Gastric Surgical Diseases and Gastric cancers

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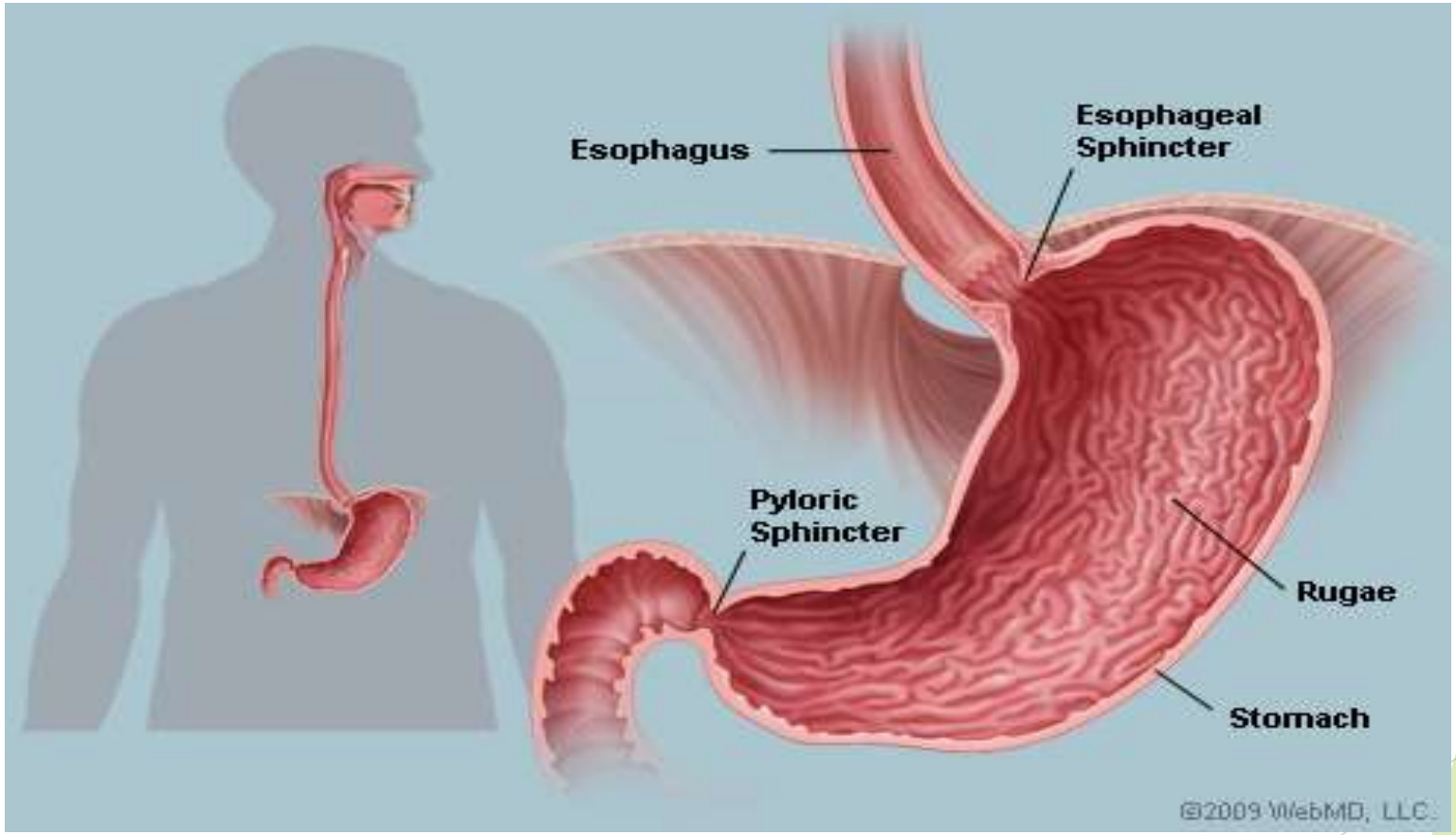
Outlines

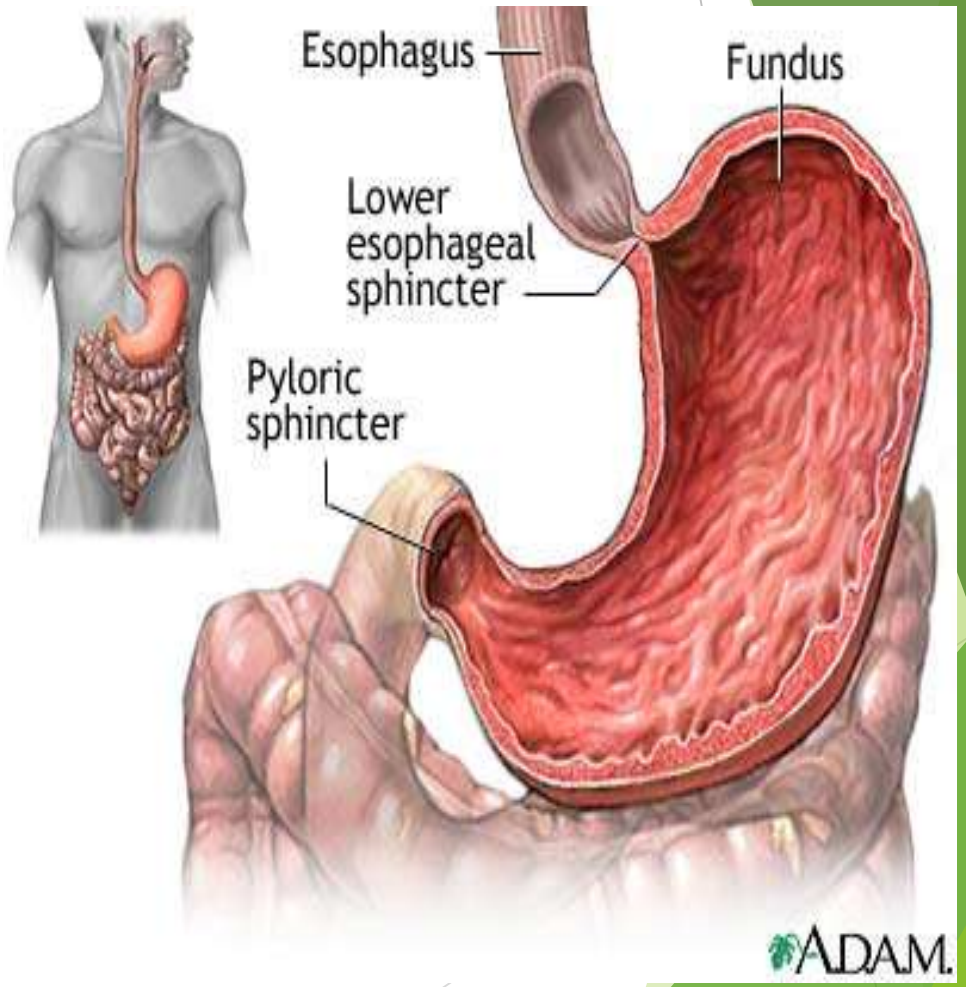
- Surgical anatomy of the stomach.
- H.PYLORI infection
- Peptic ulcer disease
- Post gastrectomy syndromes
- Gastric adenocarcinoma
- Gastric lymphoma
- Gastrointestinal stromal tumors (GIST)

STOMACH ANATOMY

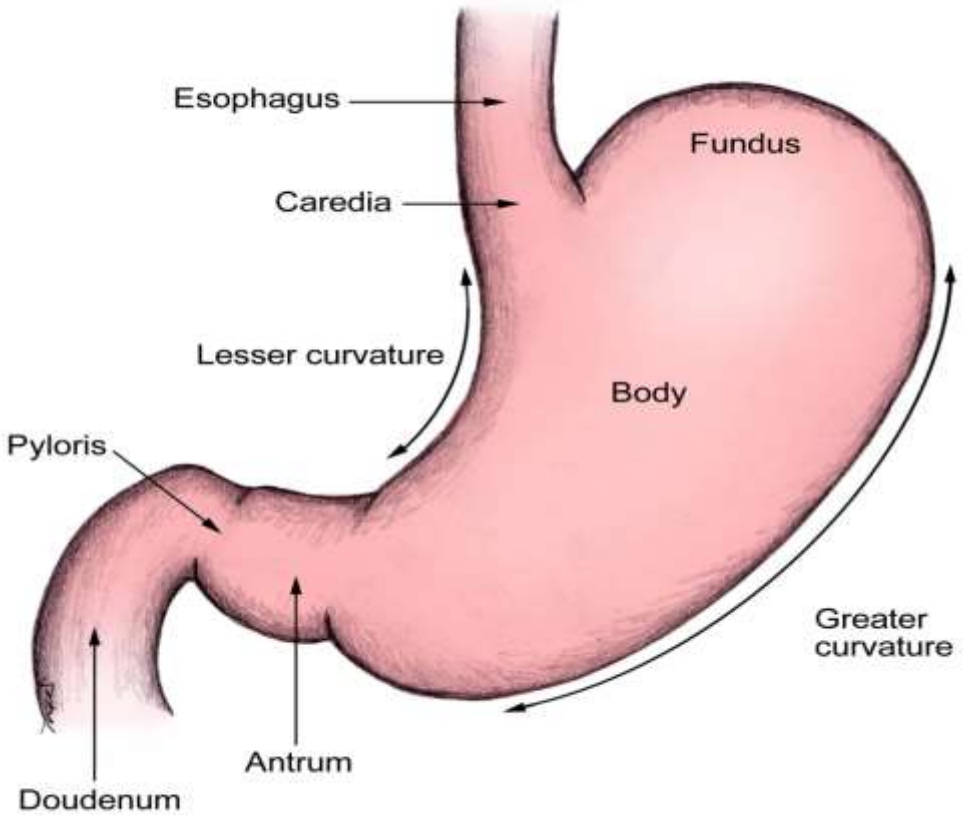


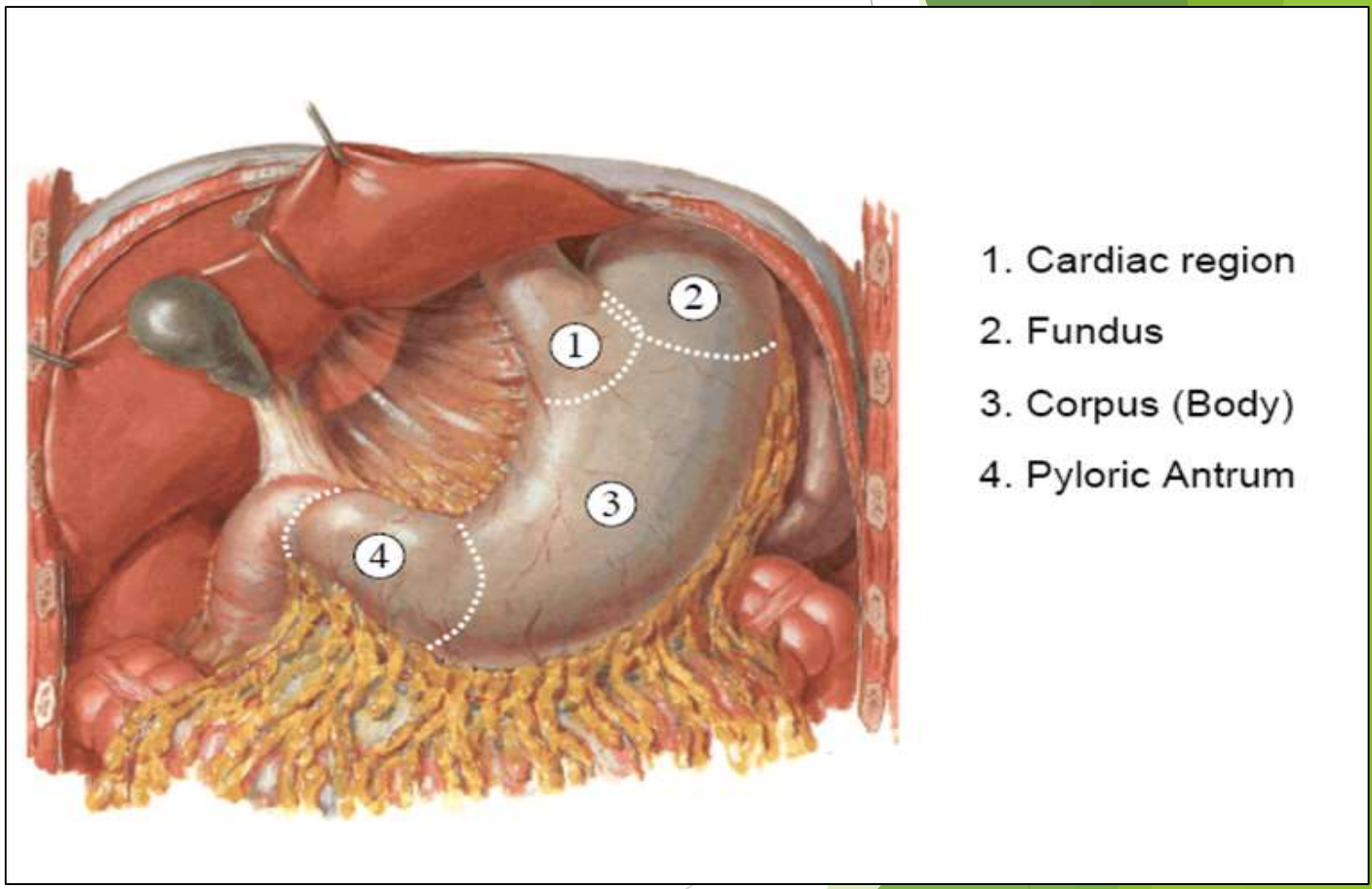
The stomach

- 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



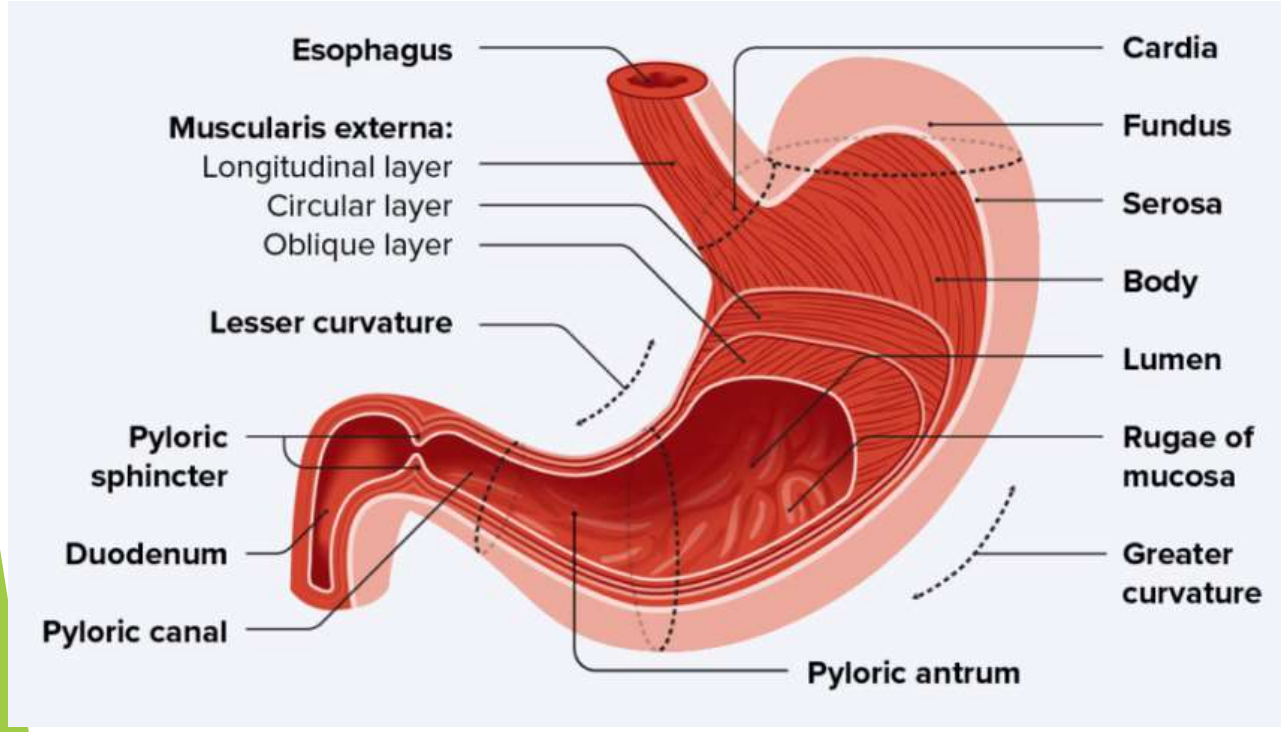
The Stomach

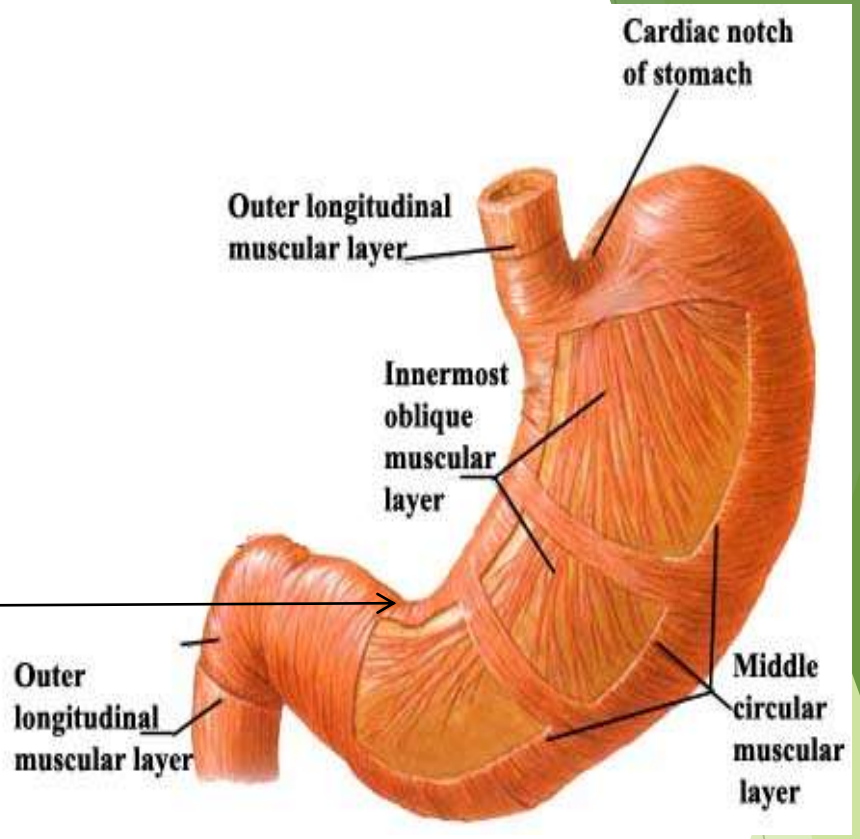

Esophagus
Caredia
Fundus
Lesser curvature
Body
Pyloris
Greater curvature
Antrum
Doudenum

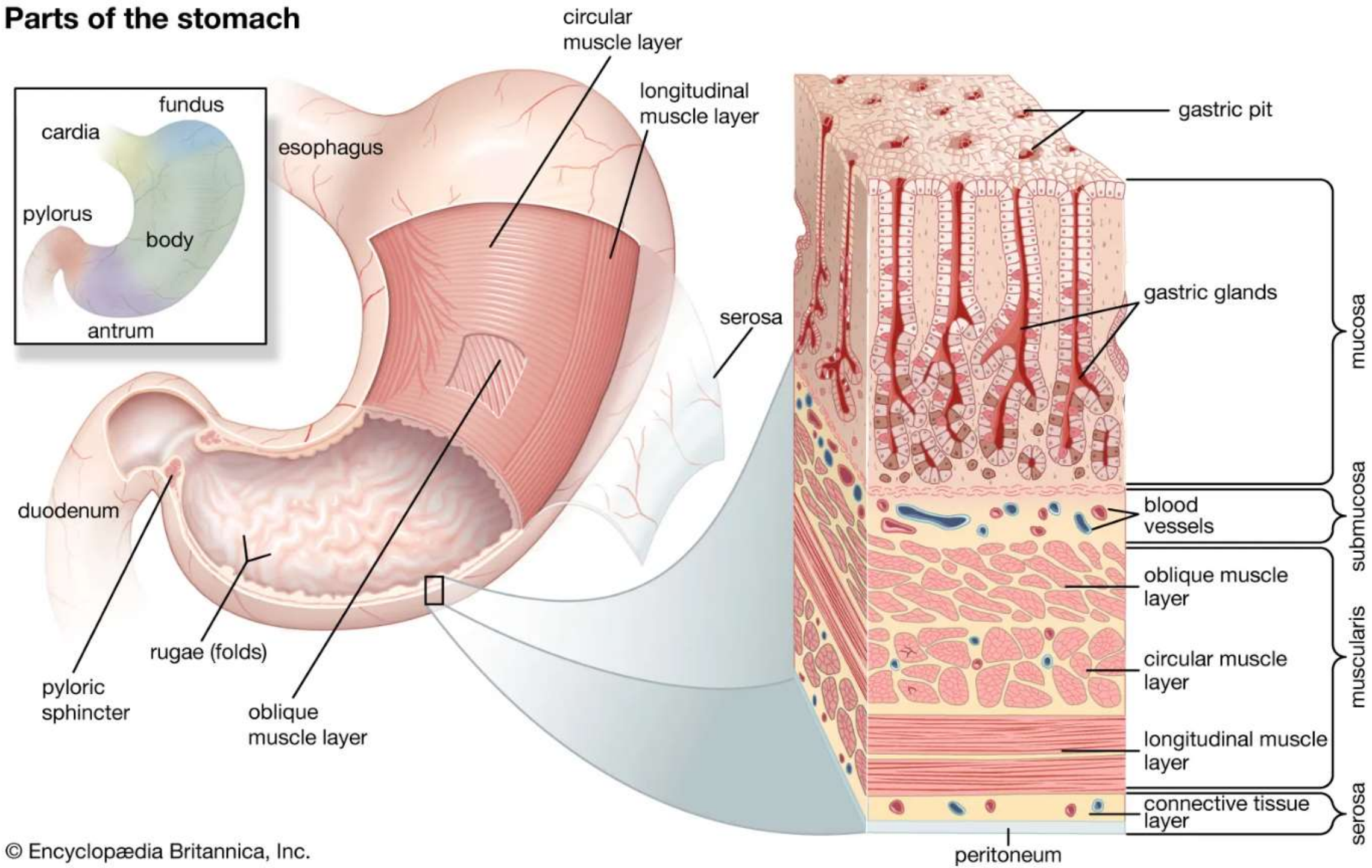

1
2
3
4
1. Cardiac region
2. Fundus
3. Corpus (Body)
4. Pyloric Antrum

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.






