Chapter 17

Digestive System Disorders

Common Manifestations of Digestive System Disorders

Anorexia, Nausea, Vomiting, and Bulimia

- May be signs of digestive disorder or other condition elsewhere in the body
  - Systemic infection
  - Uremia
  - Emotional responses
  - Motion sickness
  - Pressure in the brain
  - Over indulgence of food, drugs
  - Pain

Anorexia, Nausea, Vomiting, and Bulimia

- Anorexia and vomiting // Can cause serious complications -- Dehydration, acidosis, malnutrition
- Anorexia - Often precedes nausea and vomiting
- Nausea // Unpleasant subjective feeling
  - Simulated by distention, irritation, inflammation of digestive tract
  - Also stimulated by smells, visual images, pain, and chemical toxins and/or drugs

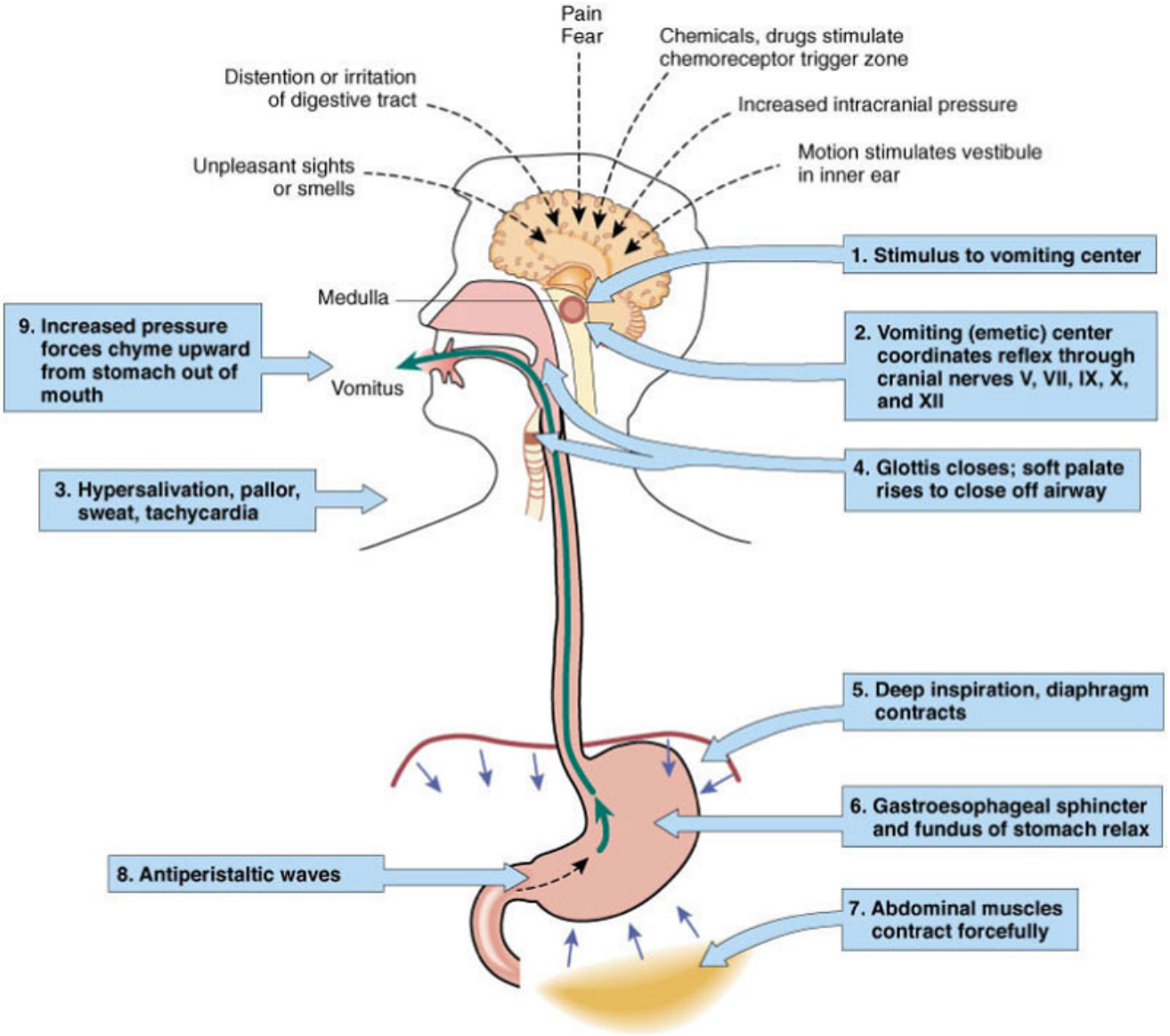
Anorexia, Nausea, Vomiting, and Bulimia

- Vomiting (emesis) // Vomiting center located in the medulla
  - Coordinates activities involved in vomiting
  - Protects airway during vomiting
  - Forceful expulsion of chyme from stomach // Sometimes includes bile from intestine
- Bulimia - eating disorder
  - Damage to structures of the GI tract caused by recurrent vomiting -- Oral mucosa / Teeth / Esophagus

Vomiting (Emetic) Center Activation

- Distention or irritation in digestive tract
- Stimuli from various parts of the brain // Response to unpleasant sights or smells, ischemia
- Pain or stress
- Vestibular apparatus of inner ear (motion)
- Increased intracranial pressure // Sudden projectile vomiting without previous nausea
- Stimulation of chemoreceptor trigger zone // By drugs, toxins, chemicals

Vomiting Reflex



Vomiting Reflex Activities

- Deep inspiration
- Closing the glottis, raising the soft palate
- Ceasing respiration // Minimizes risk of aspiration of vomitus into lungs
- Relaxing the gastroesophageal sphincter
- Contracting the abdominal muscles // Forces gastric contents upward
- Reversing peristaltic waves // Promotes expulsion of stomach contents

Characteristics of Vomitus

- Presence of blood - hematemesis
  - Coffee ground vomitus - brown granular material indicates action of HCl on hemoglobin
  - Hemorrhage - red blood may be in vomitus
- Yellow or green-stained vomitus // Bile from the duodenum
- Deeper brown color // May indicate content from lower intestine
- Recurrent vomiting of undigested food // Problem with gastric emptying or infection

Diarrhea

- Excessive frequency of stools // Usually of loose or watery consistency
- May be acute or chronic
- Frequently with nausea and vomiting when infection or inflammation develops
- May be accompanied by cramping pain
- Prolonged diarrhea may lead to dehydration, electrolyte imbalance, acidosis, malnutrition

Common Types of Diarrhea

- Large-volume diarrhea (secretory or osmotic)
  - 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

Common Types of Diarrhea

- Small-volume diarrhea
  - Often caused by inflammatory bowel disease
  - Stool may contain blood, mucus, pus
  - May be accompanied by abdominal cramps and tenesmus

Common Types of Diarrhea

- Steatorrhea = “fatty diarrhea”
  - Frequent bulky, greasy, loose stools
  - Foul odor
  - Characteristic of malabsorption syndromes // Celiac disease, cystic fibrosis
  - Fat usually the first dietary component affected // Presence interferes with digestion of other nutrients.
  - Abdomen often distended

Blood in Stool

- Blood may occur in normal stools with diarrhea, constipation, tumors, or an inflammatory condition.
  - Frank blood // Red blood—usually from lesions in rectum or anal canal
  - Occult blood // Small hidden amounts, detectable with stool test // May be caused by small bleeding ulcers
  - Melena // Dark-colored, tarry stool // May result from significant bleeding in upper digestive tract

Gas

- From swallowed air, such as drinking from a straw
- Bacterial action on food
- Foods or alterations in motility
- Excessive gas causes:
  - Eructation
  - Borborygmus
  - Abdominal distention and pain
  - Flatus

Constipation

- Less frequent bowel movements than normal
- Small hard stools
- Acute or chronic problem
- May be caused by decreased peristalsis // Increased time for reabsorption of fluid
- Periods of constipation may alter with periods of diarrhea.
- Chronic constipation may cause hemorrhoids, anal fissures, or diverticulitis.

Causes of Constipation

- Weakness of smooth muscle because of age or illness
- Inadequate dietary fiber
- Inadequate fluid intake
- Failure to respond to defecation reflex
- Immobility
- Neurological disorders
- Drugs (i.e., opiates)
- Some antacids, iron medications
- Obstructions caused by tumors or strictures

Fluid and Electrolyte Imbalances

- Dehydration and hypovolemia are common complications of *digestive tract disorders*.
- Electrolytes // Lost in vomiting and diarrhea
- Acid-base imbalances
  - Metabolic alkalosis // Results from loss of hydrochloric acid with vomiting
  - Metabolic acidosis // Severe vomiting causes a change to metabolic acidosis because of the loss of bicarbonate of duodenal secretions. // Diarrhea causes loss of bicarbonate.

Pain: Visceral Pain (the viscera)

- Burning sensation // Inflammation and ulceration in upper digestive tract
- Dull, aching pain // Typical result of stretching of liver capsule
- Cramping or diffuse pain // Inflammation, distention, stretching of intestines
- Colicky, often severe pain // Recurrent sooth muscle spasms or contraction --- Response to severe inflammation or obstruction

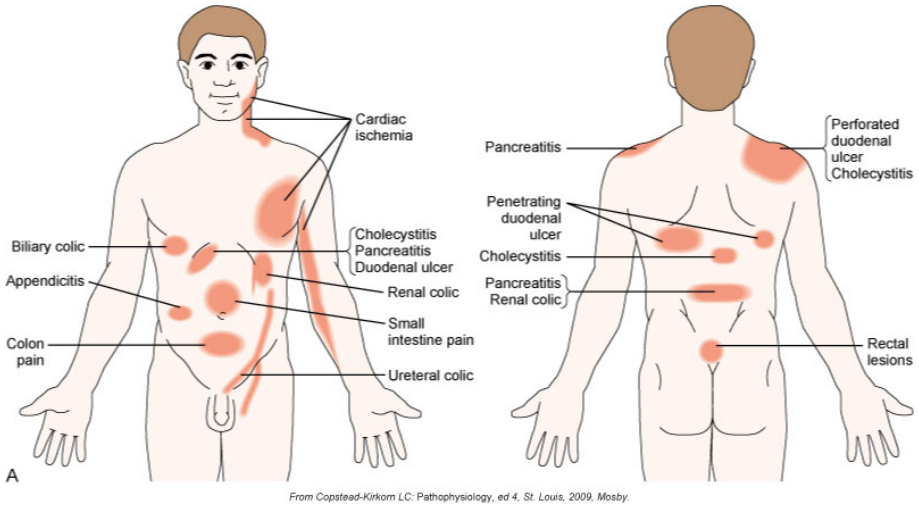
Pain: Somatic Pain (muscles & joints)

- Somatic pain receptors directly linked to spinal nerves // May cause reflex spasm of overlying abdominal muscles
- Steady, intense, often well-localized abdominal pain
- Involvement or inflammation of parietal peritoneum
- Rebound tenderness—identified over area of inflammation when pressure is *released*

Pain: Referred Pain

- Common phenomenon
- Pain is perceived at a site different from origin.
- Results when visceral and somatic nerves converge at one spinal cord level
- Source of visceral pain is perceived as the same as that of the somatic nerve.
- May assist or delay diagnosis, depending on problem

Pain: Referred Pain



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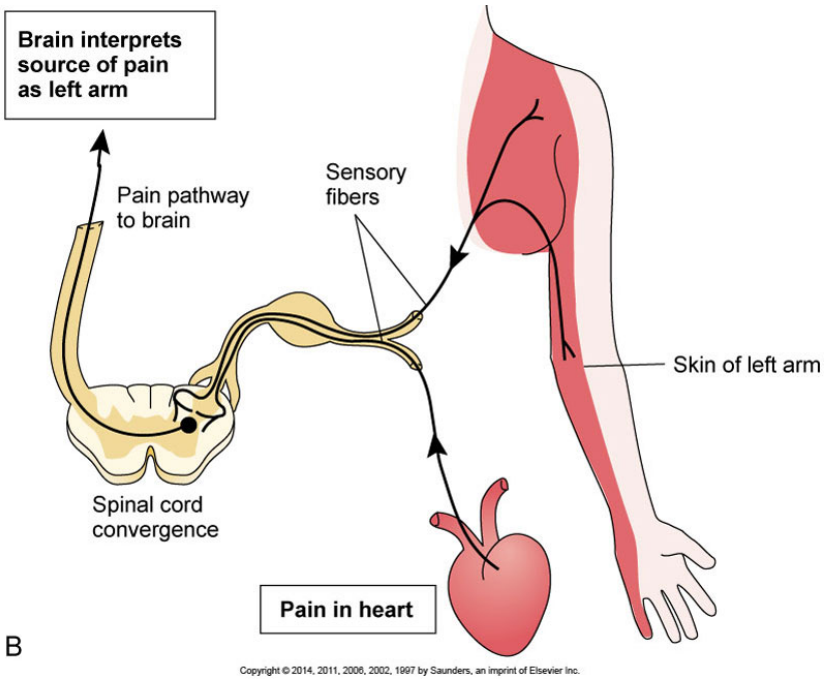


Malnutrition

- May be limited to a specific nutrient or general
- Causes of limited malnutrition - specific problem // Vitamin B_{12} deficiency & Iron deficiency
- Causes of generalized malnutrition
  - Chronic anorexia, vomiting, diarrhea
  - Other systemic causes // Chronic inflammatory bowel disorders / Cancer treatments / Wasting syndrome / Lack of available nutrients

Basic Diagnostic Tests

- Radiography // Contrast medium may be used.
- Ultrasound // May show unusual masses
- Computed tomography (CT)
- Magnetic resonance imaging (MRI)
- CT and MRI may use radioactive tracers. // Can be used for liver and pancreatic abnormalities

Basic Diagnostic Tests

- Fiberoptic endoscopy used in upper GI tract // Biopsy may be done during procedures.
- Sigmoidoscopy and colonoscopy // Biopsy and removal of polyps may be done
- Laboratory analysis of stool specimens // Check for infection, parasites and ova, bleeding, tumors, malabsorption
- Blood tests // Liver function, pancreatic function, cancer markers

Common Therapies and Prevention

- Dietary modifications
  - Example - gluten-free diet (celiac disease)
  - Reduced intake of alcohol and coffee
  - Increased fiber and fluid intake
- Stress reduction techniques // Stress impairs immune function and tissue healing.
- Drugs // Variety of medications are available.

