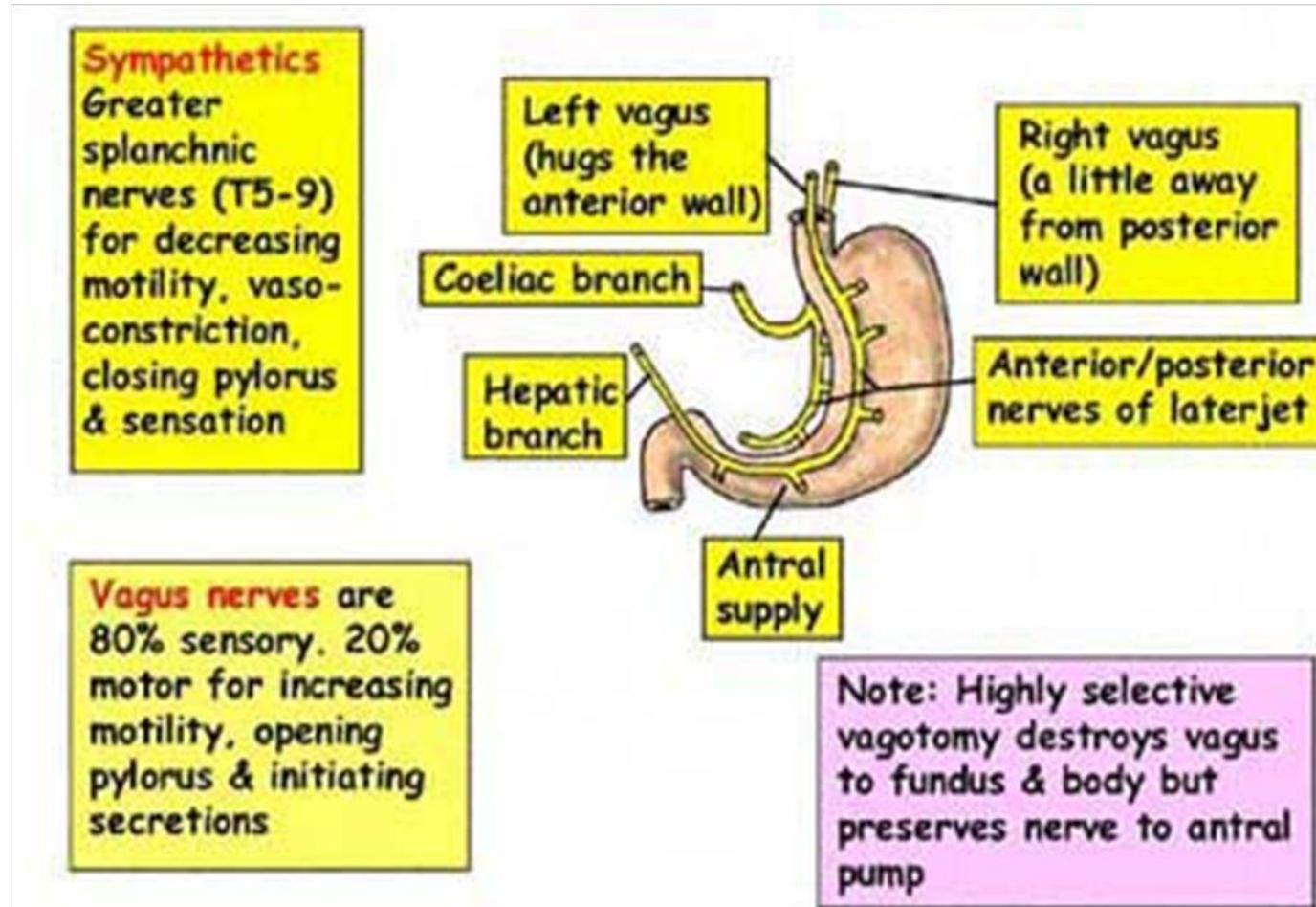
- ▶ **Truncal vagotomy** just below the diaphragmatic esophageal hiatus before it gives off celiac and hepatic branches.
- ▶ **selective vagotomy** is performed distal to this location and spares the celiac and hepatic branches.
- ▶ **Highly selective vagotomy** (parietal cell vagotomy) divides individual terminal branches of the nerve of Latarjet in the fundus and corpus of the stomach but spares the vagal branches to the antrum and pylorus, which control gastric motility and emptying—thus spares the need for a drainage procedure.

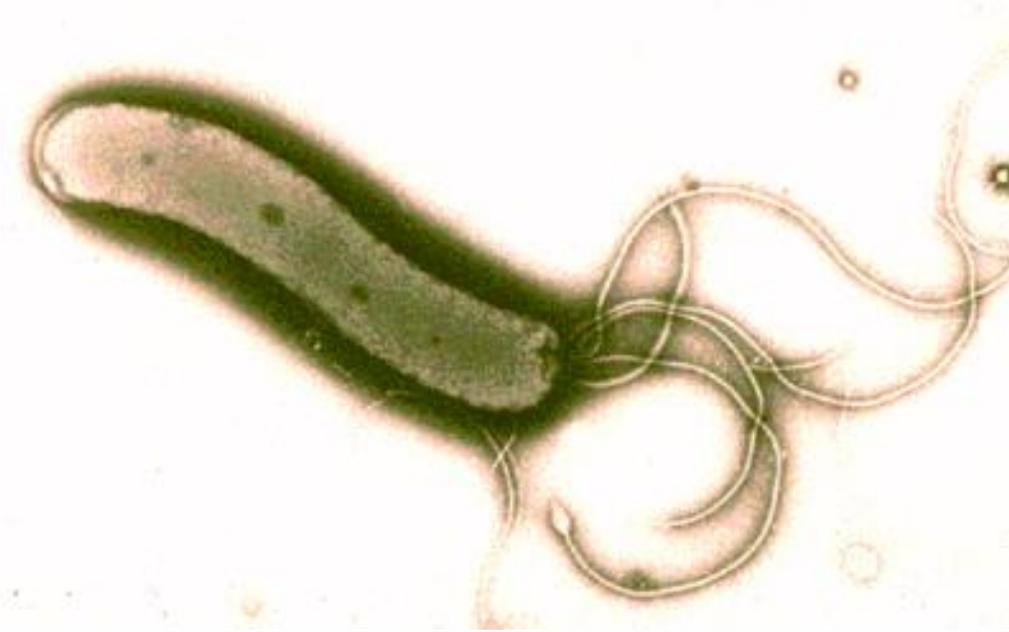


↳ less contribution than Parasympathetic.

- ▶ **sympathetic** innervation is provided by the **celiac plexus** from the fifth to twelfth thoracic spinal nerves (**T5-T10**) and travel to the celiac plexus via the **greater splanchnic nerves**.
- ▶ Sympathetic innervation is responsible for **inhibiting gastric motility** and **constricting the pyloric sphincter**, thus preventing gastric emptying.

Stomach Innervations





Helicobacter pylori infection



contributed to most Gastric disease.

▶ H. pylori is a curved or S-shaped, gram-negative microaerophilic motile bacterium whose natural habitat is the human stomach.

▶ although 2/3 of population are infected with H. pylori, only 10 – 15% develop PUD & the majority are asymptomatic

▶ Conditions associated with H.Pylori infection :

1 ▶ Acute gastritis → chronic gastritis

2 ▶ PUD : 90% of DU , 75% of Gastric Ulcers

▶ if Long standing → chronic gastritis → gastric atrophy & increased risk of metaplasia

4 ▶ Gastric adenocarcinoma

5 ▶ More than 90% of MALT lymphomas have H. pylori & low grade tumours regress with H. pylori eradication only (no need for chemotherapy if responded)

Mechanisms by Which *H. pylori* can Damage the Gastric Mucosa

not important

Local effects
Elaboration of toxins
vacA
cagA
Effect on immune response
Elaboration of cytokines
Elaboration of interleukin 8
Recruitment of inflammatory cells
Release of inflammatory mediators
Production of immunoglobulins
Effect on acid secretion
Initial hypochlorhydria
Subsequent hyperchlorhydria
Elevated serum gastric levels
Reduced gastric antral somatostatin levels
Increased levels of gastric fundic N-methylhistamine
Hypergastrinemia may contribute to greater parietal cell mass
Effect on duodenal bicarbonate secretion
Reduced secretion of duodenal bicarbonate in patients colonized with <i>H. pylori</i>

reduce HCO_3^- secretion

- ▶ In patients infected with *H pylori*, high levels of gastrin and pepsinogen and reduced levels of somatostatin have been measured. In infected patients, exposure of the duodenum to acid is increased. Virulence factors produced by *H pylori*, including urease, catalase, vacuolating cytotoxin, and lipopolysaccharide, are all well described.
- ▶ Impair duodenal bicarb secretion which predisposes to DU .

Pathogenesis

- 1-Increases fasting & postprandial gastrin
- 2-Increases pepsinogen secretion
- 3-Decreases gastric mucosal resistance
- 4-Suppresses somatostatin release
- 5-Releases tissue damaging cytotoxins

Diagnoses

▶ Noninvasive tests

most used to detect Eradication success.

① urea breath test

detect urease enzyme function of H. pylori

~~✗~~

- ▶ analyzes breath for labeled carbon dioxide produced by bacterial urease from the conversion of ingested labeled urea. Because of its noninvasiveness plus high sensitivity and specificity (95%), the urea breath test is considered the test of choice for screening and for documentation of H. pylori eradication.

② serology. (serum antibodies, fecal antigen)

- ▶ Serologic tests are quick and inexpensive but cannot differentiate between active infection and previous exposure. Serology is useful for the initial diagnosis of H. pylori infection in patients in whom endoscopy is not indicated.

▶ **Invasive tests** require endoscopic mucosal biopsy and include histologic examination, the rapid urease test, and culture.

1 ▶ **Histologic examination** ^{quick tests} can accurately diagnose H. pylori with two biopsy specimens with high sensitivity and specificity (90%).

2 ▶ **The rapid urease test** ^{on a Biopsy.} on a mucosal biopsy is considered the initial test of choice because of its simplicity, accuracy, and rapid results.

3 ▶ **Culture of H. pylori** ^{only for research purpose & very expensive} has the most specificity (100%) but is difficult to perform (only for research purposes or patients with suspected antibiotic resistance).

↳ most specific But usually not done.

Treatment : Eradication therapy

A) P.P.I based triple therapy with two antibiotic

B) H2 blockers based triple therapy

*Bismuth is included in some regimens

Table 1. First-line treatment recommendations

Therapy	Dosing	Duration (days)	Eradication (%)
Clarithromycin triple	PPI twice daily + clarithromycin 500 mg twice daily + amoxicillin 1 g twice daily or metronidazole 500 mg three times daily	14	70%-85%
Bismuth quadruple	PPI twice daily + bismuth salicylate 300 mg four times daily + metronidazole 500 mg three times daily + tetracycline 500 mg four times daily	14	75%-90%
Non-bismuth quadruple	PPI twice daily + amoxicillin 1 g twice daily + clarithromycin 500 mg twice daily + metronidazole 500 mg three times daily	14	90%

PPI, proton pump inhibitor

Peptic ulcer disease : PUD



▶ **Peptic ulcers** are defects in the gastric or duodenal mucosa that extend through the muscularis mucosa.

▶ Loss of normal balance between protective mechanisms and acid secretion/ aggressive factors

▶ Normal protective mechanisms :

- tight intercellular junctions, mucus production, bicarbonate, mucosal blood flow, cellular restitution, Prostaglandin E , and epithelial renewal.

- Risk factors :

- 1 H pylori infection
- 2 Drugs (NSAIDS, steroids)
- 3 Severe physiologic stress (burns/curling ulcer, head trauma/ cushing ulcer.... etc) لا حقا بالتفصيل
- 4 Hypersecretory states (uncommon) – Zollinger Ellison , G-cell hyperplasia
- 5 Genetic factors
- 6 smoking

due to eradication Therapy.

▶ Incidence of PUD is decreasing due to H.pylori eradication and anti secretory therapy

Differential Diagnosis of Hypergastrinemia

With excessive gastric acid formation (ulcerogenic)

* Zollinger-Ellison syndrome *Pancreatic islet cells tumor (primary tumor).*

* Gastric outlet obstruction

Retained gastric antrum (after Billroth II reconstruction)

G-cell hyperplasia

Without excessive gastric acid formation (nonulcerogenic)

* Pernicious anemia

* Atrophic gastritis

Renal failure

Postvagotomy → decrease acid secretion.
→ Body will increase Gastrin secretion as a feedback.

Short gut syndrome (after significant intestinal resection)

History and Exam

- ▶ *Chronic mostly.*
Epigastric pain, (gnawing or burning)
- Dyspepsia, including belching, bloating, distention, and fatty food intolerance
- Heartburn
- Chest discomfort
- Hematemesis or melena. → *as a complication.*
- anemia (eg, fatigue, dyspnea)
- Sudden onset of symptoms may indicate perforation.
- NSAID-induced gastritis or ulcers may be silent, especially in elderly patients.
- ▶ On exam : normal or minimal findings if uncomplicated (minimal epigastric tenderness)
- ▶ Signs of GOO (if strictures and obstruction) or peritonitis (if perforation occurred)

	<u>Duodenal Ulcer</u>	<u>Gastric ulcer</u>
Relived by	Eating	<u>Lying down</u> or <u>vomiting</u>
Duration	1-2 months	Few weeks A
Vomiting	Uncommon	<u>Common</u> (to relieve the pain)
Appetite	Good	<u>Pt afraid to eat</u>
Diet	Good , eat to relieve the pain	Avoid fried food
Weight	No wt. loss	<u>wt. Loss maybe present</u>
At night	<u>Pain awaken pt</u>	Less night pain
	A	

~~XA~~ ~~XA~~

Alarming symptoms:

1. Relentless pain *→ pain resistance to Tx.*
 2. Bleeding or anemia ~~XA~~
 3. Early satiety
 4. Unexplained weight loss
 5. Progressive dysphagia
 6. Recurrent vomiting
 7. Family history of gastrointestinal cancer
- → all need endoscopy + biopsy to be performed

workup

**This not for all patients
→ every patient has specific test.*

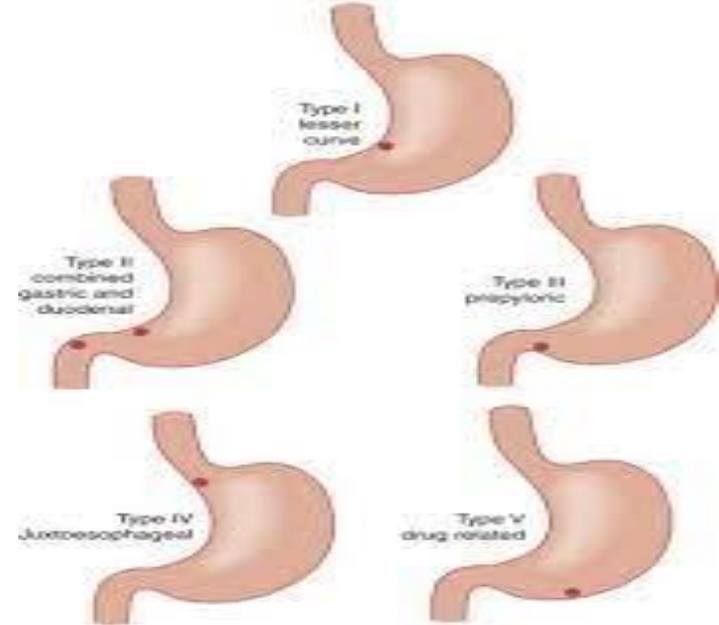
- 1 H. pylori testing
- 2 Endoscopy + biopsy → for all suspected PUD especially if alarming symptoms present or new onset/refractory symptoms in >50 years old patients.
- 3 Erect CXR (if perforation suspected) → for Acute Abdomen in ER.
- 4 Serum gastrin level (if multiple, unusual location, +FHx, unidentifiable cause, +diarrhea, +steatorrhea, weight loss, ulcer refractory to treatment) → to exclude ZES
- 5 Secritin stimulation test (for gastrinoma / ZES)
- 6 Angiography+ Embolization (if massive bleeding)

Treatment :

- ▶ H.pylori eradication (triple therapy)
- ▶ Confirm eradication of H.pylori and healing of ulcer with followup endoscopy
- ▶ Stop NSAIDs, PGE analogue (misoprostol) , prophylactic PPI
- ▶ SURGICAL treatment :
 - Now largely abandoned because of high success rate for medical therapy.
*resistance PUD & complication. *indication for surgery become rare nowadays.*
 - Surgery is indicated only in refractory ulcers not responding to medical treatment (12weeks)
 - or if complications developed (bleeding not responding to endoscopic management)
 - Perforation, GOO , Suspicion of malignancy

Modify Johnson's classification

بسته اسید



Type	Location	Acid Hypersecretion
I	Lesser curvature, incisura	No
II	Body of stomach, incisura, and duodenal ulcer (active or healed)	Yes
III	Prepyloric	Yes
IV	High on lesser curve, near gastroesophageal junction	No
V	Anywhere (medication induced)	No

ass. with Acid Hypersecretion.

بسته اسید

Antrectomy only

Type II, III treated as duodenal ulcer, preferred tx include antrectomy (to include ulcer) + truncal vagotomy with Billroth I OR II

Modified Antrectomy including ulcer+ billroth I OR subtotal gastrectomy with Roux en Y without vagotomy

SURGICAL OPTION

- VAGOTOMY

- ✓ Truncal and drainage
- ✓ Selective
- ✓ Highly selective
- ✓ Posterior vagotomy and anterior seromyotomy

- GASTRECTOMY

- Billroth I
- Billroth II
- Subtotal gastrectomy

damage control surgery ←

- GRAHAM'S OMENTAL PATCH

→ when there is Perforation.

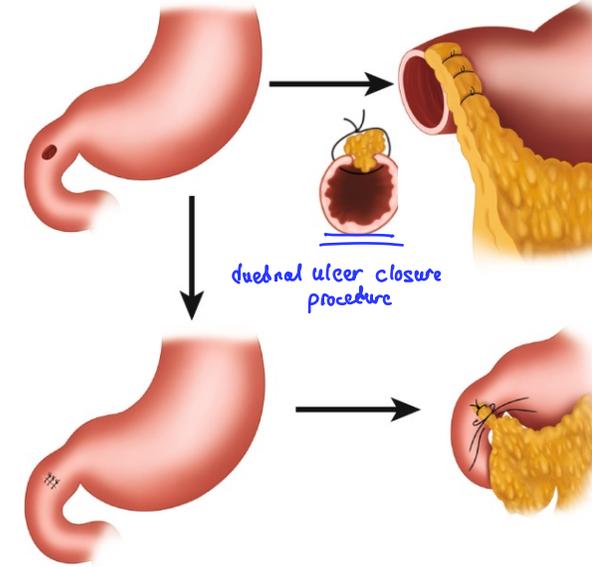
- SUTURE LIGATION OF GASTRODUODENAL ARTERY

→ for bleeding control

- UNDER-RUNNING AN ULCER BASE

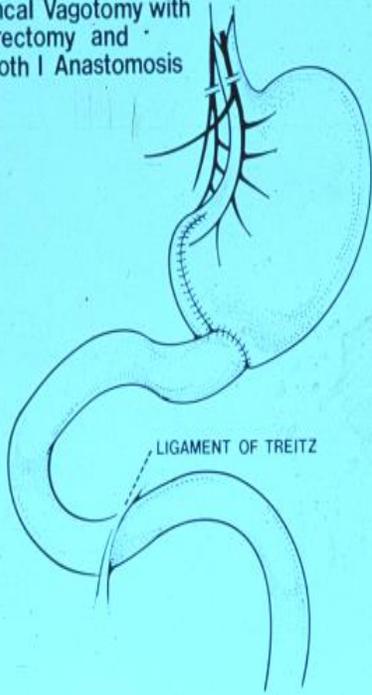
- After excision of the edge
- Vagotomy

Graham omental Patch

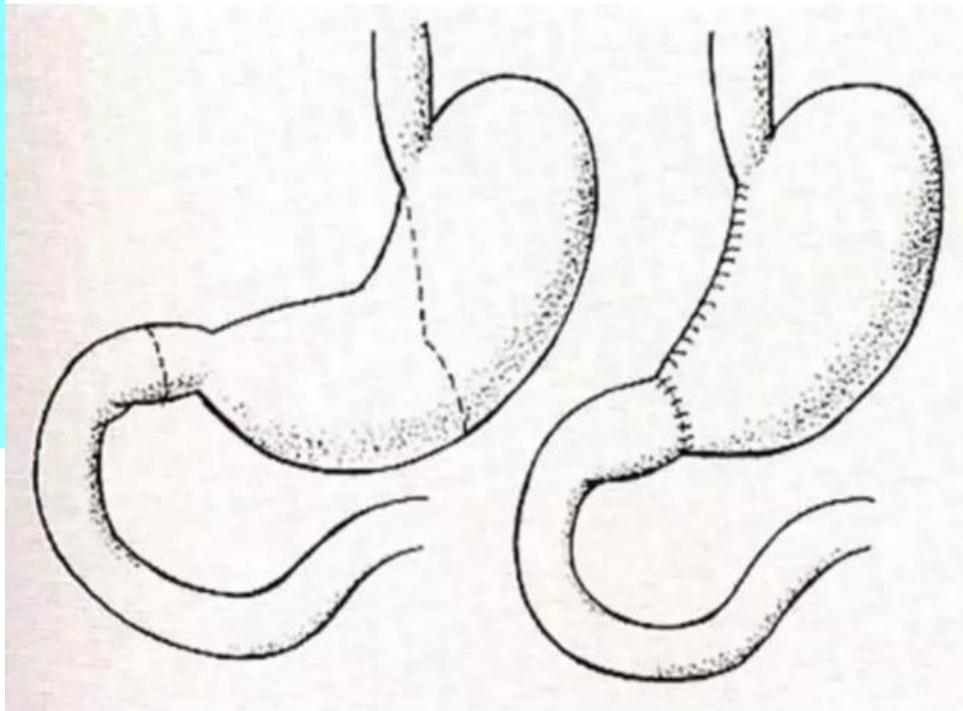


Billroth B → one limb.
↳ stomach continuous with duodenum.
تقطيع و تقييد

Truncal Vagotomy with Antrectomy and Billroth I Anastomosis



Billroth I – partial gastrectomy gastro-duodenostomy end-to-end
Done for gastric ulcer in the antrum

