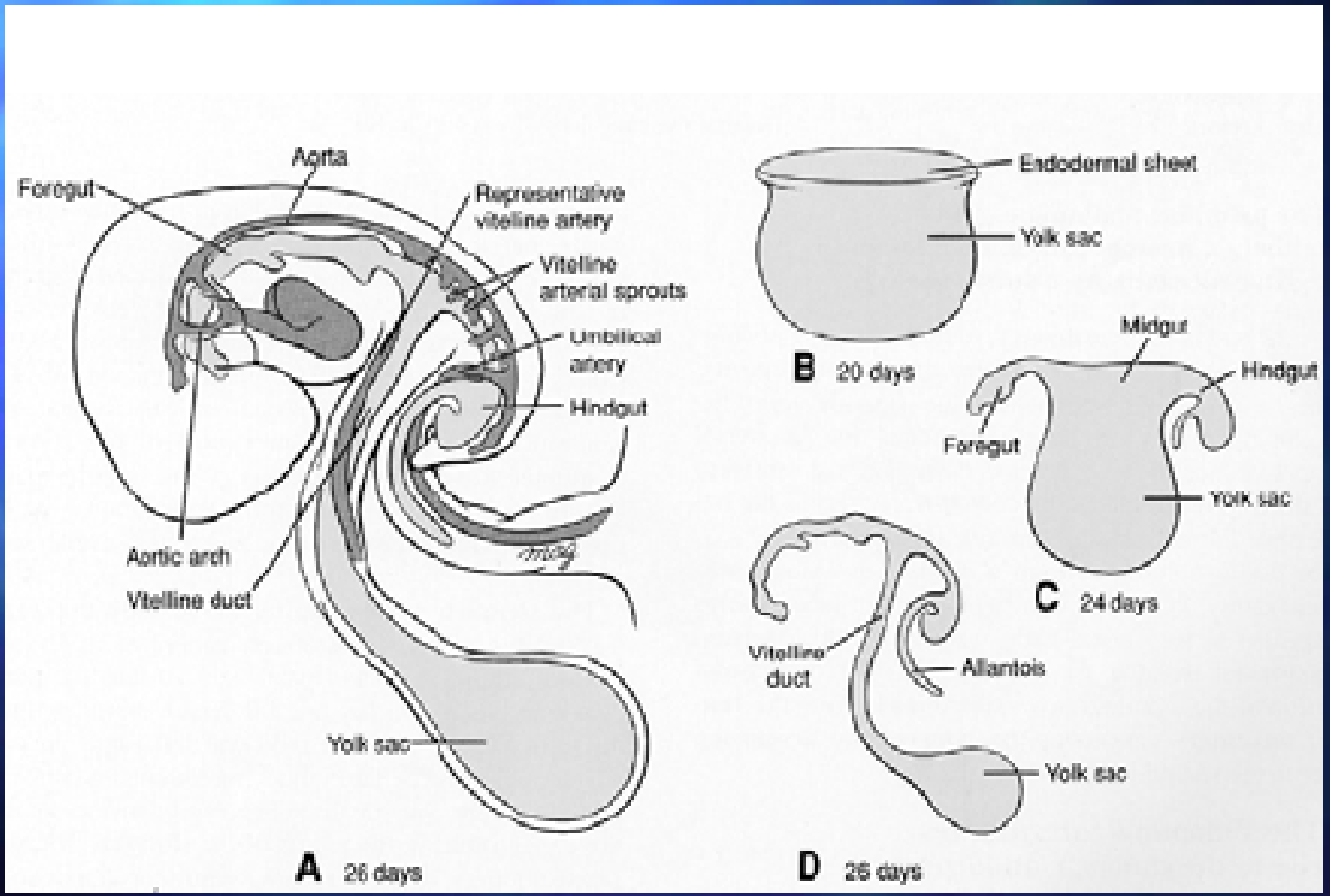
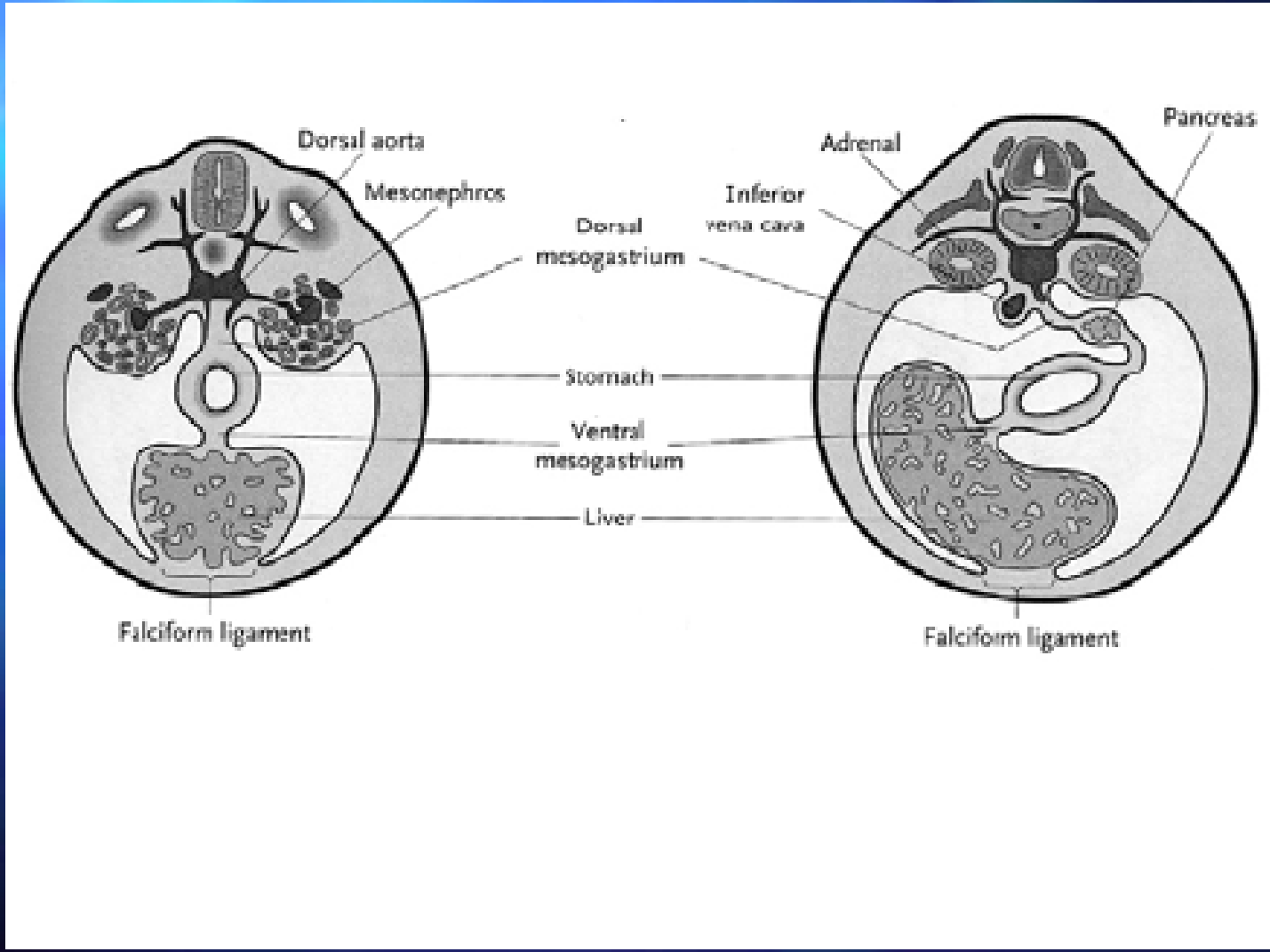
Stomach

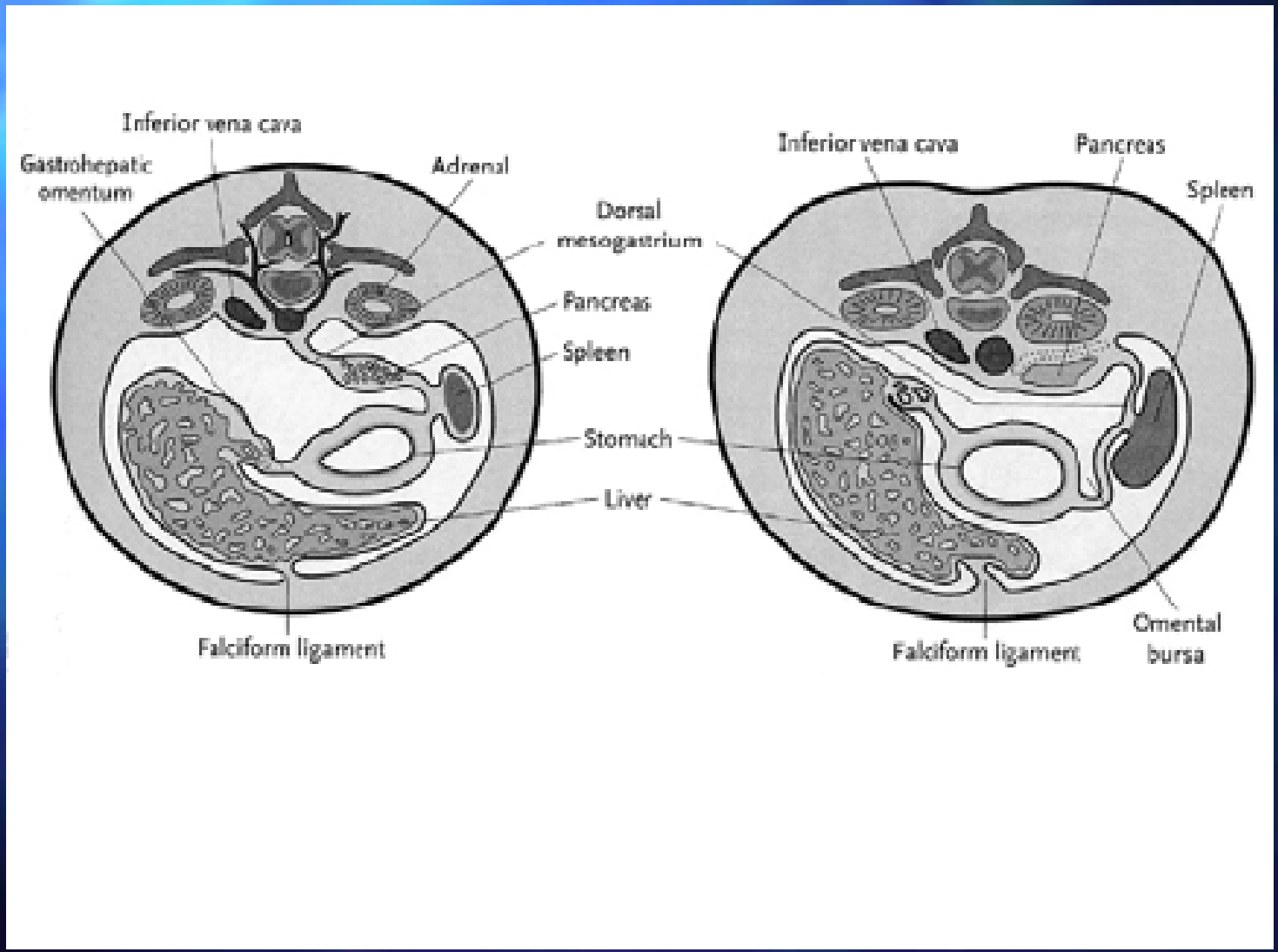
Embryology


Aorta
Foregut
Representative vitelline artery
Vitelline arterial sprouts
Umbilical artery
Hindgut
Aortic arch
Vitelline duct
Yolk sac
A 26 days
Endodermal sheet
Yolk sac
B 20 days
Midgut
Hindgut
Foregut
Yolk sac
C 24 days
Vitelline duct
Allantois
Yolk sac
D 26 days

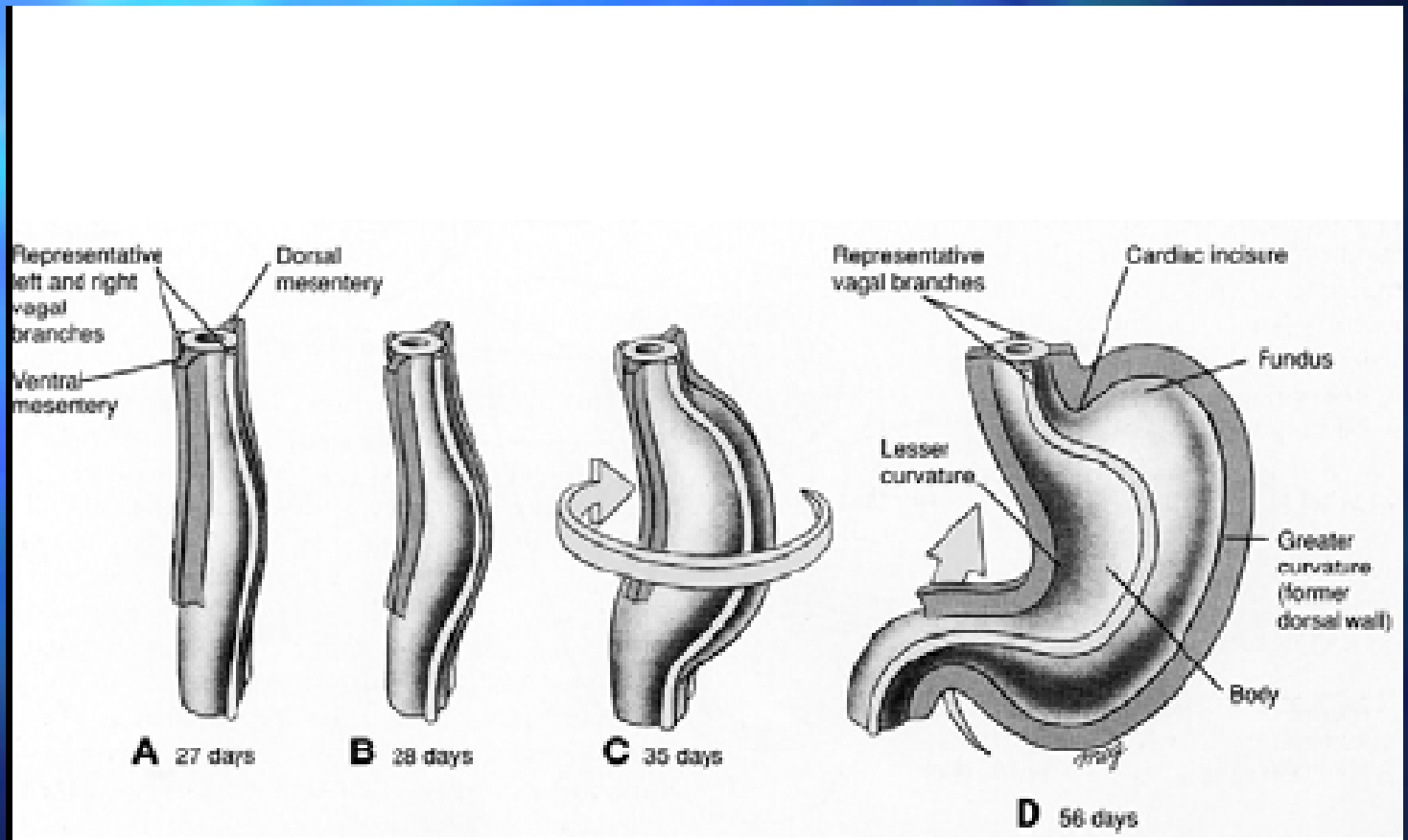
Embryology


Dorsal aorta
Mesonephros
Dorsal mesogastrium
Stomach
Ventral mesogastrium
Liver
Falciform ligament
Adrenal
Inferior vena cava
Pancreas
Falciform ligament

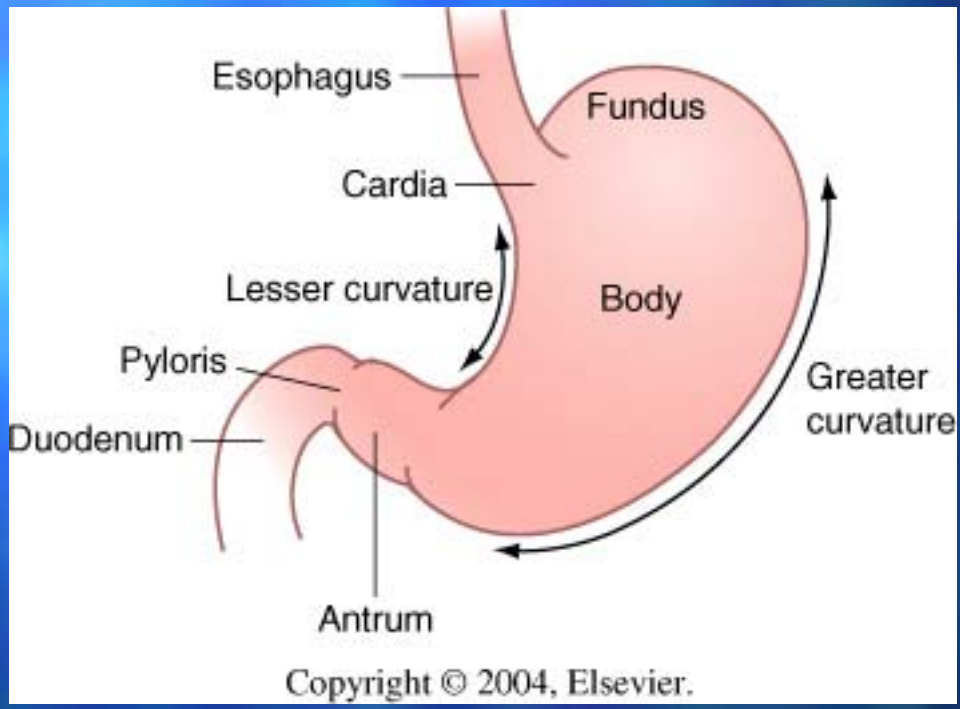
Embryology


Inferior vena cava
Gastrohepatic omentum
Adrenal
Dorsal mesogastrium
Pancreas
Spleen
Stomach
Liver
Falciform ligament
Inferior vena cava
Pancreas
Spleen
Falciform ligament
Omental bursa

Embryology


Representative left and right vagal branches
Dorsal mesentery
Ventral mesentery
A 27 days
B 28 days
C 35 days
Representative vagal branches
Cardiac incisure
Fundus
Lesser curvature
Greater curvature (former dorsal wall)
Body
D 56 days