Parts of the stomach
fundus
cardia
pylorus
body
antrum
esophagus
circular muscle layer
longitudinal muscle layer
serosa
duodenum
rugae (folds)
pyloric sphincter
oblique muscle layer
gastric pit
gastric glands
blood vessels
oblique muscle layer
circular muscle layer
longitudinal muscle layer
connective tissue layer
mucosa
submucosa
muscularis
serosa
peritoneum
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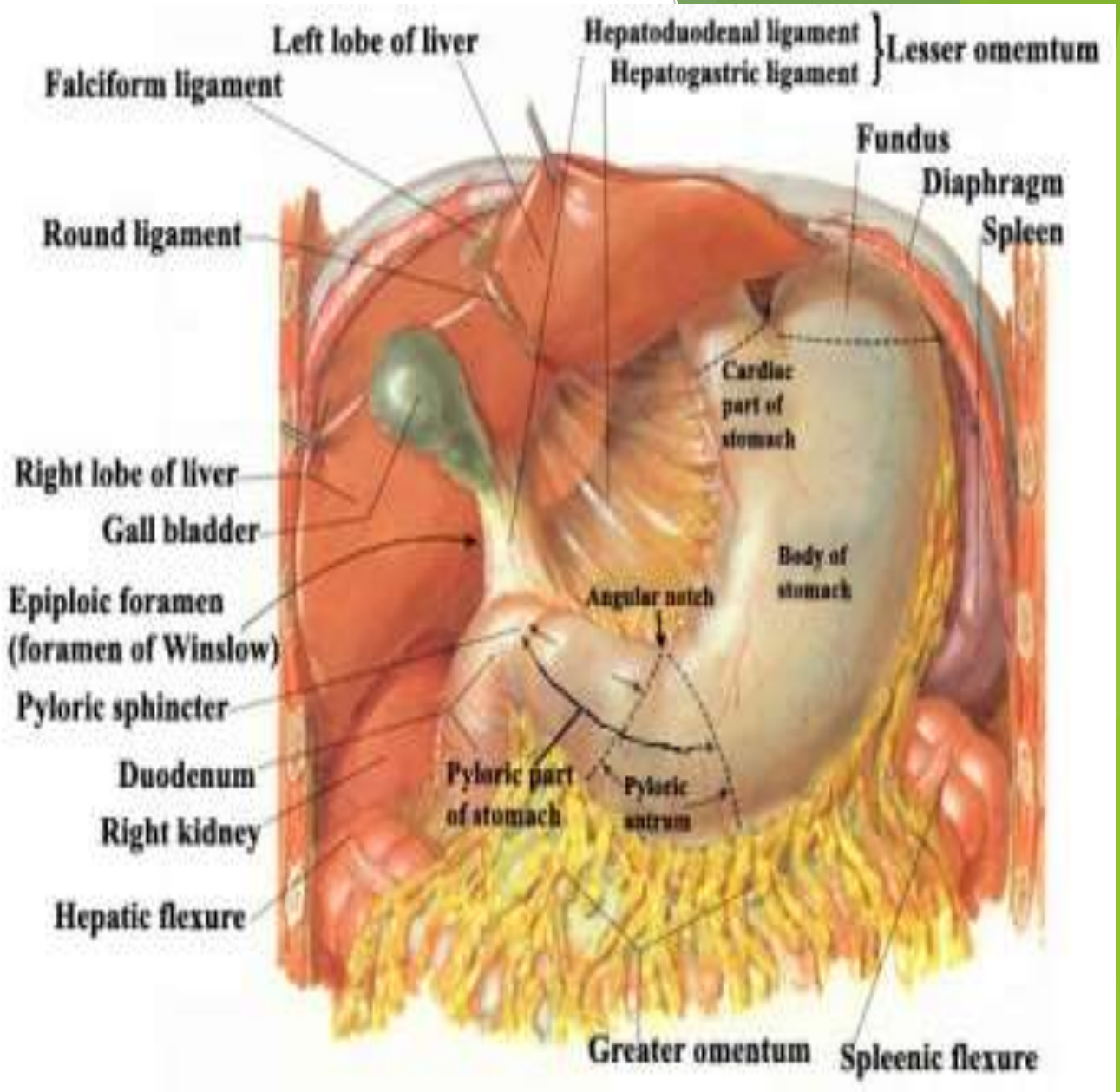
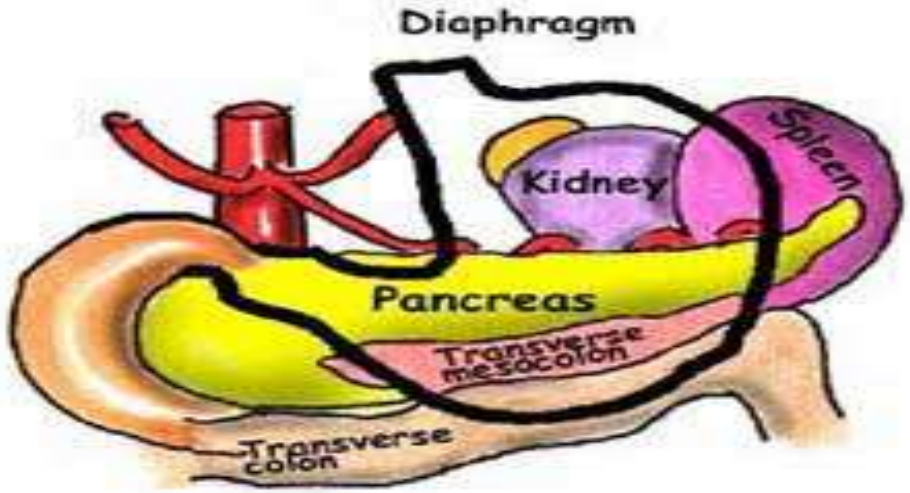
The stomach

- Layers of peritoneum attached to the stomach:
  - Lesser omentum: attaches the liver to the lesser curvature.
  - Greater omentum: attaches the greater curvature to the posterior body wall.



Stomach Anatomical Relation

STOMACH - RELATIONS



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

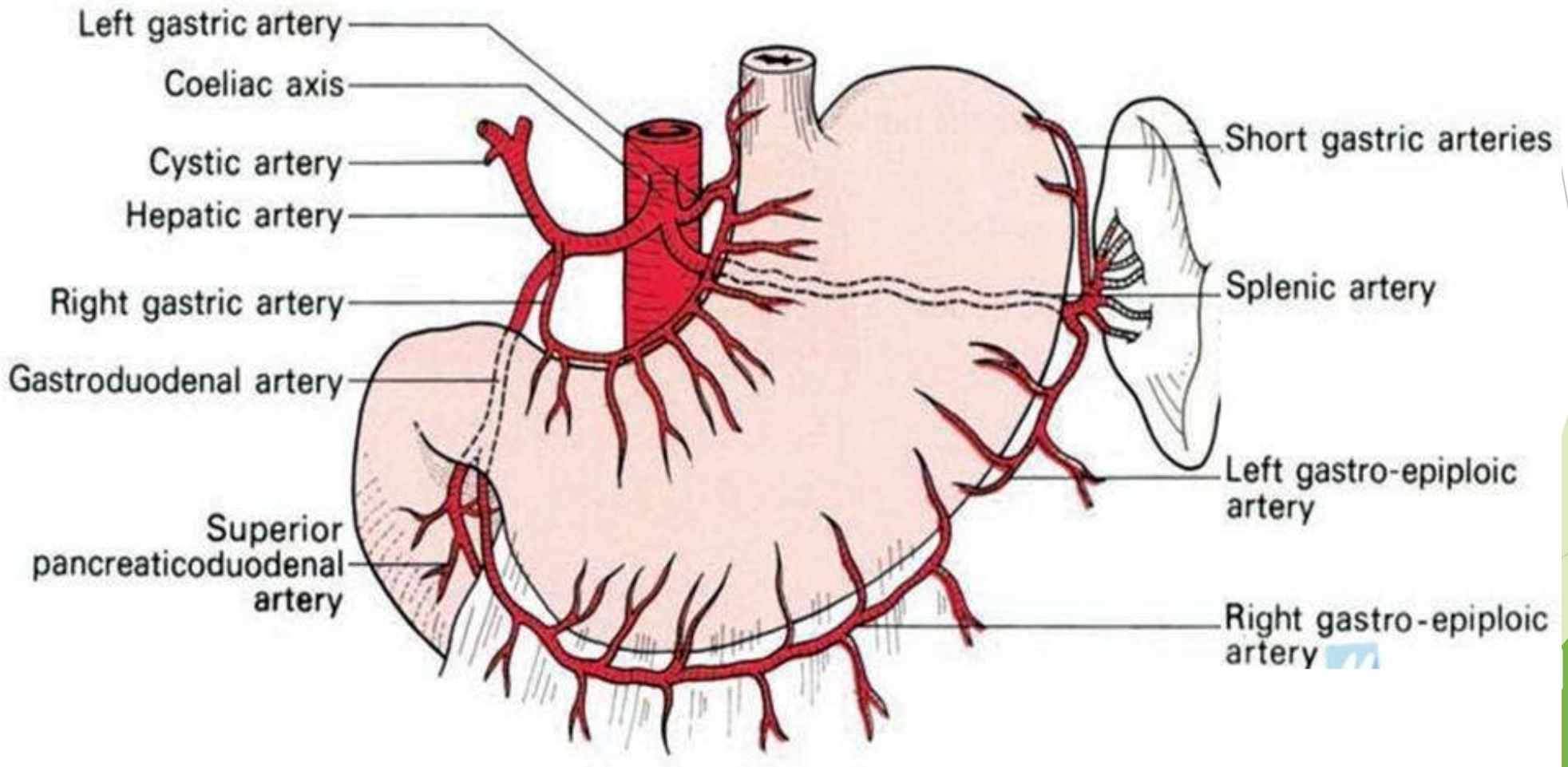
Arterial blood supply:

- 3 Branches
  - Left Gastric Artery
    - Supplies the cardia of the stomach and distal esophagus
  - Splenic Artery
    - Gives rise to 2 branches which help supply the greater curvature of the stomach
      - Left Gastroepiploic
      - Short Gastric Arteries
  - Common Hepatic or Proper Hepatic Artery
    - 2 major branches
      - Right Gastric- supplies a portion of the lesser curvature
      - Gastroduodenal artery

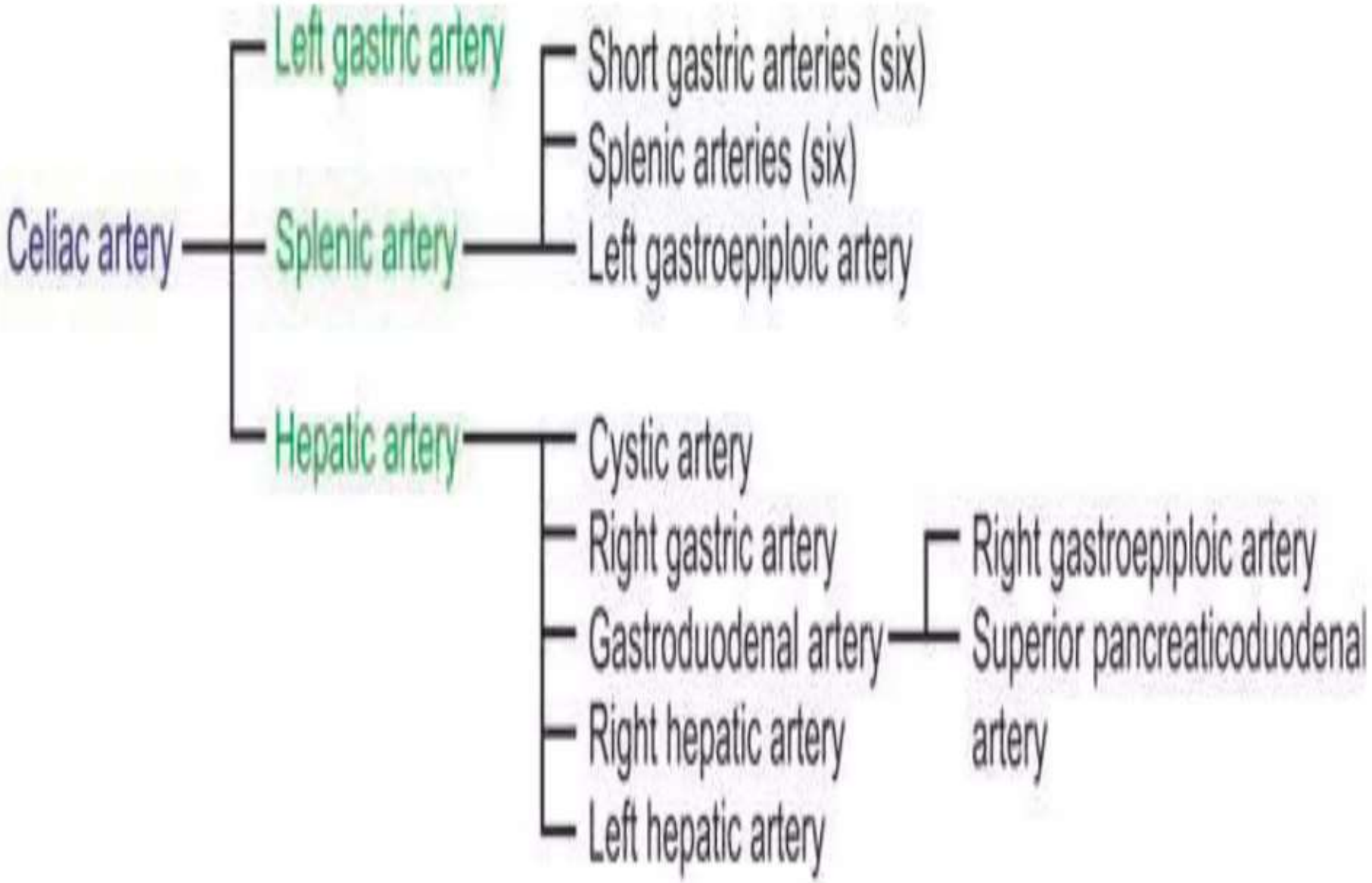
        -Gives rise to Right Gastroepiploic artery

