Stomach & Duodenom

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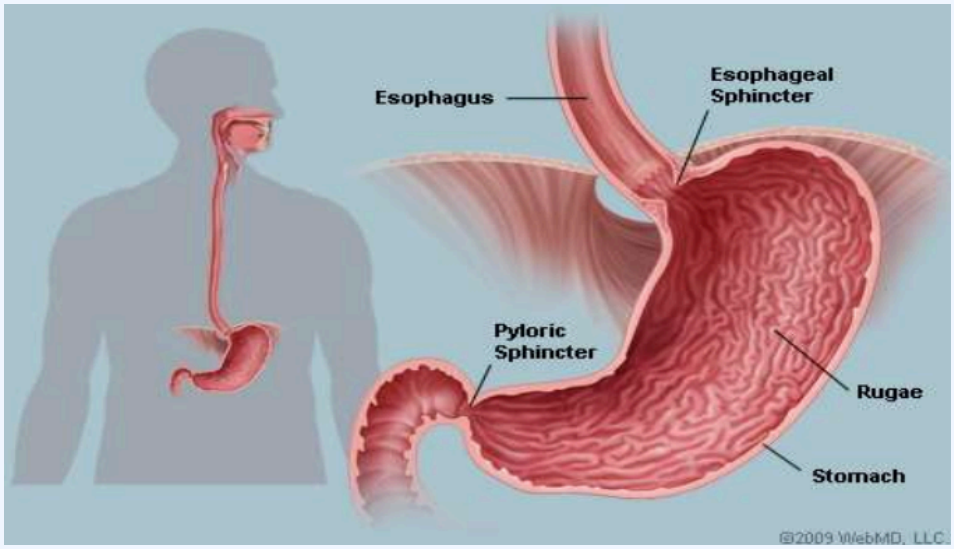
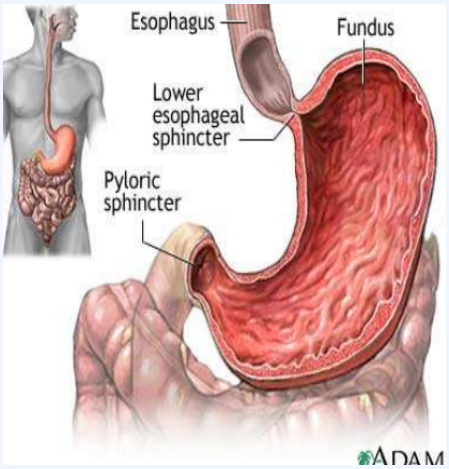
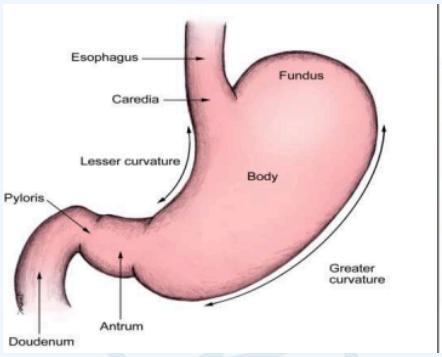
STOMACH ANATOMY

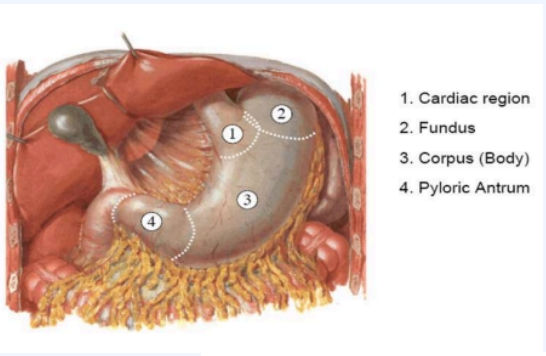


- **The stomach**
- **The stomach is a dilated part of the alimentary canal between the esophagus and the small intestine.** Fundus
- **It is a J-shaped muscular sac.**
- **located at level of T10 and L3 vertebral.**
- **Position of the stomach varies with body habitus**

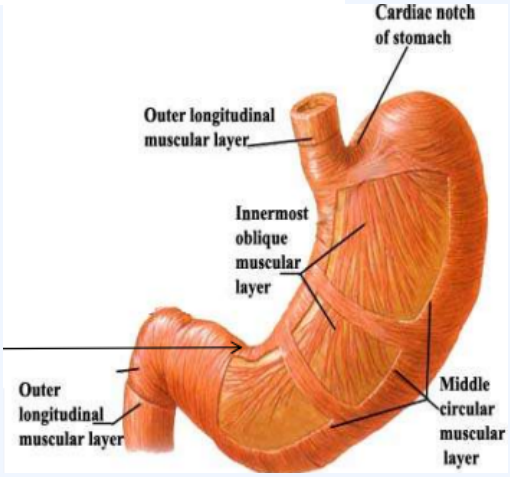


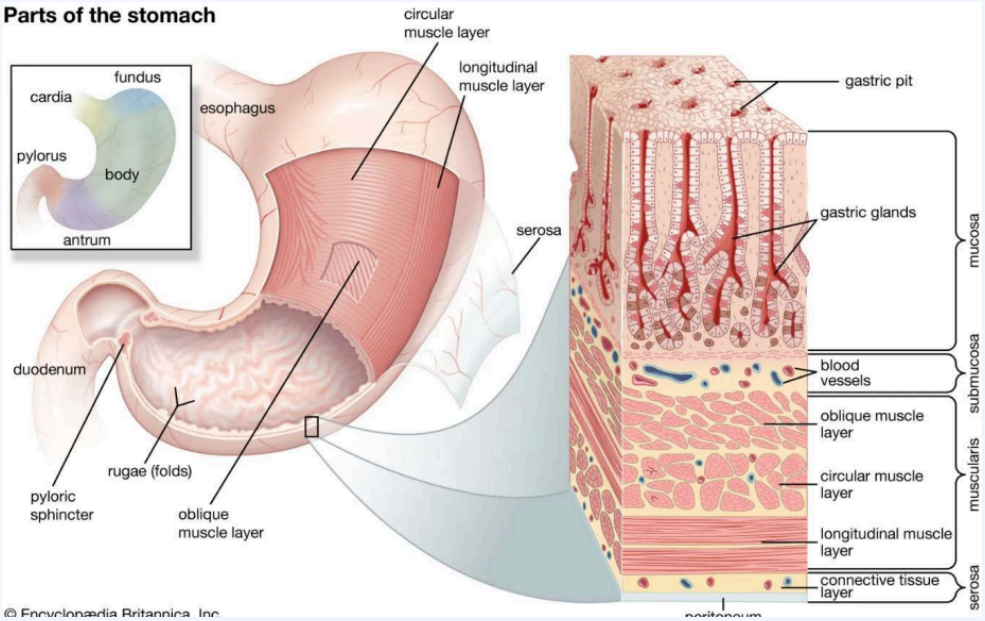
خزان يمنع وصول الاكل مباشرة للامعاء الامتصاص السريع للأغذية في حالة عدم وجود معدة يؤدي إلى زيادة كمية الأنسولين المفرزة بشكل كبيربالتالي هبوط نسبة السكر في الدم



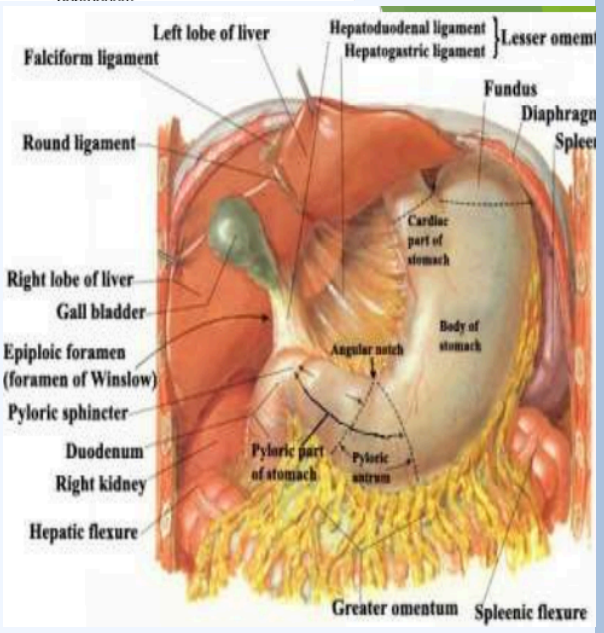


- **the cardial notch, which is the superior angle created when the esophagus enters the stomach. the angular notch, which is a bend on the lesser curvature.**

