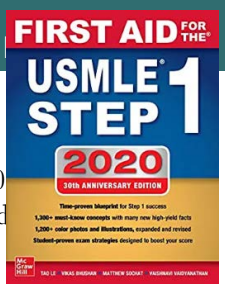
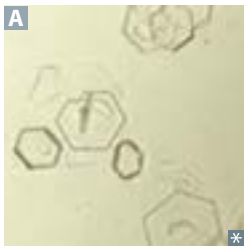


Cystinuria



Hereditary defect of renal PCT and intestinal amino acid transporter that prevents reabsorption of Cystine, Ornithine, Lysine, and Arginine (COLA).

Excess cystine in the urine can lead to recurrent precipitation of hexagonal cystine stones A.

Treatment: urinary alkalinization (eg, potassium citrate, acetazolamide) and chelating agents (eg, penicillamine) ↑ solubility of cystine stones; good hydration.

Autosomal recessive. Common (1:700

Urinary cyanide-nitroprusside test is d

Cystine is made of 2 cysteines connected by a disulfide bond.

Organic acidemias

Most commonly present in infancy with poor feeding, vomiting, hypotonia, high anion gap metabolic acidosis, hepatomegaly, seizures. Organic acid accumulation:

- Inhibits gluconeogenesis → ↓ fasting blood glucose levels, ↑ ketoacidosis → high anion gap metabolic acidosis
- Inhibits urea cycle → hyperammonemia

Propionic acidemia	Deficiency of propionyl-CoA carboxylase → ↑ propionyl-CoA, ↓ methylmalonic acid.	Treatment: low-protein diet limited in substances that metabolize into propionyl-CoA: Valine, Odd-chain fatty acids, Methionine, Isoleucine, Threonine (VOMIT).
Methylmalonic acidemia	Deficiency of methylmalonyl-CoA mutase or vitamin B_{12}.	

Glycogen regulation by insulin and glucagon/epinephrine

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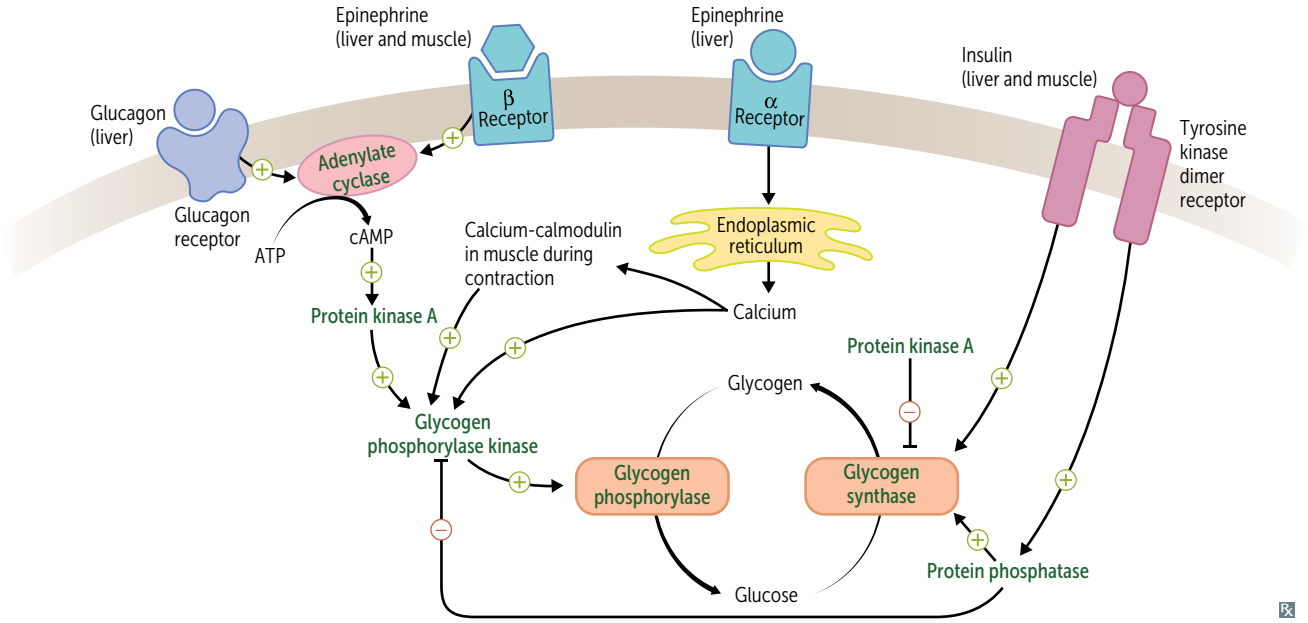

