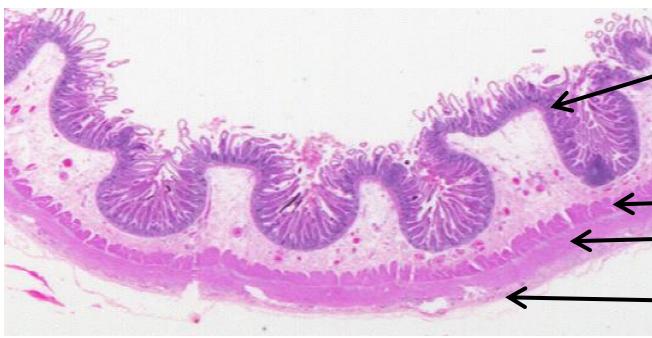
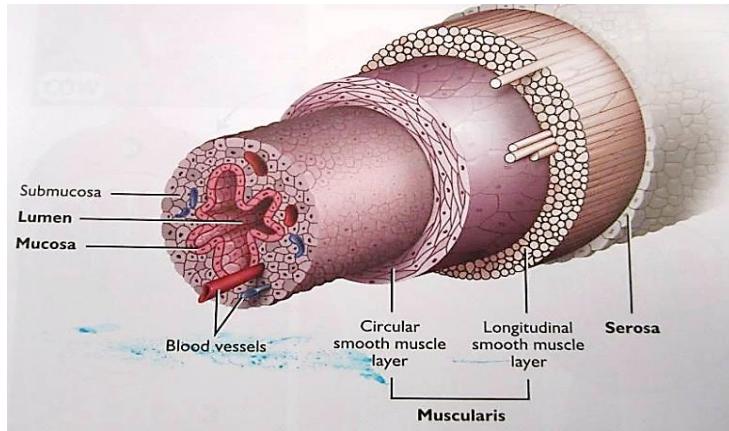


## BIOS 2015 ... CHAPTER 17- Digestive System Disorders

Page	Note
	Roles of Gastrointestinal (GI) System: 1. Digest food to a point that nutrients can be absorbed. 2. To absorb nutrients. 3. To participate in fluid balance, absorbing water from ingested fluids and solids.
	Nutrients are absorbed by active transport. Water is absorbed by osmosis.
	<b>Pathway of Digestion:</b> Digestion starts in mouth: ' teeth break up food. - salivary amylase digests sugar.
	Digestion continues in stomach: - stomach churns or mixes food till it becomes a liquid called "Chyme" - the digestion process uses acid and enzymes that digest proteins, examples: Pepsin and Trypsin
	Chyme is released into the small intestine (duodenum), this triggers several events: - cholecystokinin and secretin are hormones released by the intestinal cells that cause the gallbladder to contract and the pancreas to release pancreatic fluid. - the bile from the gallbladder contains detergents that break up fat. The bile also contains toxins and waste that the liver has filtered from the blood and put in the bile for elimination in the feces. - the pancreatic fluid contains an enzyme, lipase, that breaks up fat, but it also is rich in bicarbonate that neutralizes the stomach acid and leaves the fluid basic or alkaline (this is why diarrhea loses base and can result in metabolic acidosis).
	After the duodenum, fluid passes into the jejunum and ileum (parts of the small intestine) where nutrients are absorbed by active transport.
	Food then goes into the colon where water is absorbed and a solid stool is formed.
	<b>Anatomy of the gut:</b>
	 <p>         Mucosal lining cells (mucosa)          Submucosa          Muscularis          Inner circular          outer longitudinal       </p>

	The mucosa is different in the esophagus, stomach, small intestine and large intestine, but all have the same basic architecture of mucosa, submucosa, muscularis, and serosa.
	The muscularis also varies by region.
	Upper esophagus - striated muscle
	Lower esophagus- smooth muscle
	Stomach - 3 layers of smooth muscle (less well layered than intestine)
	Small and large intestine - 2 layers of smooth muscle (inner circular, outer longitudinal)



Circular muscle cells wrap around the long axis of the intestine (like your fingers wrap around a drinking glass).

Longitudinal muscle cells run perpendicular to circular cells and run down the long axis of the intestine (like vertical stripes down a pair of pants).

#### Role of Liver:

- filters blood draining from the intestines into the hepatic portal vein.
- removes toxins, drugs, and waste and puts them in the bile for excretion in the feces.
- makes bile for digestion of fat.
- stores carbohydrates as glycogen
- processes carbohydrate, protein and fat.
- produces plasma proteins and clotting factors.
- role in red blood cell breakdown by conjugating (adding sugar molecules to) bilirubin.