Anatomy


Esophagus
Fundus
Cardia
Lesser curvature
Body
Pyloris
Greater curvature
Duodenum
Antrum
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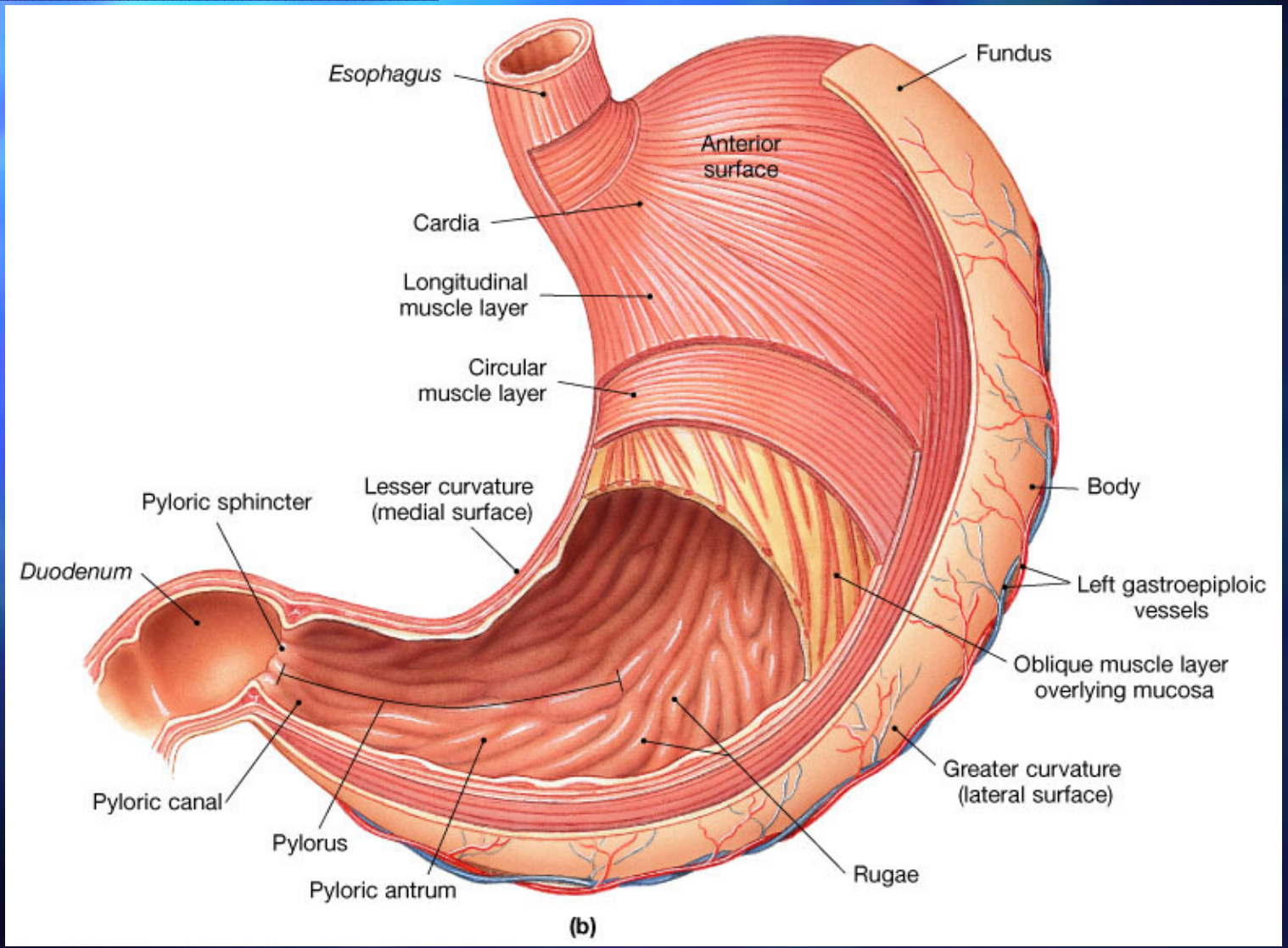
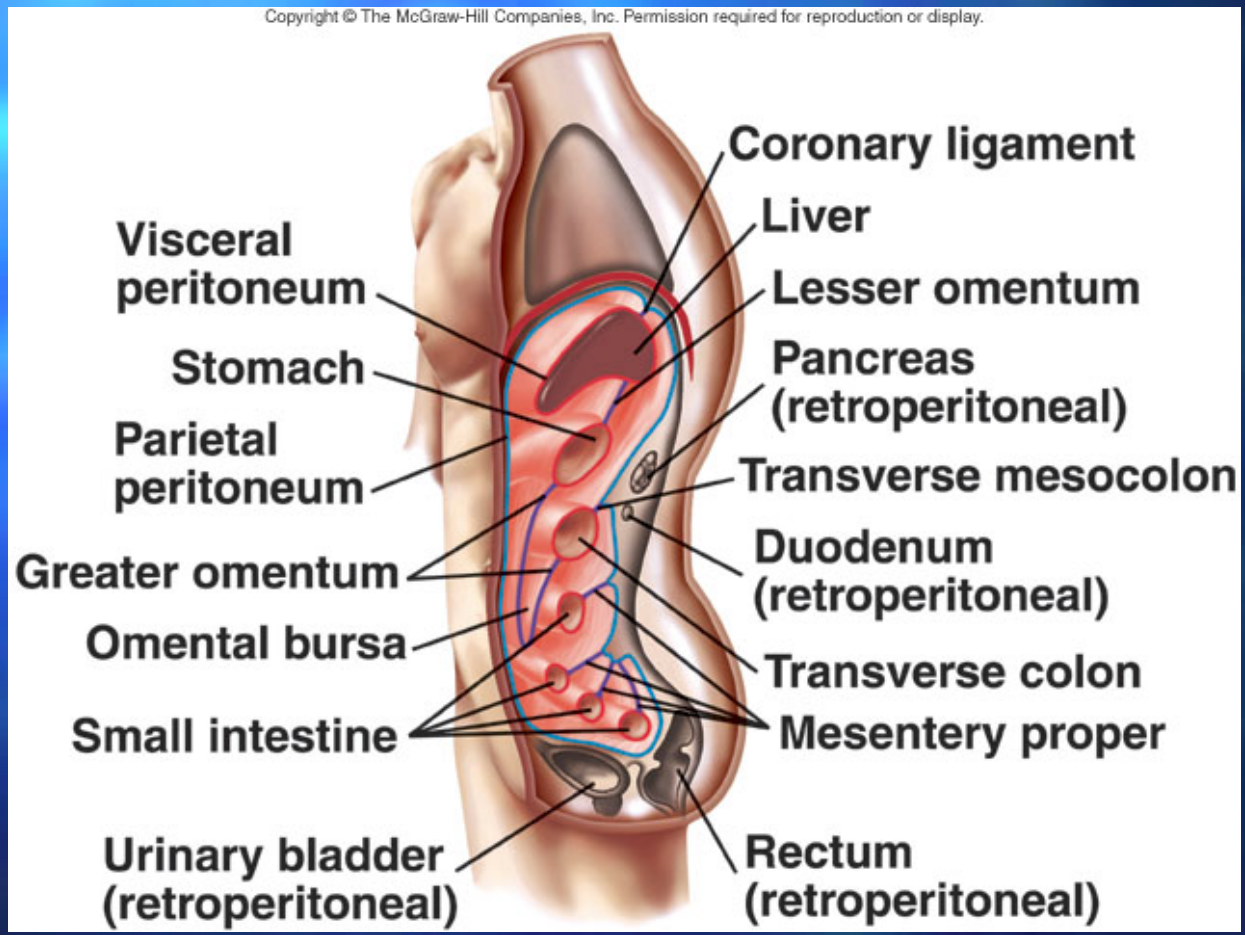
Anatomy


Esophagus
Fundus
Anterior surface
Cardia
Longitudinal muscle layer
Circular muscle layer
Lesser curvature (medial surface)
Pyloric sphincter
Duodenum
Body
Left gastroepiploic vessels
Oblique muscle layer overlying mucosa
Greater curvature (lateral surface)
Pyloric canal
Pylorus
Pyloric antrum
Rugae
(b)

Anatomy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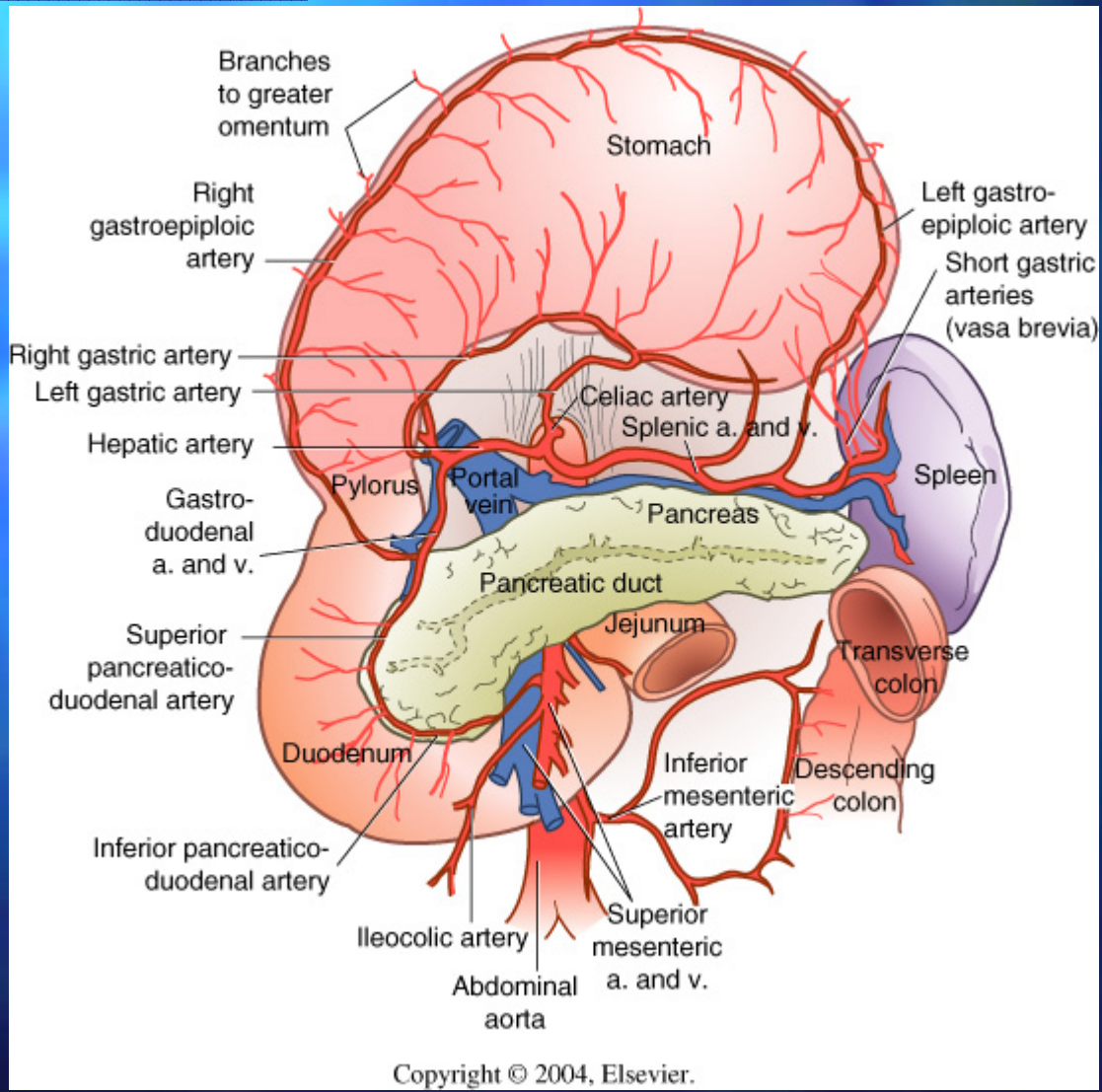
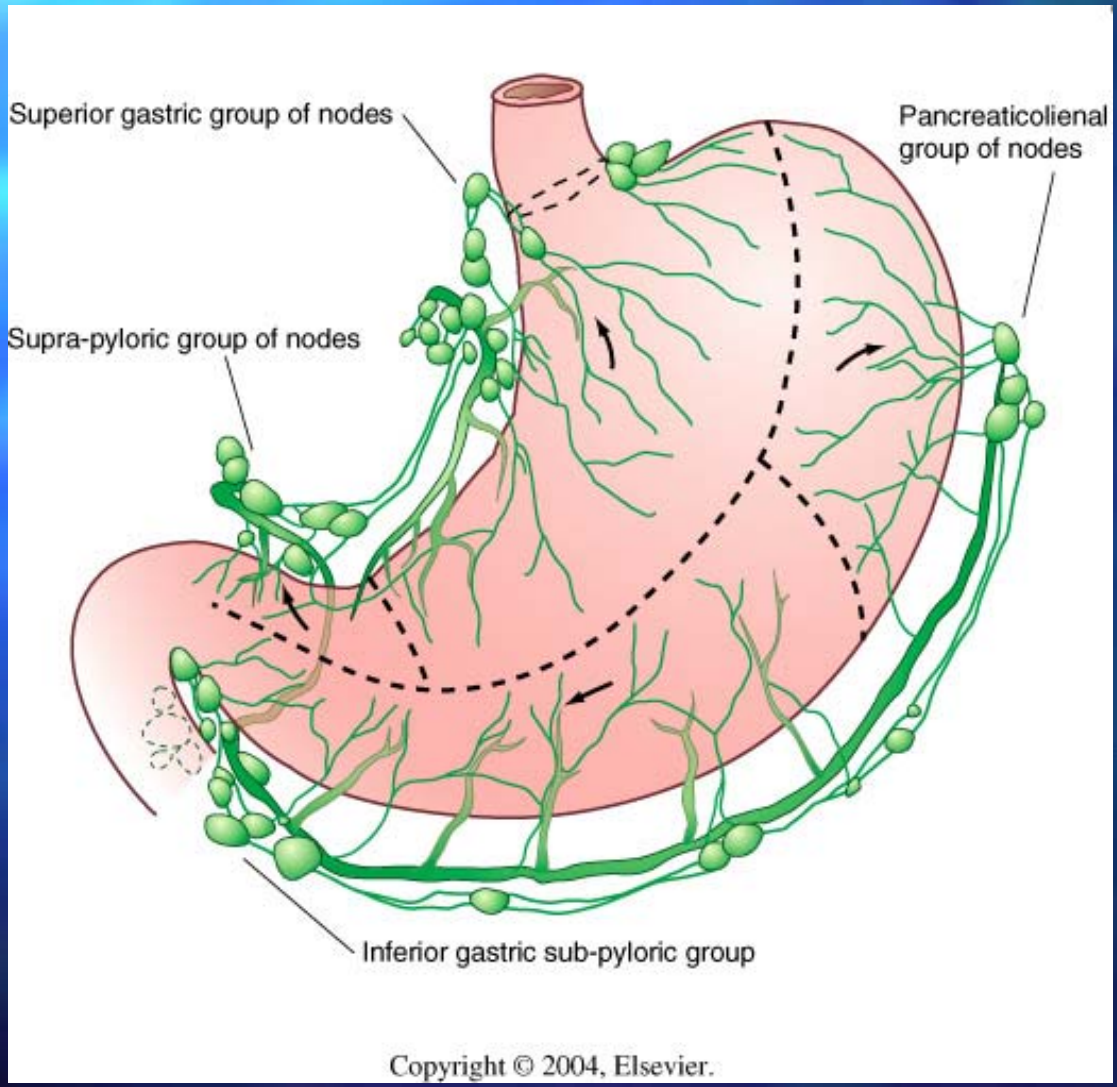
Coronary ligament
Liver
Visceral peritoneum
Lesser omentum
Stomach
Pancreas (retroperitoneal)
Parietal peritoneum
Transverse mesocolon
Duodenum (retroperitoneal)
Greater omentum
Omental bursa
Transverse colon
Small intestine
Mesentery proper
Urinary bladder (retroperitoneal)
Rectum (retroperitoneal)