▸ IMMUNOLOGY—LYMPHOID STRUCTURES

Immune system organs

1° organs:
- Bone marrow—immune cell production, B cell maturation
- Thymus—T cell maturation

2° organs:
- Spleen, lymph nodes, tonsils, Peyer patches
- Allow immune cells to interact with antigen

Lymph node

A 2° lymphoid organ that has many afferents, 1 or more efferents. Encapsulated, with trabeculae A B. Functions are nonspecific filtration by macrophages, circulation of B and T cells, and immune response activation.

Follicle

Site of B-cell localization and proliferation. In outer cortex. 1° follicles are dense and quiescent. 2° follicles have pale central germinal centers and are active.

Medulla

Consists of medullary cords (closely packed lymphocytes and plasma cells) and medullary sinuses. Medullary sinuses communicate with efferent lymphatics and contain reticular cells and macrophages.

Paracortex

Contains T cells. Region of cortex between follicles and medulla. Contains high endothelial venules through which T and B cells enter from blood. Not well developed in patients with DiGeorge syndrome.

Paracortex enlarges in an extreme cellular immune response (eg, EBV and other viral infections → paracortical hyperplasia → lymphadenopathy).

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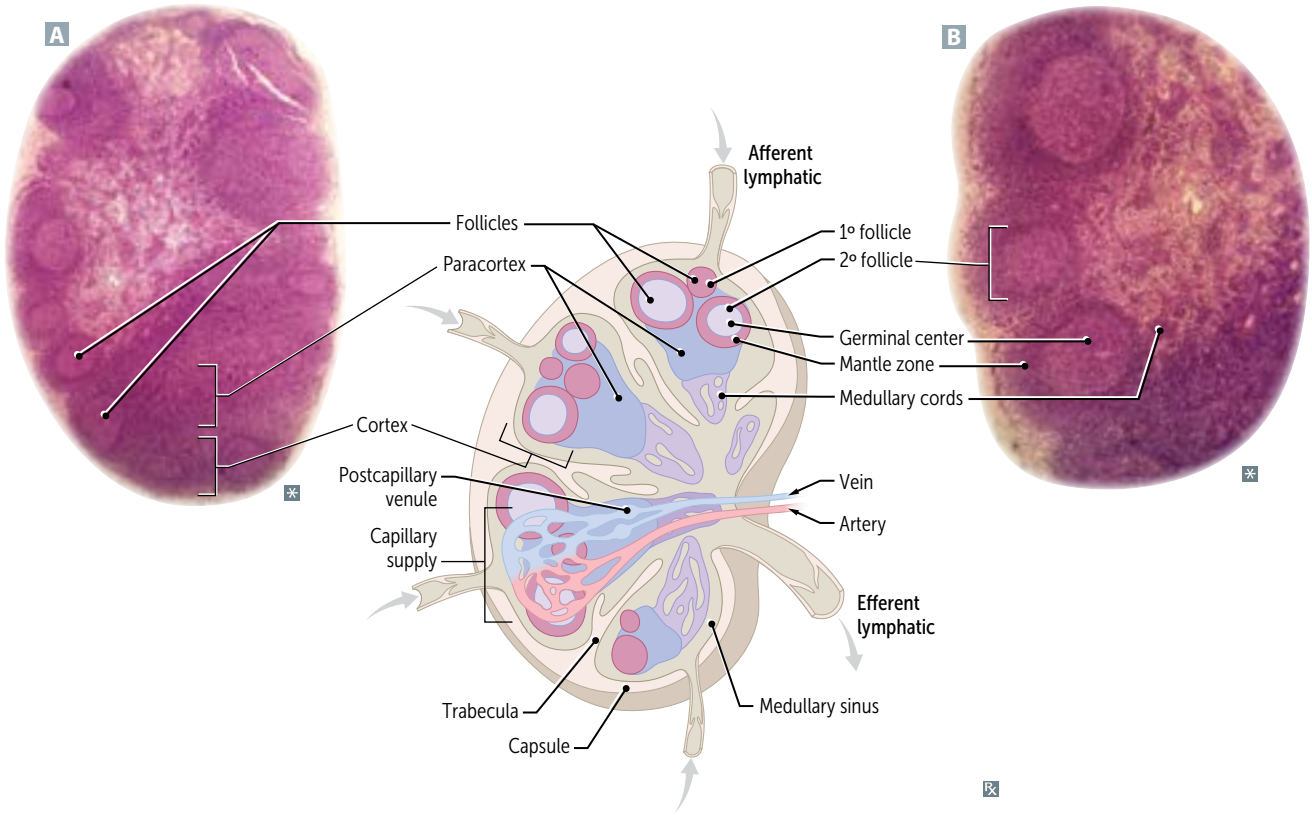
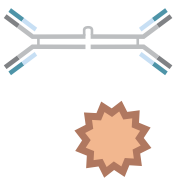
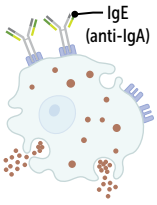
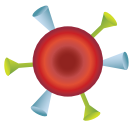
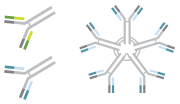
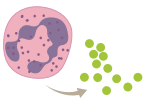
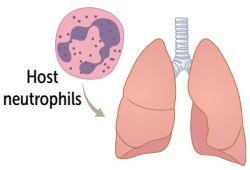
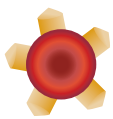


Blood transfusion reactions

TYPE	PATHOGENESIS	TIMING	CLINICAL PRESENTATION	DONOR BLOOD	HOST BLOOD