#### Neural Controls:

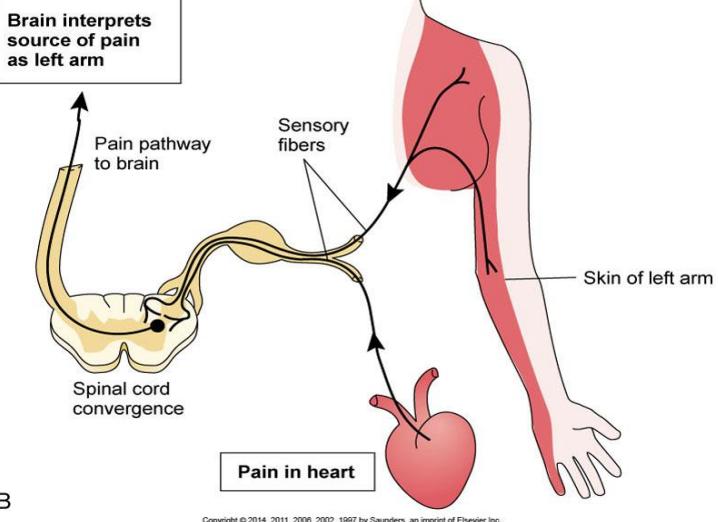
Autonomic nervous system (sympathetic and parasympathetic) act in an opposite fashion to how they affect the heart because of you are in a fight or flight mode, you do not want blood diverted to the intestines for digestion.

Sympathetic: speeds up heart but slows down gut.

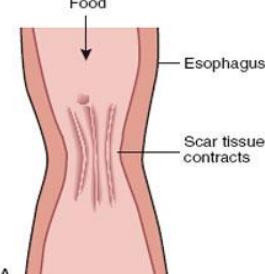
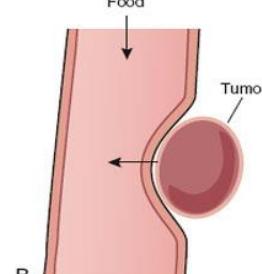
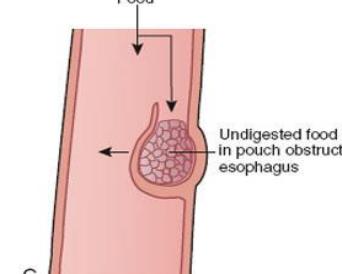
Parasympathetic (vagus nerve): slows down heart but speeds up gut.

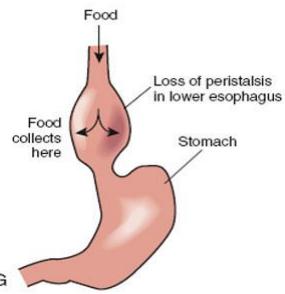
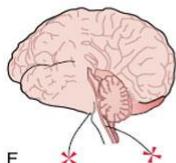
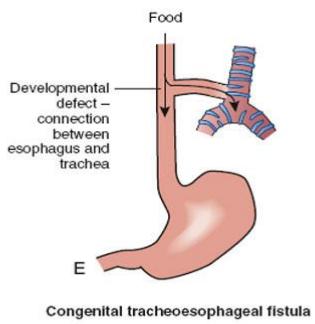
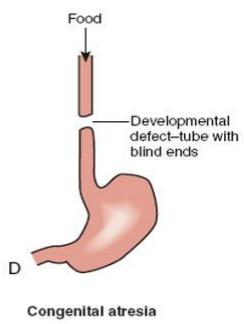
Hormonal Control:			
Hormone	Source	Stimulus	Effects
Gastrin	Gastric cells	Food in the stomach Protein, caffeine, or high pH of chyme	Increases gastric secretions and motility and promotes gastric emptying
Cholecystokinin	Intestinal mucosal cells	Protein and fat in the duodenum	Inhibits gastric secretions and motility; stimulates pancreatic enzyme secretion; stimulates gallbladder contractions and release of bile
Secretin	Intestinal mucosal cells	Acidic chyme in the duodenum	Stimulates bile and pancreatic secretions with high bicarbonate content
Neural Controls	Source	Stimulus	Effects
Parasympathetic nervous system	Vagus nerve	Taste food	Increases secretions and peristalsis
Sympathetic nervous system	SNS	Stress	Decreases secretions and peristalsis Stimulates vasoconstriction in the mucosa
<p><b>Note:</b> that cholecystokinin and secretin not only affect the gall bladder and pancreas but also signal the stomach to slow down so that the latest delivery from the stomach to the duodenum can be processed.</p>			
<b>Vitamins:</b> Fat Soluble: A,D,E,K (these are stored in fat, so overconsumption can lead to toxicity).  Water Soluble: B and C (these are readily lost in the urine, B vitamins known for turning the urine bright yellow).			
<b>GI Symptoms:</b> <b>Vague GI symptoms like anorexia, nausea and vomiting</b> can be triggered by a wide variety of conditions:			
Systemic infection Emotional responses Motion sickness Pressure in the brain Overindulgence of food, drugs Pain			
Vomiting center in brain (medulla) coordinates a number of events that must be orchestrated to achieve vomiting - sending food backwards in the GI tract.			
Different color and texture in vomit may give information: - blood in the stomach is digested by acid and forms "Coffee ground emesis" - Bile reflux into the stomach can cause green or yellow stained vomit. - Dark brown vomit suggests origin from the small intestines.			
Repeated vomiting may be a sign of an intestinal obstruction.			