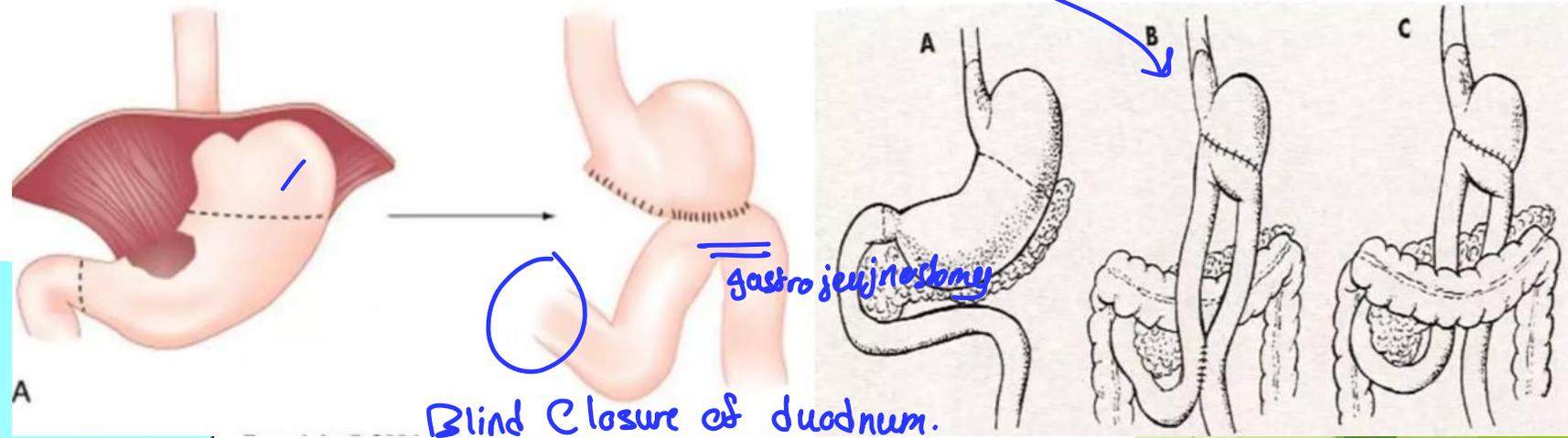


Billroth I gastroduodenal anastomosis completed

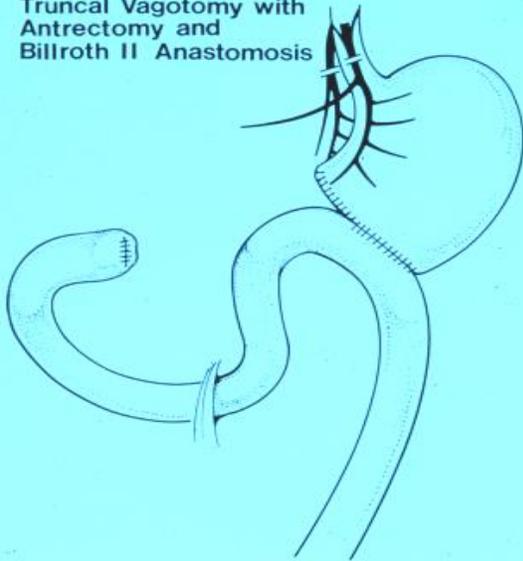
Billroth II 2 ends.

Partial gastro-jejunostomy end-to-side with blind closure of duodenum

Done for a proximal gastric ulcer



Truncal Vagotomy with Antrectomy and Billroth II Anastomosis



Roux-en-Y.

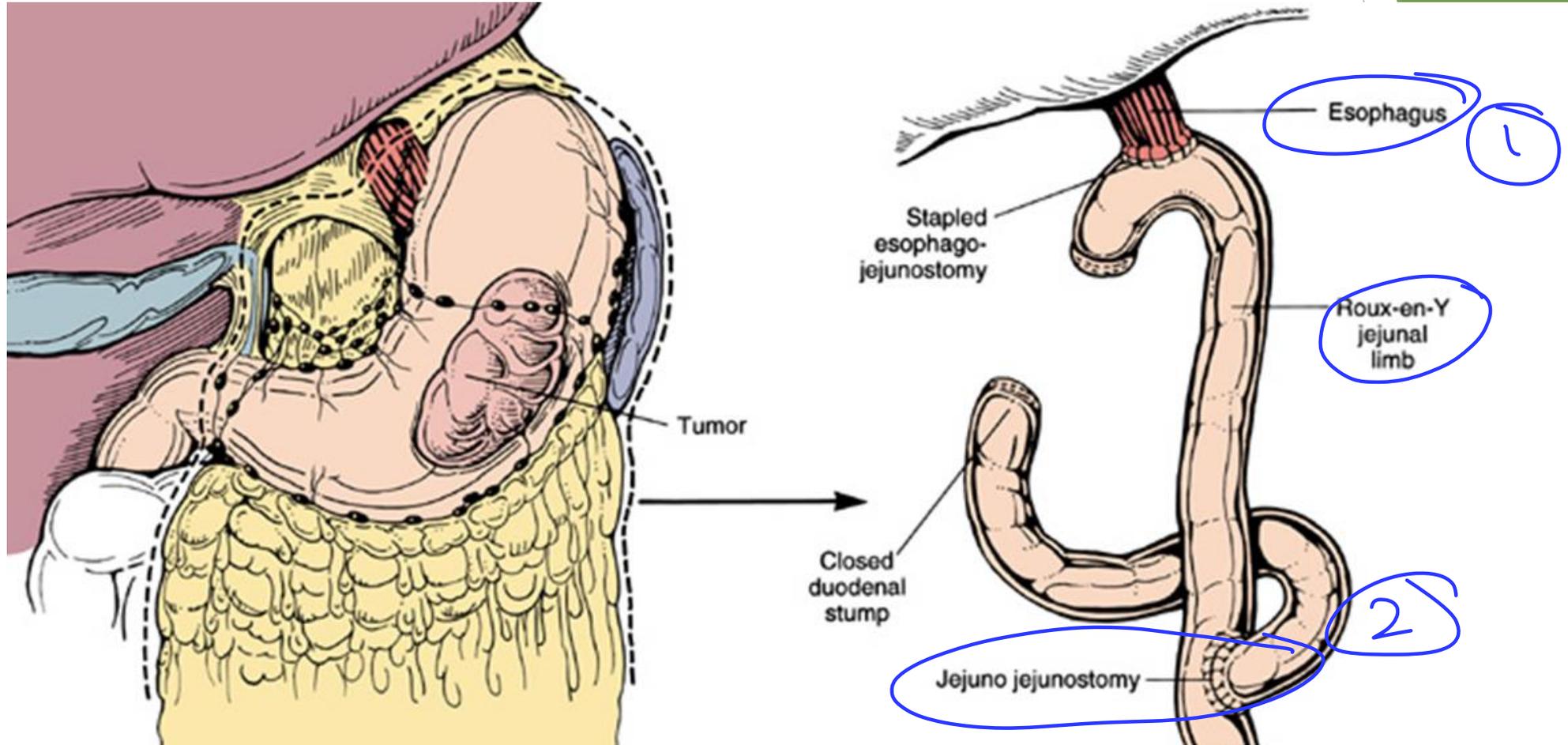


Table 25-12 Surgical Options in the Treatment of Duodenal and Gastric Ulcer Disease

منه مطلوب

Indication	Duodenal	Gastric
Bleeding	<ol style="list-style-type: none"> 1. Oversew^a 2. Oversew, V+D 3. V+A 	<ol style="list-style-type: none"> 1. Oversew and biopsy^a 2. Oversew, biopsy, V+D 3. Distal gastrectomy^b
Perforation	<ol style="list-style-type: none"> 1. Patch^a 2. Patch, HSV^b 3. Patch, V+D 	<ol style="list-style-type: none"> 1. Biopsy and patch^a 2. Wedge excision, V+D 3. Distal gastrectomy^b
Obstruction	<ol style="list-style-type: none"> 1. HSV + GJ 2. V+A 	<ol style="list-style-type: none"> 1. Biopsy; HSV + GJ 2. Distal gastrectomy^b
Intractability/nonhealing	<ol style="list-style-type: none"> 1. HSV^b 2. V+D 3. V+A 	<ol style="list-style-type: none"> 1. HSV and wedge excision 2. Distal gastrectomy

to R/O cancer.

^aUnless the patient is in shock or moribund, a definitive procedure should be considered.

^bOperation of choice in low-risk patient.

V : vagotomy
 HSV: highly selective vagotomy
 D: drainage pyloroplasty
 A: antrectomy
 GJ: gastrojejunostomy

امراض
الجهاز الهضمي

Duodenal	Highly-selective anterior vagotomy combined with posterior truncal vagotomy or seromyotomy combined with posterior truncal vagotomy
Gastric	Type I: Distal gastrectomy Type II: Antrectomy with vagotomy Type III: Antrectomy with vagotomy Type IV: Subtotal gastrectomy
Uncertain diagnosis*	The same than in case of refractory ulcer
Gastric outlet obstruction*	Highly-selective vagotomy with gastrojejunostomy (if balloon dilatation fails)
Bleeding duodenal ulcer	Partial gastrectomy (less rebleeding rate) Suture oversewing (less long-term side effects)
Bleeding gastric ulcer	Ulcer excision (variable from wedge excision to partial gastrectomy)
Perforated duodenal ulcer	Simple closure Acid secretion reduction procedure if continuation of NSAIDs is predictable
Perforated gastric ulcer	Partial gastrectomy Biopsy and simple closure in case of patient poor condition

Table 2 Indications for surgery and recommended techniques. *After excluding diseases other than peptic

For duodenal ulcers :

* duodenal ulcer unstable with bleeding → over suture *Artery* *بند*

*

- Acid can be reduced by eliminating vagal stimulation, removing the antral source of gastrin, and removing the parietal cell mass.
- subtotal two-thirds gastrectomy has the highest mortality rate. (abandoned)
- Truncal vagotomy with antrectomy ^{→ to ↓ acid production} has the lowest recurrence rate.
- Procedures involving antrectomy, pyloroplasty, or truncal vagotomy may be complicated by diarrhea, postprandial dumping, or bile reflux.
- Selective vagotomy, which preserves the hepatic and celiac vagal branches, has been associated with a lower rate of diarrhea than truncal vagotomy has.
- Highly selective vagotomy, preserve innervations to the pyloro-antral region and thus maintain more normal gastric emptying. This operation carries the lowest mortality rate, the lowest incidence of side effects (morbidity) , but the highest recurrence rate, which ranges from 5% to 15%.

Table 25-13 Clinical Results of Surgery for Duodenal Ulcer

	<i>(Highly selective)</i> Parietal Cell Vagotomy	Truncal Vagotomy and Pyloroplasty	Truncal Vagotomy and Antrectomy
Operative mortality rate (%)	0	<1	1
Ulcer recurrence rate (%)	5-15	5-15	<2
Dumping (%)			
Mild	<5	10	10-15
Severe	0	1	1-2
Diarrhea (%)			
Mild	<5	25	20
Severe	0	2	1-2

*الدراقام مش حفظ.

(Highly selective)

ass. with highest recurrence rate

↳ the lowest mortality.

↳ the most ass. with recurrence

↳ best result there is no recurrence

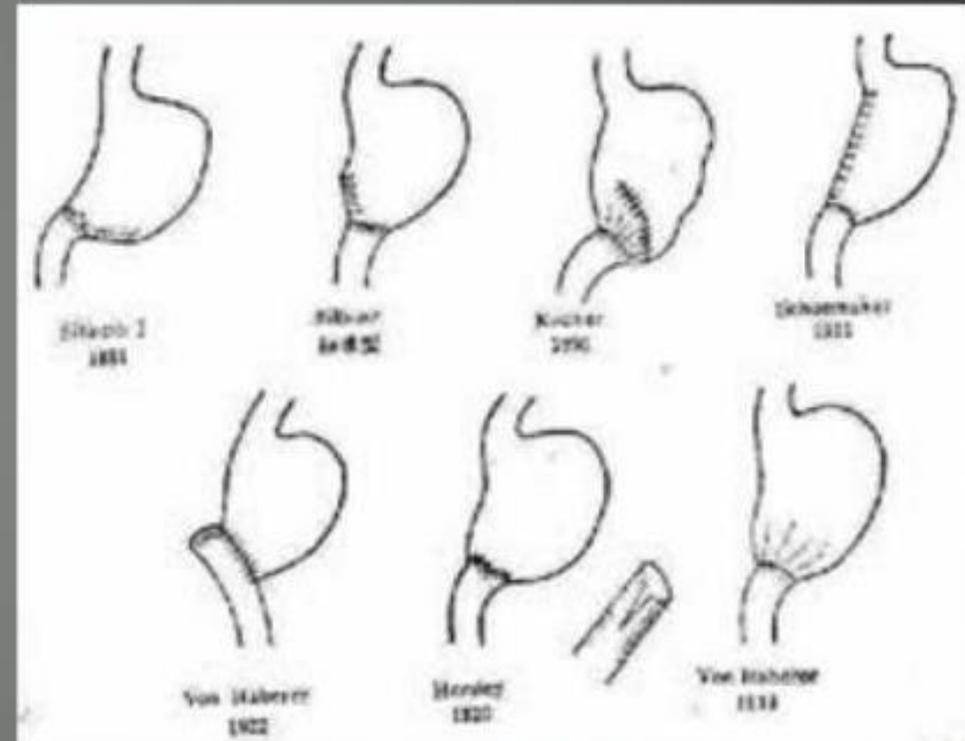
↳ The highest mortality rate.

SOURCE: Modified with permission from Mulholland MW, Debas HT: Chronic duodenal and gastric ulcer. *Surg Clin North Am* 67:489, 1987.

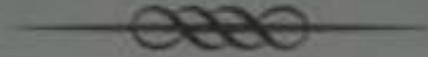
Billroth I



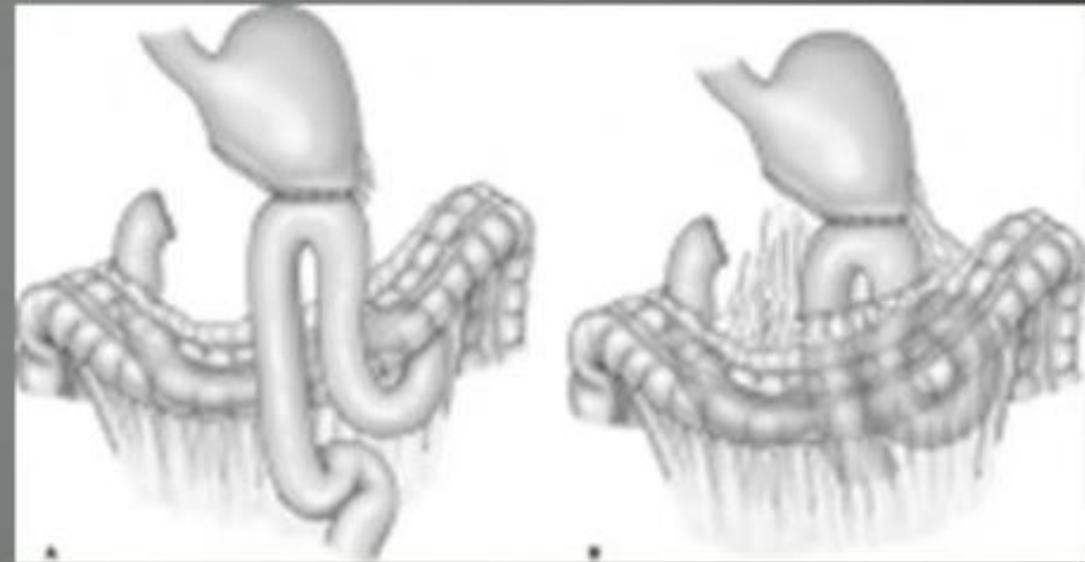
- ⌘ Preserves duodenal passage
- ⌘ Preserves pancreatic function
 - ⌘ Altered after gastrojejunostomy
 - ⌘ Better fat digestion
- ⌘ Less incidence of gastritis and reflux
- ⌘ Can use only with distal limited gastric resections
tension



Billroth II



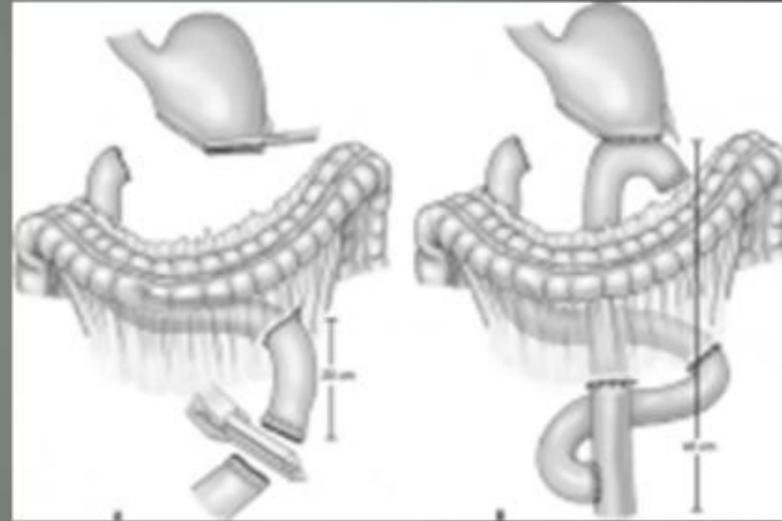
- Can be used for larger gastric resections
- Can result in dumping syndrome



Roux-en-Y



- ❧ Described by Cesar Roux
 - ❧ Late 1800's
 - ❧ Gastric outlet obstruction
- ❧ Better control of enterogastric reflux
- ❧ Method of choice for early dumping or reflux



Post-gastrectomy Syndromes

- ▶ A wide spectrum of diseases occurs after gastrectomy
- ▶ Due to denervation of stomach → the pyloric mechanism becomes incompetent and the control of stomach emptying is impaired

- ① ▶ Early Dumping syndrome ✓
- ② ▶ Late dumping syndrome ✓
 - ▶ Post-vagotomy Diarrhea
- ③ ▶ Afferent loop syndrome / efferent loop syndrome ✓
 - ▶ Blind loop syndrome
- ④ ▶ Alkaline Reflux gastritis ✓
 - ▶ Roux stasis syndrome
 - ▶ Recurrent ulcers
 - ▶ Gastric atony
 - ▶ Metabolic disturbances

الحمى
بسه هزول.

TABLE 1. Post gastrectomy syndromes: classification

Acute complications	<ul style="list-style-type: none">- Anastomotic leak- Duodenal stump blowout
Gastric reservoir dysfunction	<ul style="list-style-type: none">- <u>Dumping syndrome</u> (Types I and II)- Metabolic alterations
<u>Vagal denervation</u>	<ul style="list-style-type: none">- <u>Gastric stasis and hurry</u>- <u>Gallstones</u>
Aberrations of reconstruction	<ul style="list-style-type: none">- Bile reflux gastritis- Afferent loop syndrome- Efferent loop syndrome- Jejunogastric intussusception- Roux stasis syndrome- Gastro-jejuno-colic fistula
Long term complication	<ul style="list-style-type: none">- Recurrent ulcerations- Remnant stump cancer

loss of pylorus junction
فقدان نقطة
small bowel.

Dumping syndrome :

▶ Pathophysiology : **loss of normal pylorus mechanism** , food directly introduced into small bowel

▶ **Early** dumping :

- **Occurs 15-30 mins after meal**

osmotic effect → Rapid fluid shift → Hypotension.

- **High osmolarity food** dumped directly into small bowel → **Fluid shifts rapidly from intravascular space into intraluminal space** producing acute intravascular volume depletion.

- **tachycardia**, **diaphoresis**, **palpitations** , **Abd pain/cramps**, **borborygmi** , diarrhea, weakness.

- As simple sugars are absorbed and dilution of hypertonic solution occurs the symptoms decreased.