        -helps supply greater curvature in conjunction with Left Gastroepiploic Artery

Blood supply of the Stomach

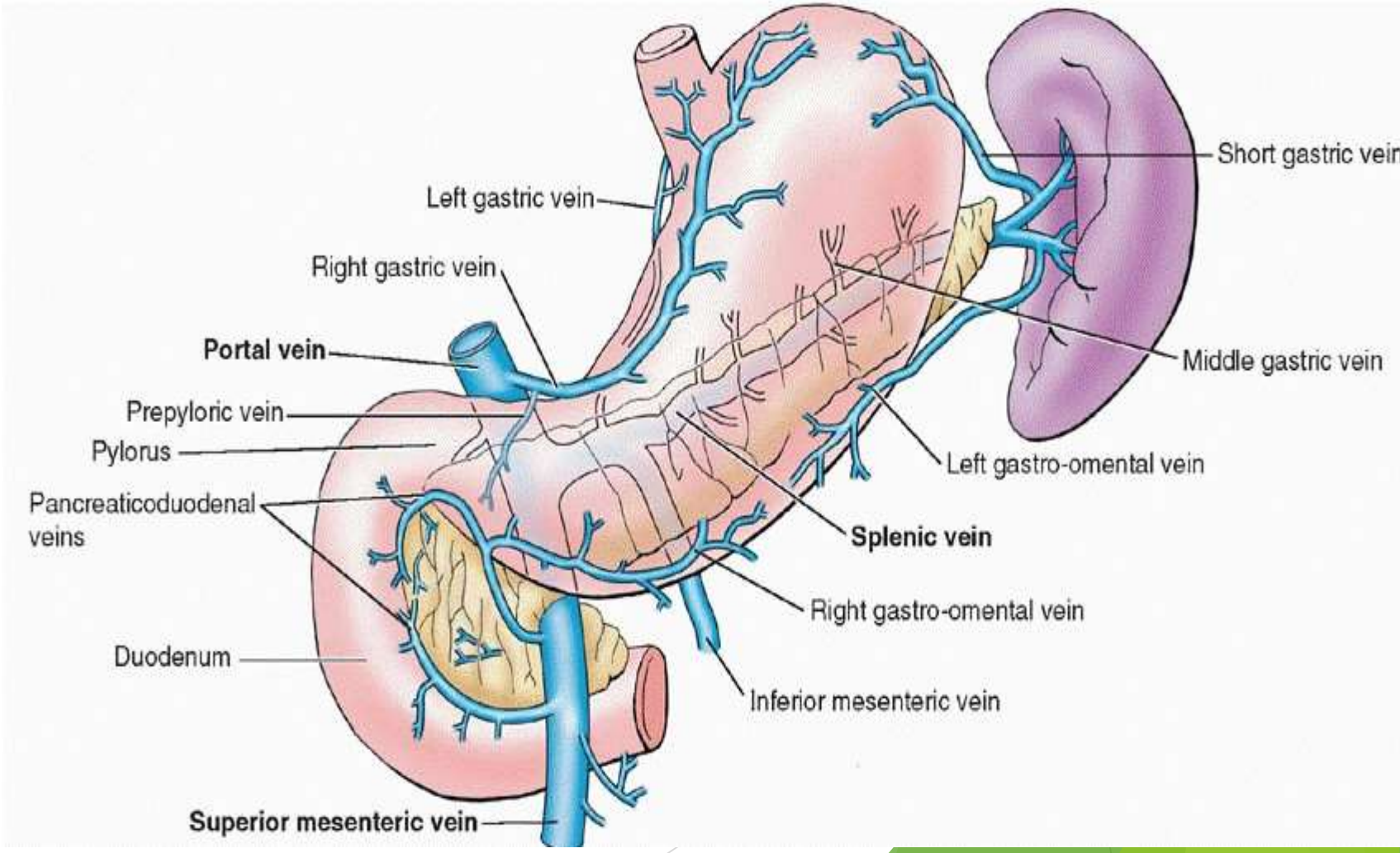


Arterial Supply Of Stomach

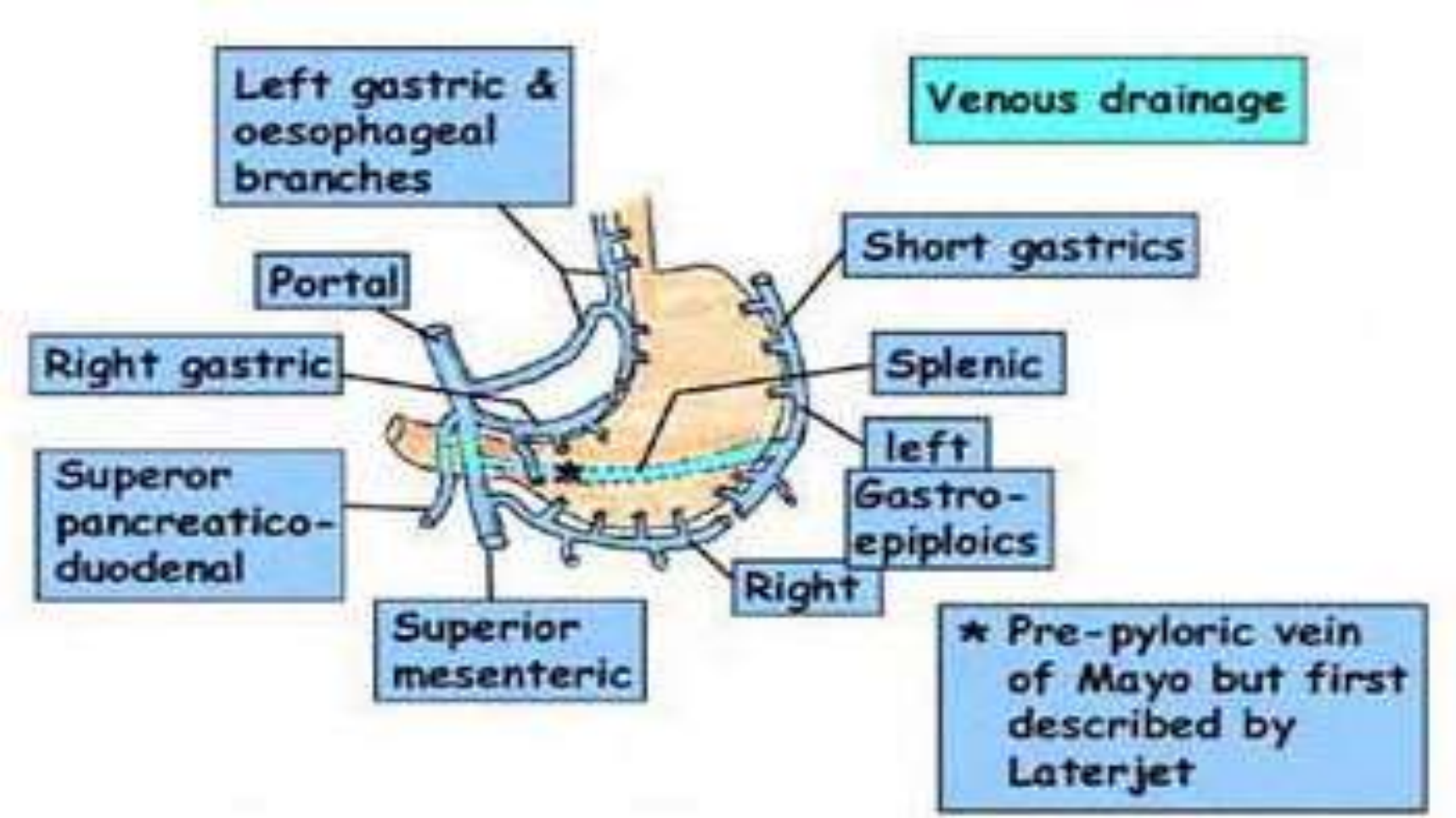


Stomach Venous Drainage

- **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



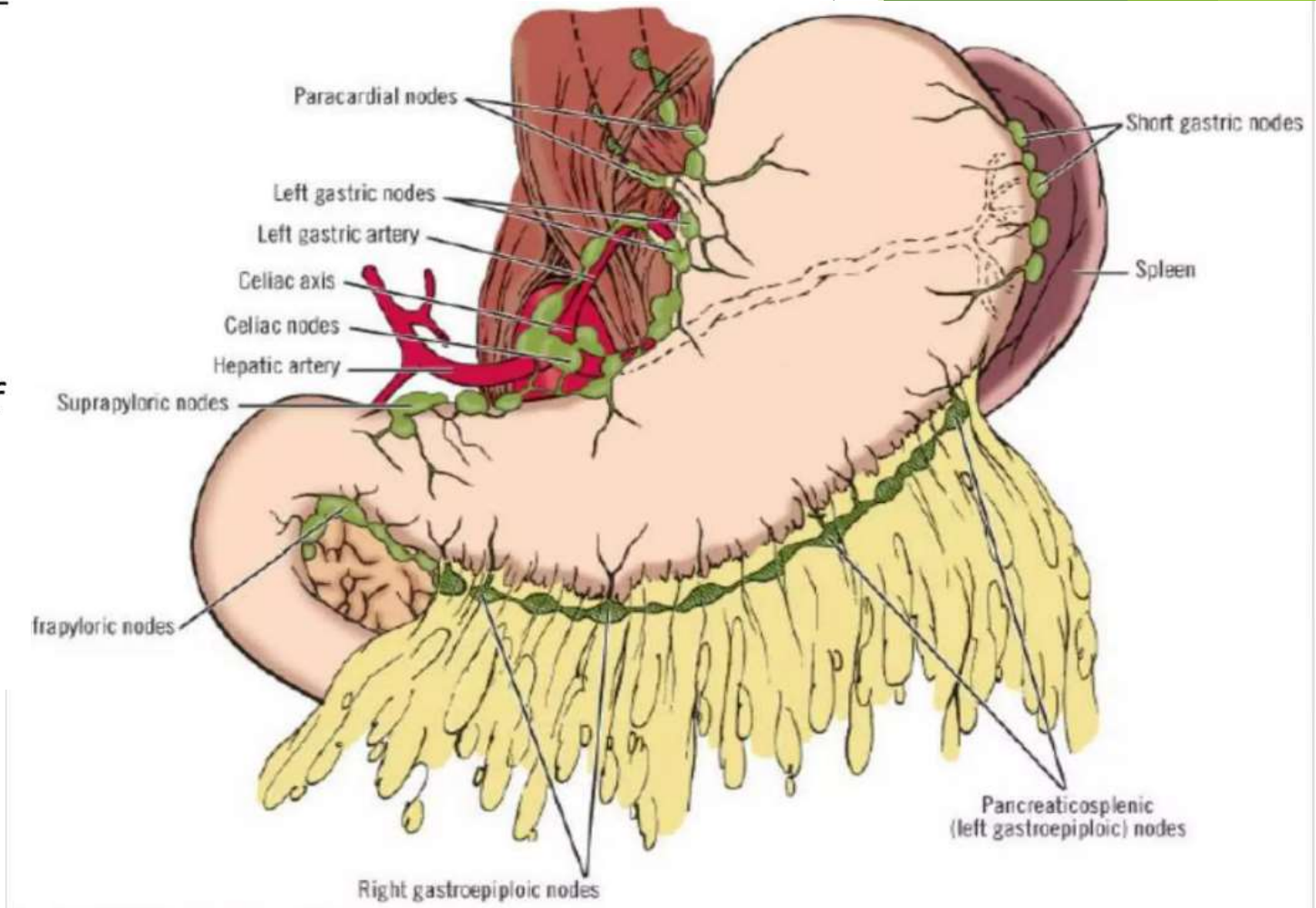
Stomach Venous Drainage



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



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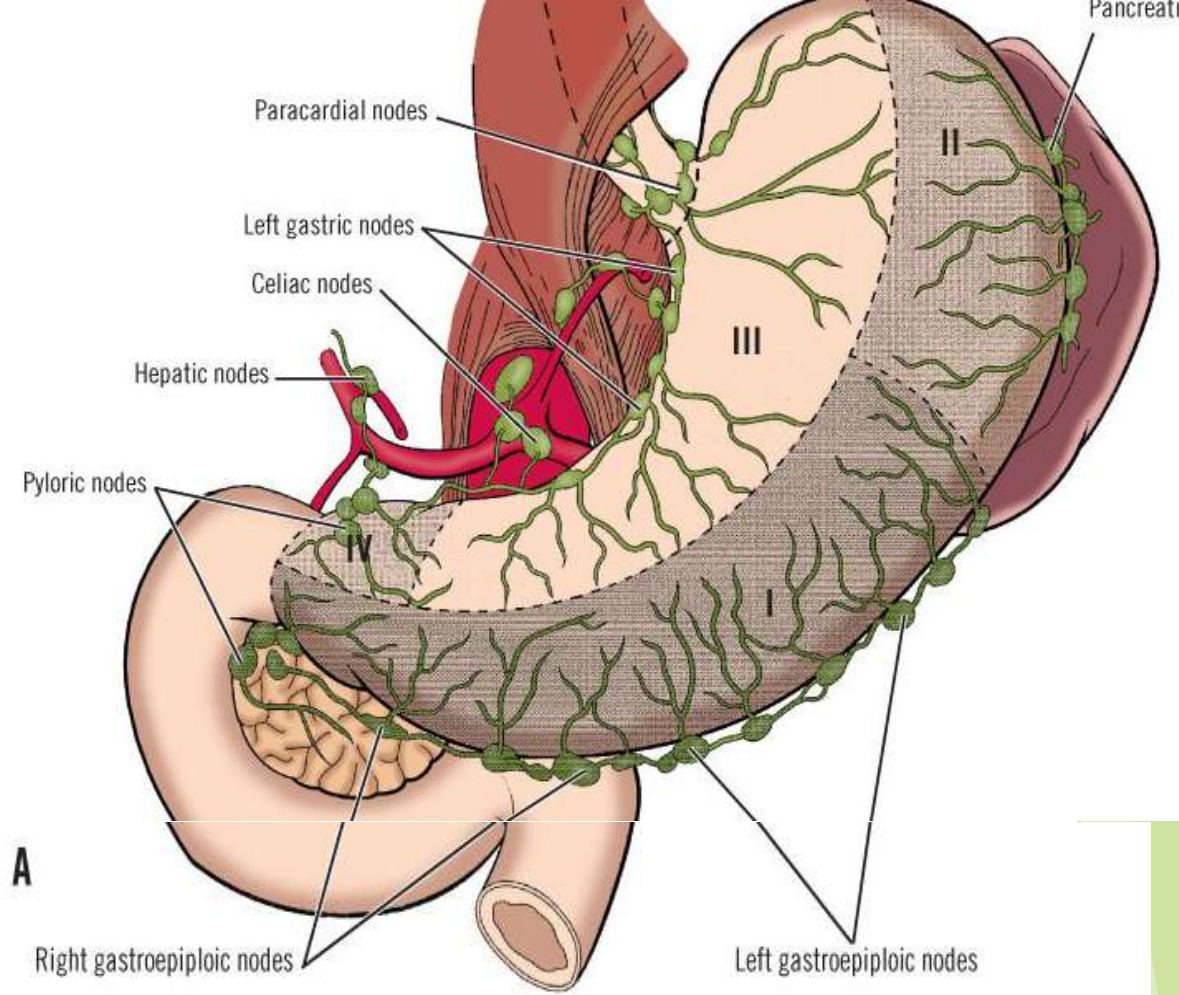
Eight groups of lymph nodes of the stomach.

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

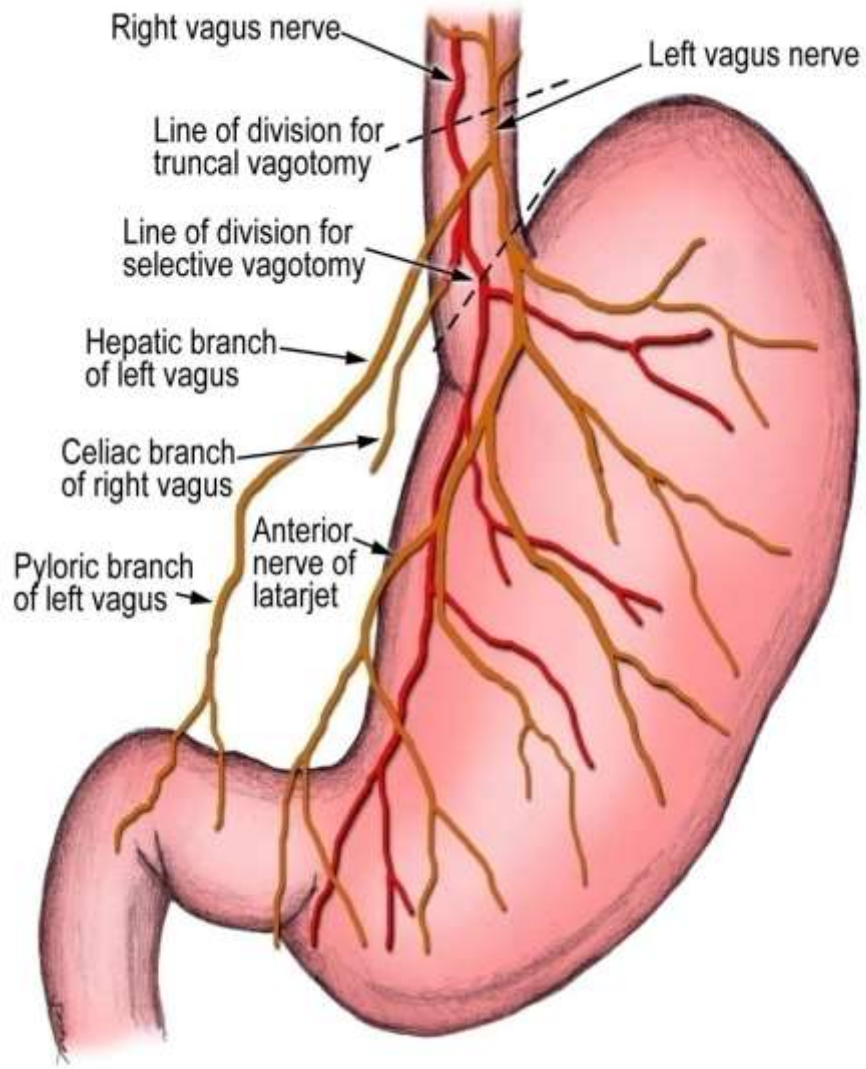
4. Hepatic-pyloric-left gastric nodes: drain the pyloric part of the stomach



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

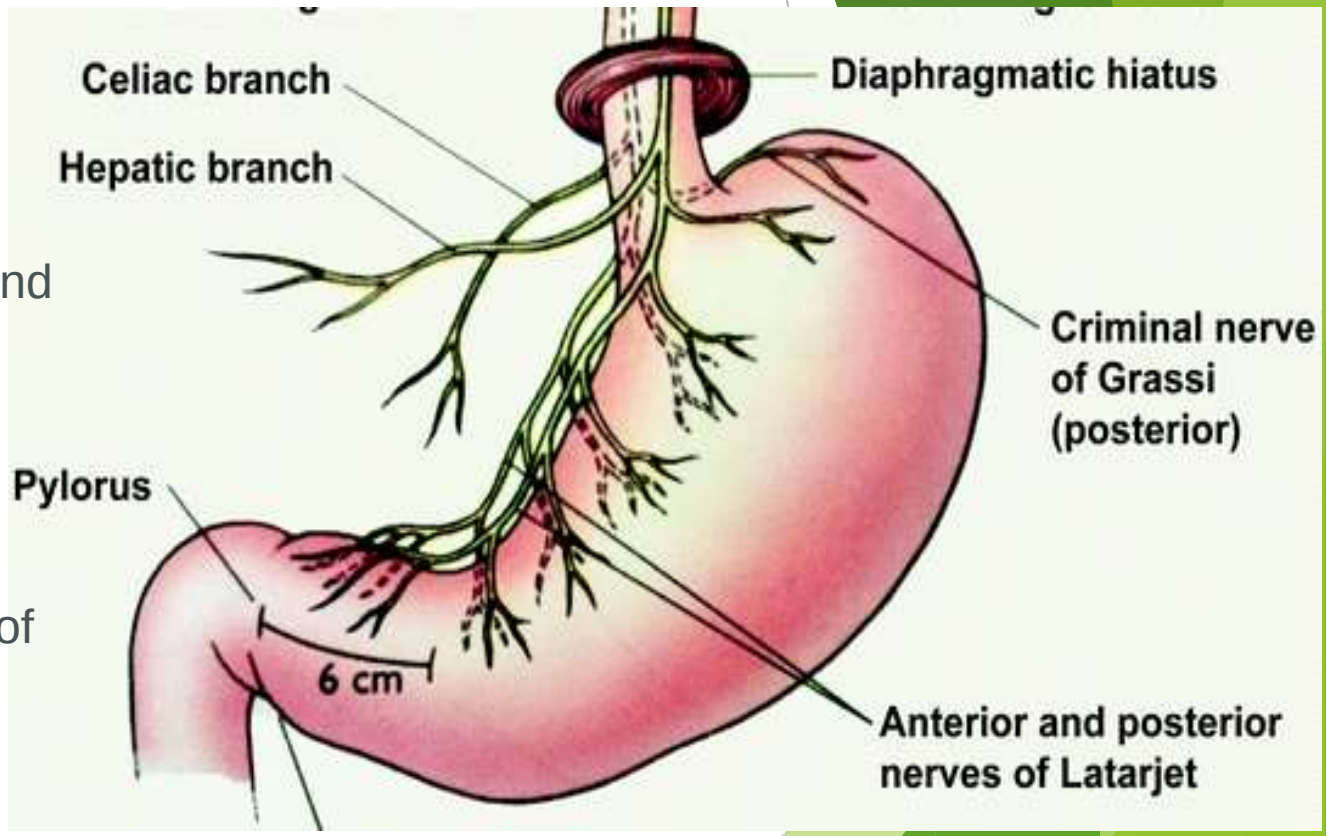
Stomach Innervations

- Parasympathetic innervation of Stomach- Vagus Nerve
- 80% of fiber 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



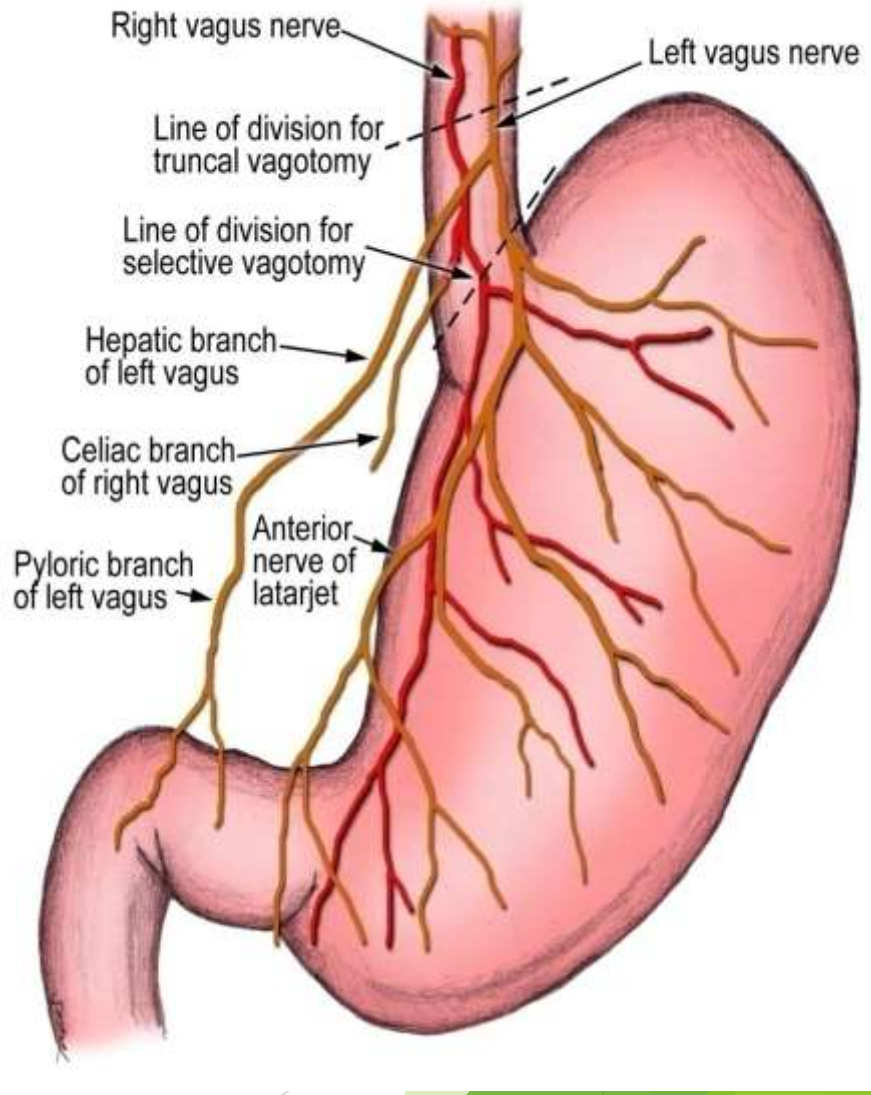
Vagus trunk

- 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** proximal branche of the posterior vagal trunk as a possible cause of recurrent ulcers if left undivided during selective vagotomy.



Levels of vagotomy

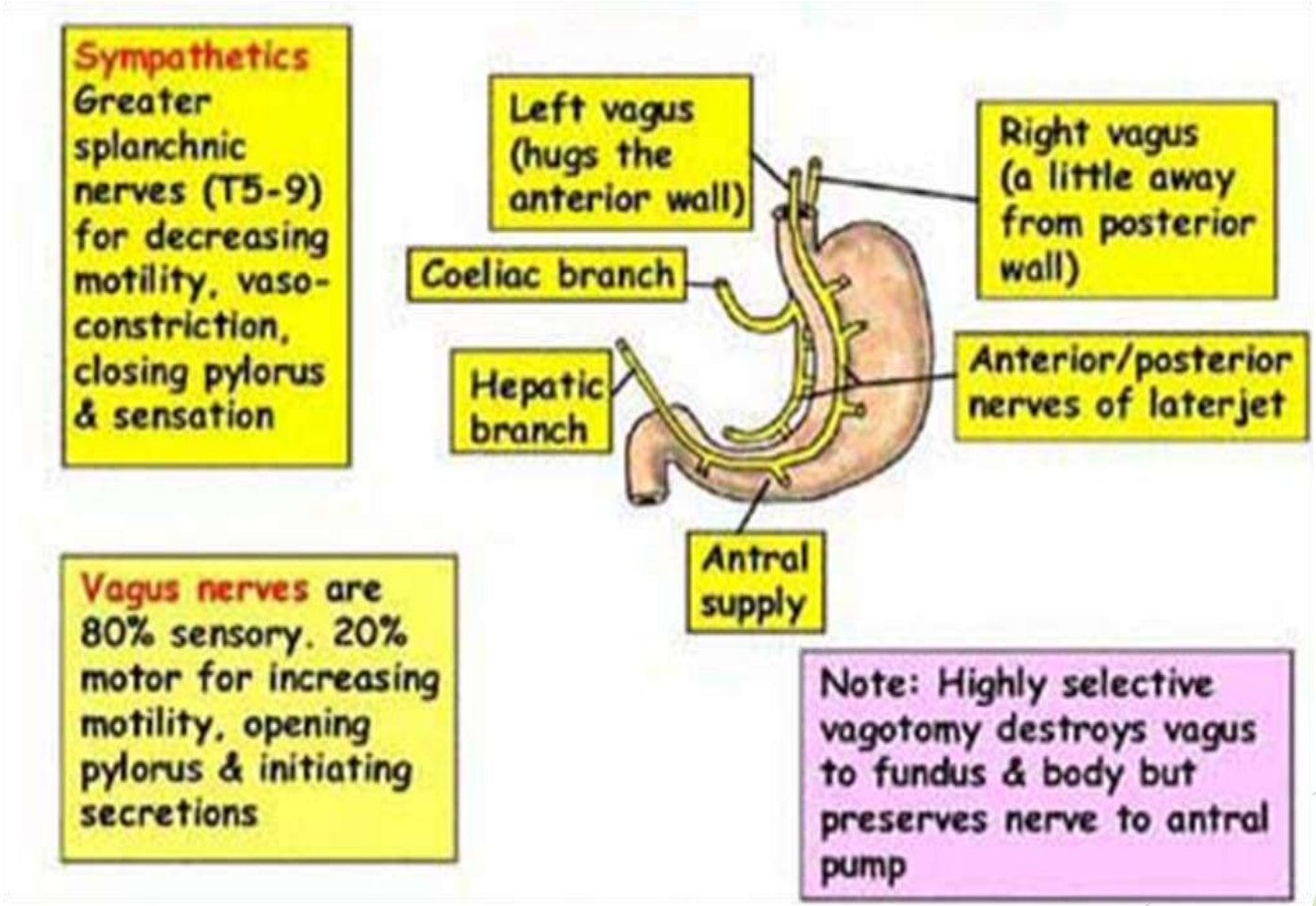
- **Truncal vagotomy** just below the diaphragmatic esophageal hiatus before it gives off celiac and hepatic branches.
- **selective vagotomy** is performed distal to this location and spares the celiac and hepatic branches.
- **Highly selective vagotomy** (parietal cell vagotomy) divides individual 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.

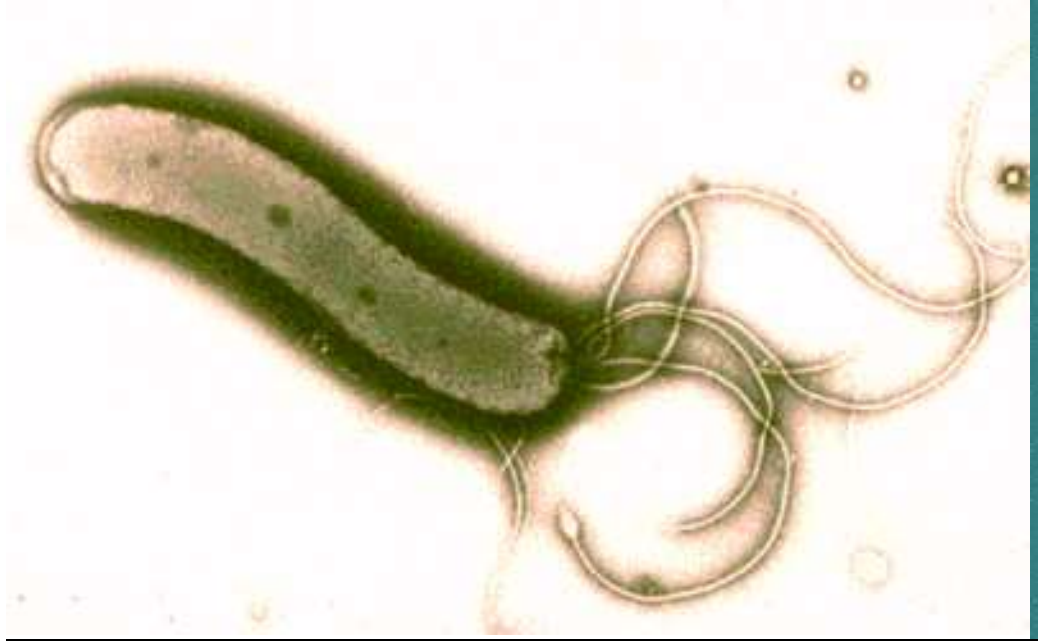


- **sympathetic** innervation is provided by the celiac plexus from the fifth to twelfth thoracic spinal nerves (T5-T10) and travel to the celiac plexus via the greater splanchnic nerves.