	<b>Diarrhea:</b>
	<p><b>Large-volume diarrhea (secretory or osmotic)</b></p> <ul style="list-style-type: none"> <li>- Watery stool resulting from increased secretions into intestine from the plasma</li> <li>- Often related to infection</li> <li>- Limited reabsorption because of reversal of normal carriers for sodium and/or glucose</li> </ul> <p><b>Small-volume diarrhea</b></p> <ul style="list-style-type: none"> <li>- Often caused by inflammatory bowel disease</li> <li>- Stool may contain blood, mucus, pus</li> <li>- May be accompanied by abdominal cramps and tenesmus</li> </ul>
	<b>Steatorrhea:</b> Fatty diarrhea, bulky greasy loose stool with foul odor (and floats). Seen in malabsorption in Celiac disease and Cystic fibrosis.
	<b>Blood in Stool:</b>
	<p>Blood may occur in normal stools with diarrhea, constipation, tumors, or an inflammatory condition.</p> <p><b>Frank blood</b></p> <ul style="list-style-type: none"> <li>- Red blood—usually from lesions in rectum or anal canal</li> </ul> <p><b>Occult blood</b></p> <ul style="list-style-type: none"> <li>- Small hidden amounts, detectable with stool test</li> <li>- May be caused by small bleeding ulcers</li> </ul> <p><b>Melena</b></p> <ul style="list-style-type: none"> <li>- Dark-colored, tarry stool</li> <li>- May result from significant bleeding in upper digestive tract</li> </ul>
	<b>Motility and stool consistency:</b>
	<p>It takes time for water to be absorbed from material in the colon to form a desirable consistency to stool.</p> <p>If peristalsis is too rapid, not enough water is absorbed and the stool is loose.</p> <p>If peristalsis is too slow, too much fluid is absorbed and the stool is hard (constipation).</p>
	Fluid and Electrolytes:
	Vomiting loses acid >>> metabolic alkalosis.
	Diarrhea loses base (bicarbonate) >>> metabolic acidosis.
	<b>PAIN:</b>
	<b>Visceral Pain (often has vague associations).</b>
	Burning, epigastric: stomach or duodenal ulcer.
	Dull aching in right upper quadrant: stretching of liver capsule.
	Diffuse cramping pain: Distension of intestines, gas is a typical cause.
	<b>Somatic Pain (more focused and localized).</b>
	- local inflammation may involve peritoneum.
	- peritoneal inflammation may show "rebound tenderness" - push in on abdomen and rapidly release, causes a sharp pain. Commonly seen in appendicitis.
	<b>Acute Appendicitis:</b>
	Generalized abdominal pain followed by localization with point tenderness in right lower quadrant with associated rebound tenderness.

<p><b>Referred Pain:</b></p>	<ul style="list-style-type: none"> <li>- Pain is perceived at a site different from origin.</li> <li>- Results when visceral and somatic nerves converge at one spinal cord level</li> <li>Source of visceral pain is perceived as the same as that of the somatic nerve (see diagram below).</li> <li>- May assist or delay diagnosis, depending on problem</li> </ul>
	 <p><b>Classic Example of Referred Pain:</b> Left Arm Pain when having a heart attack.</p> <p>Visceral nerve impulses from the heart activate sensory nerves from the arm and make the brain think there is arm pain and that is what you feel.</p>
<p><b>Classic Pain Associations to Remember:</b></p>	
	<ol style="list-style-type: none"> <li>1. Pain in left arm and shoulder. Cardiac pain (heart attack).</li> <li>2. Pain in right lower quadrant of abdomen: Appendix (acute appendicitis).</li> <li>3. Pain in right upper quadrant of abdomen: Gall Bladder (cholecystitis).</li> <li>4. Epigastric pain: Duodenum (acute ulcer), Pancreas (acute pancreatitis).</li> <li>5. Pain in left lower quadrant of abdomen: Sigmoid colon (diverticulitis).</li> <li>6. Back pain: Kidneys (kidney stones).</li> </ol>
<p><b>Drugs used in Digestive Disorders:</b></p>	
	<p><b>Antacids and Proton pump inhibitors (PPI):</b></p> <ul style="list-style-type: none"> <li>- To relieve gastric pain.</li> <li>- Reduce gastric secretion (PPI).</li> </ul> <p><b>Antiemetics (Anti + Emesis, Emesis is vomiting):</b></p> <ul style="list-style-type: none"> <li>- To relieve vomiting (often by dulling nausea)</li> </ul> <p><b>Laxatives or enemas:</b></p> <ul style="list-style-type: none"> <li>- Treatment of acute constipation</li> </ul> <p><b>Antidiarrheals:</b></p> <ul style="list-style-type: none"> <li>- Reduction of peristalsis</li> <li>- Relieve cramps</li> </ul>