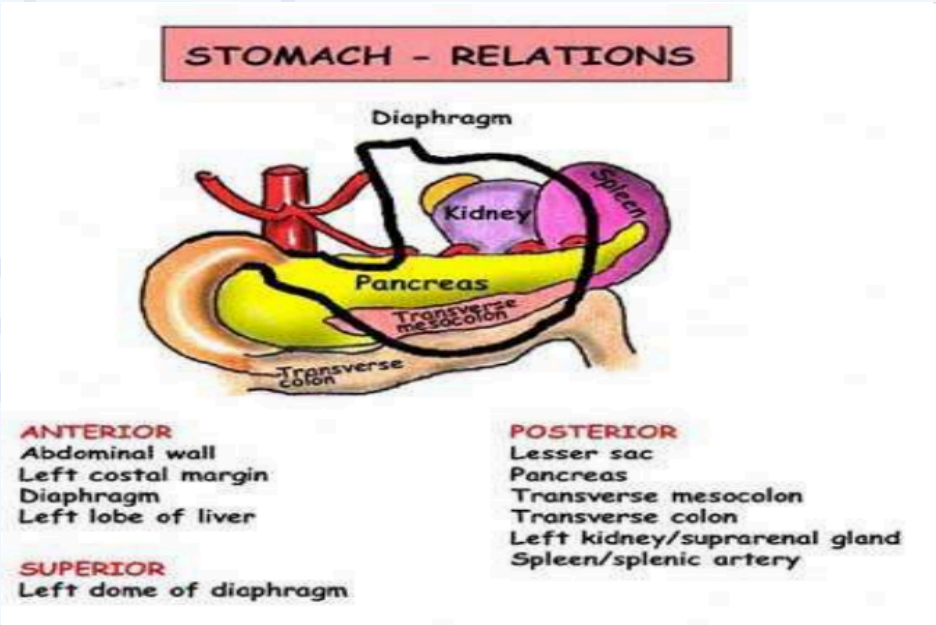




Layers of peritoneum attached to the stomach: ⊠ Lesser omentum: attaches the liver to the lesser curvature. ⊠ Greater omentum: attaches the greater curvature to the posterior body wall.



Stomach Anatomical Relation

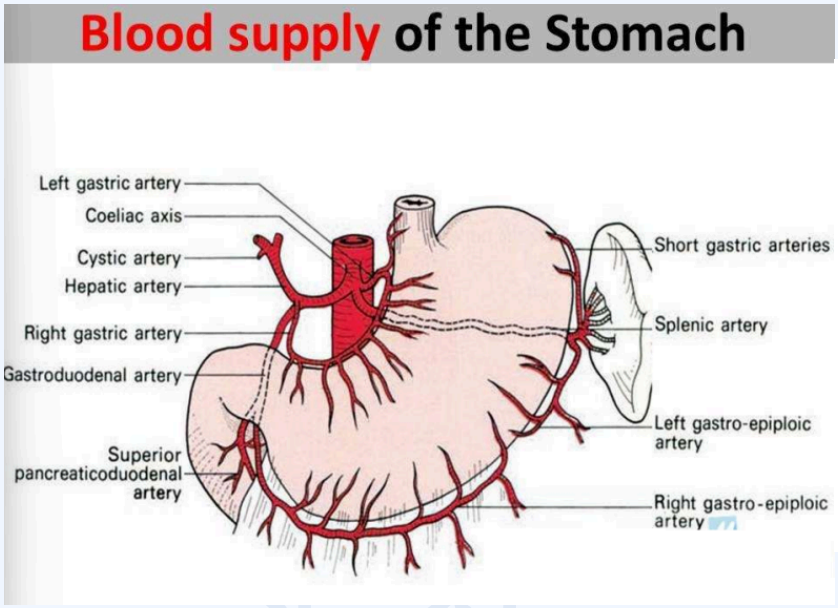


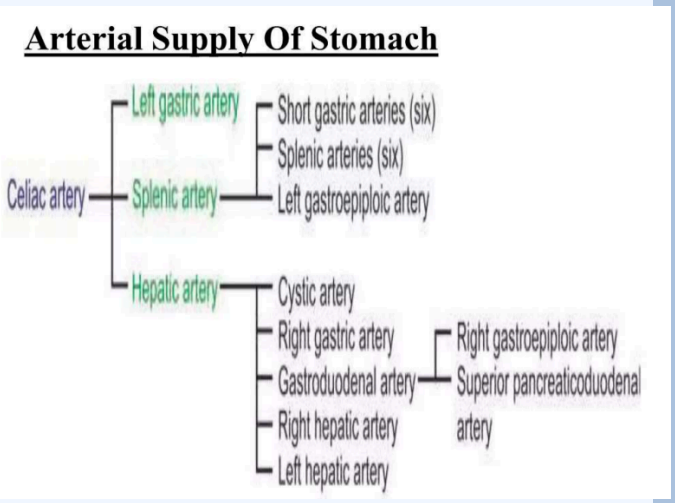
Arterial blood supply of stomach:

□ 3 Branches

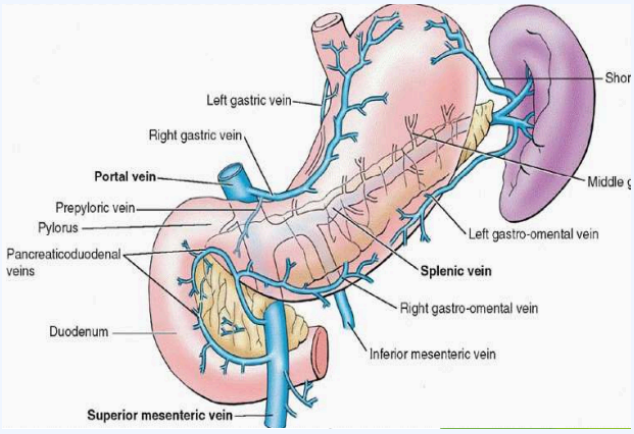
- **□ Left Gastric Artery □ Supplies the cardia of the stomach and distal esophagus**
- **□ Splenic Artery □ Gives rise to 2 branches which help supply the greater curvature of the stomach □ Left Gastroepiploic □ Short Gastric Arteries**
- **□ Common Hepatic or Proper Hepatic Artery □ 2 major branches □ 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**

Good B supply so Stomache resist ischemia





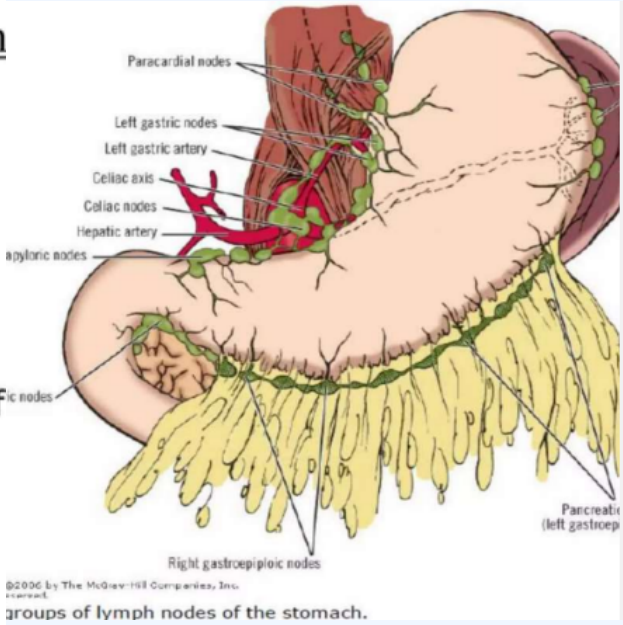
- **Stomach Venous Drainage**
- **Venous Drainage**
- **□Parallel to arterial supply**
- **□ Rt &Lt gastric veins drain to the portal**
- **□ Rt gastroepiploic drains to the SMV**
- **□ Lt gastroepiploic drains to the splenic**
- **□ Short gastric V. into splenic**



Stomach Lymphatic Drainage

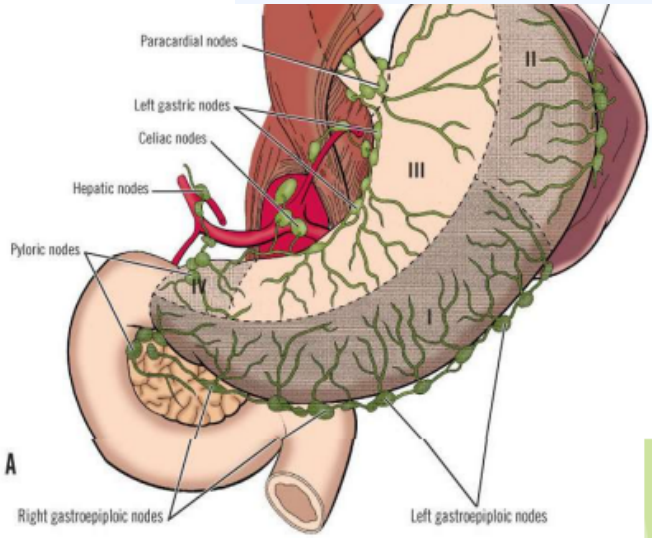
- Eight groups of lymph nodes of the stomach

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



groups of lymph nodes of the stomach.