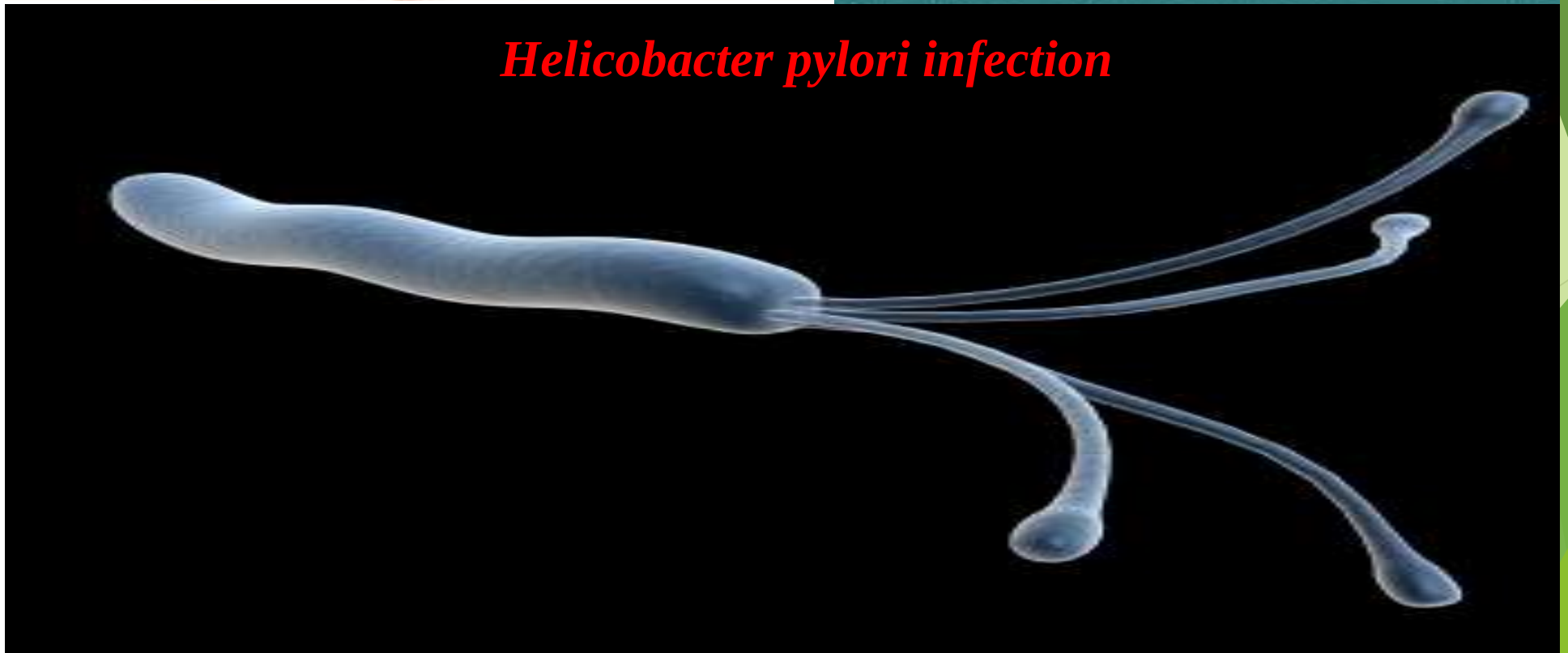
- Sympathetic innervation is responsible for inhibiting gastric motility and constricting the pyloric sphincter, thus preventing gastric emptying.

Stomach Innervations





Helicobacter pylori infection



- 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
- acute gastritis ——chronic gastritis
- PUD : 90% of DU , 75% of Gastric Ulcers
- Long standing chronic gastritis ——gastric atrophy & increased risk of metaplasia
- Gastric adenocarcinoma
- More than 90% of MALT lymphomas have H. pylori & low grade tumours regress with H. pylori eradication

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

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 *H. pylori*

- 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 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**
- **H. Pylori stool Antigen Test.**
- **urea breath test**
- 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.

- **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.
- **Histologic examination** can accurately diagnose H. pylori with two biopsy specimens with high sensitivity and specificity (90%).
- **The rapid urease test** on a mucosal biopsy is considered the initial test of choice because of its simplicity, accuracy, and rapid results.
- **Culture of H. pylori** has the most specificity (100%) but is difficult to perform (only for research purposes or patients with suspected antibiotic resistance.

Treatment : Eradication therapy

* triple therapy :

For the population with low clarithromycin resistant rate 15%

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

Surgical peptic ulcer diseases either:

* perforation
* bleeding
*gastric outlet obstruction

- 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 :

- *H pylori* infection
- Drugs (NSAIDS, steroids)
- Severe physiologic stress (burns/curling ulcer, head trauma/ cushing ulcer…. etc
- Hypersecretory states (uncommon) – Zollinger Ellison , G-cell hyperplasia
- Genetic factors
- smoking

- 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
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
Short gut syndrome (after significant intestinal resection)

History and Exam

- Epigastric pain, (gnawing or burning)
- Dyspepsia, including belching, bloating, distention, and fatty food intolerance
- Heartburn
- Chest discomfort
- Hematemesis or melena.
- 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 or peritonitis (if perforation occurred)

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

Alarming symptoms :

- Bleeding or anemia
- Early satiety
- Unexplained weight loss
- Progressive dysphagia or odynophagia
- Recurrent vomiting
- Family history of gastrointestinal cancer

- → all need endoscopy to be performed

workup

- H.pylori stool antigen testing (if presentation at the clinic)
- Endoscopy + biopsy → for all suspected PUD especially if alarming symptoms present or new onset/refractory symptoms in >50 years old patients.
- CXR (if perforation suspected)
- Serum gastrin level (if multiple, unusual location, +FHx, unidentifiable cause, +diarrhea,+steatorrhea,weight loss, ulcer refractory to treatment)
- Secritin stimulation test
- Angiography (if massive bleeding)

Treatment :

- H.pylori eradication (triple therapy for low clarithromycin resistant rate less than 15%) otherwise quadruple therapy, rifabutin for refractory cases
- Confirm eradication of H pylori and healing of ulcer with endoscopy or H pylori stool antigen after 2 weeks of cessation of PPI, 4 weeks of the antibiotic course .
- Stop NSAIDs, PGE analogue (misoprostol) , prophylactic PPI
- SURGICAL treatment :

- Now largely abandoned because of high success rate for medical therapy.
- 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

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

Antrectomy only

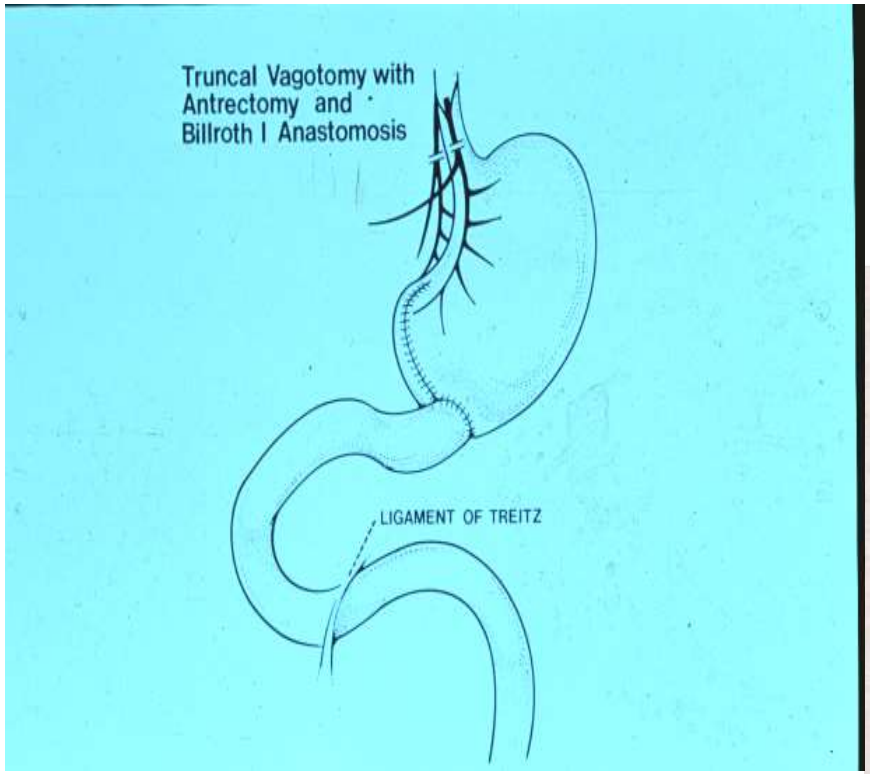
Type II,III treated as duodenal ulcer, preferred tx include antrectomy (to include ulcer) + truncal vagotomy with Billroth I OR II

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

SURGICAL OPTION

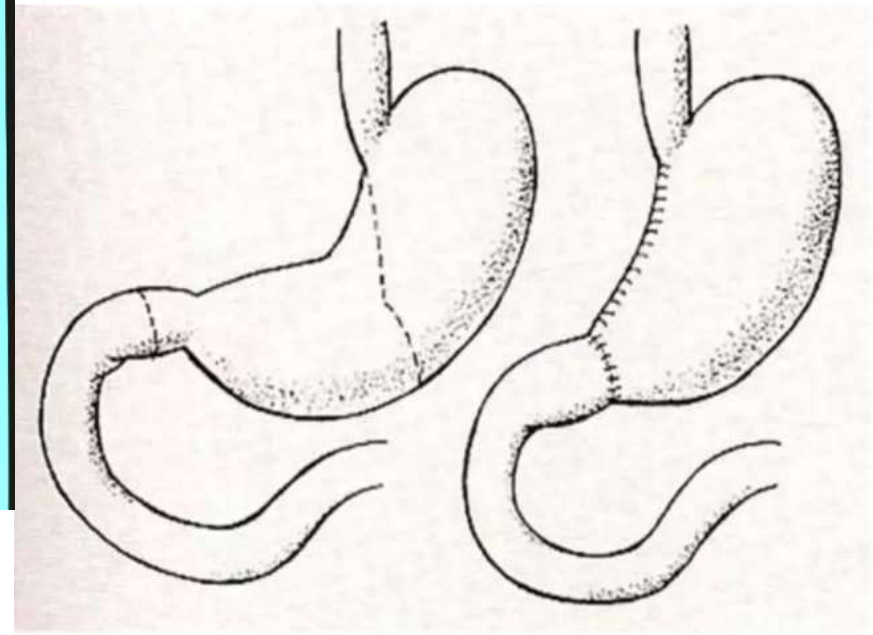
- VAGOTOMY
  - Truncal and drainage
  - Selective
  - Highly selective
  - Posterior vagotomy and anterior seromyotomy
- GASTRECTOMY
  - Billroth I
  - Billroth II
  - Subtotal gastrectomy
- GRAHAM'S OMENTAL PATCH
- SUTURE LIGATION OF GASTRODUODENAL ARTERY
- UNDRER-RUNNING AN ULCER BASE
  - After excision of the edge
  - Vagotomy

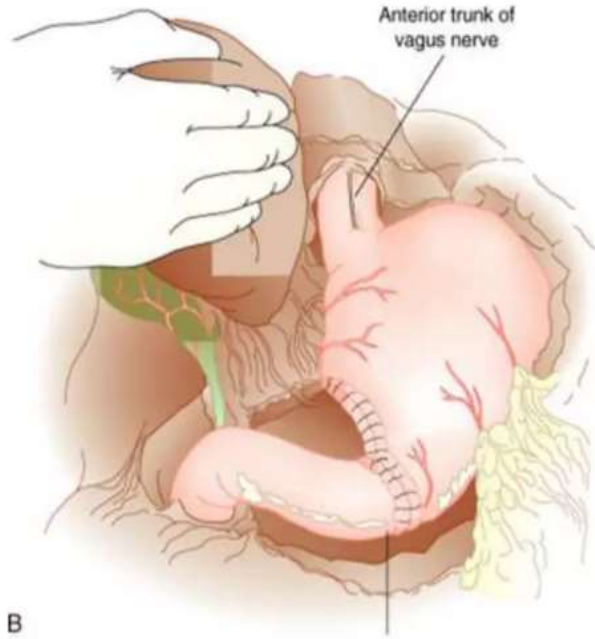
Billroth I



Billroth I – partial gastrectomy gastro-duodenostomy end-to-end

Done for gastric ulcer in the antrum





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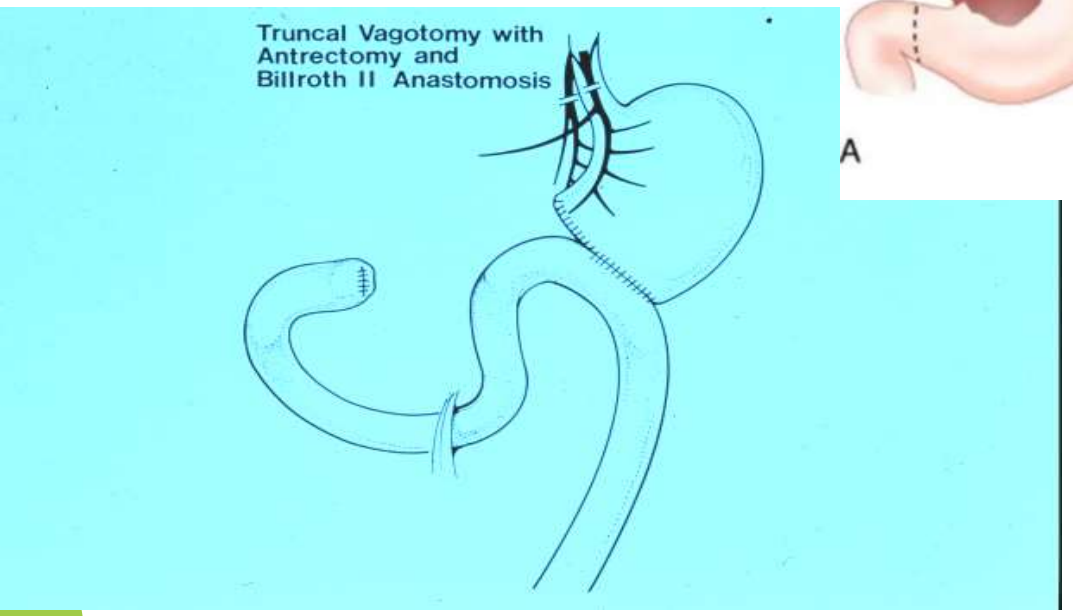
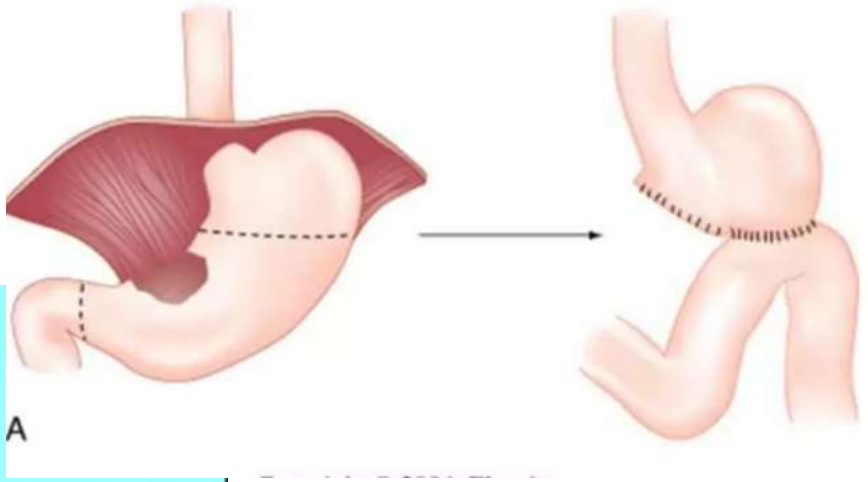
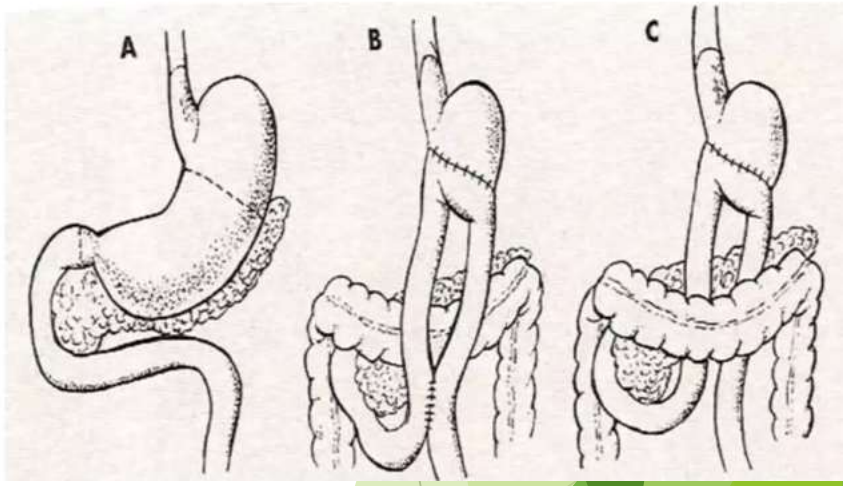
Billroth II

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

Done for a proximal gastric ulcer







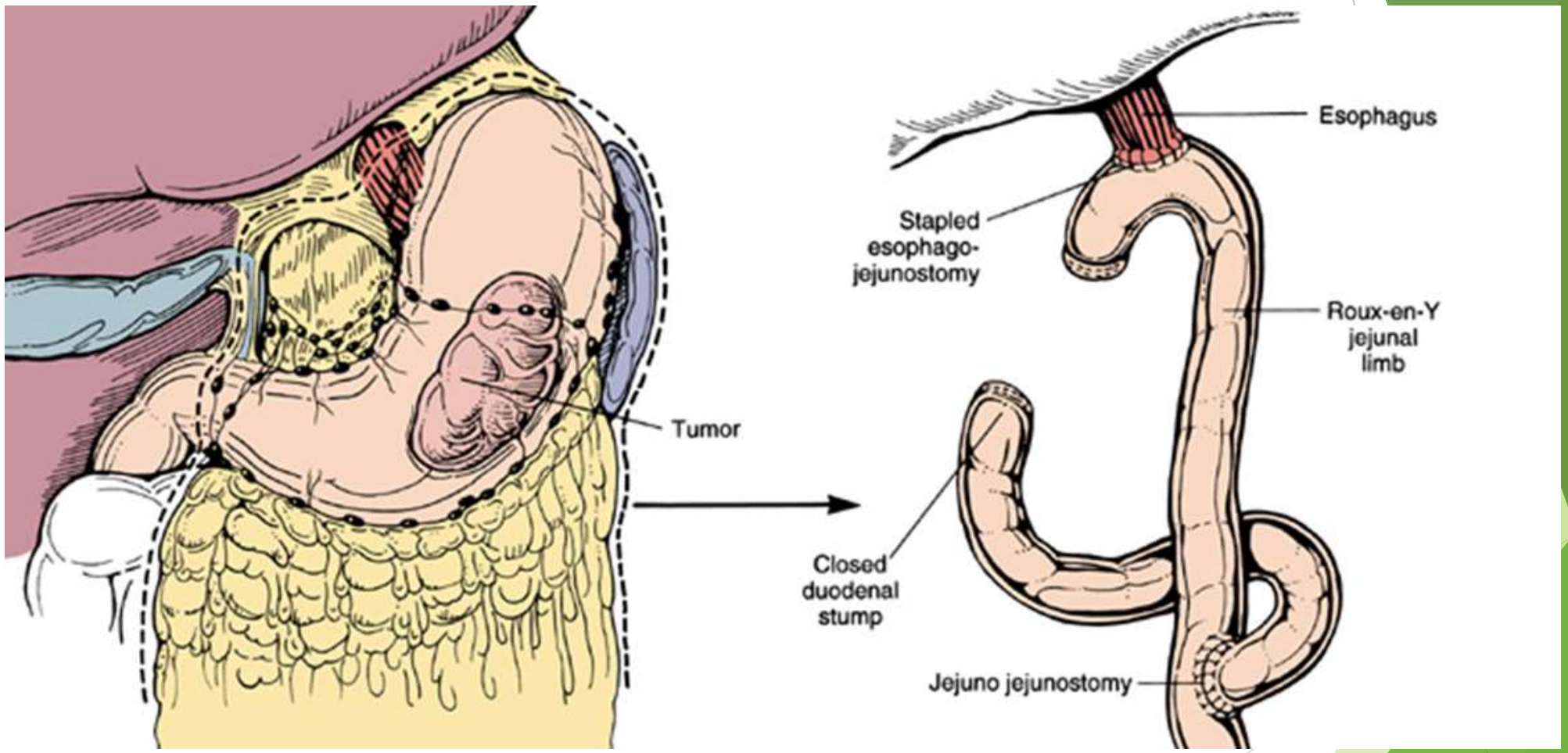

Tumor
Esophagus
Stapled esophago-jejunostomy
Roux-en-Y jejunal limb
Closed duodenal stump
Jejuno jejunostomy

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

Indication	Duodenal	Gastric
Bleeding	1. Oversew[a] 2. Oversew, V+D 3. V+A	1. Oversew and biopsy[a] 2. Oversew, biopsy, V+D 3. Distal gastrectomy[b]
Perforation	1. Patch[a] 2. Patch, HSV[b] 3. Patch, V+D	1. Biopsy and patch[a] 2. Wedge excision, V+D 3. Distal gastrectomy[b]
Obstruction	1. HSV + GJ 2. V+A	1. Biopsy; HSV + GJ 2. Distal gastrectomy[b]
Intractability/nonhealing	1. HSV[b] 2. V+D 3. V+A	1. HSV and wedge excision 2. Distal gastrectomy

[a]Unless the patient is in shock or moribund, a definitive procedure should be considered.

[b]Operation of choice in low-risk patient.

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 :

- 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 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, but the highest recurrence rate, which ranges from 5% to 15%.