Allergic/ anaphylactic reaction	Type I hypersensitivity reaction against plasma proteins in transfused blood IgA-deficient individuals should receive blood products without IgA	Within minutes to 2-3 hr (due to release of preformed inflammatory mediators in degranulating mast cells)	Allergies: urticaria, pruritus Anaphylaxis: wheezing, hypotension, respiratory arrest, shock		
Acute hemolytic transfusion reaction	Type II hypersensitivity reaction Typically causes intravascular hemolysis (ABO blood group incompatibility)	During transfusion or within 24 hr (due to preformed antibodies)	Fever, hypotension, tachypnea, tachycardia, flank pain, hemoglobinuria (intravascular), jaundice (extravascular)		
Febrile nonhemolytic transfusion reaction	Cytokines created by donor WBCs accumulate during storage of blood products Reactions prevented by leukoreduction of blood products	Within 1-6 hr (due to preformed cytokines)	Fever, headaches, chills, flushing More common in children		
Transfusion-related acute lung injury	Two-hit mechanism: ▪ Neutrophils are sequestered and primed in pulmonary vasculature due to recipient risk factors ▪ Neutrophils are activated by a product (eg, antileukocyte antibodies) in the transfused blood and release inflammatory mediators → ↑ capillary permeability → pulmonary edema	Within minutes to 6 hr	Respiratory distress, noncardiogenic pulmonary edema		
Delayed hemolytic transfusion reaction	Anamnestic response to a foreign antigen on donor RBCs (most commonly Rh or other minor blood group antigens) previously encountered by recipient Typically causes extravascular hemolysis	Onset over 24 hr Usually presents within 1-2 wk (due to slow destruction by reticuloendothelial system)	Generally self limited and clinically silent Mild fever, hyperbilirubinemia		