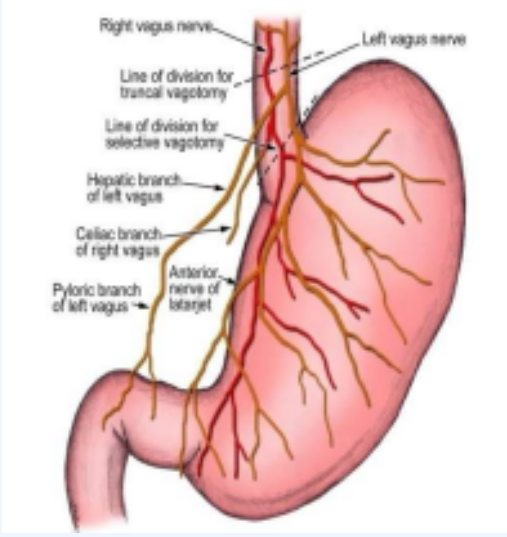
1. Left gastric lymph nodes: drains areas of both anterior and posterior gastric walls
2. Pancreaticosplenic nodes: drains gastric fundus and body
3. Right gastroepiploic nodes: drains the right half of the greater curvature, occasionally including the pylorus
4. Hepatic-pyloric-left gastric nodes: drain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



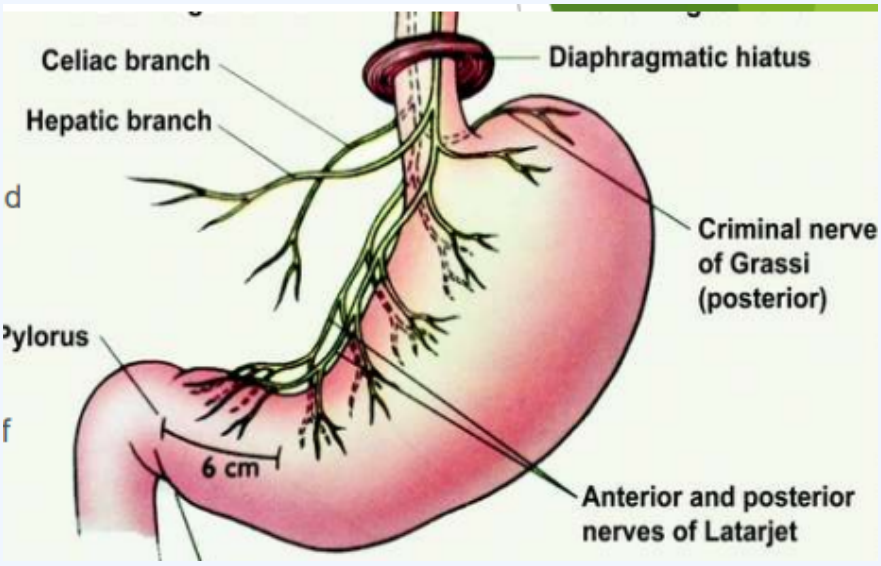
When a gastric tumor we don't have to remove spleen but once we remove it we remove fatty tissue surrounding it help us in collection the LN to know the exact grade of the cancer

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.**

Most of stomach innervation from parasympathetic nurve

Vagus if devide before diaphragm ➡4 branches if after 2 branches



Levels of vagotomy

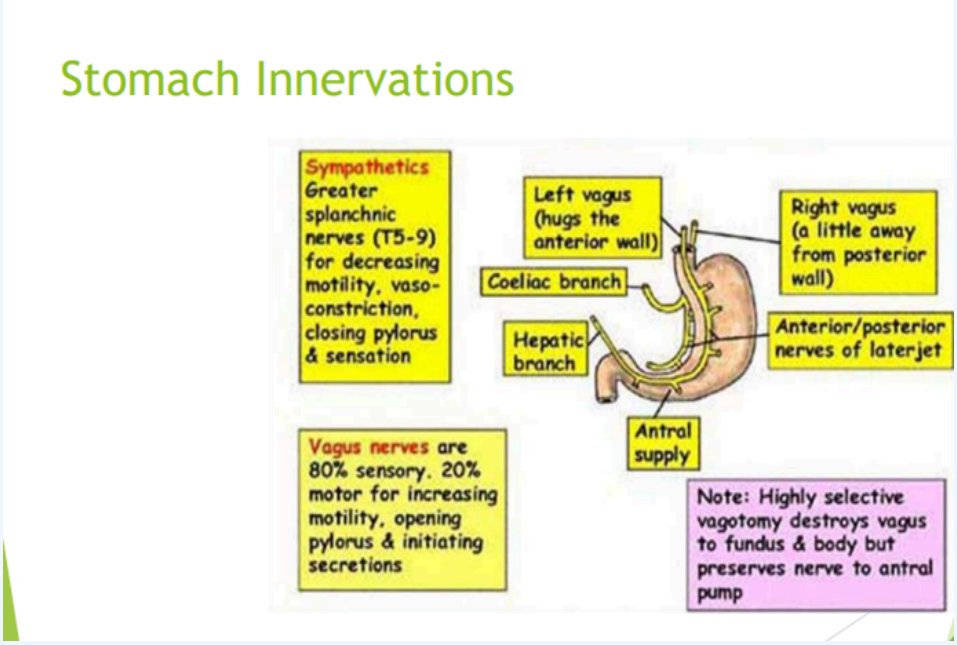
- **Truncal vagotomy just below the diaphragmatic esophageal hiatus before it gives off celiac and hepatic branches.**
- **selective vagotomy is performed distal to this location and spares the celiac and hepatic branches.**
- **Highly selective vagotomy (parietal cell vagotomy) divides individual terminal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

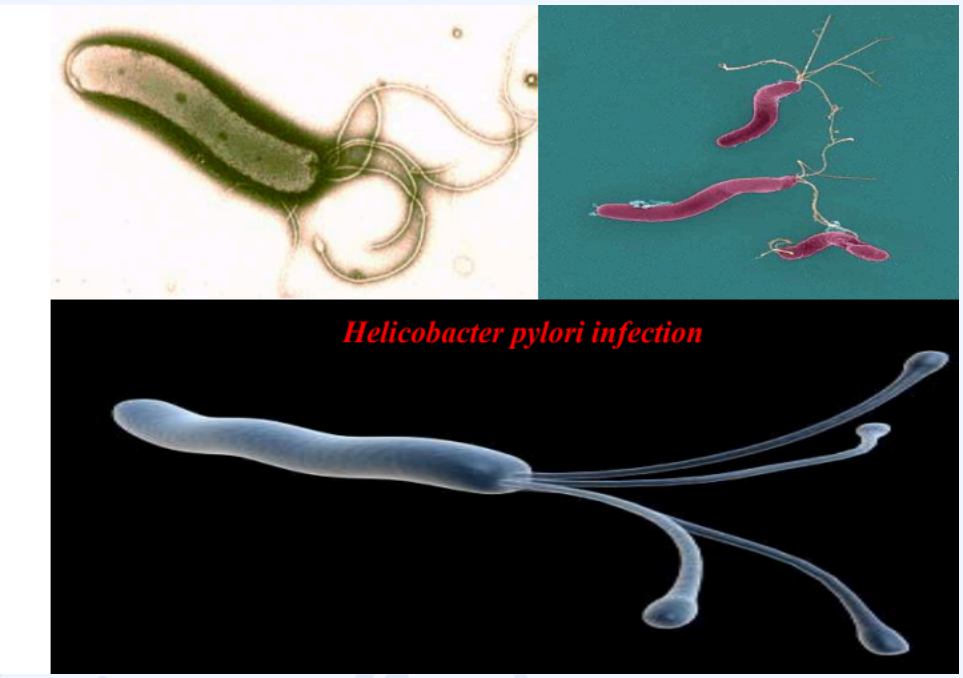
vagus related to prestaltic movement so if cut we must do drainage procedure (widening the pylorus)

Any upper abdominal pain radiate to back

 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.