Table 25-13 Clinical Results of Surgery for Duodenal Ulcer

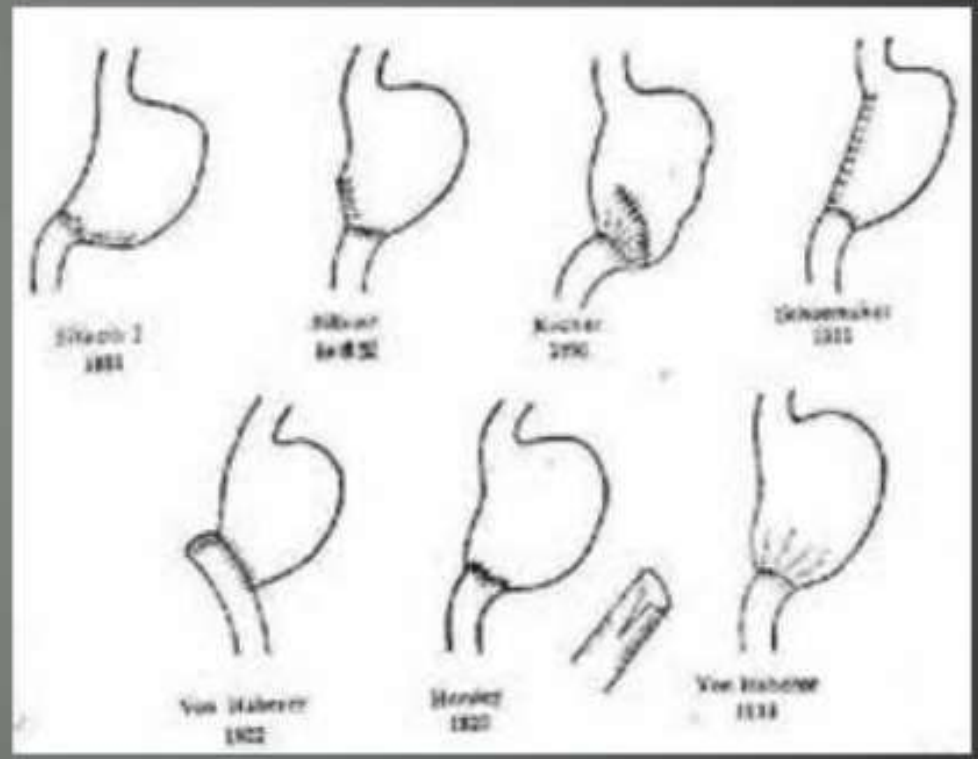
	Parietal Cell Vagotomy	Truncal Vagotomy and Pyloroplasty	Truncal Vagotomy and Antrectomy
Operative mortality rate (%)	0	<1	1
Ulcer recurrence rate (%)	5–15	5–15	<2
Dumping (%)			
Mild	<5	10	10–15
Severe	0	1	1–2
Diarrhea (%)			
Mild	<5	25	20
Severe	0	2	1–2

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

Billroth I

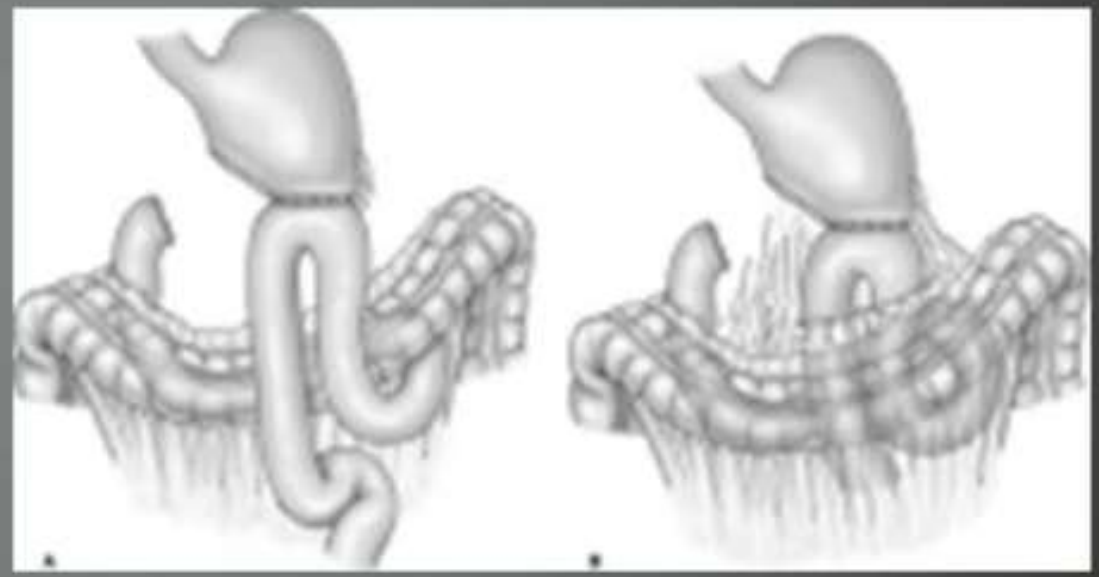
- Preserves duodenal passage
- Preserves pancreatic function
  - Altered after gastrojejunostomy
  - Better fat digestion
- Less incidence of gastritis and reflux
- Can use only with distal limited gastric resections

tension



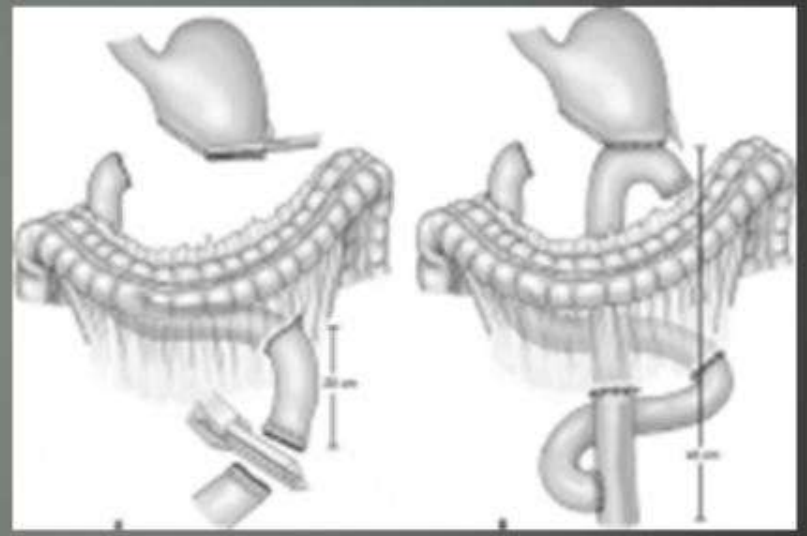
Billroth II

- 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



Postgastrectomy 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
- Early Dumping syndrome
- Late dumping syndrome
- Post-vagotomy Diarrhea
- Afferent loop syndrome
- Blind loop syndrome
- Alkaline Reflux gastritis
- Roux stasis syndrome
- Recurrent ulcers
- Gastric atony
- Metabolic disturbances

Table 1: Post gastrectomy syndromes: classification

Acute complications	- Anastomotic leak - Duodenal stump blowout
Gastric reservoir dysfunction	- Dumping syndrome (Types I and II) - Metabolic alterations
Vagal denervation	- Gastric stasis and hurry - Gallstones
Aberrations of reconstruction	- Bile reflux gastritis - Afferent loop syndrome - Efferent loop syndrome - Jejunogastric intussusception - Roux stasis syndrome - Gastro-jejuno-colic fistula
Long term complication	- Recurrent ulcerations - Remnant stump cancer

Dumping syndrome :

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

- **Early** dumping :

- Occurs 15-30 mins after meal
- 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 .

- Sudden anxiety, diaphoresis, tremor, tachycardia, palpitations, weakness, fatigue.
- It is not associated with borborygmi or diarrhea.

- 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. 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 :

- Diet modification (most pts will improve)
- Smaller more frequent meal bulks.
- Lower carbs in food (less osmolar food)
- 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 peristalitic interposition jejunal loop
- Conversion of Billroth II TO Billroth I can be an option

Vagal Denervation : Diarrhea

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

Vagal Denervation : Diarrhea

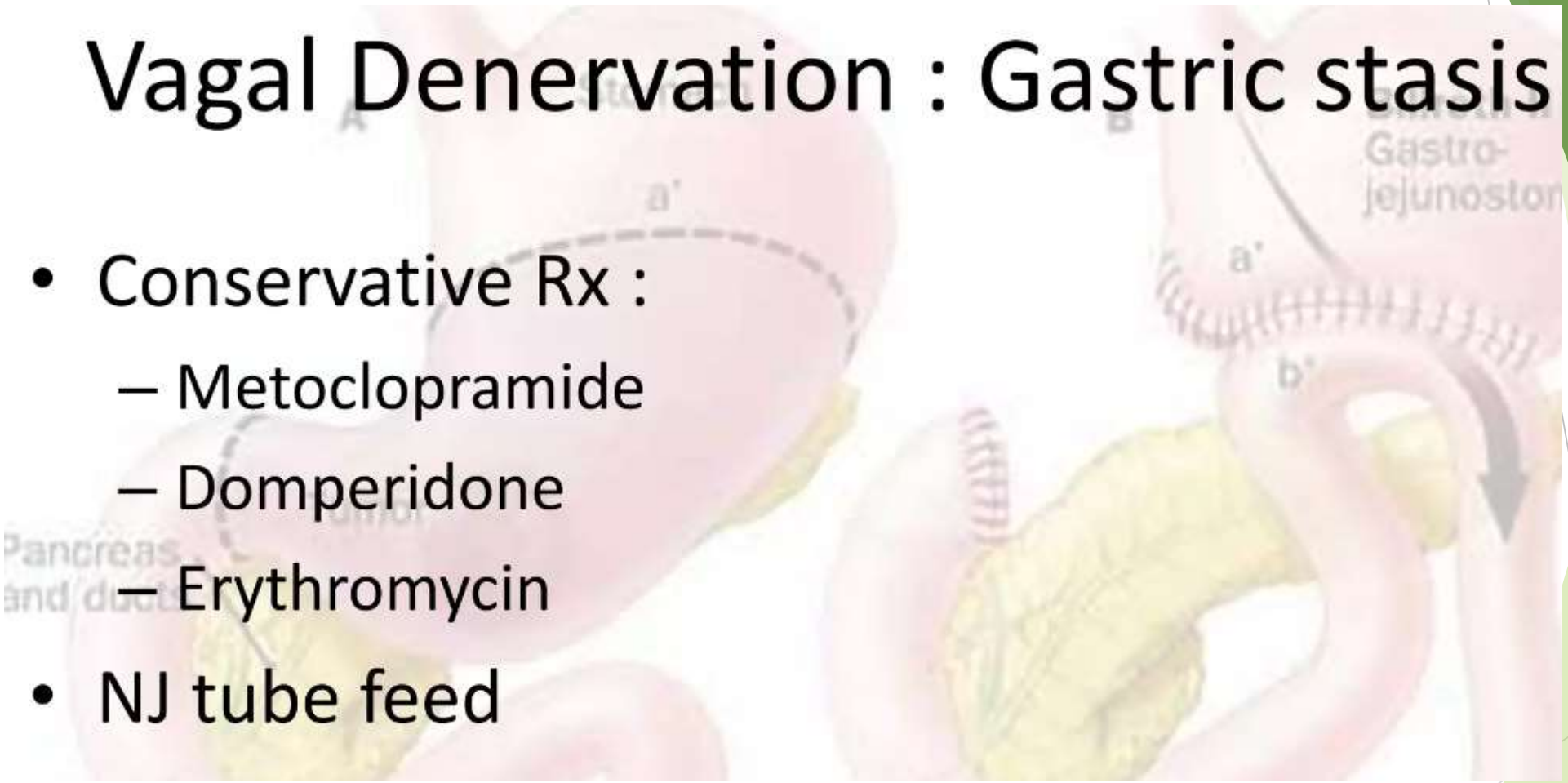
Conservative Rx :

- 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

- 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
- Gastric emptying studies – normal: 60% solid, 80% liquid clearance at 60 min.
  - Nuclear medicine or thin barium vs gastrografin

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

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

Abberation in Reconstruction

- Bile reflux gastritis
- Afferent and efferent loop obstruction
- Jejunogastric intussusception
- Roux syndrome

Bile Reflux Gastritis

- Most patient no symptoms
- Reflux symptoms: epigastric pain, N/bilious vomiting
- Dx by clinical + evidence of bile reflux (scope or scan)
- Scope :
  - mucosal erythema that involve parastomal region
  - bile staining or pooling
  - observed enterogastric reflux

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

- EGD – classic beefy red, edematous gastric mucosa
  - Sharp demarcation of mucosa at anastomosis
- 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

Conservative

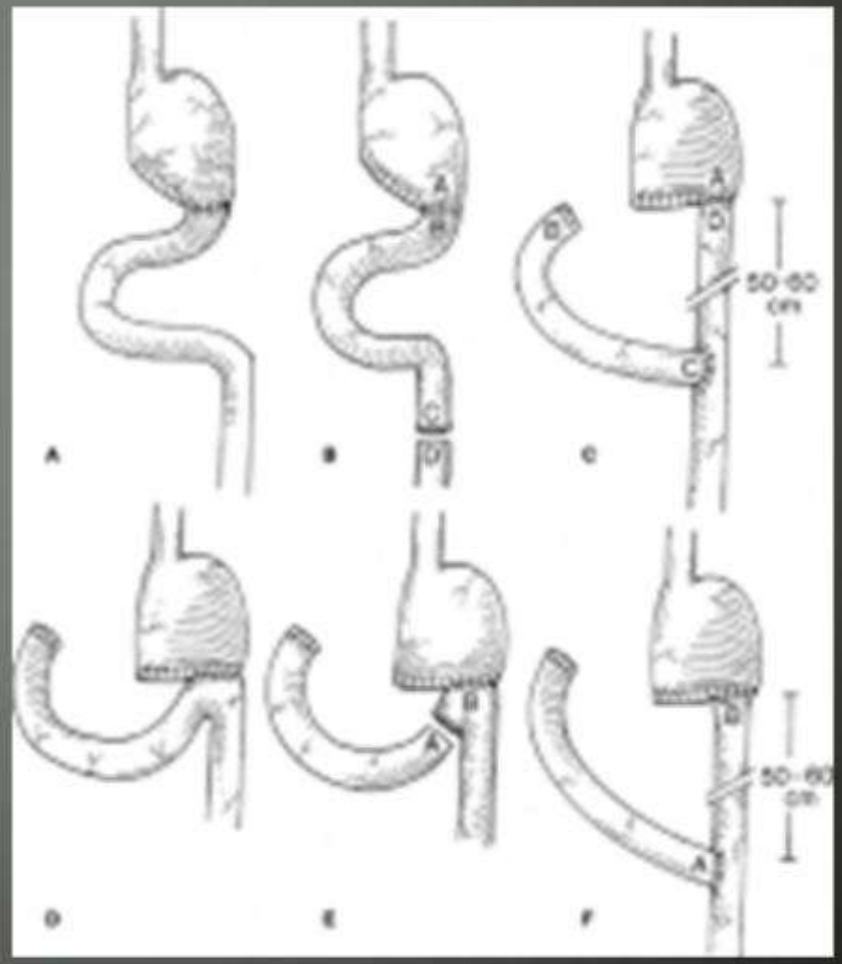
- Doesn't work as well
- High fat/protein diet
- PPI, carafate, reglan
- Cholestyramine – binds bile
- Most promising – Ursodeoxycholic acid
  - Resolution of symptoms

Surgical

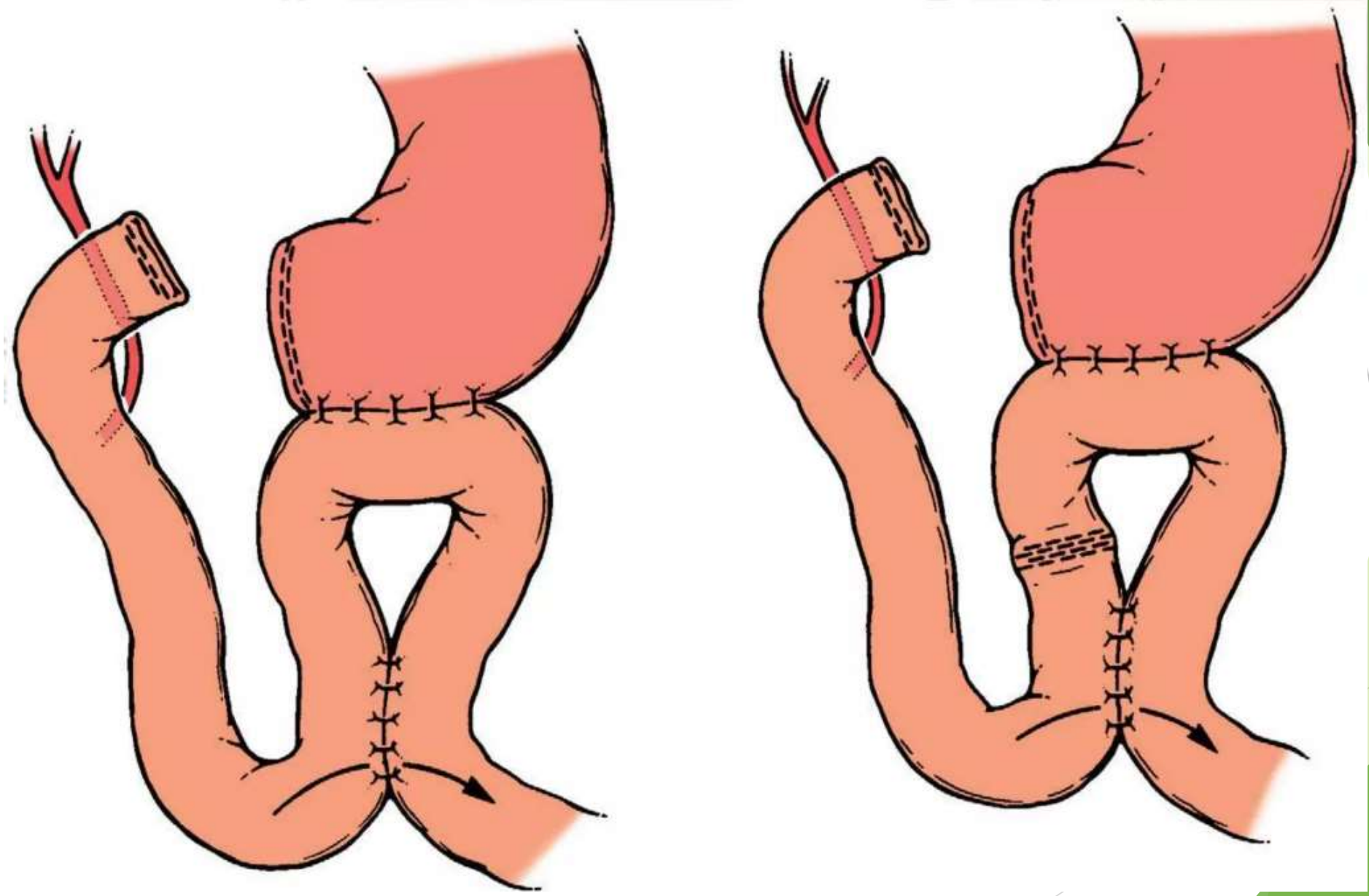
- Braun procedure
- Distal enteroenterostomy
- Henley loop
- Roux-en-Y reconstruction

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

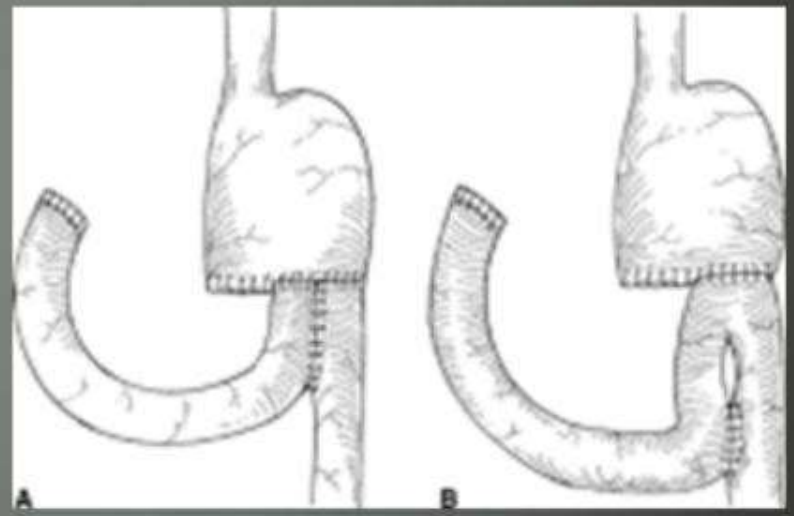


Bile Reflux Gastritis



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



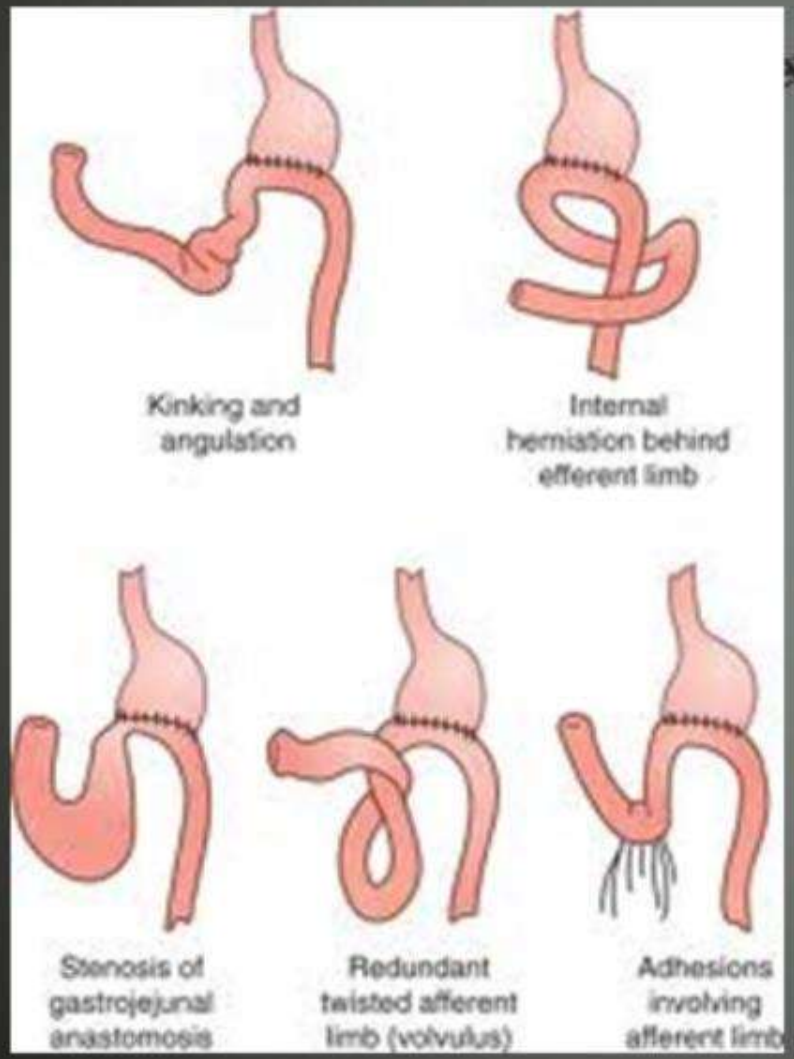
Afferent and Efferent Limb Syndromes

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

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
  - Dramatic clinical relief after vomiting
  - Some can be presented with diarrhea