	<b>Disorders of Oral Cavity</b>
	<b>Cleft lip and palate:</b>
 	<p>Congenital abnormalities      Cleft lip and cleft palate  <b>Feeding problems of the infant</b>  <b>High risk of aspirating fluid into respiratory passages</b>  <b>Speech development impaired</b>      Surgical repair done as soon as possible      Therapy with speech-language pathologist and orthodontist</p>
	<b>Aphthous ulcers:</b>
	<p>Inner lip, small painful, self limited,      possibly Strep. sanguis</p>
	<b>Oral Candidiasis (Thrush):</b>
	<p>Patchy white plaques in mouth and on tongue.      Grows from local flora.      Opportunistic infection.      Seen with certain antibiotics, chemotherapy and</p>
	<b>Oral Herpes (HSV type I):</b>
	<p>Herpes simplex virus type 1 (HSV-1)  <b>Transmitted by kissing or close contact</b>  <b>Virus remains dormant in sensory ganglion</b>  <b>Activated by stress, trauma, other infection</b>      Formation of blisters, ulcers, clear fluid release—      contains virus; can be autoinoculated to other areas      Lesions heal spontaneously in 7 to 10 days.      Acute stage may be alleviated by antiviral medication.      May spread to eyes: Conjunctivitis and keratitis</p>

	<p><b>Syphilis (caused by spirochete <i>Treponema pallidum</i>):</b></p> <p>May cause oral lesions  <b>Highly contagious during first and second stages</b></p> <p><b>Primary stage</b>  <b>Chancre</b>, a painless ulcer on tongue, lip, palate      Heals spontaneously (1 or 2 weeks)</p> <p><b>Secondary stage</b>  <b>Red macules or papules on palate</b>—highly infectious      Heals spontaneously      Both stages treated with long-acting penicillin</p>
	<p><b>Dental Caries (Cavities in teeth):</b></p> <p>Bacteria break down sugar to acid, and acid dissolves mineral in tooth enamel.</p>
	<p>Gingivitis - inflammation of the gums</p> <p>Periodontitis - inflammation of deeper tooth structures around roots.</p>
	<p><b>Hyperkeratosis - Leukoplakia:</b></p>  <div style="border: 1px solid black; padding: 5px; margin-left: 10px;"> <p>Whitish plaque or epidermal thickening of mucosa      Occurs on buccal mucosa, palate, lower lip      May be related to smoking or chronic irritation      Lesions require monitoring.  <b>Epithelial dysplasia beneath plaque may develop into</b></p> </div>
	<p><b>Sialadenitis</b> - inflammation of salivary glands.</p>
	<p><b>Dysphagia</b> - difficult swallowing, various causes.</p> <div style="display: flex; justify-content: space-around; align-items: flex-end;"> <div style="text-align: center;">  <p><b>Fibrosis</b></p> </div> <div style="text-align: center;">  <p><b>Compression</b></p> </div> <div style="text-align: center;">  <p><b>Diverticulum</b></p> </div> </div> <p style="text-align: center; font-size: small;">Copyright © 2014, 2011, 2006, 2002, 1997 by Saunders, an imprint of Elsevier Inc.</p>



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### Esophageal Cancer:

Associated with chronic irritation because of:

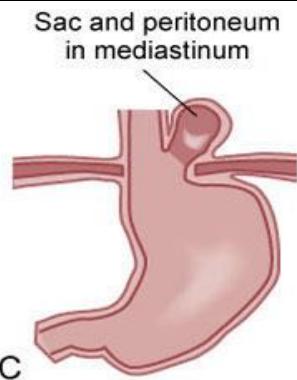
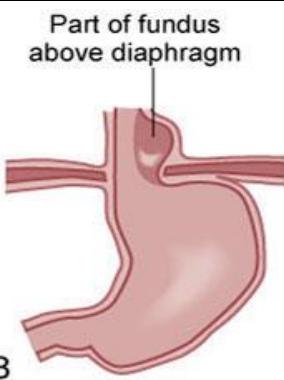
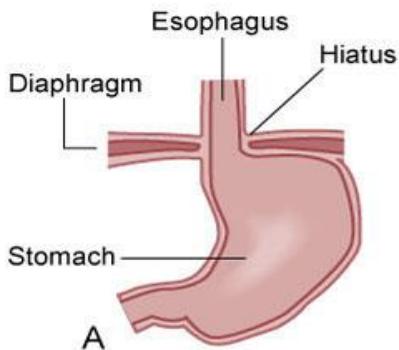
Chronic esophagitis