 H. pylori is a curved or S-shaped, gram-negative microaerophilic motile bacterium .whose natural habitat is the human stomach
develop PUD %15_ although 2/3 of population are infected with H. pylori, only 10 & the majority are asymptomatic
chronic gastritis___ Conditions associated with H.Pylori infection acute gastritis
 PUD : 90% of DU , 75% of Gastric Ulcers
 Long standing chronic gastritis
gastric atrophy & increased risk of metaplas___
ia Gastric adenocarcinoma
 More than 90% of MALT lymphomas have H. pylori & low grade tumours regress with H. pylori eradication

Hcl from praytal cell secrete by 1.vagal stimulation "so mostly at night parasympathetic,could be by tumor in brain,2 by gastrin hormone from g cell in antrum,3 histamine
the following increase gastrin level
Both
Zollinger-Ellison Syndrome(tumor) and H pylory are correct

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

If +ve culture of H pylory but Àsymptomatic don't cure to avoid resistance

metronidazole could replace amoxicillin if there if ptenicillin allergy
Treatment needs 14 days
Best treatment is triple therapy:2Antibiotic+1PPI
Tetracyclin is recommended when resistance Of clanthomycin
If tetra resist use rifabutine
Doxacycline could be used if tetracycline aint present

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

ولحتى نعالج اي السر لازم اعالج البكتيريا والالسر لهيك بعطي ppi لعلاج الالسر مع الانتي بيوتيك للبكتيريا

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)**

Brain injury could rise vagal stimulation lead to ⬆ HCl then ulcer called stress ulcer

	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 (by Tumer gross factor alpha)**
- **Progressive dysphagia or odynophagia**
- **Recurrent vomiting**
- **Family history of gastrointestinal cancer**
- **all need endoscopy to be performed**

⬆Hcl could lead to GORD

Usually neither Guarding nor rigidety are present In physical exam of gastritis,if present this means perforation(complication)

SoH pylory have a relation to both lymphoma "when rise inflammatory reaction) and gastric ca

One of the alarm feature of Gastric CA is early satiety ،Stool Ag isn't enough,we should do upper endoscopy

كل العيلة If one in the family with Hereditary Nonpolyposis Colorectal Cancer(HNPCC) rise the risk of gastric CA ؛ so must do upper endoscopy

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)**

Chronic élévation of gastrin ➡rise Hcl secretion in stomache so gastric atrophy ➡increase risk of metaplasia

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

Investigation of perforation done by chest x-ray;when stomach perforation gass will accomulate below diaphragm

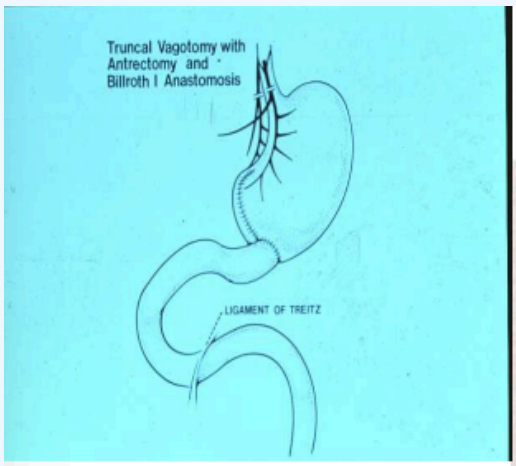
Modify Johnson's classification

Type	Location	Acid Hypersecretion	
I	Lesser curvature, incisura	No	Antrectomy only
II	Body of stomach, incisura, and duodenal ulcer (active or healed)	Yes	Type II,III treated as duodenal ulcer, preferred tx include antrectomy (to include ulcer) + truncal vagotomy with Billroth I OR II
III	Prepyloric	Yes	
IV	High on lesser curve, near gastroesophageal junction	No	Modified Antrectomy including ulcer+ billroth I OR subtotal gastrectomy with Roux en Y without vagotomy
V	Anywhere (medication induced)	No	

SURGICAL OPTION

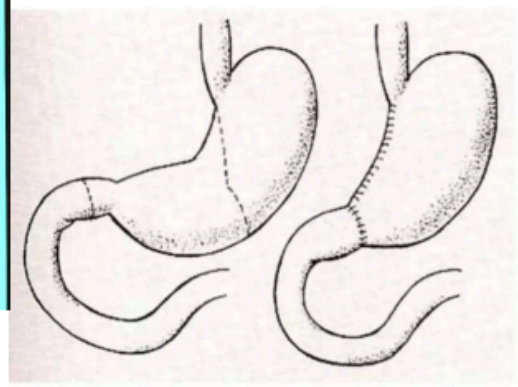
- VAGOTOMY
  - Truncal and drainage
  - Selective
  - Highly selective
  - Posterior vagotomy and anterior seromyotomy
- GASTRECTOMY
  - Billroth I
  - Billroth II
  - Subtotal gastrectomy
- GRAHAM'S OMENTAL PATCH
- SUTURE LIGATION OF GASTRODUODENAL ARTERY
- UNDRER-RUNNING AN ULCER BASE
  - After excision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

