
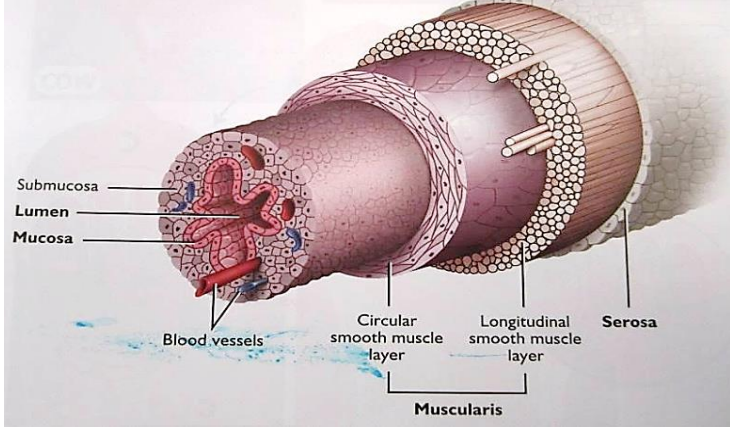


BIOS 2015 ... CHAPTER 17- Digestive System Disorders






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.
	Pathway of Digestion:
	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:
	- cholecystikinin 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.
	Anatomy of the gut:
	 <div style="display: flex; flex-direction: column; align-items: flex-end; margin-top: 10px;"> <div>Mucosal lining cells (mucosa)</div> <div>Submucosa</div> <div>Muscularis</div> <div>Inner circular</div> <div>outer longitudinal</div> </div>


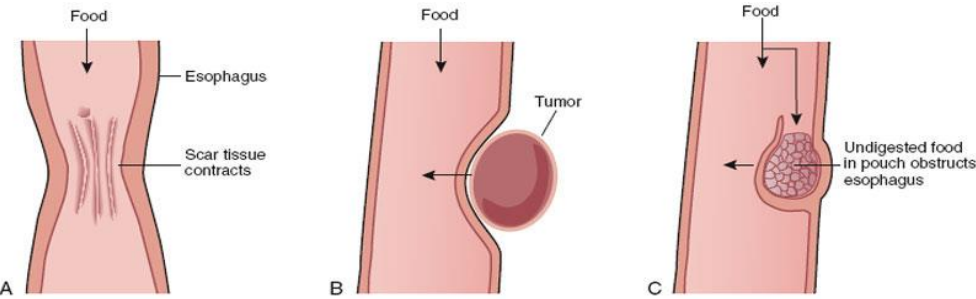
	The mucosa is different in the esophagus, stomach, small intestine and large intestine, but all have the same basic architecture of mucosa, submucosa, muscularis, and serosa.
	The muscularis also varies by region.
	Upper esophagus - striated muscle
	Lower esophagus- smooth muscle
	Stomach - 3 layers of smooth muscle (less well layered than intestine)
	Small and large intestine - 2 layers of smooth muscle (inner circular, outer longitudinal)
	 <div data-bbox="902 436 1448 793"> <p>Circular muscle cells wrap around the long axis of the intestine (like your fingers wrap around a drinking glass).</p> <p>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).</p> </div>
	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.

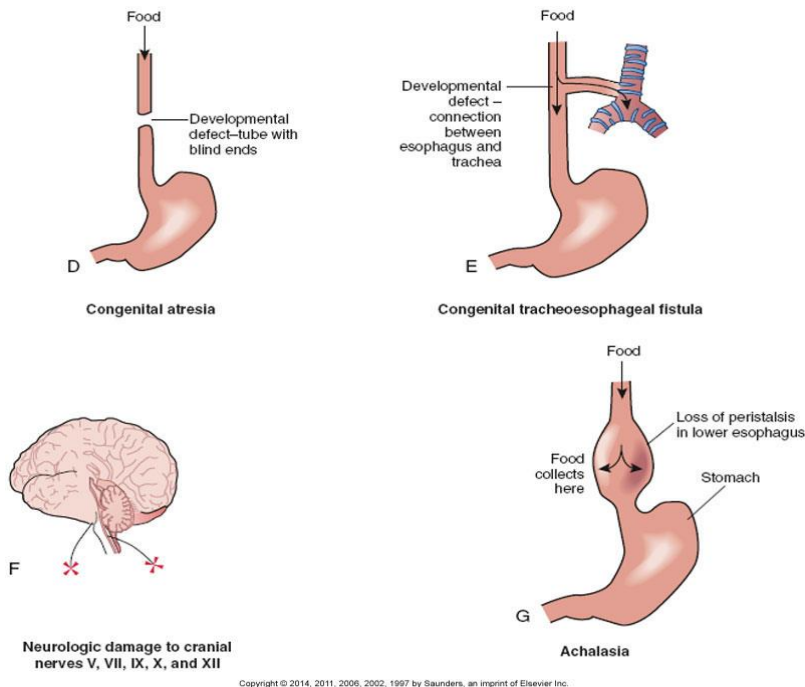
	Hormonal Control:		
Hormone	Source	Stimulus	Effects
Gastrin	Gastric cells	Food in the stomach Protein, caffeine, or high pH of chyme	Increases gastric secretions and motility and promotes gastric emptying
Cholecystikinin	Intestinal mucosal cells	Protein and fat in the duodenum	Inhibits gastric secretions 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
	Note: that cholecystikinin 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.		
	Vitamins:		
	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).		
	GI Symptoms:		
	Vague GI symptoms like anorexia, nausea and vomiting 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.		