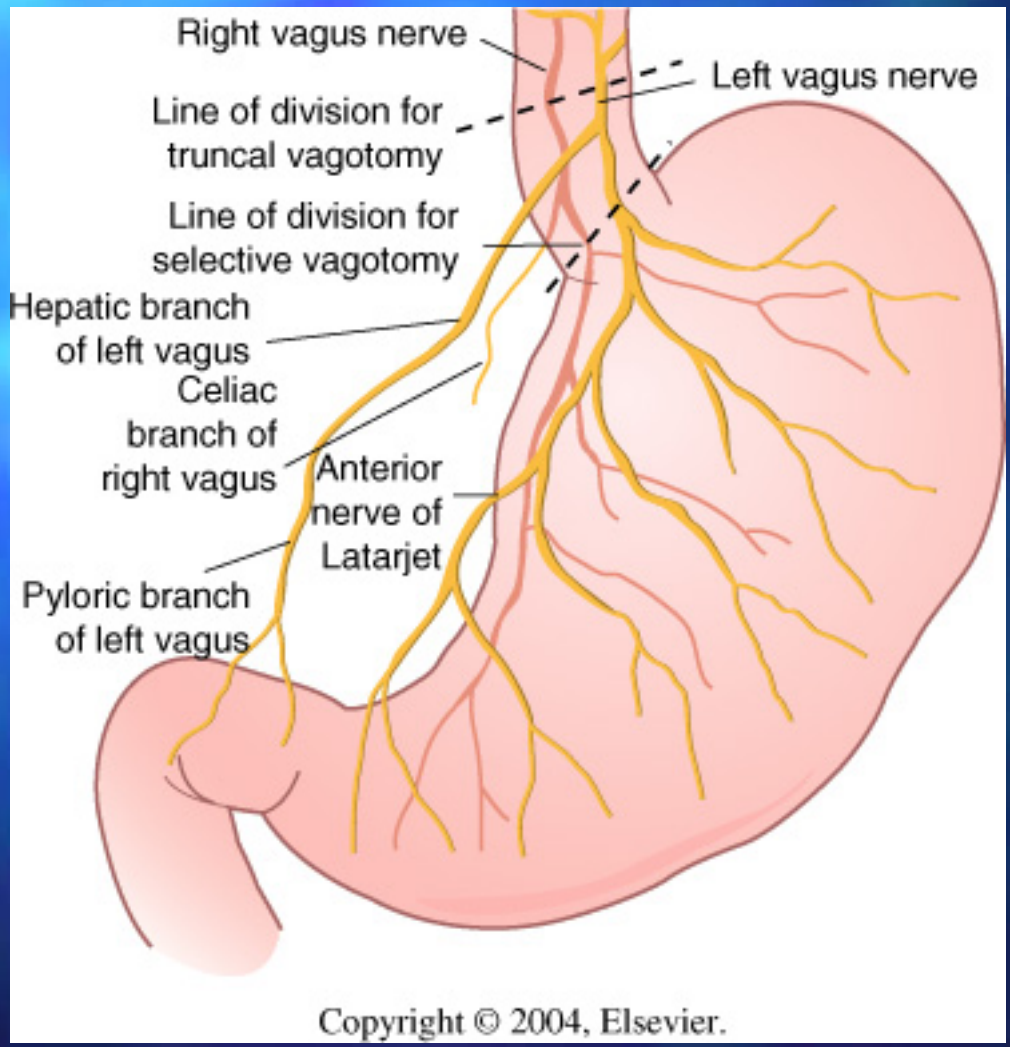
Anatomy: Blood Supply



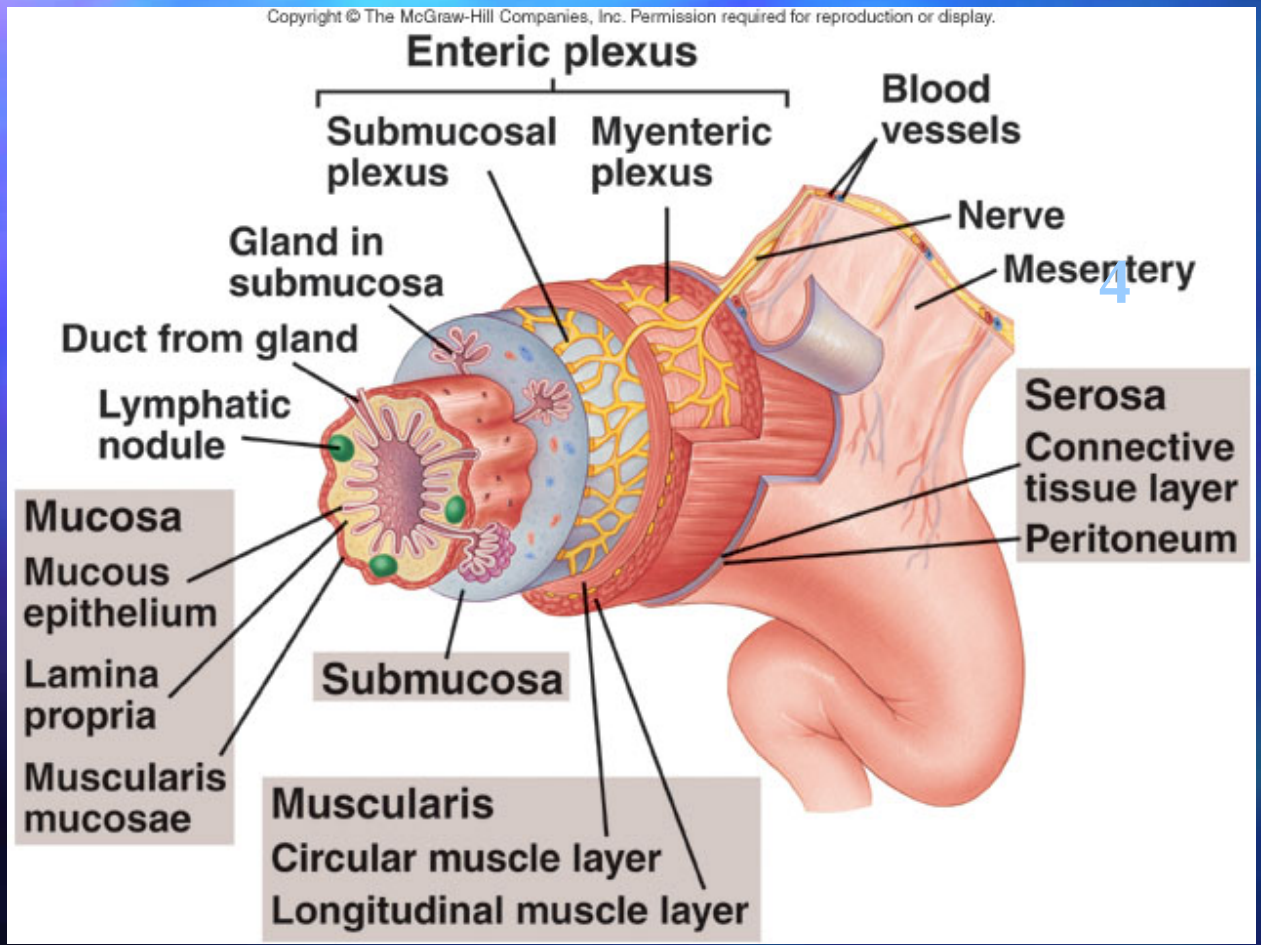
Anatomy: Lymphatic Drainage



Anatomy: Innervation

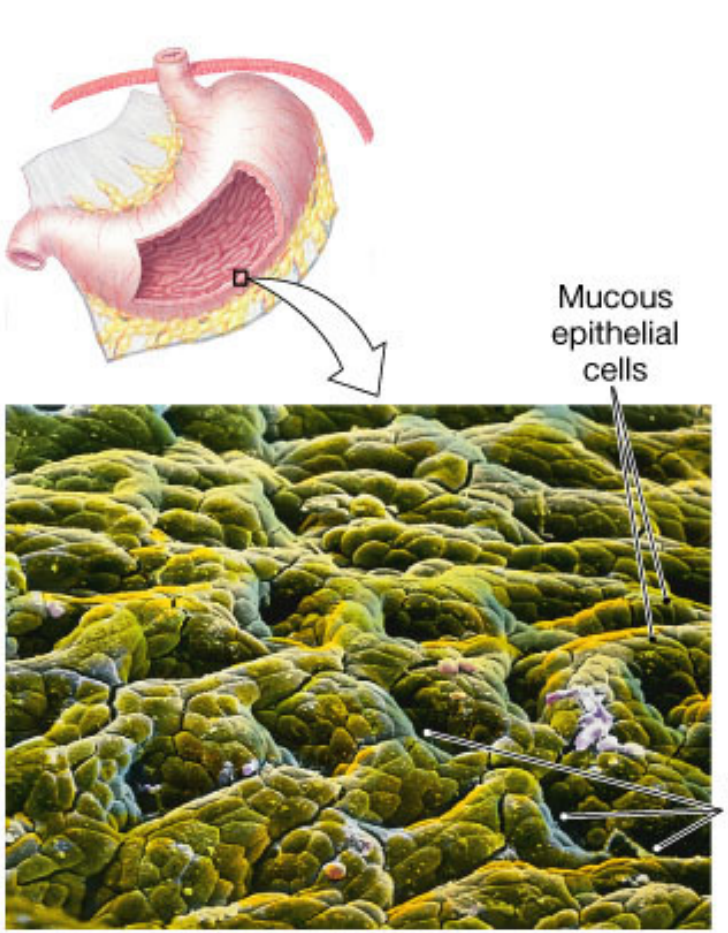


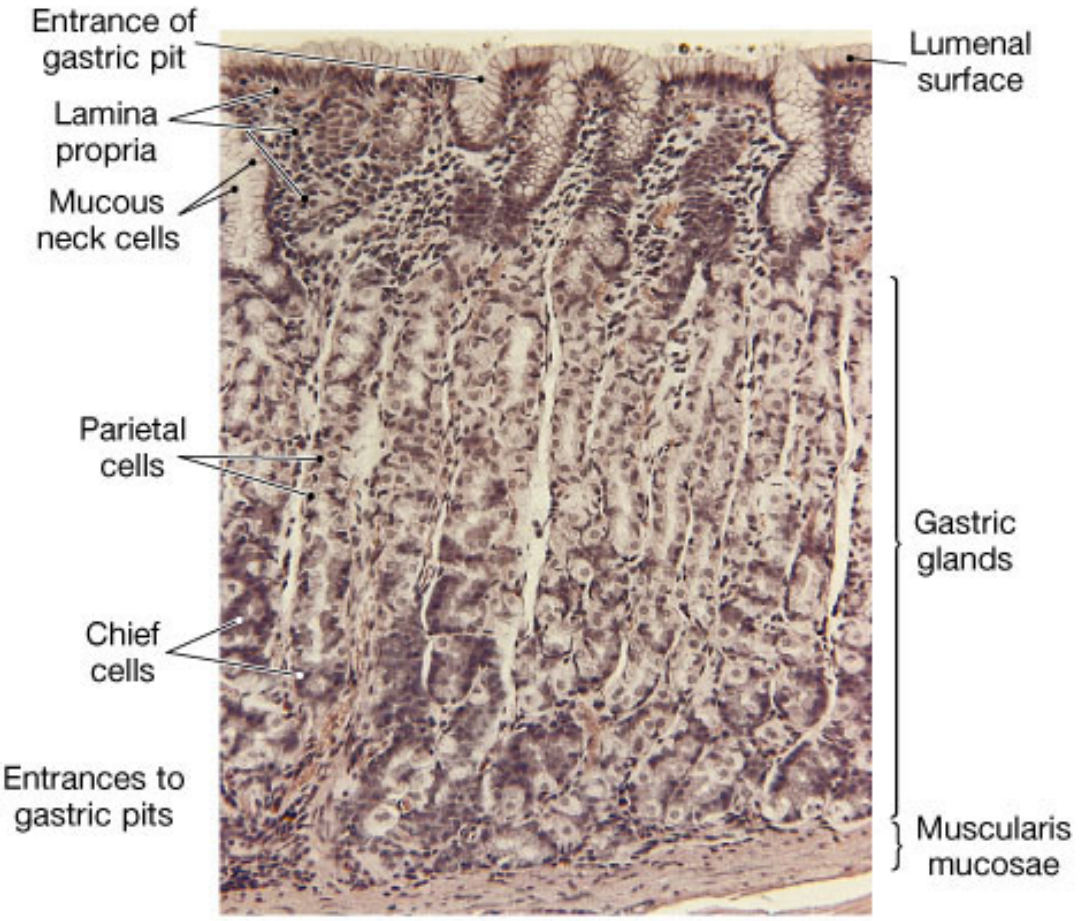
Histology



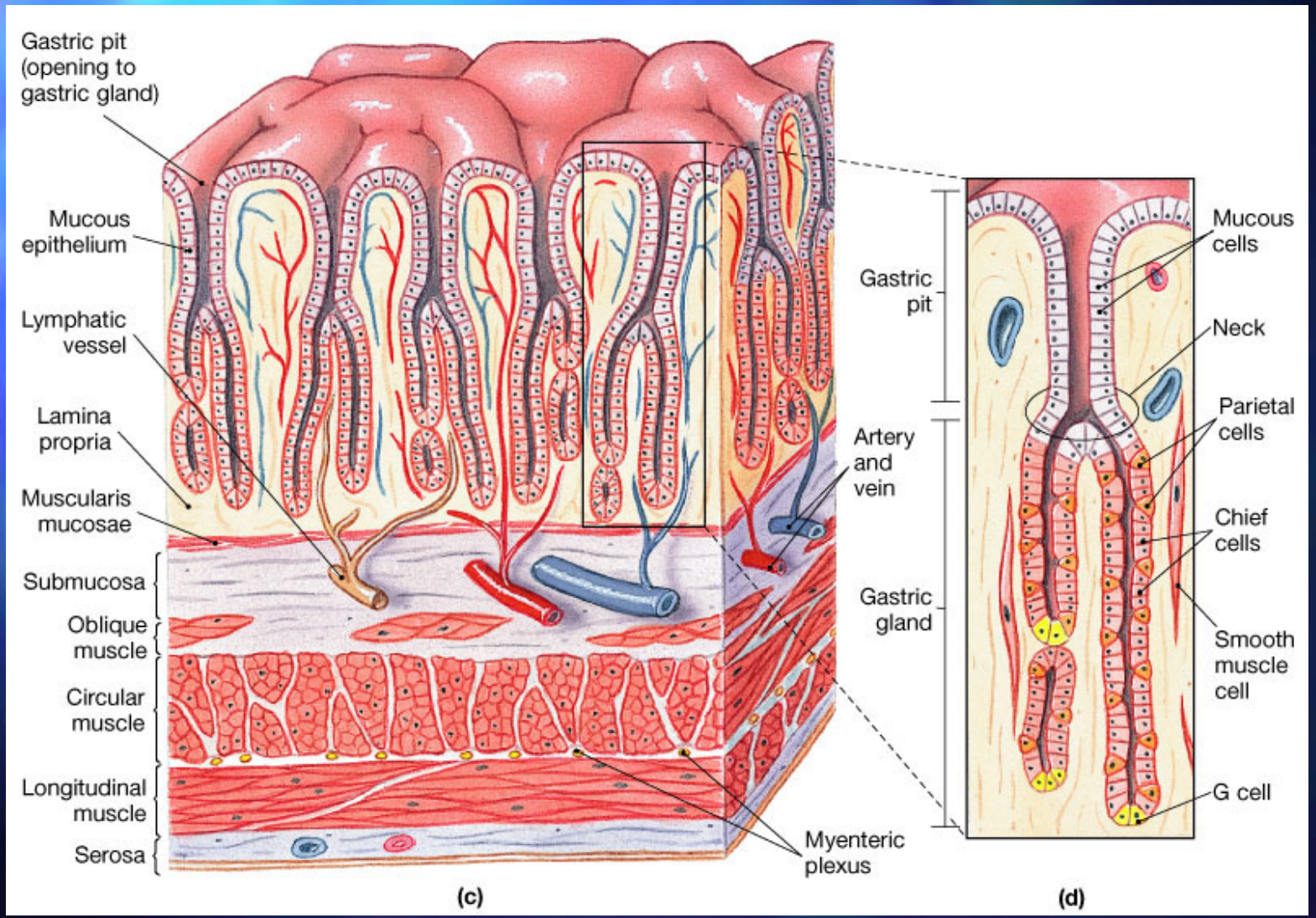
- Layers
  - Serosa or visceral peritoneum
  - Muscularis: Three layers
    - Outer longitudinal
    - Middle circular
    - Inner oblique
  - Submucosa
  - Mucosa
    3 sub layers

Histology

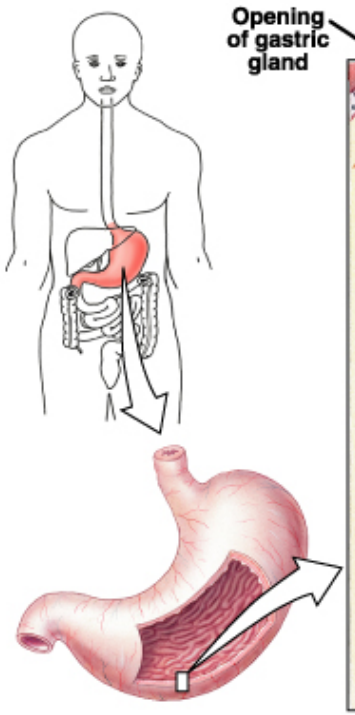

Mucous epithelial cells
Entrances to gastric pits
(a)

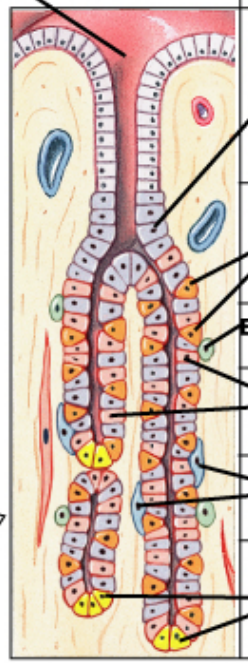

Entrance of gastric pit
Lamina propria
Mucous neck cells
Parietal cells
Chief cells
Lumenal surface
Gastric glands
Muscularis mucosae
(b)

Histology


Gastric pit (opening to gastric gland)
Mucous epithelium
Lymphatic vessel
Lamina propria
Muscularis mucosae
Submucosa
Oblique muscle
Circular muscle
Longitudinal muscle
Serosa
Myenteric plexus
(c)
Gastric pit
Artery and vein
Gastric gland
Mucous cells
Neck
Parietal cells
Chief cells
Smooth muscle cell
G cell
(d)

Histology



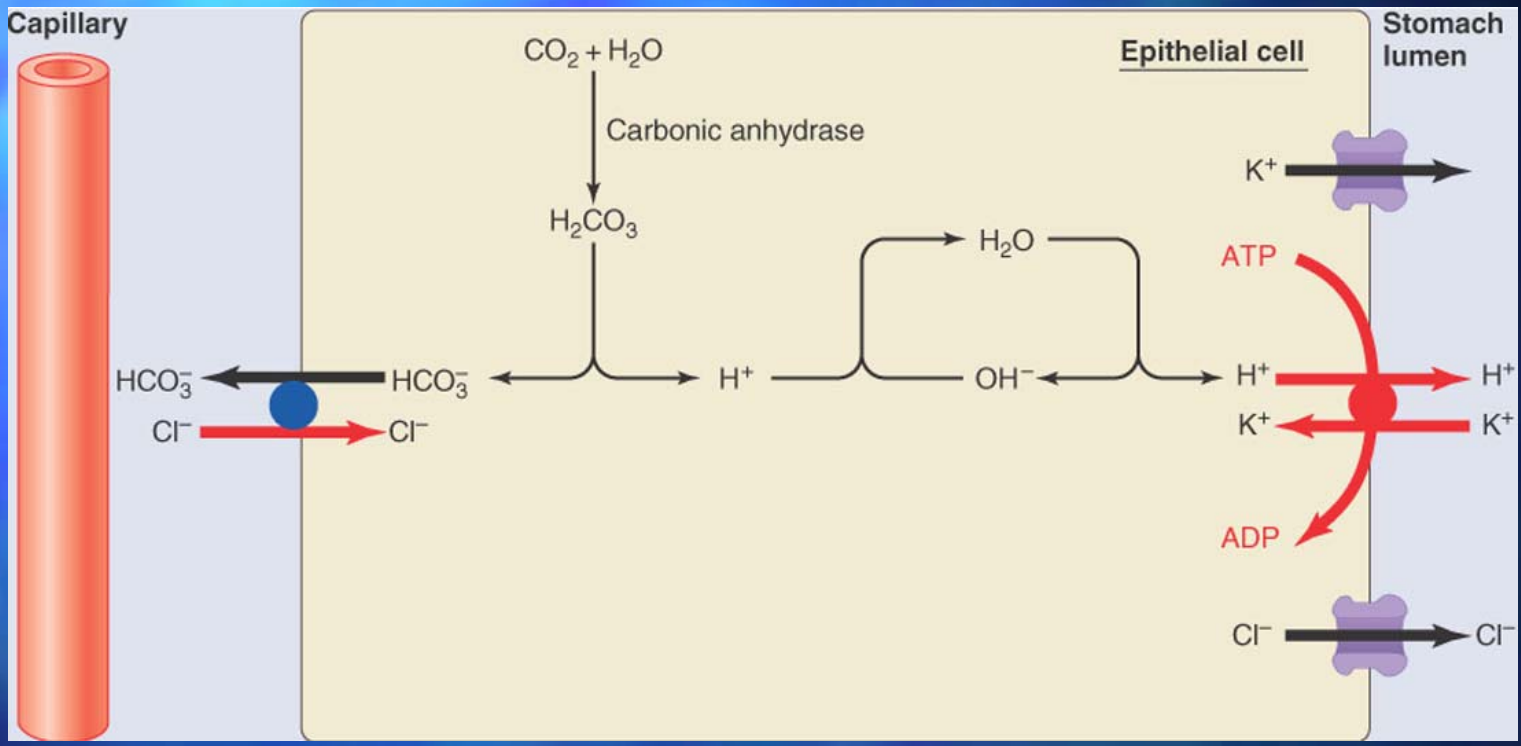


Source	Substance Secreted	Stimulus for Release	Function
Mucous neck cell	Mucus	Tonic secretion; increased with irritation of mucosa	Physical barrier between lumen and epithelium
	Bicarbonate	Secreted with mucus	Buffers gastric acid to prevent damage to epithelium
Parietal cells	Gastric acid (HCl)	Acetylcholine, gastrin, histamine	Activates pepsin; kills bacteria
	Intrinsic factor		Complexes with vitamin B_{12} to permit absorption
Enterochromaffin-like cell	Histamine	Acetylcholine, gastrin	Stimulates gastric acid secretion
Chief cells	Pepsin(ogen)	Acetylcholine, acid, secretin	Digests proteins
	Gastric lipase		Digests fats
D cells	Somatostatin	Acid in the stomach	Inhibits gastric acid secretion
G cells	Gastrin	Acetylcholine, peptides, and amino acids	Stimulates gastric acid secretion

Physiology: Functions of the Stomach

- Bulk storage of undigested food
- Mechanical breakdown of food
- Disruption of chemical bonds via acids and enzymes (pepsin)
- Production of intrinsic factor
- Very little absorption of nutrients
  - Some drugs, however, are absorbed
- Enteroendocrine cells

Physiology: Gastric Acid Secretion



Acid production by the parietal cells in the stomach depends on the generation of carbonic acid; subsequent movement of hydrogen ions into the gastric lumen results from primary active transport.

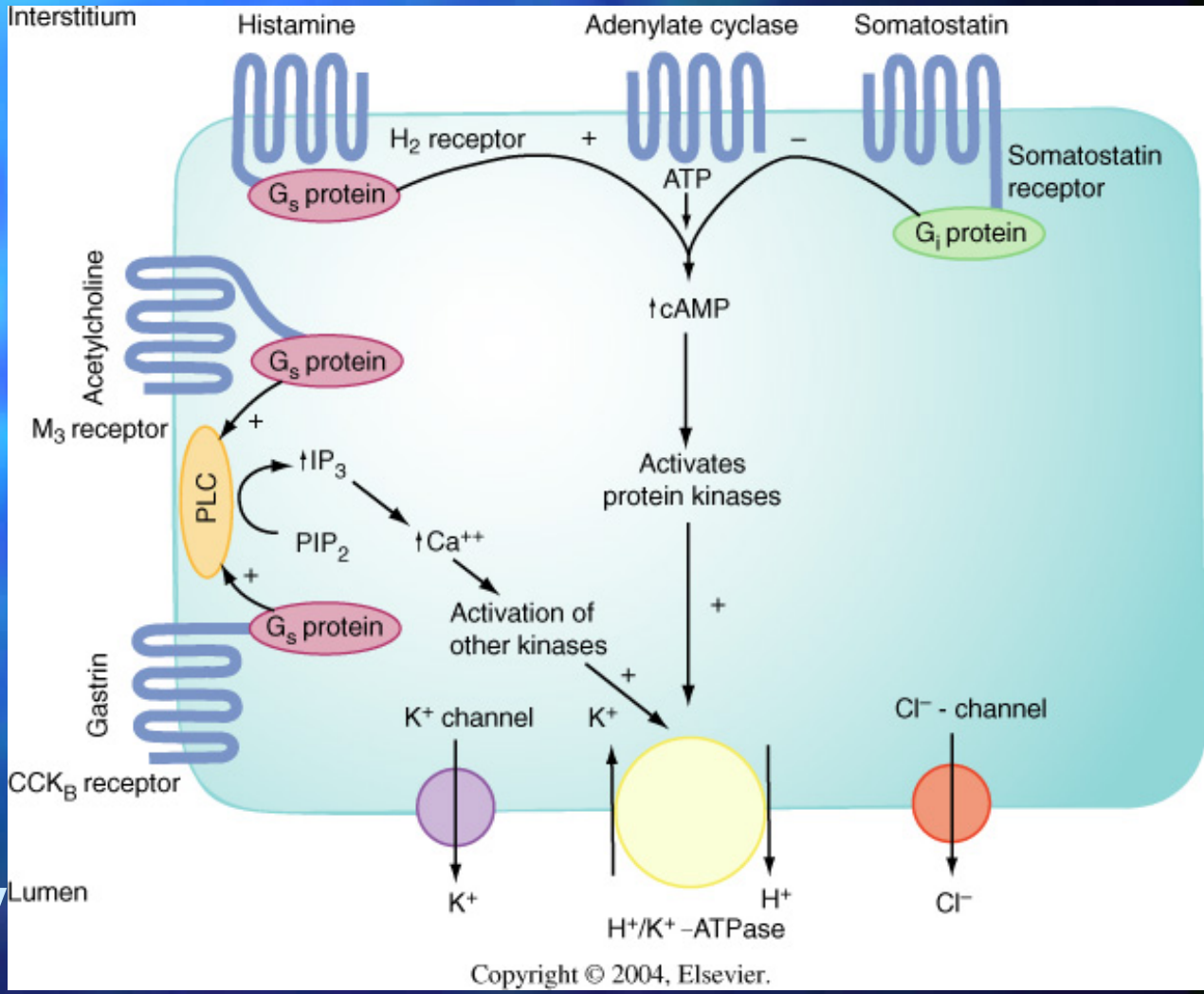
Physiology: Gastric Acid Secretion

One inhibitory and three stimulatory signals that alter acid secretion by parietal cells in the stomach.

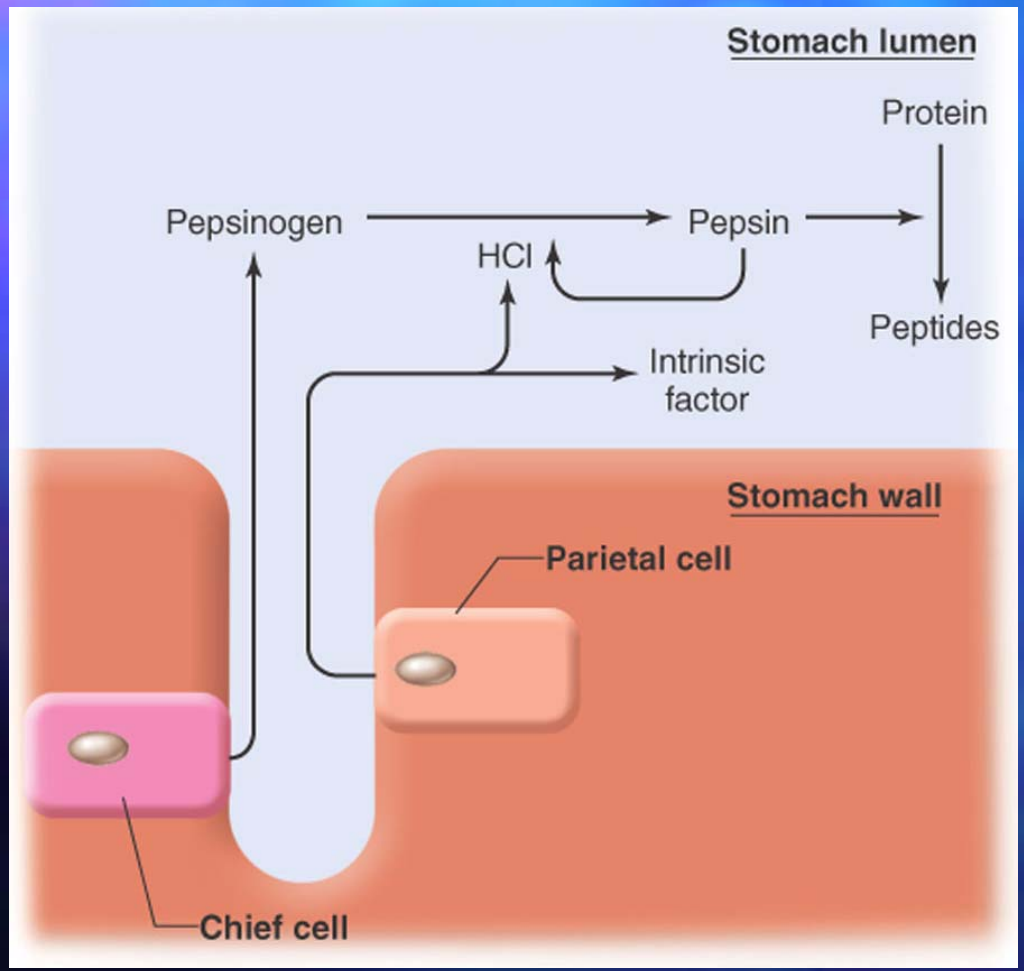
Gastrin and Ach work by increasing $[Ca^{++}]_I$ and activate Protein Kinases

Histamine works via a H_2 receptor and by a cAMP mechanism

All 3 work synergistically



Physiology: Gastric Acid Secretion



The acidity in the gastric lumen converts the protease precursor pepsinogen to pepsin; subsequent conversions occur quickly as a result of pepsin's protease activity.