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Influenza viruses

Orthomyxoviruses. Enveloped, ⊖ ssRNA viruses with 8-segment genome. Contain hemagglutinin (binds sialic acid and promotes viral entry) and neuraminidase (promotes progeny virion release) antigens. Patients at risk for fatal bacterial superinfection, most commonly *S aureus*, *S pneumoniae*, and *H influenzae*.

Reformulated vaccine ("the flu shot") contains viral strains most likely to appear during the flu season, due to the virus' rapid genetic change.
Killed viral vaccine is most frequently used.
Live attenuated vaccine contains temperature-sensitive mutant that replicates in the nose but not in the lung; administered intranasally.
Treatment: supportive +/– neuraminidase inhibitor (eg, oseltamivir, zanamivir).

Genetic/antigenic shift

Infection of 1 cell by 2 different segmented viruses (eg, swine influenza and human influenza viruses) → RNA segment reassortment → dramatically different virus (genetic shift) → major global outbreaks (pandemics).

Sudden shift is more deadly than gradual drift.

Genetic/antigenic drift

Random mutation in hemagglutinin or neuraminidase genes → minor changes (antigenic drift) → local outbreaks (epidemics).

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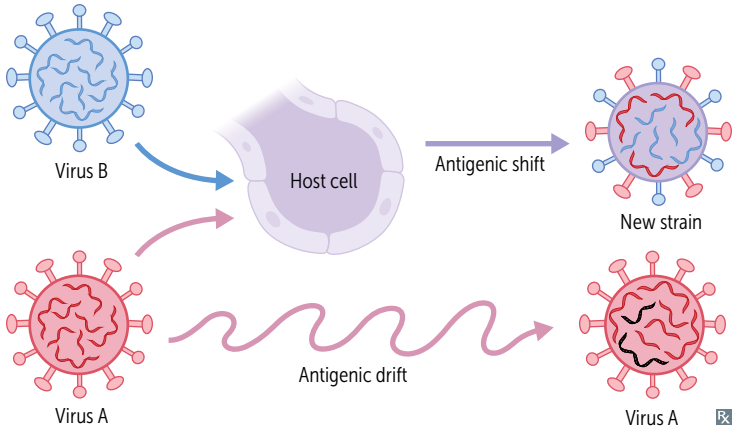
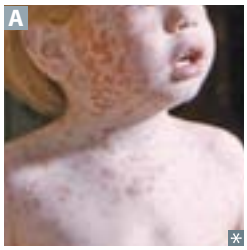


Rubella virus



A togavirus. Causes rubella, once known as German (3-day) measles. Fever, postauricular and other lymphadenopathy, arthralgias, and fine, maculopapular rash that starts on face and spreads centrifugally to involve trunk and extremities A.
Causes mild disease in children but serious congenital disease (a TORCH infection). Congenital rubella findings include "blueberry muffin" appearance due to dermal extramedullary hematopoiesis.

Paramyxoviruses

Paramyxoviruses cause disease in children. They include those that cause parainfluenza (croup), mumps, measles, RSV, and human metapneumovirus, which causes respiratory tract infection (bronchiolitis, pneumonia) in infants. All contain surface F (fusion) protein, which causes respiratory epithelial cells to fuse and form multinucleated cells. Palivizumab (monoclonal antibody against F protein) prevents pneumonia caused by RSV infection in premature infants.
Palivizumab for Paramyxovirus (RSV) Prophylaxis in Preemies.

Tumor nomenclature

Carcinoma implies epithelial origin, whereas **sarcoma** denotes mesenchymal origin. Both terms generally imply malignancy.