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

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

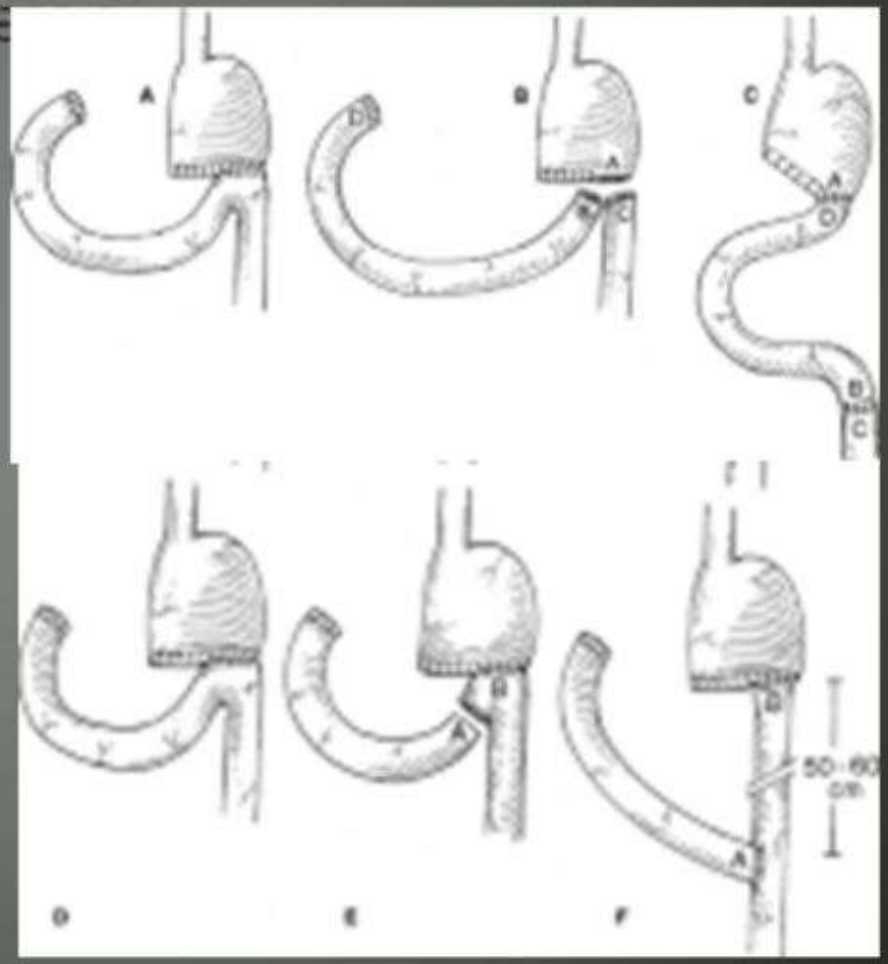
- Pain unrelated to meals
- Vomitus: Bile and food, no change in pain, nonprojectile
- Occult bleeding common
- Cause: enterogastric reflux

Treatment

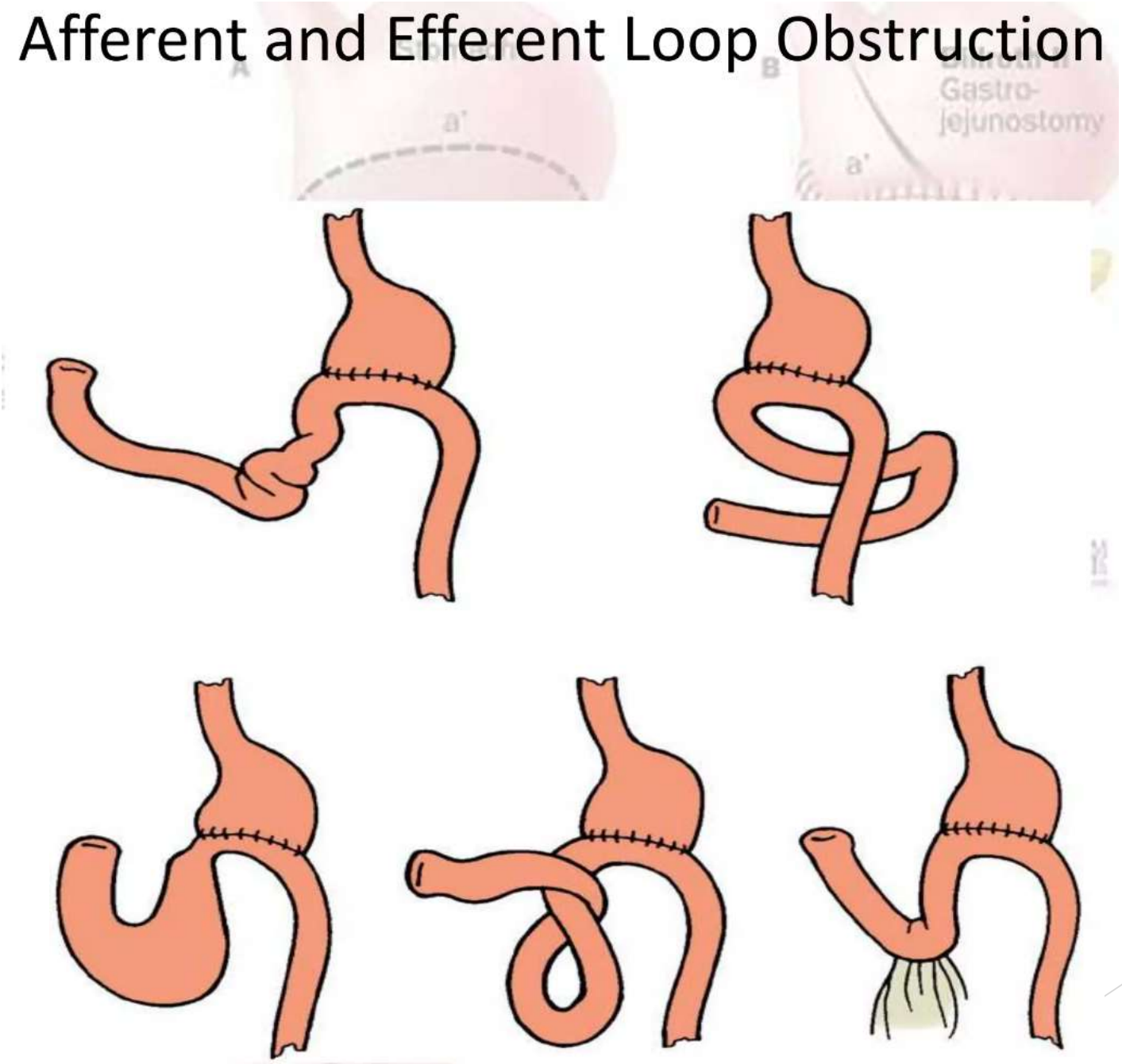
Surgical

Two most accepted

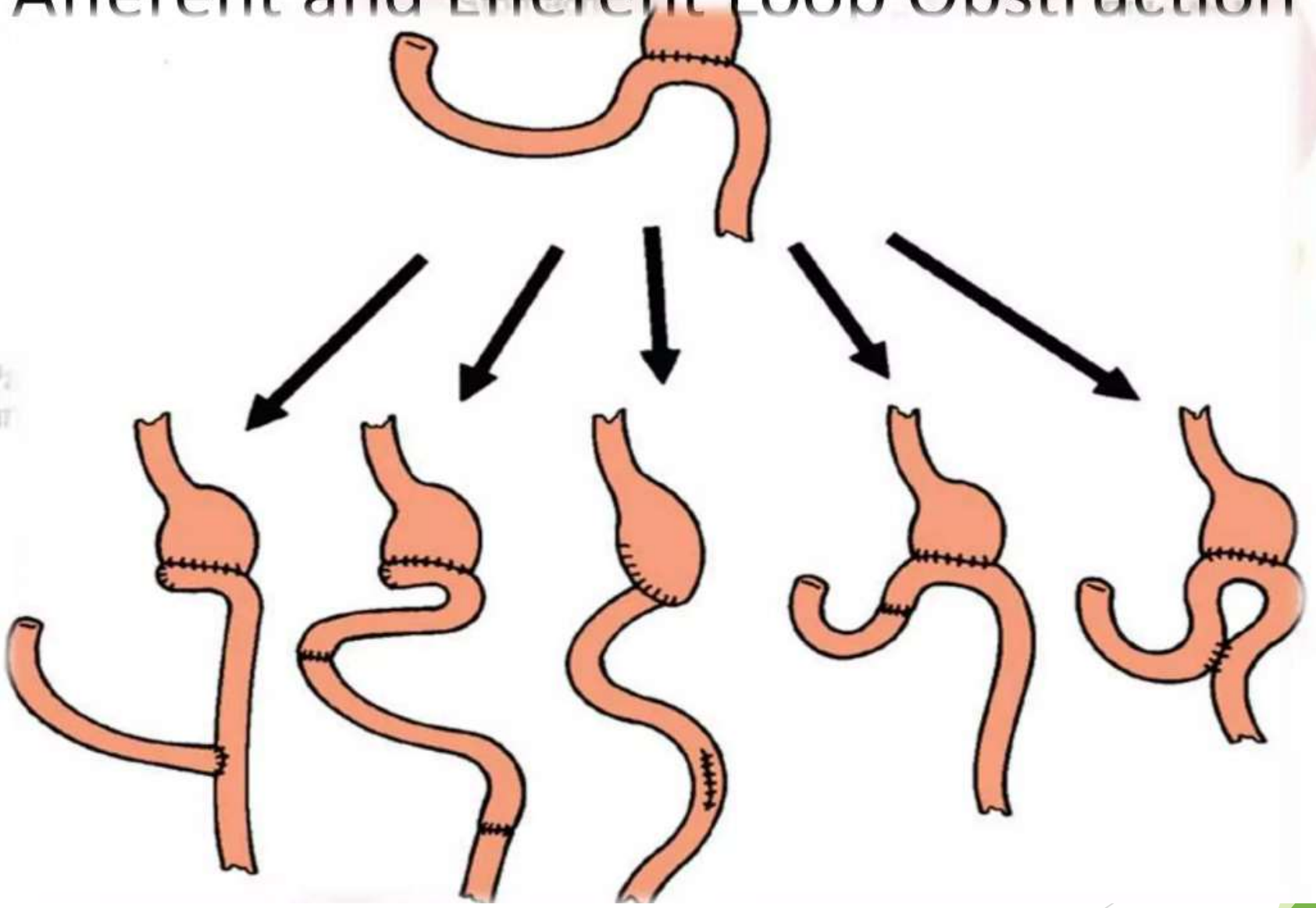
- Convert BII to BI
  - Can be difficult to get to duodenal stump
- Roux-en-Y – important to remember to perform complete vagotomy
  - Prevents marginal ulcers

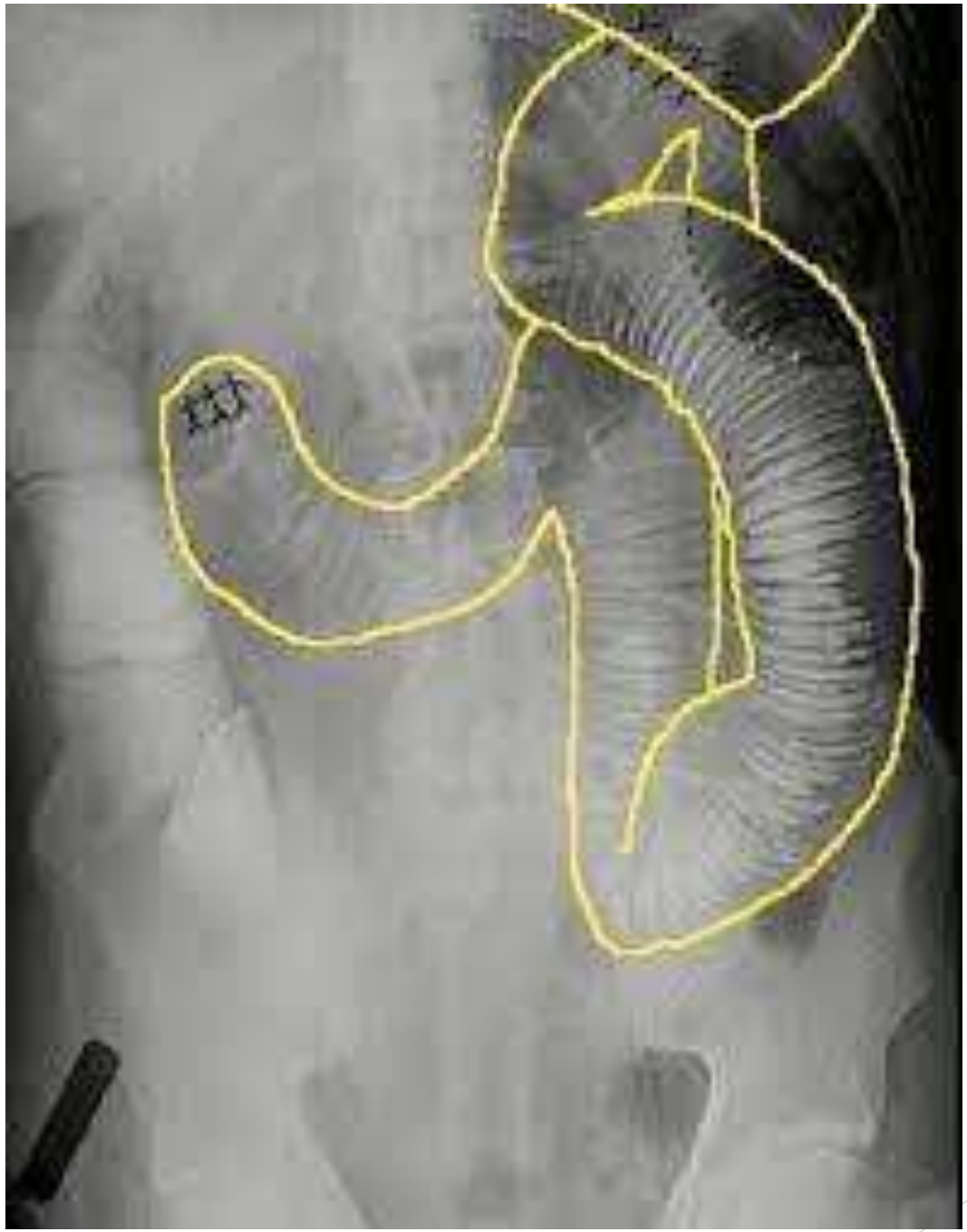


Afferent and Efferent Loop Obstruction



Afferent and Efferent Loop Obstruction





Efferent Limb Syndrome

- 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 : Promotality agents VS Sx

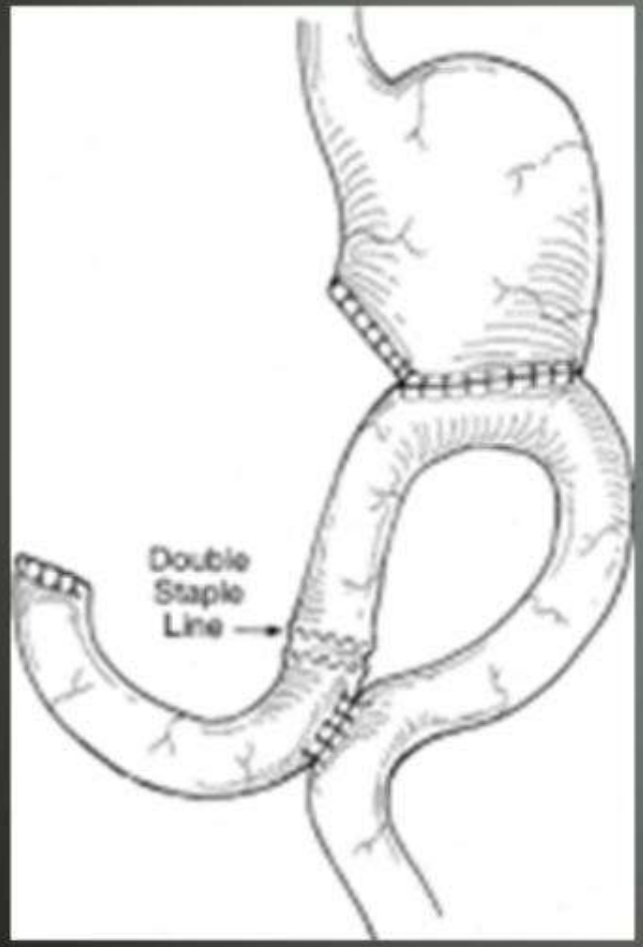
Roux Limb Syndrome

- 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

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 treaments.

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 carbohy - drates) into small bowel.

Leads to net fluid retention due to the osmotic gradient + releases one or more vasoactive hormones, eg. serotonin and VIP

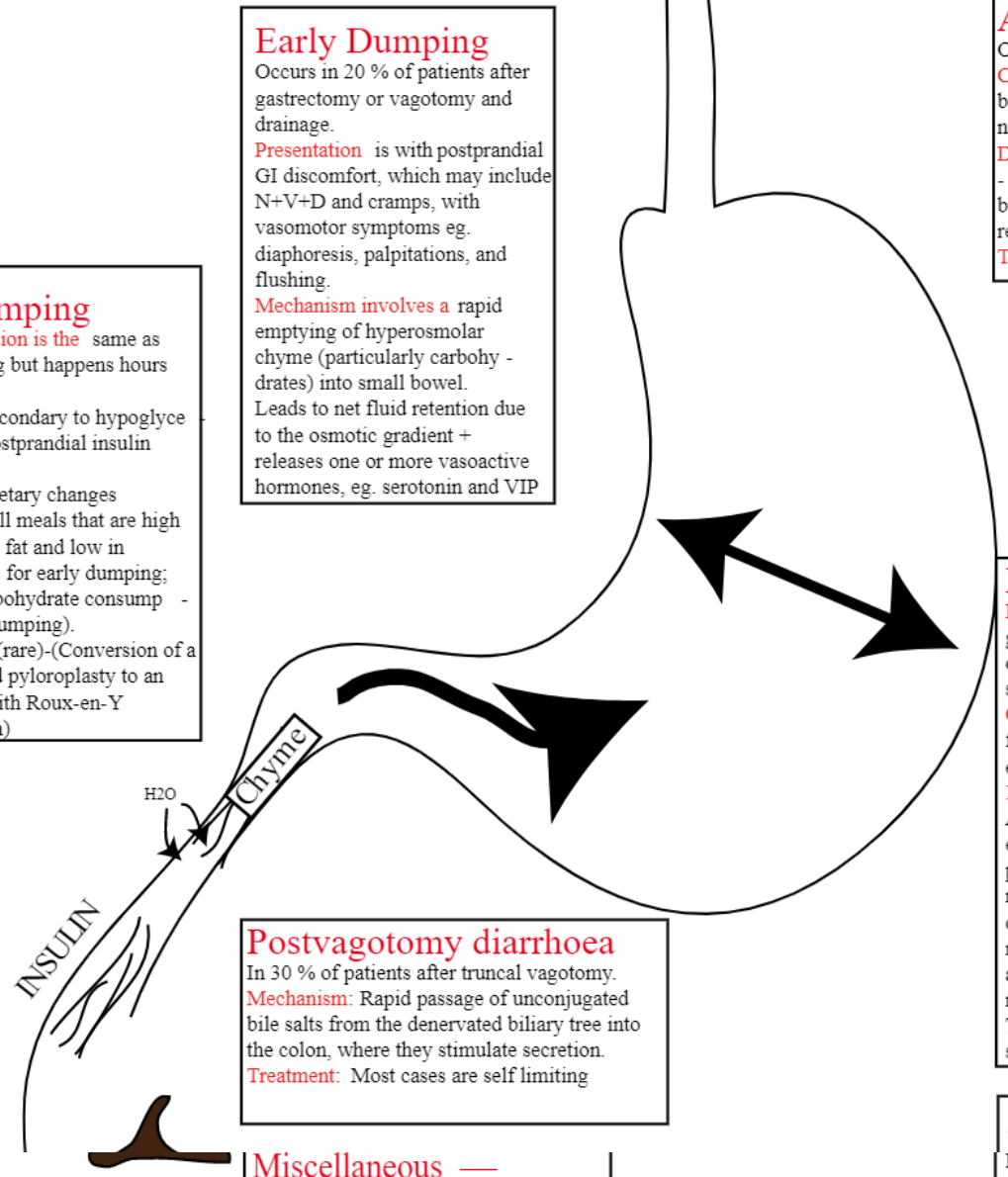
Late dumping

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

Mechanism:Secondary to hypoglyce mia 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 consump - tion for late dumping).