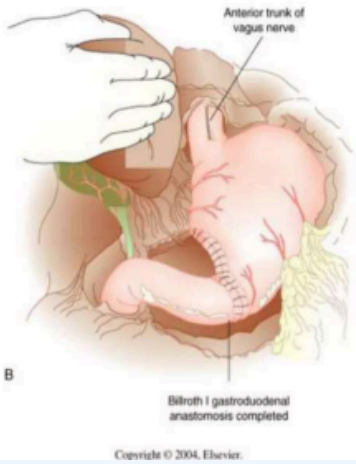
Billroth I



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

Done for gastric ulcer in the antrum

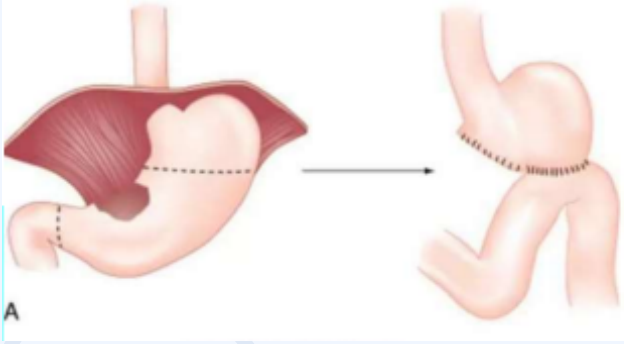


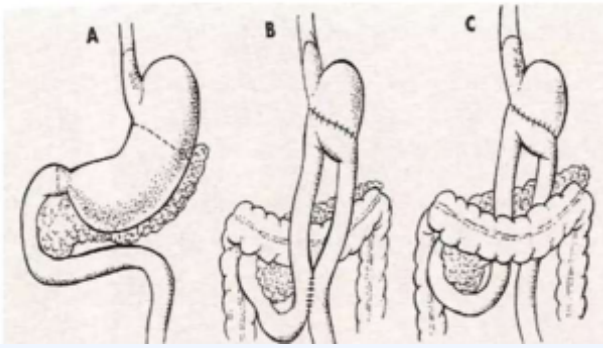


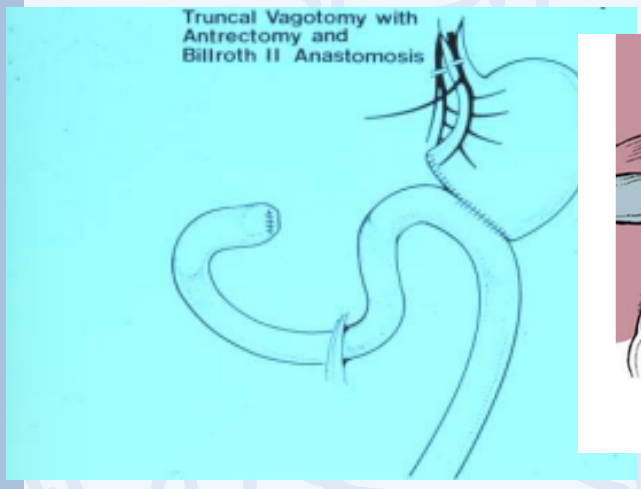
Billroth II

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

Done for a proximal gastric ulcer







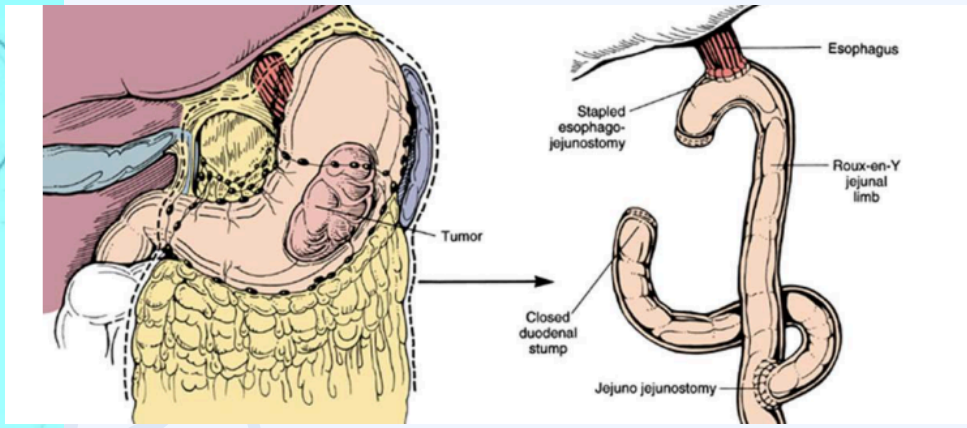


Table 25-12 Surgical Options in the Treatment of Duodenal and Gastric Ulcer Disease		
Indication	**Duodenal**	**Gastric**
Bleeding	1. Oversew[a] 2. Oversew, V+D 3. V+A	1. Oversew and biopsy[a] 2. Oversew, biopsy, V+D 3. Distal gastrectomy[b]
Perforation	1. Patch[a] 2. Patch, HSV[b] 3. Patch, V+D	1. Biopsy 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

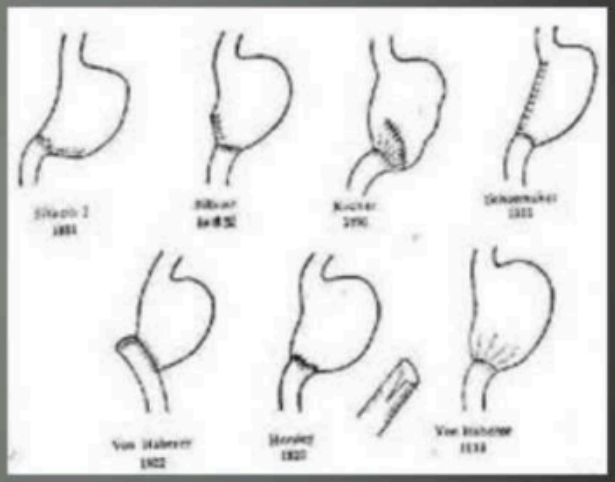
SOURCE: Modified with permission from Mulholland MW, Debas HT: Chronic duodenal and gastric ulcer. *Surg Clin North Am* 67:489, 1987.

When the ulcer caused by weakness of mucosal barrier mostly in lesser carveture or around cardium&
In body or insesora caused by rise acid secretion
To lower Hcl we can cut the vagus but we should also dilate pylorus with it "drainage procedure"
The perforation better closed by the omentum "called Graham patch"then give Ppi
Antrum could be removed

Billroth I

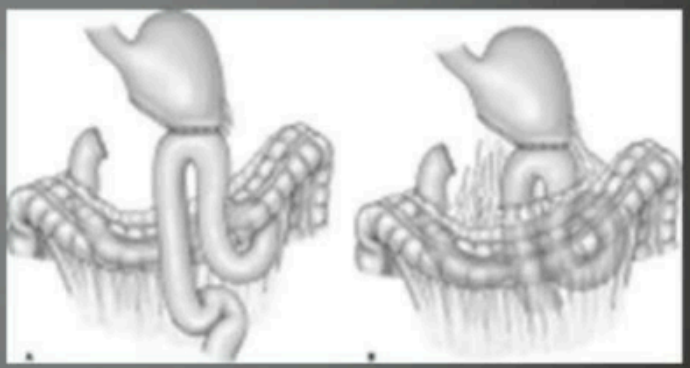
- Preserves duodenal passage
- Preserves pancreatic function
  - Altered after gastrojejunostomy
  - Better fat digestion
- Less incidence of gastritis and reflux
- Can use only with distal limited gastric resections

tension

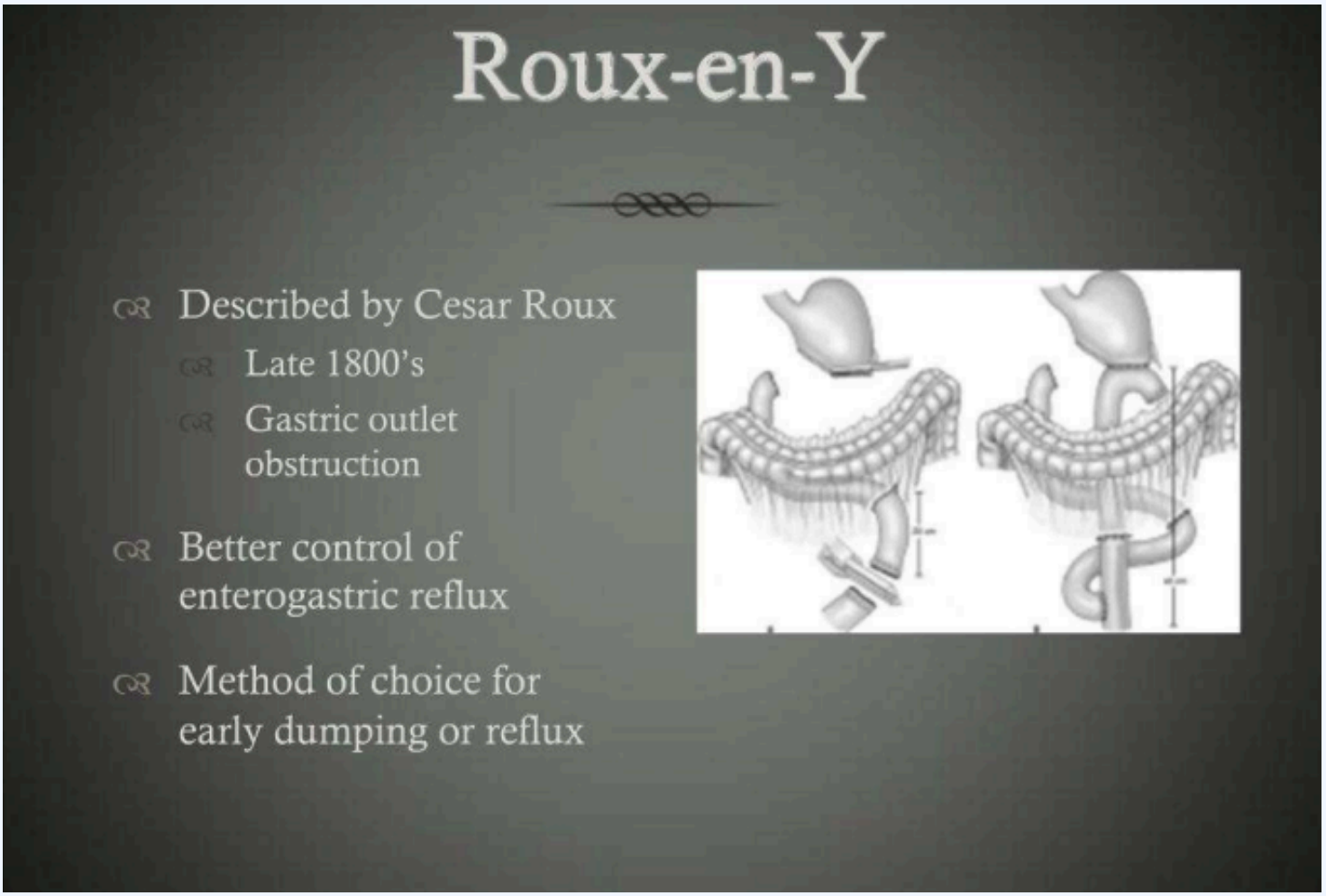


Billroth II

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



The dudenum home many enzymes so hard to seal it ,the best solution is billroth 2
But tin this way ,food directly go to bowel so intestine swell causing severe abdominal pain and diarrhea,the absorption fast so elevate insulin which lowers the BP this called Dumping syndrome
(Stages 1 diarrhea 2 hypotension)
If there is bleeding ➡ oversewing"best"
If a PT with previous perforation and already we've done Graham patch,we must cut vagus with drainage procedure
The last resort is gastrectomy



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**

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. Dumping syndrome first diarrhea then hypotension

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.