Drugs Used in Digestive System Disorders

- Antacids // To relieve pyrosis
- Antiemetics // To relieve vomiting
- Laxatives or enemas // Treatment of acute constipation
- Antidiarrheals // Reduction of peristalsis & Relieve cramps

Drugs Used in Digestive System Disorders

- Sulfasalazine // Anti-inflammatory and antibacterial // Used for acute episodes of inflammatory bowel disease
- Clarithromycin or azithromycin // Effective against *Helicobacter pylori* infection // Usually combined with a proton pump inhibitor
- Sucralfate // Coating agent -- Enhance gastric mucosal barrier against irritants such as nonsteroidal anti-inflammatory drugs (NSAIDs)

Drugs Used in Digestive System Disorders

- Anticholinergic drugs // Reduce PNS activity by reducing secretions and motility
- Histamine 2 antagonists // Useful for gastric reflux
- Proton pump inhibitors // Reduce gastric secretion

TABLE 17-3 Examples of Drugs Used in Digestive System Disorders

Classification	Example	Action
Antiemetic	Dimenhydrinate (Dramamine) Prochlorperazine (Stemetil)	Reduces vomiting resulting from drugs, motion sickness, and radiation treatment
Antidiarrheal	Loperamide (Imodium) Codeine, paregoric	Reduces intestinal motility
Anti-inflammatory	Prednisone (Deltasone—a glucocorticoid) Sulfasalazine (Azulfidine)	Reduces inflammation Prednisone blocks immune response Sulfasalazine has antibacterial action
Acid-reduction	Ranitidine (Zantac): blocks H_2 receptors Lansoprazole (Prevacid): proton (H^+) pump inhibitor	Reduces secretion of hydrochloric acid in the stomach
Antimicrobial	Clarithromycin (Biaxin) Metronidazole (Flagyl) Tetracycline Cefoperazone Amoxicillin	Combination therapy for *Helicobacter pylori* infection Drugs as indicated by culture and sensitivity
Coating Agent	Sucralfate (Carafate)	Covers ulcer to allow healing
Antacid	Aluminum-magnesium combinations (Maalox)	Reduces hyperacidity
Laxative	Psyllium (Metamucil) (bulk) or docusate sodium (Colace) (stool softener)	With water, increases fecal bulk and intestinal motility
Anticholinergics	pirenzepine, propantheline bromide	reduces PNS activities—reduced secretions and mobility
Histamine-2 blockers	Tagamet, Zantac	Inhibits acid production in stomach
Proton Pump Inhibitors	Prevacid, Prilosec	reduce gastric secretions

Upper Gastrointestinal Tract Disorders

Disorders of the Oral Cavity

- Congenital abnormalities // Cleft lip and cleft palate
  - Arise in sixth to seventh week of gestation
  - Most likely of multifactorial origin
  - 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

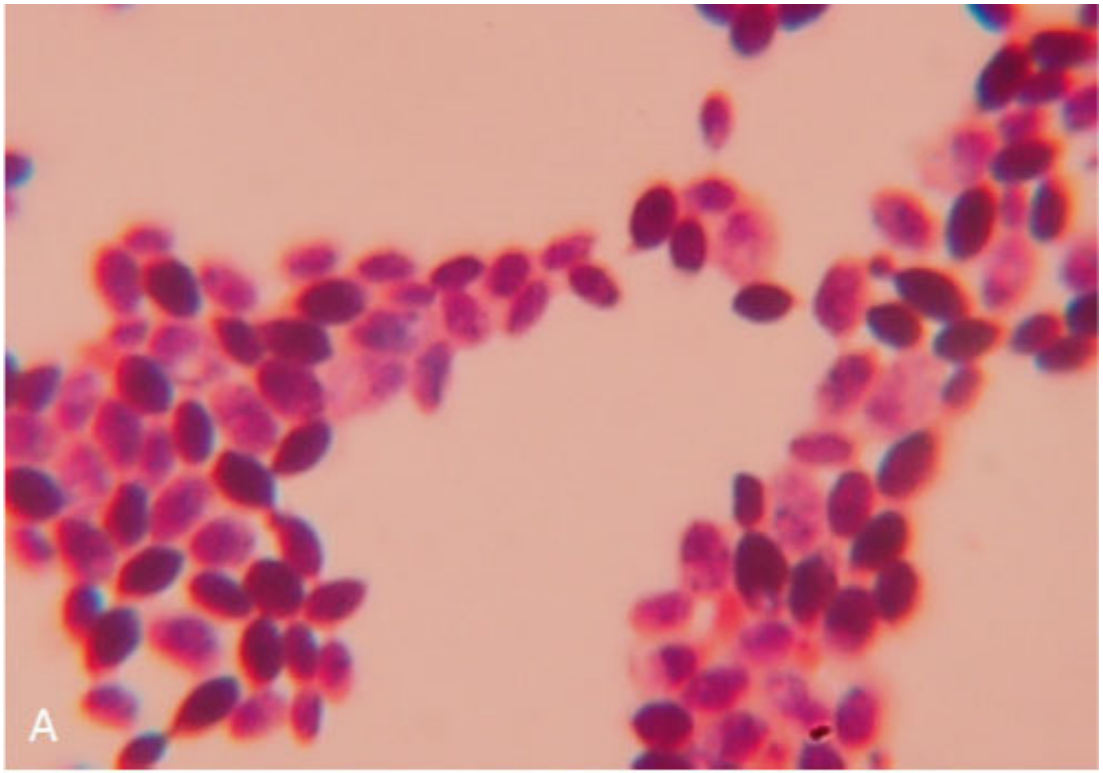
Disorders of the Oral Cavity

- Inflammatory lesions—aphthous ulcers
  - *Streptococcus sanguis* may be involved.
    // Part of the oral resident flora
  - Small painful lesions on:
    - Movable mucosa
    - Buccal mucosa
    - Floor of the mouth
    - Soft palate
    - Lateral borders of the tongue
  - Usually heal spontaneously

Disorders of the Oral Cavity: Infections

- Candidiasis
  - *Candida albicans* - causative agent
    - Often part of the resident flora
    - Opportunistic organism
  - Oral candidiasis (thrush)
    - People receiving broad-spectrum antibiotics
    - During and after cancer therapy
    - Immunocompromised individuals or those with diabetes
  - May appear as red, swollen areas
  - May be irregular patches of a white curdlike material

Oral Candidiasis



A. *From VanMeter K, Hubert R:* Microbiology for the Healthcare Professional, *St. Louis, 2010, Elsevier.* B. From Zitelli BJ, Davis HW: *Atlas of pediatric physical Diagnosis,* ed 4. St. Louis, 2002, Mosby.

Disorders of the Oral Cavity: Infections

- Herpes simplex type 1 infection
  - 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

Disorders of the Oral Cavity: Infections

- Syphilis // Caused by *Treponema pallidum*
  - May cause oral lesions
  - Highly contagious during first and second stages
  - Primary stage // Chancre, a painless ulcer on tongue, lip, palate // Heals spontaneously (1 or 2 weeks)
  - Secondary stage // Red macules or papules on palate - highly infectious // Heals spontaneously
  - Both stages treated with long-acting penicillin

Disorders of the Oral Cavity:
Dental Problems

- Caries // *Streptococcus mutans* - initiating microbe
  - *Lactobacillus* follows in large numbers.
  - Bacteria break down sugars and produce large quantities of lactic acid.
  - Lactic acid dissolves mineral in tooth enamel
  - Tooth erosion and caries formation
  - Caries is promoted by frequent intake of sugars and acids.
  - Fluoride - anticaries treatment

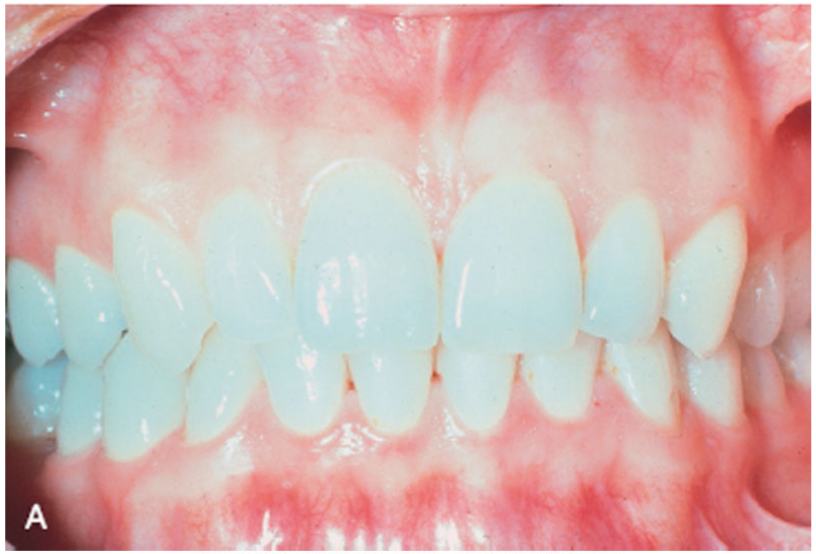
Disorders of the Oral Cavity:
Dental Problems

- Gingivitis
  - Changes in the gingivae may be a local or systemic problem.
  - Inflammation of the gingiva // Tissue becomes red, soft, swollen, bleeds easily // May be a result of accumulated plaque
  - Inadequate oral hygiene
  - Toothbrush trauma
    - Results from improper or excessive brushing
    - Creates extensive grooving on tooth surface
    - Increase plaque retention and damage to gingivae

Disorders of the Oral Cavity: Dental Problems

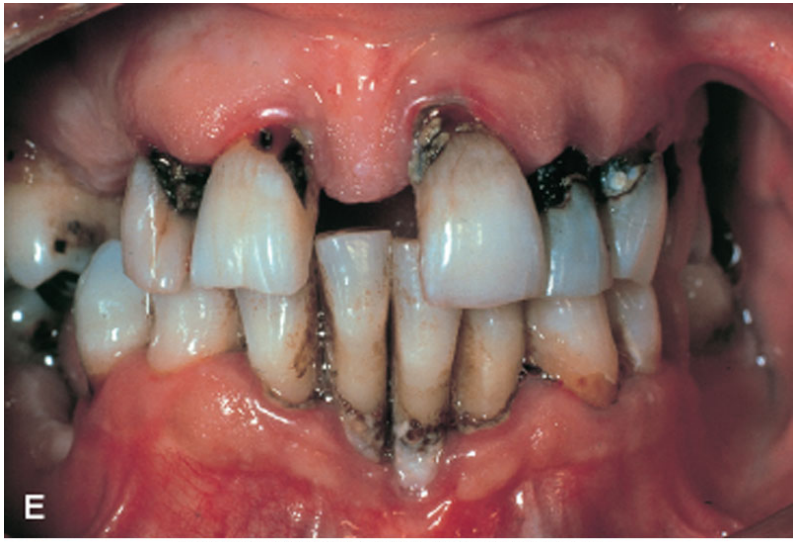
- Periodontal disease
  - Infection and damage to the periodontal ligament and bone
  - Predisposing condition is gingivitis
  - Caused by microorganisms as a result of poor dental hygiene
  - Subsequent loss of teeth possible
  - Several categories, depending on degree of disease
  - May be aggravated by systemic disease and medications that reduce salivary secretions

Healthy Periodontium



A-E, Courtesy Evie Jesin, George Brown College of Applied Arts and Technology, Toronto, Ontario, Canada.

Periodontal Disease



A-E, Courtesy Evie Jesin, George Brown College of Applied Arts and Technology, Toronto, Ontario, Canada.

Disorders of the Oral Cavity: Dental Problems

- Periodontitis occurs when organisms enter the gingival blood vessels and travel to the connective tissues and bone of the dental arch.
- Reabsorption of bone and loss of ligament fibers result in weakened attachment of teeth.
- May result in total loss of tooth from socket
- Treated by antimicrobials, local surgery of gingiva, and improved dental hygiene

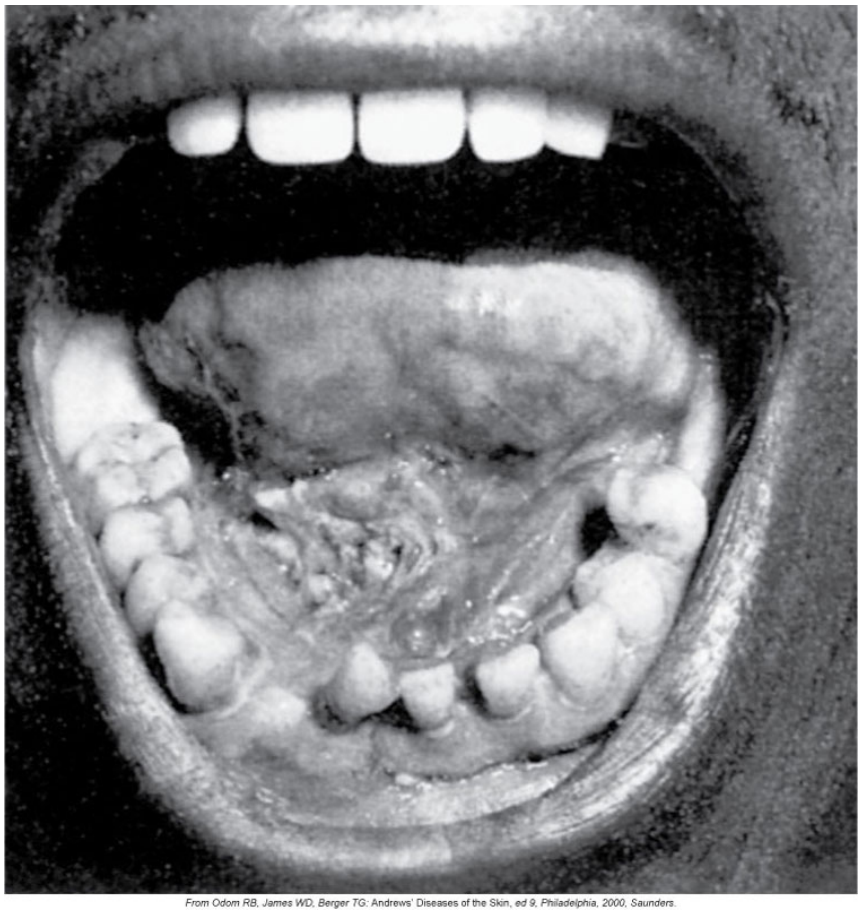
Disorders of the Oral Cavity: Dental Problems

- Hyperkeratosis
  - Leukoplakia (example)
  - 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 squamous cell carcinoma.

Disorders of the Oral Cavity: Cancer of the Oral Cavity

- Squamous cell carcinoma—common type
- Often develops in persons older than 40 years
  - Smokers, preexisting leukoplakia, alcohol abuse
  - Floor of the mouth, lateral borders of the tongue
  - Multiple lesions possible
- Kaposi sarcoma in patients with AIDS
- Lip cancer has a better prognosis. // Common in smokers, particularly pipe smokers

Squamous Cell Carcinoma on Floor of the Mouth



From Odom RB, James WD, Berger TG: Andrews' Diseases of the Skin, ed 9, Philadelphia, 2000, Saunders.

Disorders of the Oral Cavity: Salivary Gland Disorders

- Sialadenitis
  - Inflammation of the salivary glands
  - May be infectious or noninfectious
  - Most commonly affected - parotid gland
- Mumps - infectious parotitis
  - Viral infection
  - Vaccine available

Disorders of the Oral Cavity: Salivary Gland Disorders

- Noninfectious parotitis
  - Often seen in older adults who lack adequate fluid intake and mouth care
- Most malignant tumor of salivary glands is mucoepidermoid carcinoma

Dysphagia

- Difficulty swallowing
- Causes // Neurological deficit // Muscular disorder // Mechanical obstruction
- Results and presentation
  - Pain with swallowing
  - Inability to swallow larger pieces of solid material
  - Difficulty swallowing liquids

Dysphagia // Neurological deficit

- Infection
- Stroke
- Brain damage
- Achalasia // Failure of the lower esophageal sphincter to relax because of lack of innervation

Dysphagia // Muscular disorder

- Impairment from muscular dystrophy

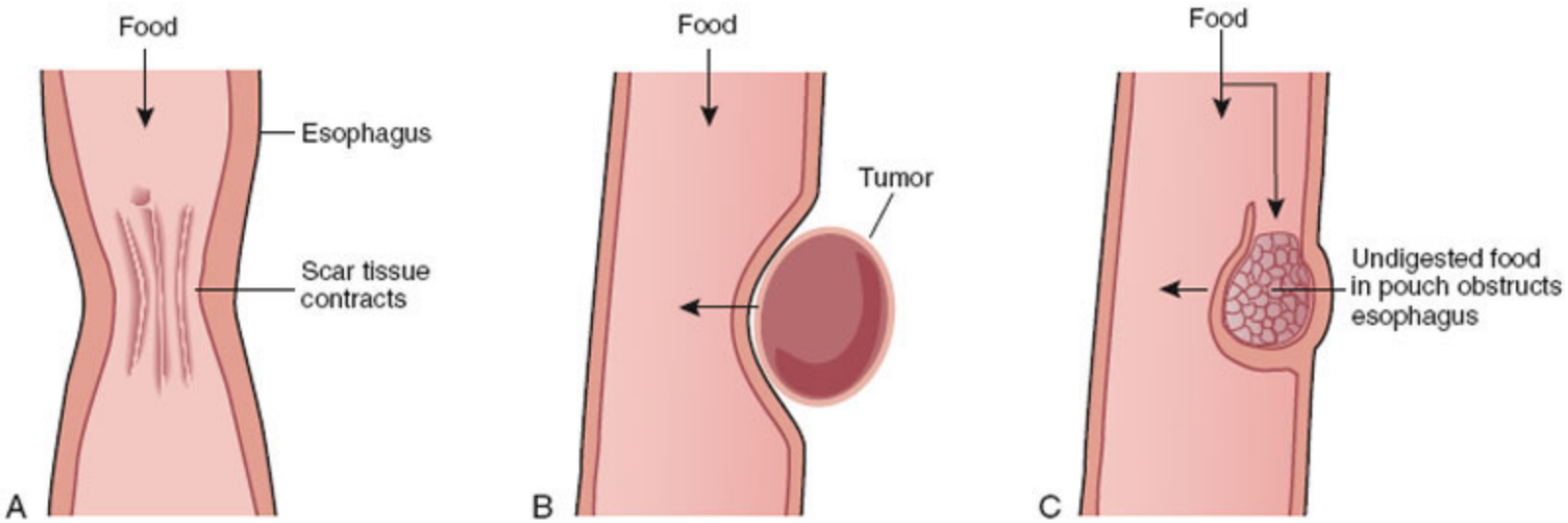
Dysphagia // Mechanical obstruction

- Congenital atresia // Developmental anomaly
  - Upper and lower esophageal segments are separated.
- Stenosis // Narrowing of the esophagus
  - May be developmental or acquired
  - May be secondary to fibrosis, chronic inflammation, ulceration, radiation therapy
  - Stenosis or stricture may also result from scar tissue
  - May require treatment with repeated mechanical dilation