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



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 metoclopra - mide 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

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

Afferent and efferent

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

Miscellaneous —

Weight Loss and Dietary Resriction (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%)

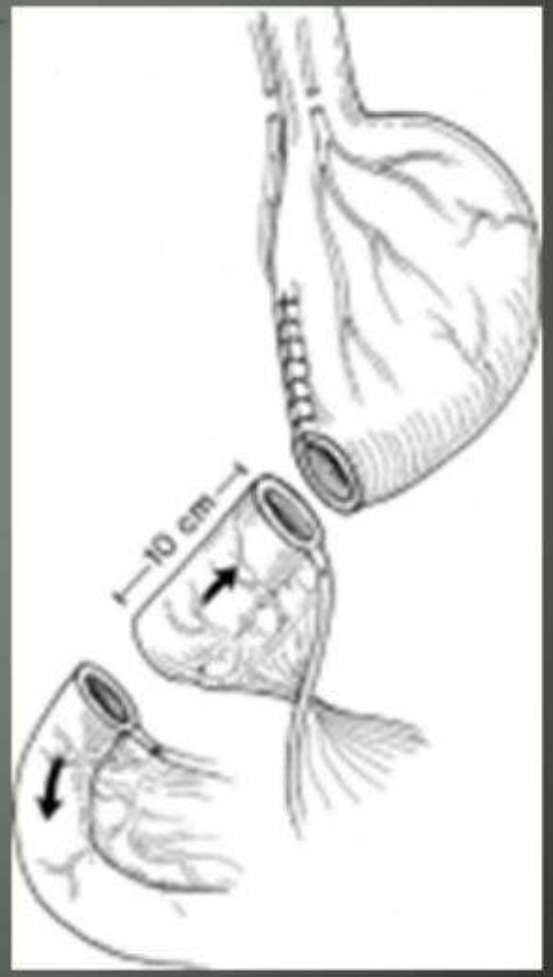
Metabolic bone disease

TREATMENT SUMMARY

Dumping syndrome	• Interposition of jejunal loop: Iso/ anti-peristaltic. • Conversion of loop gastrojejunostomy to RouxenY reconstruction (57,58).
Post vagotomy diarrhoea	• Anti-peristaltic jejunal loop distal interposition (59).
Gastric stasis	• Gastric pacing/Electric pacemaker (60). • Venting gastrostomy. • Re-operative near total gastrectomy.
Bile reflux gastritis	• Conversion to a Roux-en-Y reconstruction.
Afferent loop syndrome and Efferent loop syndrome	• Endoscopic insertion of double pigtail stent (61). • Conversion to RouxenY. • Conversion to Billroth I. • Braun entero-enterostomy.
Jejuno-gastric intussusception	• Revision of the GJ/Resection of the intussuscepted segment. • Revision of the GJ/Resection of the Conversion to a Rouxen Y.
Roux stasis syndrome	• 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%),
- Lymphoma (4%),
- Malignant GIST (1%)

Rare Primary

- Carcinoid, Angiosarcoma, Carcinosarcoma, and Squamous cell carcinoma

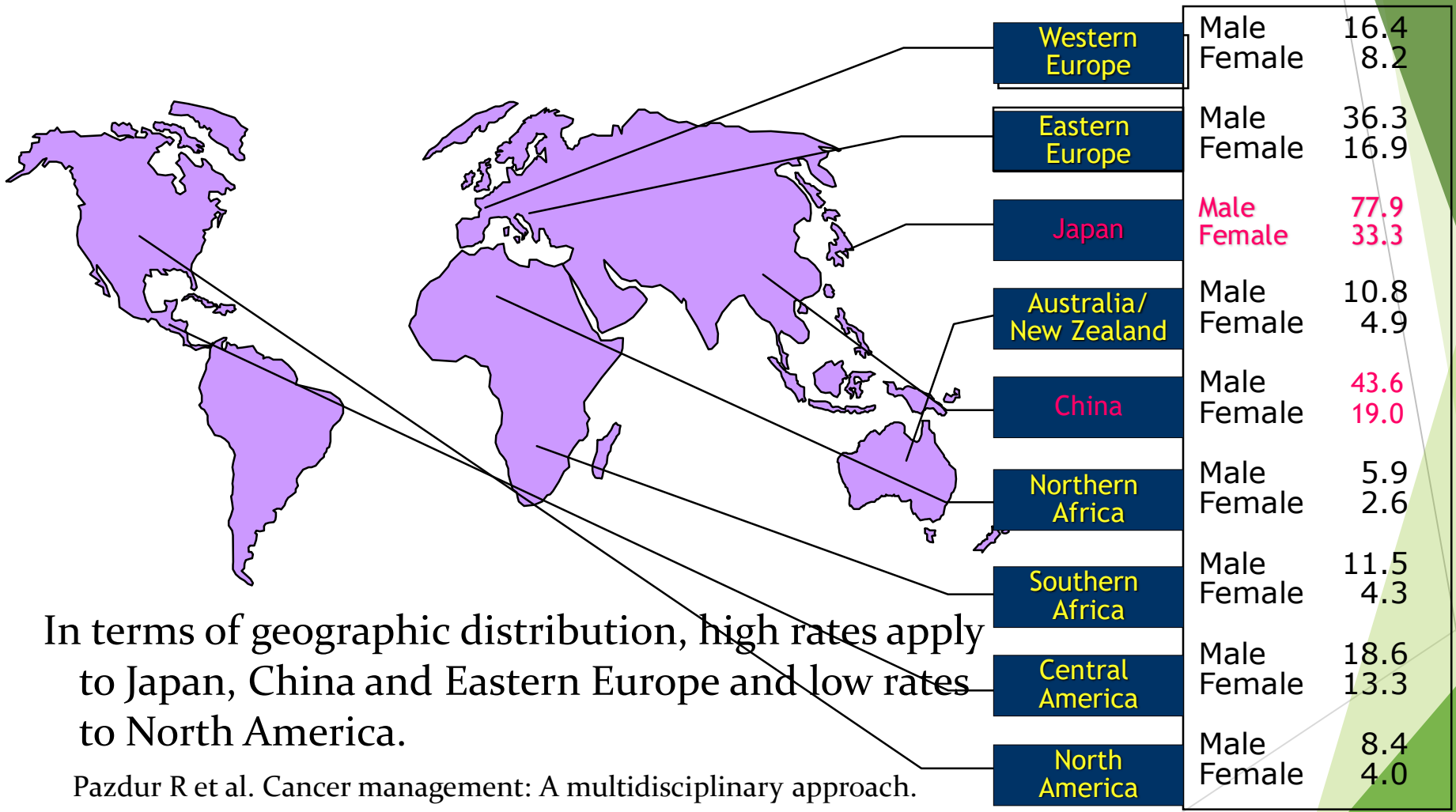
Secondary From :

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

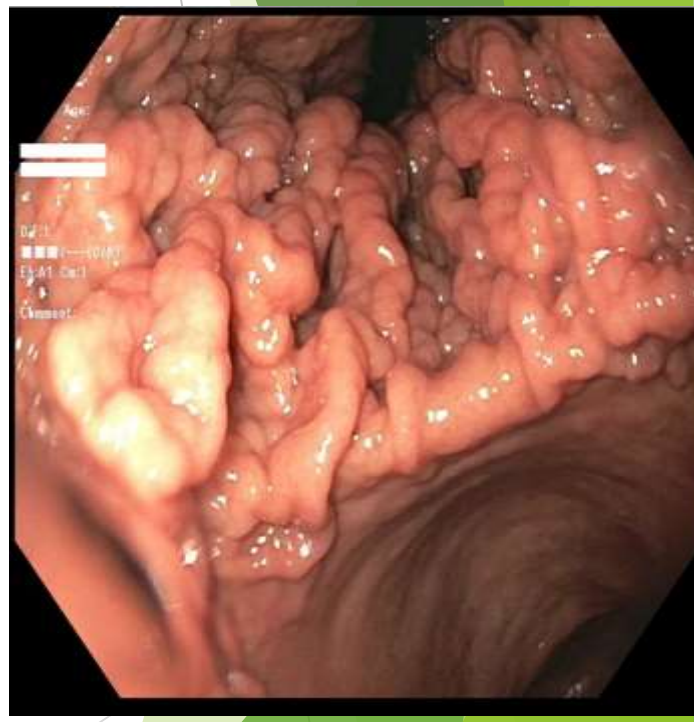
Risk factors :

- **Smoking**
- Alcohol is **NOT** a risk factor
- **Hyperplastic polyps** : less risk factor than in colon ca (villous, >1 cm)
- **Chronic atrophic gastritis** (need *H.pylori* eradication
- **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
- 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 :**
- 3% of gastric CA are inherited, most are sporadic.
- P53 (Li-fraumini).
- HNPCC (Lynch), MUTYH polyposis
- Familial adenomatous polyposis FAP./ APC mutation
- P-TEN (Cowden) hamartomas.
- BRCA-2
- Peutz-Jegher Syndrome.
- **CDH-1 mutation :** AD , 70% risk for hereditary diffuse gastric CA
- → 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)

Pathology :

- Arises from the mucus producing cells, not the acid producing ones.

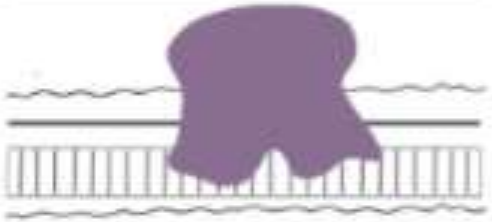
- Various classifications schemes for gastric CA.

- Anatomical : (according to the site):

- Proximal (cardia) → might be treated as pure gastric or considered distal esophageal (siewert)
- Distal (non cardia) – more with H.pylori infection.
- Diffuse (9%) , linitis plastica

2- Bormann's Classification

- Classification on gross appearance of the lesion

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

- 3- Histopatholigical (**lauren** classification) → the most adapted classification
- **Intestinal type :**
- - more in elderly, males.
  - usually well to moderately differentiated.
  - associated with metaplasia / chronic / atrophic gastritis.
  - More distal and localized than proximal
  - more tendency to spread through lymphatics and hematogenously.
  - liver is the m.c site of mets.
  - Better prognosis
- **Diffuse type :**
- - more in younger , females
- - usually poorly differentiated + signet ring cells
- - 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 :

- Asymptomatic, discovered incidentally
- Early gastric CA :

- No obvious signs and symptoms.
- 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)

- Dysphagia also 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)

- 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).

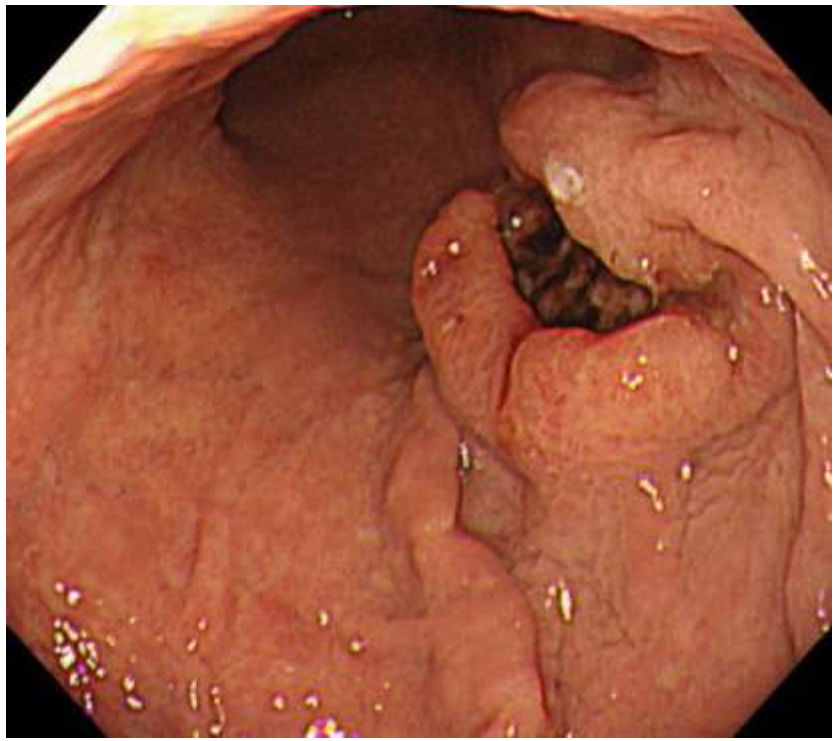
- Elective vs. Emergency presentation (G.O.O , perforating malignant gastric ulcer, upper GI bleeding)

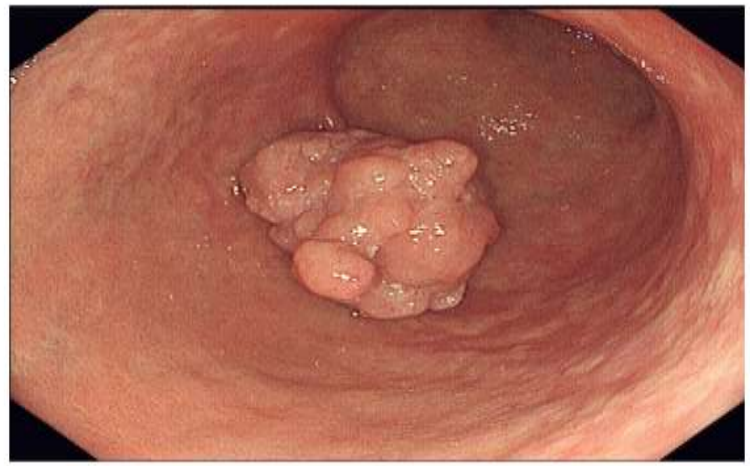
Diagnosis / Assessment / Staging

- 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)
- CEA :
  - 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 :**

- 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

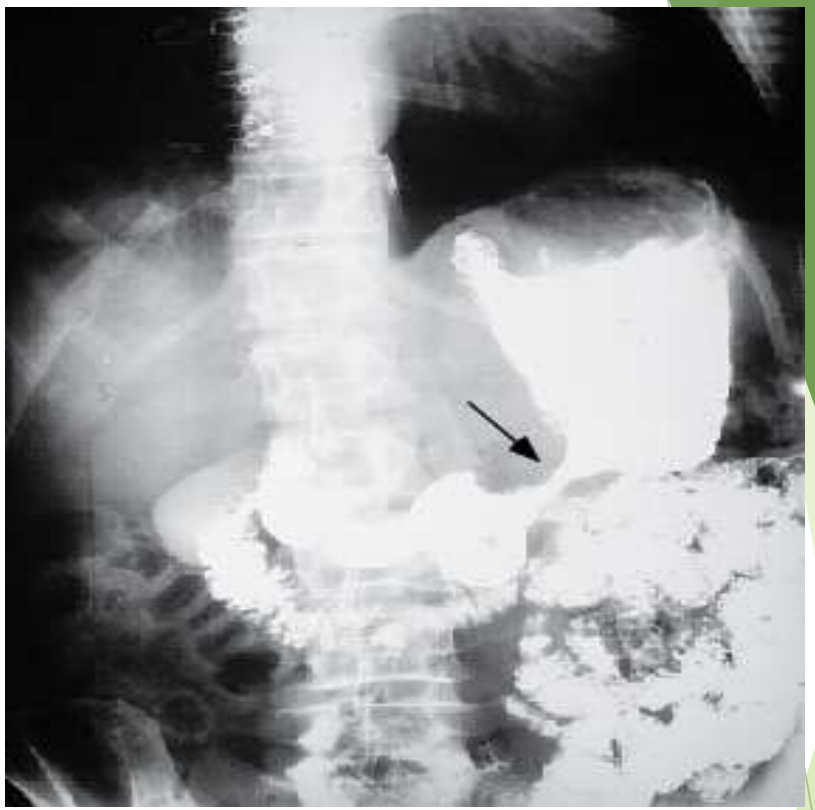




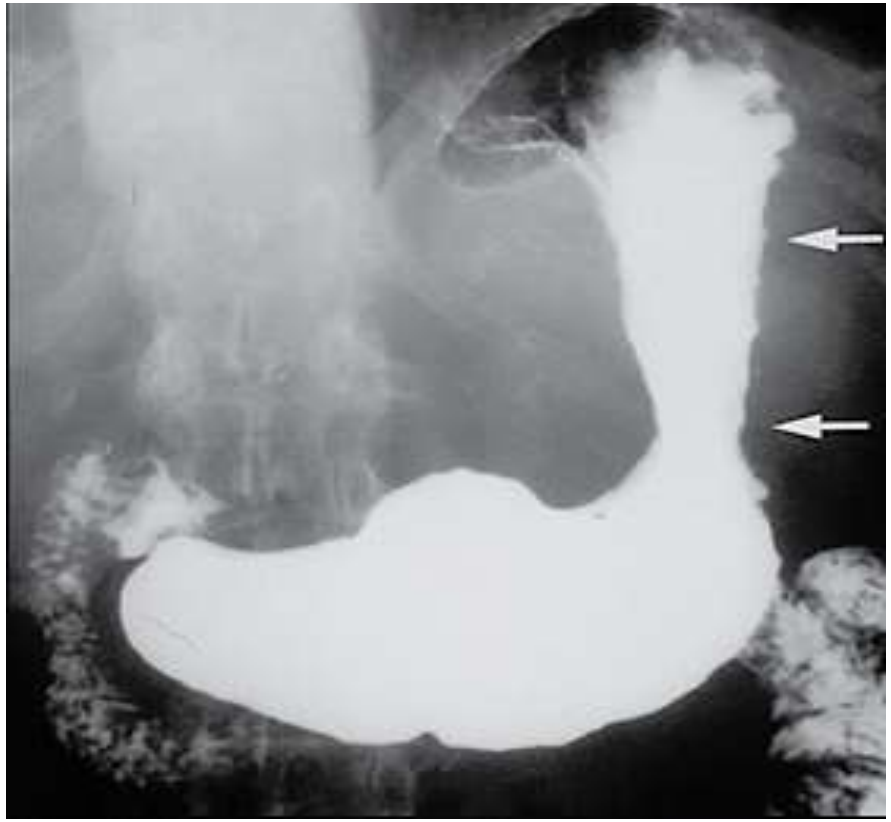
Barium swallow

75% accuracy

for obstructive lesions only

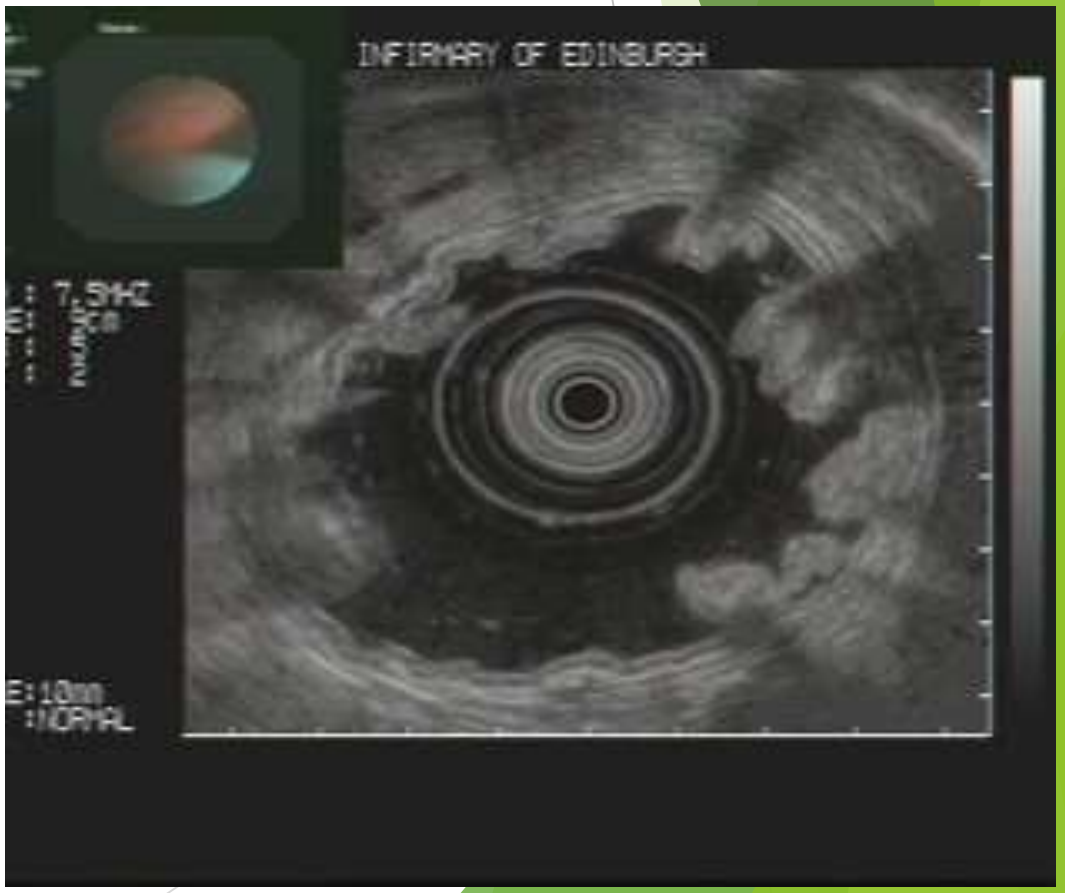


Apple core sign”



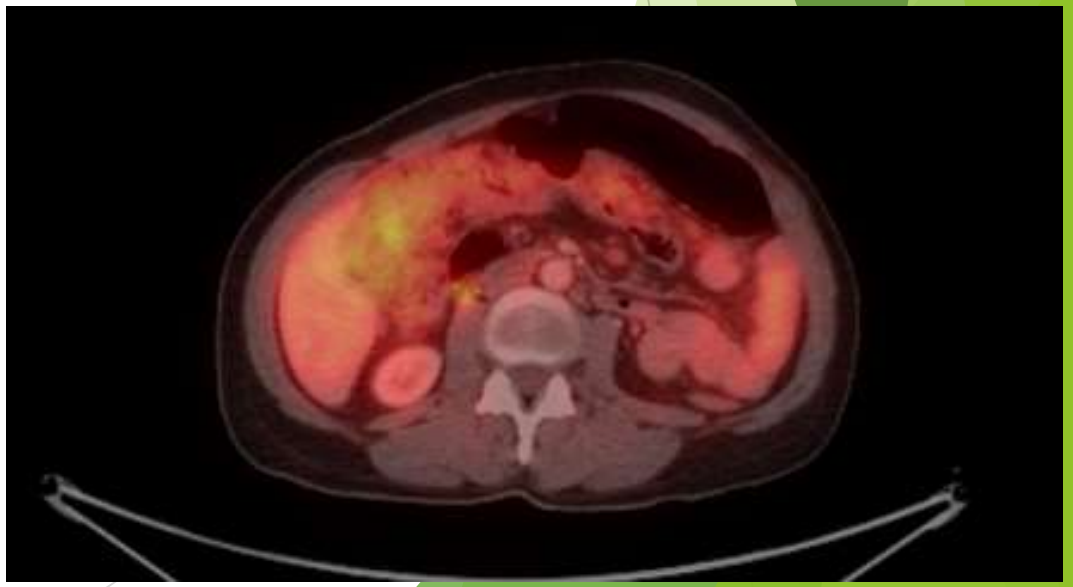
Linitis plastica "leather-flask" appearing stomach

- **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
- for locally advanced gastric CA.



- 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 :
- 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.



- **Staging Laparoscopy :**
- 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, mesentry, 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)

- 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.
- T1 : tumor invading submucosa/**lamina propria.**
- T2 : tumor invading **muscularis** propria.
- T3 : tumor invading subserosa without visceral peritoneium.
- T4 : tumor invading serosa (visceral peritoneium) or adjacent organs.