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

	Referred Pain:
	<ul style="list-style-type: none"> - Pain is perceived at a site different from origin. - Results when visceral and somatic nerves converge at one spinal cord level <p>Source of visceral pain is perceived as the same as that of the somatic nerve (see diagram below).</p> <ul style="list-style-type: none"> - May assist or delay diagnosis, depending on problem
	<div data-bbox="147 369 863 894"> <p>Brain interprets source of pain as left arm</p> <p>Pain pathway to brain</p> <p>Sensory fibers</p> <p>Spinal cord convergence</p> <p>Pain in heart</p> <p>Skin of left arm</p> <p>B</p> <p><small>Copyright © 2014, 2011, 2006, 2002, 1997 by Saunders, an imprint of Elsevier Inc.</small></p> </div> <div data-bbox="915 401 1421 646"> <p>Classic Example of Referred Pain: 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> </div>
Classic Pain Associations to Remember:	
	<ol style="list-style-type: none"> 1. Pain in left arm and shoulder. Cardiac pain (heart attack). 2. Pain in right lower quadrant of abdomen: Appendix (acute appendicitis). 3. Pain in right upper quadrant of abdomen: Gall Bladder (cholecystitis). 4. Epigastric pain: Duodenum (acute ulcer), Pancreas (acute pancreatitis). 5. Pain in left lower quadrant of abdomen: Sigmoid colon (diverticulitis). 6. Back pain: Kidneys (kidney stones).
Drugs used in Digestive Disorders:	
	<p>Antacids and Proton pump inhibitors (PPI):</p> <ul style="list-style-type: none"> - To relieve gastric pain. - Reduce gastric secretion (PPI). <p>Antiemetics (Anti + Emesis, Emesis is vomiting):</p> <ul style="list-style-type: none"> - To relieve vomiting (often by dulling nausea) <p>Laxatives or enemas:</p> <ul style="list-style-type: none"> - Treatment of acute constipation <p>Antidiarrheals:</p> <ul style="list-style-type: none"> - Reduction of peristalsis - Relieve cramps

	Disorders of Oral Cavity
	Cleft lip and palate:
 <p>Baby with cleft lip</p> 	<p>Congenital abnormalities Cleft lip and cleft palate Feeding problems of the infant High risk of aspirating fluid into respiratory passages Speech development impaired Surgical repair done as soon as possible Therapy with speech-language pathologist and orthodontist</p>
	Aphthous ulcers:
	<p>Inner lip, small painful, self limited, possibly Strep. sanguis</p>
	Oral Candidiasis (Thrush):
	<p>Patchy white plaques in mouth and on tongue. Grows from local flora. Opportunistic infection. Seen with certain antibiotics, chemotherapy and</p>
	Oral Herpes (HSV type I):
	<p>Herpes simplex virus type 1 (HSV-1) Transmitted by kissing or close contact Virus remains dormant in sensory ganglion Activated by stress, trauma, other infection Formation of blister, 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>