Achalasia

Hiatal hernia

Alcohol abuse, smoking

### Hiatal Hernia (Part of the stomach protrudes into the thoracic cavity):

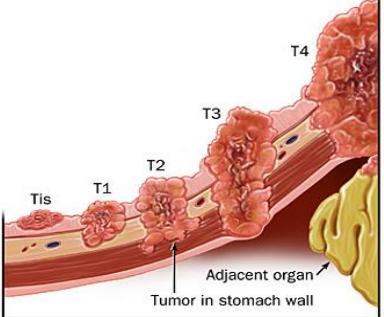
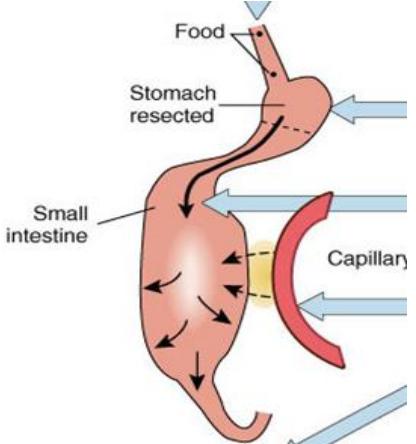


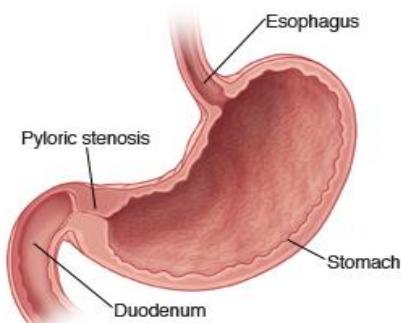
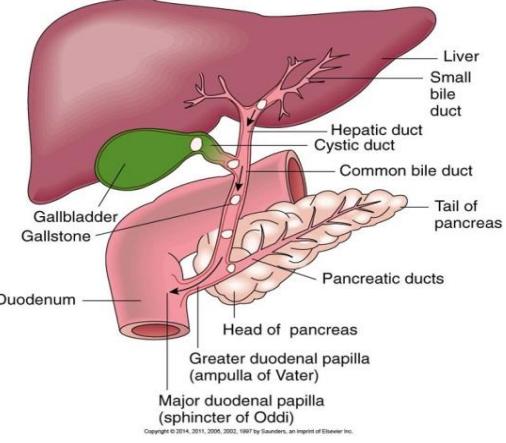
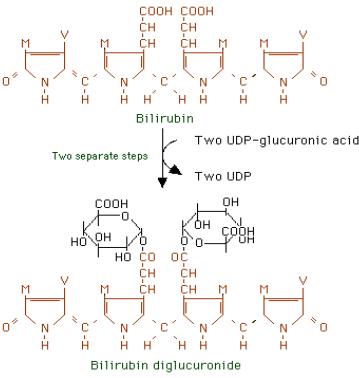
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### Gastroesophageal Reflux Disease:

- Periodic reflux of gastric contents into distal esophagus causes erosion and inflammation

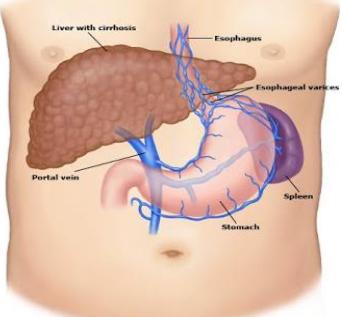
	<b>Acute Gastritis (many causes):</b>
	<p><b>Infection by microorganisms</b>            Allergies to foods            Spicy or irritating foods</p> <p><b>Excessive alcohol intake</b></p> <p><b>Ingestion of aspirin or other NSAIDs</b>            Ingestion of corrosive or toxic substances            Radiation or chemotherapy</p>
	<b>Chronic Gastritis:</b>
	<p>Characterized by atrophy of stomach mucosa            Loss of secretory glands</p> <p><b>Reduced production of intrinsic factor</b></p> <p><b>Helicobacter pylori infection is often present.</b>            Signs may be vague.            Mild epigastric discomfort, anorexia, intolerance for certain foods</p> <p><b>Increased risk of peptic ulcers and gastric carcinoma</b>  <b>Certain autoimmune disorders are associated with one type of chronic gastric atrophy.</b></p>
	<b>Escherichia coli Infection</b>
	<p>Although E. coli is usually harmless as a resident in the human intestine, infective strains can cause significant problems.</p> <p><b>Infective strains</b></p> <p>Enterotoxigenic E. coli            Enteroinvasive E. coli            Enteropathogenic E. coli            Enteroaggregative E. coli            Enterohemorrhagic E. coli</p>
	<b>Peptic Ulcer: Gastric and Duodenal Ulcers</b>
	<p><b>Most caused by H. pylori infection</b>            Usually occur in the proximal duodenum (duodenal ulcers)            Also found in the antrum of the stomach (gastric ulcers)            Development begins with breakdown of mucosal barrier            Decreased mucosal defense            More common in gastric ulcer development</p> <p><b>Increased acid secretion predominant factor in duodenal ulcers</b></p>