- (N) :

- 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)
- Endoscopic resection (very early disease, selected patients)
- Surgical resection (mainstay of treatment, the only curative one)

Endoscopic Mucosal Resection (EMR)

- 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 :

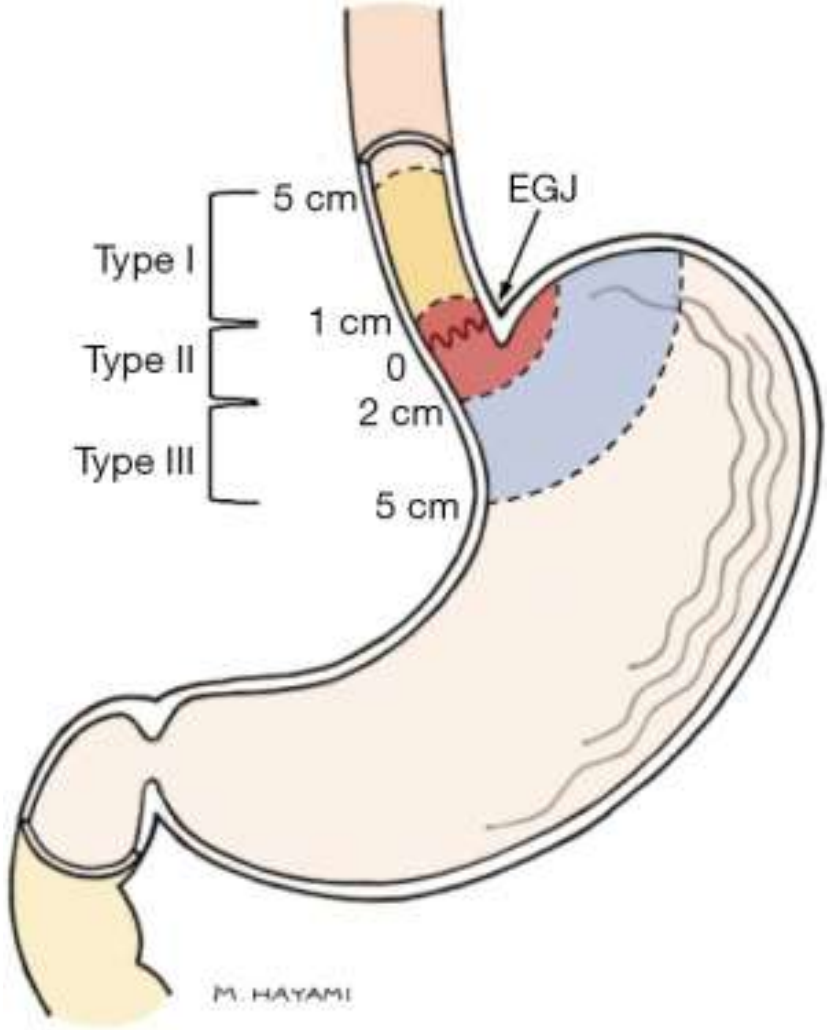
- performed only in specialized high volume centers with good experience (Japan)
- Need closer surveillance / followup

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 antraum) 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
- Type 3 (2-5 cm into the stomach are gastric CA)
- All proximal pure gastric CA should undergo total gastrectomy (not partial)

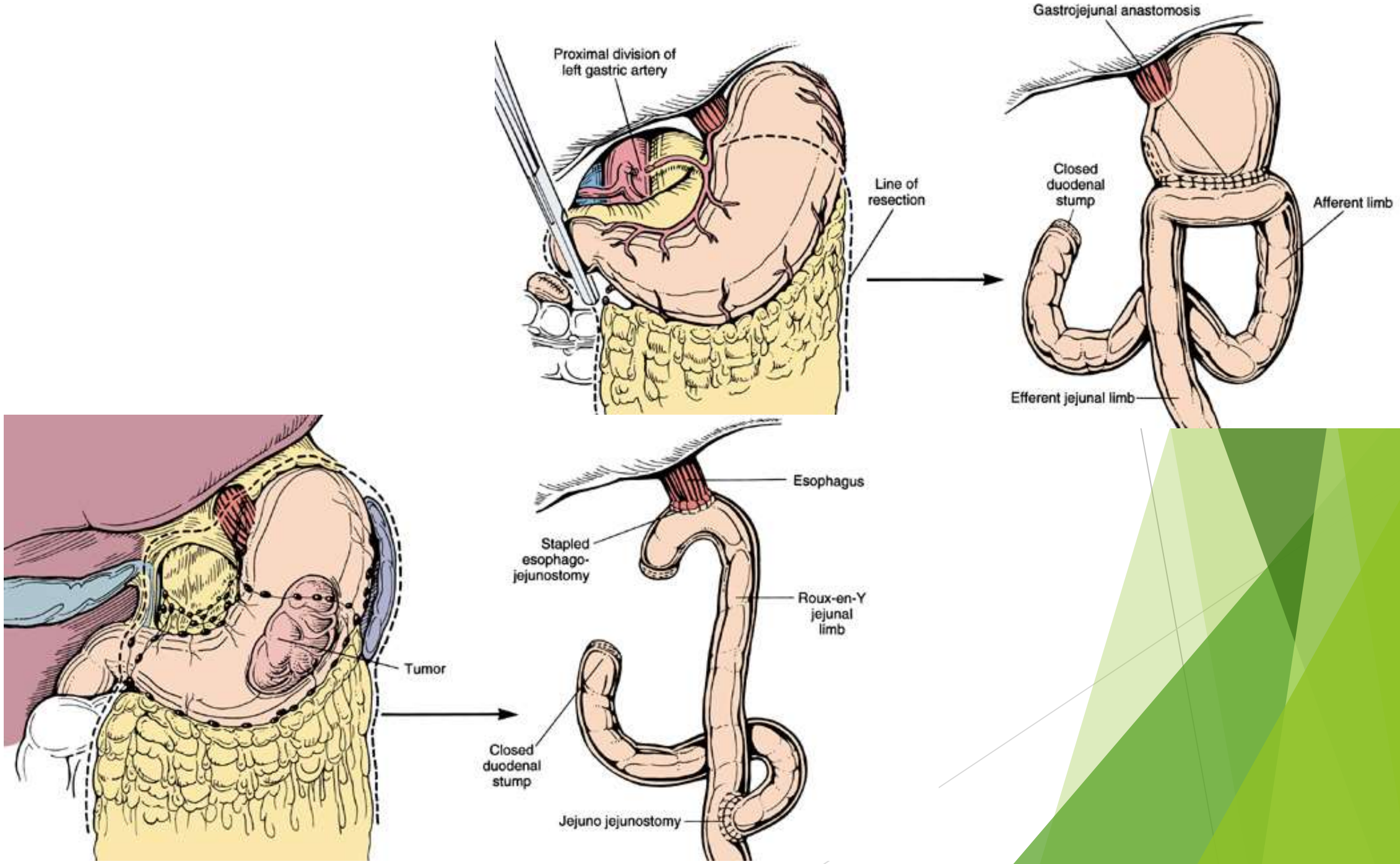


- Total gastrectomy : (for proximal and midbody tumors)
- Removal of the whole stomach and reconstruction with **ROUX en-Y** configuration –
- Partial (distal/subtotal) gastrectomy: (for antral/distal tumors)
- Removing part of the stomach with remaining proximal gastric pouch (supplied by short gastric vessels).

  reconstruction can be done with **Billroth II** OR with **ROUX en-Y** configuration.

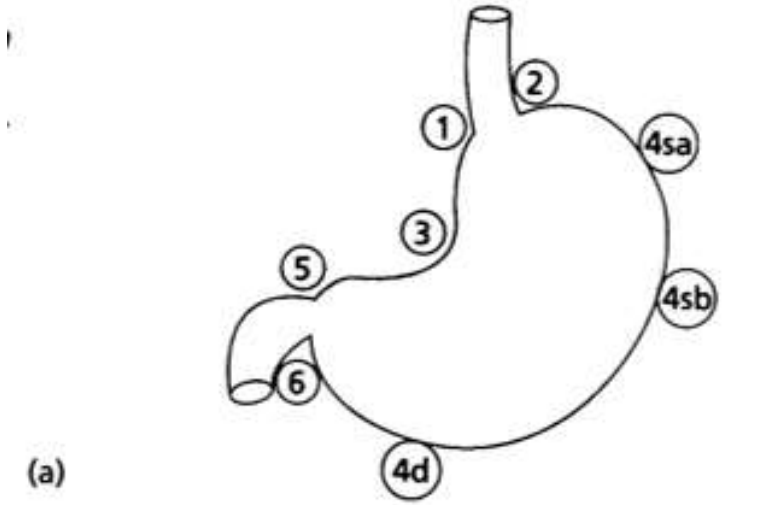
- **open** vs. **laparoscopic**
- Both offer same survival/outcome/completeness of resection.
- Lap. Surgery has lower post op pain, faster recovery and earlier return to daily activities. But require a more skilled surgeon.

- Feeding jejunostomy insertion at surgery for all patients.


Proximal division of left gastric artery
Line of resection
Gastrojejunal anastomosis
Closed duodenal stump
Afferent limb
Efferent jejunal limb
Tumor
Esophagus
Stapled esophago-jejunostomy
Roux-en-Y jejunal limb
Closed duodenal stump
Jejuno jejunostomy

- **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 lymphadenecomy.**
- 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)
- D3 (D2++ paraortic,retropancreatic) → higher morbidity, no added survival benefits
- D3 only performed in Japan

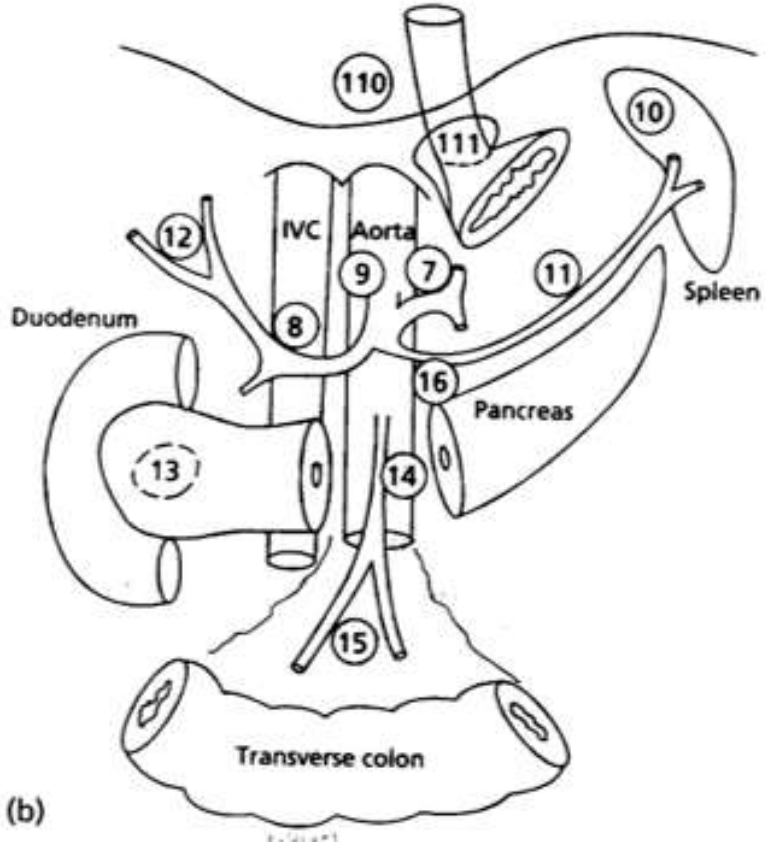
- Japanese literature shows survival benefit of extended D2 dissection:
- 39% 5 year survival after D2 vs. 18% for D1
-
- Western literature shows no survival benefit but increased morbidity and mortality of D3 dissection



(a)

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



(b)

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

- **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

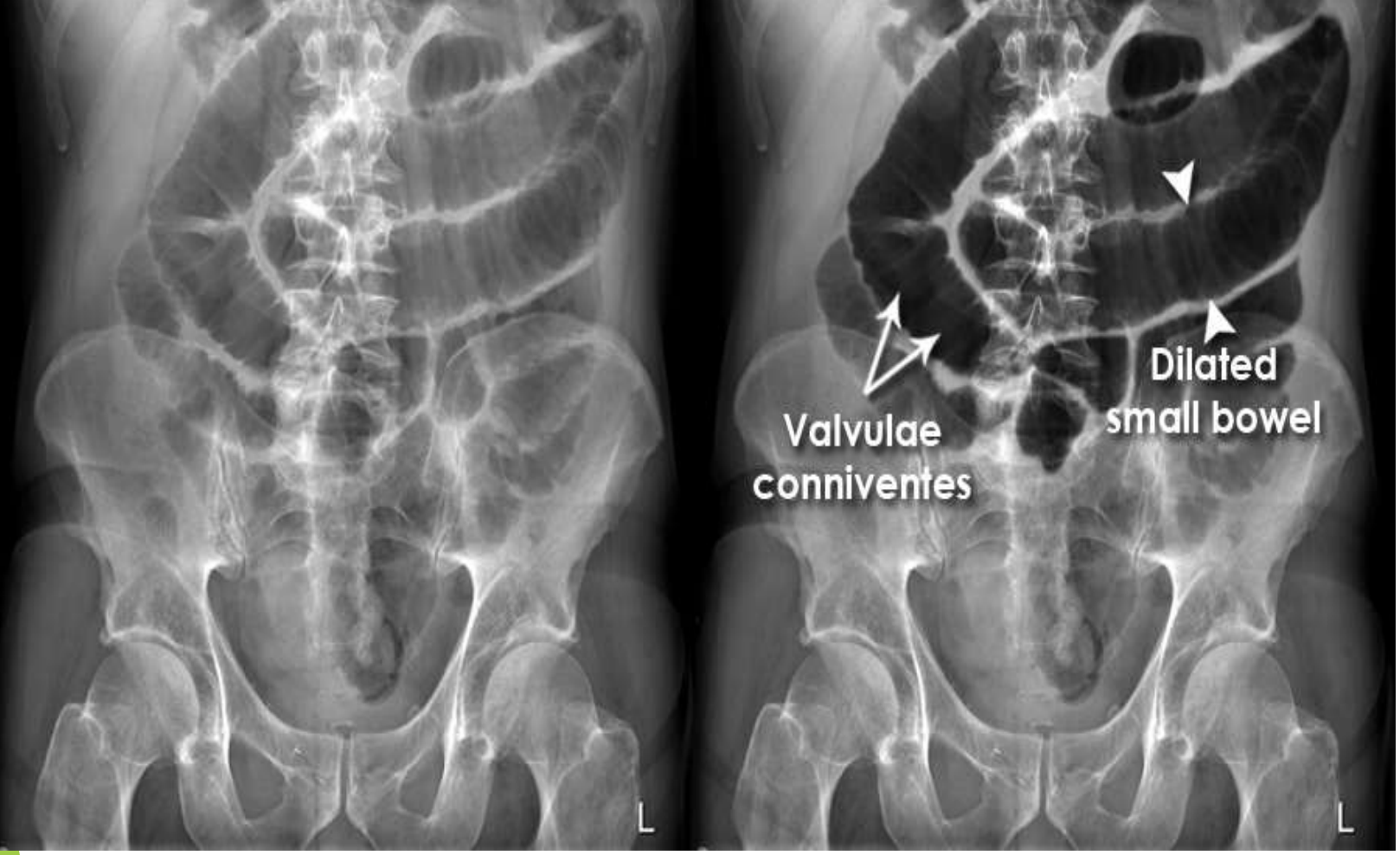
- 2-10%
- Can be early or late (up to 40 days post op)
- Leak in the first few days is usually a technical failure
- Dx : fever, leukocytosis, tachycardia, signs of peritonitis or food/juice in drains if free leak
- CT with oral contrast or GG swallow detects leak
- Some surgeons perform routine contrast imaging before initiation of oral feeding (post op day 5)

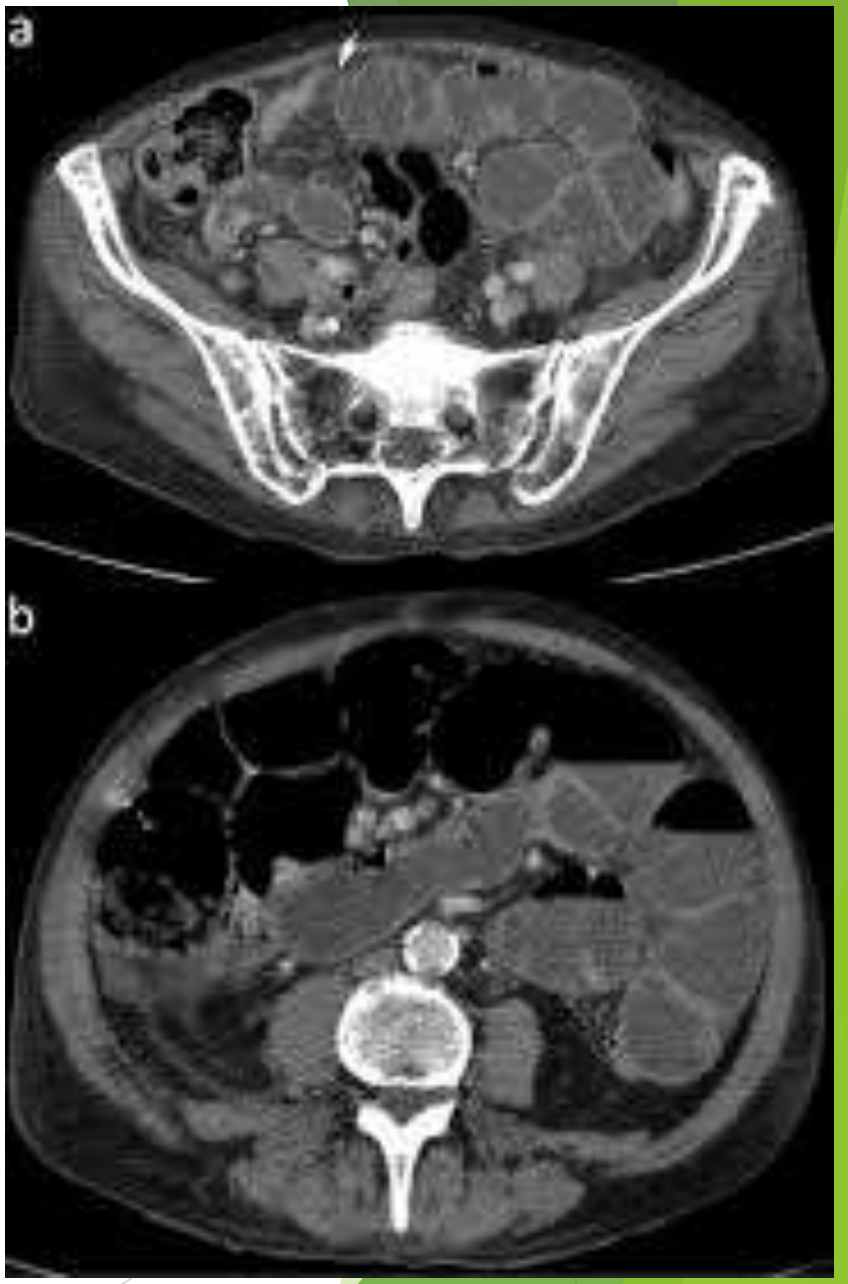
- Management :

- 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.
- Normal 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 : Keep NPO, treat the cause, early ambulation of patient.


Valvulae
conniventes
Dilated
small bowel
L
L


a
b

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.
- 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

- **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 :

- Palliative bypass (gastrojejunostomy)
- Gastrostomy tube for decompression
- Palliative resection not advised for most of patients

- **Survival :**
- Depends on stage, completeness of resection
- 5 yrs- survival :
- local disease 50-80%,
- +ve LN 30%,
- Metastatic 5%

- **Recurrence :**

- High even after complete resection (30%)
- Most occur in the first 2 years (80%)
- Can be local (surgery site) ,
- distant (liver) ,
- peritoneal mets
- Carry a dismal prognoses (6 months survival)
- Treated as palliative only (usually chemotherapy)

- **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 annualy

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 (88%), anorexia(47%), weight loss (25%), bleeding (19%), and vomiting (18%).
- Obstruction and perforation, Anaemia and epigastric mass.

Investigations

- Labs: CBC, LDH , B2-macroglobulin, bone marraow 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 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)

- Historically, these lesions were classified as leiomyomas or leiomyosarcoma
- <1 % of gastric CA
- 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 extraluminaly 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
- - The stomach is the most common site of GIST. Other includes small bowel, colon and rectum

GIST tumours

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

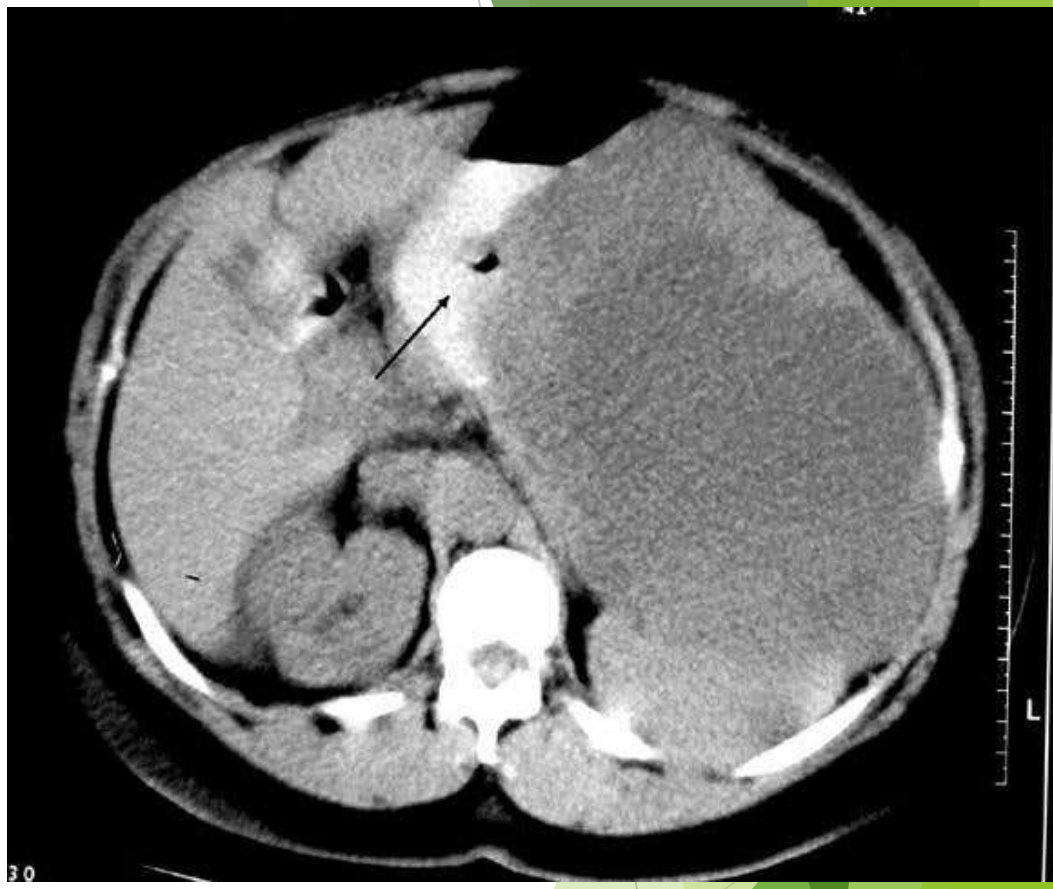
Investigations:

- Labs
- Barium swallow, u/s , and AXR gives limited informations
- 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.
- Biopsy is not always indicated if dx is suggestive and surgery Is needed anyway



GIST tumours

- Treatment
- 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 prognoses
- 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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