Physiology: Regulation of Gastric Acid Secretion

(a) The Cephalic Phase

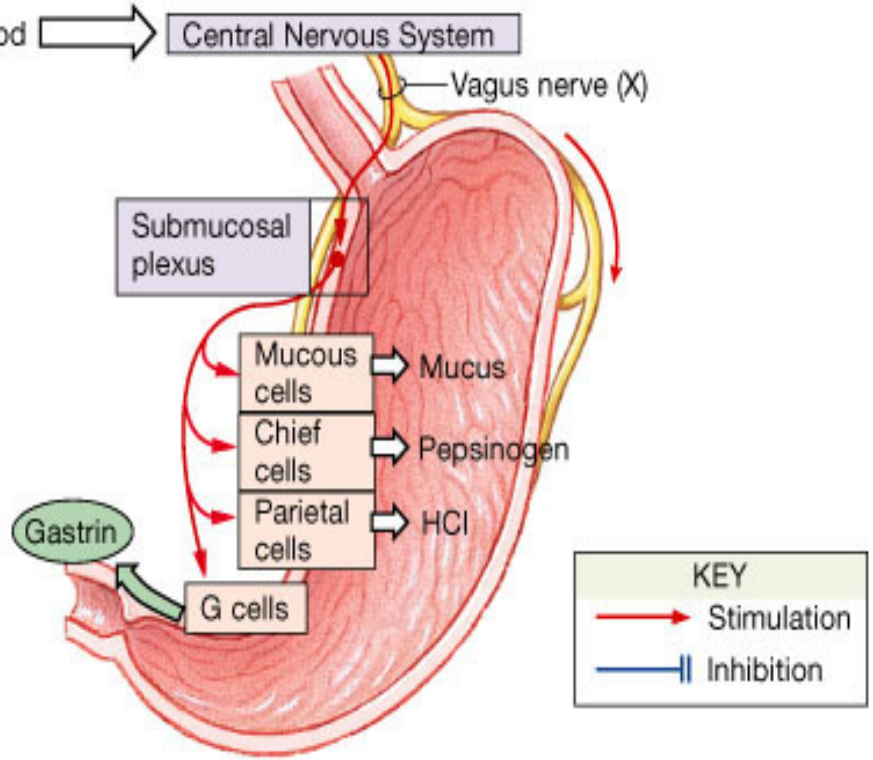
Sight, smell, taste, or thoughts of food → Central Nervous System

Function:
Prepare stomach for arrival of food

Duration:
Short (minutes)

Mechanism:
Neural, via preganglionic fibers in vagus nerve and synapses in submucosal plexus

Actions:
Primary: stimulation of mucus, enzyme, and acid production, leading to increased volume of gastric juice
Secondary: stimulation of gastrin release by G cells



Physiology: Regulation of Gastric Acid Secretion

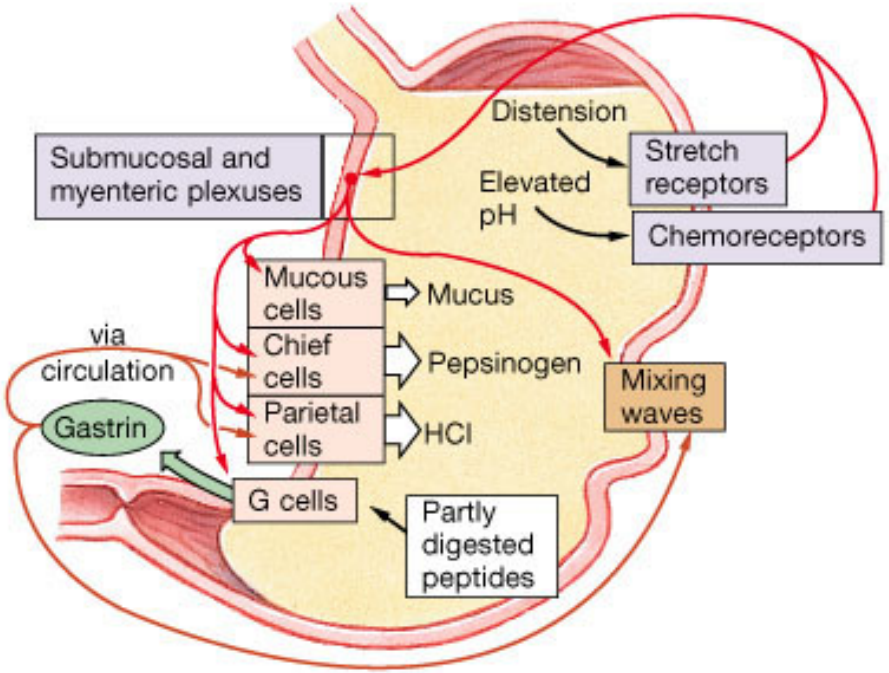
(b) The Gastric Phase

Functions:
- Enhance secretion started in cephalic stage; homogenize and acidify chyme; initiate digestion of proteins by pepsin

Duration:
- Long (3–4 hours)

Mechanisms:
- *Neural:* short reflexes triggered by
  - (1) stimulation of stretch receptors as stomach fills
  - (2) stimulation of chemoreceptors as pH increases
- *Hormonal:* stimulation of gastrin release by G cells through parasympathetic activity and presence of peptides and amino acids in chyme
- *Local:* release of histamine by mast cells as stomach fills (not shown)

Actions:
- Increased acid and pepsinogen production; increased motility and initiation of mixing waves



Physiology: Regulation of Gastric Acid Secretion

(c) The Intestinal Phase

Function:
- Control rate of chyme entry into duodenum

Duration:
- Long (hours)

Mechanisms:
- *Neural:* short reflexes (enterogastric reflex) triggered by distension of duodenum
- *Hormonal:*
- Primary: stimulation of cholecystokinin (CCK), gastric inhibitory peptide (GIP), and secretin release by presence of acid, carbohydrates, and lipids
- Secondary: release of gastrin stimulated by presence of undigested proteins and peptides (not shown)

Actions:
- Feedback inhibition of gastric acid and pepsinogen production; reduction in gastric motility

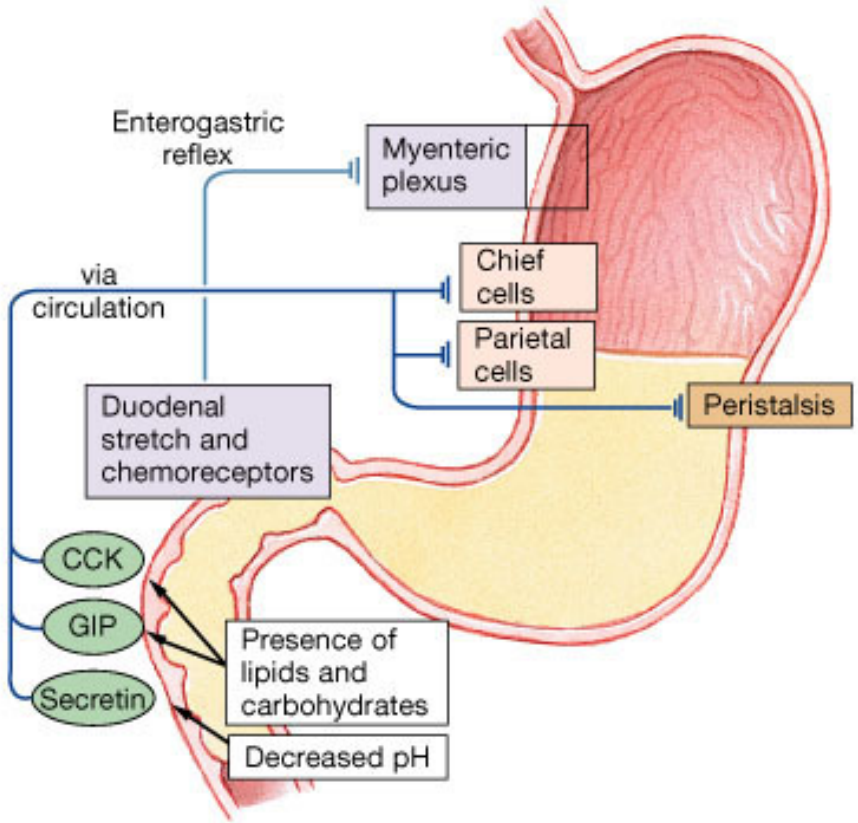
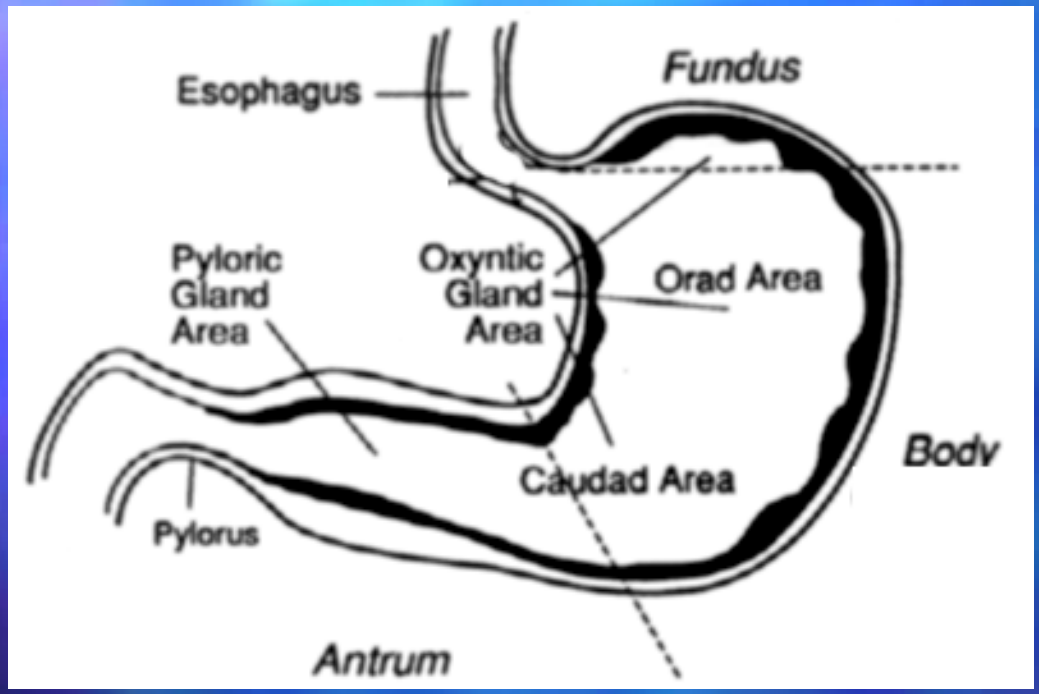


Table 24.3 Functions of the Gastrointestinal Hormones

Site of Production	Method of Stimulation	Secretory Effects	Motility Effects
Gastrin			
Stomach and duodenum	Distention; partially digested proteins, autonomic stimulation, ingestion of alcohol or caffeine	Increases gastric secretion	Increases gastric emptying by increasing stomach motility and relaxing the pyloric sphincter
Secretin			
Duodenum	Acidity of chyme	Inhibits gastric secretion; stimulates pancreatic secretions high in bicarbonate ions; increases the rate of bile and increases intestinal secretion; mucus secretion	Decreases gastric motility
Cholecystokinin			
Intestine	Fatty acids and other lipids	Slightly inhibits gastric secretion; stimulates pancreatic secretions high in digestive enzymes; and causes contraction of the gallbladder and relaxation of the hepatopancreatic ampullar sphincter	Decreases gastric motility
Gastric Inhibitory Polypeptide			
Duodenum and proximal jejunum	Fatty acids and other lipids	Inhibits gastric secretions	Decreases gastric motility

Physiology: Motility



Proximal stomach

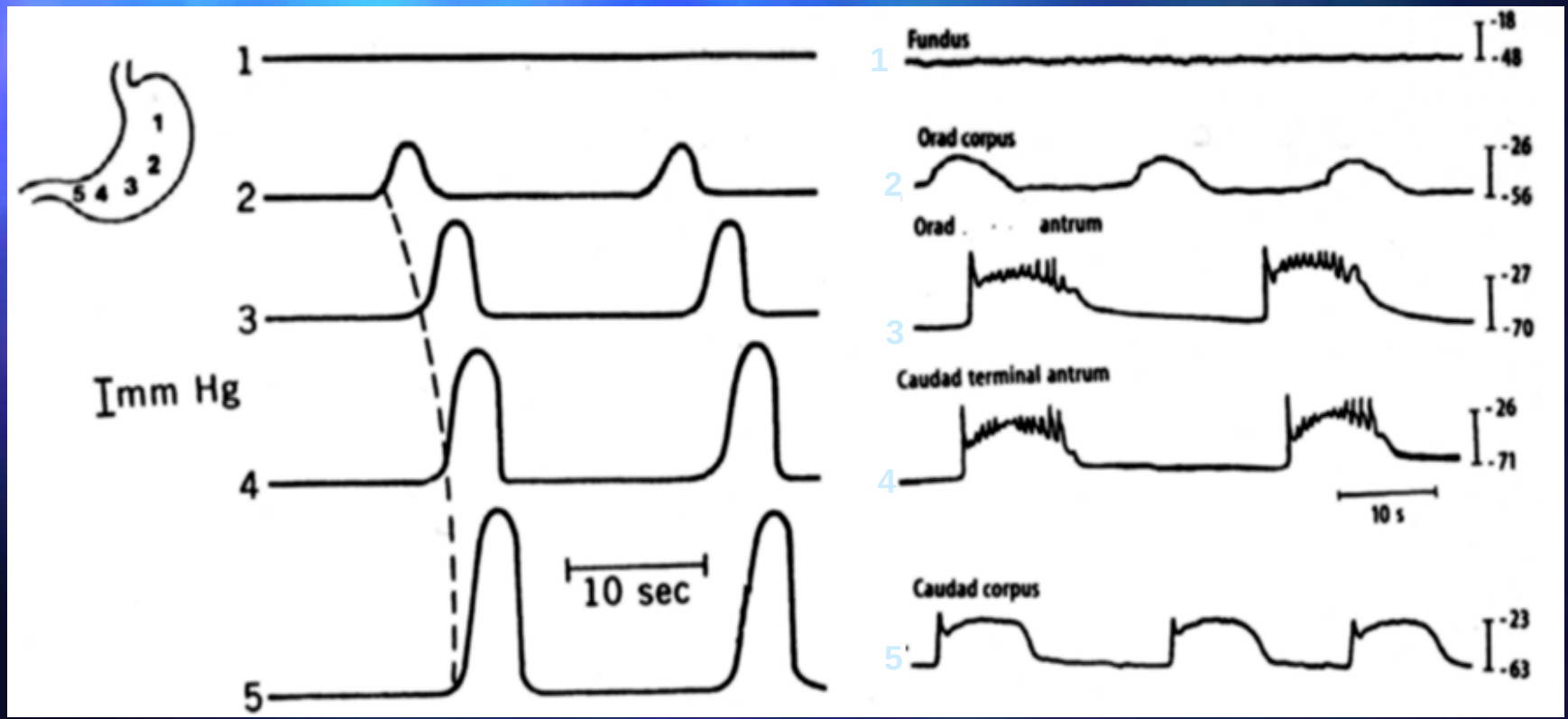
- No basal electrical activity
- Slow tonic contraction
- High distensibility
- Gastric reservoir

Distal stomach

- Basal electrical activity
- Peristaltic phasic contractions
- Low distensibility
- Grinding of solids