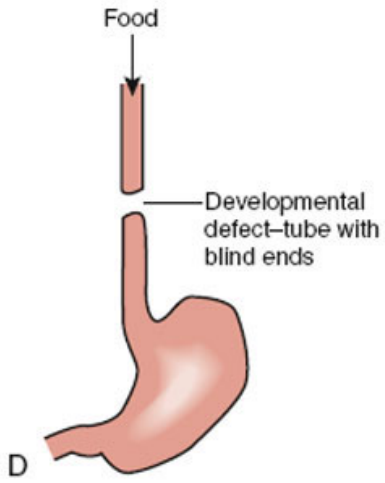
Dysphagia // Mechanical obstruction

- Esophageal diverticula
  - Outpouchings of the esophageal wall
  - Congenital or acquired following inflammation
  - Causes irritation, inflammation, scar tissue
  - Signs include dysphagia, foul breath, chronic cough, hoarseness
- Tumors // May be internal or external

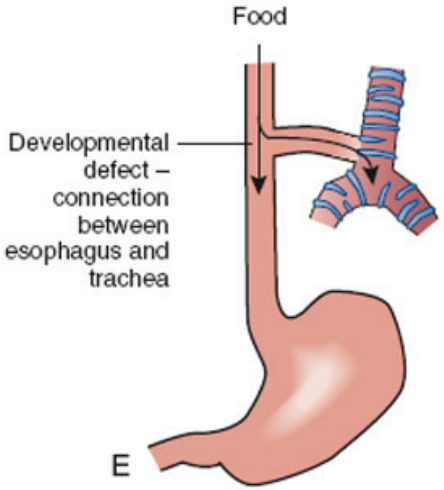
Causes of Dysphagia



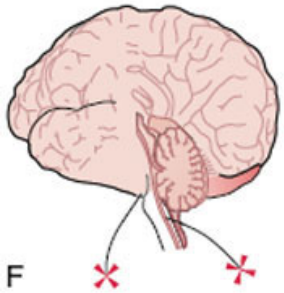
Causes of Dysphagia



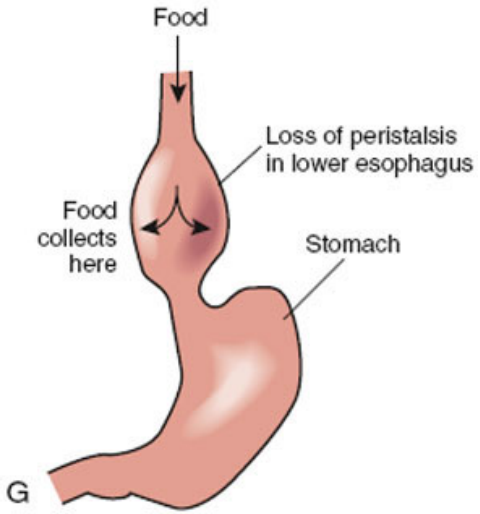
Congenital atresia



Congenital tracheoesophageal fistula



Neurologic damage to cranial nerves V, VII, IX, X, and XII



Achalasia

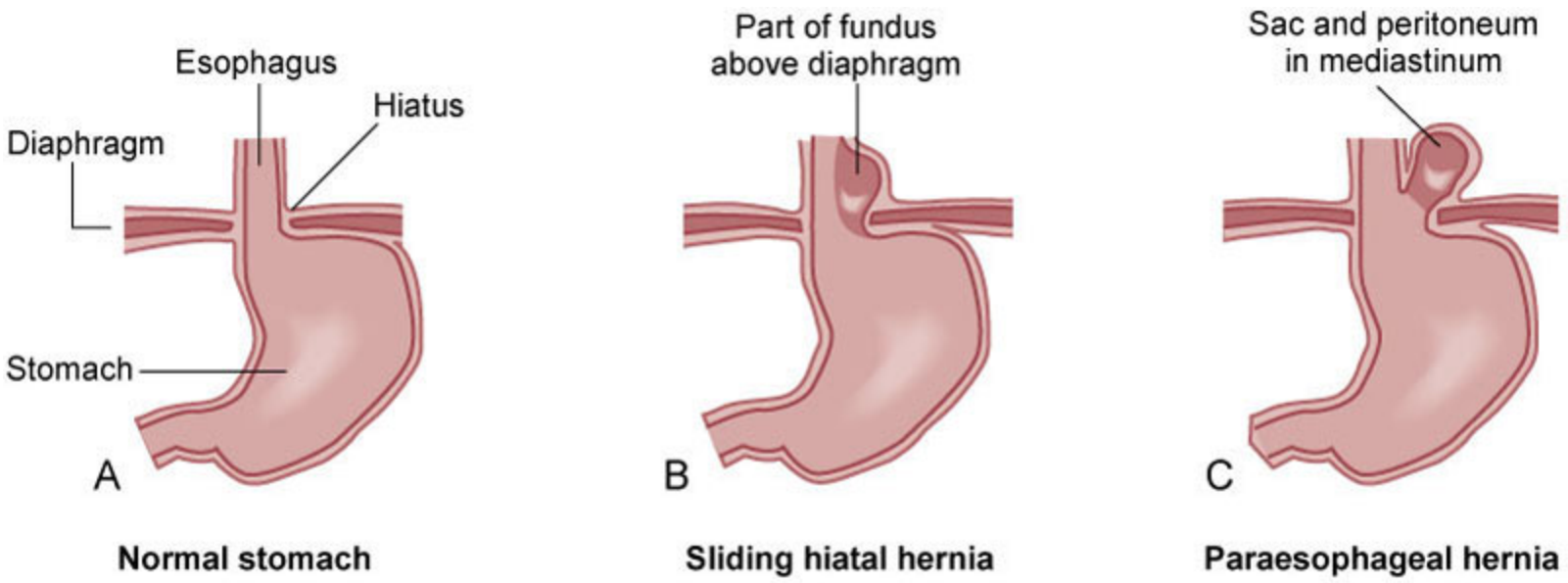
Esophageal Cancer

- Primarily squamous cell carcinoma
- Usually in distal esophagus
- Significant dysphagia in later stages
- Poor prognosis because of late manifestations
- 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.
- Sliding hernia // More common type
  - Portions of the stomach and gastroesophageal junction slide up above the diaphragm.
- Rolling or paraesophageal hernia
  - Part of the fundus of the stomach moves up through an enlarged or weak hiatus in the diaphragm and may become trapped.

Types of Hiatal Hernia



Hiatal Hernia

- Food may lodge in pouch of the hernia
  - Causes inflammation of the mucosa
  - Reflux of food up the esophagus
  - May cause chronic esophagitis
- Signs
  - Heartburn or pyrosis
  - Frequent belching
  - Increased discomfort when laying down
  - Substernal pain that may radiate to shoulder and jaw

Gastroesophageal Reflux Disease

- Periodic reflux of gastric contents into distal esophagus causes erosion and inflammation.
- Often seen in conjunction with hiatal hernia
- Severity depends on competence of the lower esophageal sphincter.
- Delayed gastric emptying may be a factor.
- Avoidance of: // Caffeine, fatty and spicy foods, alcohol, smoking, certain drugs
- Use of medication may reduce reflux and inflammation

Gastritis: Acute Gastritis

- Gastric mucosa is inflamed.
- May be ulcerated and bleeding
- May result from
  - 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

Gastritis: Acute Gastritis

- Basic signs of gastrointestinal irritation
  - Anorexia, nausea, vomiting may develop
  - Hematemesis caused by bleeding
  - Epigastric pain, cramps or general discomfort
  - With infection, diarrhea may develop.
- Acute gastritis is usually self-limiting.
  - Complete regeneration of gastric mucosa
  - Supportive treatment with prolonged vomiting
  - May require treatment with antimicrobial drugs

Gastritis: 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.

Gastritis: Gastroenteritis

- Inflammation of stomach and intestine
- Usually caused by infection
- May also be caused by allergic reactions to food or drugs
- Microbes can be transmitted by fecal contaminated food, soil, and/or water
  - Most infections are self-limiting.
  - Serious illness may result in compromised host or virulent organisms.
  - May cause epidemic outbreaks in refugee or disaster settings
  - Safe sanitation essential for prevention

TABLE 17-4 Common Infections Transmitted by Food and Water

Pathogen	Source	Incubation	Pathophysiology	Manifestations
Staphylococcus aureus	Food handlers Inadequate cooking or refrigeration of custards, salad dressing, cold meats	1-7 hr (2-4, average)	Enterotoxin (exotoxin), heat-stable	Sudden severe nausea, vomiting, and cramps; sometimes diarrhea Subnormal body temperature and low blood pressure
Escherichia coli (traveler's diarrhea)	Fecal contamination of food and water	10-12 hr	Various strains may release enterotoxins (increase secretions) or invade the mucosa	Profuse watery diarrhea, sometimes with blood or mucus Vomiting and abdominal cramps often present
Salmonella species	Fecal contamination of food or undercooked or raw eggs, poultry, shellfish Contaminated work surfaces	6-72 hr	Organisms multiply in intestine, causing inflammation and ulceration	Sudden diarrhea, abdominal pain, and fever Sometimes vomiting
Rotavirus	Oral-fecal (infants)	24-72 hr	Inflammation and loss of villi	Vomiting, severe watery diarrhea, fever
Norwalk virus	Oral-fecal (adults/older children) shellfish, fomites	24-48 hr	Damage villi	Vomiting, diarrhea, and cramps, headache, fever
Entamoeba histolytica (amebic dysentery)	Fecal contamination of water and vegetables	2-4 wk	Protozoan parasite with cyst stage and active trophozoite stage; may invade mucosa, causing abscesses	Diarrhea with blood and mucus, may alternate with constipation Fever and chills
Listeria sp.	Found in soil and water	30-70 days	Granulomas	Flu-like with diarrhea.
Clostridium botulinum	Spores in poorly canned food or prepared meat	12-36 hr	Neurotoxin (exotoxin) causes nerve paralysis	Visual problems, dysphagia, then flaccid paralysis and respiratory failure Possibly early vomiting or diarrhea
Campylobacter sp.	Handling raw poultry, undercooked poultry, raw milk, contaminated water	2-5 days	infection causes tissue damage to the ileum, jejunum and colon	diarrhea, nausea

Escherichia coli Infection

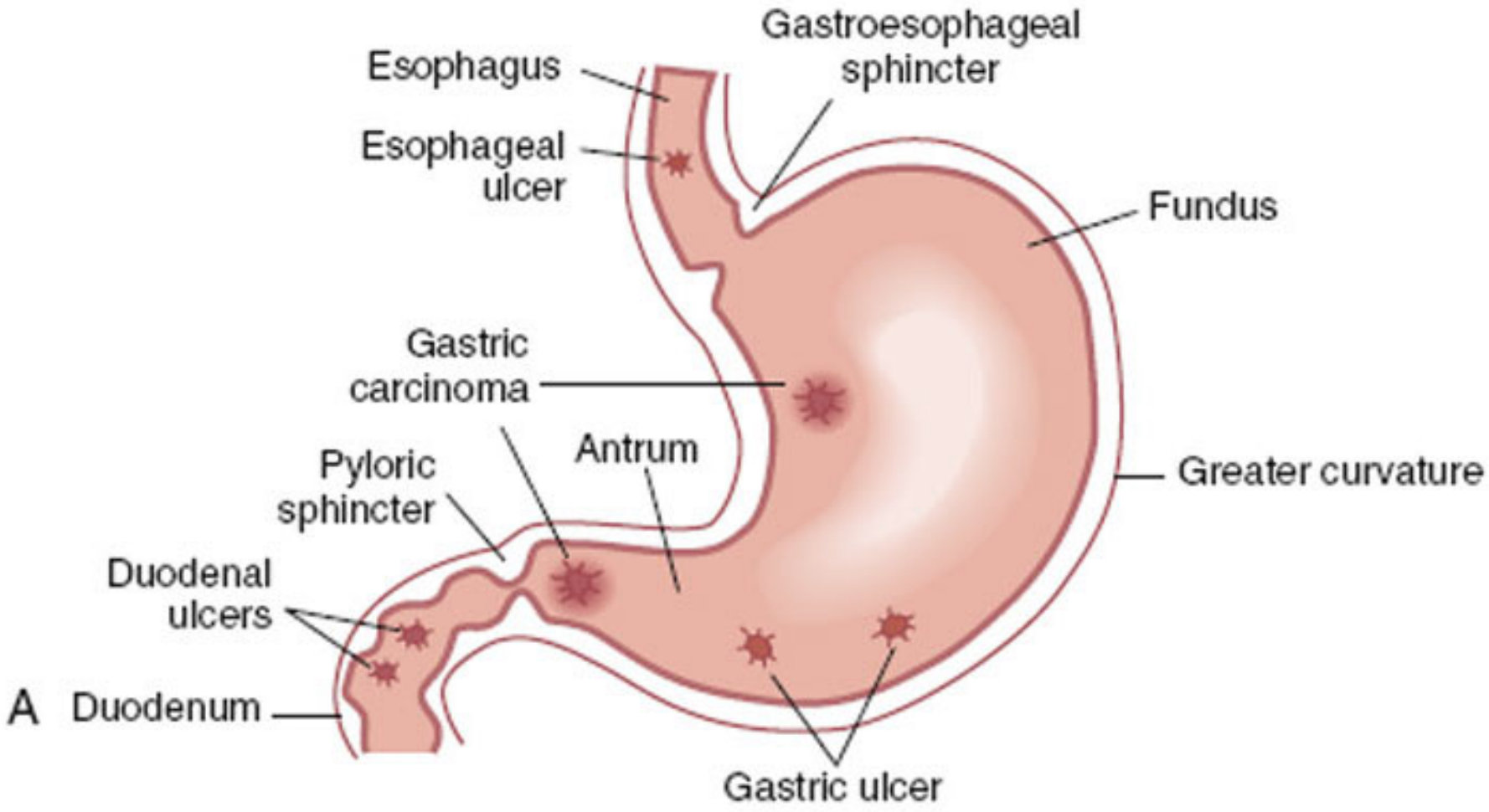
Although *E. coli* is usually harmless as a resident in the human intestine, infective strains can cause significant problems.

