Stomach and Duodenum

2007-2008 Student Lecture Series

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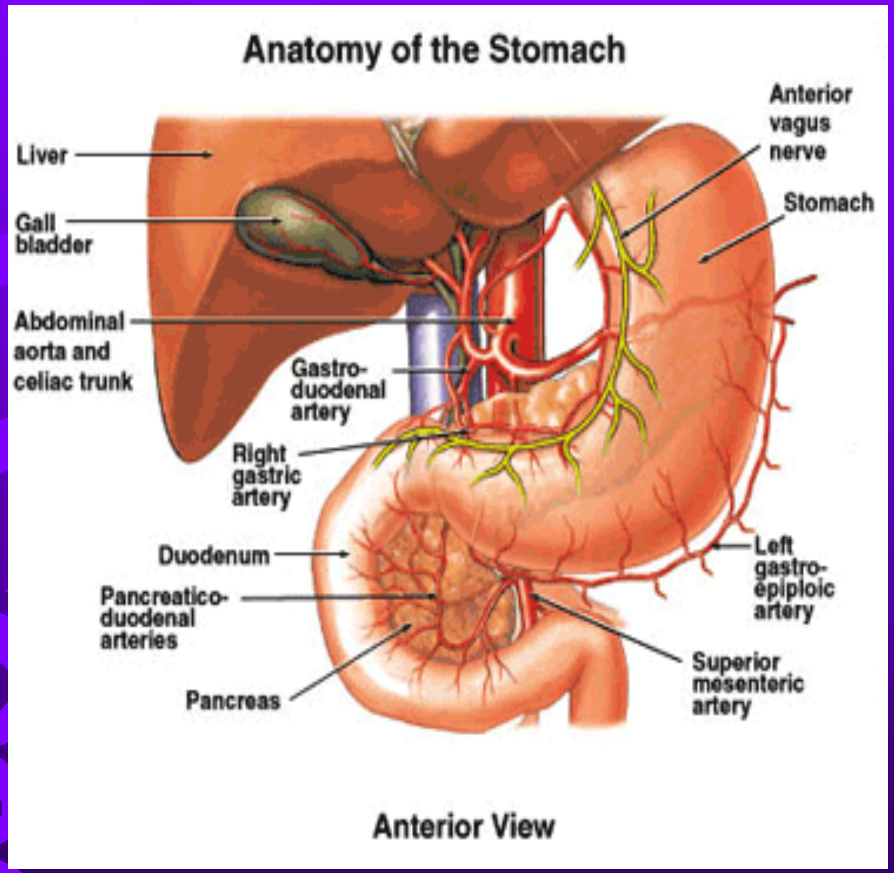
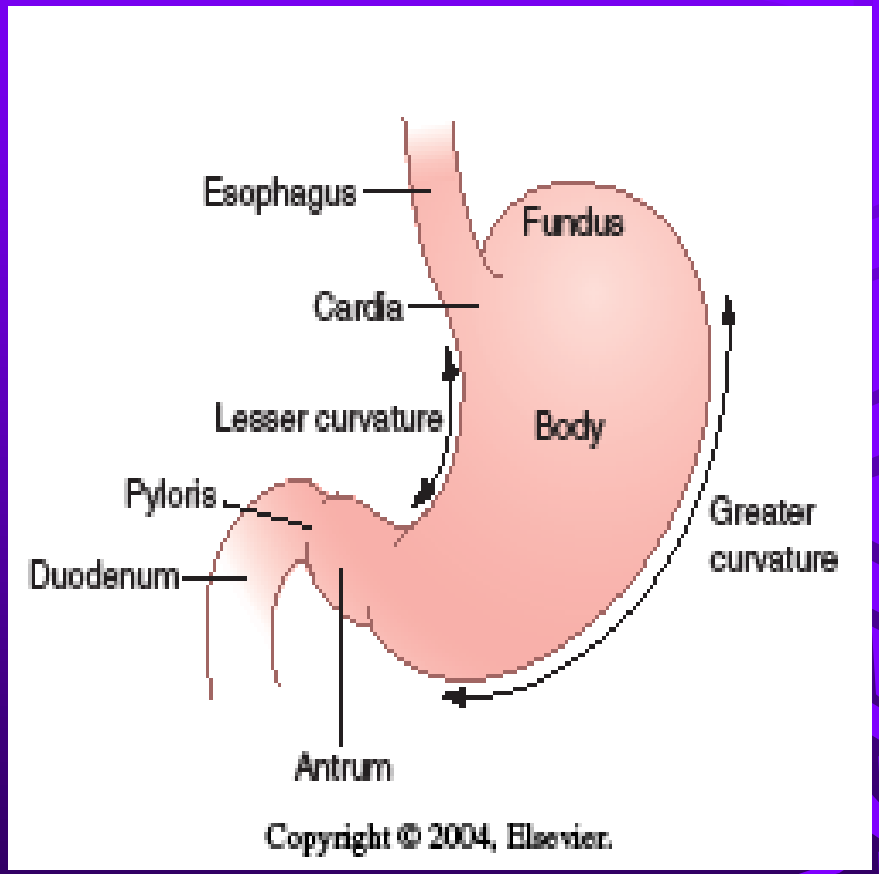
Anatomy

- Appears in week 5.
- A pliable, saccular organ.
- Located in the LUQ and epigastrium.
- Separated from the GI tract (2 locations).

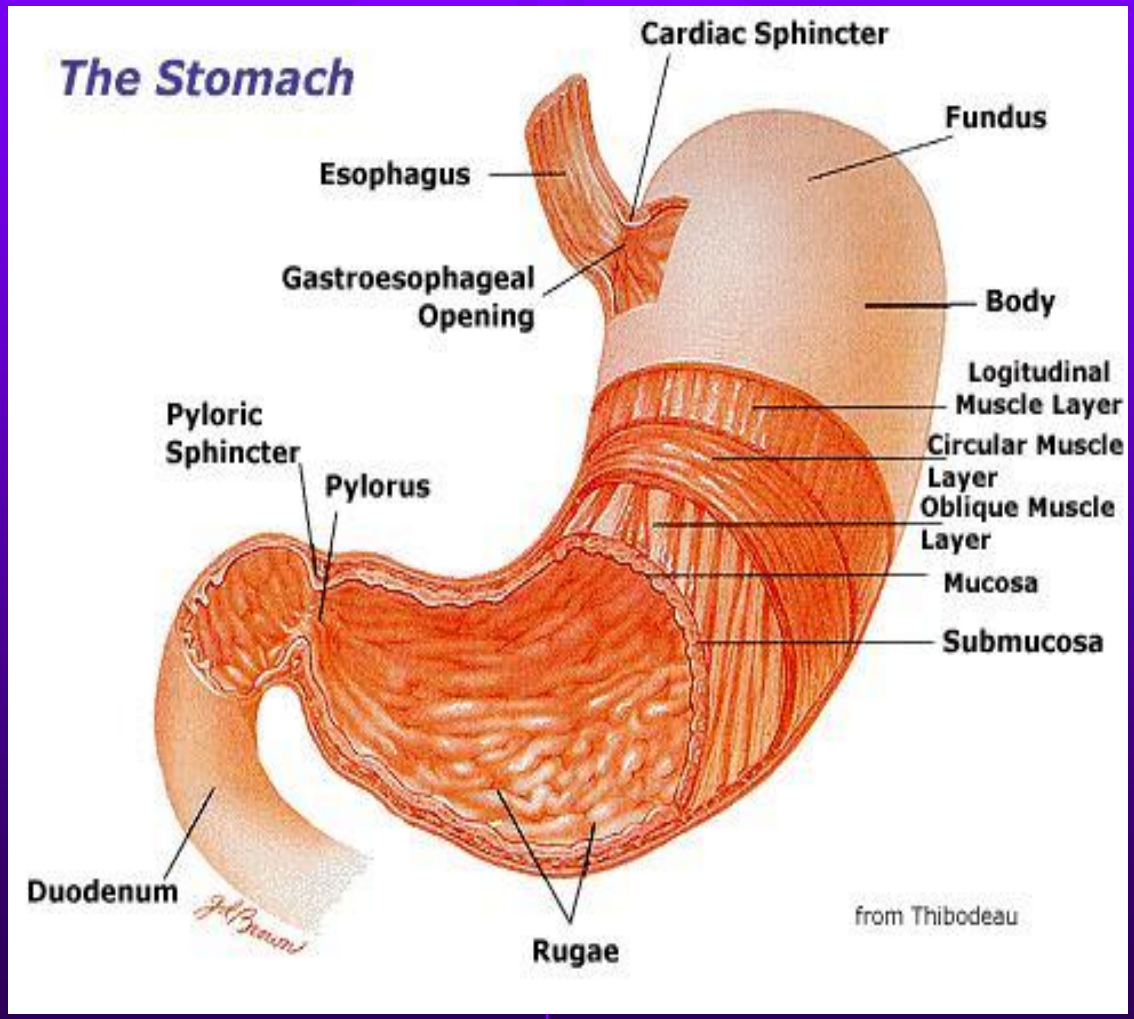


Gross Anatomy



- Proximal= Cardia (attaches to esophagus) attaches at the LES.
- Fundus= most superior portion, receives food.
- Body= largest portion, contains parietal, chief and ECL cells.
- Distal= antrum, contains the G cells.

Anatomy


The Stomach
Cardiac Sphincter
Fundus
Esophagus
Gastroesophageal Opening
Body
Logitudinal Muscle Layer
Circular Muscle Layer
Oblique Muscle Layer
Mucosa
Submucosa
Pyloric Sphincter
Pylorus
Duodenum
Rugae
from Thibodeau

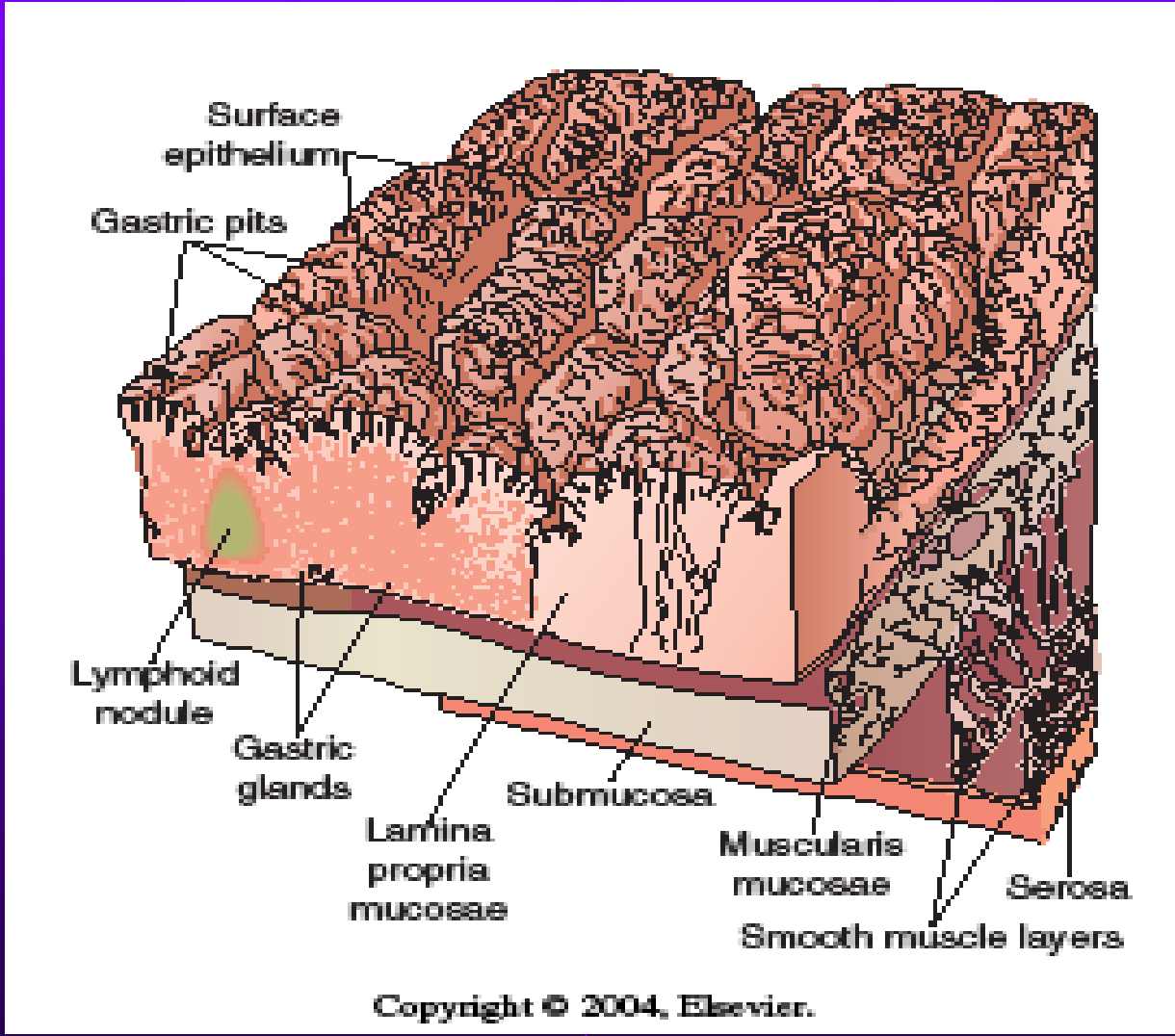
- The stomach is almost entirely covered with peritoneum.
- The peritoneum forms the outer gastrc serosa.
- Beneath the serosa is the muscularis propria.
- The MP is made up of 3 layers of smooth muscle.
  - The middle layer is the circular muscle and is the only “complete” layer of muscle

- As you progress distally the middle layer of muscle begins to thicken and form the _____? Which functions as a true sphincter.
- This and the GE junction form the gastric "borders" and are the two "fixed" points of the stomach.
- The outer muscle layer (longitudinal) is contiguous with the outer layer of the esophagus.

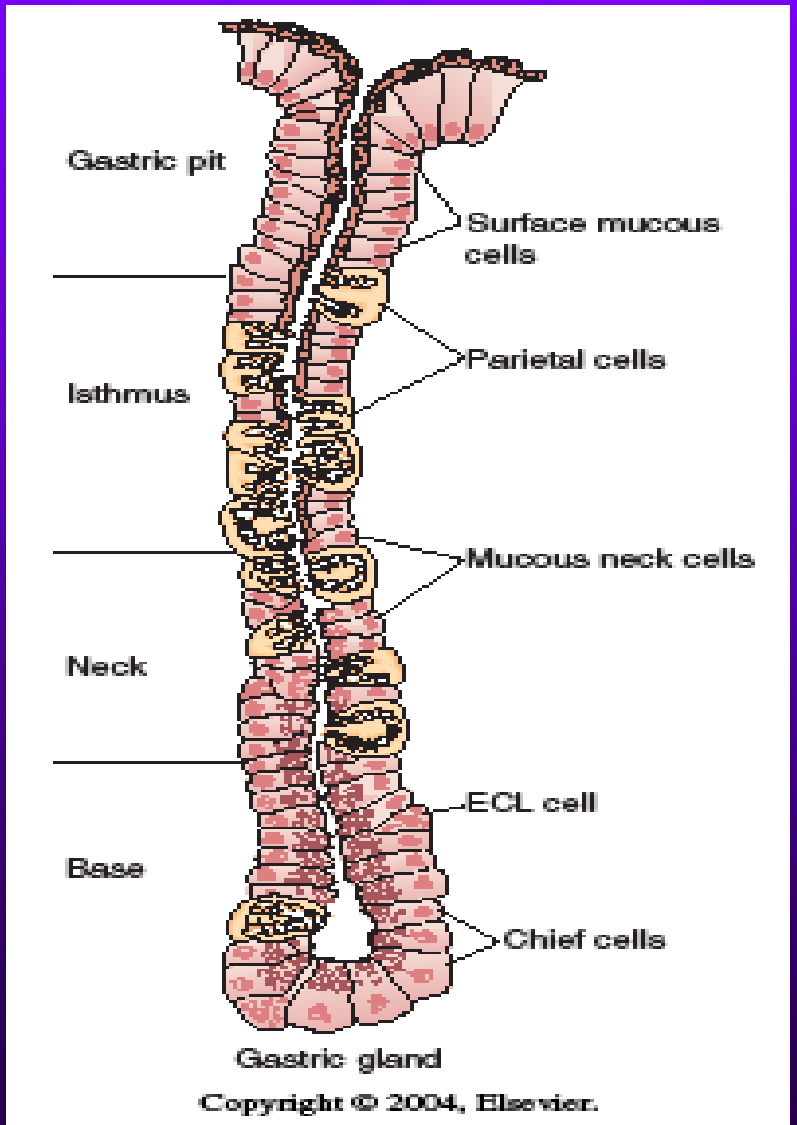
- Within the layers of the MP, there is a rich plexus of autonomic nerves and ganglia called_____.
- The submucosa lies between the MP and the mucosa. It is a collagen rich layer of connective tissue and is the *weakest/strongest* layer of the gastric wall.
- The submucosa also contains the rich blood vessel network and the lypmhatics as well as Meissner's plexus.