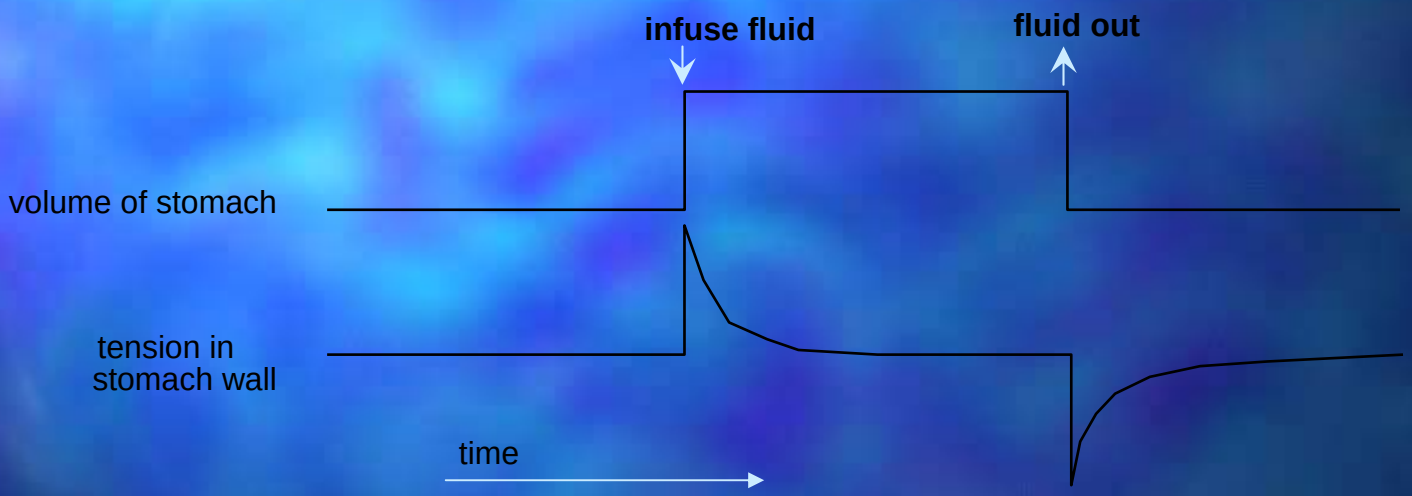
Physiology: Motility

Peristaltic strength increases: corpus to antrum



Physiology: Motility

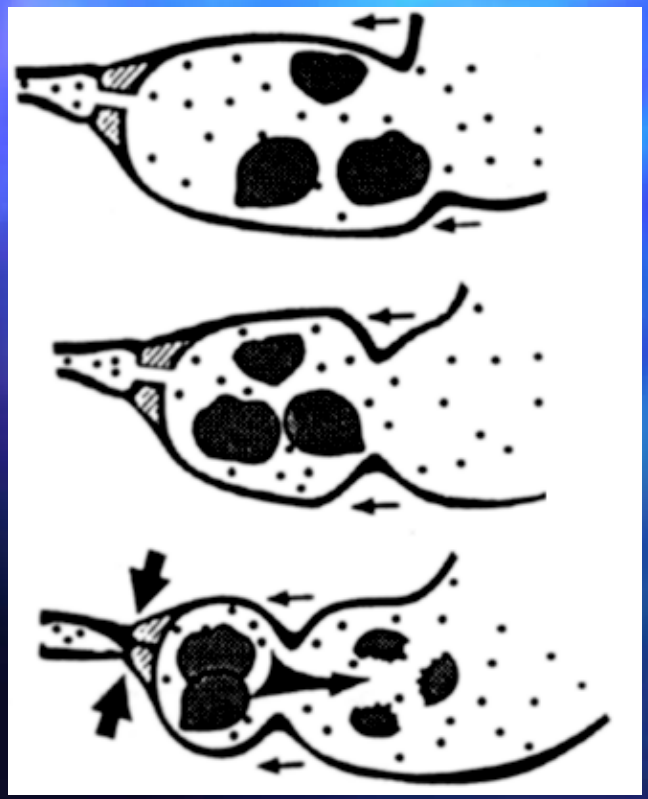
Isotonic Relaxation and Contraction



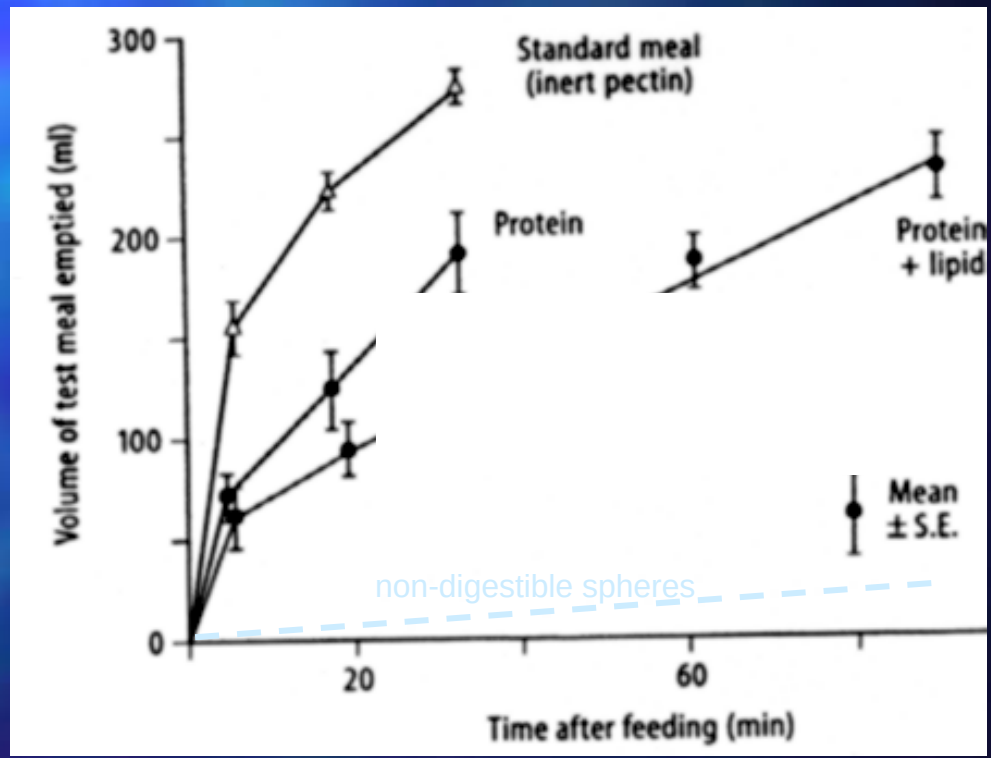
Physiology: Motility

Gastric Emptying

Propulsion-Retropulsion mechanism in the antrum



Content regulates gastric emptying



Pathology

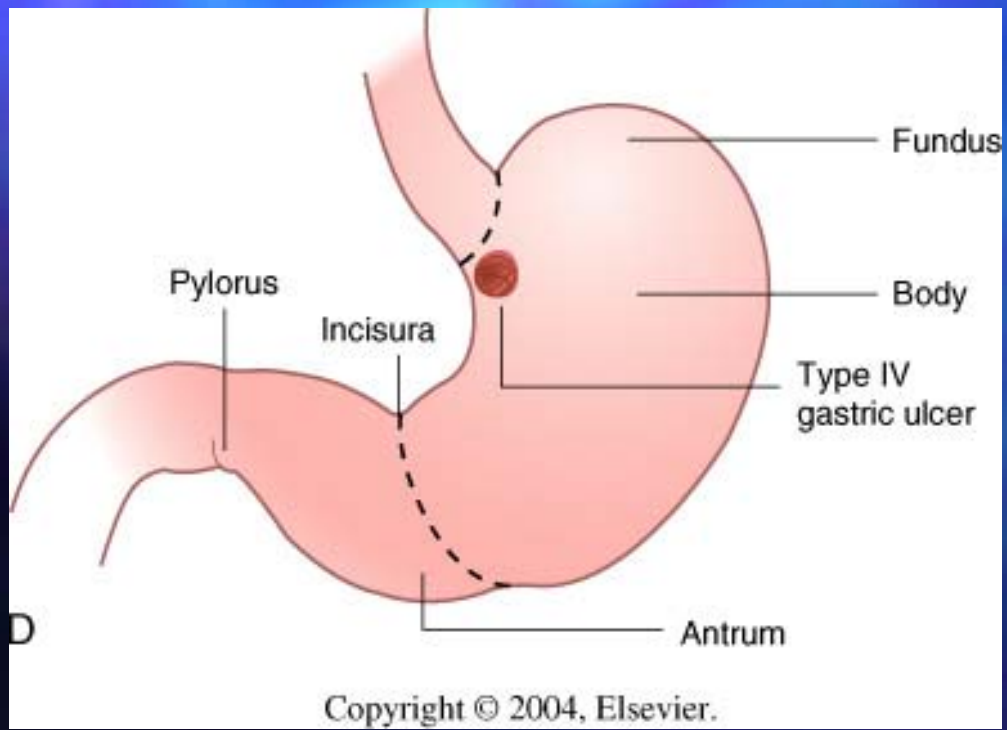
- Peptic Ulcer Disease
- Neoplasia
- Other

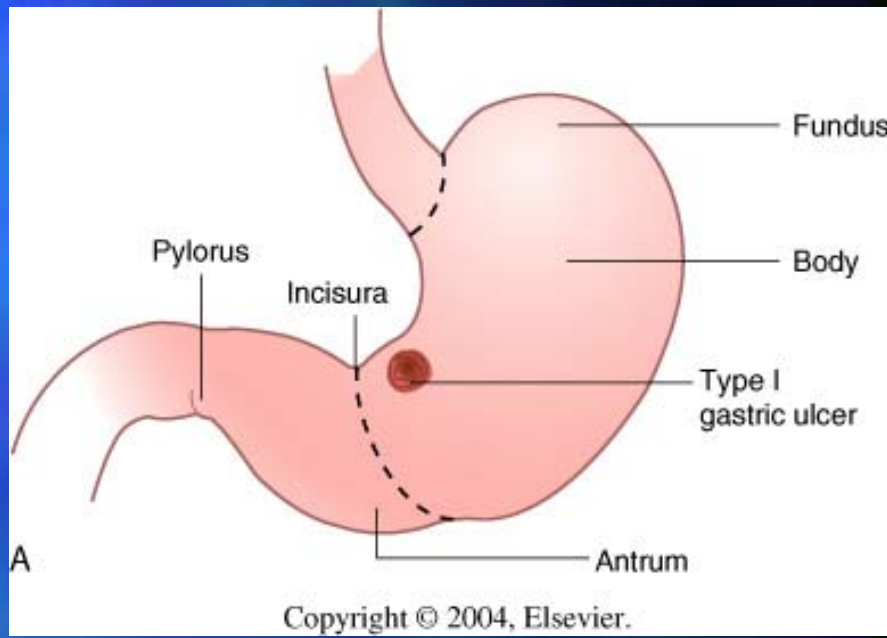
Peptic Ulcer Disease (PUD)

- Definition: ulcer x erosion (musc mucosa)
- Epidemiology
  - 500,000 new cases per year
  - Prevalence > incidence
  - 3-4 million pts seen by MD every year
  - 130,000 operations for PUD per year
  - 9,000 deaths from PUD complications
  - Over last 20y:
    - Increase in emergency operations
    - Decrease in elective operations

PUD: Location and Types of Ulcers

- Type IV:
  - Rare in the US and Europe
  - Common in Latin America

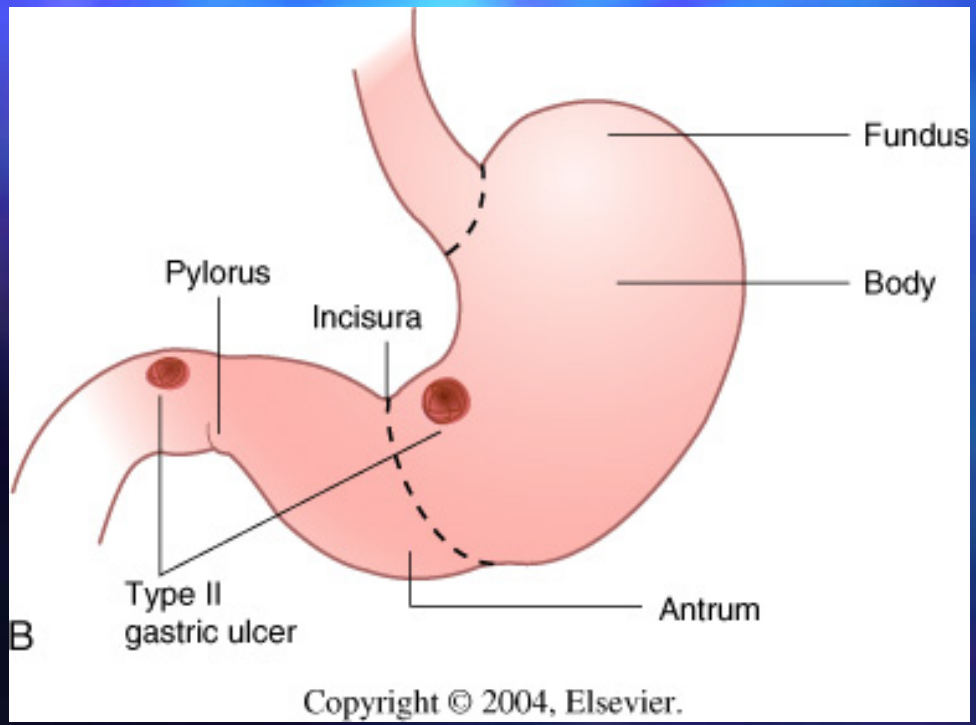


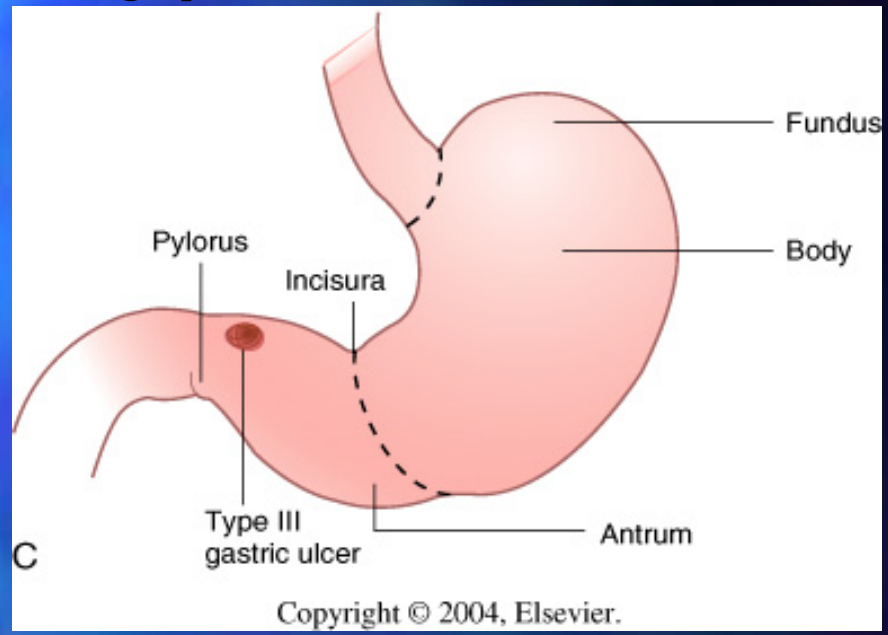


- Type I:
  - More common (60-70%)
  - Normal or low acid secretion
  - Not assoc with gastric or duodenal mucosa abnormalities.

PUD: Location and Types of Ulcers

- Type II:
  - ~ 15%
  - Assoc with active or chronic duodenal ulcer disease





- Type III:
  - ~ 20%
  - Assoc with diffuse antral gastritis

PUD: Pathogenesis

- Helicobacter pylori
  - Urease producting GNR
  - Association: 90% duodenal; 75% gastric
  - ? Mechanism
    - Local mucosal injury by toxic products
    - Induction of local immune response
    - Increased acid secretion: increased gastrin (D cell destruction)
  - Associated with low socioeconomic status (?)
  - Lifetime risk of PUD if + H.pylori: 15% (3%-)

PUD: Pathogenesis

- NSAIDS
  - 2nd most cause of PUD and increasing
  - 3 million on NSAIDS; 1 in 10 has active ulcer
  - Risk of gastric complication: increased 2-10x
  - Risk is proportional to anti-inflam potency
  - Acute or chronic injury
    - Acute: within 1-2 weeks of use
    - Chronic: after 1 month
  - Ulcers are more frequent in the stomach

PUD: Pathogenesis

- Acid secretion
  - Basal acid secretion: 1-8 mmol/h
  - Response to pentagastrin: 6-40 mmol/h
  - Gastric ulcers type II and III and duodenal ulcers are associated with increased gastric acid secretion
  - Pernicious anemia, gastric cancer, gastric atrophy, gastric ulcers tipe I and IV are associated with decreased basal and post-pentagastrin acid output

PUD: Pathogenesis

- Duodenal Ulcer
  - Multiple etiologies
  - Only relatively absolute requirements:
    - Acid and pepsin secretion
    - Combination with either NSAIDS or H. pylori
  - Multiple secretory abnormalities:
    - Decr duodenal bicarb secretion: 70%
    - Incr nocturnal acid secretion: 70%
    - Incr duodenal acid load: 65%
    - Incr sensitivity to gastrin: 35% ...

PUD: Clinical Manifestations

- Duodenal Ulcer
  - Abdominal pain:
    - varies; most common is well localized mid-epigastric pain
    - relieved by food
    - May be episodic or seasonal in spring and fall or by stress
    - Constant pain: ? Deeper penetration of the ulcer
    - Back pain: ? Perforation/penetration into the pancreas

PUD: Clinical Manifestations

- Duodenal Ulcer
  - Perforation
    - 5%: free perforation into the peritoneal cavity
    - Patient recalls exact time of onset
    - Accompanied by fever, tachycardia, dehydration, ileus
    - Abdominal PE: tenderness, rigidity, rebound
    - Hallmark: free air underneath the diaphragm on XRay

PUD: Clinical Manifestations

- Duodenal Ulcer
  - Bleeding
    - Most common cause of death (usually >65 yo and with multiple comorbidities)
    - Gastroduodenal arteries lie directly posterior
    - However, most present as minor bleeding by more superficial ulcers

PUD: Clinical Manifestations

- Duodenal Ulcer
  - Obstruction
    - Acute: functional GOO with associated inflammation
      - Delayed gastric emptying with N/V, anorexia
      - Prolonged vomiting may cause hypo Cl,K,H.
    - Chronic: recurrent inflammation/healing/scarring leading to obstruction
      - Painless vomiting of large amounts
      - Similar metabolic abnormalities
      - Stomach may become massively dilated
      - Malnutrition

PUD: Clinical Manifestations

- Gastric Ulcer
  - Abdominal pain: similar to duodenal (Sabiston)
  - Concern about potential malignancy
  - Surgical intervention needed for complications: 8-20%
  - Hemorrhage: 35-40%. Patients usually have worse medical condition than in duod ulcers
  - Peforation is the most frequent complication
    - Usually along the anterior aspect of the lesser curve

PUD: Clinical Manifestations

- Zollinger-Ellison Syndrome
  - Clinical triad:
    - Gastric acid hypersecretion
    - Severe peptic ulcer disease
    - Non-beta islet cell of the pancreas. Tumors secret gastrin (gastrinomas).
  - Tumors usually in the panc head, duod wall or regional lymph nodes (gastrinoma triangle: CBD, neck of the pancreas, 3rd portion of the duodenum)
  - 50% are multiple; 2/3 are malignant; ¼ are MEN I
  - Diarrhea due to increased acid secretion
  - Steatorrhea because of decreased duod/jej pH and inactivation of lipase

PUD: Clinical Manifestations

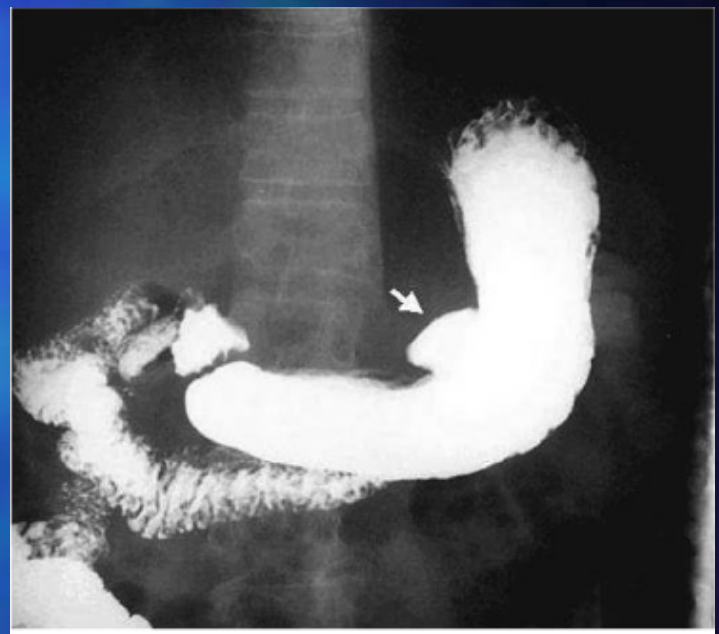
- Zollinger-Ellison Syndrome
  - Hypercalcemia and other signs of MEN I
  - Dx usually does not require provocative tests (secretin): fasting/stimulated gastrin levels are high enough to make diagnosis
  - CT to show tumor
  - Tx is surgical removal of tumor and/or PPIs

PUD: Diagnosis

- H/P of limited value for gastric x duodenal ulcer dz
- Routine labs, CXR
- Gastrin level for refractory ulcers
- CXR

PUD: Diagnosis

- Image
  - Contrast radiograph:
    - Less expensive
    - 90% accurate (double-contrast)
    - But
      - 5% of “benign” ulcers are actually malignant
      - 50% of duodenal ulcers may be missed by single-contrast studies
  - EGD
    - Accuracy 97%; the most reliable method
    - Ability to biopsy lesions and sample for H.pylori dx



PUD: Diagnosis

- H. pylori testing
  - Noninvasive
    - Serology:
      - ELISA or others
      - 90% sensitivity / specificity
      - Test of choice when EGD cannot be done.
      - May be positive for ~ 1 year after eradication.
    - Carbon-labeled urea breath test:
      - 95% sensitivity / specificity
      - Patient ingests carbon-labeled urea. Urea is metabolized to ammonia and labeled bicarb (urease)
      - Test of choice to document eradication
      - Needs to be done ~ 4 weeks after tx because of possible false negatives.

PUD: Diagnosis

- H. pylori testing
  - Invasive
    - Rapid urease test
      - Method of choice for diagnosis with EGD (cheap)
      - Mucosal biopsies are placed in a medium containing urea and a pH indicator. If urease +, it will become alkaline
      - Sensitivity 90%; specificity 98%
      - Cheap, except for endoscopy
    - Histology
      - Direct visualization of H. pylori
      - Gold standard of tests; Sensitivity 95%; specificity 99%
    - Culture
      - Sensitivity 80%; specificity 100%
      - Requires 3-5 days

PUD: Treatment

- Medical Management
  - Lifestyle modifications:
    - Smoking cessation
    - Coffee
    - Alcohol
  - D/C aspirin or NSAIDS (? switch to COX2)
  - Eradication of H. pylori
    - Duodenal ulcer recurrence: 75% with no maint tx, 25% with maint tx, < 2% with H.pylori eradication
    - All patients with ulcer + H. pylori should be treated
    - Amoxicillin, tetracycline, metronidazol x 2 weeks
  - Medications

PUD: Treatment

- Medical Management
  - Medications
    - Antacids
      - Oldest form of therapy
      - Inhibit pepsin action by raising pH by reacting with HCl
      - Most effective when ingested 1 hour after meal
      - Minimal side effects
      - ~ 80% of ulcer healing in 1 month
      - Magnesium can cause diarrhea
      - Phosphorus can cause constipation
      - Need to be taken several times/day and large amounts

PUD: Treatment

- Medical Management
  - Medications
    - H2-Receptor antagonists
      - Structure is similar to histamine
      - Hepatic metabolism / excreted by the kidneys
      - Continuous infusion is more efficacious than intermittent
      - 70-80% of duodenal ulcer healing after 4 weeks
      - 80-90% of duodenal ulcer healing after 8 weeks
    - Proton-Pump Inhibitors
      - Most potent class
      - Irreversebly bind to proton pump
      - More prolonged and complete inhibition than H2Rs
      - More rapid healing of ulcers (85% 4w; 95% 8w)
      - Do not associate with H2Rs

PUD: Treatment

- Medical Management
  - Medications
    - Sucralfate
      - Structure is similar to heparin; not an anti coagulant
      - Aluminum salt of sulfated sucrose dissociates under acidic conditions in the stomach. ? Then binds to protein in the ulcer crater and provide a protective coating?
      - Ulcer healing is comparable to H2Rs
      - Magnesium can cause diarrhea
      - Phosphorus can cause constipation
      - Need to be taken several times/day and large amounts

PUD: Treatment

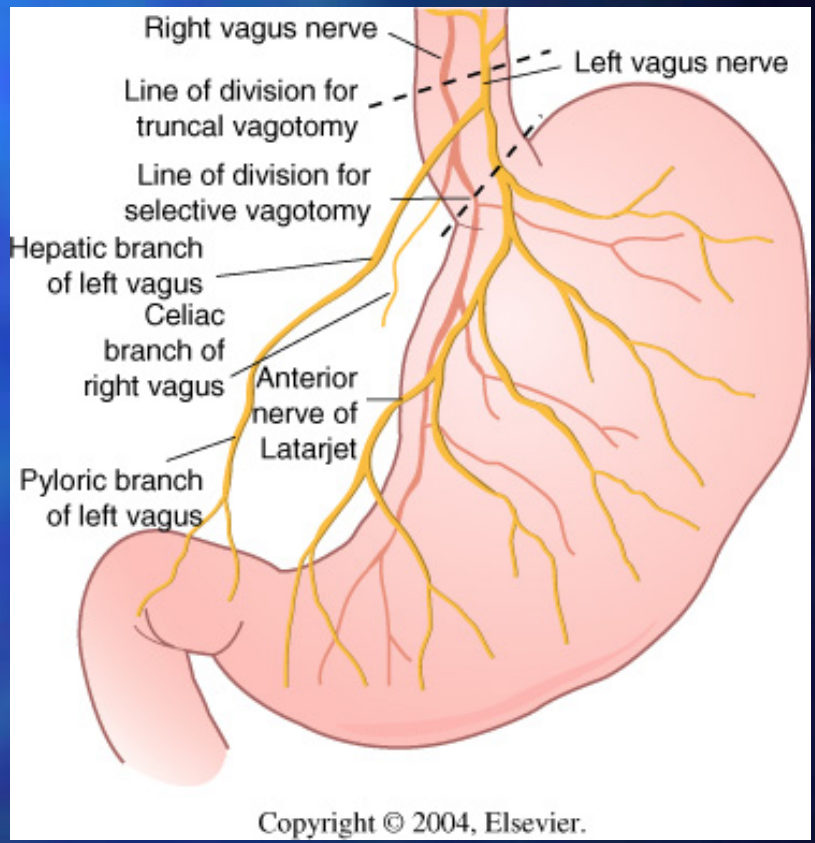
- Bleeding from PUD
  - 80% upper GI bleed are self-limited
  - 8-10% require intervention and number has not changed
  - Initial resuscitation
  - EGD: diagnostic AND therapeutic
  - Worse prognosis: age > 60, shock, high transfusion requirement, recurrent bleeding, inhospital bleeding, visible vessel on EGD