- 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

Peptic Ulcer: Common Locations



A. *Courtesy of RW Shaw, North York General Hospital, Toronto, Ontario, Canada.* From Cooke RA, Stewart B. *Colour Atlas of Anatomical Pathology,* ed 3, Sydney, 2004, Churchill Livingstone

Peptic Ulcer: Gastric and Duodenal Ulcers

- Damage to mucosal barrier predisposes to development of ulcers and is associated with:
  - Inadequate blood supply // 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. // Aspirin, NSAIDs, alcohol
  - Atrophy of gastric mucosa // Chronic gastritis

Peptic Ulcer: Gastric and Duodenal Ulcers

- Increased acid pepsin secretions
  - Increased gastrin secretion
  - Increased vagal stimulation
  - Increased sensitivity to vagal stimuli
  - Increased number of acid pepsin secretory cells in the stomach (genetic anomaly)
  - Increased stimulation of acid pepsin secretion // Alcohol, caffeine, certain foods
  - Interference with normal feedback mechanisms
  - Rapid gastric emptying

Peptic Ulcer: Gastric and Duodenal Ulcers

- Complications of peptic ulcer
  - Hemorrhage
    - Caused by erosion of blood vessels
    - Common complication
    - May be the first sign of a peptic ulcer
  - Perforation
    - Ulcer erodes completely through the wall.
    - Chyme can enter the peritoneal cavity.
    - Results in chemical peritonitis
  - Obstruction // May result later because of the formation of scar tissue

Peptic Ulcer: Gastric and Duodenal Ulcers

- Signs and symptoms // Epigastric burning or localized pain, usually following stomach emptying

- Diagnostic tests

  - Fiberoptic endoscopy
  - Barium x-ray
  - Endoscopic biopsy

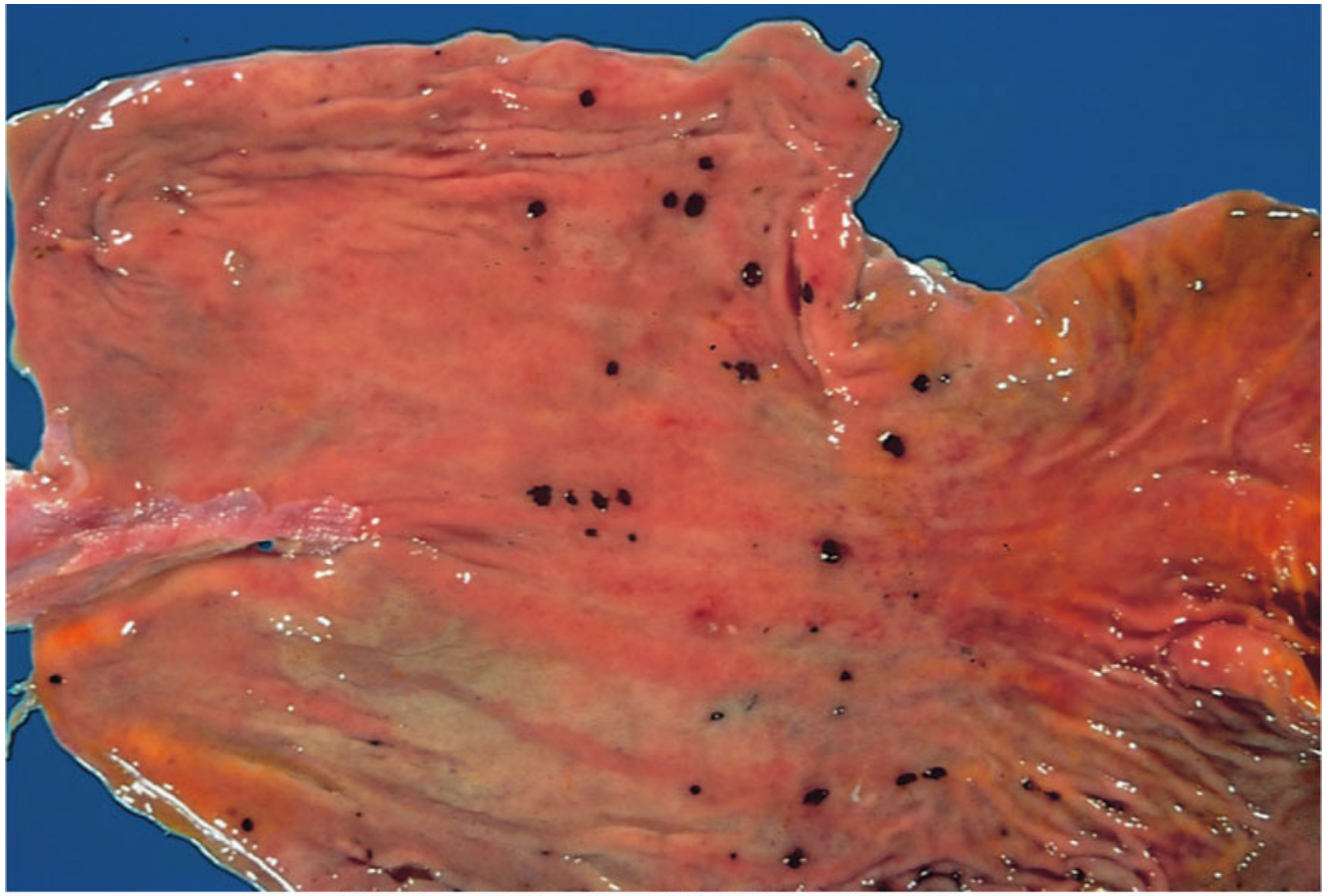
- Treatment

  - Combination of antimicrobial and proton pump inhibitor to eliminate *H. pylori*
  - Reduction of exacerbating factors

Stress Ulcers

- Associated with severe trauma or systemic problems
  - Burns, head injury
  - Hemorrhage or sepsis
- Rapid onset
  - Multiple ulcers (usually gastric) may form within hours of precipitating event
  - First indicator—hemorrhage and severe pain

Multiple Stress Ulcers of the Stomach



From Kumar V, Abbas AK, Fausto M: Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.*

Gastric Cancer

- Arises primarily in mucous glands
- Mostly in the antrum or pyloric area
- Early carcinoma // Confined to mucosa and submucosa
- Later stages // Involves muscularis // Eventually invades serosa and spreads to lymph nodes
- Asymptomatic in the early stages // Often, prognosis is poor on diagnosis

Gastric Cancer

- Diet seems to be a key factor, particularly smoked foods, nitrites, and nitrates.
- Genetic influences also play a role.
- Symptoms vague until cancer is advanced.
  - Reason for late diagnosis
  - Surgery together with chemotherapy and radiation may relieve symptoms.
  - Survival rate less than 20%

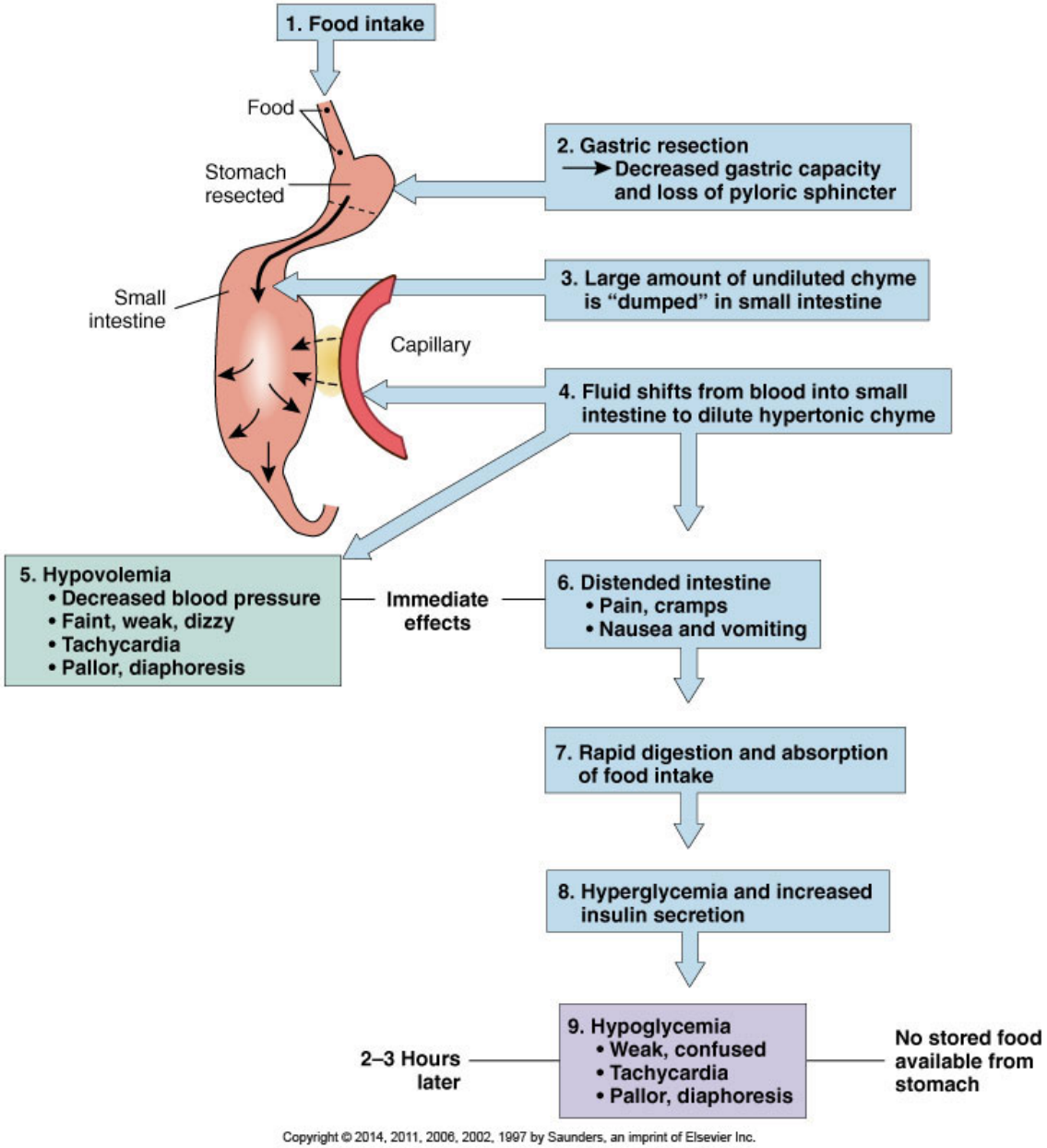
Dumping Syndrome

- Control of gastric emptying is lost, and gastric contents are “dumped” into the duodenum without complete digestion.
- May follow gastric resection
- Hyperosmolar chyme draws fluid from vascular compartment into intestine
  - Intestinal distention
  - Increased intestinal motility
  - Decreased blood pressure → anxiety and syncope

Dumping Syndrome

- Occurs during or shortly after meals // Abdominal cramps, nausea, diarrhea
- Hypoglycemia 2 to 3 hours after meal // High blood glucose levels in chyme stimulate increased insulin secretion → drop in blood glucose levels
- May be resolved by dietary changes // Frequent small meals - high in protein, low in simple carbohydrates
- Often resolves over time

Dumping Syndrome



Pyloric Stenosis

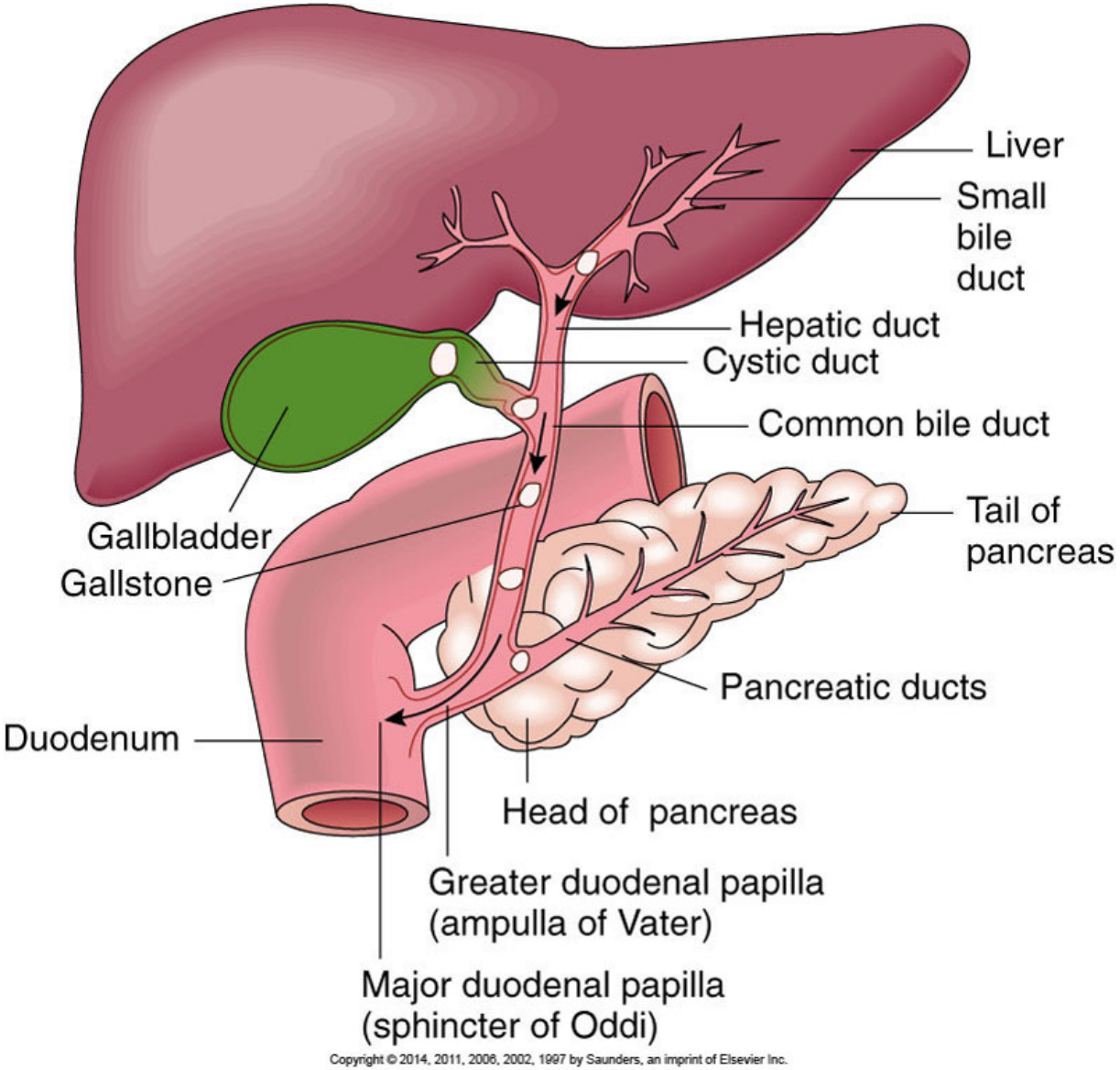
- Narrowing and obstruction of pyloric sphincter
- May be developmental anomaly
- Signs appear within several weeks after birth.
  - Projectile vomiting immediately after feeding
  - Firm mass can be palpated at pylorus.
  - Infant fails to gain weight, dehydration, persistent hunger
- Surgery required to remove obstruction.
- May be acquired later in life // Persistent feeling of fullness // Increased incidence of vomiting

Disorders of the Liver and Pancreas

Gallbladder Disorders

- Cholelithiasis // Formation of gallstones - Solid material (calculi) that form in bile
- Cholecystitis // Inflammation of gallbladder and cystic duct
- Cholangitis // Inflammation usually related to infection of bile ducts
- Choledocholithiasis // Obstruction of the biliary tract by gallstones

Biliary Ducts and Pancreas with Possible Locations of Gallstones



Gallbladder Disorders

- Gallstones vary in size and shape.
- Form in bile ducts, gallbladder, or cystic duct
- May consist of // Cholesterol or bile pigment // Mixed content with calcium salts
- Small stones // May be silent and excreted in bile
- Larger stones // Obstruct flow of bile in cystic or common bile ducts; cause severe pain, which is often referred to subscapular area

Gallbladder Disorders

- Risk factors for gallstones
  - Women twice as likely to develop stones
  - High cholesterol in bile
  - High cholesterol intake
  - Obesity
  - Multiparity
  - Use of oral contraceptives or estrogen supplements
  - Hemolytic anemia
  - Alcoholic cirrhosis
  - Biliary tract infection