- The mucosa consists of 3 layers:
  - Surface epithelium (columnar).
  - Lamina propria
    - Connective tissue layer that supports the surface epithelium.
  - Muscularis mucosae (probably the reason for rugal folds).
    - The MM is the boundary for invasive/noninvasive gastric cancer.

Anatomy/Morphology



Anatomy/Morphology



Cell Types

- Parietal:
  - Location: Body
  - Function: secrete acid and intrinsic factor
- Mucus:
  - Location: Body, Antrum
  - Function: mucus production
- Chief:
  - Location: Body
  - Function: produce Pepsin

Cell Types

- Surface epithelium:
  - Location: Diffuse
  - Function: produce mucus, bicarb, prostaglandins(?)
- ECL:
  - Location: Body
  - Function: Histamine production
- G cells:
  - Location: Antrum
  - Function: Gastrin production

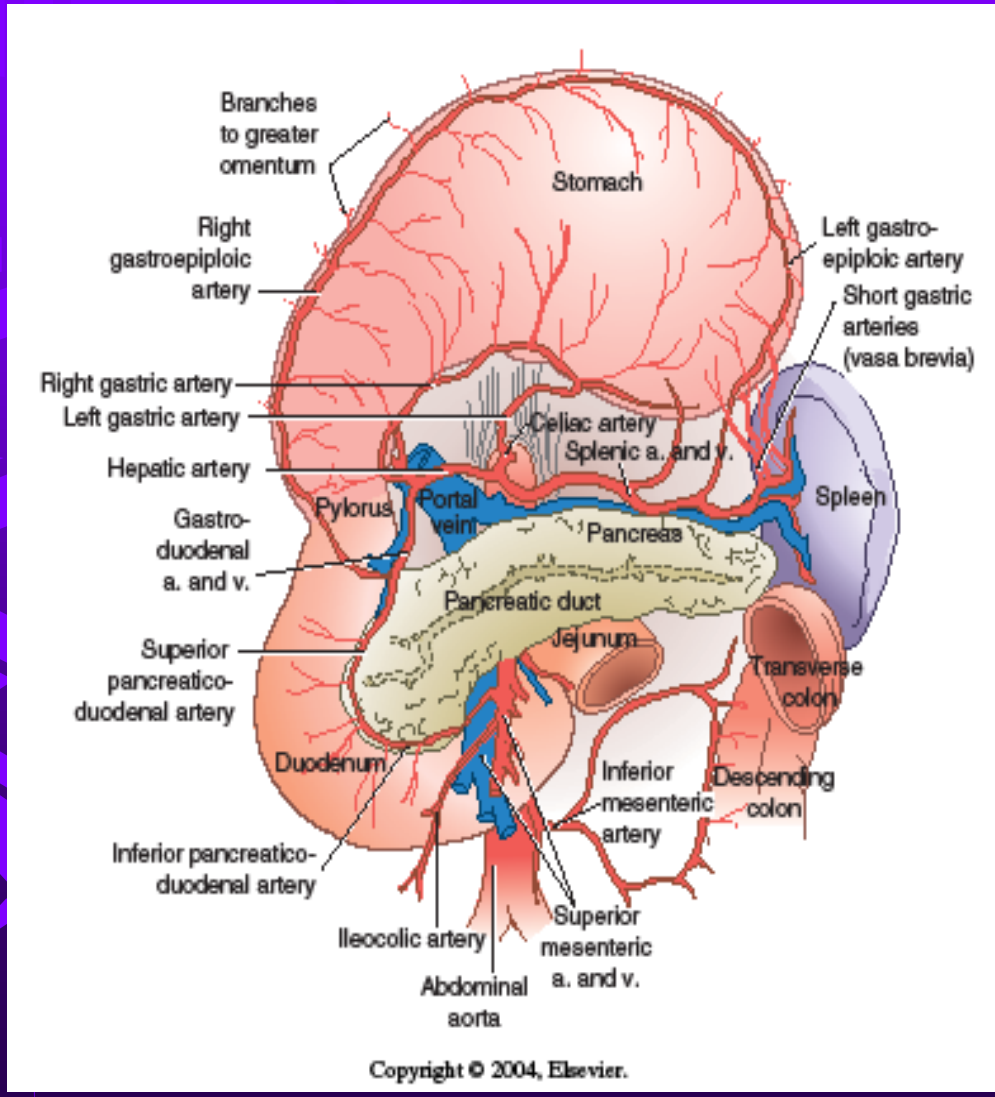
Cell Types

- D cells:
  - Location: Body, Antrum
  - Function: produce Somatostatin
- Gastric mucosal interneurons:
  - Location: Body, Antrum
  - Function: produce Gastrin-releasing peptide
- Entric Neurons:
  - Location: Diffuse
  - Function: CGRP, others production

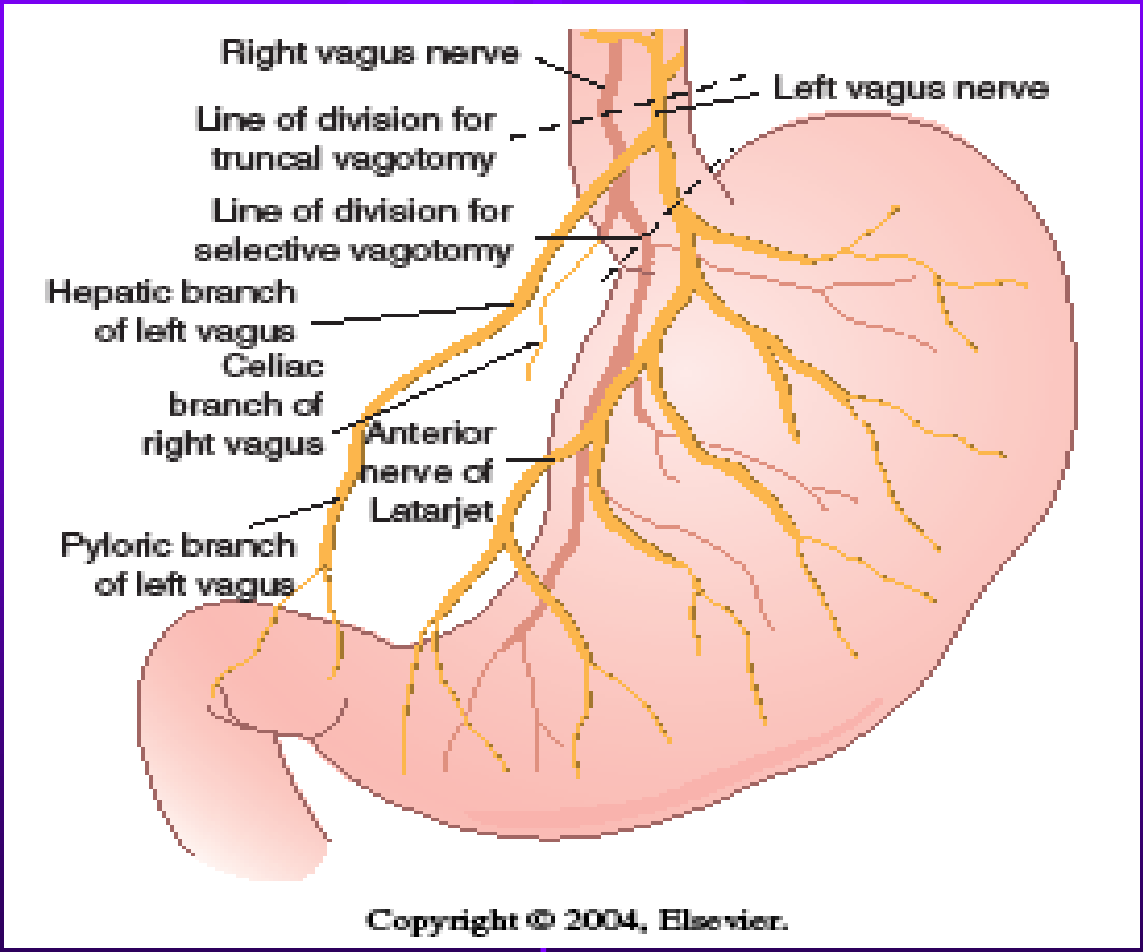
Vascular Supply

- R&L gastrics
- R&L gastroepiploics
- Short gastrics
- Inferior phrenics
- Gastroduodenal

- Venous drainage:
- R&L gastric veins drain to the portal, R gastroepiploic drains to the SMV, L ge drains to the splenic



Nerve supply



Gastric Physiology

- Principle Function:
  - Storage:
    - Receptive relaxation
  - Start digestion:
    - Separates meal into fat/protein/carbohydrates

Regulation of Function

- The stomach is under both neural and hormonal control.

Gastric Hormones

- Gastric Hormones:
  - Chemical messengers that regulate intestinal and pancreatic function.
  - The "gut" is the largest endocrine organ in the body.
  - The messengers can act as:
    - Endocrine: distant target
    - Paracrine: close target
    - Autocrine: self target
    - Neurocrine: neurotransmitter or stimulator.

Gastric Hormones

- Gastric Hormones:
  - Synthesized as inactive precursors
  - Converted to active form by post-translational modification
  - #1 stimulus for release is: FOOD
    - Composition of food dictates timing and specific hormone release.

Gastric Hormones

- Gastric Hormones:
  - Inhibition:
    - Removal of stimulus
    - Negative feed-back loops
    - Inhibitory peptides, ie. Somatostatin

Gastric Hormones

- Gastrin
- Somatostatin
- Gastrin-releasing peptide (GRP)
- Histamine

Gastrin

- Synthesis: G-cells in the antrum
- Release:
  - AA, protein, vagal tone, antral distention, GRP, pH > 3.0, ETOH, Histamine.
- Inhibition:
  - pH < 3.0, somatostatin, secretin, CCK, VIP, GIP, glucagon.
- Target cells:
  - Parietal and Chief cells

Gastrin

- Action(s):
  - Stimulates acid secretion
    - Direct action on parietal cells
    - Potentiating interaction with histamine
    - Possible: releasing of histamine
  - Increases release of lytes & water from stomach, pancreas, liver and Brunner's glands
  - Stimulates motility in stomach, intestine, and gall bladder
  - Inhibits contraction of pylorus and sphincter of Oddi.
  - Stimulates GI mucosal growth.

Somatostatin

- Tetradecapeptide
- Synthesis:
  - CNS, antrum, fundus, sm. bowel, colon, and D-cells in pancreas.
- Release:
  - Antral acidification
  - Fats, protein, acid in duodenum
  - Pancreatic: glucose, amino acids, CCK
- Inhibition:
  - Release of acetyl-choline from vagal nerve fibers

Somatostatin

- Action(s):
  - The “master off switch”
  - Inhibits the release of most GI hormones
  - Inhibits pancreatic and GI secretion(s)
  - Inhibits intestinal motility.
- Clinical:
  - Octreotide- decrease fistula output
  - Treatment of esophageal variceal bleed
  - Can ameliorate symptoms of endocrine tumors

GRP

- Mammalian equivalent of Bombesin
- Synthesis:
  - Gastric antrum, small bowel mucosa
- Release: vagal stimulation

GRP

- Action(s):
  - The "master on switch"
  - Stimulates the release of all GI hormones (? Secretin).
  - Stimulates GI secretion
  - Stimulates GI motility
  - * most important: stimulates gastric acid secretion and release of antral gastrin
  - Stimulates bowel and pancreatic mucosal growth and stimulates various GI and pancreatic CA's

Histamine

- Stimulates parietal cells
- Found in the acidic granules of ECL cells and resident Mast cells.
- Release is stimulated by:
  - Gastrin, acetyl-choline, epinephrine
- Inhibited by Somatostatin.
- ? A necessary intermediary of acid production.

Acid Secretion

- Two forms:
  - Basal Acid Secretion
  - Stimulated Acid Secretion

Stimulated Acid Secretion

- Three Phases:
  - Cephalic phase
  - Gastric phase
  - Intestinal Phase
- These phases occur *concurrently* NOT *consecutively*.

Cephalic Phase

- Originates with the sight, smell, thought or taste of food.
- Stimulates the cortex and hypothalamus.
- Signals cause Vagus to release Ach, Ach causes increase in parietal cell acid production.
- Accounts for 20-30% of acid production.

Gastric Phase

- Begins when food enters the gastric lumen (gastric distention).
- Digestion products stimulate the G cells, they release gastrin, parietal cells release acid.
- Distention alone can increase acid production.
- Accounts for 60-70% of acid production.
- Phase lasts until the stomach is empty.

Intestinal Phase

- Poorly understood.
- (?) initiated by chyme entering the small bowel.
- Accounts for ~10% of acid production.