- **Occurs more with Billroth I, II** . **LESS in Roux en Y reconstruction.**

Late dumping :

- ▶ 2-3 hours after meals, due to excessive insulin release causing transient hypoglycemia and insulin/glucose imbalance .

Exaggerated insulin release → hypoglycemia.

- ▶ Sudden anxiety, diaphoresis, tremor, tachycardia, palpitations, weakness, fatigue.

- ▶ It is not associated with borborygmi or diarrhea. *Bez no fluid shift*

- ▶ Pathophysiology:

- ▶ Rapid changes in serum glucose and insulin levels.

- ▶ Large glucose-bolus containing chyme presented to small intestine , glucose is absorbed faster than when the intact pylorus controls emptying of stomach.

→ improve by decrease glucose.

- ▶ This causes high levels of serum glucose shortly after meal and causes a big released of insulin. Insulin response exceeds what is necessary to clear glucose from blood and hypoglycemia symptoms happens. Symptoms are due to rapid fluctuations in serum glucose levels.

Diagnosis



- ❧ Usually made clinically - classic history
 - ❧ Onset of symptoms 10-30mins after meal
 - ❧ Most commonly after high carb meal
 - ❧ Crampy discomfort, belching, nausea, fecal urgency
 - ❧ Light headedness, blurred vision, diaphoresis, flushing, dizziness, hypotension, syncope
 - ❧ Usually no true abdominal pain
- ❧ 50% glucose solution – reproduces symptoms
- ❧ Gastric emptying studies – rapid emptying

Treatment of dumping syndrome :

90% of patients will improve.

► Diet modification (most pts will improve)

- Smaller more frequent meal bulks.

*less Bolus meal

- Lower carbs in food (less osmolar food)

* صبح بدال ما یا کل ۳ وجبات کبار ← یا کل ۷ وجبات صغیر *

- Avoid mixing liquids and solids (Liquids should be ingested before meals or 30 min after meals.)

- Ingest some fat to slow gastric emptying



► somatostatin analogue (octreotide) for refractory cases.

► In minority of pts who don't improve → might need conversion of Billroth to Roux en Y OR creaton of anti peristaltic interposition jejunal loop

► Conversion of Billroth II to Billroth I can be an option

early patient need surgery. → less than 10% of patient

Vagal Denervation : Diarrhea

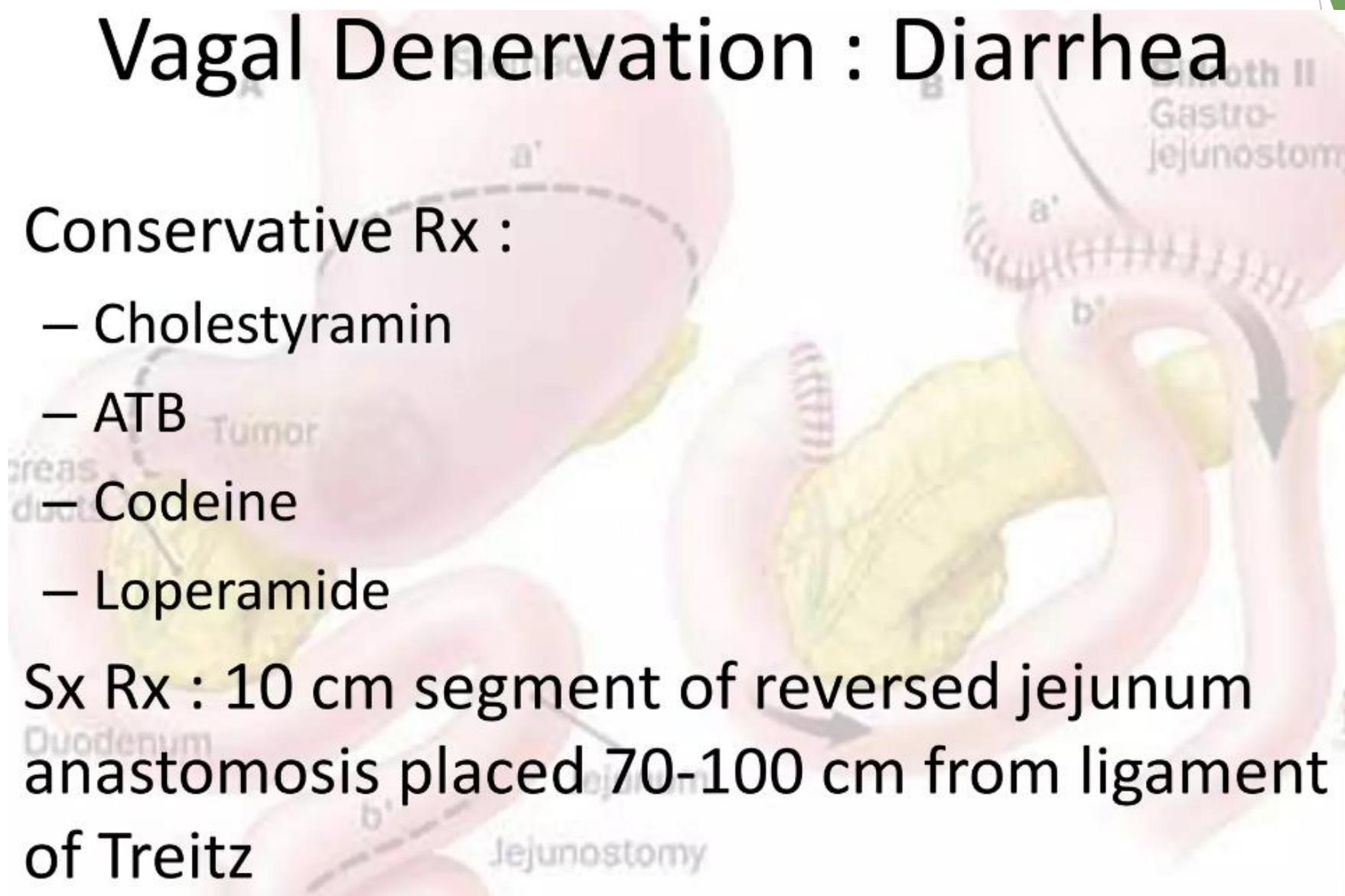
- Presented with diarrhea
- Mostly diarrhea from dumping syndrome, but some may not
- Post vagal resection >> uncontrolled bowel movement >> increased stool frequency
- Other mechanism: bile acid malabsorption, rapid gastric emptying time and bacterial overgrowth

Vagal Denervation : Diarrhea

Conservative Rx :

- Cholestyramin
- ATB
- Codeine
- Loperamide

Sx Rx : 10 cm segment of reversed jejunum anastomosis placed 70-100 cm from ligament of Treitz



Vagal Denervation : Gastric stasis

- Conservative Rx :
 - Metoclopramide
 - Domperidone
 - Erythromycin
- NJ tube feed

Gastroparesis
- Early satiety
- distended stomach
- recurrent vomiting.

Pancreas
and duct

Gastro-
jejunostom

Gastroparesis



- ⌘ Most common syndrome
- ⌘ ~50% of pts thought to have symptoms
- ⌘ Definition is highly variable
 - ⌘ Hard to get true incidence

Diagnosis



- ⌘ Most pts diagnosed if not taking adequate po intake 7-14 days post-op after gastric procedure
- ⌘ Symptoms: nausea, bloating, fullness, early satiety, vomiting
- ⌘ nuclear study.
⌘ Gastric emptying studies – normal: 60% solid, 80% liquid clearance at 60 min.
- ⌘ Nuclear medicine or thin barium vs gastrograffin

Acute Gastroparesis



Causes:

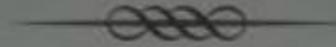
Metabolic/Neuronal

- Release of norepi and inhibition of acetylcholine in immediate post-op period
- Electrolytes – hypomagnesemia, hypokalemia
- Endocrine – hypothyroidism, DM
- Medications – opiates, anticholinergics, antidepressants

Functional

- Preoperative gastric outlet obstruction-affects contraction
- Effects of truncal vagotomy – mostly solids
- Stomal edema, adhesions, kinking, hematoma, intussusception

Treatment



Conservative

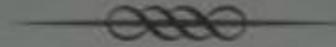
- ⌘ NGT decompression
- ⌘ Prokinetic agents
 - ⌘ Bethanechol, Reglan, Erythromycin Cisapride – off market
- ⌘ Correction of lytes
- ⌘ patience

After failed treatment

- ⌘ Minimum of 3-4 wks
- ⌘ No improvement – re-explore
- ⌘ Look for mechanical causes
- ⌘ Place feeding tube – jejunostomy
- ⌘ Gastric pacing – experimental

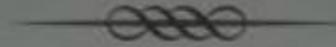


Chronic Gastroparesis



- ⌘ Diagnosis of exclusion – rule out stricture, internal hernia, stomal edema, intussusception
- ⌘ ~2% of patients after gastric surgery – mostly after truncal vagotomy
- ⌘ Symptoms start later in the post-op period

Chronic Gastroparesis



Diagnosis

- ⌘ Symptoms – early satiety, nausea, vomiting, postprandial bloating, hiccups, belching
 - ⌘ Increase throughout the day
- ⌘ Emesis of food ingested days earlier – pathognomonic
- ⌘ Need UGI and EGD to rule out other syndromes

Treatment



Conservative treatment

- ☞ Same as acute gastroparesis
- ☞ More emphasis on maximizing response to prokinetic agents

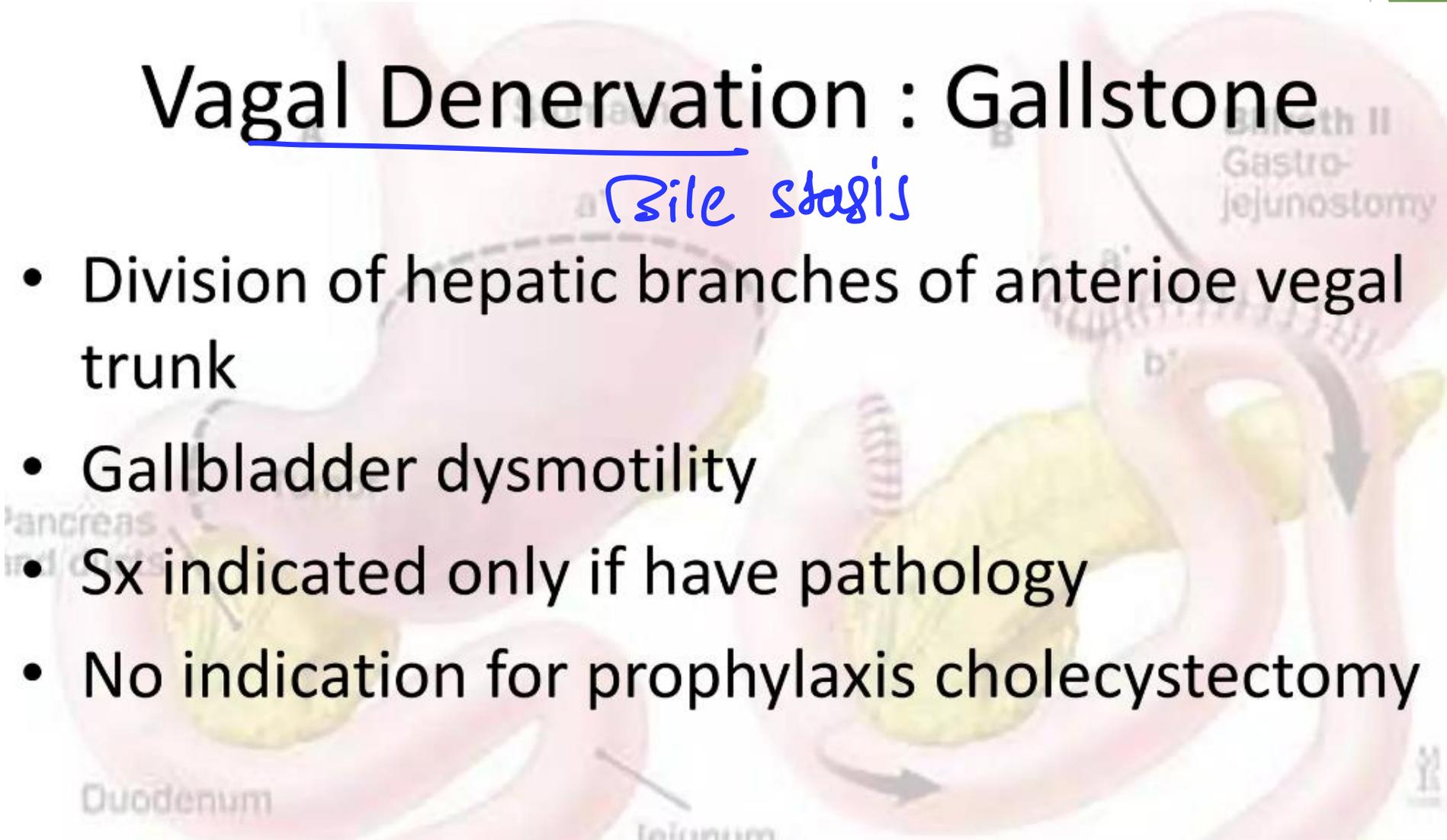
Surgical treatment

- ☞ Resection of atonic portion
- ☞ Using a different type of reconstruction
- ☞ Only total gastrectomy may be curative
- ☞ Gastric pacing – some improvement at 6 mo, but disappears at 1 yr

Vagal Denervation : Gallstone

Bile stasis

- Division of hepatic branches of anterior vagal trunk
- Gallbladder dysmotility
- Sx indicated only if have pathology
- No indication for prophylaxis cholecystectomy



Abberation in Reconstruction

- ✱ Bile reflux gastritis *Bile go up to stomach in Billroth II rather than going down → lead to Billous vomiting.*
- ✱ • Afferent and efferent loop obstruction
 - Jejunogastric intussusception
 - Roux syndrome

Bile Reflux Gastritis

usually in Billroth II.

- Most patient no symptoms
- Reflux symptoms: epigastric pain, N/bilious vomiting
- Dx by clinical + evidence of bile reflux (scope or scan)
 - if pain doesn't improved by vomiting*
- Scope :
 - mucosal erythema that involve parastomal region
 - bile staining or pooling
 - observed enterogastric reflux

most important feature

Bile Reflux Gastritis

- Rx : no significant medication benefit
- Sx : divert bile and pancreatic secretion from stomach

• ~~Roux-en-Y gastrojejunostomy (Roux limb at least 45 cm)~~

~~– Interposition 40 cm of isoperistaltic jejunal loop~~

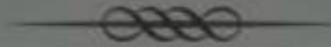
~~– Braun enteroenterostomy~~

Alkaline Reflux Gastritis



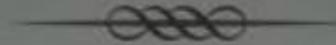
- ⌘ Reflux of intestinal contents into the stomach
 - ⌘ Significant mucosal injury causing symptoms
- ⌘ Debilitating in 1-2% of post gastrectomy pts.
- ⌘ ~20% of healthy controls have duodenogastric reflux
 - ⌘ Also increased in pts with cholelithiasis/ cholecystectomy
- ⌘ More common in Billroth II > B I > V&P

Pathophysiology



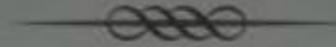
- ⌘ Refluxate contents – bile, pancreatic enzymes, other intestinal secretions
- ⌘ Some disagreement – bile salts + gastric acid = decreased barrier function of mucosa
 - ⌘ Causes gastritis and symptoms

Diagnosis



- ⌘ Again a diagnosis of exclusion
- ⌘ Symptoms: usually start ~1yr post-op
 - ⌘ Fatigue, malaise, weight loss
 - ⌘ Constant, burning epigastric pain, worse with food
 - ⌘ Persistent nausea, always bilious emesis
 - ⌘ Pain characteristically not alleviated with emesis

Diagnosis



- ⌘ **Endoscopy** EGD – classic beefy red, edematous gastric mucosa
 - ⌘ Sharp demarcation of mucosa at anastomosis
- ⌘ **نوعی** **scan** HIDA – to evaluate gastric reflux index
 - ⌘ <5% - healthy, normal
 - ⌘ 5% - 45% - asymptomatic, after BII
 - ⌘ >60% - symptomatic
- ⌘ Bernstein test – reproduction of symptoms with gastric infusion of Na hydroxide

Treatment

المعالم

*conversion

Billroth II → Roux-en-Y

Conservative

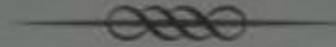
- Doesn't work as well
- High fat/protein diet
- PPI, carafate, reglan
- Cholestyramine – binds bile
- Most promising –
Ursodeoxycholic acid
Resolution of symptoms

Surgical

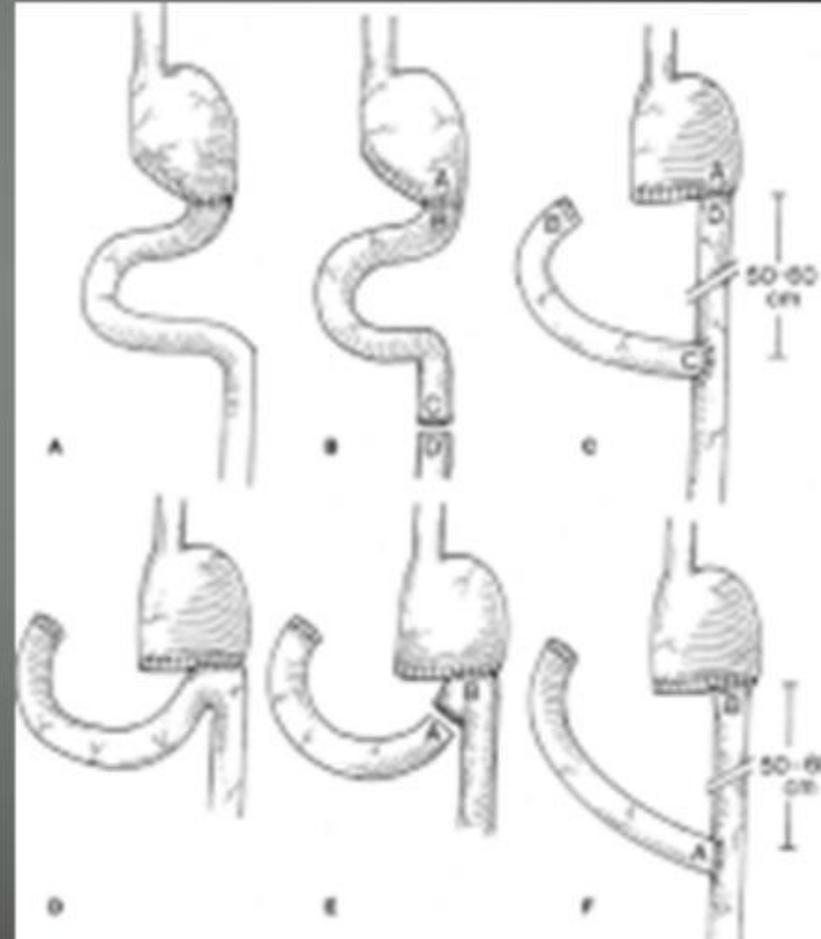
mostly rather than conservative

- Braun procedure
- Distal enteroenterostomy
- Henley loop
- Roux-en-Y reconstruction