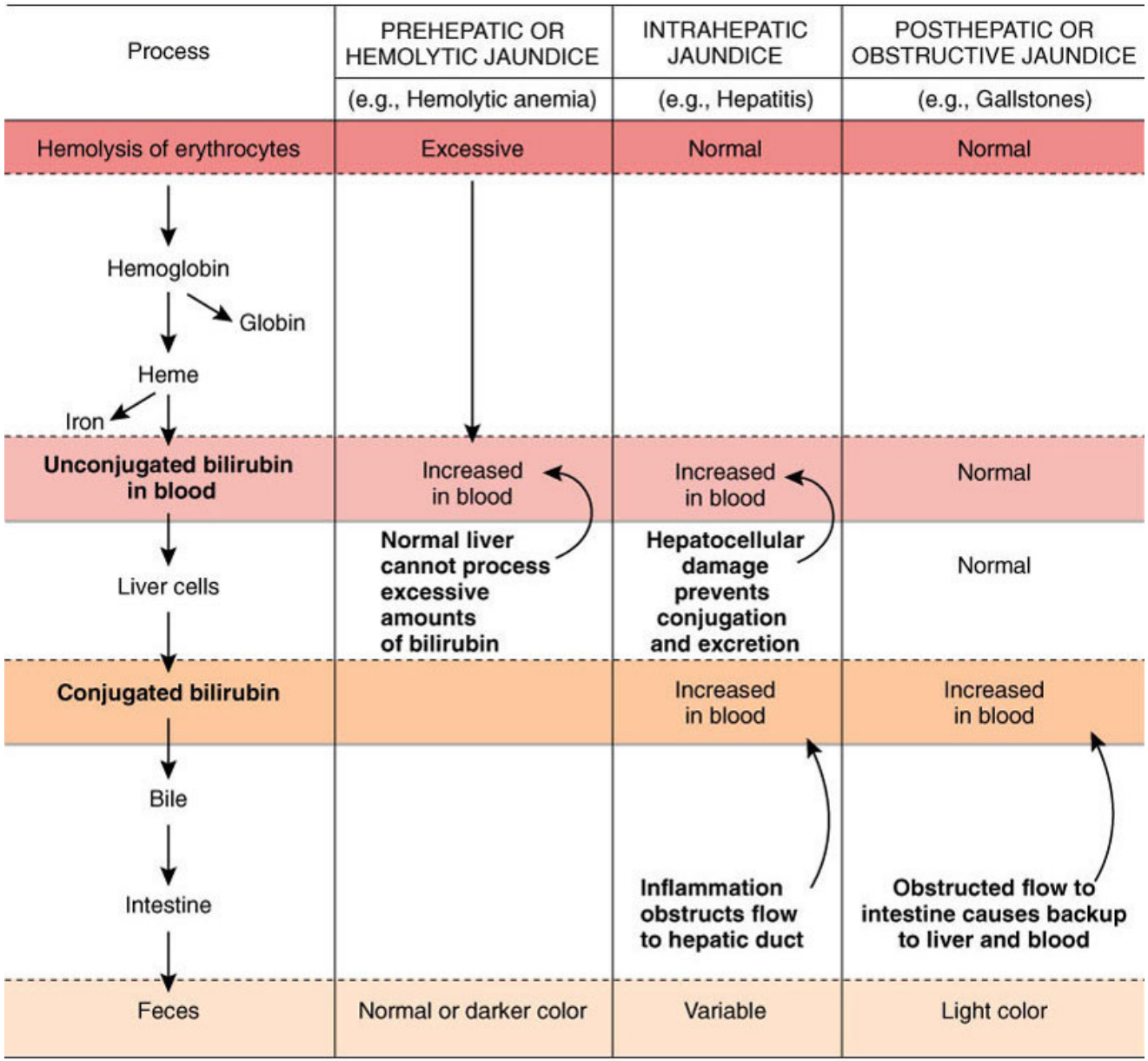
Gallbladder Disorders

- Obstruction of a duct by a large calculi
  - Sudden severe waves of pain // Radiating pain
  - Nausea and vomiting usually present
  - Pain continues, and jaundice develops.
    - Bile backs up into the liver and blood.
    - Risk of ruptured gallbladder if obstruction persists
    - Pain decreases if stone moves into duodenum
  - Surgical intervention may be necessary. // May be removed using laparoscopic surgery // Low-fat diet necessary following surgery

Jaundice

- Prehepatic jaundice // Result of excessive destruction of red blood cells // Characteristic of hemolytic anemias or transfusion reactions
- Intrahepatic jaundice // Occurs with disease or damage to hepatocytes // Hepatitis or cirrhosis
- Posthepatic jaundice // Caused by obstruction of bile flow into gallbladder or duodenum // Tumor, cholelithiasis

Types of Jaundice



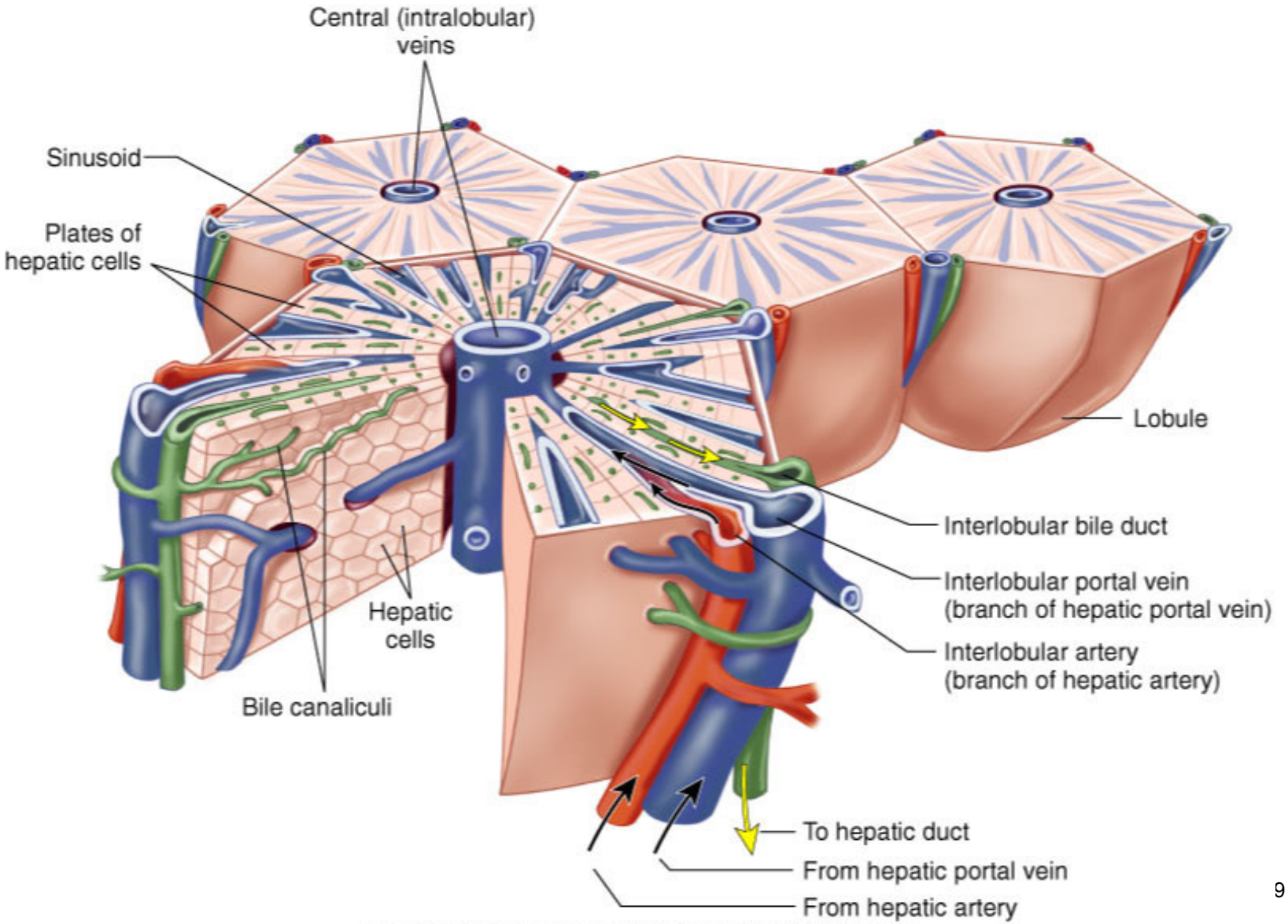
Bilirubin Measurement in Jaundice

- Direct or conjugated bilirubin can be measured in the blood.
- Total bilirubin is measured in blood.
- Total bilirubin minus direct bilirubin = indirect or unconjugated bilirubin.

Jaundice

- Type of jaundice indicated by increase in serum bilirubin level and changes in stools
- Prehepatic jaundice // Unconjugated bilirubin level elevated
- Intrahepatic jaundice // Both unconjugated and conjugated bilirubin levels may be elevated.
- Posthepatic jaundice // Increased conjugated bilirubin level // Light-colored stool caused by absence of bile

Structure of Liver Lobule



From Patton KT, Thibodeau GA: Anatomy & Physiology, ed 8, St. Louis, 2013, Mosby.

Hepatitis

- Inflammation of the liver
- Alcoholic // Fatty liver
- Idiopathic // fatty liver
- Viral hepatitis // Local infection
- Infection elsewhere in body // Examples - infectious mononucleosis or amebiasis
- Chemical or drug toxicity

Viral Hepatitis

- Cell injury results in inflammation and necrosis in the liver. // Degrees of inflammation and damage vary.
- Liver is edematous and tender.
- Causative viruses
  - Hepatitis A virus (HAV)
  - Hepatitis B virus (HBV)
  - Hepatitis C virus (HCV)
  - Hepatitis D virus (HDV)
  - Hepatitis E virus (HEV)

Viral Hepatitis // Hepatitis A (HAV)

- Small RNA virus
- Infectious hepatitis
- Transmitted by fecal-oral route in areas of inadequate sanitation or hygiene // Often from contaminated water or shellfish
- Sexual transmission has occurred during anal intercourse.
- Acute but self-limiting infection
- No carrier or chronic state
- Fecal shedding of virus before onset of signs
- Vaccine available for travelers, food care workers, and health care workers

Viral Hepatitis // Hepatitis B (HBV)

- Partially double-stranded DNA virus
- Over 50% of HIV-positive patients are positive for HBV.
- 50% of patients are asymptomatic but contagious because of carrier state.
- Chronic inflammation can occur.
- Transmission primarily by infected blood
- Sexual transmission has been noted.
- Tattooing and body piercing may transmit the virus.
- Vaccine available, routinely given to children

Viral Hepatitis // Hepatitis C (HCV)

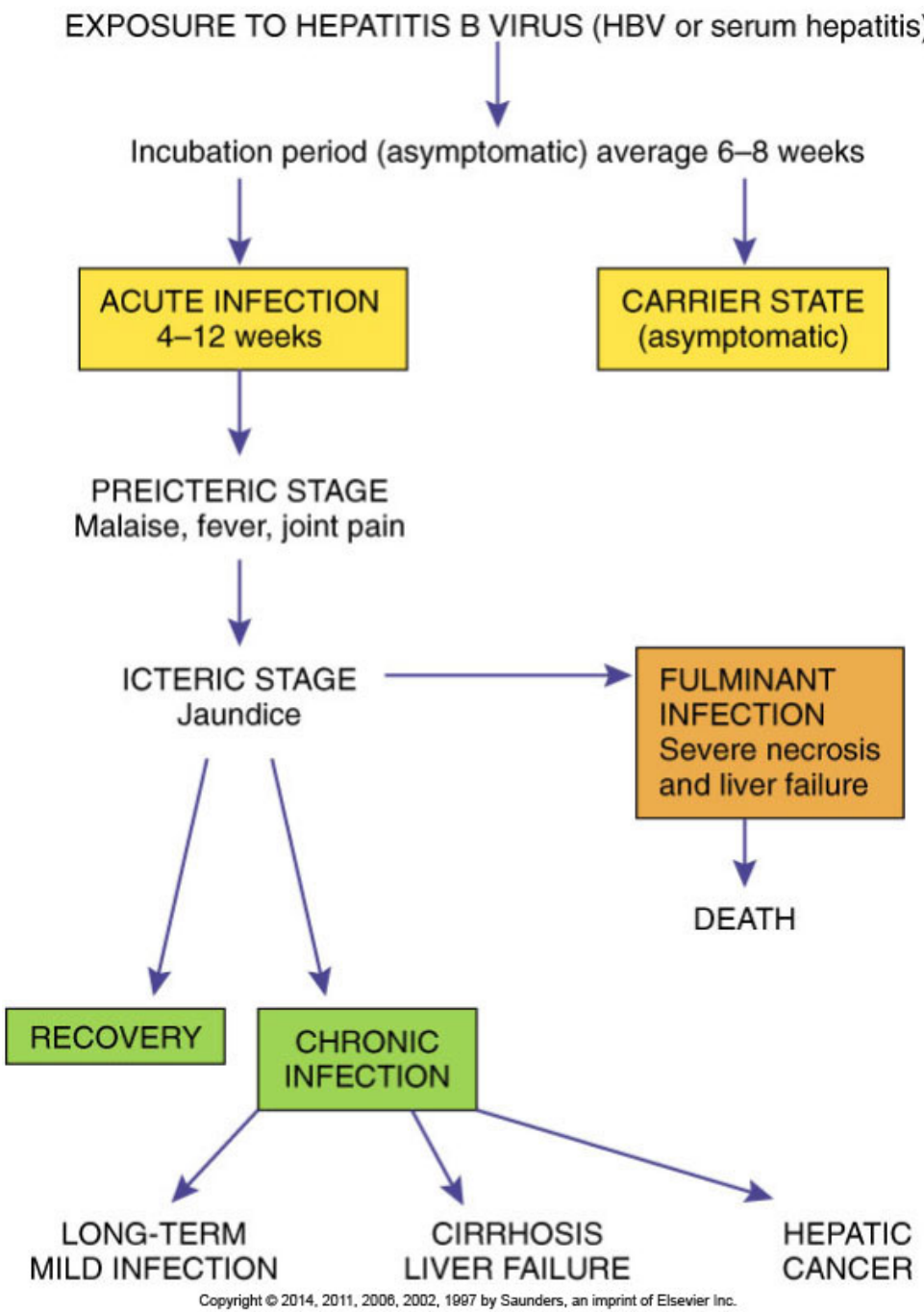
- Single-stranded RNA virus
- Most common type transmitted by blood transfusion
- May exist in a carrier state
- About 50% of patients enter the chronic state.
- Increases risk of hepatocellular carcinoma
- Treated with interferon injections

Viral Hepatitis

- Hepatitis D (HDV)
  - Also called delta virus
  - Incomplete RNA virus // Requires HBV to replicate and produce active infection
  - HDV infection increases severity of HBV infection
  - Transmitted by blood
- Hepatitis E (HEV)
  - Single-stranded RNA virus
  - Transmitted by oral-fecal route
  - No chronic or carrier state

Viral Hepatitis: Signs and Symptoms

- Preicteric stage
  - Fatigue and malaise
  - Anorexia and nausea
  - General muscle aching
- Icteric stage
  - Onset of jaundice
  - Stools light in color, urine becomes darker
  - Liver tender and enlarged, mild aching pain
- Posticteric stage—recovery stage
  - Reductions in signs
  - Weakness persists for weeks



Course of Hepatitis B Infection

Viral Hepatitis

- Only body defense is formation of antibodies via vaccination
- Supportive measures // Rest, diet high in protein, carbohydrate, and vitamins
- Chronic hepatitis can be treated with interferon.
  - Decreases viral replication
  - Effective in only 30% to 40% of individuals
  - Drug combination (slow-acting interferon plus antiviral drug) more effective

Toxic or Nonviral Hepatitis

- Variety of hepatotoxins can cause inflammation and necrosis of the liver.
  - Drugs include: // Acetaminophen, halothane, phenothiazines, tetracycline
  - Chemicals include: // Carbon tetrachloride (not used currently), toluene, ethanol
- Direct effect of toxins
- May result from sudden exposure to large amounts or from lower dose and long-term exposure

Cirrhosis

- Progressive destruction of the liver
- Causes
  - Alcoholic liver disease
  - Biliary cirrhosis // Associated with immune disorders
  - Postnecrotic cirrhosis // Linked with chronic hepatitis or long-term exposure to toxic materials
  - Metabolic // Usually caused by genetic metabolic storage disorders

Cirrhosis

- Extensive diffuse fibrosis // Interferes with blood supply // Bile may back up.
- Loss of lobular organization
- Degenerative changes may be asymptomatic until disease is well advanced.
- Liver biopsy and serologic test to determine cause and extent of damage

Cirrhosis: Alcoholic Liver Disease

- Initial stage - fatty liver // Enlargement of the liver // Asymptomatic and reversible with reduced alcohol intake
- Second stage - alcoholic hepatitis // Inflammation and cell necrosis // Fibrous tissue formation—irreversible change
- Third stage - end-stage cirrhosis // Fibrotic tissue replaces normal tissue. // Little normal function remains.