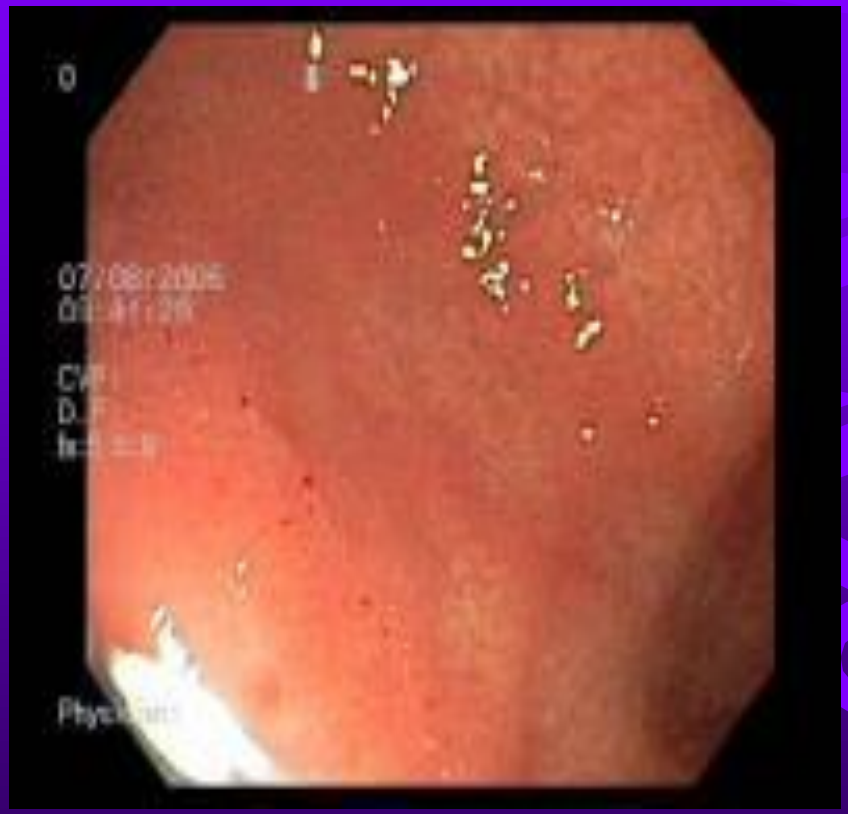
Other functions

- Gastric acid suppression
- Mucosal protection
- B_{12} absorption

Benign Gastric Disease(s)

- Acute/Stress Gastritis
- Gastric (peptic) Ulcer Disease
- Hypertrophic Gastritis
- Mallory-Weiss Syndrome
- Gastric Polyps
- Bezoars

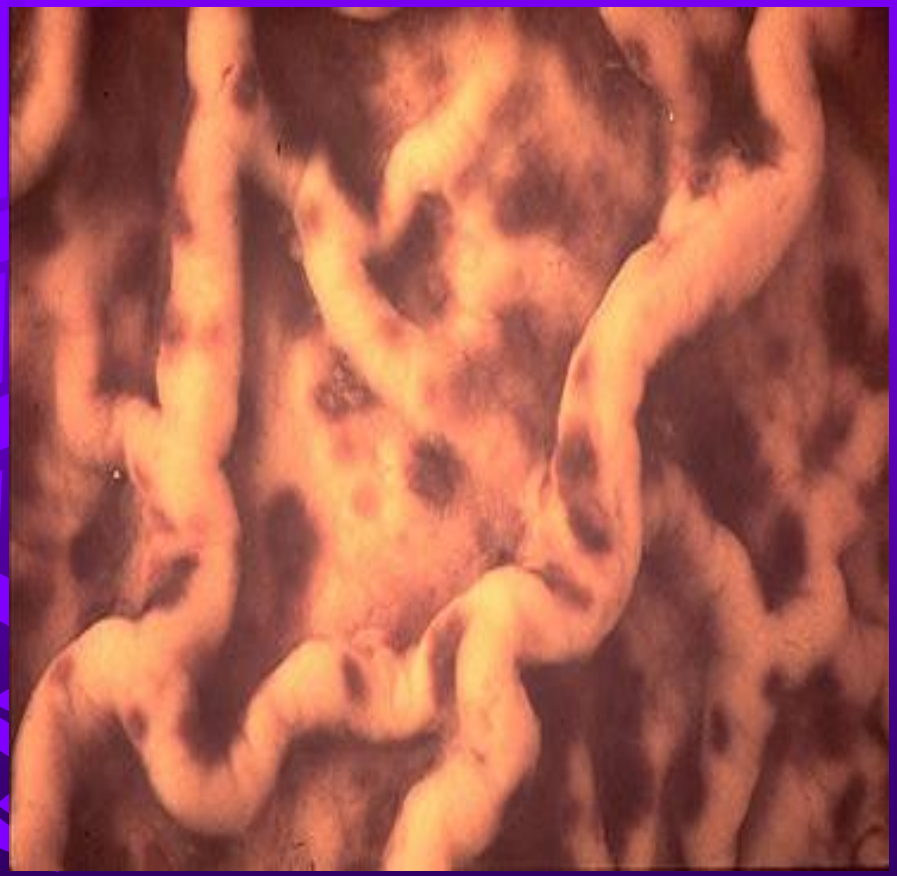
Gastritis (acute or stress)



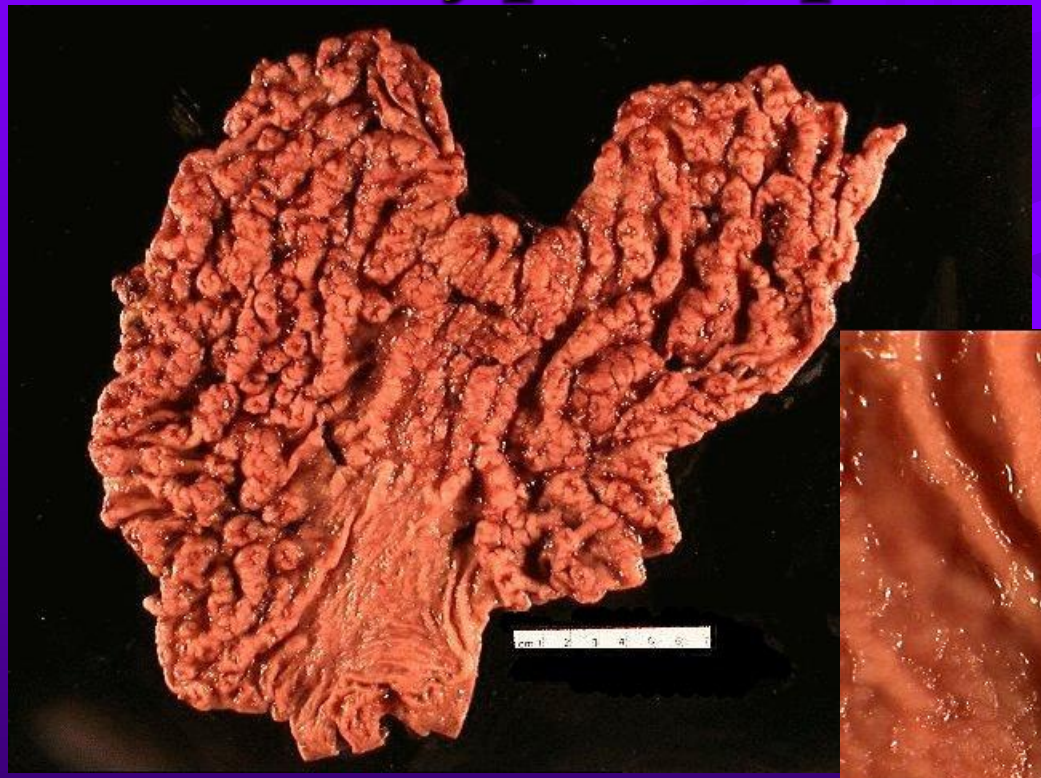
- Produces inflammation of the mucosa.
- Can be associated with erosions and bleeding.
- Causes:
  - *H. pylori*, NSAIDS, bile reflux, Etoh, radiation, local trauma, physiologic stress.

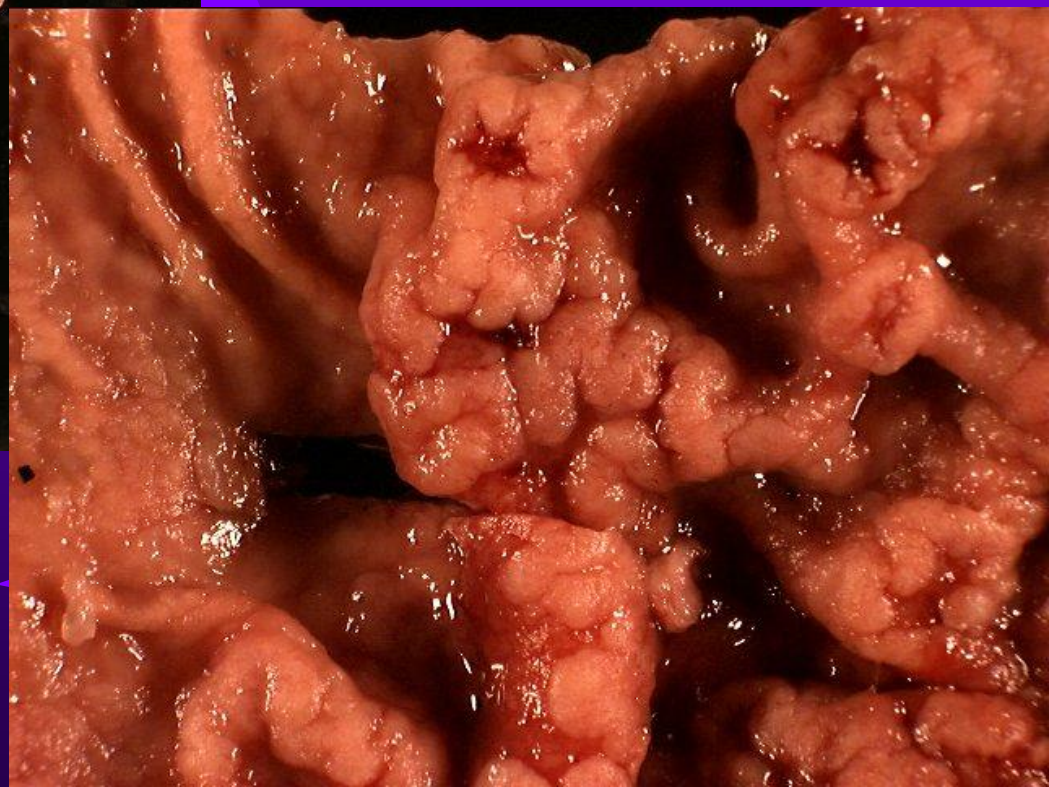
Gastritis

- S&S:
  - Nausea, emesis, hematemesis, melena, hematochezia, etc.
- Treatment:
  - Prevention, removal of offending agent, acid supression, occ gastric decompression and support.



Menetrier's Disease (aka Hypertrophic Gastritis)





Menetrier's Disease (aka Hypertrophic Gastritis)

- Rare.
- Characterized by massive hypertrophy of the gastric rugae.
- Etiology unknown.
- (?) autoimmune.
- (?) over-expression of TNF-β.

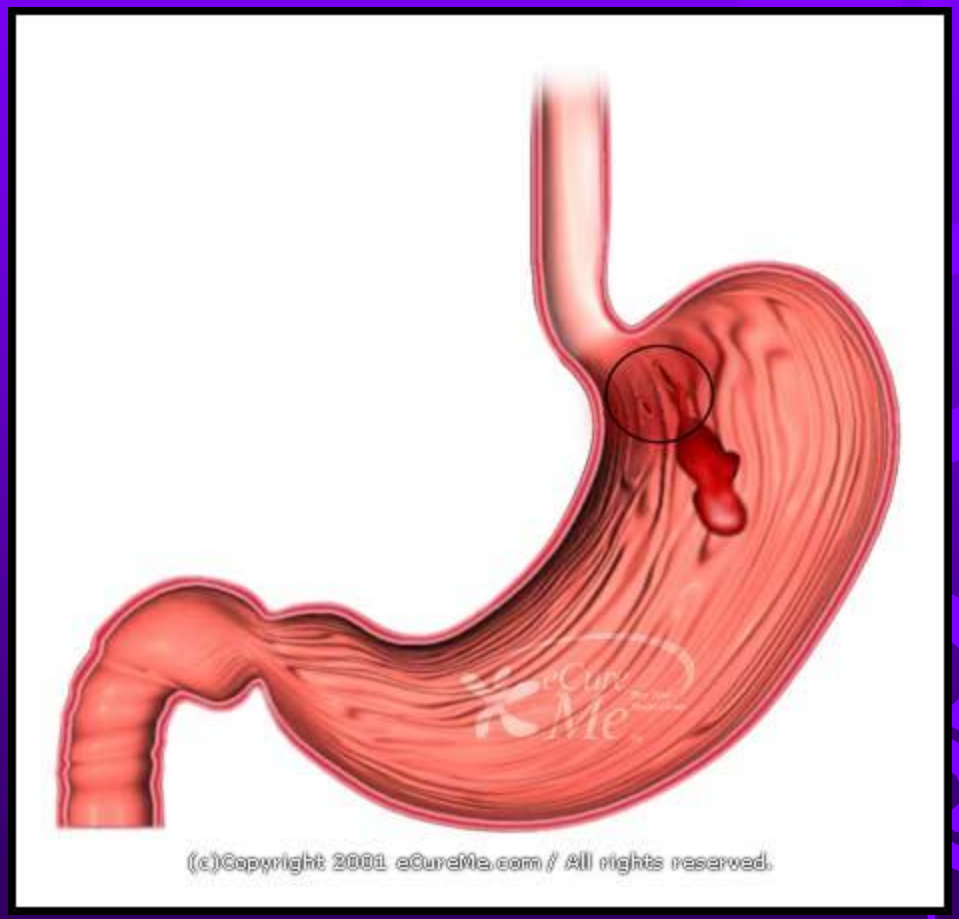
Menetrier's Disease (aka Hypertrophic Gastritis)

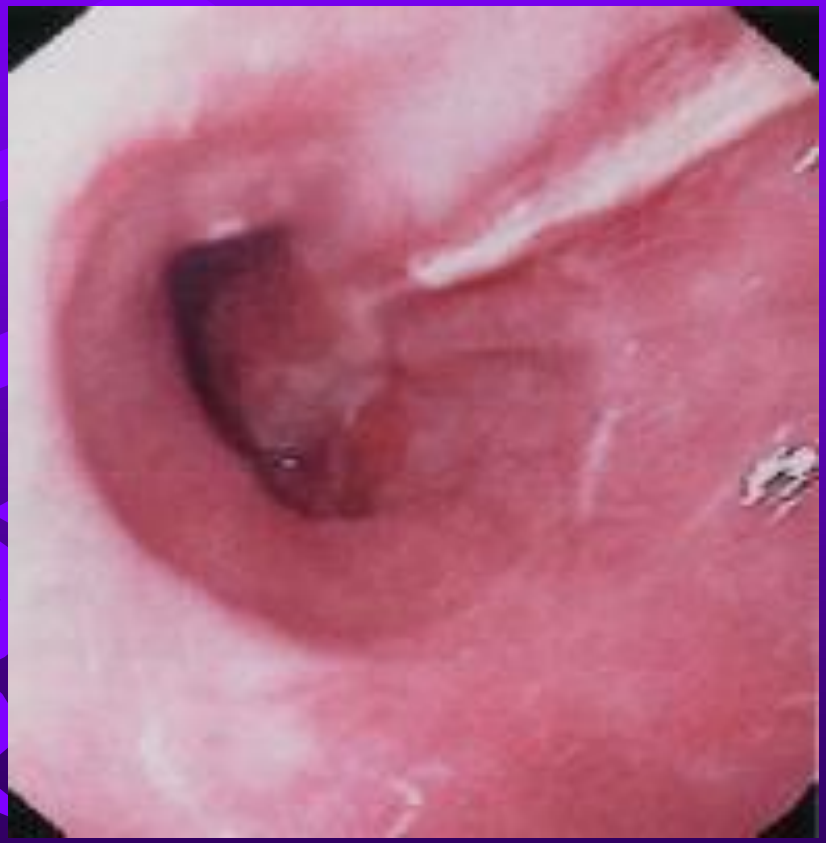
- Patient's usually present with:
  - Pain, N/V, occult hemorrhage, anorexia, wt loss and diarrhea.
- Disease progression is marked by protein-losing gastropathy.
- DX: UGI endoscopy w/ biopsy.
- Tx: typically medical, surgery is rare, Menetrier's is a risk factor for gastric CA