PUD: Surgical Options

- Truncal vagotomy
- Highly selective vagotomy (Parietal cell vagotomy)
- Truncal vagotomy and antrectomy
- Subtotal Gastrectomy
- Laparoscopy

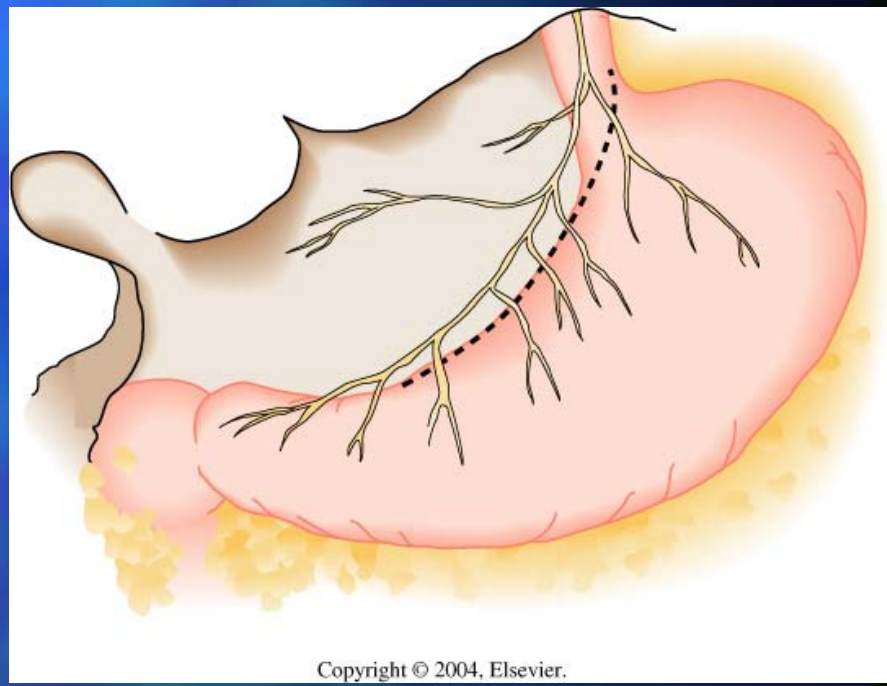
PUD: Surgical Options

- Truncal vagotomy
  - Division of L+R vagus nerves just above the GEj
  - Most common op for duodenal UDz
  - Requires drainage procedure
  - Can be performed quickly, with few complications (good for bleeding ulcers)



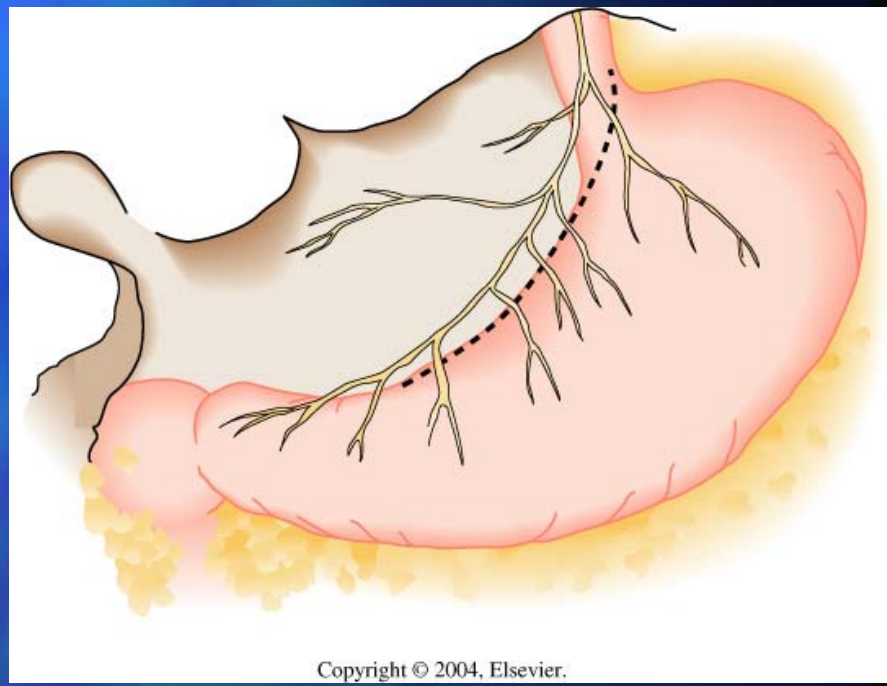
PUD: Surgical Options

- Highly selective vagotomy (parietal cell vagotomy)
  - Nerves of Latarjet are divided at the crow's feet (5 cm above GEj to 7 cm proximal to the pylorus)
  - Preserves vagal innervation of the gastric antrum
  - Does not require drainage procedure
  - Decr post op complications



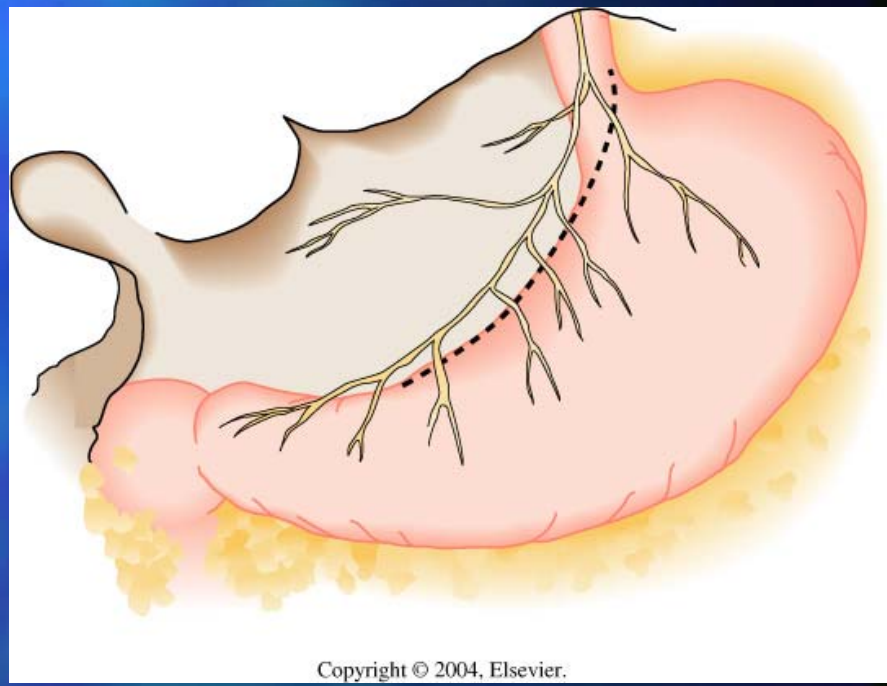
PUD: Surgical Options

- Highly selective vagotomy (parietal cell vagotomy)
  - "Criminal nerve of Grassi": very proximal branch of the posterior trunk
  - Recurrence rates are variable and depend on surgeon's skills and duration of follow up: 10-15% by skilled surgeons (similar to truncal vagot, but with lower complication rates)



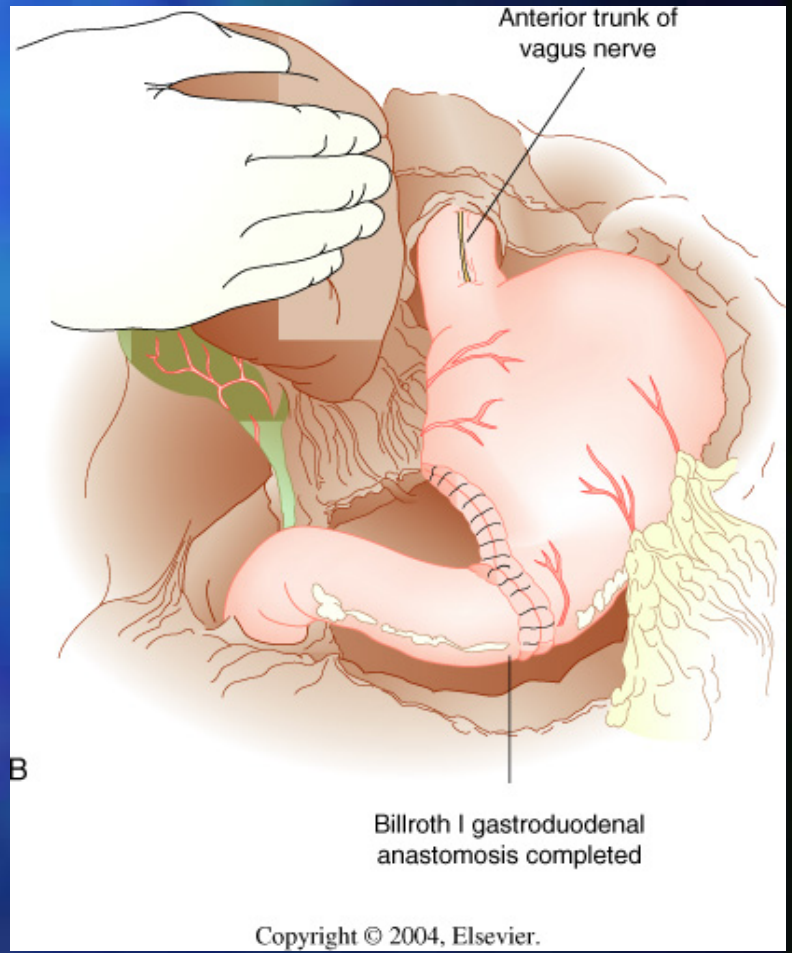
PUD: Surgical Options

- Highly selective vagotomy (parietal cell vagotomy)
  - Recurrences can be treated with PPIs
  - Pre pyloric ulcers recur >>> than duodenal. Therefore, may not be the procedure of choice.



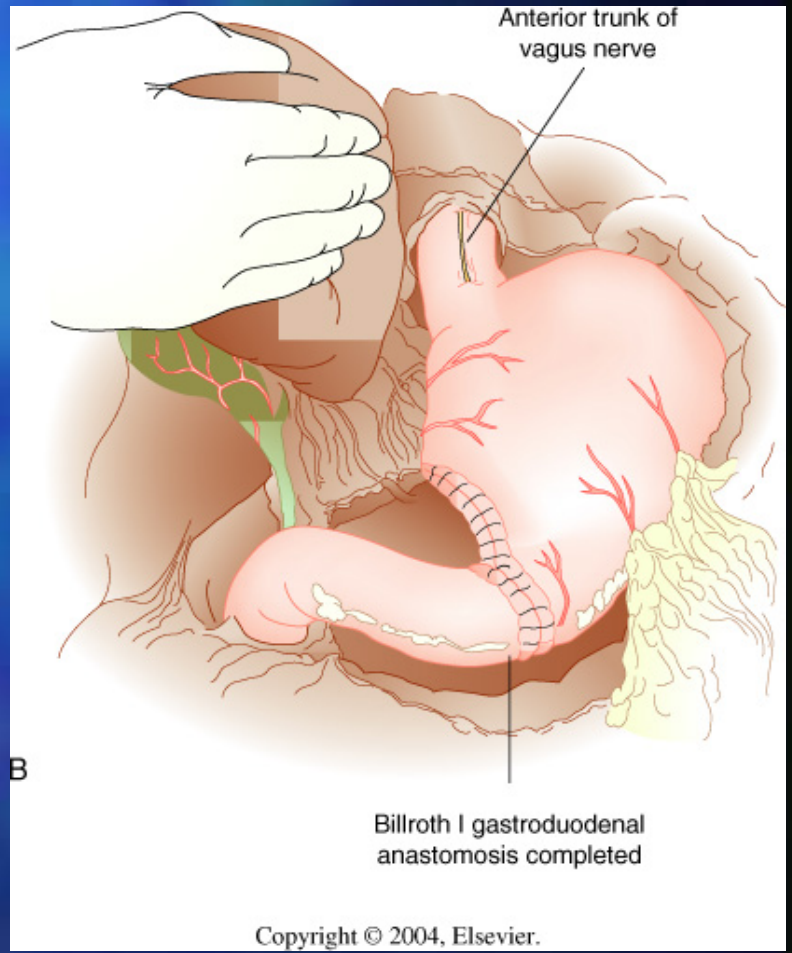
PUD: Surgical Options

- Truncal vagotomy and antrectomy
  - Indications: duod or gastric UD, large benign gastric TU
  - Contra indications: cirrhosis, extensive duodenal scarring, previous op of the prox duod
  - More effective than TV or HSV (recurrence rate 0-2%)
  - But, >>> post op complications (post vagot, post gastrec sds - 20%)



PUD: Surgical Options

- Truncal vagotomy and antrectomy
  - May reconstruct with BI or BII
  - BI is favored for benign dz
  - BII is favored if duodenum is significantly scarred



PUD: Surgical Options

- Subtotal Gastrectomy
  - Rare operation
  - Indicated for malignancies, recurrence after TV+A
  - Reconstruction with BII or Roux-en-Y

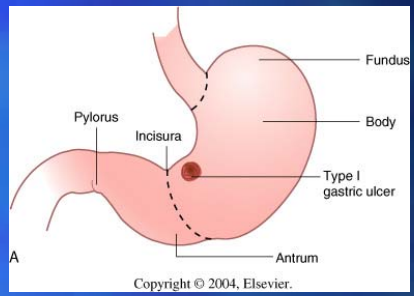
PUD: Surgical Indications

- Intractability
- Bleeding
- Perforation
- Obstruction

PUD: Surgical Indications

- Intractability: Failure of an ulcer to heal after 8-12 weeks of Tx or recurrence after Tx is discontinued
  - Duodenal ulcers
    - Very unusual
    - Do parietal cell vagotomy (? Laparoscopic)
      - Morbidity <1%
      - Mortality <0.5%
      - Recurrence: 5-25%
    - Some prefer Taylor procedure: laparoscopic posterior truncal vagotomy + seromyotomy across the ant portion of the stomach to divide vagal fibers coursing through the seromuscular layer

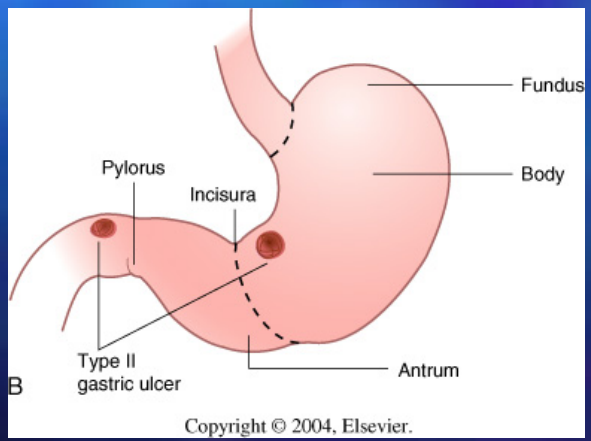
PUD: Surgical Indications

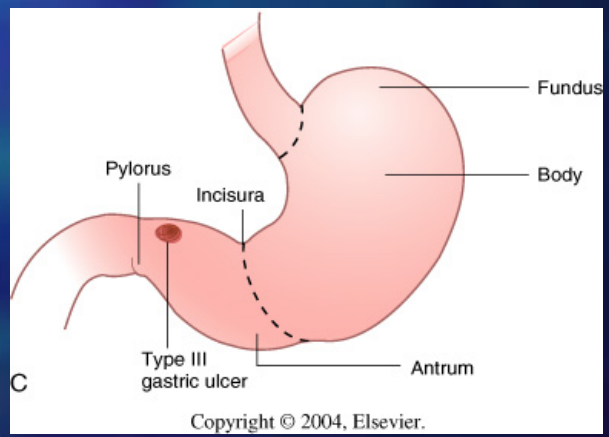


- Intractability
  - Type I gastric ulcer
    - Most will heal with appropriate medical Tx
    - Malignancy is a great concern
    - Surgery: distal gastrectomy + BI or BII
      - No need for vagotomy because is not dependent on acid secretion
      - BI is desired, as long as there is no malignancy
      - Morb: 3-5%; Mortal: 1-2%; Recur: < 2%
    - If malignant: subtotal gastrectomy + BII or ReY

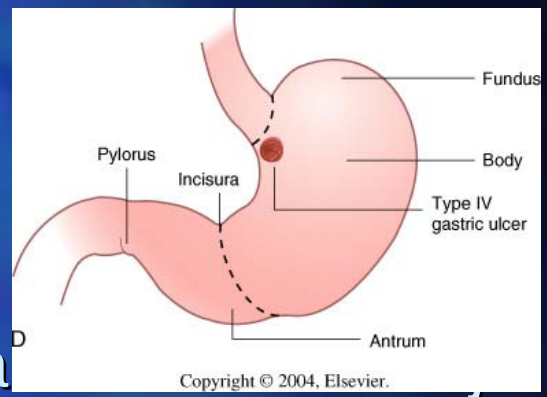
PUD: Surgical Indications

- Intractability
  - Type II or III gastric ulcer
    - Distal gastrectomy + vagotomy (selective or truncal)
    - HSV for these ulcers has poorer outcome
    - Option: laparoscopic HSV followed by resection IF recurrence





PUD: Surgical Indications



- Intractability
  - Type IV gastric ulcer
    - Tx depends on ulcer size, distal
      and degree of surrounding inflammation
    - Ulcer should be excised whenever possible
    - Distal gastrectomy including small portion of the esophageal wall and ulcer + ReY
    - For ulcers within 2-5 cm of GEj: distal gastrectomy + end-to-end gastroduodenostomy

PUD: Surgical Indications

- Bleeding ulcers
  - Duodenal
    - Fewer indications; sicker patients
    - Endoscopic treatment of recurrence of bleeding is safe
    - If failed conservative management:
      - Open duodenum and oversew the bleeding vessel
      - Truncal vagotomy preferred because of usual poor patient's condition
      - Gastrectomy is rarely indicated
    - Remember to dx/tx H. pylori

PUD: Surgical Indications

- Bleeding ulcers
  - Gastric
    - For bleeding type I: distal gastrectomy + BI
    - ? Adding vagotomy for patients who will remain on NSAIDs (also misoprostol or changing to COX2)
    - Type II and III: distal gastrectomy + vagotomy

PUD: Surgical Indications

- Perforated ulcers
  - Duodenal
    - Patching followed by Tx of H. pylori
    - Truncal vagotomy if patient is known not to be infected by H. pylori
    - Either laparoscopic or open procedure
    - ? Non-operative management ? (sicker patients were the ones who failed in Hong Kong)
  - Gastric
    - Type I in HD stable patients: distal gastrectomy + BI
    - Also simple patching after biopsy of the ulcer (malig)
    - Types II and III: treat as duodenal

PUD: Surgical Indications

- Gastric Outlet Obstruction (GOO)
  - More common with duodenal and type III gastric ulcers, but also type II
  - If type I should suspect of malignancy
  - Patients require pre op NG decompression for several days; also correction of metabolic abn
  - Acute: non op Tx with NGT and IV resuscitation
  - Chronic: surgery for desobstruction + acid reducing procedure (HSV + gastrojej)

PUD: Post Op Complications

- Mortality
  - Vagotomy w or wo antrectomy: < 1%
  - HSV: ~ 0.05%
- HSV has the lowest morbidity: ~1%
- Overall, ~25% develop some form of post gastrectomy syndrome; only ~1% remains permanently disabled
- Current trend is to avoid reoperation and use conservative Tx instead

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric resection
  - Dumping Syndromes
    - Early is more common than late
    - Disruption of the pyloric sphincter mechanism
    - Early and Late dumping syndromes
  - Metabolic disturbances

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric resection
  - Early Dumping
    - 20-30 min after a meal
    - GI s/s: N/V, sense of epigastric fullness, eructation, crampy abdominal pain, explosive diarrhea
    - CV s/s: tachycardia, palpitations, diaphoresis, fainting, dizziness, flushing, blurred vision
    - GI s/s are more common than CV
    - After any gastric resection, but >>> after gastrec + BII: 50-60% incidence

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric resection
  - Early Dumping
    - Mechanism is not completely understood:
      - Rapid passage of food of high osmolarity into the duod – rapid shift of extracellular fluid into the lumen – distention – autonomic response
      - s/s seem to be secondary to the release of several humoral agents, such as serotonin, bradykinin-like substances, neurotensin, enteroglucagon
    - Dx can be made on clinical presentation only. Otherwise:
      - Gastric emptying scans
      - Provocative test with 200cc of 50% glucose

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric resection
  - Early Dumping
    - Treatment
      - Most will respond to dietary measures
        - Avoid large amounts of sugar
        - Frequent feeding of small meals rich in prot and fat
        - Separating liquids from solids
      - Somatostatin
      - 1% who fail everything: surgery
        - Interposition of 10-20cm jejunal segment between stomach and SB. It dilates overtime and promotes reservoir function
        - Roux-en-Y