Roux-en-Y Reconstruction

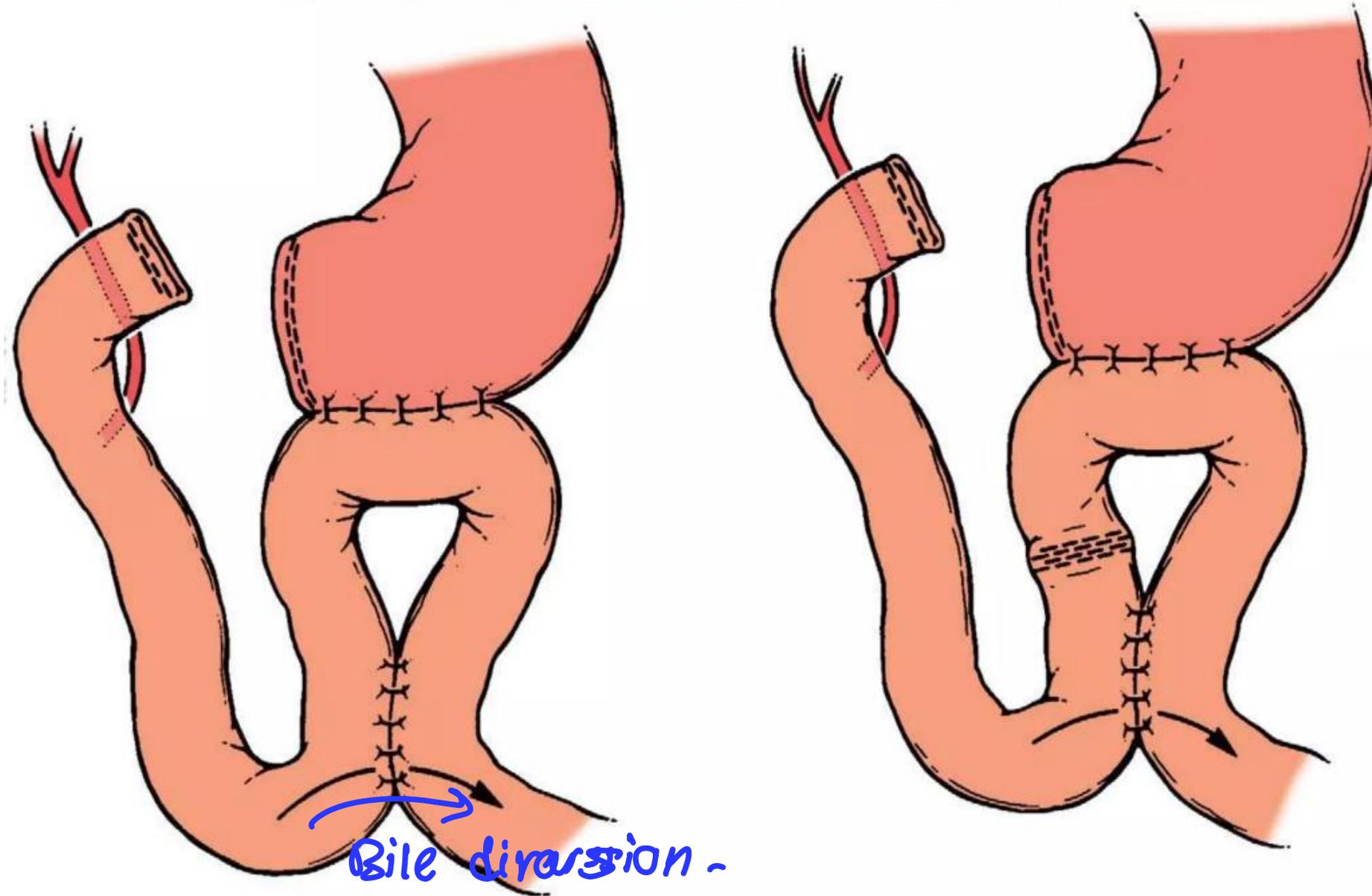


- ❧ Procedure of choice
- ❧ Long Roux limb – 45-60cm
- ❧ Symptoms relieved in 80% of pts
- ❧ Ulcerogenic, ~30% gastroparesis
 - ❧ Evaluate original path for complete antrectomy and gastric motility



Bile Reflux Gastritis

Billroth II

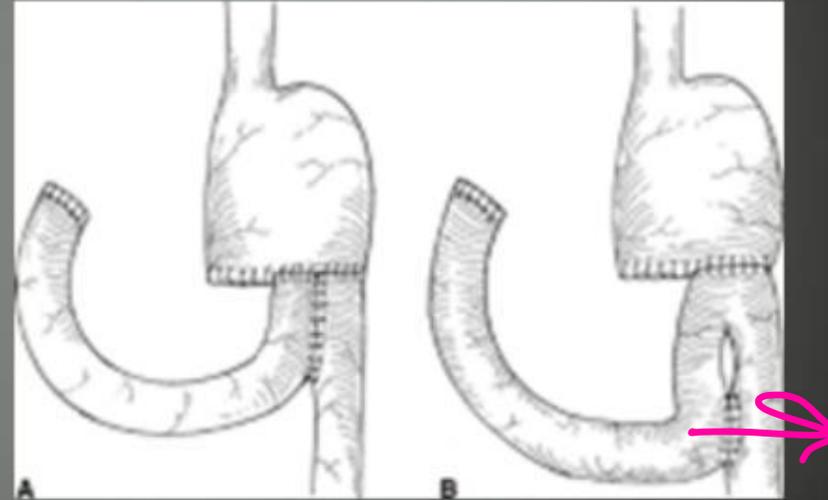


Braun Procedure

Distal enteroenterostomy



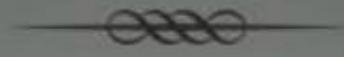
- Both decrease amount of refluxant but not enough protection from reflux
- Goal should be to divert all intestinal contents away from gastric remnant



Afferent and Efferent Limb Syndromes

حکایت

دانش



- ❧ Caused by partial or complete obstruction of jejunal limbs
- ❧ Characteristic signs and symptoms
- ❧ Afferent is more common than efferent

same as SBO

→ obstruction.

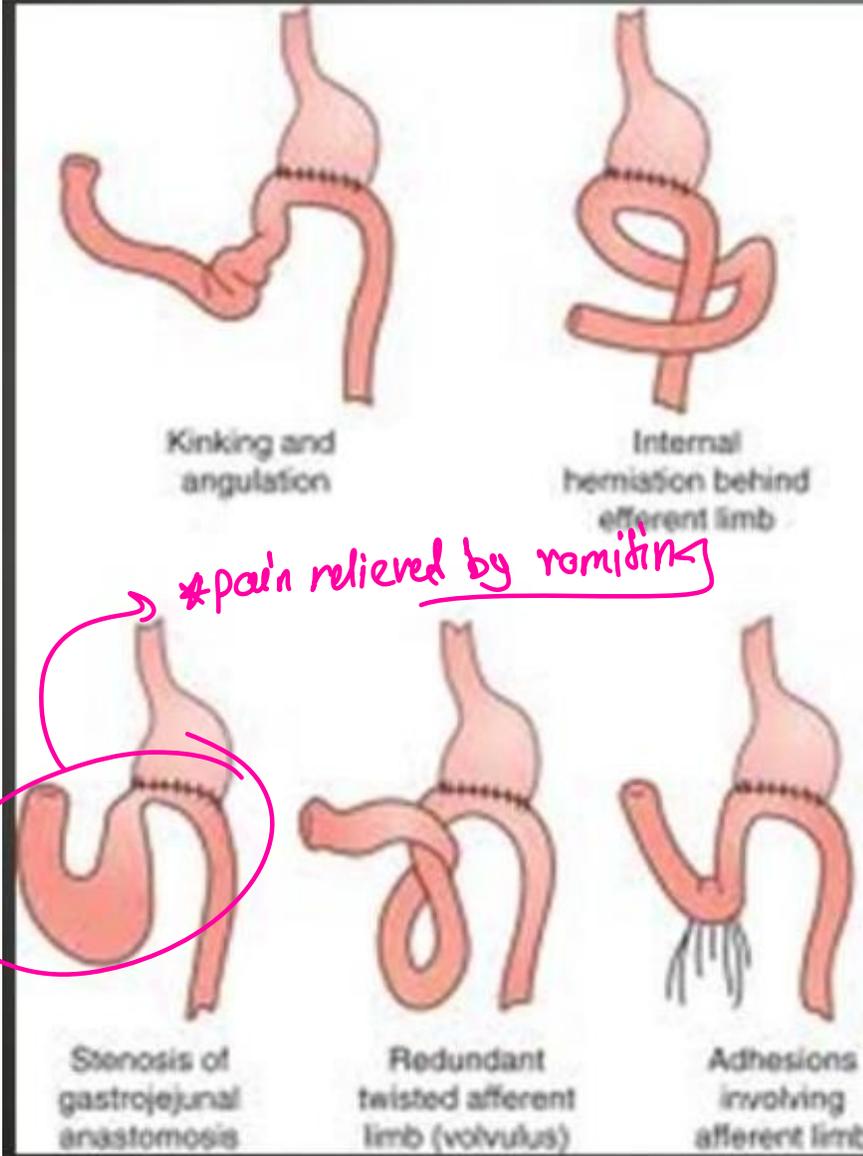
Afferent and Efferent Loop Obstruction

- Afferent loop syndrome
 - Afferent limb length > 30-40 cm can be obstruction
 - Chronic > acute
 - Severe postprandial epigastric pain (30-60 mins)
 - Projectile bilious vomiting → Bilious vomiting will relieve pain
 - Dramatic clinical relief after vomiting
 - Some can be presented with diarrhea

→ due to Adhesion or internal hernia

→ mesentery
* ممكن داخل العملية اذا نسيت الـ mesentery
فانج ← تفوت فيها bowel

Afferent Limb Syndrome



- Occurs only with BII
- Almost always due to too long of a limb
- Acute and chronic forms
- Acute = closed loop obstruction, usually immediately post-op, needs surgery

Diagnosis



- ❧ Symptoms of intermittent RUQ pain, relieved with vomiting
- ❧ Hyperamylasemia
 - ❧ ?pancreatitis
- ❧ EGD, gastrograffin study, CT - dilated loop
- ❧ HIDA - delayed films can show +/- obstruction

Chronic Afferent Limb Syndrome



- More common than acute
- Due to partial limb obstruction
- Increased incidence if anastomosis is retrocolic and above the mesocolonic defect
- Bacterial overgrowth – blind loop syndrome, vit B12 deficiency

ما تقدر التفاهيل
بسه اعرف المبدأ

Manifestations of Chronic Afferent Limb Syndrome and Alkaline Reflux Gastritis



Chronic Afferent Limb Syndrome

- ☞ Pain after meals
- ☞ Vomitus: Bile, no food, relieves pain, projectile
- ☞ Occult bleeding rare
- ☞ Cause: limb obstruction

Alkaline Reflux Gastritis

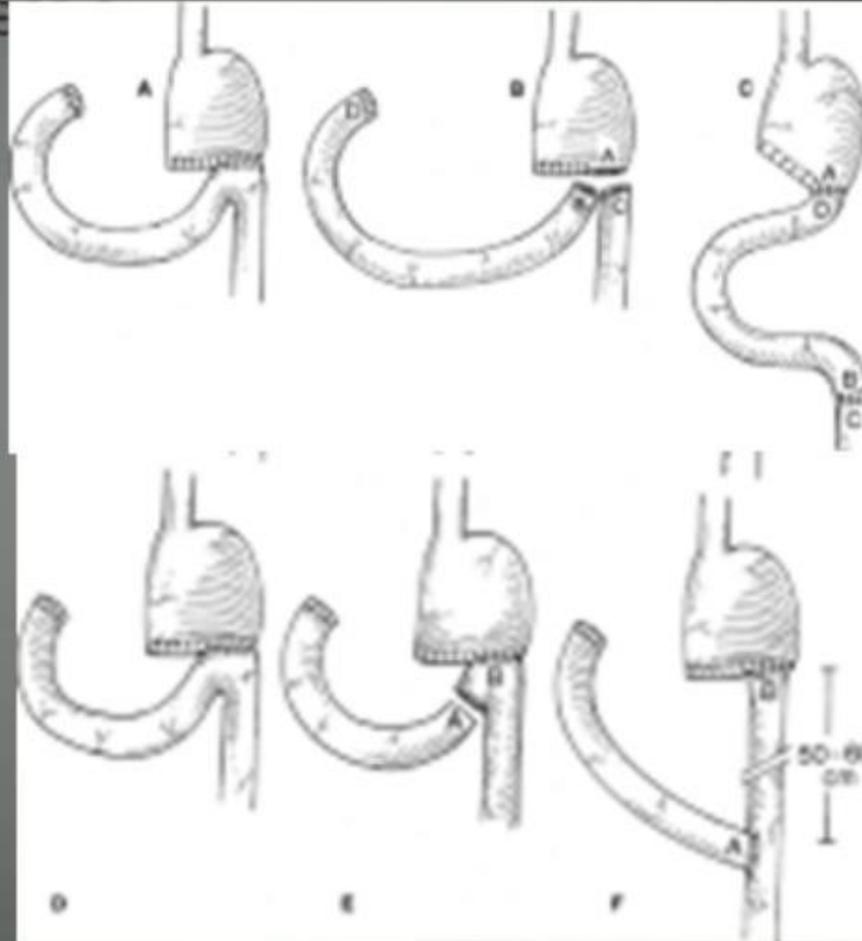
- ☞ Pain unrelated to meals
- ☞ Vomitus: Bile and food, no change in pain, nonprojectile
- ☞ Occult bleeding common
- ☞ Cause: enterogastric reflux

Treatment

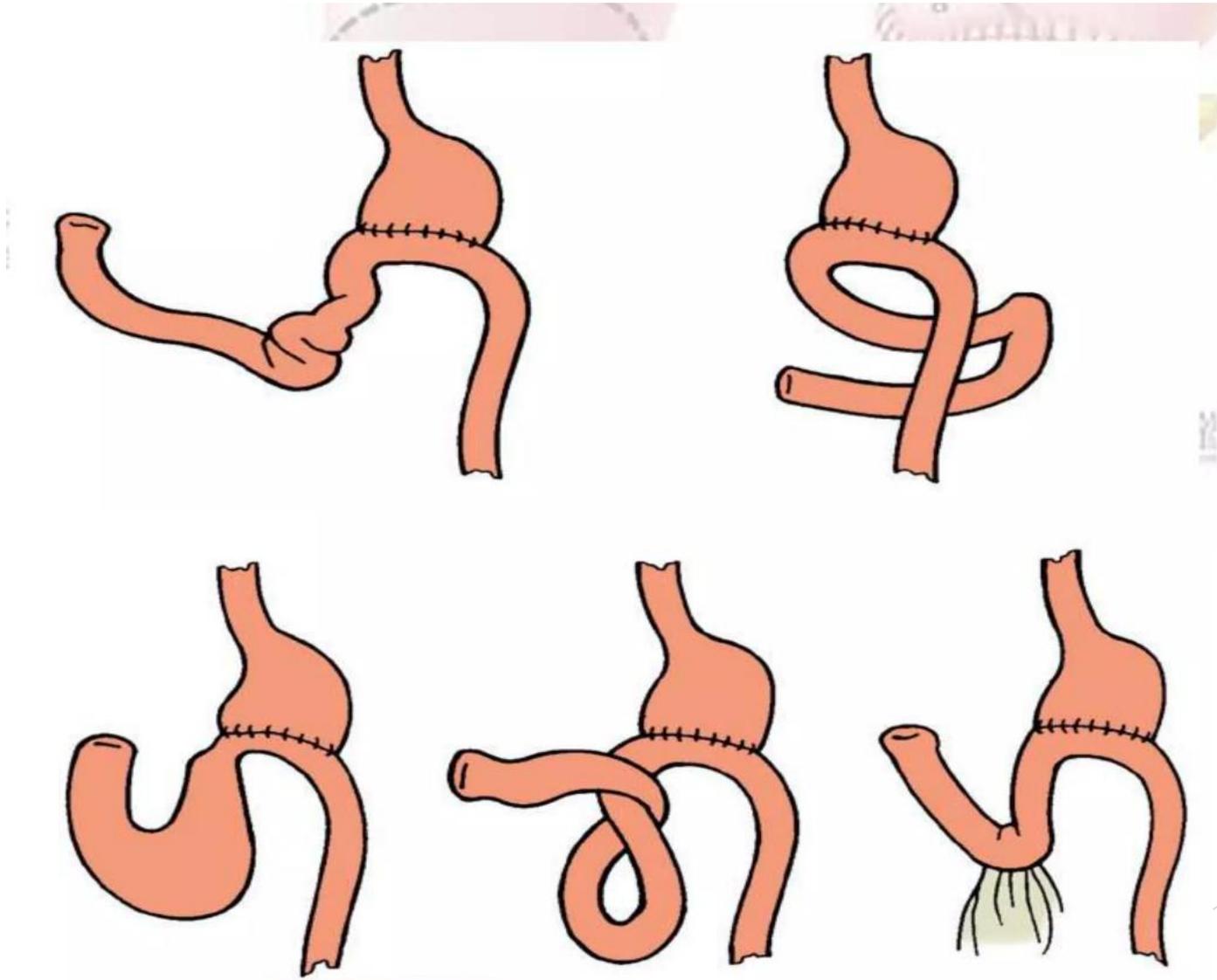
Surgical

Two most accepted

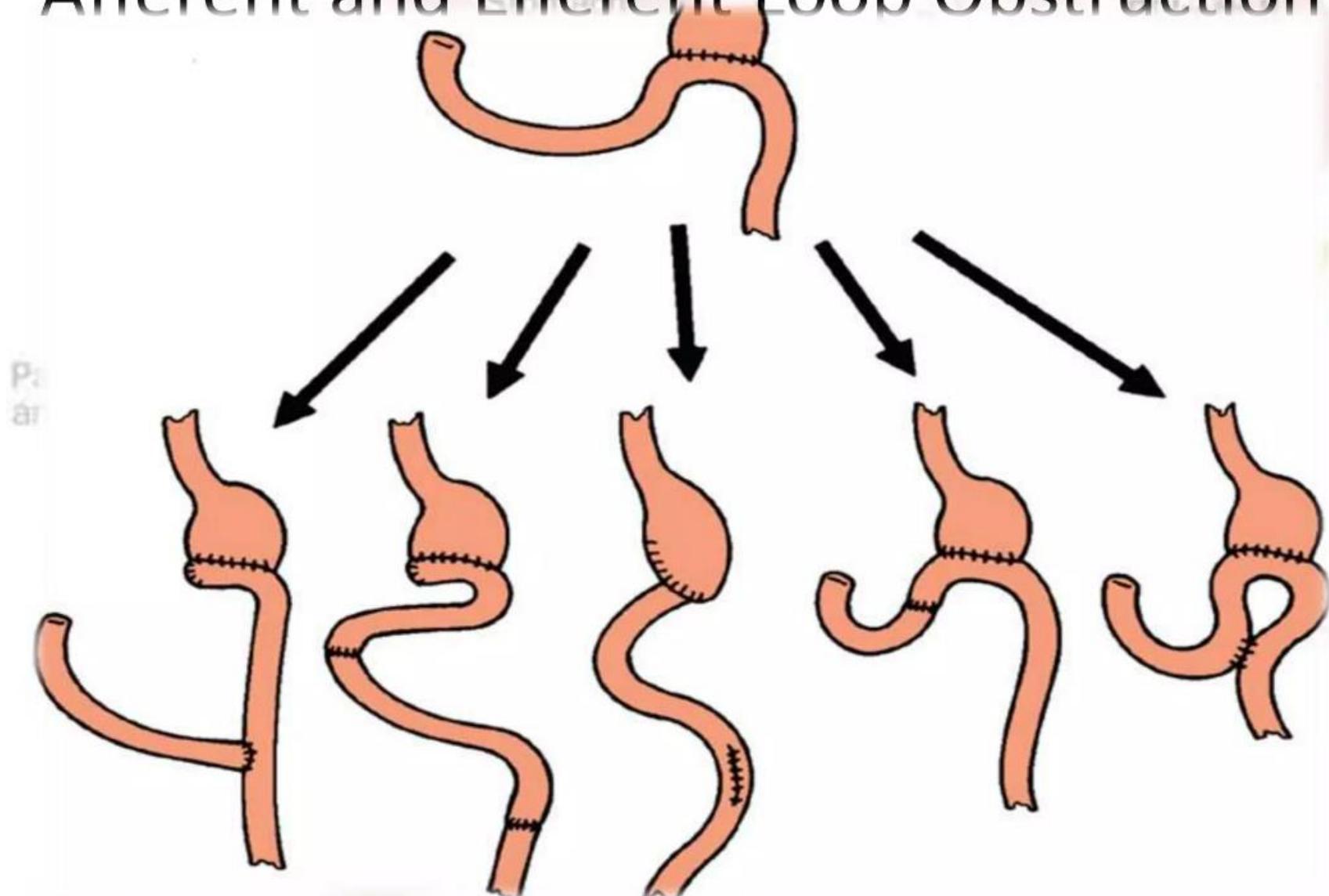
- ↳ Convert BII to BI
 - ↳ Can be difficult to get to duodenal stump
- ↳ Roux-en-Y – important to remember to perform complete vagotomy
 - ↳ Prevents marginal ulcers

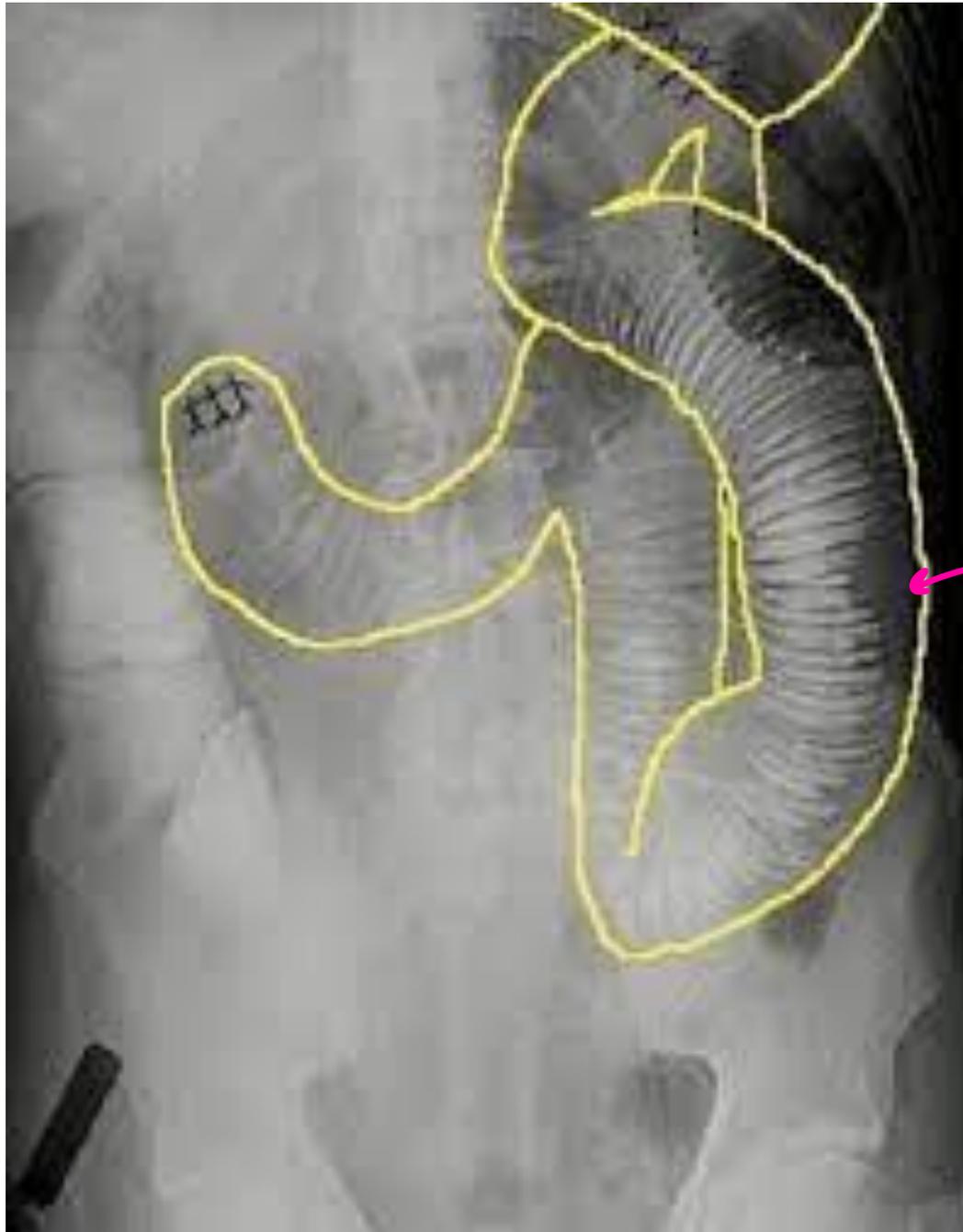


Afferent loop syndrome



Afferent and Efferent Loop Obstruction





Affera obstruction.

valvula conventis

Efferent Limb Syndrome

obstruction in distal limb.