Mallory-Weiss Syndrome

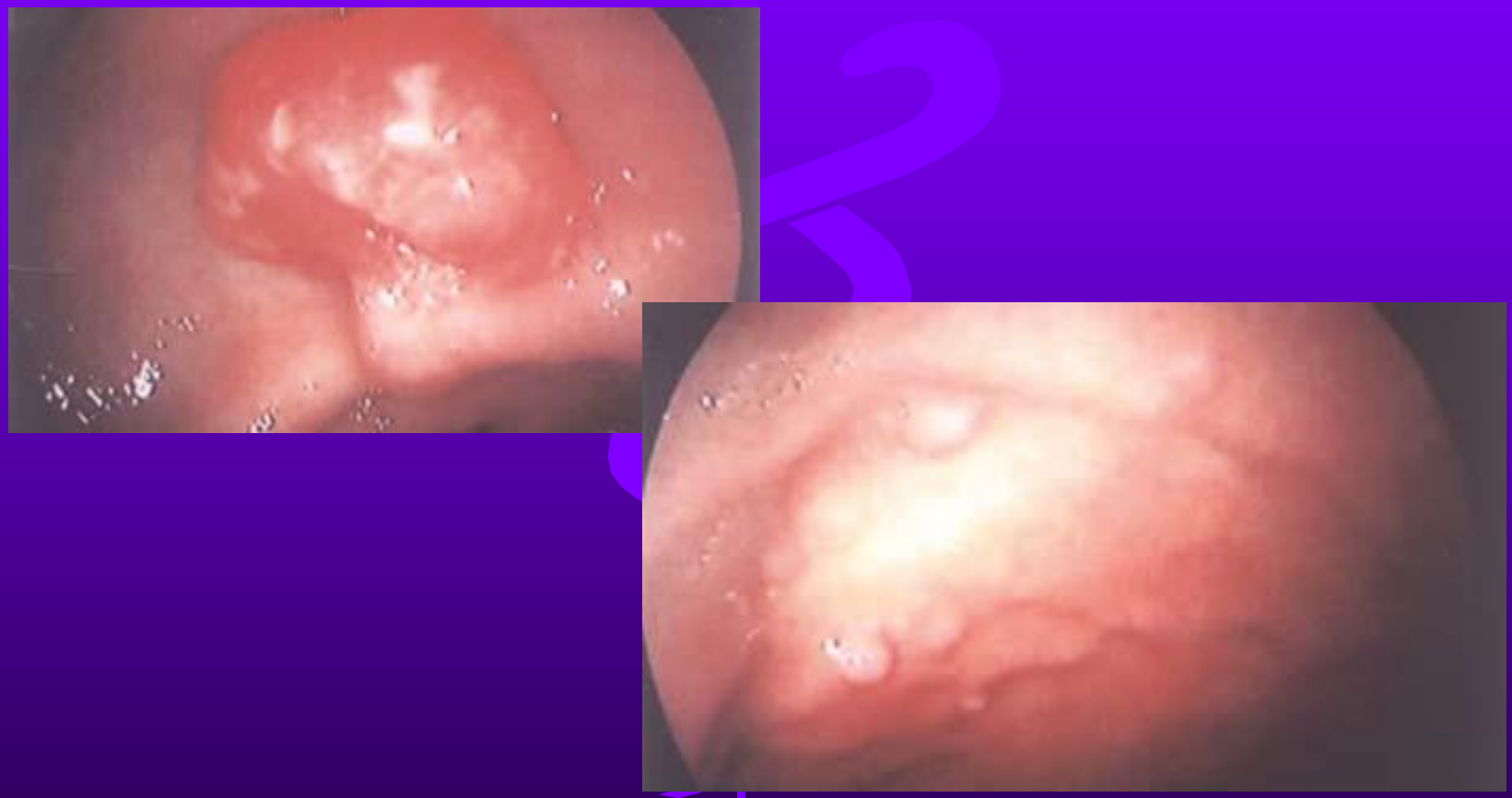
- UGI bleeding caused by linear tears at/near the GE junction.
- *Common Test Scenario:
  - Alcoholic, intense episode of emesis, now with UGI bleeding.

Mallory-Weiss Syndrome





Gastric Polyps



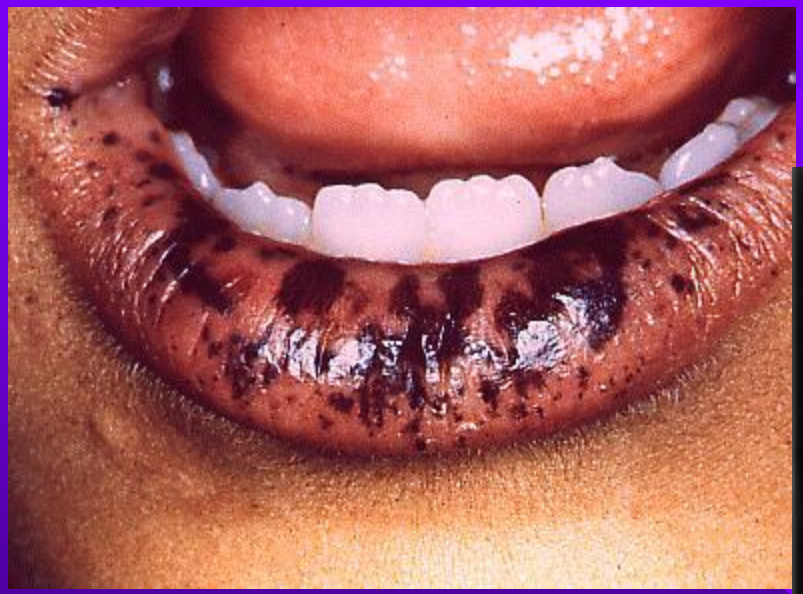
Gastric Polyps

- Rare, but frequency is increasing due to increasing numbers of UGI endoscopy.
- There are two types:
  - Hyperplastic
  - Adenomatous
- Hyperplastic polyps:
  - More common
  - Typically are benign (but can transform)

Gastric Polyps

- Adenomatous polyps:
  - Greater risk of malignancy.
  - "Size Matters"
    - < 0.5 cm in diameter = very low risk.
    - > 1.5 cm in diameter = very high risk.
- Once a polyp is diagnosed, one should evaluate for more.

Peutz-Jegher's Syndrome



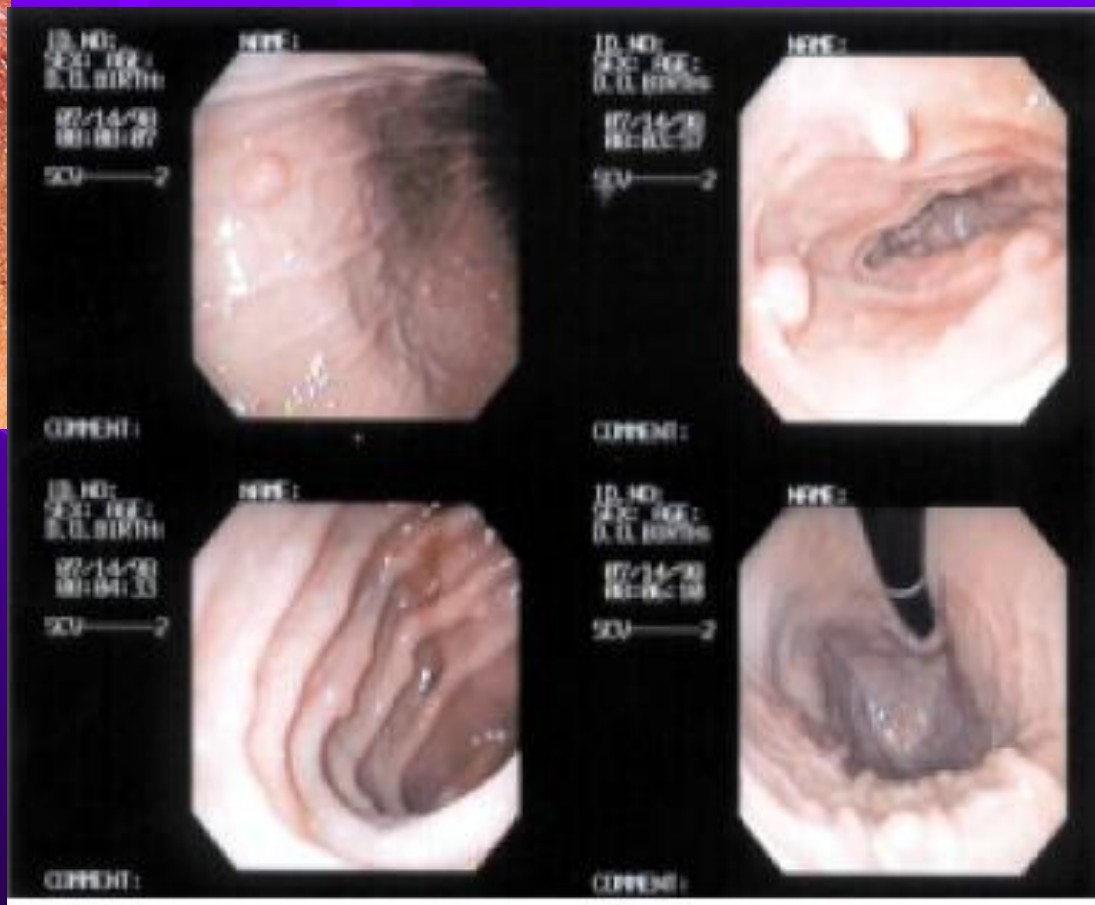
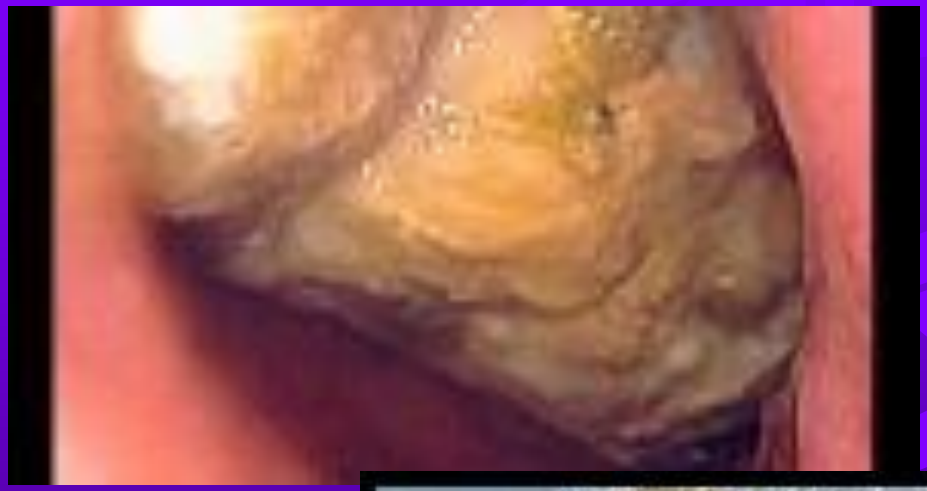


Figure 4 - Endoscopic view of sessile polyps in stomach.

Peutz-Jegher's

- Characterized by:
  - Melanous spots on the lips and buccal mucosa.
  - Multiple benign gastric and small bowel polyps.
- Autosomal dominant w/ high degree of penetrance.
- Treatment is conservative, polyps are hamartomas and are infrequently malignant.

Bezoars



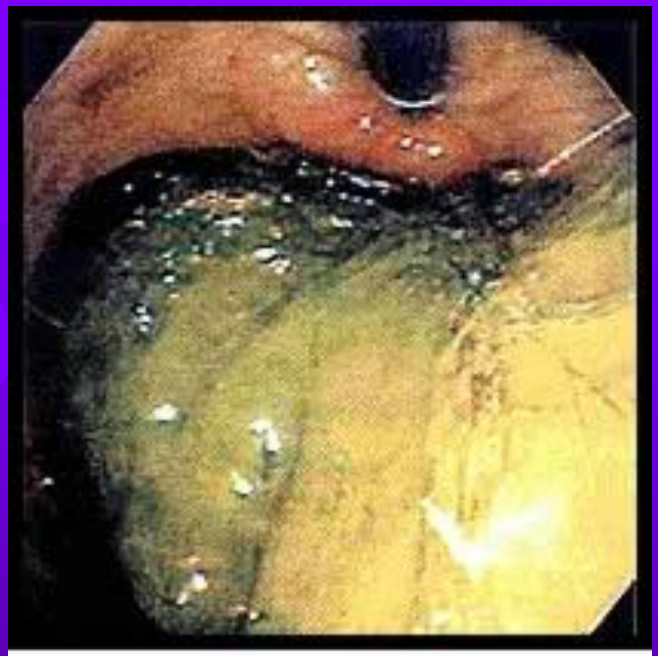


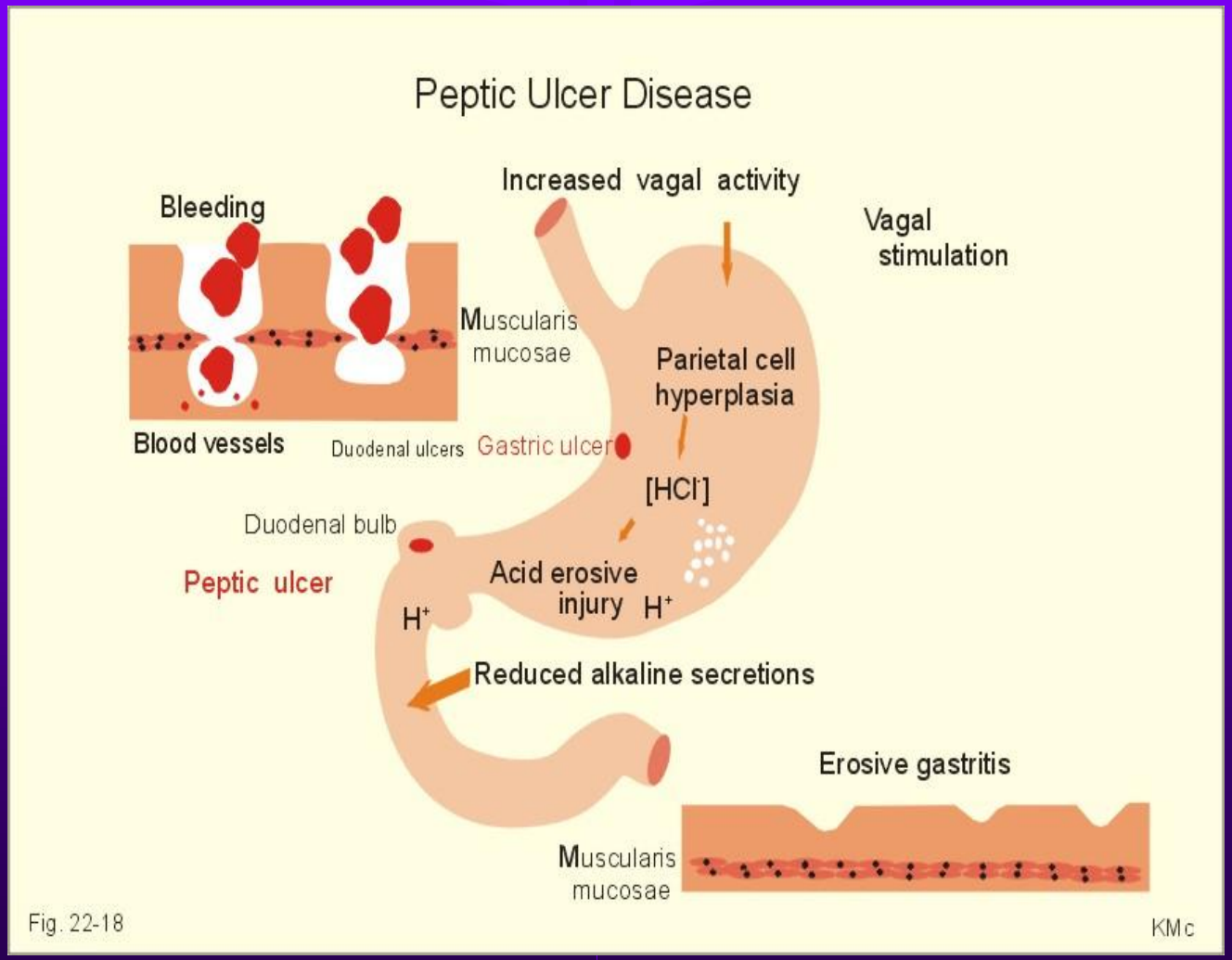
Image 5

Image 4

Bezoars

- Large masses of indigestible fiber(s) within the stomach.
- Phytobezoars = vegetable matter.
- Trichobezoars = hair.