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric resection
  - Late Dumping
    - 2-3 hours after a meal
    - Rapid release of CH into the SB – quickly absorbed – hyperglycemia – overshooting of insulin secretion – hypoglycemia – release of catecholamines by adrenals
    - Diaphoresis, tremulousness, lightheadedness, tachycardia, confusion

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric resection
  - Late Dumping
    - Treatment
      - Dietary measures: similar to early dumping
      - Pectin or acarbose: delay CH absorption by impairment of intra luminal digestion
      - If everything fails: surgery – interposition of anti peristaltic jejunal loop

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric resection
  - Metabolic disturbances:
    - BII>BI; severity is proportional to extent of resection
    - Anemia
      - Iron
        - Most common (30% of patients)
        - Not fully understood: decrease iron intake, impaired absorption, chronic subclinical blood loss at margins of stoma
        - Oral supplements correct problem
      - Vitamin B12
        - Usually when resection > 50% stomach; rare with antrectomy
        - Megaloblastic anemia occurs 2nd to decr intrinsic factor
        - Treatment: IM cyanocobalamin

PUD: Post Op Complications

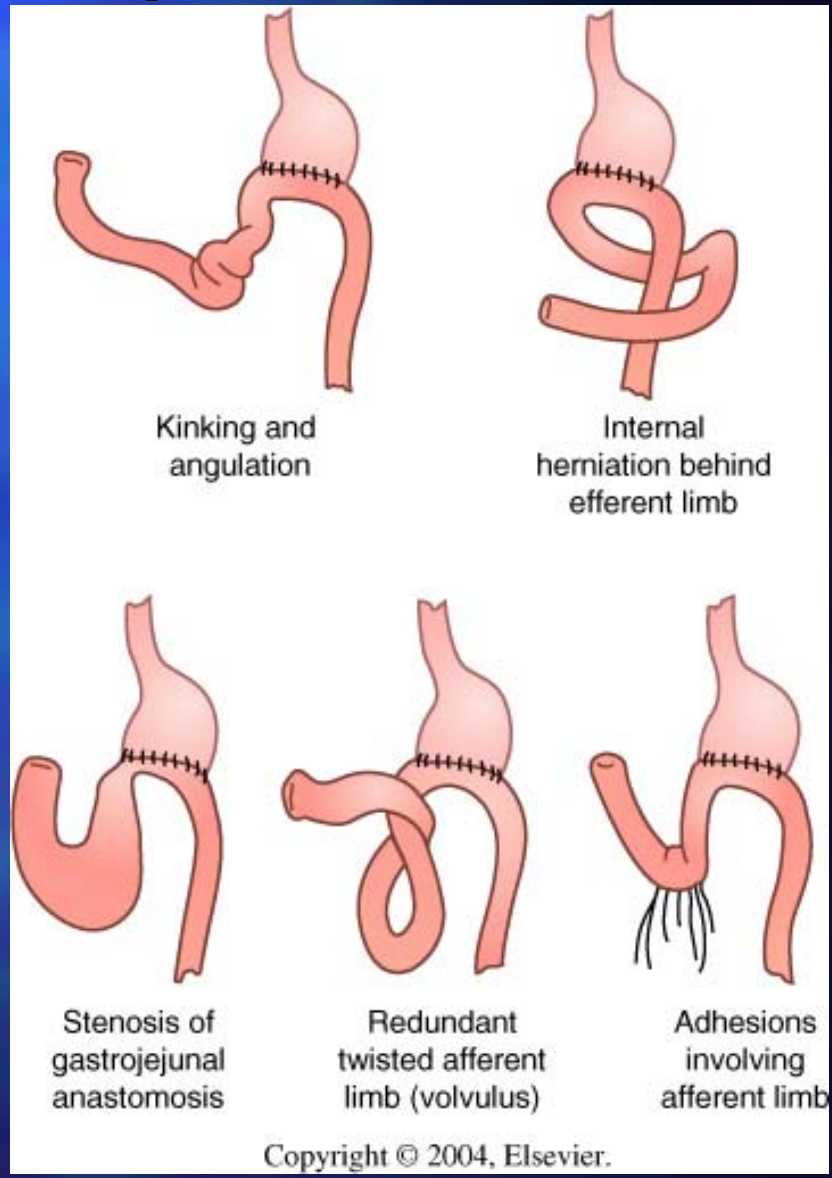
- Post Gastrec Synds 2nd to gastric resection
  - Metabolic disturbances:
    - Impaired absorption of fat
      - Inadequate mixing of bile salts and pancreatic lipase with ingested fat
      - May be associated with deficiency of lipid soluble vitamins
      - Tx: pancreatic enzymes if steatorrhea
    - Osteoporosis and osteomalacia
      - Decreased levels of calcium; may be worsened by impaired absorption of fat (saponification)
      - Bone disease usually starts 4-5 years after surgery
      - Tx: calcium supplement + vitamin D

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric reconstruc
  - Afferent loop syndrome
  - Efferent loop syndrome
  - Alkaline reflux gastritis
  - Retained antrum syndrome

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric reconstruc
  - Afferent loop syndrome
    - Partial obstruction of the afferent limb → accumulation of pancreatic and hepatobiliary secretion → distention → pain
    - ↑ pressure empties afferent loop → bilious vomiting with no food (stomach is already empty) → relief of symptoms
  - Chronic > acute; partial > complete
  - Perforation may occur if complete obstruction
  - More common when
    - Limb > 30-40 cm
    - Anastomosed to gastric remnant in an anticolic fashion



PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric reconstruc
  - Afferent loop syndrome
    - May be associated with blind loop syndrome (vit B12 deficiency)
    - Acute form may occur few days or many years post op
    - Diagnosis
      - barium study
      - EGD
      - ? radionucleotide studies of the biliary tree
    - Treatment of acute or chronic: surgery
      - BII → I or
      - BII → R-Y

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric reconstruc
  - Efferent Loop Obstruction
    - Rare
    - Most common cause: herniation of the limb behind the anastomosis R → L fashion
    - Can occur anytime, but >50% in 1st month post op
    - Diagnosis: barium study
    - Treatment: surgical
      - reduce hernia
      - close retroanastomotic space to prevent recurrence

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric reconstruc
  - Alkaline Reflux Gastritis
    - ↑↑ in BII
    - Reflux of bile is fairly common. In a small % of patients:
      - Severe epigastric pain; not relived by food or antacids
      - Bilious vomiting (@ anytime, even when sleeping)
      - Weight loss
    - Diagnosis:
      - HIDA scan (visible in stomach, esophagus)
      - EGD: send gastric fluid for analyses; friable and beefy mucosa
    - Medical treatment is usually unsuccessful
    - Surgery: BII → Roux-en-Y

PUD: Post Op Complications

- Post Gastrec Synds 2nd to gastric reconstruc
  - Retained Antrum Syndrome
    - Antral mucosa may extend 0.5 cm past the pyloric muscle (duodenal stump)
    - Retained antrum is continuously bathed on alkaline secretions
    - Comprises 9% of ulcer recurrences
    - Retained antrum will cause ulceration in 80% times
    - Diagnosis: technetium scan for gastric mucosa
    - Treatment
      - PPIs or H2 blockers
      - Surgery: BII → BI or excision of retained antrum

PUD: Post Op Complications

- Post Vagotomy Syndromes
  - Post vagotomy diarrhea
  - Post vagotomy gastric atony
  - Incomplete vagal transection

PUD: Post Op Complications

- Post Vagotomy Syndromes
  - Post vagotomy diarrhea
    - ~ 30% of patients after gastric surgery
    - Most are not severe and disappear in 3-4 months
    - If fails to disappear: cholestyramine
    - < 1% fail all the above and require surgery: interposition of 10 cm segment of reverse jejunum 70-100 cm from lig Treitz

PUD: Post Op Complications

- Post Vagotomy Syndromes
  - Post vagotomy diarrhea
    - Gastric emptying is delayed post op (except for highly selective)
      - Solids ↓↓ (loss of central pump)
      - Liquids ↑ (loss of receptive relaxation)
    - First exclude : DM, electrolyte imbalance, drug toxicity, neuromuscular disorders and mechanical causes
    - It's functional, not mechanical (adhesions, efferent/afferent loop obstruction, internal herniation)
    - Diagnosis confirmed by scintigraphic assessment of gastric emptying
    - EGD to rule out anastomotic blood
    - Tx is pharmocologic
      - Metoclopramide (dopamine antagonist)
      - Erythromycin (motilin receptors)

PUD: Post Op Complications

- Post Vagotomy Syndromes
  - Incomplete Vagal Transection
    - ↓ in HSV
    - ↑ in truncal (more common R - posterior, buried)
  - histologic confirmation of duodenal mucosa ↓ incidence

Stress Gastritis

- Occur after stressful situation: trauma, burns, shock, SEPSIS, hemorrhage, respiratory failure, etc
- Lesions are multiple, nonulcerating, begin in the proximal stomach and progress distally
- May be detected within hours after injury

Stress Gastritis

- Pathophysiology
  - Multifactorial etiology
  - Impaired mucosal defense
    - Reduction in blood flow
    - Reduction in bicarb secretion
    - Reduction in endogenous prostaglandins
  - There is little evidence that acid secretion ↑
  - Erosion of the mucosa into vessels -> bleeding
  - Mucosal injury may be far more common than bleeding

Stress Gastritis

- Presentation and Diagnosis
  - More than 50% develop in the first 1-2 days
  - Painless UGIB may be the only sign
    - Slow, intermitent
    - Unexplained drop in H/H
    - Stool is guaiac +, but melena/hematochezia are rare
    - Profound hemorrhage is much less common
    - Endoscopy is required for accurate diagnosis

Stress Gastritis

- Treatment
  - Prompt resuscitation: IVF, ? blood
  - Coagulation factors, platelets
  - NGT, NPO
  - Treatment of the primary cause
  - More than 80% will stop after above
  - Drugs (* Optimal pH: >5.0)
    - PPIs, H2Ri
    - Vasopressin: ↓ blood loss, does not ↑ survival
  - Angiography, embolization (?)
  - EGD has probably no benefit

Stress Gastritis

- Treatment
  - Surgery:
    - More than 6 units of blood (?)
    - Long anterior gastrostomy in the proximal stomach
    - Bleeding areas are oversewn with deep 8 stitches
    - Follow closure with truncal vagotomy and pyloroplasty
    - Total gastrectomy is rarely needed

Stress Gastritis

- Treatment
  - Prophylaxis:
    - Overall optimization of the patient
    - Feeding via enteral route
    - Drugs:
      - Antacids: 96% efficacy
      - H2 blockers: no advantage over antacids (? Cont inf)
      - Sucralfate: 90-97%
      - PPIs ?

Gastric Tumors

- Benign Tumors
  - Gastric polyps
  - Ectopic pancreas
- Malignant Tumors
  - Adenocarcinoma
  - Lymphoma
  - Sarcoma

Benign Gastric Tumors: Polyps

- Incidental finding on EGD: 2-3%
- Fundic gland polyps are 47% and have no malignant potential
- Familial adenomatous polyposis and Gardner's Sd: occur in 53%
- *** Colorectal neoplasms in up to 60% of patients with gastric polyps

Benign Gastric Tumors: Polyps

- Pathology
  - Hyperplastic polyps in 28-75%
    - Dysplastic changes may occur
    - AdenoCA in 2% of hyperplastic polyps
  - Adenomatous polyps: ~ 10%
    - More commonly antral, sessile, solitary, eroded
    - AdenoCa may be found in 21% (tubular->villous)
    - If larger than 4cm: 40% adeno CA
    - Also look for CA in other parts stomach (8-59%)

Benign Gastric Tumors: Polyps

- Treatment
  - Endoscopic polypectomy: sufficient if
    - Polyp is entirely removed
    - There is no malignancy
  - Operative management:
    - Sessile lesions > 2 cm
    - Polyps found to have areas of CA
    - Symptomatics polyps (pain, bleeding)

Benign Tumors: Ectopic Pancreas

- Implanted in the bowel during rotation and fusion of ventral/dorsal pancreas
- Incidence: 1-2% in autopsies
- Most patients are asymptomatic
- Others have s/s similar to PUD
- If mass can be seen on EGD biopsy can be attempted, but tissue is submucosal (endoscopic US can help
- Symptomatic tissue is treated surgically

Malignant Tumors: Adeno CA

- 2nd cancer in incidence worldwide
- 10th in the US and decreasing
- Geographic variation: Japan, South America
- 22,000 new pts/year; 13,000 will die
- 2 men: 1 woman; black > white
- Incidence increases with age: peak 7th dec
- Environmental exposure
- Shifting from distal to proximal stomach

Malignant Tumors: Adeno CA

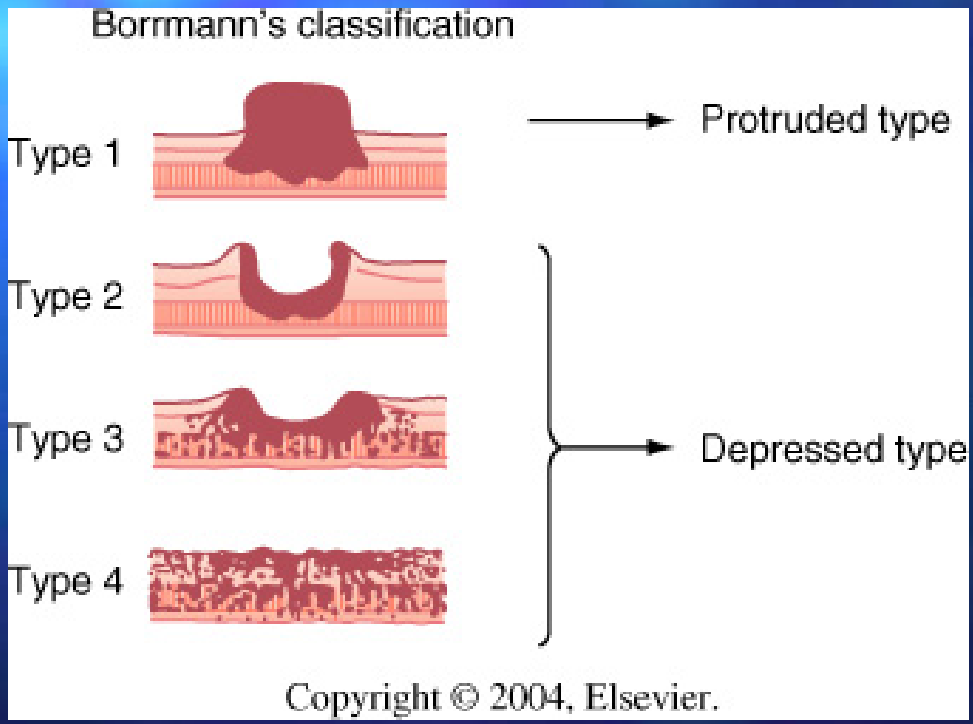
- Risk factors
  - Nutritional
    - Low in animal protein and fat, high in complex CH, high in salted meats and fish, high in nitrates
    - Lower risk: citric fruits, fibers, raw vegetables
  - Environmental
    - H pylori in drinking water
    - Poor food preparation: salted, smoked
    - Lack of refrigeration
    - Smoking

Malignant Tumors: Adeno CA

- Risk factors
  - Social
    - Low social class
  - Medical
    - Prior gastric surgery
    - H pylori infection
    - Gastric atrophy and gastritis
    - Adenomatous polyps
    - Male gender

Malignant Tumors: Adeno CA

- Pathology
  - 95% of all gastric CAs are adenocarcinomas
  - Borrmann's classification



Malignant Tumors: Adeno CA

- Pathology: Lauren Classification

- Intestinal
  - Environmental
  - Gastric atrophy, intestinal metaplasia
  - Men>women
  - Increasing incidence with age
  - Gland formation
  - Hematogenous spread
  - Microsatellite instability
  - APC gene mutations
  - P53, p16 inactivation

- Diffuse
  - Familial
  - Blood type A
  - Women>men
  - Younger age group
  - Poorly diff; signet ring cells
  - Transmural/lymphatic spread
  - Decreased E-cadherin
  - P53, p16 inactivation

Malignant Tumors: Adeno CA

- Clinical Presentation
  - Unspecific s/s -> delayed diagnosis
  - Vague epigastric discomfort/indigestion; may mimic PUD or angina
  - Pain is typically constant, nonradiating, not relieved by food
  - More advanced disease: weight loss, vomiting, anorexia, fatigue
  - GOO from distal tumors, dysphagia from proximal tumors

Malignant Tumors: Adeno CA

- Clinical Presentation
  - Clinically significant UGIB is rare (15% have hematemesis; 40% are anemic)
  - Erosion into the large bowel may obstruct colon
  - PE is unremarkable until too late; then:
    - Palpable abdominal mass
    - Virchow's or Sister Mary Joseph's lymph nodes
    - Blumer's shelf on rectal examination
    - Krukenberg's tumor: palpable ovarian mass
    - Etc with progression of disease

Malignant Tumors: Adeno CA

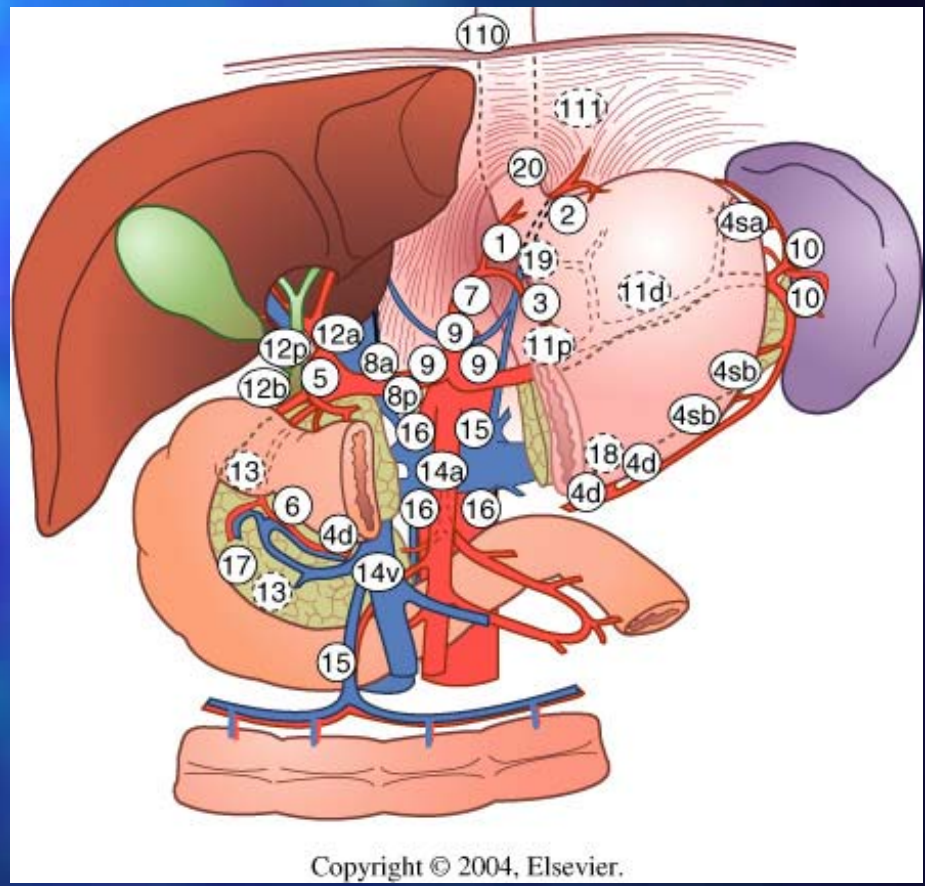
- Preoperative evaluation
  - EGD with multiple biopsies (>7; around crater): 98% sensitivity; ↑ with direct brush cytology
  - ? Endoscopic ultrasound: aids on staging, but cannot distinguish tumor from fibrosis
  - Preop labs
  - CXR, CT scan of the abdomen/pelvis (pelvic US in women)

Malignant Tumors: Adeno CA

- Preoperative evaluation
  - Laparoscopy:
    - To evaluate small macrometastases on the peritoneal surface of the liver: 23-37% of pts deemed “curable” by CT and others
    - MD Anderson: ↓ non therapeutic laparotomies by one fourth
    - Addition of lap US may increase sensitivity (?)
    - Cytology analysis of peritoneal fluid