- ❧ Less common and harder to diagnose
- ❧ Symptoms of crampy LUQ and epigastric pain associated with bilious vomiting
- ❧ Most commonly caused by internal herniation of the limb behind the anastomosis
- ❧ Diagnosed with barium UGI and EGD
- ❧ Treatment is surgical and is determined at the time of exploration

Roux Syndrome

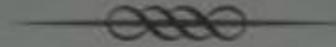
- Vomiting, epigastrium pain and weight loss after distal gastrectomy with Roux-en-Y reconstruction
- Scope : Dilate remnant stomach and Roux limb
- No mechanical obstruction from CT or upper GI series
- Rx : Promotility agents VS Sx

Roux Limb Syndrome



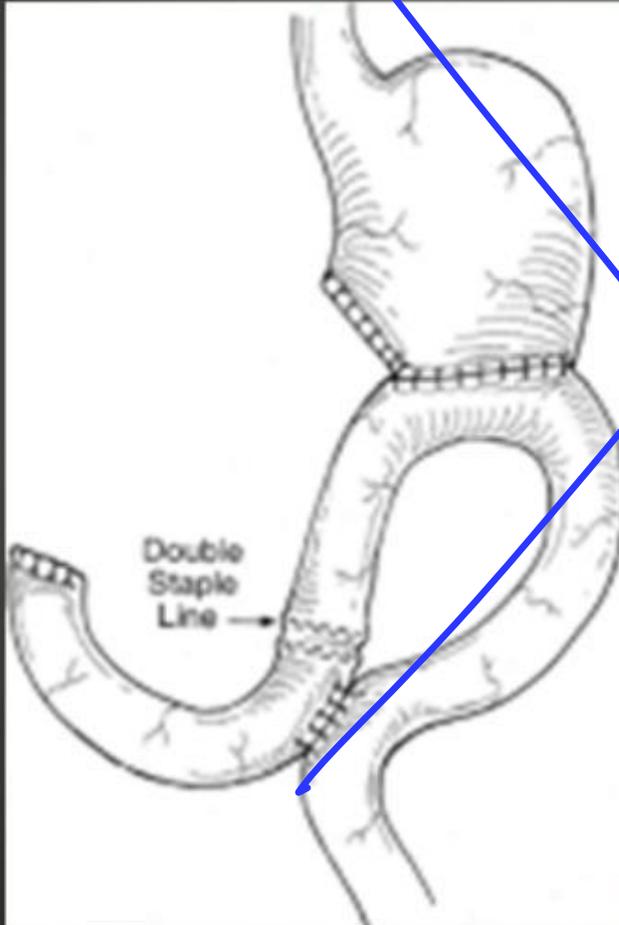
- ❧ 25-30% incidence of nausea, vomiting, postprandial epigastric fullness, and upper abdominal pain
- ❧ Hard to differentiate from gastroparesis
 - ❧ Usually a late complication
- ❧ True cause is unknown
 - ❧ Thought to occur due to disrupted jejunal myoelectric propagation
 - ❧ Loss of vagal stimulation that accompanies gastrectomy
 - ❧ No true correlation between length of Roux limb

Diagnosis



- ❧ Symptoms months to years after gastrectomy
- ❧ UGI and EGD usually normal
- ❧ Gastric emptying – significantly delayed
- ❧ Symptoms are not improved with prokinetic agents
 - ❧ Main way to differentiate from gastroparesis
- ❧ Don't improve over time

Treatment



• Surgical

• Further gastric resection with new Roux-en-Y

• To remove atonic stomach

• Conversion to BII with Braun 30cm distal

• “Uncut Roux”

• Avoids defunctionalized intestine

Early Dumping

Occurs in 20% of patients after gastrectomy or vagotomy and drainage.

Presentation is with postprandial GI discomfort, which may include N+V+D and cramps, with vasomotor symptoms eg. diaphoresis, palpitations, and flushing.

Mechanism involves a rapid emptying of hyperosmolar chyme (particularly carbohydrates) into small bowel. Leads to net fluid retention due to the osmotic gradient + releases one or more vasoactive hormones, eg. serotonin and VIP

Late dumping

Clin presentation is the same as early dumping but happens hours after eating.

Mechanism: Secondary to hypoglycemia from a postprandial insulin peak.

Treatment: Dietary changes (frequent small meals that are high in protein and fat and low in carbohydrates for early dumping; increased carbohydrate consumption for late dumping).

If intractable (rare)-(Conversion of a vagotomy and pyloroplasty to an antrectomy with Roux-en-Y reconstruction)

Postvagotomy diarrhoea

In 30% of patients after truncal vagotomy.

Mechanism: Rapid passage of unconjugated bile salts from the denervated biliary tree into the colon, where they stimulate secretion.

Treatment: Most cases are self limiting

Alkaline reflux gastritis

Occurs in 2% post-op

Clin Presentation involves persistent burning epigastric pain and chronic nausea that is aggravated by meals.

Diagnosis is usually diagnosis of exclusion - OGD may show gastritis/ technetium biliary scan can demonstrate increased reflux of bile into the stomach.

There are no effective treatments.

Miscellaneous —

Weight Loss and Dietary Restriction (mainly to reduce symptoms of eg early satiety)

Malabsorption - accelerated gastric emptying/ reduced gastric seiving (Particles >1mm go through)/ decreased pancreatic function

Bacterial overgrowth

Iron/(50%)/ B12(20%)/Folate(40%)

Metabolic bone disease

Summary

Early satiety —

It is caused by postsurgical atony, gastric stasis as a result of denervation, or from the "small gastric remnant syndrome" related to resection.

Clin Presentations involves epigastric fullness with meals, often followed by emesis.

Diagnosis and

Atony can be identified with a solid food emptying test and may respond to prokinetic agents such as metoclopramide and erythromycin. If these fail, completion gastrectomy may be required, although there is some anecdotal evidence that gastric pacing may prove useful.

The "small gastric remnant syndrome"- small and frequent meals

Afferent and efferent loop syndromes

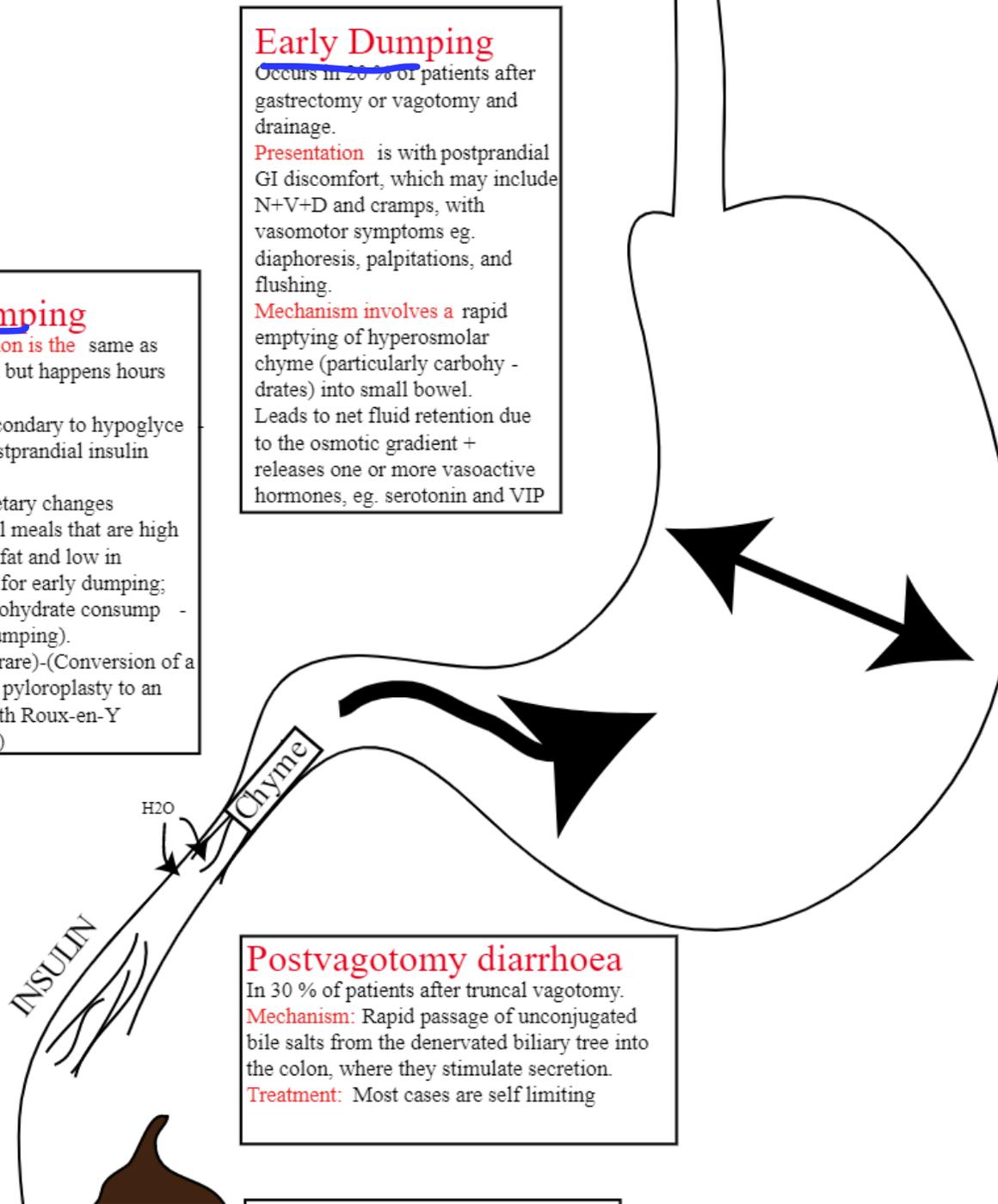
Post Billroth II reconstruction or gastroenterostomy.

It is due to mechanical obstruction of the limbs by kinking, anastomotic narrowing, or adhesions.

Clin Presentation involves postprandial epigastric pain and nonbilious vomiting relieved by projectile bilious vomiting. The detection of distended afferent loop on CT is diagnostic.

Treatments include conversion to a Roux-en-Y anastomosis.

Afferent and efferent loop syndromes —



TREATMENT SUMMARY

Dumping syndrome	<ul style="list-style-type: none">• Interposition of jejunal loop: Iso/anti-peristaltic.• Conversion of loop gastrojejunostomy to RouxenY reconstruction (57,58).
Post vagotomy diarrhoea	<ul style="list-style-type: none">• Anti-peristaltic jejunal loop distal interposition (59).
Gastric stasis	<ul style="list-style-type: none">• Gastric pacing/Electric pacemaker (60).• Venting gastrostomy.• Re-operative near total gastrectomy.
Bile reflux gastritis	<ul style="list-style-type: none">• Conversion to a Roux-en-Y reconstruction.
Afferent loop syndrome and Efferent loop syndrome	<ul style="list-style-type: none">• Endoscopic insertion of double pigtail stent (61).• Conversion to RouxenY.• Conversion to Billroth I.• Braun entero-enterostomy.
Jejuno-gastric intussusception	<ul style="list-style-type: none">• Revision of the GJ/Resection of the intussuscepted segment.• Revision of the GJ/Resection of the Conversion to a Rouxen Y.
Roux stasis syndrome	<ul style="list-style-type: none">• Resection followed by RouxenY revision.• Avoid Roux reconstruction in >50% resection of stomach (BII with Braun entero-enterostomy preferred) (62).

Jejunal Interposition

- ⌘ Isoperistaltic
 - ⌘ 10-20cm
 - ⌘ Regulate rate of gastric emptying and dilate to inc reservoir
- ⌘ Antiperistaltic
 - ⌘ No longer than 10cm
 - ⌘ Slow gastric emptying
 - ⌘ Gastric retention, reflux, obstructive symptoms



Gastric Cancers (GC)



Gastric Cancers (GC)

Common Primary

- Adenocarcinoma (95%), *most common Gastric cancer.*
- Lymphoma (4%),
- Gastrointestinal stromal tumors GIST (1%)

Rare Primary

- Carcinoid, ~~Angiosarcoma~~, ~~Carcinosarcoma~~, and ~~Squamous cell carcinoma~~

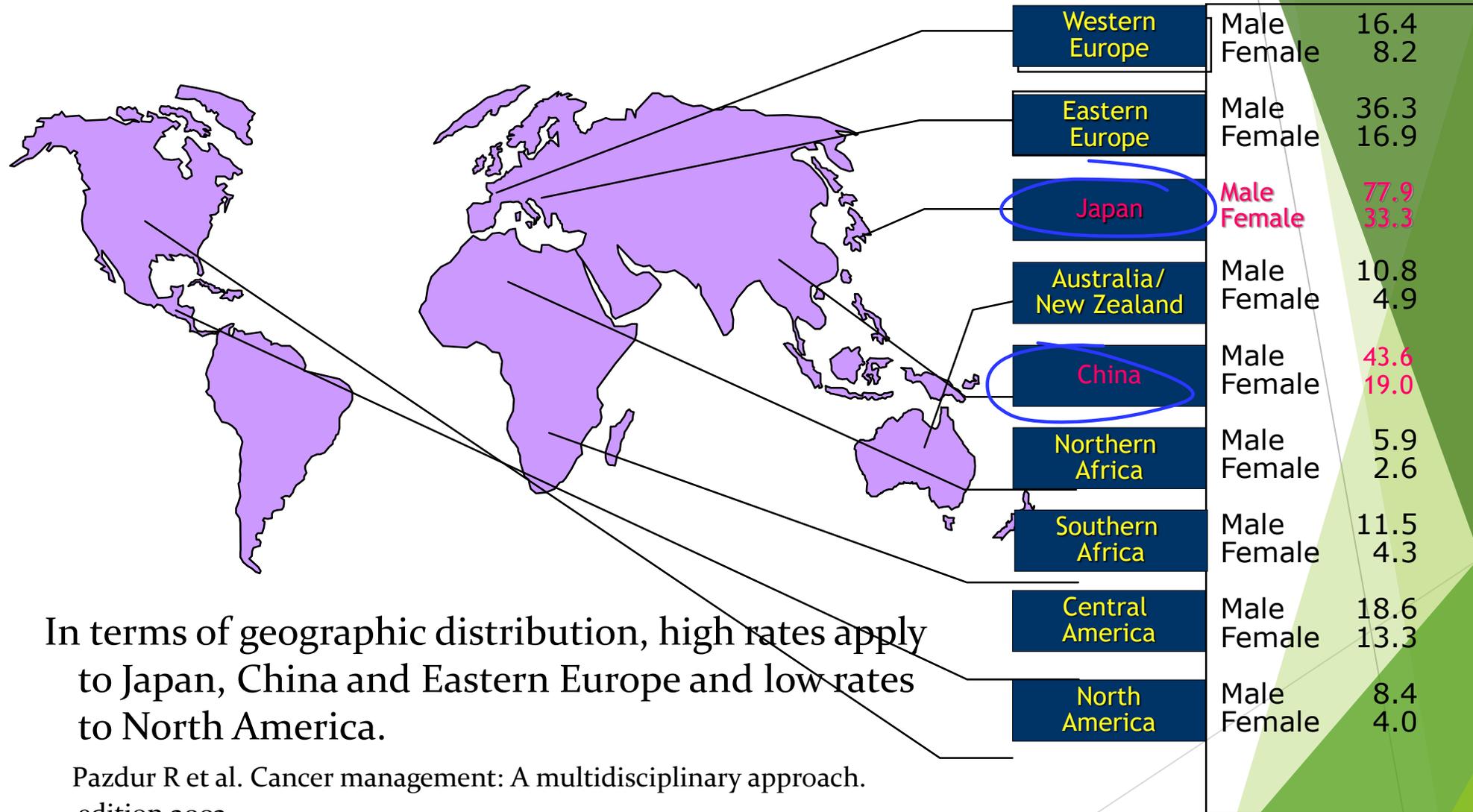
Secondary From :

- Melanoma , Breast(Blood born)
- Colon or Pancreas (Direct ext.)
- Ovary (By peritoneal seeding)

Gastric Adenocarcinoma:

- ▶ Worldwide : Fifth most common cancer and third most common cause of cancer related deaths. *most common cancer related death → lung*
- ▶ Incidence and mortality are declining in developed countries.
- ▶ Twice higher incidence and mortality in blacks/Asians.
- ▶ Male to female 2:1
- ▶ Geographical discrepancy (higher incidence in Japan/East Asia).
- ▶ Requires treatment in a high volume tertiary centers *كما يجب إتباعه في مستشفى طرفي.*
- ▶ Multidisciplinary approach for treatment offers better outcome (nutritional, endoscopic, surgical, medical, radiation oncologists)

GC Worldwide incidence



In terms of geographic distribution, high rates apply to Japan, China and Eastern Europe and low rates to North America.

Pazdur R et al. Cancer management: A multidisciplinary approach. edition,2002

Risk factors :

▶ H.pylori infection : *→ mostly lead to distal type.*

- Usually with prolonged infection >10 years *at least*
- Distal gastric ca, intestinal type in 90%

pathophysio: Atrophic gastritis → metaplasia → dysplasia → cancer

▶ **Diet :**

- salted, smoked, poorly preserved food
- Nitrate food (formation of N-Nitroso carcinogen)
- Lower risk with fruits, vegetables, vit C, vit A, antioxidants.