Peptic (Gastric) Ulcer Disease



Gastric Ulcer Disease

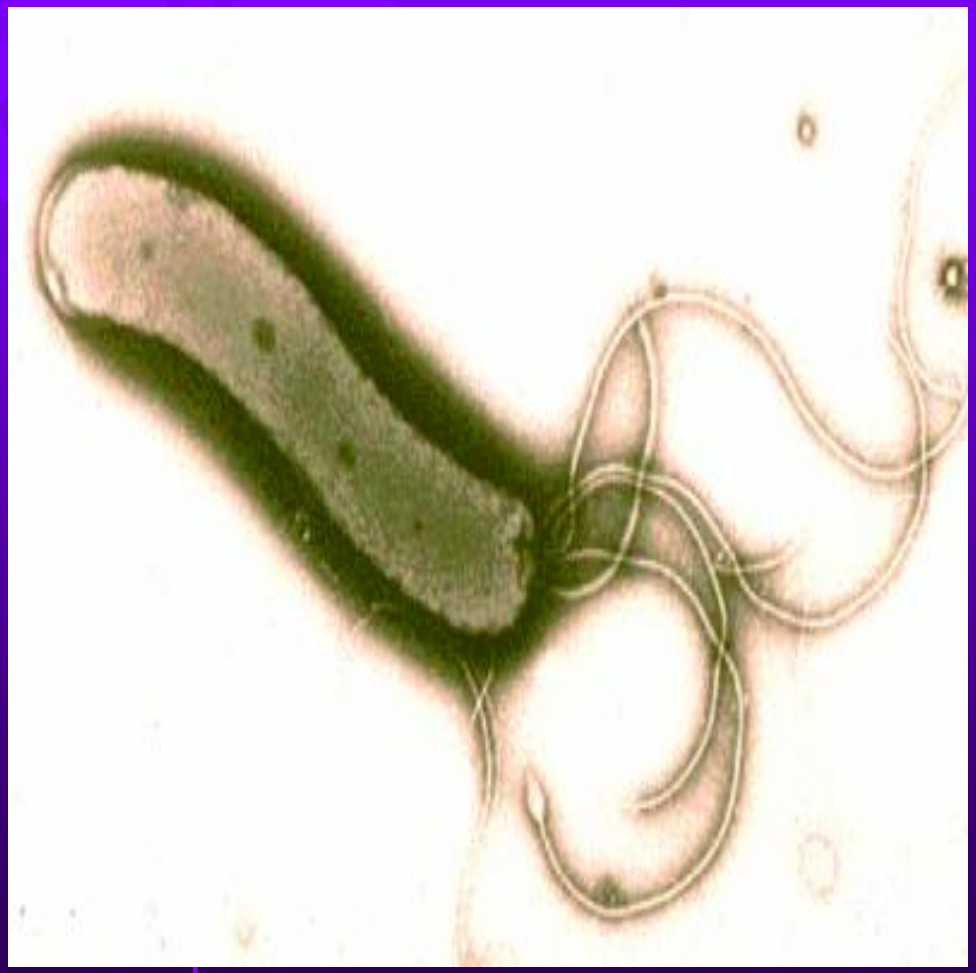
- Most common in males and elderly.
- Peak incidence: 55-65 yoa
- Approx. 90,000 new cases/year.
- Approx. 35% will have complications
- Approx. 3,000 deaths/year due to complications.
- Approx. 10% of ulcers associated w/ CA.

Gastric Ulcer Disease

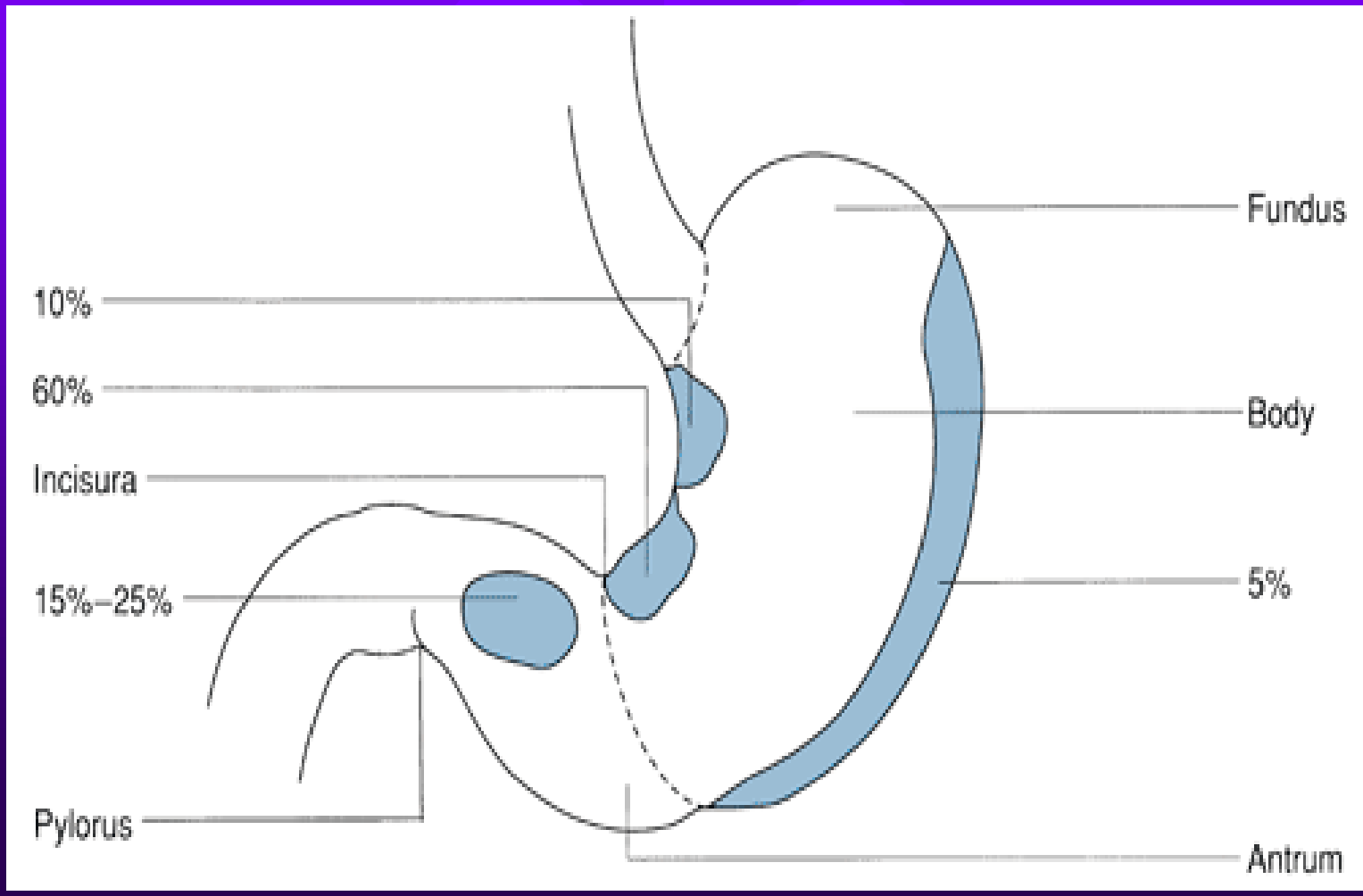
- 85%-95% of GU pts are colonized w/ H. pylori.
- Can occur anywhere in stomach.
- Most occur within 2 cm of the junction between the antral/fundic mucosa along lesser curve.
- 2/3 at incisura angularis
  - 20% distal, 10% proximal
  - Only 5% occur along greater curve.

The "Culprit"

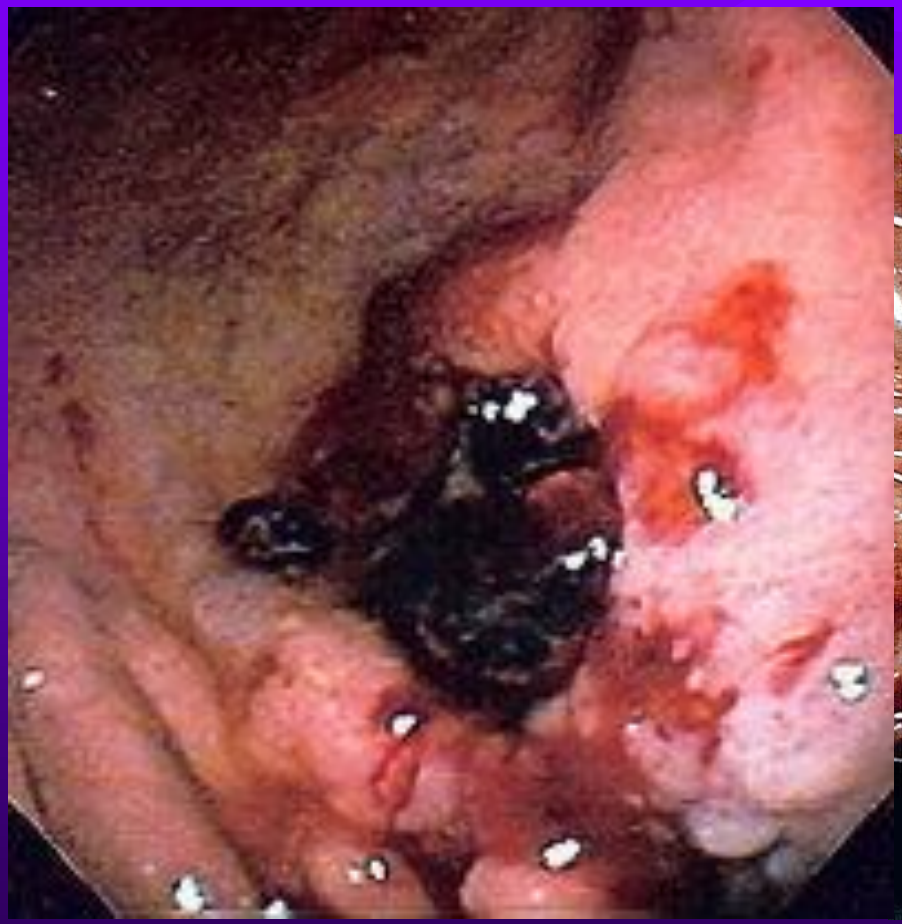
- H. pylori
- Treatment:
  - Triple therapy