Risk F for gastric cancer is blood A

Any procedure in stomache rise risk for ca

Cox 1➡prostacycline ➡VD

Thrompoxine VC

Nicotinic receptor specific for nicotine present in vessles bind so VC

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.

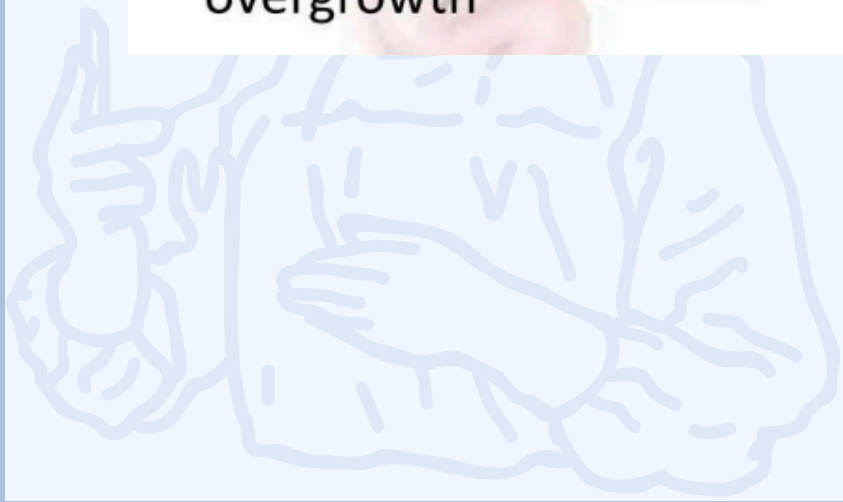
- In minority of pts who don't improve

- 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

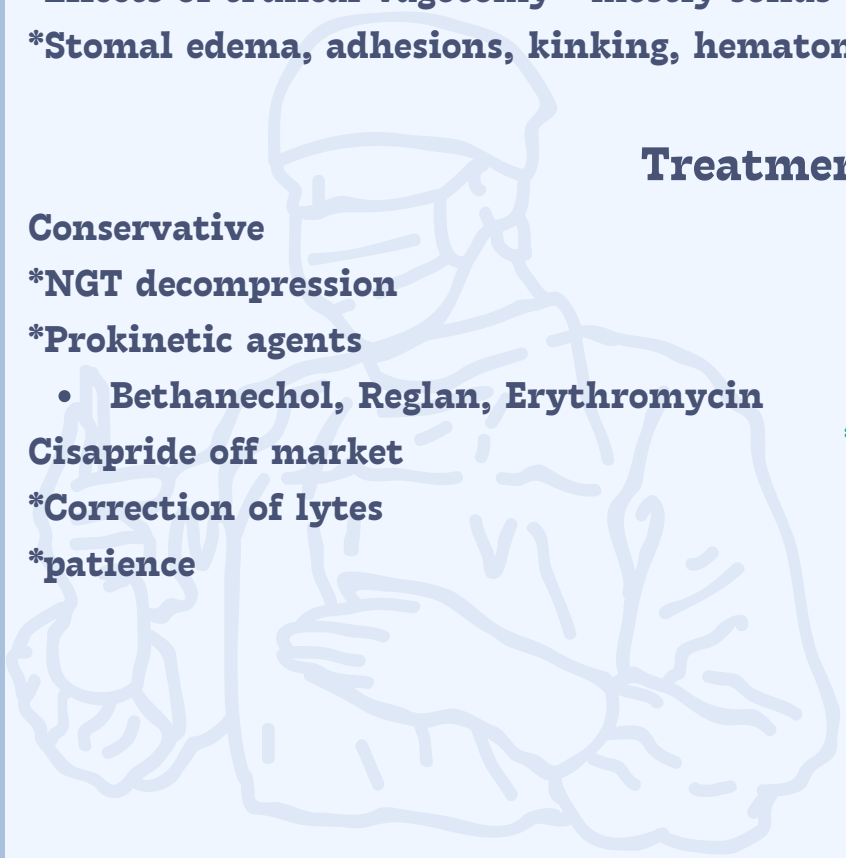
*Minimum of 3-4 wks

*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 o3 Persistent nausea, always bilious emesis
  - Pain characteristically not alleviated with emesis

*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

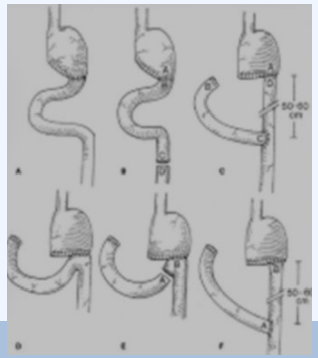
Treatment

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

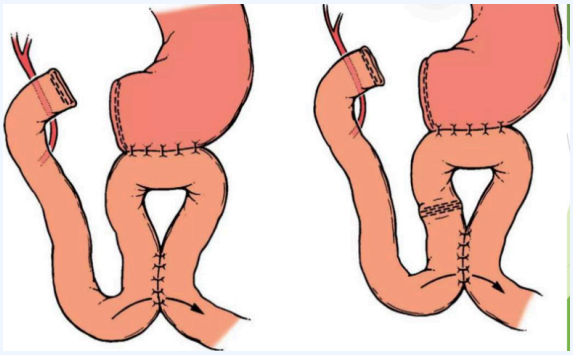
Surgical
*Braun procedure
*Distal enteroenterostomy
*Henley loop
*Roux-en-Y reconstruction

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

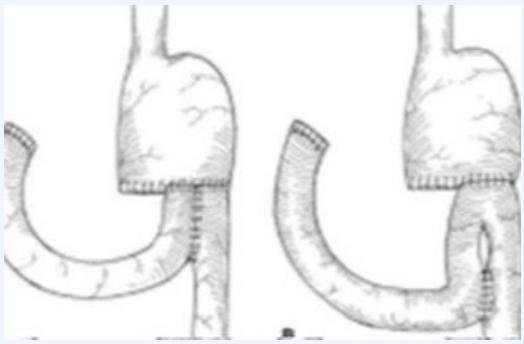


Bile Reflux Gastritis



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

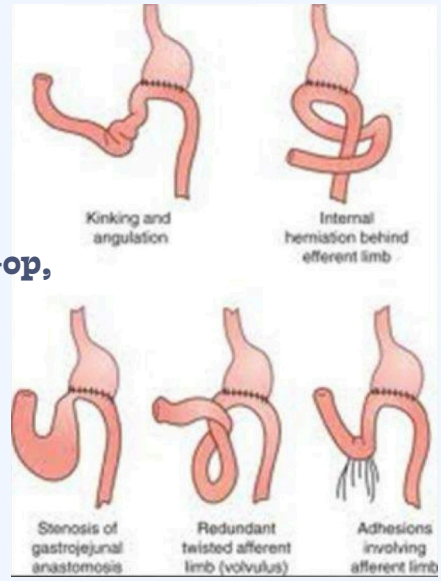
***Caused by partial or complete obstruction of jejunal limbs**
***Characteristic signs and symptoms**
***Afferent is more common than efferent**

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
- 3pancreatitis
- 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

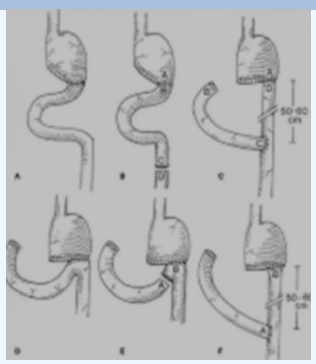
Chronic Afferent Limb Syndrome

- 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

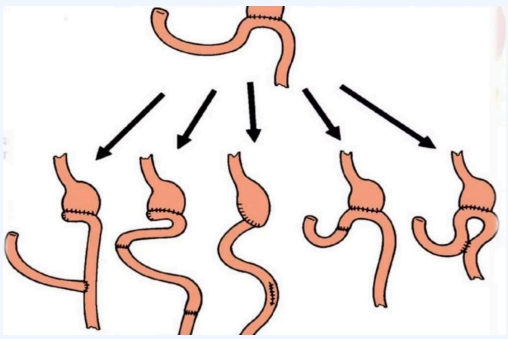
*Convert BII to BI

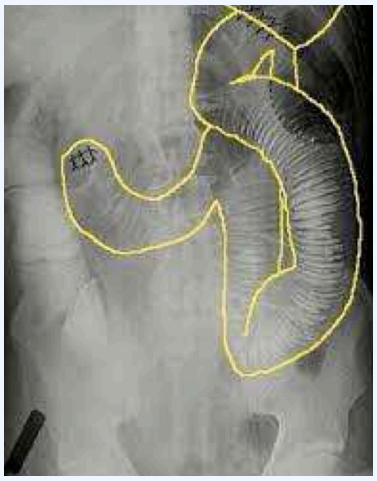
- Can be difficult to get to duodenal stump