Functional Losses with Cirrhosis

- Decreased removal and conjugation of bilirubin
- Decreased production of bile
- Impaired digestion and absorption of nutrients
- Decreased production of blood-clotting factors
- Impaired glucose and glycogen metabolism
- Impaired conversion of ammonia to urea

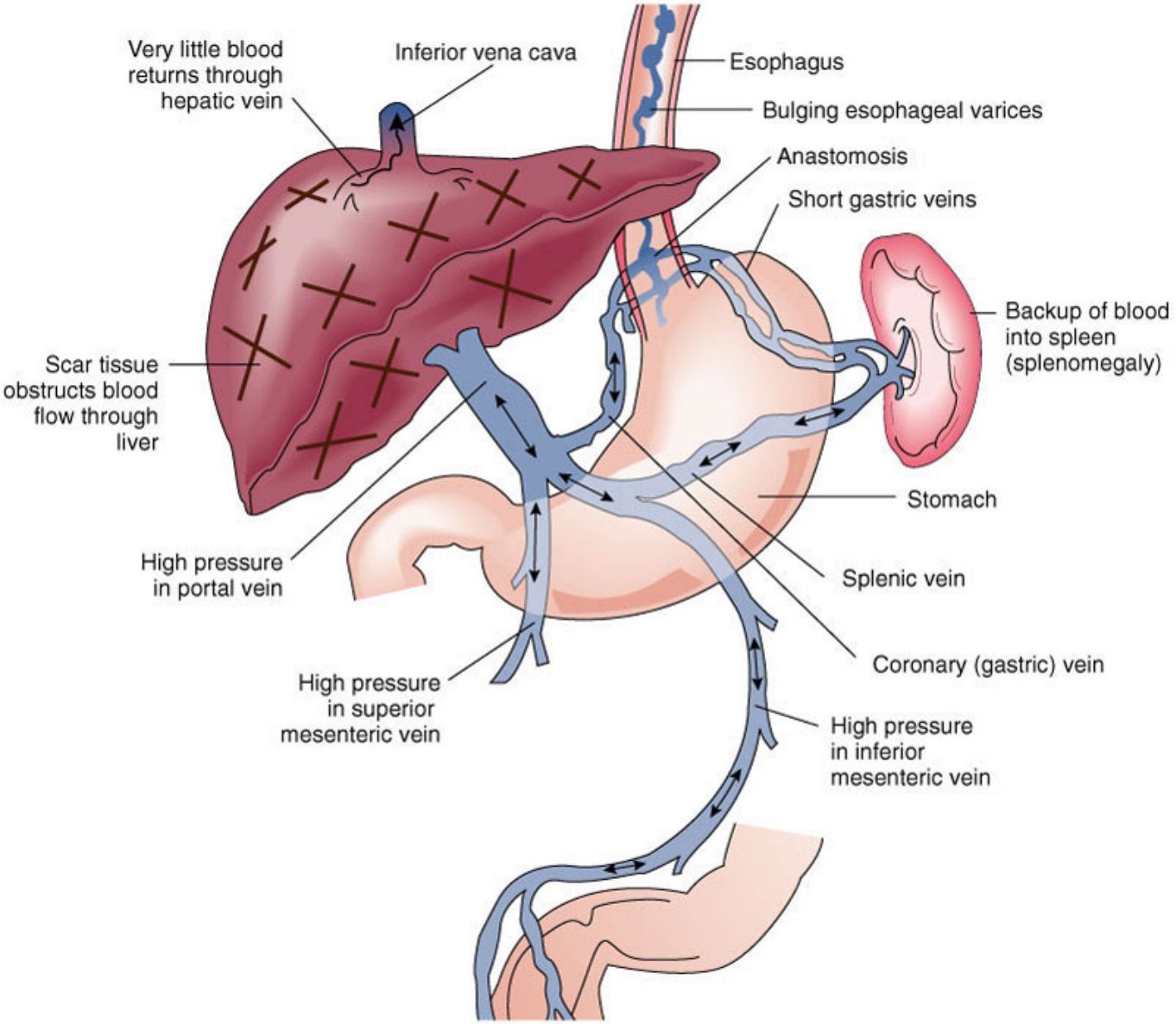
Functional Losses with Cirrhosis

- Decreased inactivation of hormones and drugs // ***Drug dosages must be carefully monitored to avoid toxicity.***
- Decreased removal of toxic substances
- Reduction of bile entering the intestine // Impairs digestion and absorption
- Backup of bile in the liver // Leads to obstructive jaundice
- Blockage of blood flow through the liver // Leads to portal hypertension
- Congestion in the spleen // Increases hemolysis

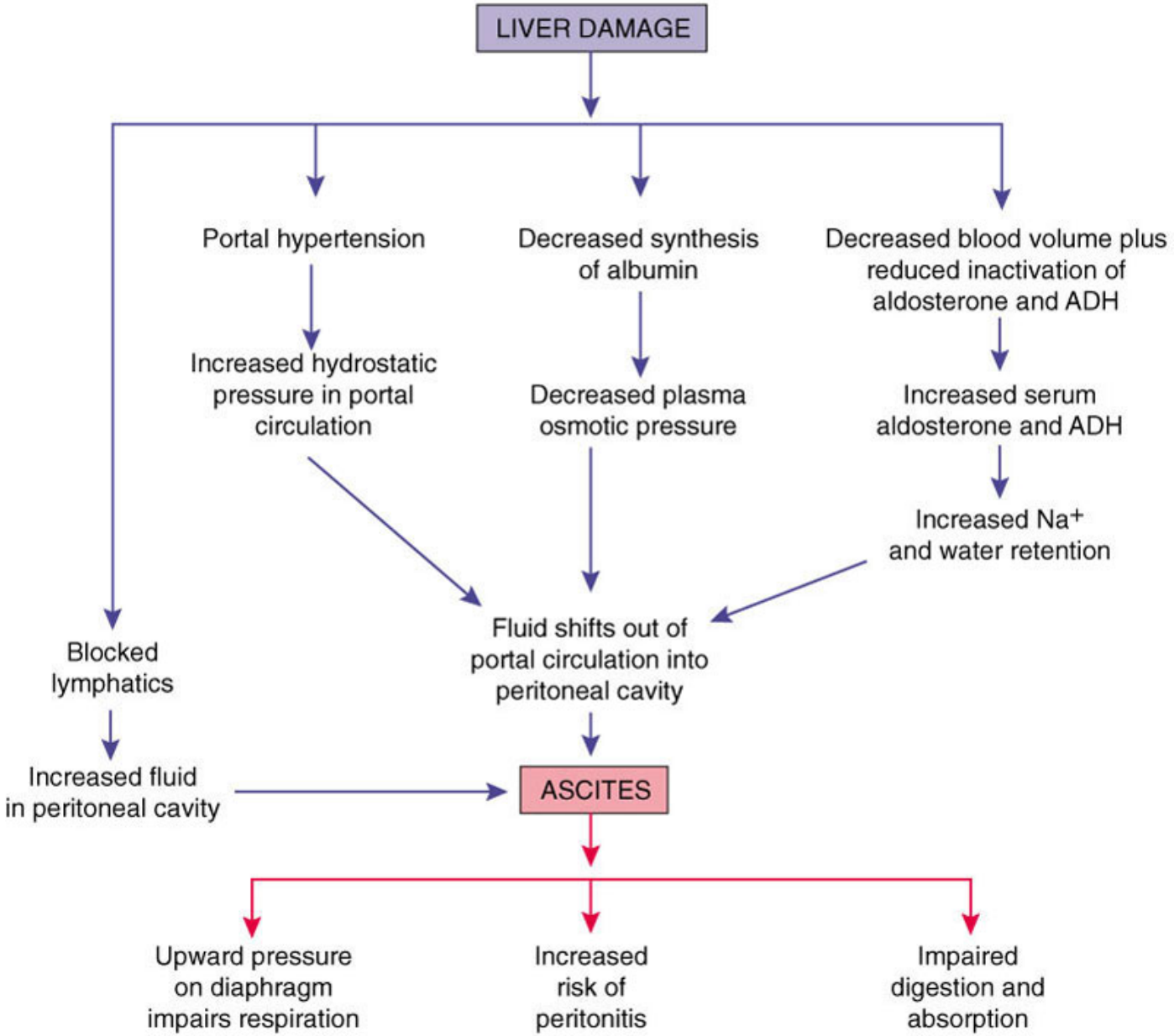
Functional Losses with Cirrhosis

- Inadequate storage of iron and vitamin B_{12}
- Congestion in intestinal walls and stomach // Impairing digestion and absorption
- Development of esophageal varices // Hemorrhage
- Development of ascites, an accumulation of fluid in the peritoneal cavity // Causes abdominal distention and pressure

Development of Esophageal Varices



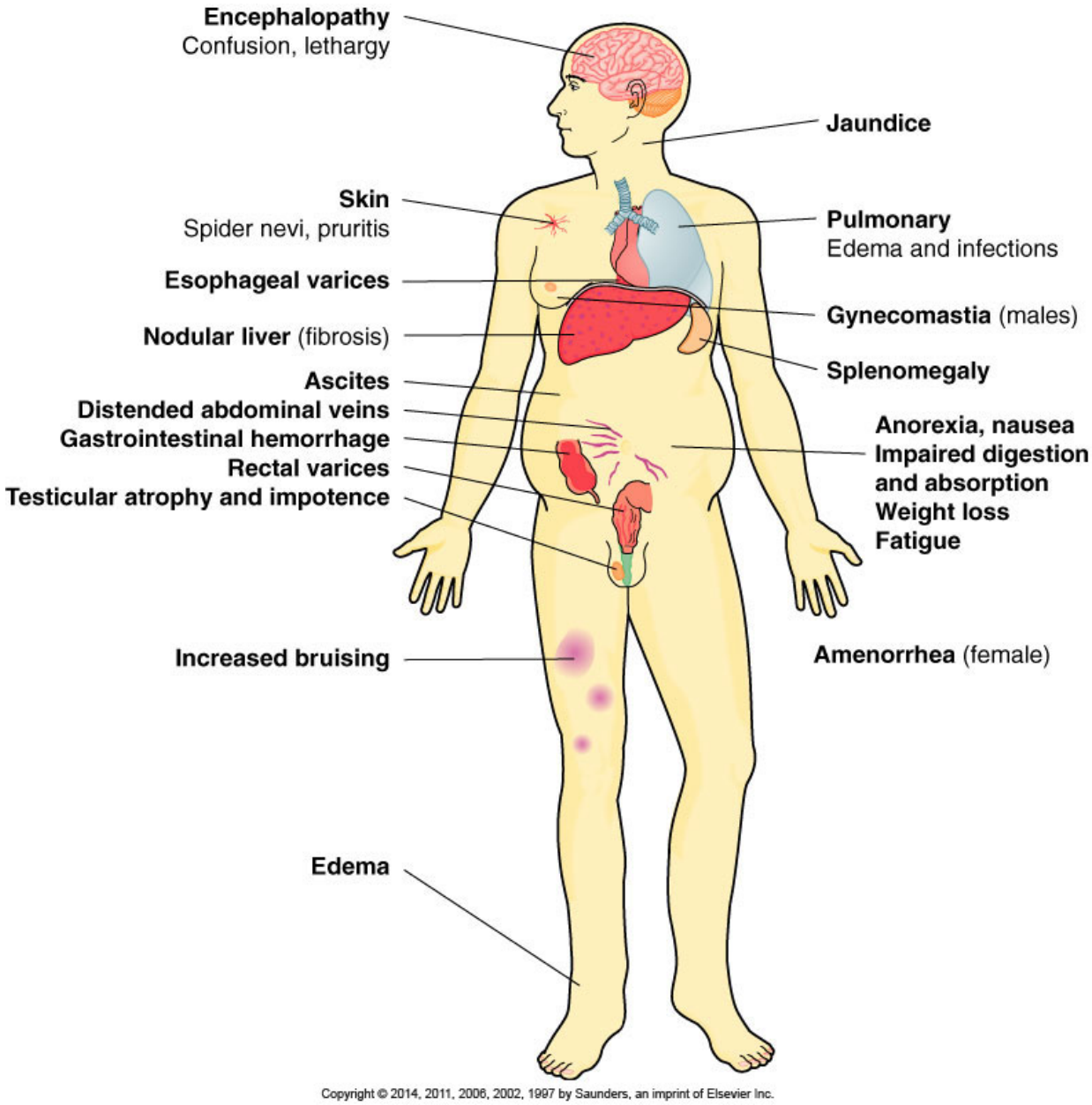
Development of Ascites with Cirrhosis



Cirrhosis

- Initial manifestations often mild and vague
  - Fatigue, anorexia, weight loss, anemia, diarrhea
  - Dull aching pain may be present in upper right abdominal quadrant.
- Advanced cirrhosis
  - Ascites and peripheral edema
  - Increased bruising
  - Esophageal varices // May rupture, leading to hemorrhage, circulatory shock
  - Jaundice, encephalopathy

Effects of Advanced Cirrhosis



Cirrhosis: Treatment

- Avoidance of alcohol or specific cause
- Supportive or symptomatic treatment
- Dietary restrictions
- Balancing serum electrolytes
- Paracentesis
- Antibiotics to reduce intestinal flora
- Emergency treatment if esophageal varices rupture
- Liver transplantation

TABLE 17-6 Common Manifestations of Liver Disease

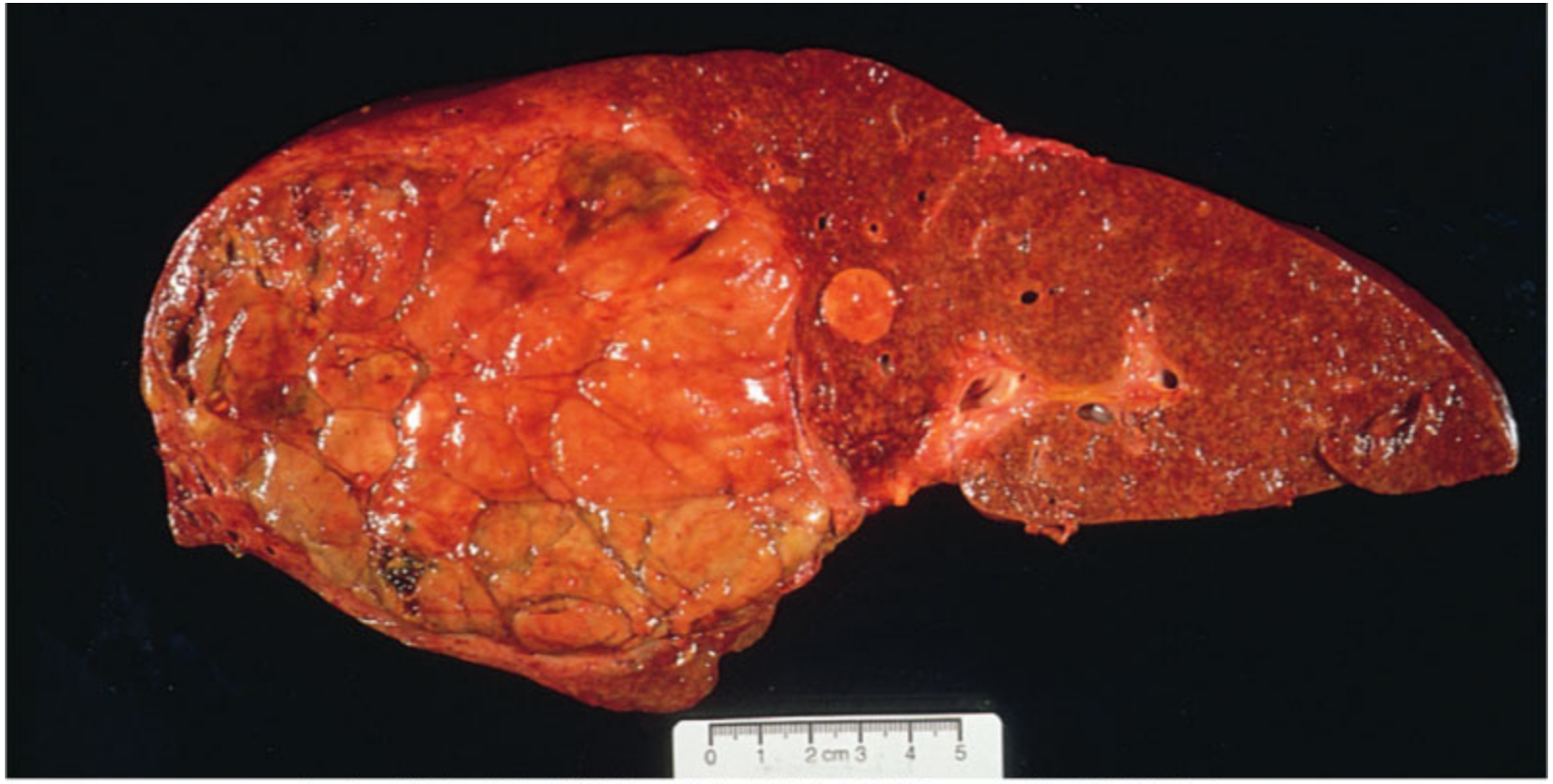
Signs or Symptoms	Pathophysiology
Fatigue, anorexia, indigestion, weight loss	Metabolic dysfunction in the liver, such as decreased gluconeogenesis; decreased bile for digestion and absorption; portal hypertension, leading to edema of intestinal wall and interfering with digestion and absorption
Ascites	Portal hypertension, elevated aldosterone and ADH levels, decreased serum albumin level, lymphatic obstruction in liver
General edema	Elevated aldosterone and ADH levels, decreased serum albumin level
Esophageal varices, hemorrhoids	Portal hypertension and collateral circulation
Splenomegaly	Portal hypertension
Anemia	Decreased absorption and storage of iron and vitamin B_{12}, malabsorption, splenomegaly, bleeding
Leukopenia, thrombocytopenia	Splenomegaly, possible bone marrow depression by ammonia and other toxins
Increased bleeding, purpura	Decreased absorption of vitamin K, decreased production of clotting factors by liver, thrombocytopenia
Hepatic encephalopathy, tremors, confusion, coma	Metabolic dysfunction with inability to remove ammonia from protein metabolism and other toxic substances
Gynecomastia, impotence, irregular menses	Impaired inactivation of sex hormones (e.g., estrogen) leads to imbalance
Jaundice	Impaired extraction and conjugation of bilirubin; decreased production of bile and obstruction of bile flow
Pruritus	Bile salts in the tissues resulting from biliary obstruction

ADH, antidiuretic hormone.