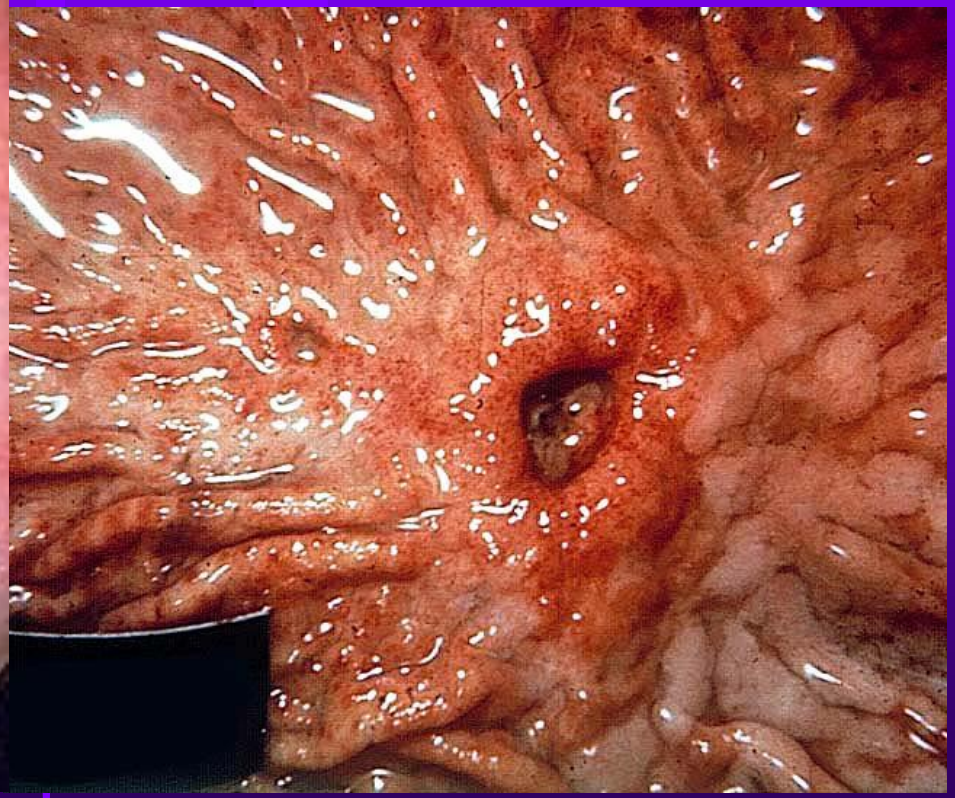


Gastric ulcers

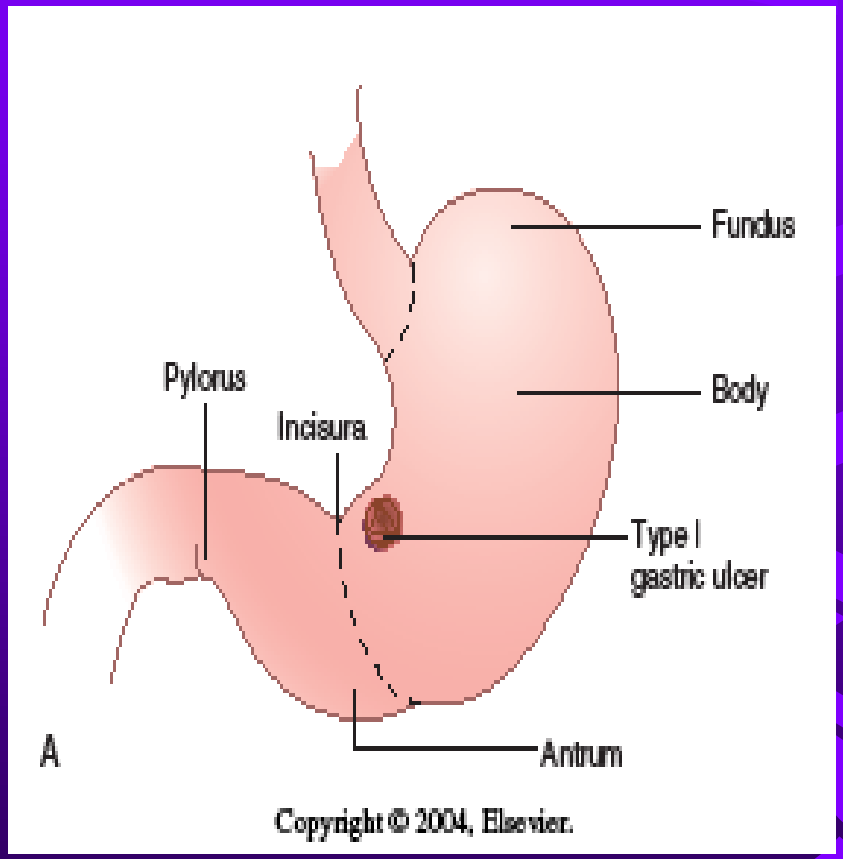


Gastric Ulcers





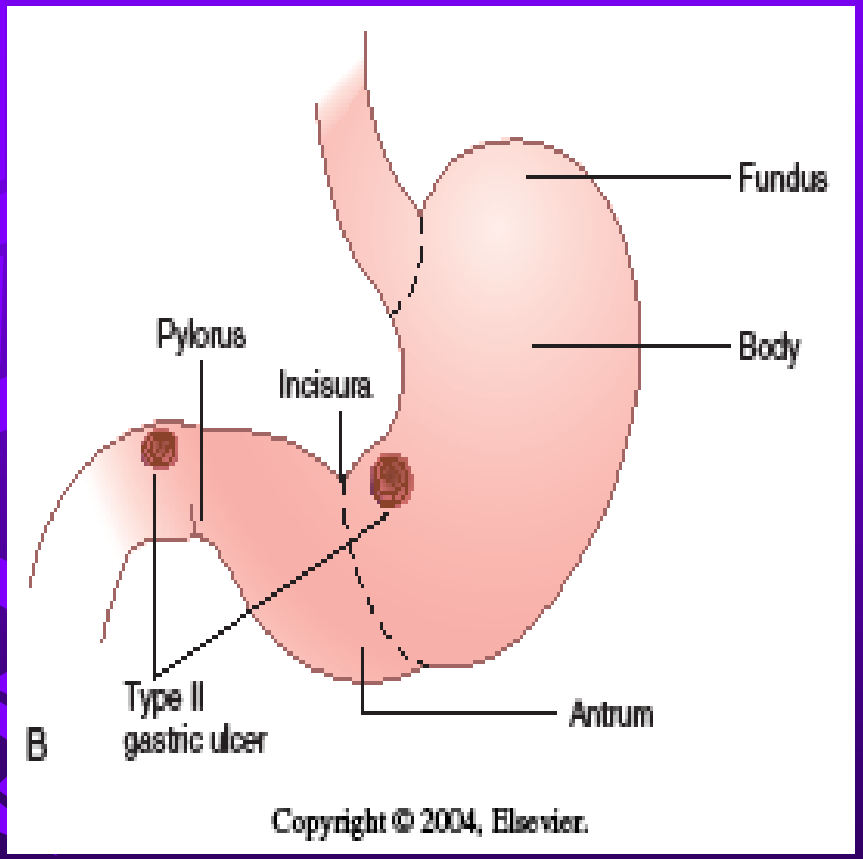
Type I



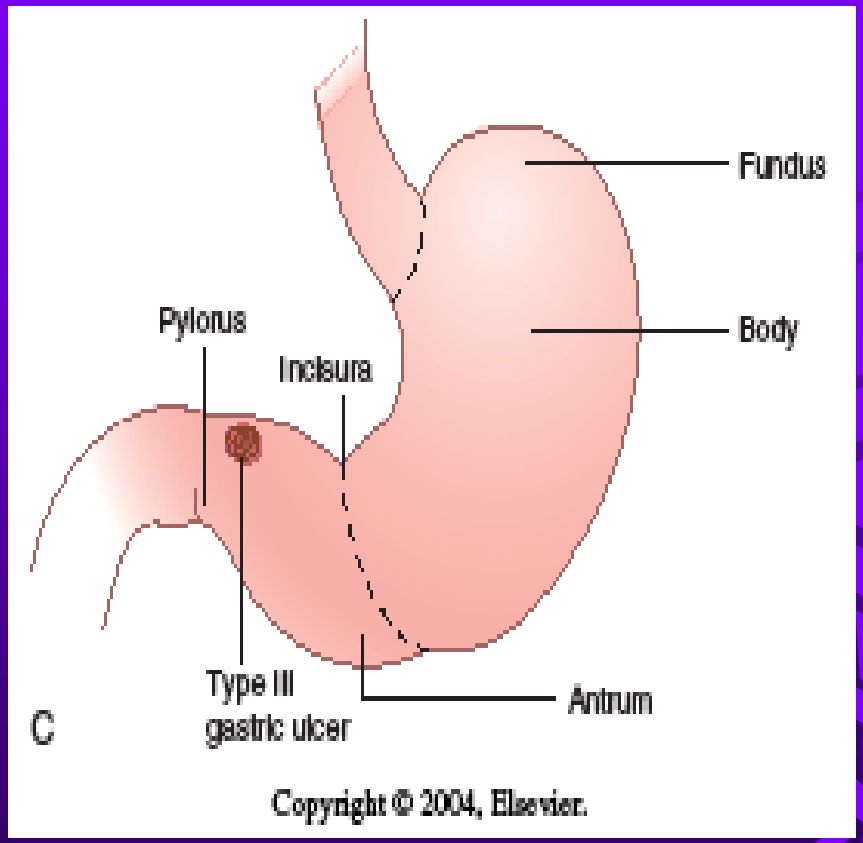
- Occur at the incisura.
- Not associated with hyperacidity, most patients have low acid output.
- Associated with ABO group "A".

Type II

- A combination of 2 ulcers, one in the body of the stomach, the other in the duodenum.
- Usually occur in hypersecretory states.
- Associated with ABO "O".



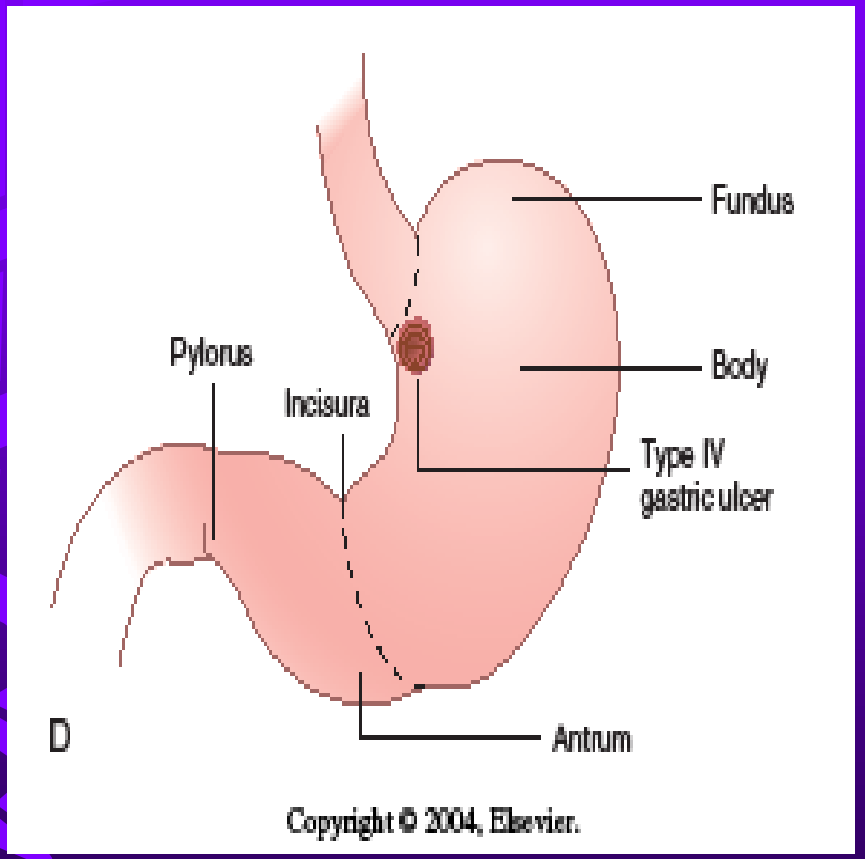
Type III



- Located pre-pyloric.
- Associated with hypersecretion.
- Type "O" blood association.
- Typically 2-3 cm from pylorus, can be multi.

Type IV

- Csendes type ulcers.
- Occur high on lesser curve at/near the GE junction.
- Not associated with hypersecretion.
- Usually result from defective mucosal defense.



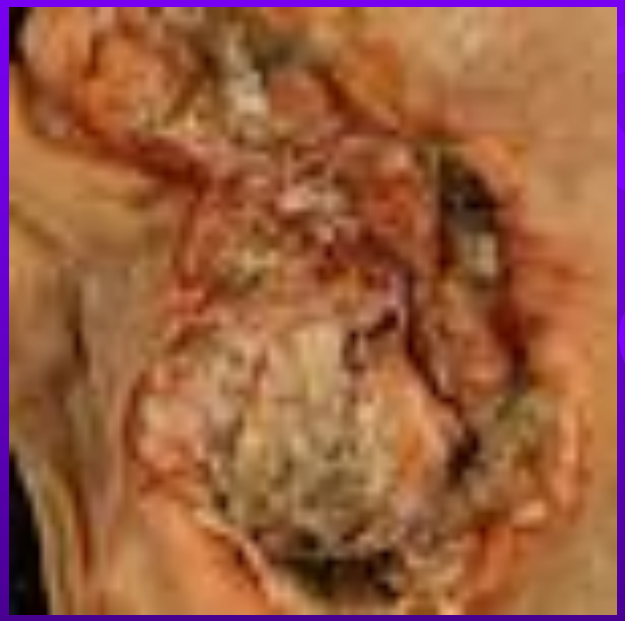
Type V

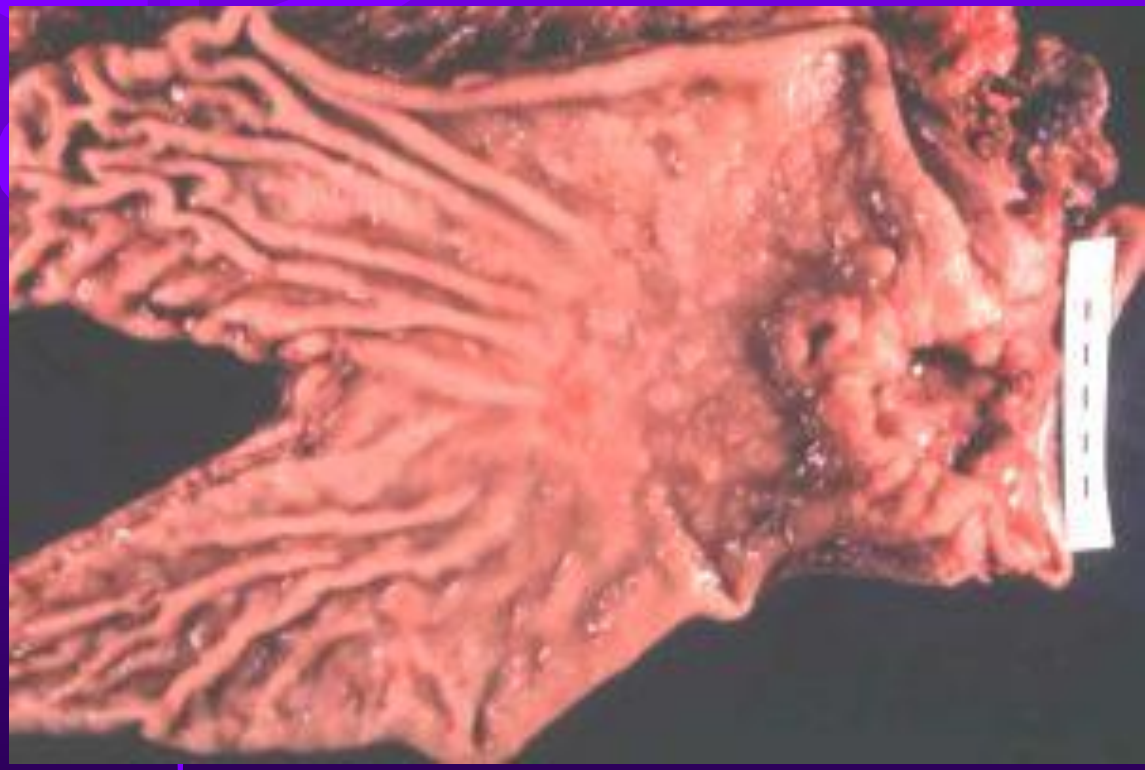
- Can occur anywhere in stomach.
- Result from chronic ASA/NSAID ingestion.

Malignant Gastric Disease

- Adenocarcinoma
- Lymphoma
- Gastrointestinal Stromal Tumor (GIST)

Gastric Adenocarcinoma





Gastric Adenocarcinoma

- Adenocarcinoma accounts for 95% of all gastric cancers.
- Worldwide is the leading cause of cancer death.
- US and Europe = low risk areas
- Asia (Japan/China), Russia, Chile = high risk areas.

Risk Factors

- H. pylori infection.
- Pernicious anemia.
- Achlorhydria.
- Chronic gastritis.
- H/o caustic injury.
- Presence of adenomatous polyps.

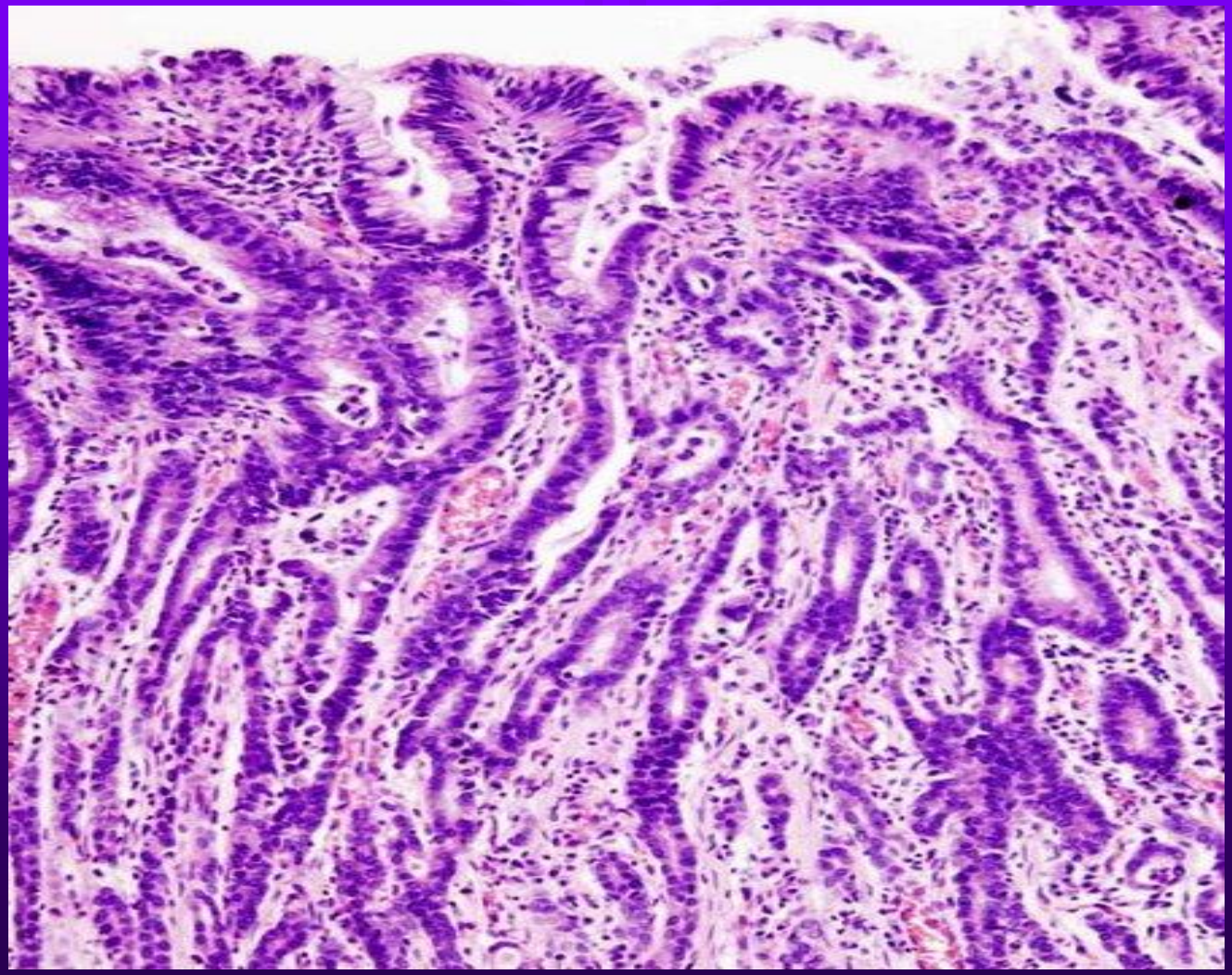
Classification

- In the US, there are 4 types:
  - Ulcerative, polypoid, scirrhous, superficial spreading.
  - Ulcerative is the most common.
- 2 distinct histologic types:
  - Intestinal
  - Diffuse

Intestinal

- Well differentiated with glandular elements.
- More common form in high incidence areas.
- Patients are usually older.
- Spread is hematogenous.