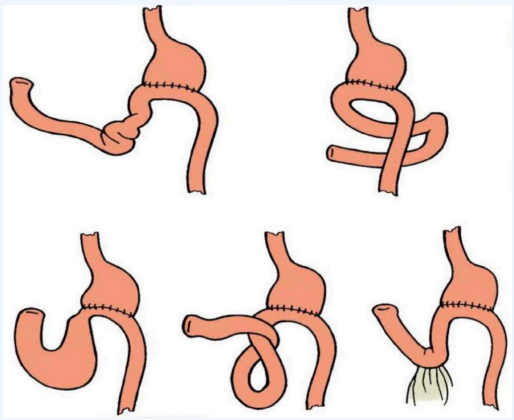
*Roux-en-Y - important to remember to perform complete vagotomy

Prevents marginal ulcers

Afferent and Efferent Loop Obstruction







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

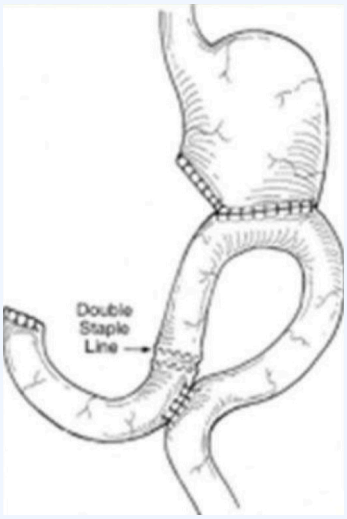
Treatment

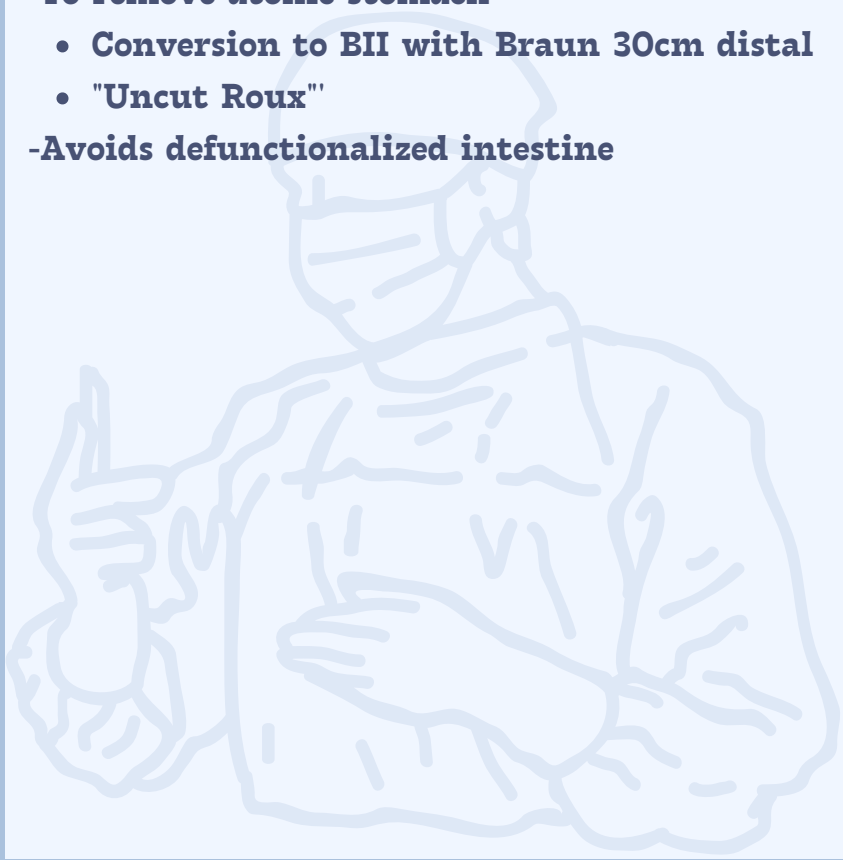
- Surgical
- Further gastric resection with new Roux-en-Y

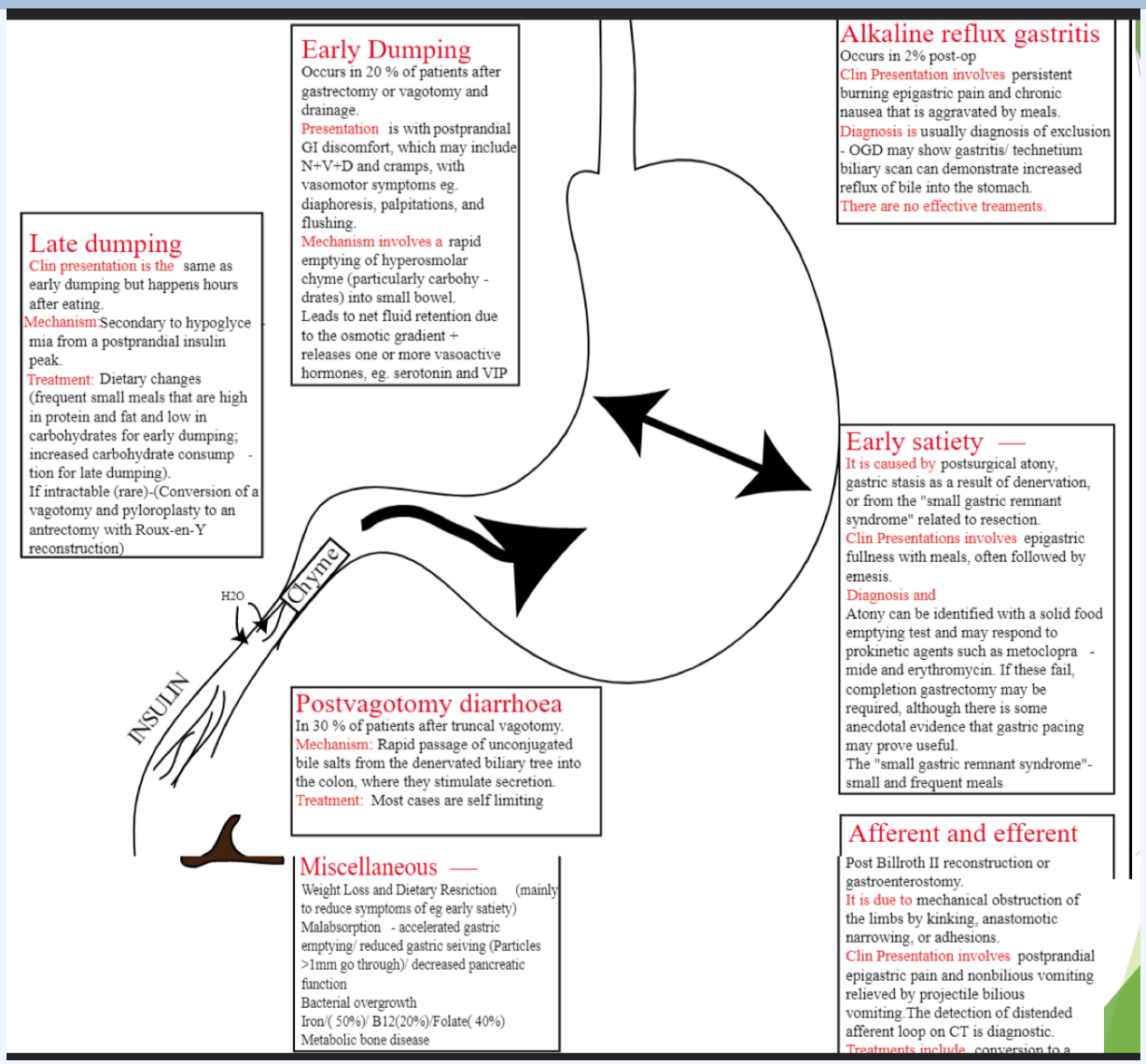
-To remove atonic stomach

- Conversion to BII with Braun 30cm distal
- "Uncut Roux"'

-Avoids defunctionalized intestine



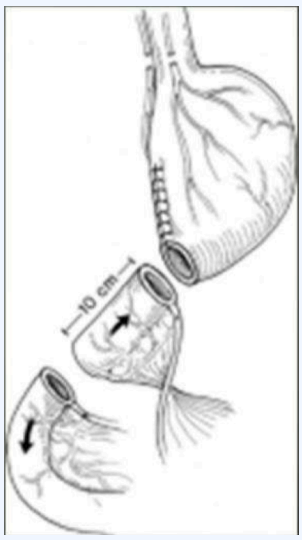




TREATMENT SUMMARY

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)

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.