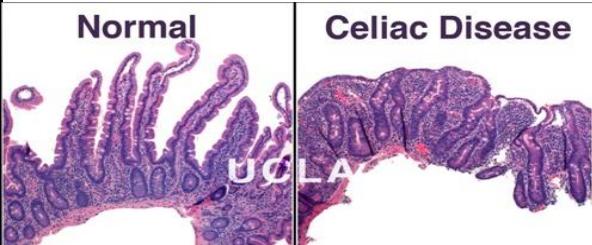
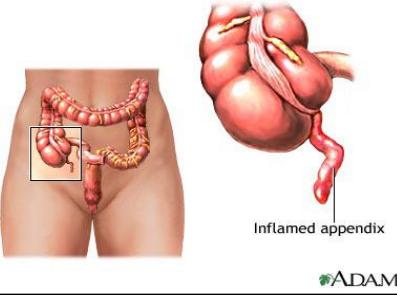
	<p><b>Damage to mucosal barrier predisposes to development of ulcers and is associated with:</b></p> <ol style="list-style-type: none"> <li><b>Inadequate blood supply</b> <ul style="list-style-type: none"> <li>- Caused by <b>vasoconstriction</b> (e.g., <b>stress, smoking, shock</b>, circulatory impairment in older adults, scar tissue, anemia)</li> <li>- Interferes with rapid regeneration of epithelium</li> </ul> </li> <li>Excessive glucocorticoid secretion or medication</li> <li><b>Ulcerogenic substances</b> break down mucous layer.           <ul style="list-style-type: none"> <li>- <b>Aspirin, NSAIDs, alcohol</b></li> </ul> </li> <li>Atrophy of gastric mucosa           <ul style="list-style-type: none"> <li>- Chronic gastritis</li> </ul> </li> </ol>
	<p><b>Increased acid pepsin secretions</b></p> <ul style="list-style-type: none"> <li>- Increased gastrin secretion</li> <li>- Increased vagal stimulation</li> </ul>
	<p><b>Complications of peptic ulcer</b></p> <p><b>Hemorrhage</b></p> <p><b>Perforation</b></p> <ul style="list-style-type: none"> <li>- Ulcer erodes completely through the wall.</li> <li>- Chyme can enter the peritoneal cavity.</li> <li>- Results in chemical peritonitis</li> </ul> <p><b>Obstruction</b></p> <ul style="list-style-type: none"> <li>- May result later because of the formation of scar tissue</li> </ul>
	<p><b>Gastric Carcinoma:</b></p> <div style="display: flex; align-items: center;">  <div style="margin-left: 20px;"> <p><b>Diet seems to be a key factor</b>, particularly <b>smoked foods, nitrites, and nitrates</b>.</p> <p><b>Early carcinoma</b></p> <ul style="list-style-type: none"> <li>- Confined to mucosa and submucosa</li> </ul> <p><b>Later stages</b></p> <ul style="list-style-type: none"> <li>- Infiltrates the muscularis</li> </ul> <p><b>Asymptomatic in the early stages</b></p> <ul style="list-style-type: none"> <li>- prognosis is poor on diagnosis</li> <li>- survival rate less than 20%</li> </ul> </div> </div>
	<p><b>Dumping:</b></p> <div style="display: flex; align-items: center;">  <div style="margin-left: 20px;"> <p>Control of gastric emptying is lost, and gastric contents are “dumped” into the duodenum without complete digestion.</p> <p><b>May follow gastric resection</b></p> <p><b>Effects:</b></p> <p>Hyperosmolar chyme draws fluid from vascular compartment into intestine causing intestinal distention and <b>decreased blood pressure</b>.</p> <p>High glucose in chyme leads to insulin release and subsequent <b>hypoglycemia</b>.</p> <p>Give frequent small meals—high in protein, low in simple carbohydrates</p> </div> </div>

<p><b>Pyloric Stenosis:</b></p>	 <p>Narrowing and obstruction of pyloric sphincter May be developmental anomaly <b>Signs appear within several weeks after birth.</b>  <ul style="list-style-type: none"> <li>- <b>Projectile vomiting immediately after feeding</b></li> <li>- <b>Firm mass can be palpated at pylorus.</b></li> <li>- Infant fails to gain weight, dehydration, persistent hunger</li> </ul> <p>Surgery required to relieve the obstruction.</p> </p>
<p><b>Gallstones:</b></p>	 <p><b>YELLOW - Cholesterol stones</b>  <b>Women (2 x men)</b>  <b>High cholesterol in bile (cholesterol stones)</b>  <b>High cholesterol intake</b>  <b>Obesity, Multiparity.</b></p>
	<p><b>BLACK - Bilirubinate stones</b>  <b>Associated with hemolytic anemia.</b></p>
	<p><b>Cholelithiasis</b>  <ul style="list-style-type: none"> <li>- Formation of gallstones</li> <li>- Solid material (calculi) that form in bile</li> </ul> <b>Choledocholithiasis</b>  <ul style="list-style-type: none"> <li>- Obstruction of the biliary tract by gallstones</li> </ul> <b>Cholecystitis</b>  <ul style="list-style-type: none"> <li>- Inflammation of gallbladder and cystic duct</li> </ul> <b>Cholangitis</b>  <ul style="list-style-type: none"> <li>- Inflammation usually related to infection of bile ducts</li> </ul> </p>
	<p>Unconjugated (indirect) bilirubin is processed in the liver to make Conjugated (direct) bilirubin.</p> <p>Total bilirubin = direct + indirect bilirubin.</p> <p>Indirect bilirubin is calculated from measurements of total and direct bilirubin.</p>