▶ **Occupations:** metal, mines, rubber, wood, asbestos workers

▶ **Obesity**

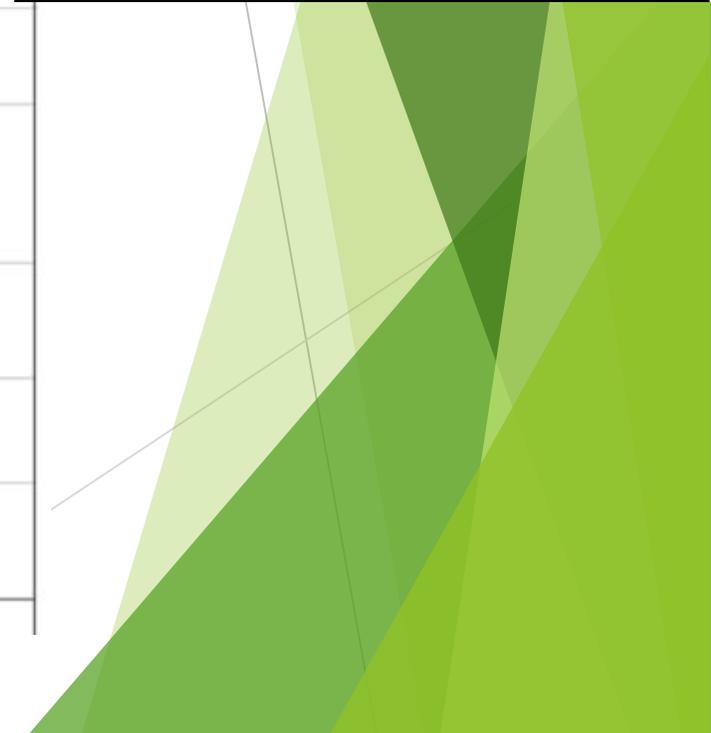
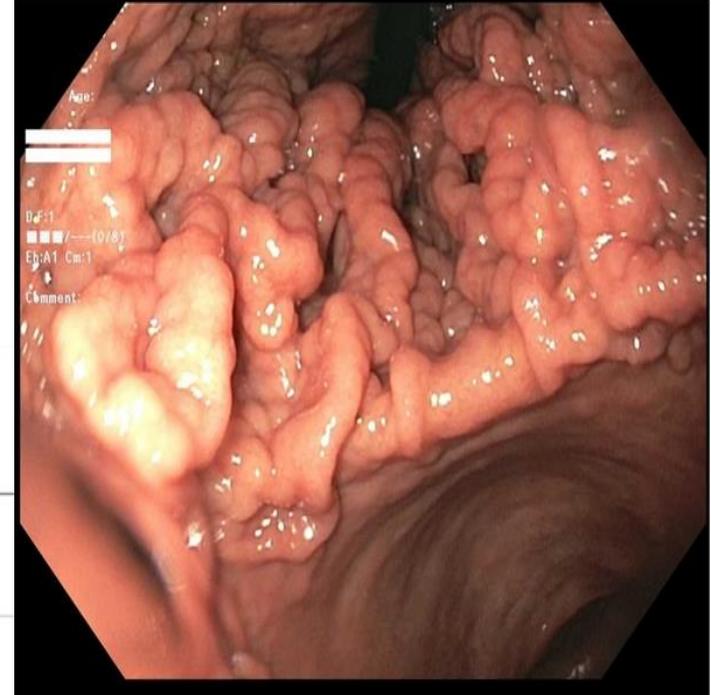
Risk factors :

- ▶ Smoking
- ▶ Alcohol is NOT a risk factor
- ▶ Hyperplastic polyps : less risk factor than in colon ca (villous, >1 cm)
- ▶ Chronic atrophic gastritis (*H.pylori*)
- ▶ Chronic non- healing gastric ulcers
- ▶ Pernicious anemia
- ▶ Blood group A

Precancerous changes

- ▶ precancerous diseases
 - ▶ chronic atrophic gastritis
 - ▶ gastric ulcer
 - ▶ gastric polyps
 - ▶ gastric remnant
 - ▶ Ménétrier's disease (*genetic + viral CMV causes.* hypoproteinemic hypertrophic gastropathy) (*rugosa of stomach.*)
- ▶ precancerous lesion
 - ▶ atypical hyperplasia

1900 Cases		
Precancerous lesion	Number of cases	%
Hyperplastic polyp	10	0.53
Adenoma	47	2.47
Chronic ulcer	13	0.68
Atrophic gastritis	1802	94.84
Verrucous gastritis	26	1.37
Stomach remnant	2	0.11
Aberrant pancreas	0	0
Total 1900		100



Risk factors :

► **Genetics** : *very minimum distribution.*

- 3% of gastric CA are inherited, most are sporadic.

- P53 (Li-fraumini) → thyroid & Breast & Gastric

- HNPCC (Lynch), MUTYH polyposis

- Familial adenomatous polyposis FAP./ APC mutation

- P-TEN (Cowden) hamartomas.

- BRCA-2 *male Breast cancer stomach & female Breast Cancer, stomach, ovarian Cancers.*

- Peutz-Jegher Syndrome. → *need prophylaxis total gastrectomy* → *may be normal on Endoscopy*

most common mutation in Gastric Cancer

Strong family Hx.

CDH-1 mutation : AD, 70% risk for hereditary diffuse gastric CA

female, young age.

- → **need prophylactic total gastrectomy** at age of 20 or 10 years before the age of the youngest diagnosed family member

- → endoscopic screening is unreliable (usually submucosal lesions)

Bcz these lesions develop submucosally

Pathology :

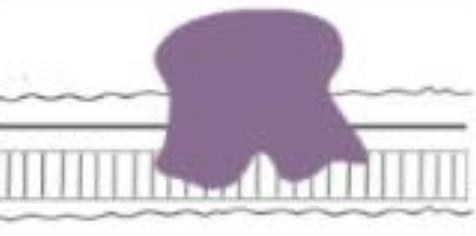
- ▶ Arises from the mucus producing cells, not the acid producing ones.
- ▶ Various classifications schemes for gastric CA.
- ▶ 1- Anatomical : (according to the site):
 - Proximal (cardia) → might be treated as pure gastric or considered distal esophageal (Siewert classification)
→ total stomach ectomy.
 - Distal (non cardia) - more with H.pylori infection.
 - Diffuse (9%) , linitis plastica / **leather-bottle stomach**

لاست اعرف الاسم

2- Bormann's Classification

منه مطلوب

- Classification based on gross appearance of the lesion

POLYPOID	FUNGATING	ULCERATED	FLAT
			
<p>Systemic symptoms Intestinal-type Low grade Expansive growth pattern Less signet-ring cells Larger LN metastases HERCEPTEST 3+</p>	<p>Older patients Systemic symptoms Intestinal-type Low grade Infiltrative growth pattern Tumor necrosis Less signet-ring cells</p>	<p>Smaller size Mainly intestinal-type High grade Infiltrative growth pattern More frequent signet-ring cells</p>	<p>Younger patients Less systemic symptoms Diffuse-type Signet-ring cells Infiltrative growth pattern High grade No tumor necrosis No MSI</p>
		<p><i>Female patients</i></p>	<p><i>Female patients Perineural infiltration</i></p>

important.

- ▶ 3- Histopathological (Lauren's classification) → the most adapted classification
- Intestinal type :
 - more in elderly, males. → good prognosis.
 - usually well to moderately differentiated.
 - associated with metaplasia / chronic / atrophic gastritis. (H. Pylori)
 - More distally localized than proximal in the stomach
 - more tendency to spread through lymphatics and hematogenously.
 - liver is the M.C site of mets.
 - Better prognosis → usually Bad prognosis.
- Diffuse type :
 - more in younger, females → But also can occur in old age.
 - usually poorly differentiated + signet ring cells → indicate worse prognosis.
 - without gastritis
 - More proximal
 - spread transmurally with local invasion, lymphatics and peritoneal mets.
 - Usually worse prognoses

Spread Patterns

- ▶ Direct invasion / transmural
- ▶ Lymph node dissemination
- ▶ hematogenous spread
- ▶ Transperitoneal/transcoelemic spread

Clinical Presentation of gastric adenocarcinoma :

*usually late presentation

▶ Asymptomatic, discovered incidentally

▶ Early gastric CA :

- No obvious signs and symptoms.

→ so patient
stage 4

بیماری

علی →

لانه اول ایش بیون
no signs

- Vague epigastric abdominal pain for months, Mistreated as dyspepsia or PUD.

▶ Progress to more obvious epigastric pain, anorexia, weight loss and vomiting +/- hematemesis (most common Sx's)

*most common Presentation

- chronic vague abdominal pain
- weight loss

▶ Dysphagia in proximal tumors.

▶ Symptoms of G.O.O in distal tumors (indicate locally advanced disease)

▶ Palpable epigastric mass in 30%.

▶ Chronic anemia

▶ Sx's & Sx's of large bowel obstruction. (invasion to transverse colon)

important.

- ▶ Signs and symptoms of metastatic disease : (40% have stage IV disease on presentation)
 - palpable left supraclavicular (Virchows) LN.
 - palpable periumbilical (Sister Mary Joseph's) nodule.
 - Blumer's shelf on DRE. (palpable peritoneal deposit)
 - ascites.
 - Jaundice.
 - Palpable ovarian mass (Krukenberg tumor) - via peritoneal seeding.
- ▶ Elective vs. Emergency presentation (G.O.O , perforating malignant gastric ulcer, upper GI bleeding)

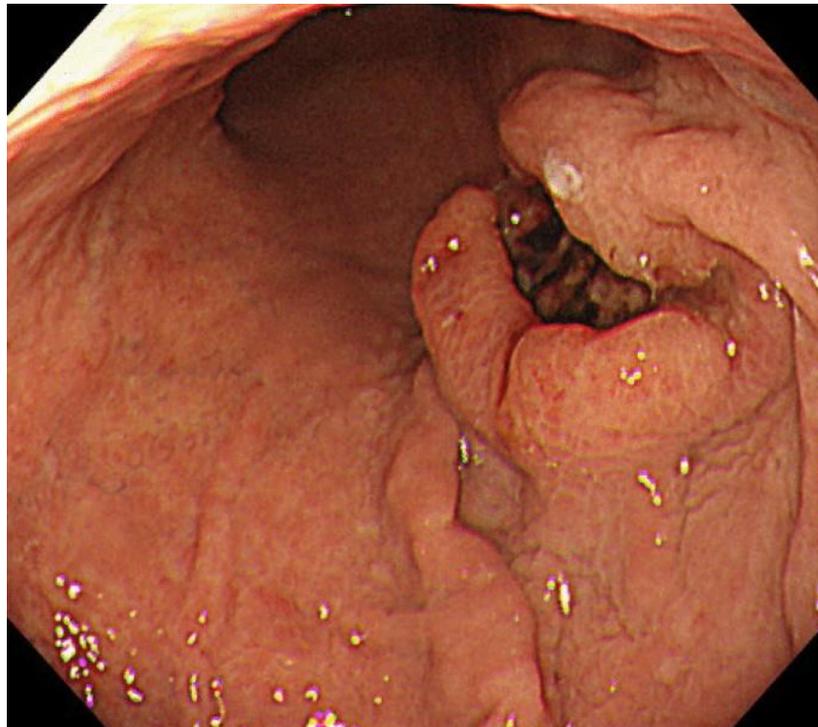
Diagnosis / Assessment / Staging

- ▶ Full Hx and PEx (age, chronicity of pain and non response to usual antacids)
- ▶ CBC (anemia) , KFT , LFT (usually normal but can be elevated in liver mets)
But mostly normal.
- ▶ *Carcino embryonic Antigen.*
CEA : → *used to detect response to Tx.*
 - High in only 30%.
 - Not useful for diagnosis / screening
 - **Useful in monitoring response to treatment**, surveillance for recurrence.
- ▶ Assessment of nutritional status (weight, BMI, serum albumin, pre-albumin, zinc, Mg⁺⁺)
- ▶ AXR : (distended stomach in GOO, perforation, obstruction)

► Upper Endoscopy : Gold standard,

- The Gold standard for diagnosis
- Allows direct anatomical localization for planning surgery.
- Confirms Dx and histopathological features with biopsy.
- Possible stenting for obstructed inoperable disease.
- 95 % accuracy

ما بتقدر تعلق
تخيل ما يكون عندك .
Gastric CA
Biopsy.

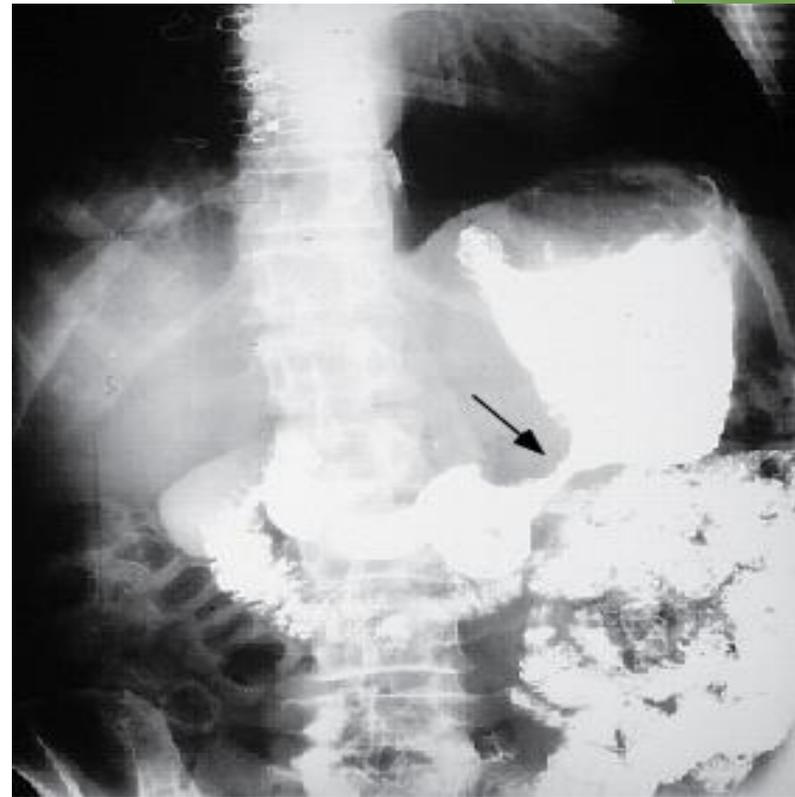
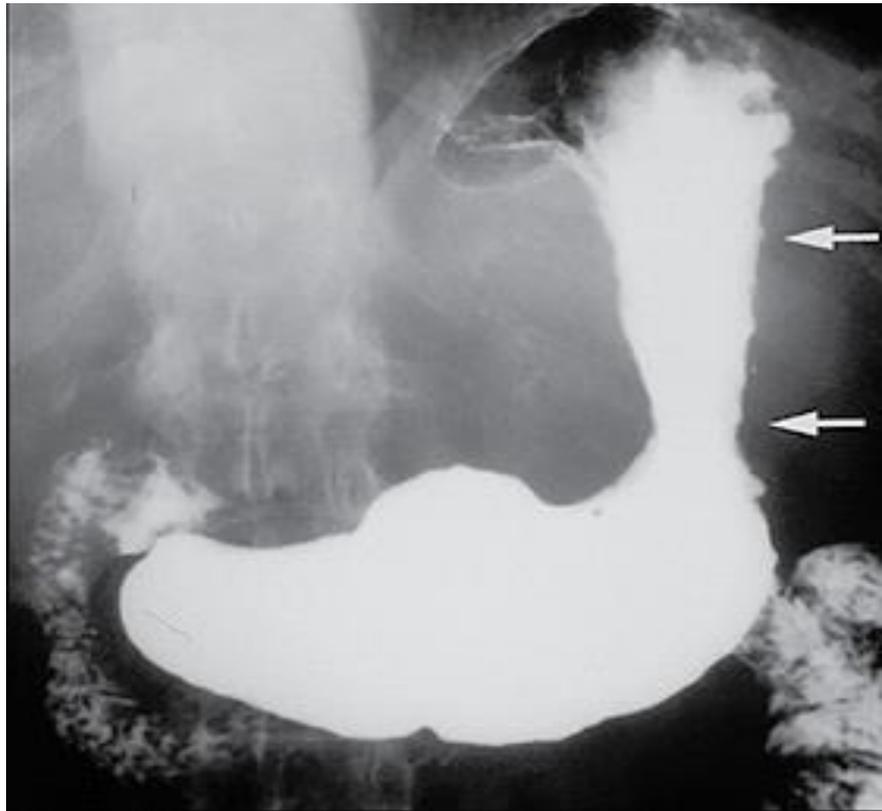


not commonly used.

Barium swallow

75% accuracy

for obstructive lesions only



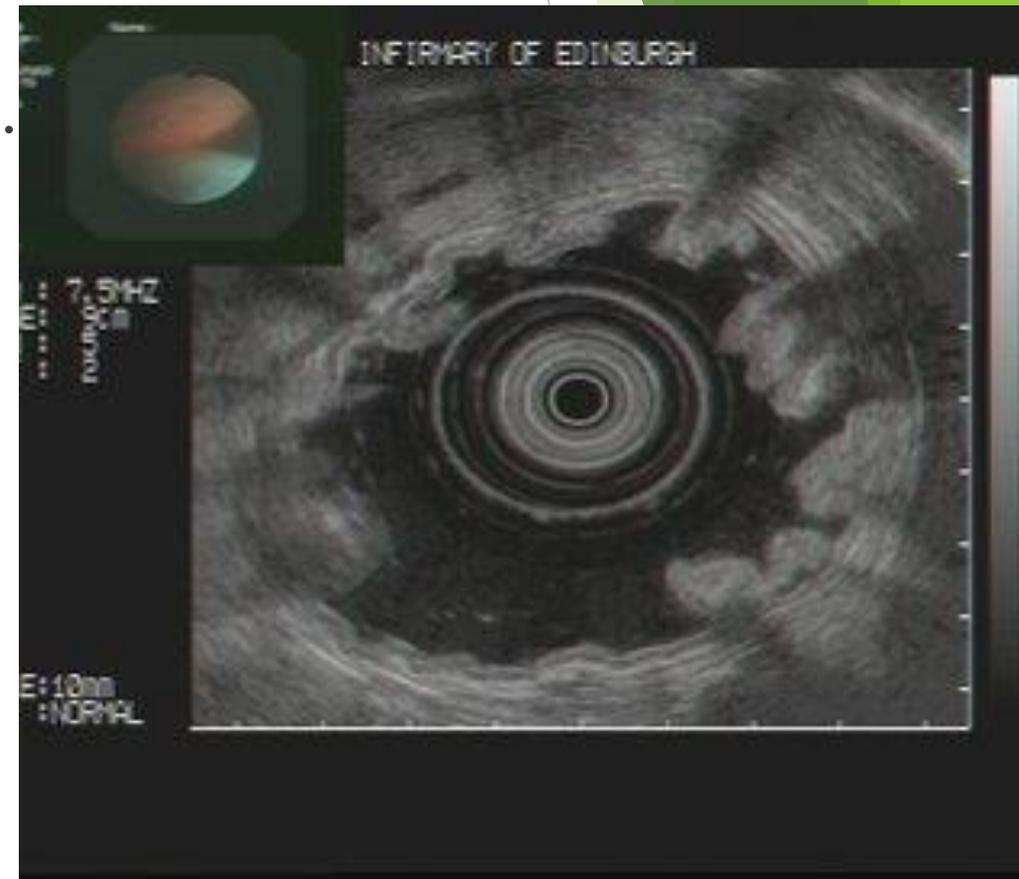
Apple core sign

Linitis plastica "leather-flask" appearing stomach

► Endoscopic Ultrasound (EUS) :

- Utilizes both endoscopy + direct Ultrasound imaging.
- The most specific and sensitive for determining the 'T' stage of the tumor
- Very accurate in evaluating the local LN status 'N' stage.
- Allows sampling of local LN
- Indicated for locally advanced gastric CA.

↳ detect invasion of Tumor in wall layers.



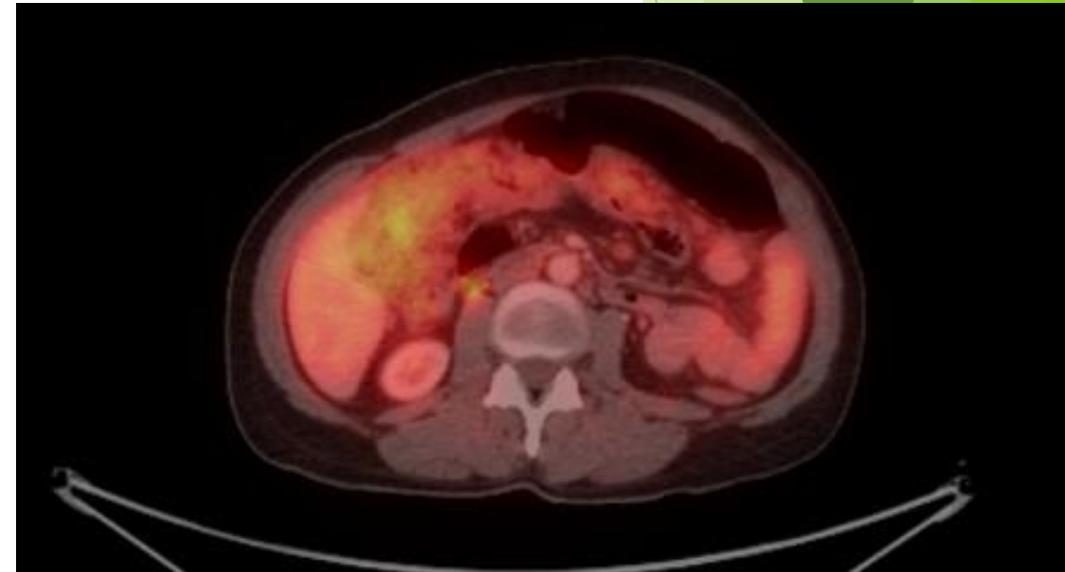
for staging.

▶ **Contrasted CT (chest/abd/pelvis) :**

- Usually for staging purposes
 - Solid organ mets, non regional LN mets
 - Ascites
 - Large peritoneal deposits
- ▶ Limitations : early gastric ca, small <5mm peritoneal and liver mets

▶ **PET/CT :** ** nodules on peritoneum less than 3mm will not be detected on PET.*

- ▶ CT combined with FDG radiolabeled glucose
- ▶ More uptake by metabolically active cells (including malignant cells)
- ▶ Improve detection of occult mets than regular CT , but still limited.