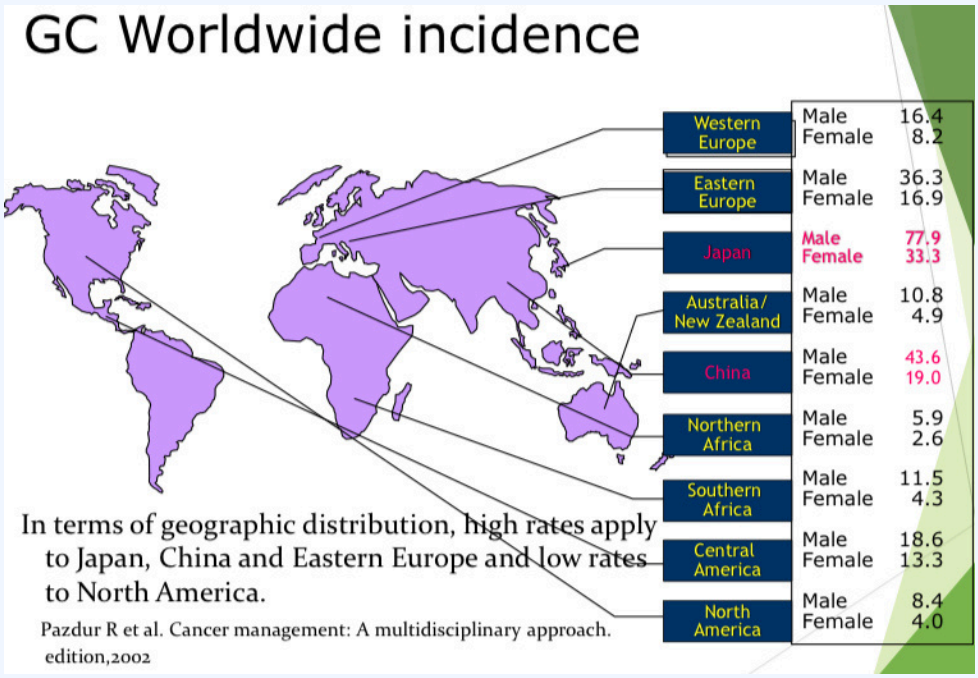
Male to female 2:1

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)

Because of high blood perfusion of stomach there is a high probability of metastasis, that's why the gastric CA lethal



Risk factors :

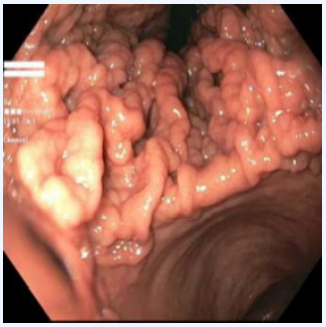
- **H.pylori infection : (most common risk factor)**
- Usually with prolonged infection >10 years
- Distal gastric ca, intestinal type in 90%
- Atrophic gastritis —>metaplasia —>dysplasia —> cancer

- **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**
- **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 (* important)**

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

*any surgery in the stomach will increase the risk of gastric cancer in

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. (adenocarcinoma)

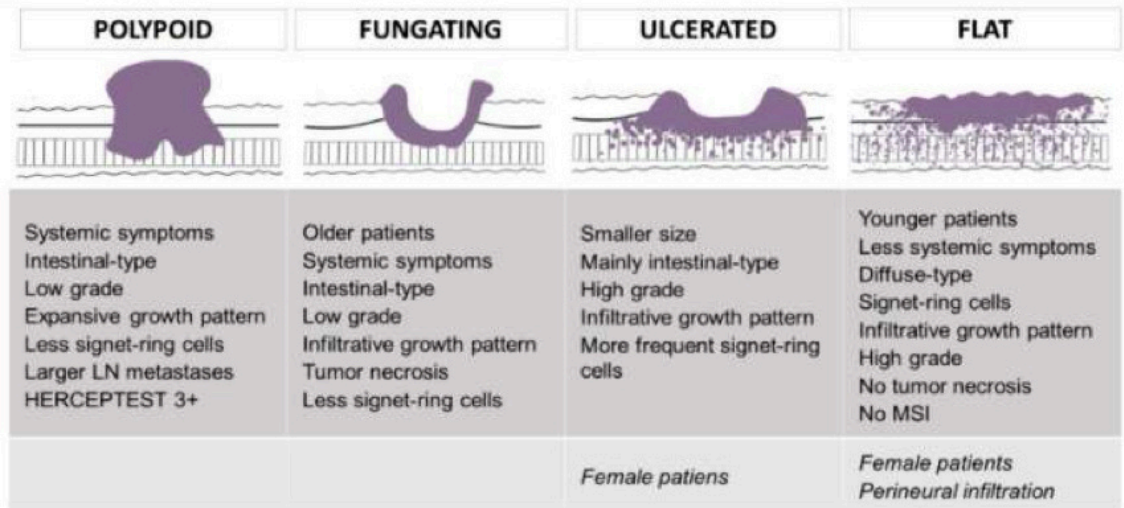
Various classifications schemes for gastric CA.

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*

If we see this lesions in upperendoscopy we should take biopsy

 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
  - (important)

Clinical Presentation :

CA is present in the stomach and will not cause severe weight loss. This common tumor grows and secretes tumor necrosis factor, which will cause severe weight loss.

- Asymptomatic, discovered incidentally
- Early gastric CA : But it can cause sever weight loss if it's near to cardia.
- 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%. The most commen Finding in Physical exam with gastric Cancer Patient is? Abdominal mass
- 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

- By upper endoscopy

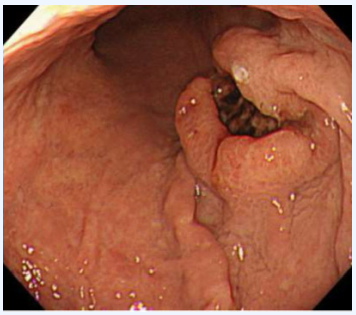
- Full Hx and PEx (age, chronicity of pain and non response to usual antacids)
- CBC (anemia) , KFT , LFT (usually normal but can be elevated in liver mets)

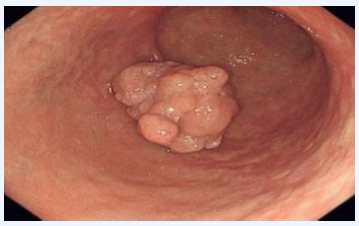
fecal occult blood → not specilic

- CEA : Carcinoembryonic antigen... helps us know if liver metastasis stage 4 and half live will be 6 months
  - 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++) The first thing I should think about is nutritional status patient.
- 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

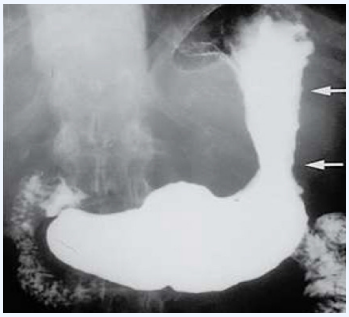




Barium swallow

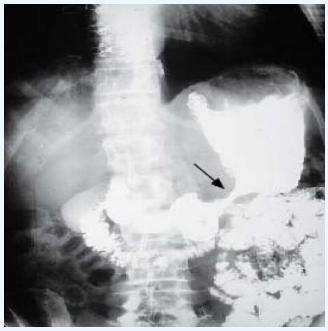
75% accuracy

for obstructive lesions only.



Linitis plastica "leather-flask" appearing stomach

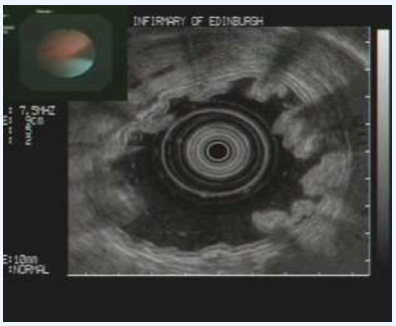
Apple core sign"



 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.

It helps us to know the stage of the tumor and how far this tumor has reached within the layer of the stomach. The stage is different in classification.
The stage tells us what condition the tumor is in, and in order for this tumor to spread, it must .invade the stomach layer



 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 : Combined nuclear radiation + CT to see activity of tumors

- 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.