Liver Cancer

- Hepatocellular carcinoma // Most common primary tumor of liver // More common in cirrhotic livers
- Secondary or metastatic cancer // Arises from areas served by the hepatic vein or spread along the peritoneal membranes
- Initial signs are mild and general.
- Diagnosis usually occurs with advanced stages
- Chemotherapy, possible lobectomy or radiofrequency ablation (RFA) procedure

Hepatocellular Carcinoma



From Kumar V, Abbas AK, Fausto M: Robbins and Cotran Pathologic Basis of Disease, *ed 7, Philadelphia, 2005, Saunders.*

Acute Pancreatitis

- Inflammation of the pancreas // Results in autodigestion of the tissue
- May be acute or chronic // Acute form considered a medical emergency
- Pancreas lacks a fibrous capsule
  - Destruction may progress into tissue surrounding the pancreas
  - Substances released by necrotic tissue lead to widespread inflammation // Hypovolemia and circulatory collapse may follow.

Acute Pancreatitis

- Chemical peritonitis results in bacterial peritonitis.
  - Septicemia may result.
  - Adult respiratory distress syndrome and acute renal failure are possible complications.
- Causes
  - Gallstones
  - Alcohol abuse
  - Sudden onset may follow intake of large meal or large amount of alcohol

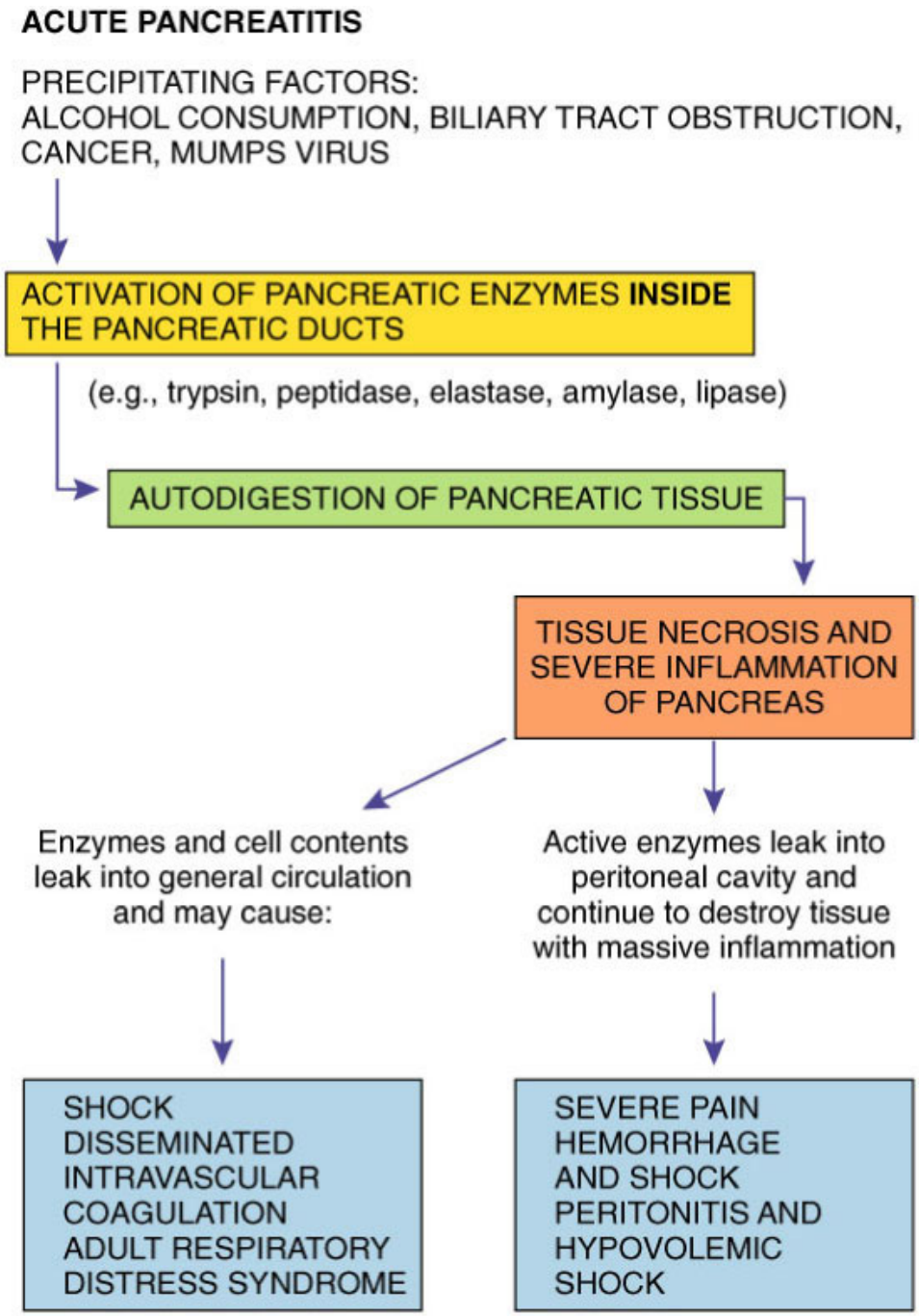
Acute Pancreatitis: Signs and Symptoms

- Severe epigastric or abdominal pain radiating to the back—primary symptoms
- Signs of shock // caused by hypovolemia
- Low-grade fever until infection develops // Body temperature may then rise significantly.
- Abdominal distention and decreased bowel sounds // Decreased peristalsis and paralytic ileus

Acute Pancreatitis

- Diagnostic tests
  - Serum amylase levels - first rise, then fall after 48 hours
  - Serum lipid levels are elevated.
  - Hypocalcemia
  - Leukocytosis
- Treatment
  - Oral intake is stopped.
  - Treatment of shock and electrolyte imbalances
  - Analgesics for pain relief

Pathophysiology of Acute Pancreatitis



Pancreatic Cancer

- Risk factors
  - Smoking
  - Pancreatitis and dietary factors
- Adenocarcinoma - most common form // Arises from the epithelial cells in the ducts
- Weight loss and jaundice early manifestations
- Frequently asymptomatic until well advanced
- Metastases occur early. // Mortality is close to 95%.

Lower Gastrointestinal Tract Disorders

Celiac Disease

- 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 – cause atrophy of villi // results in malabsorption and leads to malnutrition

Celiac Disease

- First signs appear when cereals are added to infant's diet. // At about 4 to 6 months of age
- Manifestation // Steatorrhea, muscle wasting, failure to gain weight --- Irritability and malaise common
- Diagnosed by a series of blood tests
- Gluten-free diet for treatment // Intestinal mucosa returns to normal after a few weeks without gluten intake.

Chronic Inflammatory Bowel Disease

- Two diseases // Crohn's disease and ulcerative colitis
- Causes unknown
- Genetic factor appears to be involved.
- Crohn's disease—often during adolescence
- Ulcerative colitis—second or third decade
- Many similarities between Crohn's disease and ulcerative colitis

TABLE 17-7 Inflammatory Bowel Disease

Characteristic	Crohn's Disease	Ulcerative Colitis
Region affected	Terminal ileum, sometimes colon	Colon, rectum
Distribution of lesions	Transmural, all layers Skip lesions	Mucosa only Continuous, diffuse
Characteristic stool	Loose, semi-formed	Frequent, watery, with blood and mucus
Granuloma	Common	No
Fistula, fissure, abscess	Common	No
Stricture, obstruction	Common	Rare
Malabsorption, malnutrition	Yes	Not common

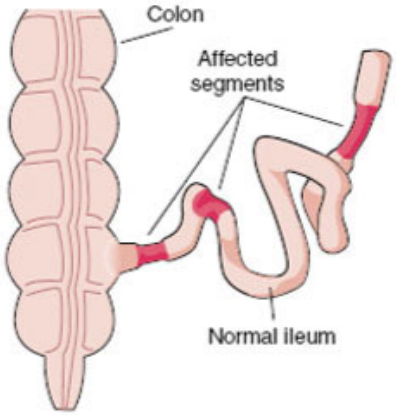
Crohn's Disease

- May affect any area of the digestive tract // Usually small intestine affected
- Inflammation occurs in characteristic distribution // "Skip lesions" - affected areas separated by areas of normal tissue
- Progressive inflammation and fibrosis may cause obstructed areas.
  - Damaged walls impair processing and absorption of food.
  - Inflammation stimulates intestinal motility.

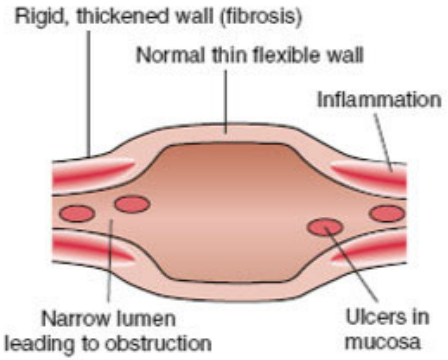
Crohn's Disease

- Interference with digestion and absorption // Hypoproteinemia, avitaminosis, malnutrition, possibly steatorrhea
- Other complications // Adhesions between loops may form and fistulas may develop.
- Children // Delayed growth and sexual maturation
- Glucocorticoid used in treatment

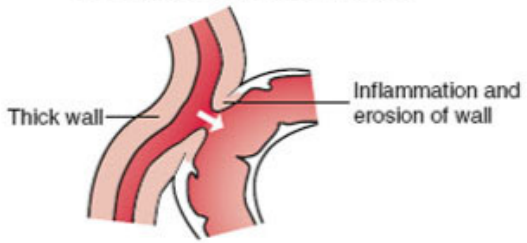
Crohn's Disease



A. "Skip lesions" — distribution of affected segments alternating with normal segments of bowel



B. Changes in the intestinal wall



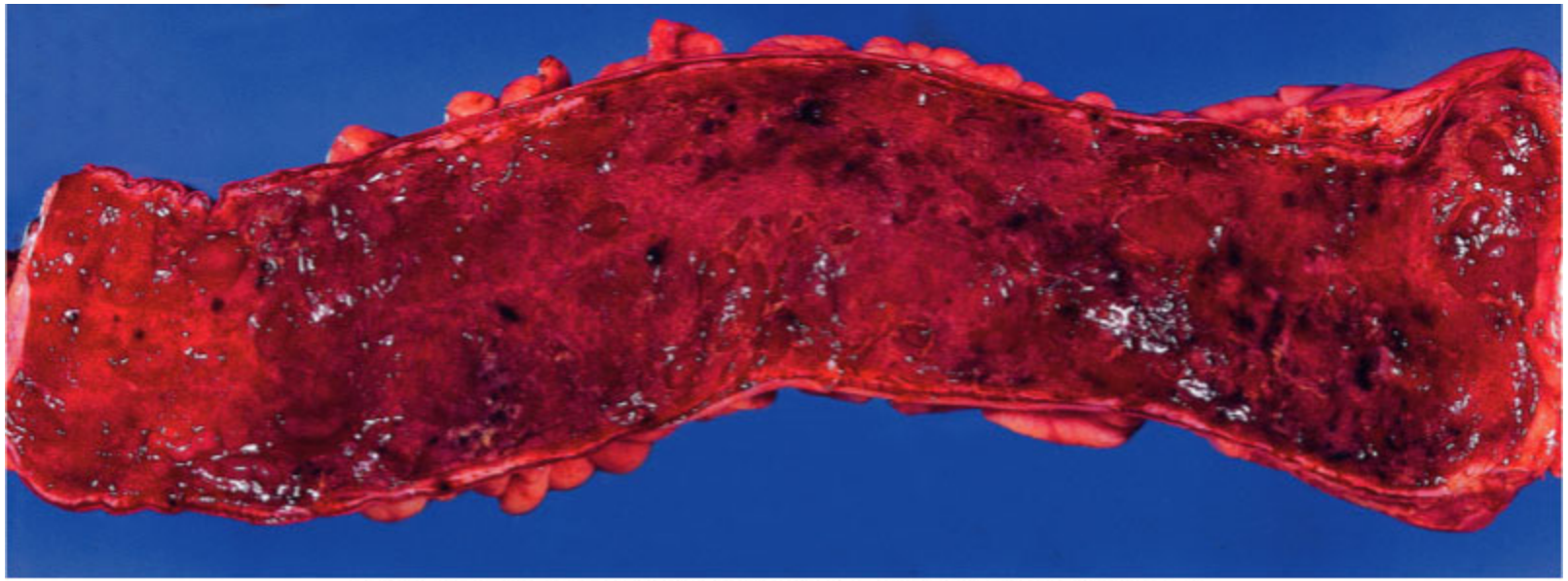
D. Fistula–abnormal opening between two structures

C and E courtesy of RW Shaw, North York General Hospital, Toronto, Ontario, Canada.

Ulcerative Colitis

- Inflammation starts in the rectum
- Progresses through the colon
- Mucosa and submucosa are inflamed. // Tissue destruction interferes with absorption of fluid and electrolytes in the colon.
- Severe acute episodes - toxic megacolon may develop.
- Marked diarrhea, with up to 12 stools per day // Contains blood and mucus -- Accompanied by cramping pain

Acute Ulcerative Colitis



From Cooke RA, Stewart B: Colour Atlas of Anatomical Pathology, *ed 3, Sydney, 2004, Churchill Livingstone.*

Treatment of IBD

- Team approach
- Anti-inflammatory medications // sulfasalazine & Glucocorticoids
- Antimotility agents
- Nutritional supplements
- Antimicrobials
- Immunotherapeutic agents
- Surgical resection // Usually ileostomy or colostomy

Irritable Bowel Syndrome

- Types
  - Abnormal gastrointestinal mobility and secretion
  - Visceral hypersensitivity
  - Postinfectious IBS
  - Overgrowth of flora
  - Food allergy or intolerance
  - Psychosocial factors

Irritable Bowel Syndrome (IBS): Manifestations and Diagnosis

- Manifestations
  - Lower abdominal pain
  - Diarrhea
  - Constipation, alternating with diarrhea
  - Bloating, nausea
- Diagnosis
  - Based on signs and symptoms
  - Testing for food allergies
  - Testing for bacterial or parasitic infections
  - No single cure for IBS

Appendicitis: Development

- Obstruction of the appendiceal lumen // By a fecalith, gallstone, or foreign material
- Fluid builds up inside the appendix. // Microorganisms proliferate
- Appendiceal wall becomes inflamed.
  - Purulent exudate forms
  - Appendix is swollen.
- Ischemia and necrosis of the wall // Results in increased permeability