Standard for staging

▶ **Staging Laparoscopy** : *most to detect peritoneal Mets.*

- ▶ Now considered a Standard of care in locally advanced gastric CA
- ▶ Before initiation of treatment.
- ▶ Performed in O.R by inserting 1,2 or 3 trocars
- ▶ Complete inspection of the abdominal cavity for small mets (peritoneal surfaces, mesentery, under surface of diaphragm and liver surface)

▶ Advantages :

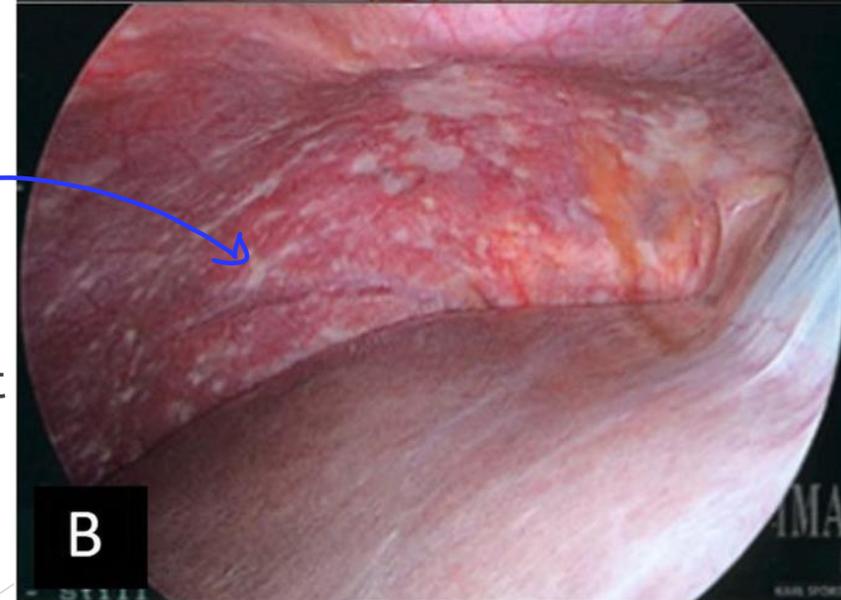
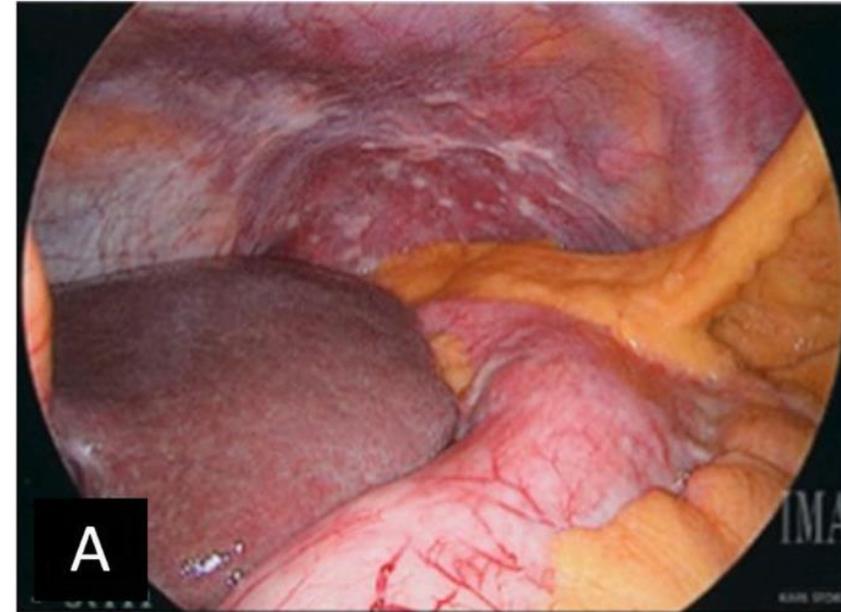
- Identify small mets that are missed by CT (<5mm).
- Obtain tissue biopsy of suspicious nodules
- Perform peritoneal lavage and fluid cytology
- Local disease status (involvement of adjacent organs)

to detect Early Mets in peritoneum.

if fluid +ve → stage 4 → no surgery

▶ Among patients deemed to have non metastatic disease initially, 36% of patients were actually stage IV disease which alter the treatment (occult peritoneal mets 21%, +ve wash cytology 13%, liver mets 6%) - in a study performed at *MD Anderson cancer center -Texas, USA.*

▶ Might need to be repeated after NACTX, before definitive surgery.



Staging of Gastric Adenocarcinoma (TNM)

▶ Necessary for 1-determining extent of disease, 2-planning of tx, 3- reflecting prognosis.

▶ (T) :

- Tis : carcinoma in situ, high grade dysplasia/ confined to mucosa

T1 : tumor invading submucosa/ lamina propria.

T2 : tumor invading muscularis propria.

T3 : tumor invading subserosa without visceral peritoneum.

T4 : tumor invading serosa (visceral peritoneum) or adjacent organs.

▶ (N) : → definitive after surgery and Pathology lab.

- N0 : no evidence of local LN involvement, N1 : 1-2 LN, N2: 3-6 LNs, N3 : 7 or more LNs.

- +ve LN makes it stage III

▶ (M): → M0 : no distant mets, M1: distant mets (stage IV)

▶ Mets to non-regional LNs (mesenteric, para-aortic, portal, retroperitoneal) is M1 (stage IV)

▶ +ve abd wash cytology is M1 (stage IV)

▶ Staging might change after surgery

Treatment :

- ▶ Various factors contribute to treatment selection:
 - Clinical stage of the disease
 - Patient related factors (Age, co-morbidities)

- ▶ Possible treatment options :
 - Chemotherapy (Neoadjuvant, Adjuvant, palliative)
 - Radiotherapy (only for palliation) → in Palliative Tx
 - Endoscopic resection (very early disease, selected patients)
 - Surgical resection (mainstay of treatment, the only curative one)
 - ↳ surgery indicated in patients without mets.

Endoscopic Mucosal Resection (EMR)

↳ usually limited only to Early cancer.

- ▶ Only for early disease confined to mucosa (Tis, T1a)N0M0
- ▶ Low probability of LN mets (0-4%)
- ▶ Absolute indications : well differentiated, <2cm, no ulceration, T1a
- ▶ Offers survival similar to surgical resection if performed for absolute indications.
- ▶ 5 yr- survival 95%
- ▶ Slightly higher incidence of local recurrence (6% vs 1% for surgical resection)

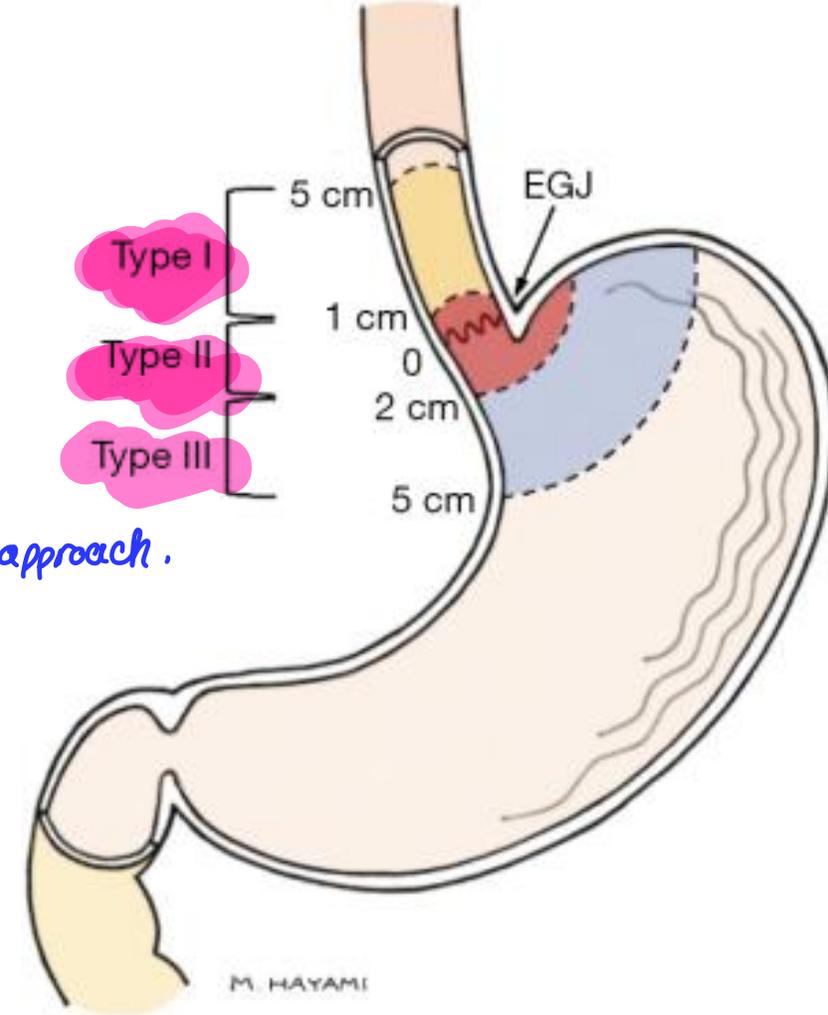
- ▶ Advantages : less invasive, less morbidity , optimal for old frail patients with co-morbidities.

- ▶ Limitations :
 - performed only in specialized high volume centers with good experience (Japan)
 - Need closer surveillance / followup

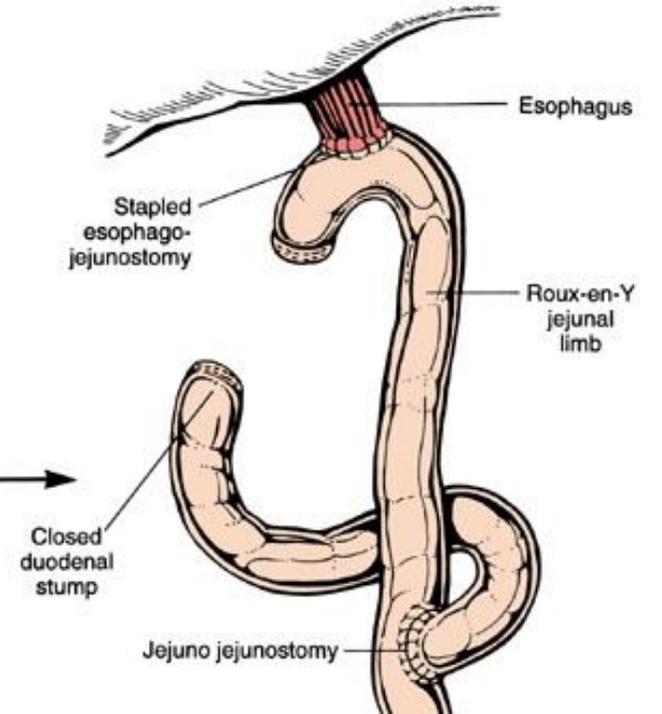
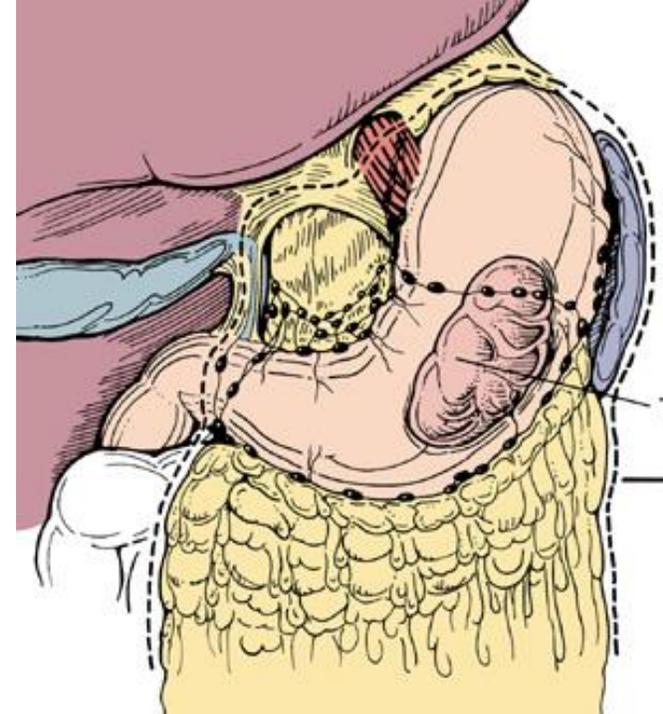
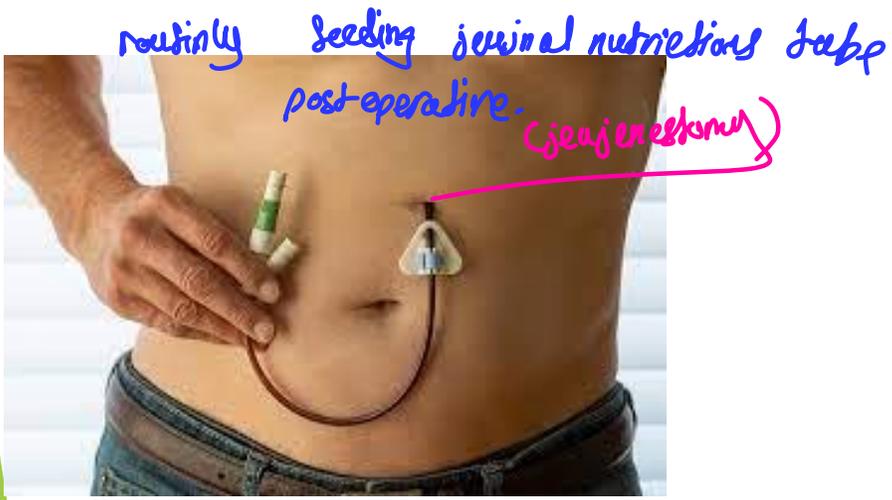
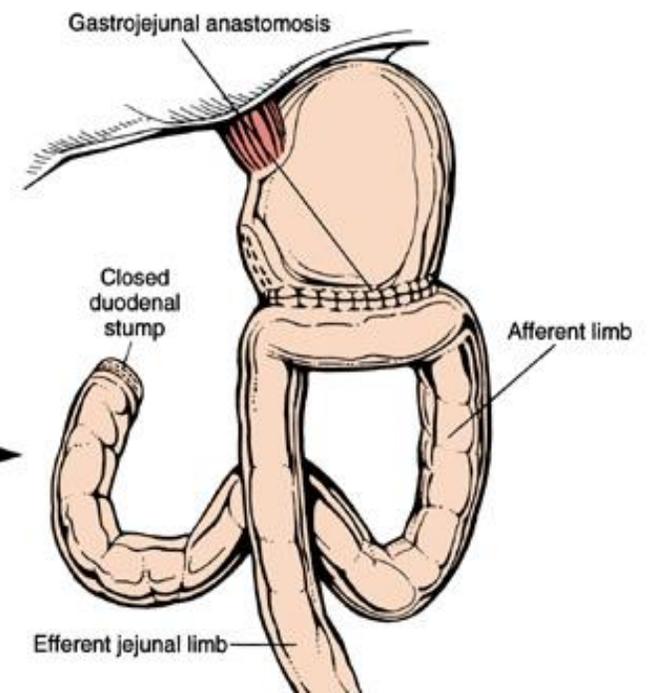
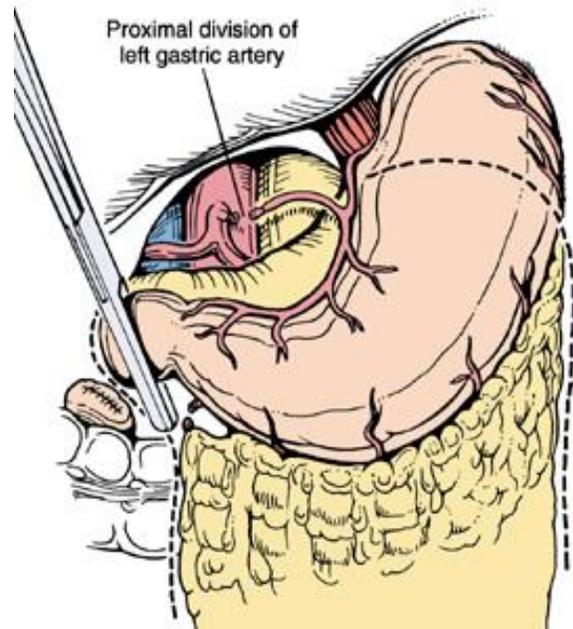
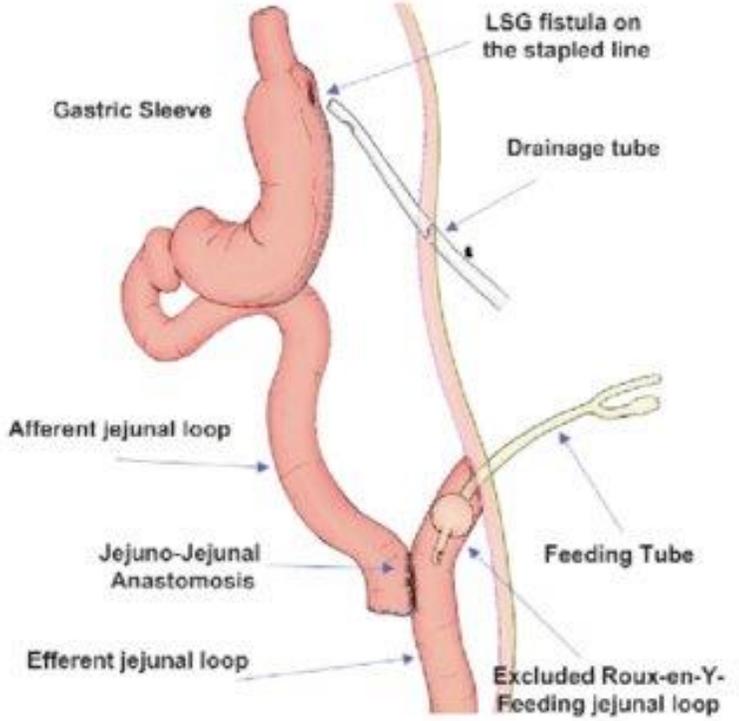
Surgical Treatment :

- ▶ The mainstay of treatment and the only curative modality (along with EMR).
- ▶ **GOAL** is to remove the tumor with at least 5cm tumor free margins and performing routine Lymphadenectomy.
- ▶ Extent of gastrectomy depends on the anatomical location of the tumor and might include **total/ subtotal or distal gastrectomy**
- ▶ for **distal** (body and antrum) tumors , distal or subtotal gastrectomy leaving a remnant proximal gastric pouch can be feasible as long as the 5 cm -ve margin is obtainable.
- ▶ Otherwise , total gastrectomy.
- ▶ All have similar outcome and survival.
- ▶ Partial gastrectomy has a better quality of life and post op nutritional status.

- ▶ Proximal tumors : (cardia and GEJ)
 - If <2cm below GEJ → treated as esophageal not gastric
 - Siewert classification : type1 (1-5cm above GEJ)
Type2: (2cm below) are esophageal → needs different Tx approach.
 - Type 3 (2-5 cm into the stomach are gastric CA)
- ▶ All proximal pure gastric CA should undergo total gastrectomy (not partial)



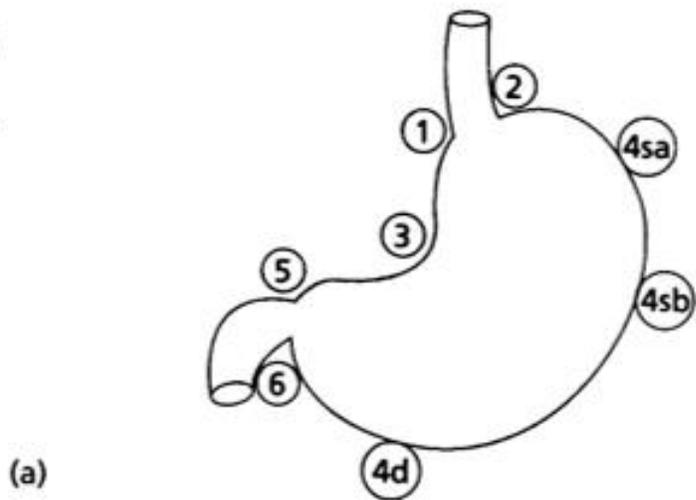
- ▶ Total gastrectomy : (for proximal and midbody tumors)
 - Removal of the whole stomach and reconstruction with **ROUX en-Y** configuration
 -
- ▶ Partial (distal/subtotal) gastrectomy: (for antral/distal tumors)
 - Removing part of the stomach with remaining proximal gastric pouch (supplied by short gastric vessels).
 - reconstruction can be done with **Billroth II** OR with **ROUX en-Y** configuration.
- ▶ **open vs. laparoscopic**
- ▶ Both offer same survival/outcome/completeness of resection.
- ▶ Lap. Surgery has lower post op pain, faster recovery and earlier return to daily activities. But require a more skilled surgeon.
- ▶ Feeding jejunostomy insertion at surgery for all patients.