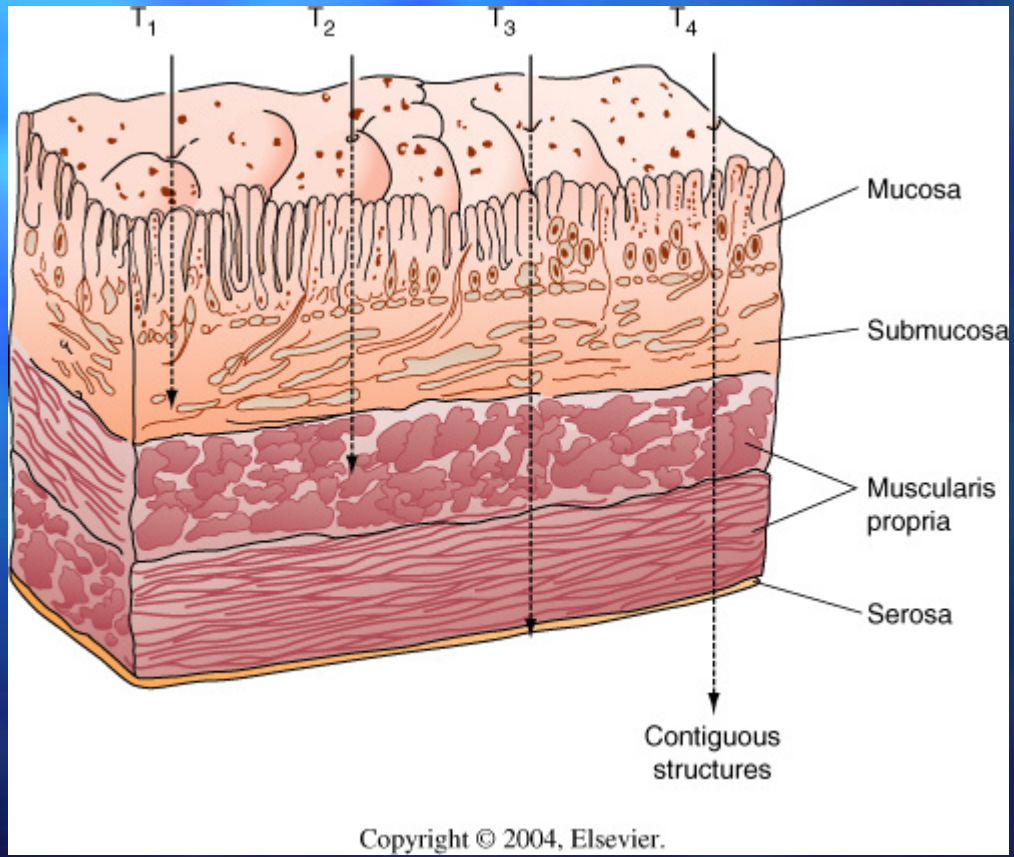
Malignant Tumors: Adeno CA

- Staging:
  - TNM
    - N
      - 1997 revision: number instead of location of LN
      - Need at least 15 LN for staging purposes
      - N1: 1-6; N2: 7-15; N3: >15



Malignant Tumors: Adeno CA

- Staging
  - TNM: T



Malignant Tumors: Adeno CA

- Staging
  - TNM
    - R: resection
      - R1: removal of all macroscopic disease but + margins
      - R2: gross residual disease

Malignant Tumors: Adeno CA

- Surgical Treatment
  - Aggressive surgical resection (in the absence of distant metastases)
  - Need negative margins: at least 6 cm from tumor margins (intramural spread)
  - Procedure: by location of tumor

Malignant Tumors: Adeno CA

- Surgical Treatment
  - Procedure: by location of tumor
    - Proximal tumors (more advanced): 35-50%
      - Total gastrectomy: 38/8% morb/mort
      - Proximal gastric resection: 52/16% morb/mort
    - Distal tumors: 35%
      - Subtotal gastrectomy
      - Total gastrectomy: no difference from subtotal (cure)

Malignant Tumors: Adeno CA

- Surgical Treatment
  - Extended lymphadenectomy
    - Controversial
    - Japanese
    - D1: group 1
    - D2: groups 1, 2
    - D3: D2 + para aortic lymph nodes
    - ? Spleen, parapancreatic by japanese only
    - higher Ds: higher morb/mort

Malignant Tumors: Adeno CA

- Palliative Treatment
  - 20-30% of CAs present with stage IV
  - Goal: relief of symptoms with minimal morbid
  - Complete staging is required for planning of palliation technique
  - Operative
    - Bypass
    - Resection
    - With or w/o endoscopic, percutaneous, radio therapeutic techniques
  - Non Operative
    - Laser recanalization
    - Endoscopic dilatation with or w/o stent placement

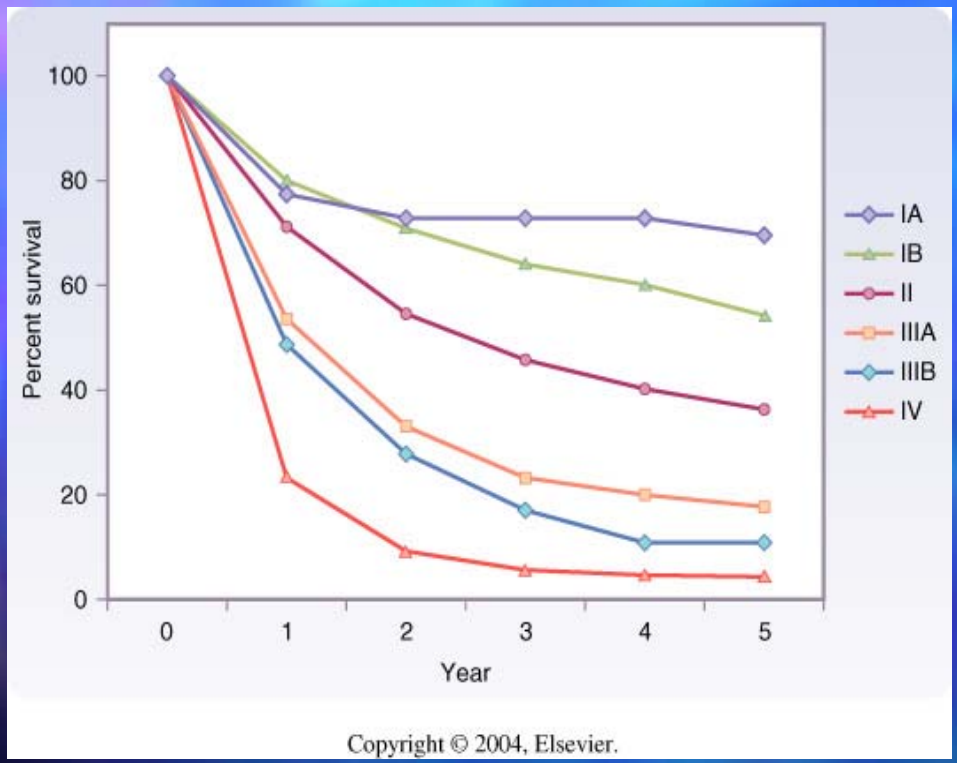
Malignant Tumors: Adeno CA

- Adjuvant Therapy
  - 1999: 71% surgery only
  - Southwest Cancer Oncology Group trial:
    - 5-fluoruracil and leucovorin + radiation for R0s
    - Median survival: 27->36 months
    - 3-year survival: 41->50%

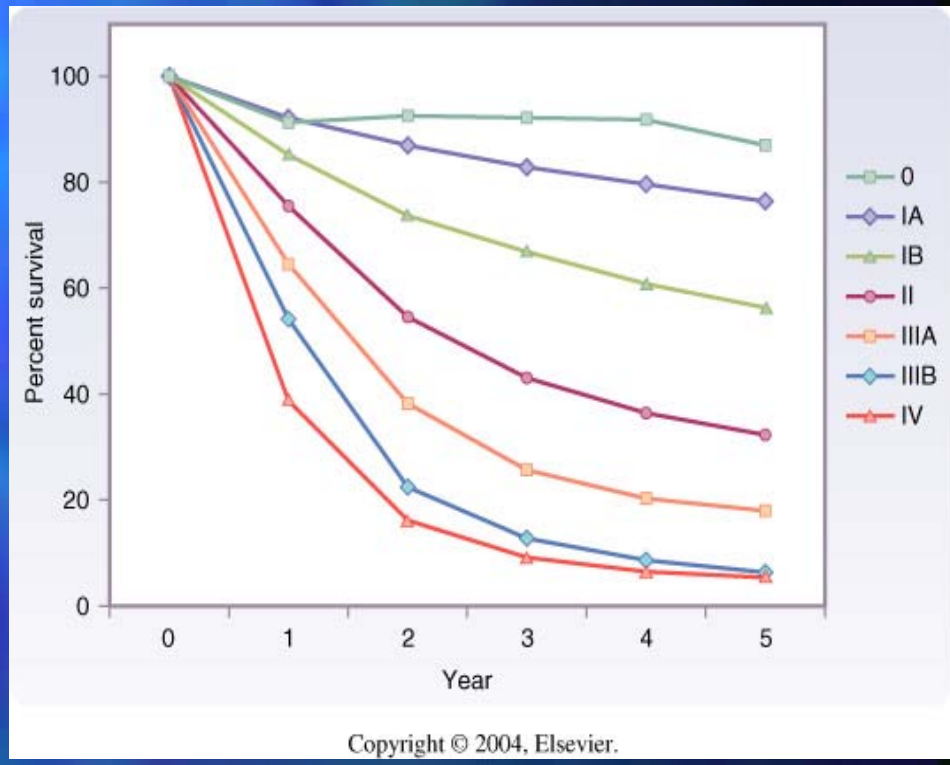
Malignant Tumors: Adeno CA

- Outcomes
  - Overall 5y survival after diagnosis: 10-21%
  - With potential for cure: 24-57% 5y survival
  - Recurrence after gastrectomy:
    - 40-80%
    - Most in the first 3 years
    - 38-45% locoregional:
      - gastric remnant at the anastomosis site
      - gastric bed
      - regional nodes
    - 54% peritoneal dissemination
    - Isolated distant metastases are uncommon

Malignant Tumors: Adeno CA



All patients with gastric CA



All patients with gastric CA + gastrectomy

Malignant Tumors: Adeno CA

- Surveillance
  - All patients need systematic follow up
  - Tighter in the first 3 years
  - H/P
    - Q 4 months x 1 year
    - Q 6 months x 2 years
    - Q year
  - ? Labs/xrays/cts
  - Endoscopy q year for subtotal gastrectomy

Malignant Tumors: Lymphoma

- Epidemiology
  - Stomach is the most common site in the GI sys
  - Primary gastric lymphoma is uncommon
    - Less than 15% of gastric malignancies
    - 2% of lymphomas
  - Older patients: 6th and 7th decades
  - 2 Males: 1 female
- Presentation
  - Vague s/s: epigastric pain, early satiety, fatigue
  - Constitutional B symptoms are rare
  - > 50% present with anemia, but UGIB is rare

Malignant Tumors: Lymphoma

- Pathology
  - Most commonly arise in the antrum
  - GASTRIC lymphoma is stomach is the exclusive or predominant site of disease
  - Histological sub types:
    - Diffuse large B-cell lymphoma (55%)
      - Most commonly are primary lesions
      - H pylori is a risk factor
    - Extranodal marginal cell lymphoma (MALT): 40%
      - H. pylori is important risk factor
    - Burkitt's lymphoma (3%)
      - Epstein Barr infection
      - younger ages
      - Cardia or body of the stomach
    - Mantle cell and follicular cell: <1% each

Malignant Tumors: Lymphoma

- Evaluation
  - EGD:
    - Nonspecific gastritis or ulcerations
    - Mass lesions are unusual
    - EUS to determine depth of invasion
  - Bone marrow biopsy
  - CT chest/abdomen: lymphadenopathy
  - Upper airway examination
  - H pylori screening

Malignant Tumors: Lymphoma

- Staging: controversial: ? TNM/others
- Treatment
  - Most patients now are treated with chemo (CHOP) and radiation alone
    - Risk of perforation after chemo: ~5%
    - Risk of complications after 30 Gy of radiation: 30% in 10y
    - Larger tumors limits usefulness of RT
  - Decreasing role for surgery
    - Complications from CT/RT: stricture, perforation, enteritis
    - Tissue for diagnosis
  - H pylori treatment alone may be sufficient for early B-cell and MALT (>75% cure)
    - Tight endoscopic follow up
    - Dormant lymphoma x disappearance

Malignant Tumors: Sarcomas

- Arise from mesenchymal components of the gastric wall
- About 3% of all gastric malignancies
- Gastrointestinal Stromal Tumors (GISTs)
  - c-kit mutation: transmembrane tyrosine kinase
- Staging
  - No current system
  - Mitotic frequency (0-5-50 /HPF)
  - Also:
    - > 5cm in size
    - Cellular atypia
    - Necrosis or local invasion

Malignant Tumors: Sarcomas

- Presentation and Evaluation
  - Most common forms of presentation
    - GI bleeding
    - Pain/dyspepsia
  - EGD: first diagnostic test; biopsy is positive in ~50% of the cases
  - CT: to evaluate true extension (intramural growth)

Malignant Tumors: Sarcomas

- Treatment
  - Surgery
    - Goal is a margin-free resection
    - Include en-bloc resection of adjacent organs
    - Frozen section intra op if diagnosis is uncertain
    - No lymphadenectomy needed
    - Most recurrences occur within 2 years: local disease + distant metastases (liver)
    - Salvage surgery for recurrences do not ↑ survival
  - Adjuvant therapy
    - Chemotherapy with Imatinib mesylate: approved for unresectable or recurrent disease only

Malignant Tumors: Sarcomas

- Prognosis
  - 5 year survival for GISTs: 48% (19-56%)
  - Survival after complete surgical resection: 32-63%
  - Worse if
    - Male sex
    - C-kit mutation
    - Mixed cytomorphology
    - Mitotic rate > 15 per 30 HPF

Other Gastric Lesions

- Hypertrophic Gastritis (Menetrier's Disease)
  - Hypoproteinemic hypertrophic gastropathy
  - Rare, pre malignant disease
  - Massive gastric folds in the fundus and corpus
  - Foveolar hyperplasia and no parietal cells on histology
  - Unknown cause: ? CMV in children/ ? H pylori

Other Gastric Lesions

- Hypertrophic Gastritis (Menetrier's Disease)
  - Epigastric pain, vomiting, weight loss, anorexia, peripheral edema
  - EGD to r/o CA or lymphoma
  - 24 h pH monitoring: hypo/achlorhydria
  - Chromium labeled albumin test: ↑ GI protein loss
  - Treatment
    - Medical:
      - Anticholinergics, acid suppression, octreotide, H pylori eradication
      - Inconsistent results
    - Surgical: total gastrectomy if continue to have massive protein loss, or dysplasia or CA

Other Gastric Lesions

- Dieulafoy's lesions
  - 0.3-7% of nonvariceal UGI bleeds
  - Bleeding is from an abnormally large (1-3 mm), tortuous artery coursing through the submucosa
  - Erosion of mucosa by arterial pulsation
  - Lesion usually is in the fundus near the cardia, 6-10 cm from the GEj
  - 2 men: 1 woman
  - Present with intermittent massive painless UGIBs

Other Gastric Lesions

- Dieulafoy's lesions
  - EGD diagnosis correctly in 80%; may need >1
  - May use EGD to treat as well
  - Angiography if unsuccessful with EGD
  - Surgical treatment:
    - Gastric wedge resection including artery
    - Difficult to locate vessel unless actively bleeding
    - ? laparoscopic

Other Gastric Lesions

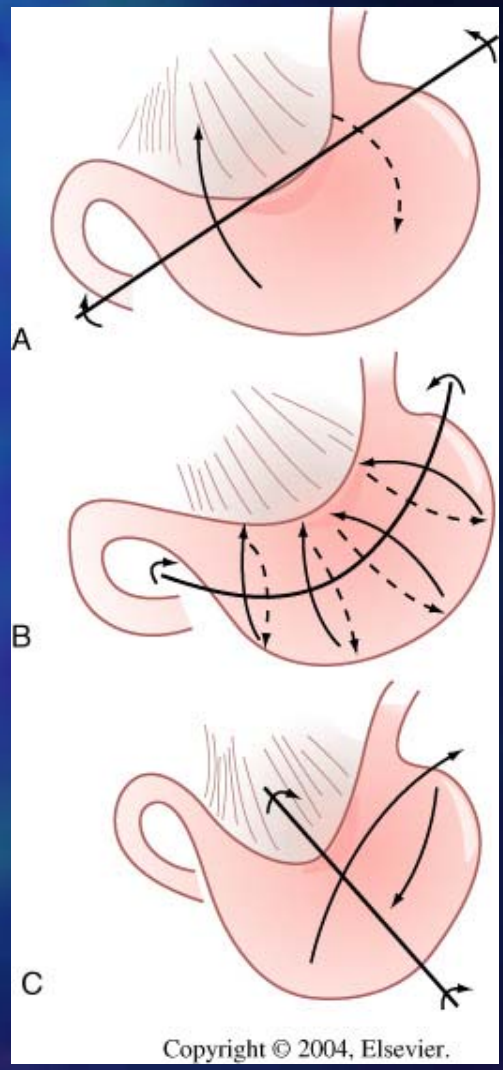
- Gastric Varices
  - Types
    - Gastroesophageal varices
    - Isolated gastric varices
      - Type 1: fundus
      - Type 2: isolated ectopic varices located anywhere in the stomach
  - Causes
    - Portal hypertension (more common)
    - Sinistral hypertension (splenic vein thrombosis)
      - Splenic blood flows retrograde through the short and posterior gastric veins into the varices -> coronary vein -> portal vein

Other Gastric Lesions

- Gastric Varices
  - Incidence of bleeding:
    - 3-30% in most series
    - As high as 78% with splenic vein thrombosis and fundic varices
  - Treatment
    - Splenic vein thrombosis: splenectomy
    - Portal hypertension:
      - Initially managed like esophageal varices
      - EGD: diagnostic and therapeutic (successful banding:89%)
      - TIPS
      - Management of portal hypertension

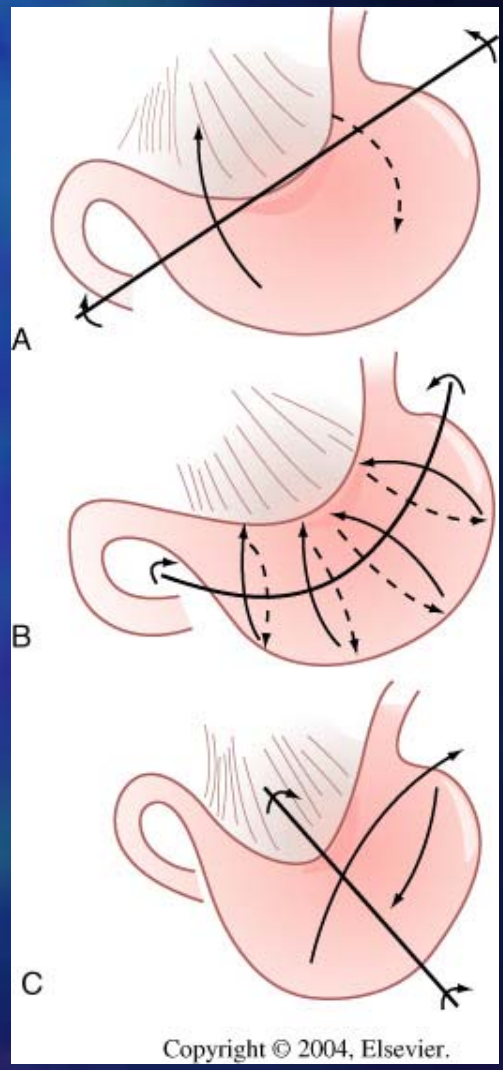
Other Gastric Lesions

- Gastric Volvulus
  - Very rare
  - 2/3 along the longitudinal axis (organoaxial)
    - Acute
    - Associated with diaphragmatic defect
  - 1/3 along the vertical axis (mesenteroaxial)
    - Incomplete (<180o)
    - Not associated with diaphragmatic defect



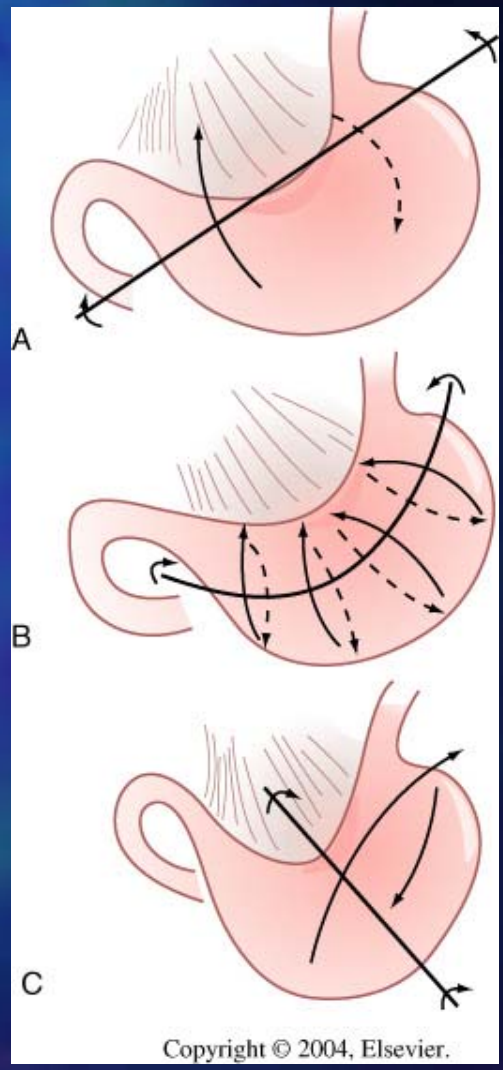
Other Gastric Lesions

- Gastric Volvulus
  - Presentation
    - Acute abdominal pain
    - Vomiting
    - Distention
    - UGI bleed
    - Borchardt's triad:
      - Acute onset of sudden and constant upper abdominal pain
      - Retching with little vomitus
      - Inability to pass a NGT



Other Gastric Lesions

- Gastric Volvulus
  - Diagnosis
    - CXR
    - Barium contrast study
    - EGD
  - Treatment
    - Is a surgical emergency
    - Uncoil stomach
    - Repair diaphragmatic defect
    - Resection for rare strangulation
    - Gastropexy if no diaphragmatic defect



Other Gastric Lesions

- Bezoars
  - Are collections of nondigestible materials, usually of
    - vegetable origin (phytobezoar) or
    - hair (trichobezoar)
  - Most commonly found after gastric resection with impaired gastric emptying
  - S/S: early satiety, n/v, pain, weight loss
  - Diagnosis by barium study or EGD

Other Gastric Lesions

- Bezoars
  - Treatment
    - Enzymatic therapy: papain (Adolph's Meat Tenderizer) or cellulase – followed by aggressive tube lavage or endoscopic fragmentation
    - Surgical removal