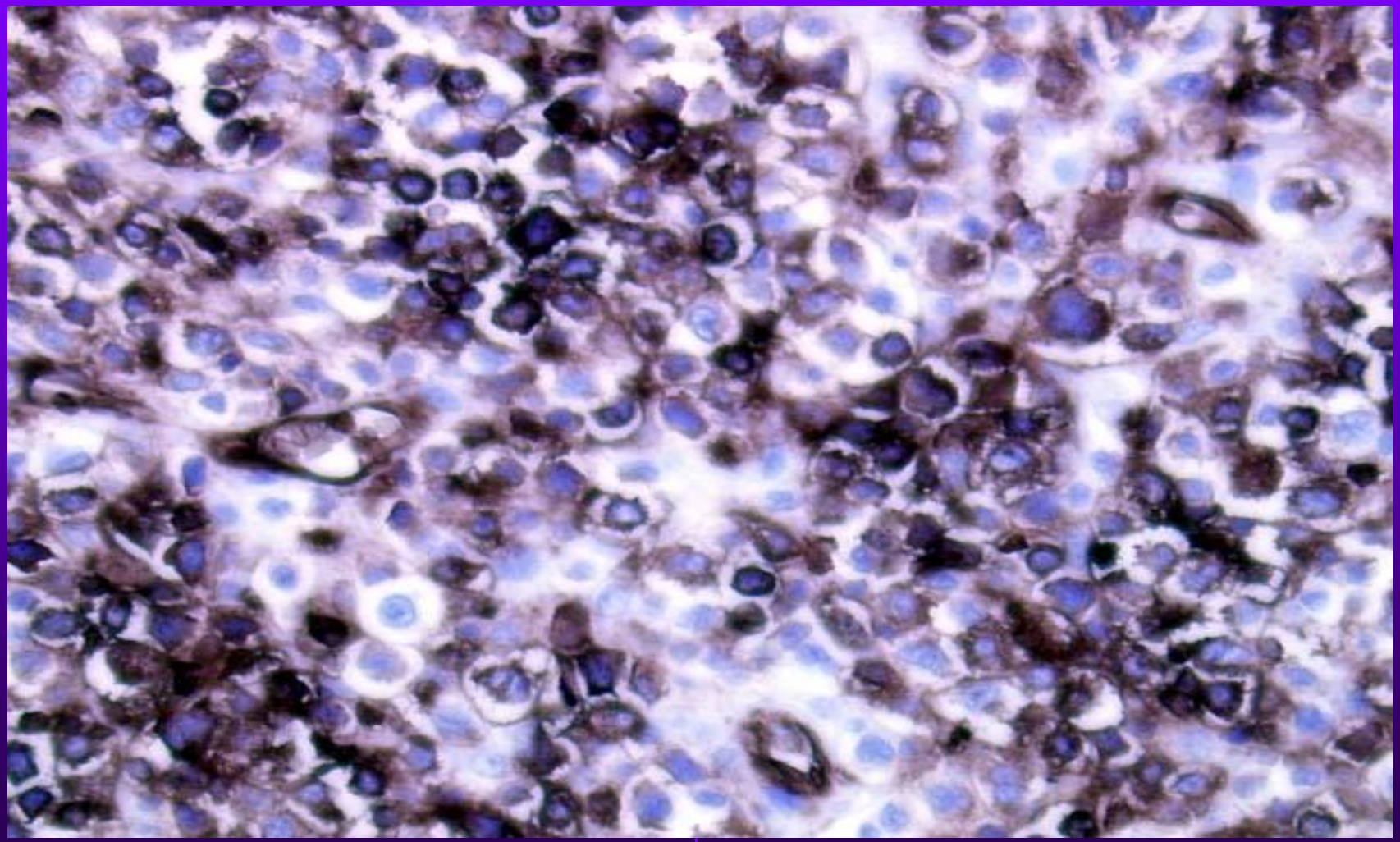
Intestinal



Diffuse

- Poorly differentiated with classic signet-ring cells.
- Patients are usually younger.
- Associated with ABO blood group “A”.
- Spreads via lymphatics and local extension.

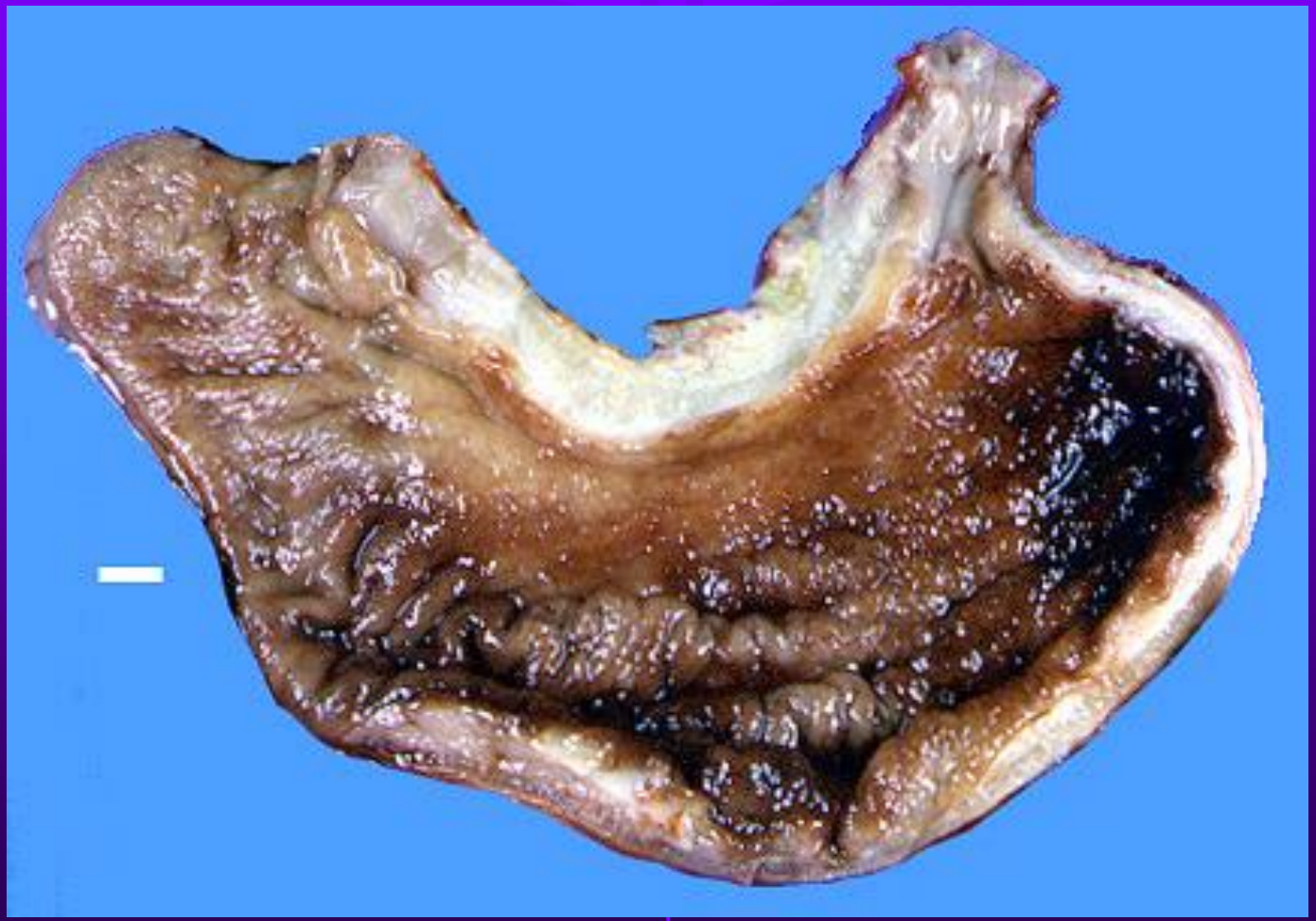
Diffuse



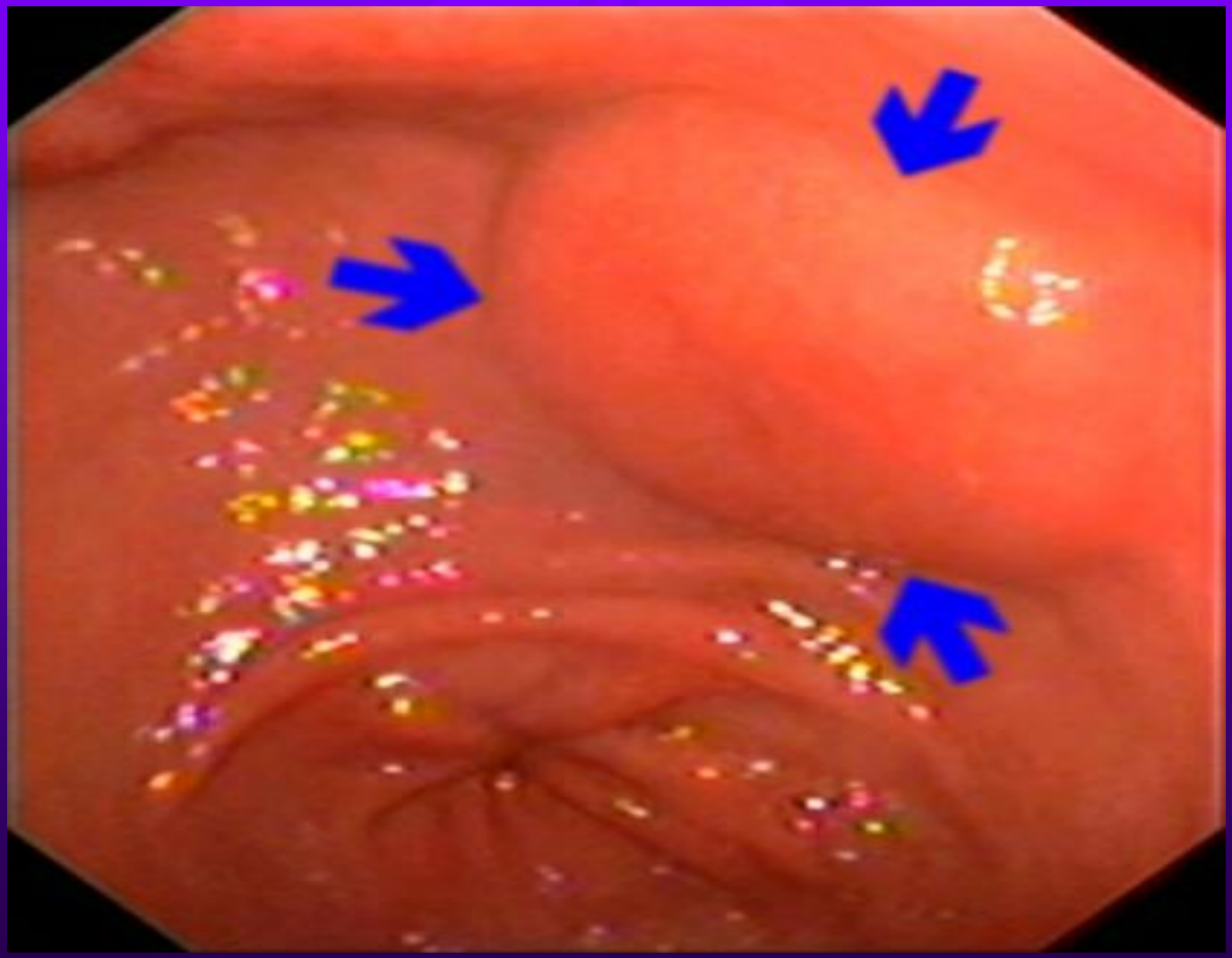
Gastric Adenocarcinoma

- Linitus plastica:
  - Complete gastric infiltration with carcinoma.
  - Has “leathery” appearance.
  - Extremely poor prognosis.
- Evaluation.
- Treatment.

Linitus Plastica



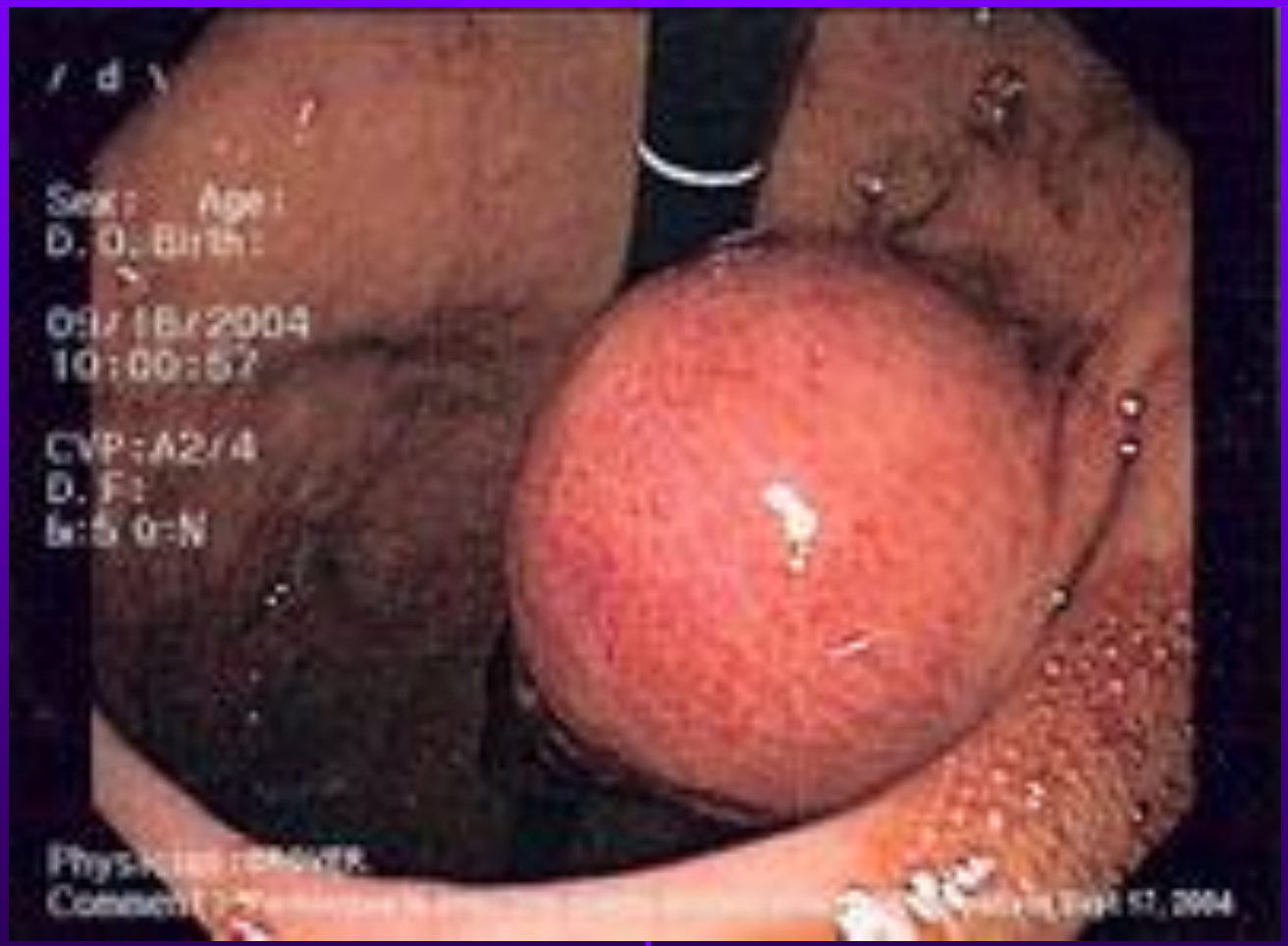
Gastric Lymphoma



Gastric Lymphoma

- Stomach is the primary site for ~2/3 of all GI lymphomas.
- Patients tend to be older.
- Predominately non-Hodgkin's variant.
- Symptoms are similar to Adeno-ca.
- Dx: via biopsy (endoscopic vs. open).
- If Dx is made prior to surgery, do lymphoma work-up.
- Treatment: chemotherapy vs. surgery.