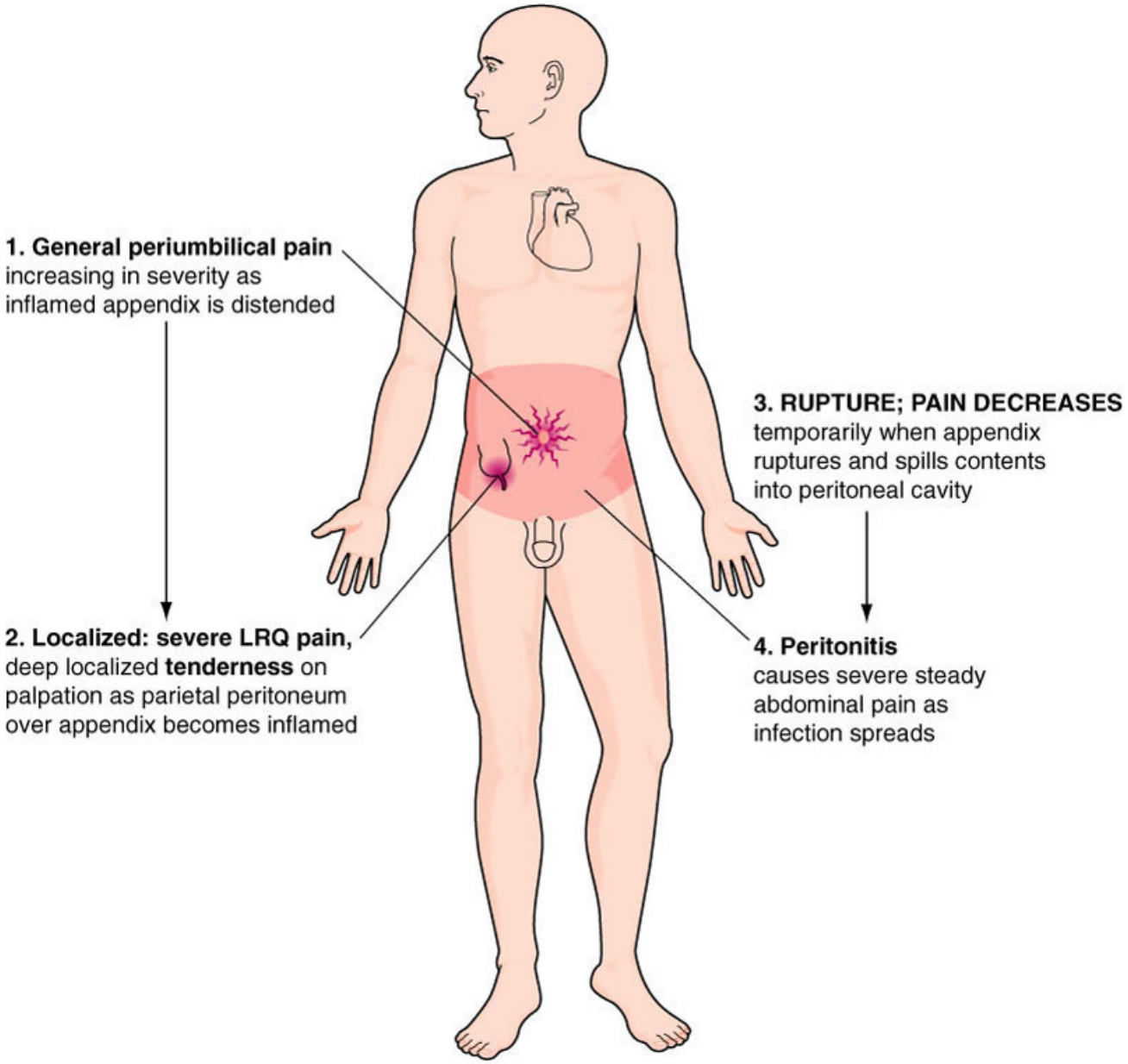
Appendicitis: Development

- Bacteria and toxins escape into surroundings.
  - Leads to abscess formation or localized bacterial peritonitis
- Abscess may develop when inflamed area is walled off. // Inflammation and pain may temporarily subside.
- Localized infection or peritonitis develops around the appendix. // May spread along the peritoneal membranes

Appendicitis: Development

- Increased necrosis and gangrene in the wall // Caused by increasing pressure in the appendix
- Appendix ruptures or perforates
  - Release of contents into peritoneal cavity
  - Generalized peritonitis // May be life-threatening
- Treatment // Surgical removal of appendix and antimicrobial drugs

Typical Progression of Pain in Acute Appendicitis



Appendicitis: Signs and Symptoms

- General periumbilical pain // Related to the inflammation
- Nausea and vomiting common
- Pain becomes severe and localized in lower right quadrant (LRQ).
- LRQ rebound tenderness develops. // Involvement of parietal peritoneum over appendix

Appendicitis: Signs and Symptoms

- After rupture // Pain subsides temporarily.
- Pain recurs - severe, generalized abdominal pain and guarding
- Low-grade fever and leukocytosis // Development of inflammation
- Boardlike abdomen, tachycardia, hypotension
  - As peritonitis develops, abdominal wall muscles spasm.
  - Toxins lead to reduced blood pressure.

Diverticular Disease

- Development of diverticula
- Diverticulum // Outpouching (herniation) of the mucosa through the muscular layer of the colon
- Diverticulosis Asymptomatic diverticular disease
- Diverticulitis // Inflammation of the diverticula

Diverticular Disease

- Form at gaps between muscle layers
- Congenital weakness of wall may be a factor
- Weaker areas bulge when pressure increases
- Many cases are asymptomatic.
- Diverticulitis stasis of material in diverticula leads to inflammation and infection. // Cramping, tenderness, nausea, vomiting // Slight fever and elevated white blood cell count
- Treatment of diverticulitis // Antimicrobial drugs // Dietary modifications to prevent stasis

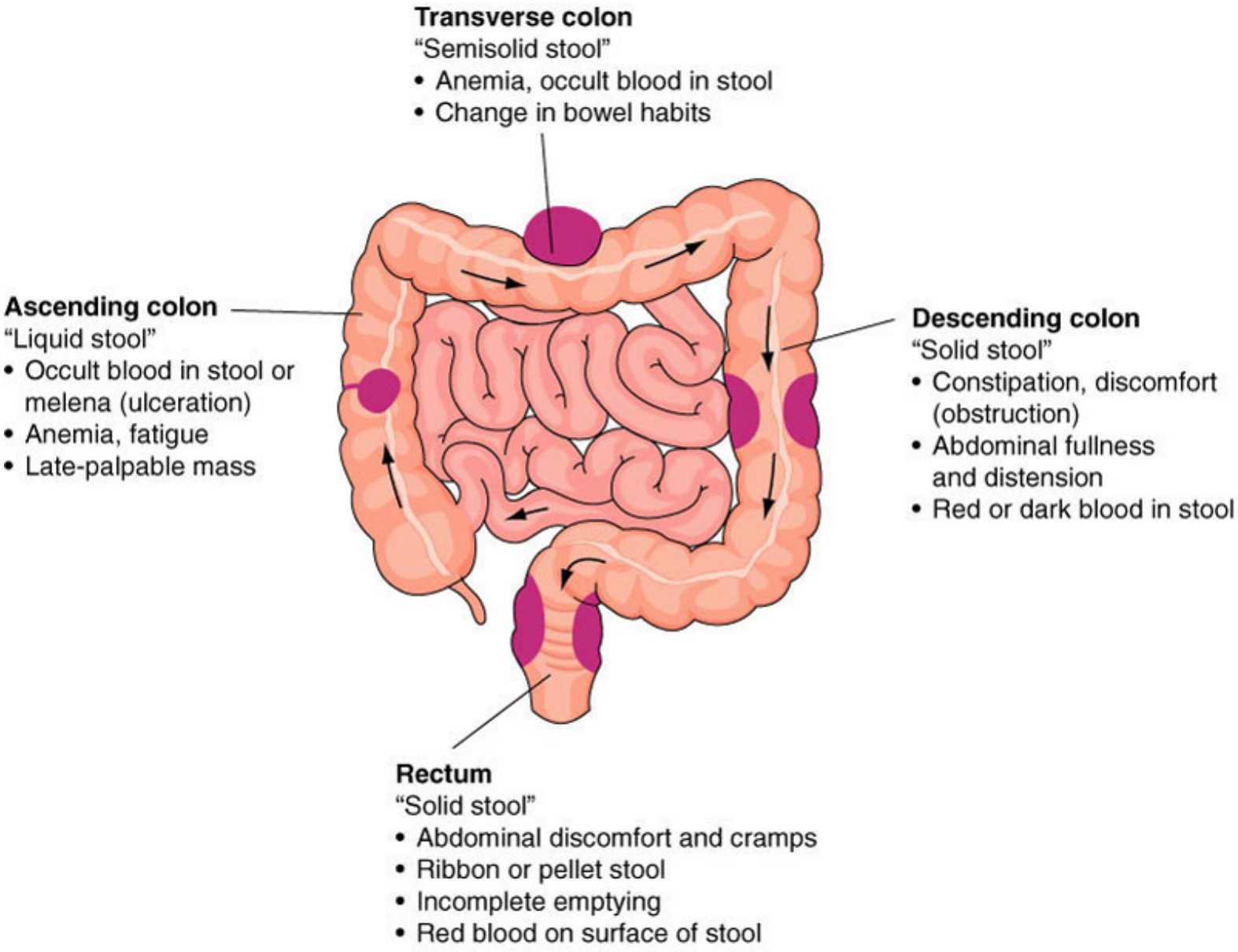
Colorectal Cancer

- Most malignancies develop from adenomatous polyps.
- Early diagnosis is essential.
- Cancer occurs primarily in persons older than 50 years.
- Risk factors
  - Familial multiple polyposis
  - Long-term ulcerative colitis
  - Genetic factors
  - Environmental factors // Diet low in fiber

Colorectal Cancer

- Initial signs depend largely on the location of the growth.
- General signs // Change in bowel habits // Alternating diarrhea and constipation
  - Bleeding
  - Fatigue, weight loss, anemia
- Treatment // Surgical removal with radiation and / or chemotherapy

Common Signs and Symptoms of Colorectal Cancer



Intestinal Obstruction

- Lack of movement of intestinal contents through the intestine // More common in small intestine
- Mechanical obstructions // Result from tumors, adhesions, hernias, other tangible obstructions
- Functional or adynamic obstructions // Result from impairment of peristalsis
    - Spinal cord injury
    - Paralytic ileus caused by toxins or electrolyte imbalance

Intestinal Obstruction

- Gases and fluids accumulate proximal to the blockage, distending the intestine.
- Increasingly strong contractions of proximal intestine // Effort to move contents along
- Pressure increases in lumen.
  - More secretions enter the intestine.
  - Compression of veins in wall // intestinal wall becomes edematous // Prevention of absorption

Intestinal Obstruction

- Intestinal distention leads to persistent vomiting.
  - Additional loss of fluid and electrolytes
  - Hypovolemia can result.
- Intestinal wall becomes ischemic and necrotic. // If obstruction is not removed, gangrene ensues.
- Ischemia and necrosis → decreased innervation and cessation of peristalsis
- Paralytic ileus occurs if it is not a cause to begin with.

Intestinal Obstruction

- Obstruction promotes rapid reproduction of intestinal bacteria.
  - Some produce endotoxins.
  - Affected wall becomes necrotic and more permeable
  - Bacteria and toxins leak into peritoneal cavity (peritonitis) or into blood (bacteremia and septicemia).
- Perforation of the necrotic segment may occur. // Generalized peritonitis and septic shock

Intestinal Obstruction

- Functional obstructions or paralytic ileus from:
  - Abdominal surgery (follows surgery)
  - Spinal shock following spinal cord injuries
  - Inflammation related to severe ischemia
  - Pancreatitis, peritonitis, infection in the abdominal cavity
  - Hypokalemia
  - Mesenteric thrombosis
  - Toxemia

Intestinal Obstruction

- Mechanical obstruction from:
  - Adhesions that twist or constrict intestine
  - Hernias & Strictures caused by scar tissue
  - Masses—tumors or foreign bodies
  - Intussusception
  - Volvulus
  - Hirschsprung's disease
  - Gradual obstruction from chronic inflammatory conditions

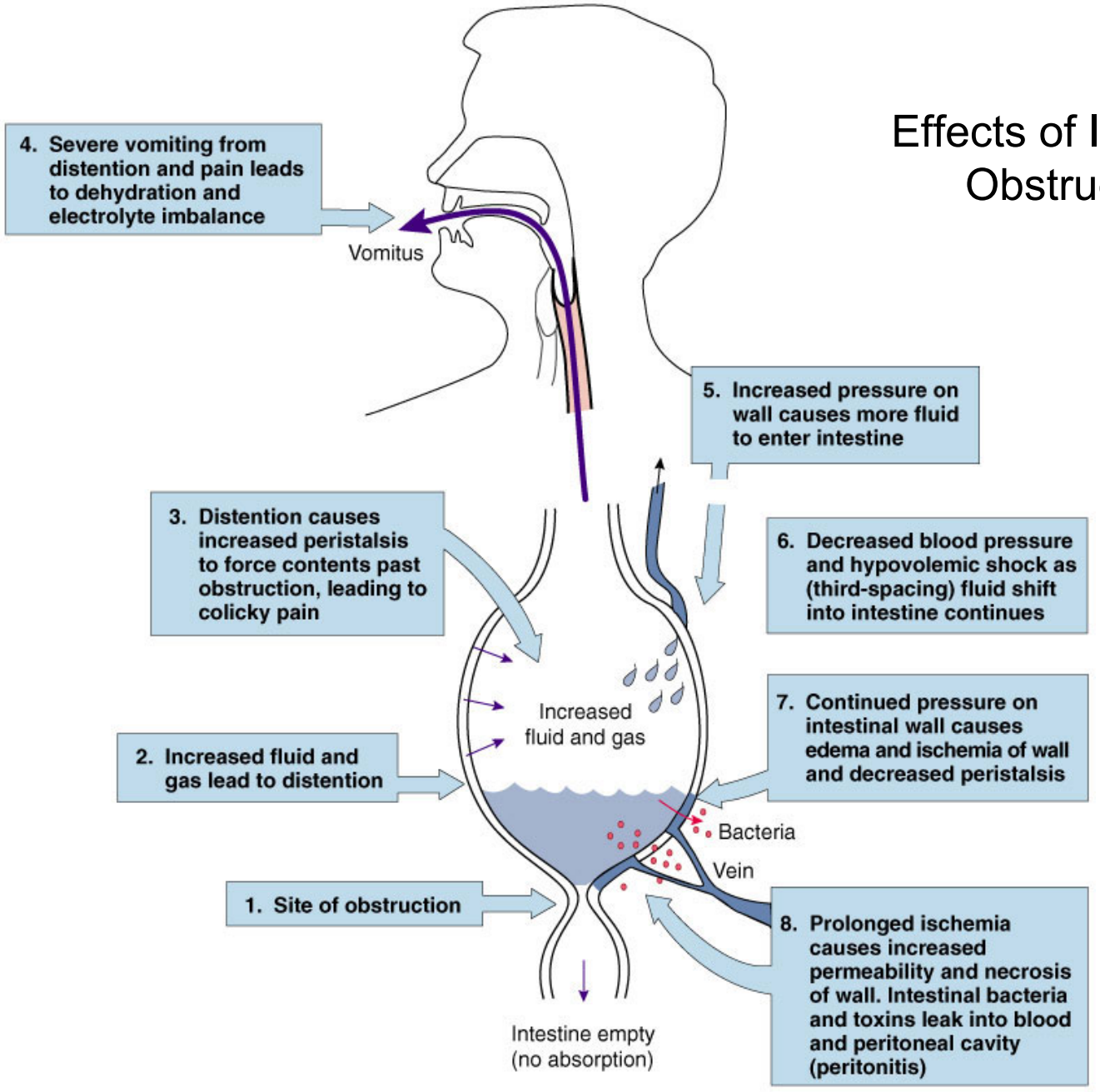
Intestinal Obstruction

- Mechanical obstruction of small intestine
  - Severe colicky abdominal pain // Intermittent bowel sounds can be heard.
- Paralytic ileus // Pain is steady. // Bowel sounds decrease or are absent.
- Vomiting and abdominal distention
  - Occurs quickly with obstruction of small intestine // Vomiting is recurrent, eventually with bile-stained content
- *Obstruction of the small intestine is a medical emergency!*

Intestinal Obstruction

- Obstruction of large intestine
  - Develops slowly, with mild signs
  - Constipation
  - Mild abdominal pain, followed by abdominal distention
  - Anorexia, vomiting, more severe pain
- Treatment
  - Treatment of underlying cause
  - Fluid and electrolyte replacement
  - Surgery and antimicrobial therapy

Effects of Intestinal Obstruction



Peritonitis

- Inflammation of the peritoneal membranes
- Chemical peritonitis may result from:
  - Enzymes released with pancreatitis
  - Urine leaking form a ruptured bladder
  - Chyme spilled from a perforated ulcer
  - Bile escaping from the ruptured gallbladder
  - Blood
  - Any other foreign material in the cavity

Peritonitis

- Bacterial peritonitis caused by:
  - Direct trauma affecting the intestine
  - Ruptured appendix
  - Intestinal obstruction and gangrene
- Any abdominal surgery // If foreign material is left or infection develops
- Pelvic inflammatory disease in women // When infection reaches the cavity through fallopian tubes

Peritonitis

- Signs and symptoms
  - Sudden, severe, generalized abdominal pain
  - Localized tenderness at site of underlying problem
  - Vomiting common, abdominal distention
  - Dehydration, hypovolemia, low blood pressure
  - Decreased blood pressure, tachycardia, fever, leukocytosis

Peritonitis

- Treatment
  - Depends on primary cause
  - Surgery might be required.
  - Massive antimicrobial drugs - specific to causative organism

Development of Peritonitis