	<p><b>Jaundice:</b></p> <div style="border: 1px solid black; padding: 10px;"> <p><b>Prehepatic jaundice</b></p> <ul style="list-style-type: none"> <li>- Result of <b>excessive destruction of red blood cells</b></li> <li>- Characteristic of hemolytic anemias or transfusion reactions</li> <li>- <b>Rise in indirect bilirubin.</b></li> </ul> <p><b>Intrahepatic jaundice</b></p> <ul style="list-style-type: none"> <li>- Occurs with <b>disease or damage to hepatocytes</b></li> <li>- Hepatitis or cirrhosis</li> <li>- <b>Rise in direct and/or indirect bilirubin.</b></li> </ul> <p><b>Posthepatic jaundice</b></p> <ul style="list-style-type: none"> <li>- Caused by <b>obstruction of bile flow</b> into gallbladder or duodenum</li> <li>- <b>Tumor, cholelithiasis</b></li> </ul> </div>
	<p><b>Hepatitis:</b></p> <p>Alcoholic (steatohepatitis, hepatitis with fatty change).</p> <p>Viral hepatitis</p> <p>Types A, B, C, D, E</p> <p>Chemical or drug toxicity</p> <p>Idiopathic (cause unknown).</p>
	<p><b>Hepatitis A (HAV)</b></p> <p>Small RNA virus</p> <p><b>Infectious hepatitis</b>, transmitted by <b>fecal-oral route</b></p> <p>Acute but self-limiting infection, <b>No carrier or chronic state.</b></p> <p>Vaccine available.</p> <p><b>Hepatitis B (HBV)</b></p> <p>Partially double-stranded DNA virus</p> <p>Transmitted by <b>IV Rx abuse and sexual intercourse.</b></p> <p>Usually <b>self limiting, sometimes chronic, can have a carrier state.</b></p> <p><b>Some develop cirrhosis and liver cancer.</b></p> <p>Vaccine available.</p> <p><b>Hepatitis C (HCV)</b></p> <p>Single-stranded RNA virus</p> <p>Most common type transmitted <b>by blood transfusion</b></p> <p><b>Chronic disease common, can have a carrier state.</b></p> <p><b>Increases risk of hepatocellular carcinoma</b></p> <p><b>Treated with interferon injections</b></p>

	<p><b>Toxic or Nonviral Hepatitis</b></p> <p>Variety of hepatotoxins can cause inflammation and necrosis of the liver.      Drugs include:  <b>Acetaminophen</b>, halothane, phenothiazines, tetracycline      Chemicals include:      Carbon tetrachloride (not used currently), toluene, ethanol      Direct effect of toxins  <b>May result from sudden exposure to large amounts or from lower dose and long-term exposure</b></p>
	<p><b>Cirrhosis</b></p> <p><b>Progressive destruction and scarring of the liver</b></p> <p>Causes</p> <ol style="list-style-type: none"> <li>1. <b>Alcoholic liver disease</b></li> <li>2. <b>Biliary cirrhosis</b> <ul style="list-style-type: none"> <li>- Associated with immune disorders, obstructed ducts.</li> </ul> </li> <li>3. <b>Post-inflammatory or post-necrotic cirrhosis</b> <ul style="list-style-type: none"> <li>- Linked with <b>chronic hepatitis</b> or long-term exposure to toxic materials</li> </ul> </li> <li>4. <b>Metabolic</b> <ul style="list-style-type: none"> <li>- Usually caused by genetic metabolic storage disorders</li> </ul> </li> </ol>
	<p><b>Initial stage—fatty liver</b></p> <ul style="list-style-type: none"> <li>- Enlargement of the liver</li> <li>- Asymptomatic and reversible with reduced alcohol intake</li> </ul> <p><b>Second stage—alcoholic hepatitis</b></p> <ul style="list-style-type: none"> <li>- Inflammation and cell necrosis</li> <li>- <b>Fibrous tissue formation—irreversible change</b></li> </ul> <p><b>Third stage—end-stage cirrhosis</b></p> <ul style="list-style-type: none"> <li>- Fibrotic tissue replaces normal tissue.</li> <li>- Little normal function remains.</li> </ul>
	<p><b>Functional Losses with Cirrhosis:</b></p> <ol style="list-style-type: none"> <li>1. Decreased removal and conjugation of bilirubin</li> <li>2. <b>Decreased production of bile</b></li> <li>3. Impaired digestion and absorption of nutrients</li> <li>4. <b>Decreased production of blood-clotting factors</b></li> <li>5. <b>Impaired glucose and glycogen metabolism</b></li> <li>6. Impaired conversion of ammonia to urea</li> </ol> <p><b>High blood ammonia is an ominous sign of serious liver failure.</b></p>