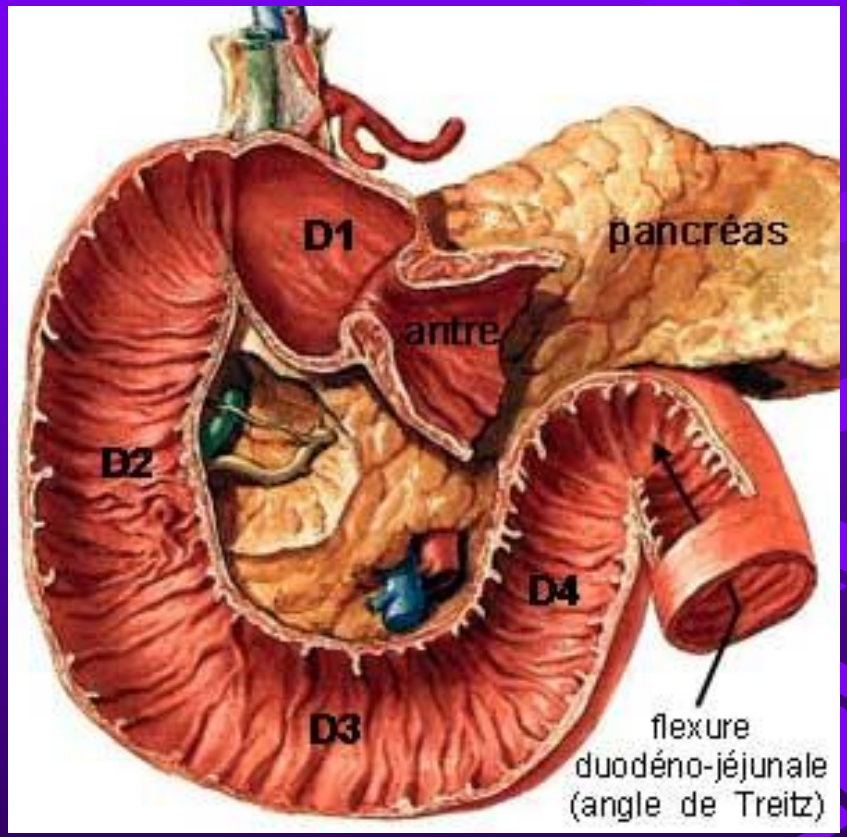
GIST



GIST

- Formerly (incorrectly) called leiomyomas or leiomyosarcomas.
- Stomach is the most common site.
- Can be “benign” or “malignant”.
  - Malignant needs direct invasion.
  - Must count mitotic figures (>10 per 50 fields = malignant).
- Work-up.
- Treatment:
  - Surgical Resection w/ margins.
  - Gleevec

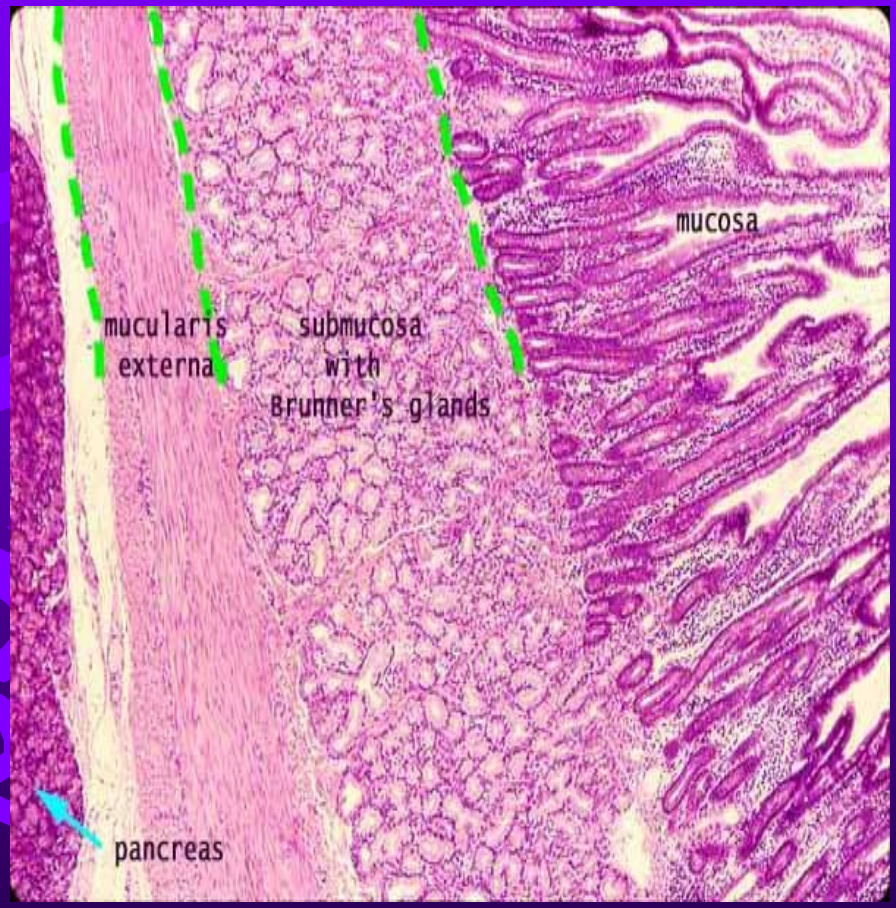
Duodenum



- 4 parts
- Metabolically active
  - Produces many enzymes
- D2: site of pacemaker
- D2: posterolateral insertion of ampulla.
- Becomes jejunum at the _____?

Duodenum

- Brunner's glands
- Blood supply:
  - GDA- superior pancreaticoduodenal
  - SMA- inferior pancreaticoduodenal



Benign Duodenal Disease

- Duodenal Ulcer Disease
  - Uncomplicated
  - Complicated
- Duodenal Polyps

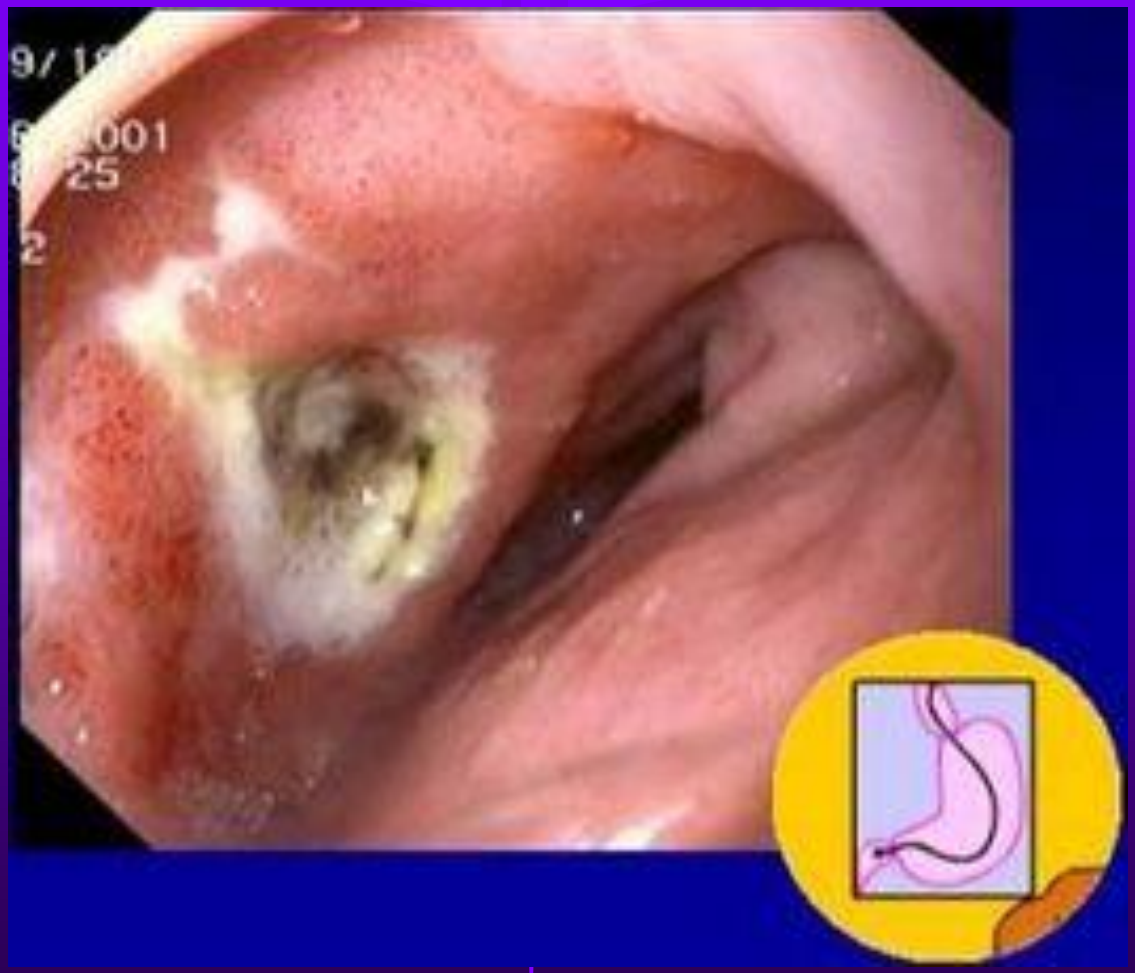
Uncomplicated Ulcer Dz.

- PUD affects:
  - ~500,000 new cases yearly.
  - ~2,000,000 active adult cases in US.
- Most are duodenal ulcers.
- Typically these form in the bulb.
- In contrast to gastric ulcers, duodenal ulcers rarely harbor malignancy.

Uncomplicated Ulcer Dz.

- Important risk factors:
  - H. pylori, NSAIDs, (?) tobacco.
- Most duodenal ulcer disease is uncomplicated.
- Treatment has shifted to mainly medical instead of surgical.

Duodenal Ulcers



Presentation

- Both types:
  - Burning, gnawing pain in the epigastrium.
  - Can radiate to the back.
  - Pain usually occurs 1-3 hours after eating.
  - Exacerbated by fasting.
  - Relief gained from OTC meds and eating.
  - THESE CAN BLEED.

Evaluation/Testing

- (+) symptoms = non-invasive H. pylori testing.
  - Serum ab, breath urease, fecal antigen.
- (+) test = H. pylori treatment.
  - Triple therapy.
- If primary treatment fails then do upper endoscopy.
  - UE allows for visualization, biopsy, eval for alternate Dx.

Treatment

- Non-operative.
- Aim/Goal: promote healing of ulcer(s), prevent recurrence of ulcer(s).
  - Remove all ulcerogenic agents.
  - Start acid suppression.
  - Eradicate H. pylori.

Treatment

- H. pylori therapy:
- First Line:
  - Amoxicillin and Clarithromycin –or-
  - Clarithromycin and Flagyl (+)
  - PPI (or H_2 blocker).
- Treat for 7 – 14 days.
- Recheck for H. pylori after treatment.
- Continue acid supression until ulcer is healed.

Complicated Ulcer Dz.

- 4 major manifestations:
  - Perforation
  - Bleeding
  - Gastric outlet obstruction
  - Intractable

Evaluation

- Same as uncomplicated unless one of the four “bad things” is present.

Treatment: Perforation

- Typical presentation.
- Resuscitation.
- Operation:
  - Graham patch vs. Modified Graham patch.
  - +/- antrectomy/drainage (vagotomy).

Treatment: Bleeding

- Resuscitation.
- Upper endoscopy w/ local treatment.
  - (-) factors: active bleeding, visible vessel, fresh clot.
- (?) angiography w/ embolization.
- Surgical intervention.

Treatment: Outlet obstruction

- Decompression, NPO, rehydration, correct lytes.
- Some resolve (swelling).
- Surgery:
  - Remove obstruction.
  - Bypass obstruction.
- Antrectomy w/ drainage.
- Gastroenterostomy.

Treatment: Intractable dz.

- Medical failure.
- Need to r/o other issues:
  - ZE syndrome, non-compliance, etc.
- Surgery.

Duodenal polyps

- Typically arise as part of a familial disorder.
  - FAP.
    - Autosomal dominant
    - High malignant potential
  - Peutz-Jegher's syndrome.

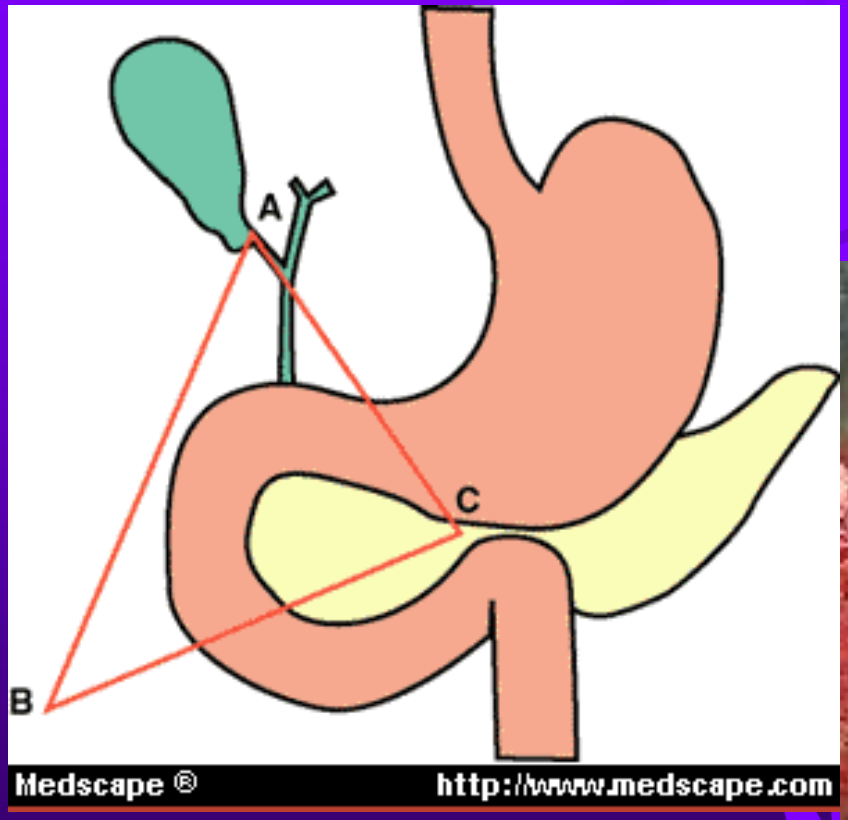
Malignant Duodenal Disease

- Zolliger-Ellison (ZE) Syndrome
- Adenocarcinoma
- Lymphoma

Z-E Syndrome

- Rare disease.
- Most well-known endocrine tumor disorder.
- Disease is a direct result of gastrinoma.
- 2/3 of gastrinomas are in the "triangle"
  - CBD/D2-D3/neck of Pancreas.
- Can be sporadic or hereditary.
  - Strong assoc. with MEN-I (3 p's)
- Treatment is resection (if possible).

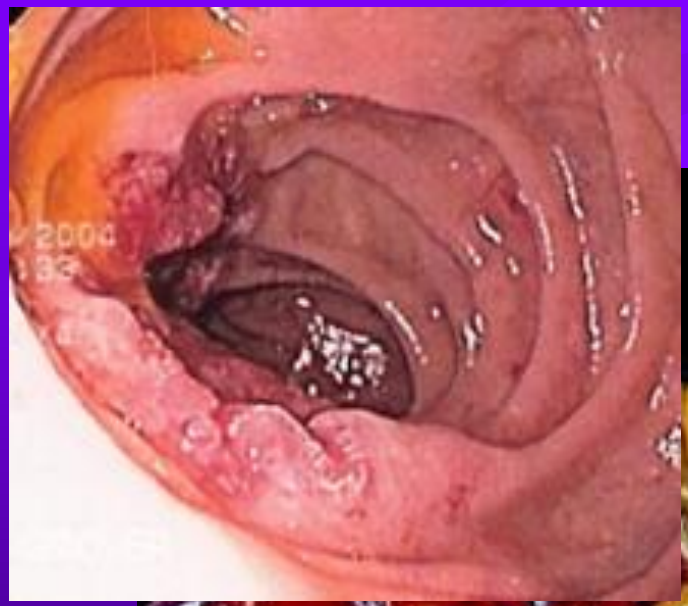
Gastrinoma



Adenocarcinoma

- Duodenum is the most common site for small bowel adenocarcinoma.
- ~ 2/3 are peri-ampullary (D2).
- Very rare, patients tend to present late in disease course.
- Only treatment is resection (if possible).
- Double bypass is unresectable.
- (+) LN's = < 15%, 5 year survival.

Adenocarcinoma



Lymphoma

- Rare, most small bowel lymphomas are ileal.
- Similar presentation to adeno CA.
- Diagnose the same as gastric lymphoma.
- Resect if resectable.
- Chemotherapy and radiation.

Lymphoma