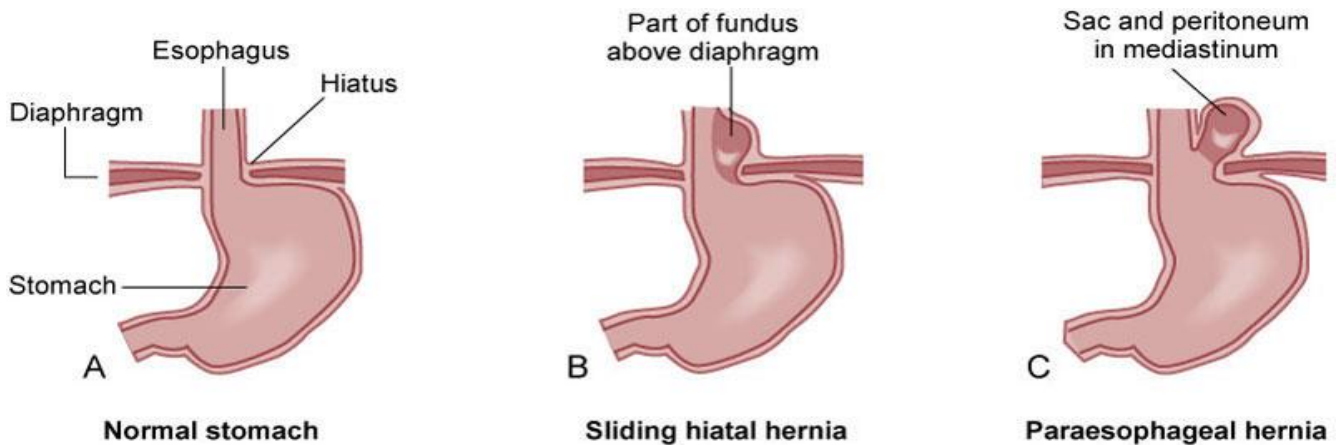
	Syphilis (caused by spirochete <i>Treponema pallidum</i>):
	<p>May cause oral lesions</p> <p>Highly contagious during first and second stages</p> <p>Primary stage</p> <p>Chancre, a painless ulcer on tongue, lip, palate</p> <p>Heals spontaneously (1 or 2 weeks)</p> <p>Secondary stage</p> <p>Red macules or papules on palate—highly infectious</p> <p>Heals spontaneously</p> <p>Both stages treated with long-acting penicillin</p>
	Dental Caries (Cavities in teeth):
	Bacteria break down sugar to acid, and acid dissolves mineral in tooth enamel.
	Gingivitis - inflammation of the gums
	Periodontitis - inflammation of deeper tooth structures around roots.
	Hyperkeratosis - Leukoplakia:
	<div>  <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. Epithelial dysplasia beneath plaque may develop into</p> </div>
	Sialadenitis - inflammation of salivary glands.
	Dysphagia - difficult swallowing, various causes.
	<div>  <p>A Fibrosis</p> <p>B Compression</p> <p>C Diverticulum</p> </div> <p>Copyright © 2014, 2011, 2006, 2002, 1997 by Saunders, an imprint of Elsevier Inc.</p>



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):



Gastroesophageal Reflux Disease:

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

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

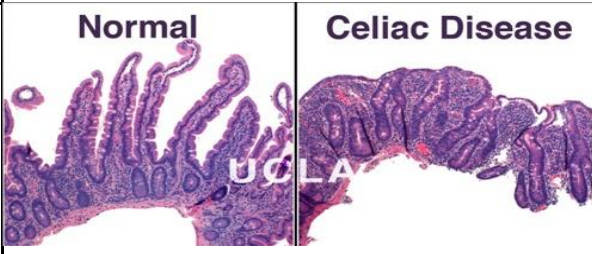
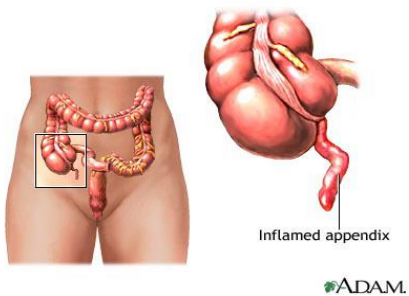
	<p>Damage to mucosal barrier predisposes to development of ulcers and is associated with:</p> <ol style="list-style-type: none"> Inadequate blood supply <ul style="list-style-type: none"> - Caused by vasoconstriction (e.g., by stress, smoking, shock, circulatory impairment in older adults, scar tissue, anemia) - Interferes with rapid regeneration of epithelium Excessive glucocorticoid secretion or medication Ulcerogenic substances break down mucous layer. <ul style="list-style-type: none"> - Aspirin, NSAIDs, alcohol Atrophy of gastric mucosa <ul style="list-style-type: none"> - Chronic gastritis
	<p>Increased acid pepsin secretions</p> <ul style="list-style-type: none"> - Increased gastrin secretion - Increased vagal stimulation
	<p>Complications of peptic ulcer</p> <p>Hemorrhage</p> <p>Perforation</p> <ul style="list-style-type: none"> - Ulcer erodes completely through the wall. - Chyme can enter the peritoneal cavity. - Results in chemical peritonitis <p>Obstruction</p> <ul style="list-style-type: none"> - May result later because of the formation of scar tissue
	<p>Gastric Carcinoma:</p>
	<div data-bbox="151 1039 527 1360" data-label="Image"> </div> <div data-bbox="609 1045 1401 1360" data-label="Text"> <p>Diet seems to be a key factor, particularly smoked foods, nitrites, and nitrates.</p> <p>Early carcinoma</p> <ul style="list-style-type: none"> - Confined to mucosa and submucosa <p>Later stages</p> <ul style="list-style-type: none"> - Infiltrates the muscularis <p>Asymptomatic in the early stages</p> <ul style="list-style-type: none"> - prognosis is poor on diagnosis - survival rate less than 20% </div>
	<p>Dumping:</p>
	<div data-bbox="170 1455 573 1896" data-label="Image"> </div> <div data-bbox="651 1476 1438 1864" data-label="Text"> <p>Control of gastric emptying is lost, and gastric contents are “dumped” into the duodenum without complete digestion.</p> <p>May follow gastric resection</p> <p>Effects:</p> <p>Hyperosmolar chyme draws fluid from vascular compartment into intestine causing intestinal distention and decreased blood pressure.</p> <p>High glucose in chyme leads to insulin release and subsequent hypoglycemia.</p> <p>Give frequent small meals—high in protein, low in simple carbohydrates</p> </div>



	<div data-bbox="131 153 321 184" data-label="Section-Header"> <p>Pyloric Stenosis:</p> </div> <div data-bbox="175 226 574 556" data-label="Image"> </div> <div data-bbox="688 216 1300 499" data-label="Text"> <p>Narrowing and obstruction of pyloric sphincter May be developmental anomaly Signs appear within several weeks after birth. - Projectile vomiting immediately after feeding - Firm mass can be palpated at pylorus. - Infant fails to gain weight, dehydration, persistent hunger Surgery required to relieve the obstruction.</p> </div>
	<div data-bbox="131 588 269 619" data-label="Section-Header"> <p>Gallstones:</p> </div> <div data-bbox="139 630 388 852" data-label="Image"> </div> <div data-bbox="453 640 980 816" data-label="Text"> <p>YELLOW - Cholesterol stones Women (2 x men) High cholesterol in bile (cholesterol stones) High cholesterol intake Obesity, Multiparity.</p> </div>
	<div data-bbox="139 879 438 1060" data-label="Image"> </div> <div data-bbox="518 890 945 957" data-label="Text"> <p>BLACK - Bilirubinate stones Associated with hemolytic anemia.</p> </div>
	<div data-bbox="131 1083 643 1520" data-label="Image"> </div> <div data-bbox="748 1110 1406 1428" data-label="Text"> <p>Cholelithiasis - Formation of gallstones - Solid material (calculi) that form in bile Choledocholithiasis - Obstruction of the biliary tract by gallstones Cholecystitis - Inflammation of gallbladder and cystic duct Cholangitis - Inflammation usually related to infection of bile ducts</p> </div>
	<div data-bbox="172 1549 534 1919" data-label="Chemical-Block"> </div> <div data-bbox="699 1560 1338 1627" data-label="Text"> <p>Unconjugated (indirect) bilirubin is processed in the liver to make Conjugated (direct) bilirubin.</p> </div> <div data-bbox="699 1667 1193 1698" data-label="Text"> <p>Total bilirubin = direct + indirect bilirubin.</p> </div> <div data-bbox="699 1738 1338 1806" data-label="Text"> <p>Indirect bilirubin is calculated from measurements of total and direct bilirubin.</p> </div>