<b>Sequelae with Cirrhosis</b>	
	 <p><b>Develop esophageal varices</b>  - Hemorrhage</p> <p><b>Develop ascites</b>, fluid in the peritoneal cavity  - Causes abdominal distention and pressure</p> <p><b>Bruising from decreased clotting factors.</b></p>
	 <p><b>Hepatocellular carcinoma</b>  Most common primary tumor of liver  <b>More common in cirrhotic livers</b>  Initial signs are mild and general.  <b>Diagnosis usually occurs with advanced stages</b>  Chemotherapy, possible lobectomy or radiofrequency ablation (RFA) procedure</p>
<b>Acute Pancreatitis</b>	
	<p>Inflammation of the pancreas</p> <p>- Results in <b>enzymatic autodigestion of the tissue</b></p> <p>May be acute or chronic</p> <p>- Acute leads to tissue destruction, <b>peritonitis, and possibly sepsis.</b></p> <p>- <b>Chronic leads to scarring of pancreas.</b></p> <p><b>Causes</b></p> <p>- Gallstones</p> <p>- Alcohol abuse</p>
	<p><b>Diagnostic Tests:</b> Elevated serum amylase and hyperlipidemia.</p>
<b>Pancreatic Cancer:</b>	
	<p><b>Risk factors: Smoking, heavy alcohol use, chronic pancreatitis.</b></p> <p>Adenocarcinoma—most common form., Arises from the epithelial cells in the ducts</p> <p>Weight loss and jaundice early manifestations</p> <p><b>Frequently asymptomatic until well advanced</b></p> <p><b>Metastases occur early.</b></p> <p><b>Mortality is close to 95%. (sometimes patient's do better if the cancer is in the head of the pancreas and it causes early symptoms, namely, obstruction of the bile ducts leading to obstructive jaundice)</b></p>

LOWER GI TRACT	
Celiac disease	
	<p><b>Malabsorption syndrome</b>  Primarily a childhood disorder  - May occur in adults in middle age  Appears to have genetic link</p> <p><b>Defect in intestinal enzyme</b>  - Prevents further digestion of gliadin (breakdown product of gluten)  - Toxic effect on intestinal villi—atrophy of villi  - Malabsorption and malnutrition result.</p>
Chronic Inflammatory Bowel Disease	
Chrohn's Disease and Ulcerative Colitis.	
<p>Chron's is in the <u>terminal ileum</u> and <u>spotty in colon</u>; has <u>transmural inflammation</u> with fissures, and <u>granulomas</u>.  Ulcerative colitis involves much of the colon (confluent); as mucosal inflammation and <u>no granulomas</u>.</p>	
Treatment:	
Anti-inflammatory medications, Antimicrobials, <b>Cytotoxic agents in serious cases.</b> Surgical resection In Chron's Disease segments that cause obstruction are resected. In Ulcerative colitis a colectomy is done for intractable inflammation or if dysplasia is detected (dysplasia is widespread and heralds cancer).	
Irritable Bowel Syndrome (IBS):	
Vague intestinal unrest with a wide variety of symptoms and causes.	
Acute Appendicitis:	
	<p><b>Obstruction of the appendiceal lumen</b>  - By a fecalith or foreign material  Appendiceal wall becomes inflamed.  - Purulent exudate fills lumen.  - Appendix is swollen.  Ischemia and necrosis of the wall</p>
Acute appendicitis presents as nausea, vomiting and generalized abdominal pain progressing to point tenderness in right lower quadrant with rebound tenderness.	

<b>Diverticular Disease:</b>	
	 <p><b>Diverticulum:</b>  - Outpouching (herniation) of the mucosa through the muscular layer of the colon</p> <p><b>Diverticulosis:</b>  - Asymptomatic diverticular disease</p> <p><b>Diverticulitis:</b>  - Inflammation of the diverticula</p>
<b>Colon Cancer:</b>	
Forms over time progressing through benign polyps before becoming cancer.	
Risk factors - Familial multiple polyposis - Long-term ulcerative colitis - Genetic factors - Environmental factors - Diet low in fiber	
<b>Because blood flow from the colon drains into the liver, the liver is a common site of metastasis.</b>	
<b>Peritonitis: Inflammation of the peritoneal membranes</b>	
<b>Chemical peritonitis may result from:</b> <b>Enzymes released with pancreatitis</b> Urine leaking from a ruptured bladder <b>Chyme spilled from a perforated ulcer</b> Bile escaping from the ruptured gallbladder Blood Any other foreign material in the cavity	
<b>Infections cause peritonitis:</b> <ul style="list-style-type: none"> <li>- <b>ruptured intestine or ruptured appendix</b> spill bacteria into peritoneal space.</li> <li>- sometimes bacterial leak into peritoneal space after <b>abdominal surgery</b>.</li> <li>- <b>Pelvic Inflammatory Disease is a portal to develop peritonitis.</b></li> </ul>	
	THE END