- ▶ **Lymphadenectomy (LN dissection) :**
- ▶ At least 16 LN , for accurate staging and cure.
- ▶ The standard is to remove level **N1** (perigastric LNs) and **N2** (celiac, C Hepatic, left gastric, splenic LNs) → **D2 lymphadenectomy.** *→ standard lymphadenectomy to Gastric Cancer.*
- ▶ D zero is incompletel lymphadenectomy,
- ▶ D1 is removal of N1 perigastric LN (all nodal tissue within 3 cm of the primary tumor (might be sufficient in early CA)
- ▶ **D2** is the standard in gastrectomy for cancer
- ▶ D3 (D2++ paraortic,retropancreatic) → higher morbidity, no added survival benefits
- ▶ D3 only performed in Japan

- ▶ Japanese literature shows survival benefit of extended D2 dissection:
 - ▶ 39% 5 year survival after D2 vs. 18% for D1

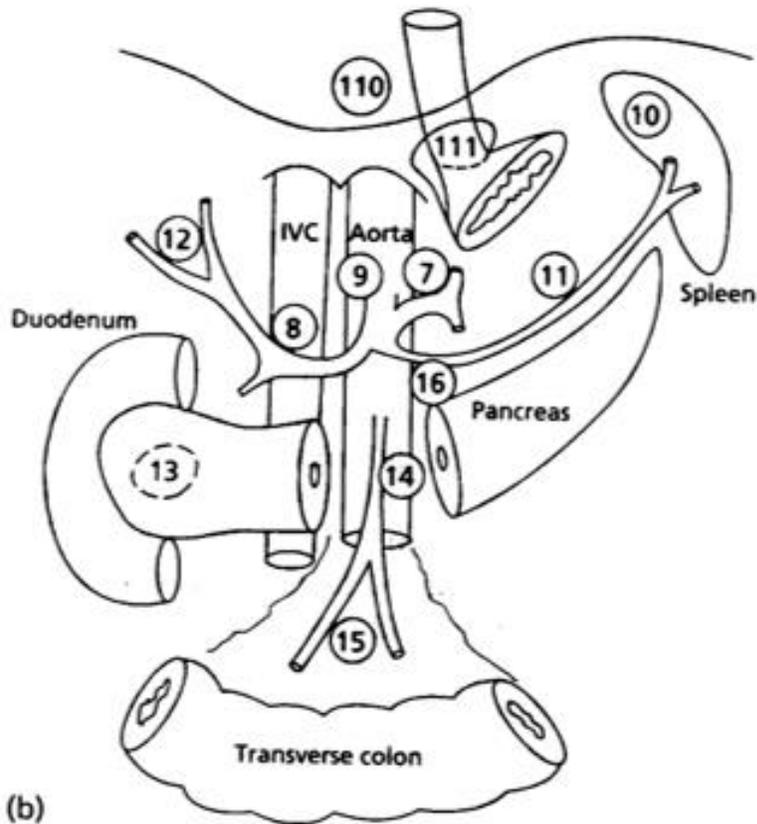
- ▶ Western literature shows no survival benefit but increased morbidity and mortality of D3 dissection



Key

- 1 = right cardiac
- 2 = left cardiac
- 3 = lesser curvature
- 4 = greater curvature :
 - sa = short gastric
 - sb = left gastroepiploic
 - d = right gastroepiploic
- 5 = suprapyloric
- 6 = subpyloric

(a)



Key:

- 7 = left gastric artery
- 8 = common hepatic artery
- 9 = coeliac axis
- 10 = splenic hilum
- 11 = splenic artery
- 12 = hepatoduodenal artery
- 13 = retropancreatic
- 14 = root of mesentery
- 15 = middle colic artery
- 16 = para-aortic
- 110 = paraoesophageal
- 111 = diaphragm

(b)

► **Splenectomy :**

- Only if spleen or splenic hilum are involved , no rule for prophylactic routine splenectomy

► **Multivisceral organ resection : (e.g. pancreas, transverse colon)**

- For T4 tumor involving adjacent organ/s
- Only if you can get -ve margins, patient can tolerate it, and no mets

Post op. complications

► General complications : 20-40%

- Pulmonary (atelectasis, pneumonia).
- Bleeding (intra op or post op)
- Infection (SSI)
- Cardiac (MI, arrhythmias)
- Venous thromboembolism (DVT, PE)

► Specific to gastrectomy:

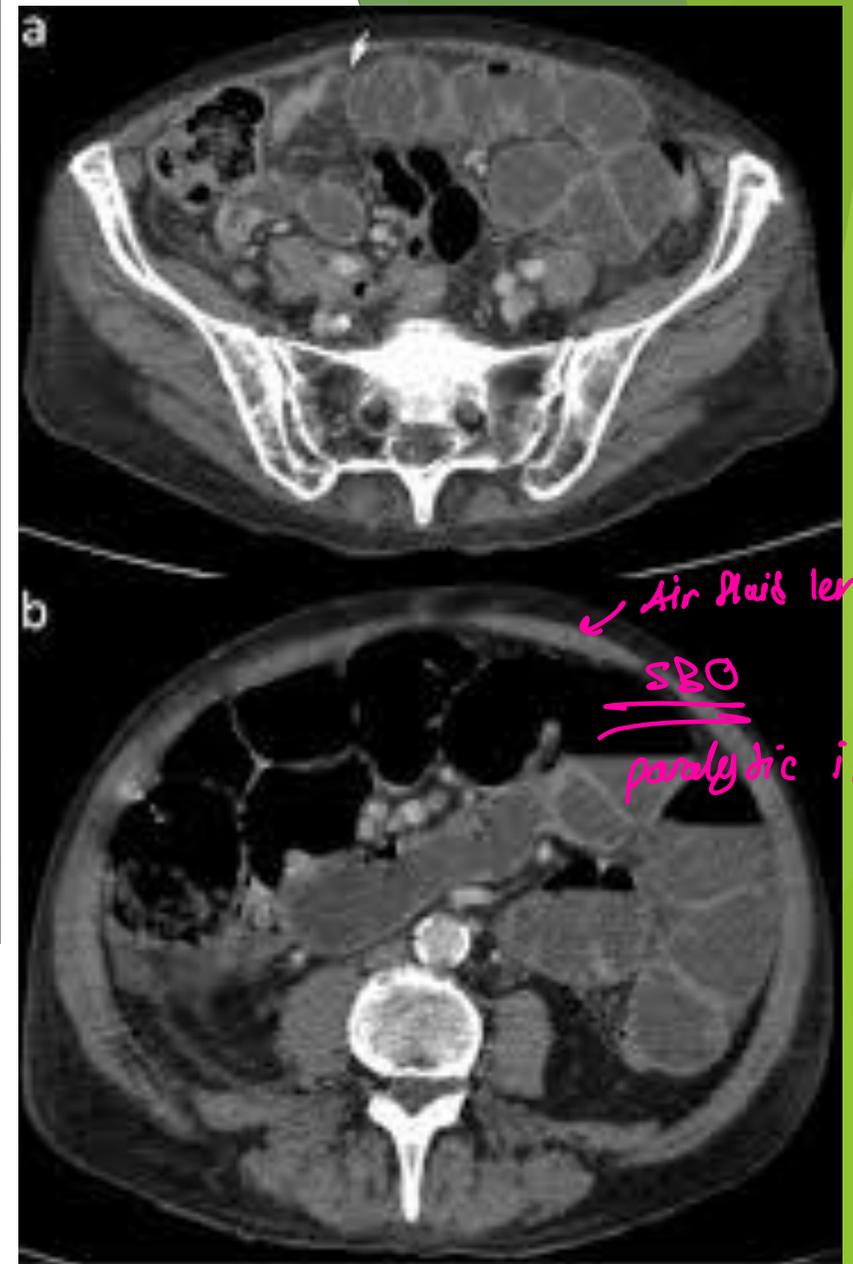
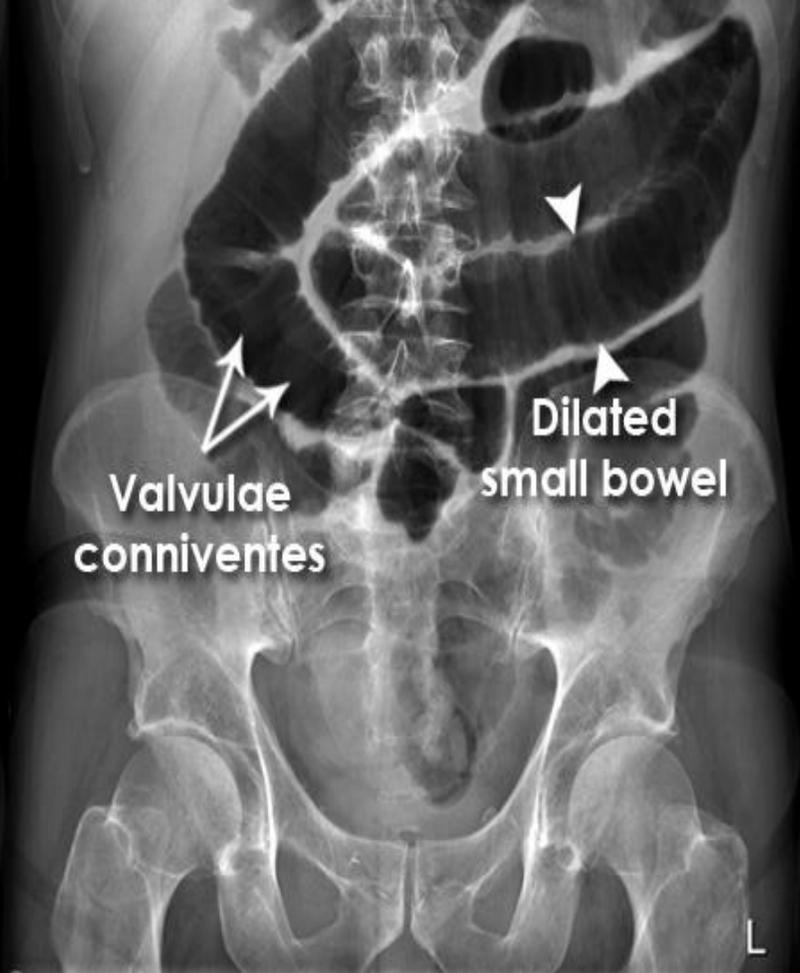
- Anastomotic site leak
- Post op ileus
- Dumping Syndrome
- Nutritional impairment (B12, calcium, iron deficiency, Food restriction)
- Diarrhea
- Gallstones
- Stomal ulceration, anastomotic site stricture

Anastomotic site leak

- ▶ 2-10%
- ▶ Can be early or late (up to 40 days post op)
- ▶ Leak in the first few days is usually a technical failure
- ▶ Dx : fever, leukocytosis, tachycardia, signs of peritonitis or food/juice in drains if free leak
 ↳ by Barium swallow 5 days post operation
 if no leak → start fluid intake.
- ▶ CT with oral contrast or GG swallow detects leak
- ▶ Some surgeons perform routine contrast imaging before initiation of oral feeding (post op day 5)
 * any unexplained Tachycardia post-op may indicate leak.
- ▶ Management :
 - non-surgical (antibiotics, nutritional support, endoscopic covered stents or percutaneous CT guided drainage (if contained leak)
 - Surgical : re-laparotomy and revision of anastomoses .

post op ileus :

- ▶ More with open surgery
- ▶ Due to extensive bowel manipulation during surgery
- ▶ Electrolytes imbalance narcotic analgesics and leak can be a cause too.
- ▶ bowel motion should return to normal 4-6 days after surgery
- ▶ Sx&Sx's : recurrent bilious vomiting, abd distention, failure to pass gas or stool, sluggish bowel sounds
- ▶ AXR shows diffusely dilated small bowel loops and multiple air-fluid levels
- ▶ Treatment : ^{↳ NG Tube} Keep NPO, treat the cause, early ambulation of patient.



Feeding post op :

- ▶ By jejunostomy feeding tube connected to a pump providing a liquid formula (ENSURE) starting from day 2-3
- ▶ Start oral feeding on day 4-7 (gradually from fluid to solid)
- ▶ Some surgeons perform oral contrasted studies routinely before oral feeding.
- ▶ May take months to return to near- normal feeding habits.
- ▶ Rule out leak and ileus before.

Chemotherapy for treatment of gastric adenocarcinoma :

▶ Neoadjuvant chemotherapy : (NACTx)

- Multiple different regimens , FLOT is the most recent one
- for any patients with locally advanced non metastatic disease on clinical pre-op staging T3/T4, N+, M0 (stage II, III)

▶ Benefits from NACTx :

- Improve resectability and chance of getting a negative margin resection.
- Test tumor behaviour/biology (aggressive vs non aggressive)
- Decrease recurrence and improve survival in locally advanced disease.
- Avoid delay in treatment with chemo that might occur after surgery if developed complications post op.

▶ Adjuvant chemotherapy :

- Given as a continuation of pre op chemo regimen or for those who have higher stage than before surgery (pts who were thought to be less than T1/2,N-)

Metastatic gastric adenocarcinoma

- ▶ 40% of pts present with metastatic disease at diagnoses.
usually M1 disease
- ▶ 36% of patients who were initially deemed to have a non metastatic disease found to be metastatic
- ▶ Some patients might be non metastatic but not fit for curative treatment and surgery (severe comorbidities, locally unresectable disease)
- ▶ Some pts may have recurrence after curative treatment
- ▶ The goal of treatment in all of these patients is only palliative (improve symptoms and quality of life only)- less chance of bleeding, perforation, relief of obstruction, pain and improve nutrition

▶ Palliative treatment options :

- ▶ Chemotherapy (minimal prolongation of survival, improve symptoms)
- ▶ Radiotherapy (decrease tumor size, less chance of bleeding/obstruction/perforation)
- ▶ Ascites : repeated tapping.
- ▶ Endoscopic : (stenting for obstruction, management of bleeding)
- ▶ Surgical ^{Tumor site.} ^{تحويل عن طريقه}
 - Palliative bypass (gastrojejunostomy)
 - Gastrostomy tube for decompression
 - Palliative resection not advised for most of patients

► Survival :

- Depends on stage, completeness of resection (if candidate)
- 5 yrs- survival :
- local disease 50-80%,
- +ve LN 30%,
- Metastatic 5%

► Recurrence :

- High even after complete resection (30%) *very high rate.*
- Most occur in the first 2 years (80% of recurrences)
- Can be local (surgery site) , *need follow up.*
- distant (liver) ,
- peritoneal mets
- Carry a dismal prognoses (6 months survival)
- Treated as palliative only (usually chemotherapy) or other modalities of palliative treatment *no repeated surgery after recurrence.*

► Post op follow up / surveillance:

- In clinic every 3 months in the first 2 years , then every 6 months
- Include full Hx, PEx, labs
- Endoscopy at 1,2,5 years if partial gastrectomy only
- CT scan every 6 months in the first 2 years , then annually

Gastric Lymphoma :

- ▶ 5% of gastric neoplasms.
- ▶ The Stomach is the commonest extranodal primary site for non-Hodgkin's lymphoma
- ▶ Secondary Lymphoma is commonly seen in stomach from another site
- ▶ The histologic types
 - low-grade B-cell lymphomas of the mucosa-associated lymphoid tissue (MALT) type.
 - high-grade diffuse large B-cell lymphoma (DLBCL);
 - **Mixed** containing both
- ▶ chronic gastritis and infection with H. pylori is the main predisposing factor
- ▶ Symptoms :
 - ▶ pain (68%^{ml}), anorexia(47%), weight loss (25%), bleeding (19%), and vomiting (18%).
 - ▶ Obstruction and perforation, Anaemia and epigastric mass.

due to H. pylori → Tx of H. pylori will cure patient.

Investigations

- Labs: CBC, ^{pull labs} LDH, ^{lymphoma Labs} B2-macroglobulin, bone marrow biopsy
- Staging CT
- Endoscopy + biopsy (histology + test for H.pylori)
- Might need deep biopsies
- EUS is the best modality.

Treatment :

- ▶ Early low grade MALT lymphoma → H.pylori eradication therapy
- ▶ Assessment of response with endoscopy, most pt will have complete remission
- ▶ persistent MALT lymphoma after *H. pylori* therapy → external beam radiation (90% to 100% rate of complete response)
- ▶ Single agent chemotherapy +/- Rituximab
- ▶ For DLBCL : combination of chemotherapy (R-CHOP)
- ▶ Autologous stem cell transplantation
- ▶ surgical intervention only in complication. in bleeding, perforation, obstruction, and residual disease after primary therapy.
- ▶ 5 yrs-survival : 90% for low grade, 50% for high grade tumors.

Gastrointestinal Stromal Tumors (GIST)

- <1 % of gastric CA ** usually as Extraluminal mass.*
- mesenchymal tumors
- Arises from interstitial cells of Cajal (pacemakers of GI motility)
- 95% stain with Ab against c-KIT (CD117) 70% stain for CD34
- 3-5% of the remainder of KIT-negative GISTs contain PDGFR-alpha mutations.
- Solitary , grows extraluminally to a large size
- Variable clinical behavior, from slow-growing indolent tumors to aggressive malignant cancers with the propensity to invade adjacent organs, metastasize to the liver, and recur locally within the abdomen.
- Site, size (>5cm) and mitoses(>5HPF) determines indolent vs more aggressive tumors *According to this → can be Benign or Malignant.*
- The stomach is the most common site of GIST. Other includes small bowel, colon and rectum

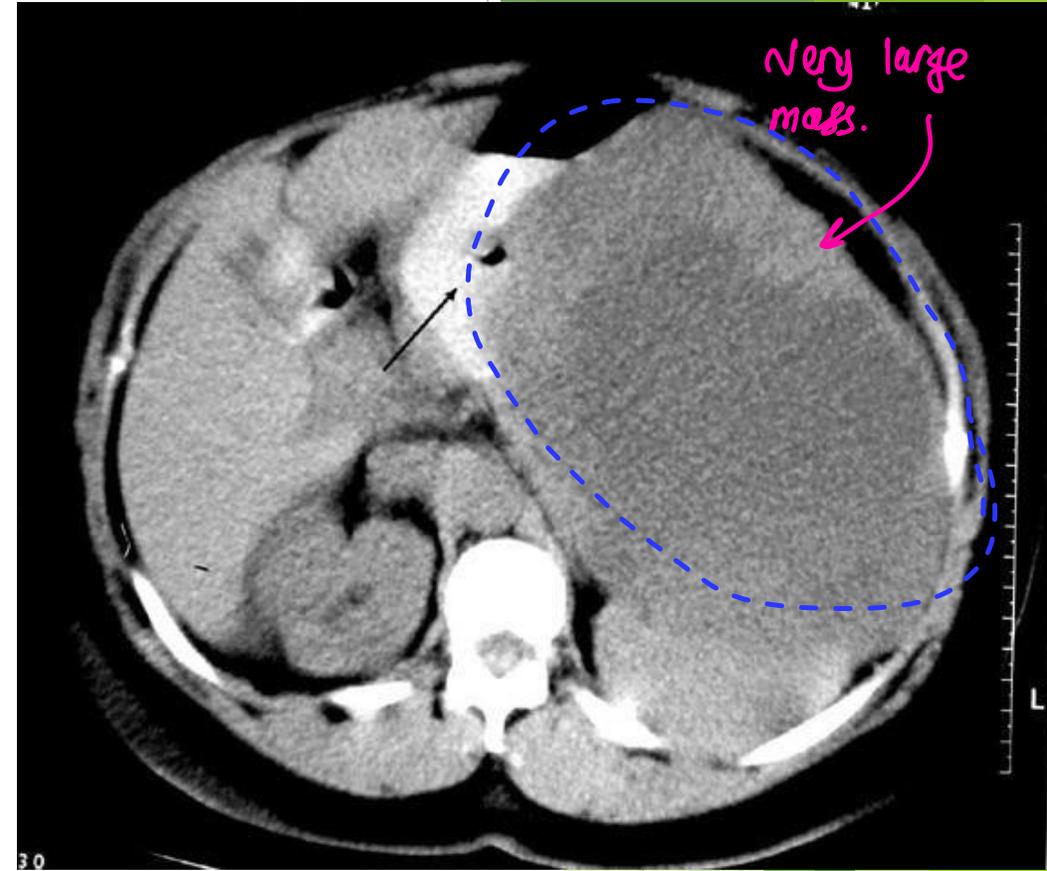
GIST tumors

► Presentation:

- might be asymptomatic / discovered incidentally or
↳ diagnosed Accidentally.
- Abdominal pain
- Anorexia
- Nausea, Vomiting
- Weight loss
- Epigastric fullness
- Early satiety
- Bleeding/perforation/obstruction

Investigations:

- ▶ Routine Labs
- ▶ Barium swallow, U/S , and AXR gives limited information
- ▶ *mostly* CT scan → very important to obtain
 - site, size, characteristics of tumor, relation to surrounding organs, mets)
- ▶ Endoscopy : (sometimes misses submucosal lesions)
- ▶ EUS better than endoscopy , also can obtain biopsy but limited value in large lesions.
- ▶ Biopsy is not always indicated if dx is suggestive and surgery is needed anyway



GIST tumours

- ▶ Treatment
 - *They don't spread to lymphatic so no need for lymph node resection.
- ▶ Surgery-local excision of tumor with 1cm -ve margins
 - Lymph node clearance unnecessary as spread is not common
 - Large tumors may need formal gastrectomy+/- adjuvant therapy (imatinib)
 - Tumor rupture/spillage during surgery and positive margin significantly worsens the prognosis
- ▶ Medical therapy consists of tyrosine kinase inhibitors (TKI), c-KIT inhibitor
 - *imatinib mesylate (Gleevec).
 - Preoperatively, to enable resectability or decrease morbidity by shrinking the tumor
 - After surgery, to decrease recurrence
 - In metastatic disease, as definitive treatment
 - In recurrent, unresectable disease, as definitive treatment

Done