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

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

	Sequelae with Cirrhosis
	<div data-bbox="154 210 495 535" data-label="Image"> <p>The diagram illustrates the anatomical changes in cirrhosis. The liver is shown as a shrunken, nodular organ. The portal vein is dilated, leading to the development of esophageal varices (enlarged veins in the esophagus) and splenomegaly (enlarged spleen). The stomach is also shown, and the overall abdominal cavity is depicted.</p> </div> <div data-bbox="649 210 1372 535" data-label="Text"> <p>Develop esophageal varices - Hemorrhage</p> <p>Develop ascites, fluid in the peritoneal cavity - Causes abdominal distention and pressure</p> <p>Bruising from decreased clotting factors.</p> </div>
	<div data-bbox="154 588 560 777" data-label="Image"> <p>The photograph shows a gross specimen of a liver with a large, irregular, reddish-brown mass, characteristic of hepatocellular carcinoma. The mass is surrounded by normal liver tissue, and a ruler is visible for scale.</p> </div> <div data-bbox="625 588 1339 850" data-label="Text"> <p>Hepatocellular carcinoma Most common primary tumor of liver More common in cirrhotic livers Initial signs are mild and general. Diagnosis usually occurs with advanced stages Chemotherapy, possible lobectomy or radiofrequency ablation (RFA) procedure</p> </div>
	Acute Pancreatitis
	<p>Inflammation of the pancreas</p> <ul style="list-style-type: none"> - Results in enzymatic autodigestion of the tissue <p>May be acute or chronic</p> <ul style="list-style-type: none"> - Acute leads to tissue destruction, peritonitis, and possibly sepsis. - Chronic leads to scarring of pancreas. <p>Causes</p> <ul style="list-style-type: none"> - Gallstones - Alcohol abuse
	Diagnostic Tests: Elevated serum amylase and hyperlipidemia.
	Pancreatic Cancer:
	<p>Risk factors: Smoking, heavy alcohol use, chronic pancreatitis.</p> <p>Adenocarcinoma—most common form., Arises from the epithelial cells in the ducts</p> <p>Weight loss and jaundice early manifestations</p> <p>Frequently asymptomatic until well advanced</p> <p>Metastases occur early.</p> <p>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)</p>

LOWER GI TRACT		
Celiac disease		
		<p>Malabsorption syndrome Primarily a childhood disorder - May occur in adults in middle age Appears to have genetic link Defect in intestinal enzyme - 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		
Crohn's Disease and Ulcerative Colitis.		
<p>Chron's is in the <u>terminal ileum</u> and <u>spotty</u> in colon; has <u>transmural inflammation</u> with fissures, and <u>granulomas</u>. Ulcerative colitis involves <u>much of the colon (confluent)</u> ; as <u>mucosal inflammation</u> and <u>no granulomas</u>.</p>		
Treatment:		
<p>Anti-inflammatory medications, Antimicrobials, Cytotoxic agents in serious cases. 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).</p>		
Irritable Bowel Syndrome (IBS):		
Vague intestinal unrest with a wide variety of symptoms and causes.		
Acute Appendicitis:		
		<p>Obstruction of the appendiceal lumen - By a fecalith or foreign material Appendiceal wall becomes inflamed. - Purulent exudate fills lumen. - Appendix is swollen. Ischemia and necrosis of the wall</p>
<p>Acute appendicitis presents as nausea, vomiting and generalized abdominal pain progressing to point tenderness in right lower quadrant with rebound tenderness.</p>		

	Diverticular Disease:
	<div data-bbox="500 212 1258 451"> <p>Diverticulum:</p> <ul style="list-style-type: none"> - Outpouching (herniation) of the mucosa through the muscular layer of the colon <p>Diverticulosis:</p> <ul style="list-style-type: none"> - Asymptomatic diverticular disease <p>Diverticulitis:</p> <ul style="list-style-type: none"> - Inflammation of the diverticula </div>
	Colon Cancer:
	Forms over time progressing through benign polyps before becoming cancer.
	<p>Risk factors</p> <ul style="list-style-type: none"> - Familial multiple polyposis - Long-term ulcerative colitis - Genetic factors - Environmental factors - Diet low in fiber
	Because blood flow from the colon drains into the liver, the liver is a common site of metastasis.
	Peritonitis: Inflammation of the peritoneal membranes
	<p>Chemical peritonitis may result from:</p> <p>Enzymes released with pancreatitis</p> <p>Urine leaking from a ruptured bladder</p> <p>Chyme spilled from a perforated ulcer</p> <p>Bile escaping from the ruptured gallbladder</p> <p>Blood</p> <p>Any other foreign material in the cavity</p>
	Infections cause peritonitis:
	- ruptured intestine or ruptured appendix spill bacteria into peritoneal space.
	- sometimes bacterial leak into peritoneal space after abdominal surgery .
	- Pelvic Inflammatory Disease is a portal to develop peritonitis.
	THE END