Benign tumors are usually well-differentiated and well-demarcated, with low mitotic activity, no metastases, and no necrosis.

Malignant tumors (cancers) may show poor differentiation, erratic growth, local invasion, metastasis, and ↓ apoptosis.

Terms for non-neoplastic malformations include hamartoma (disorganized overgrowth of tissues in their native location, eg, Peutz-Jeghers polyps) and choristoma (normal tissue in a foreign location, eg, gastric tissue located in distal ileum in Meckel diverticulum).

CELL TYPE	BENIGN	MALIGNANT
Epithelium	Adenoma, papilloma	Adenocarcinoma, papillary carcinoma
Mesenchyme		
Blood cells		Leukemia, lymphoma
Blood vessels	Hemangioma	Angiosarcoma
Smooth muscle	Leiomyoma	Leiomyosarcoma
Striated muscle	Rhabdomyoma	Rhabdomyosarcoma
Connective tissue	Fibroma	Fibrosarcoma
Bone	Osteoma	Osteosarcoma
Fat	Lipoma	Liposarcoma
Melanocyte	Nevus/mole	Melanoma

Tumor grade vs stage

Differentiation—degree to which a tumor resembles its tissue of origin. Well-differentiated tumors (often less aggressive) closely resemble their tissue of origin, whereas poorly differentiated tumors (often more aggressive) do not.

Anaplasia—complete lack of differentiation of cells in a malignant neoplasm.

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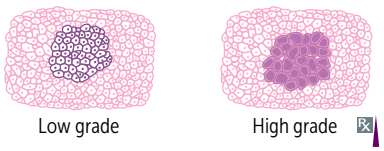
Grade

Degree of cellular differentiation and mitotic activity on histology. Ranges from low grade (well-differentiated) to high grade (poorly differentiated, undifferentiated, or anaplastic).



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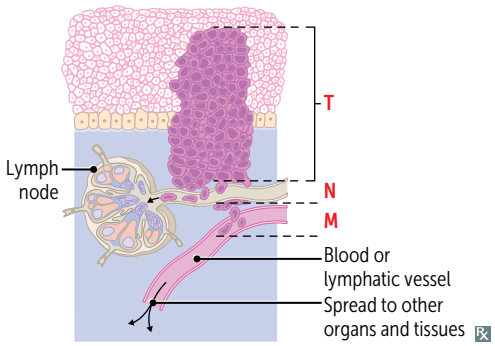
Stage

Degree of localization/spread based on site and size of 1° lesion, spread to regional lymph nodes, presence of metastases. Based on clinical (c) or pathologic (p) findings. Stage generally has more prognostic value than grade (eg, a high-stage yet low-grade tumor is usually worse than a low-stage yet high-grade tumor). Stage determines Survival.

TNM staging system (Stage = Spread):
T = Tumor size/invasiveness, N = Node involvement, M = Metastases, eg, cT3N1M0. Each TNM factor has independent prognostic value; N and M are often most important.



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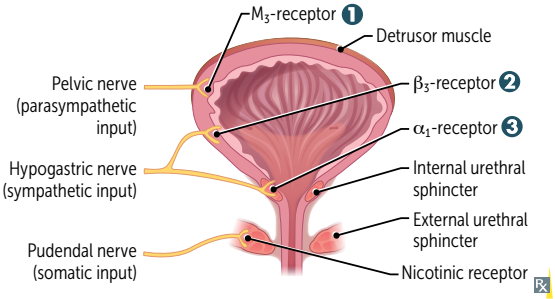
Micturition control

Micturition center in pons regulates involuntary bladder function via coordination of sympathetic and parasympathetic nervous systems.

- ⊕ sympathetic → ↑ urinary retention
- ⊕ parasympathetic → ↑ urine voiding. Some autonomic drugs act on smooth muscle receptors to treat bladder dysfunction.



DRUGS	MECHANISM	USE
1 **Muscarinic antagonists** (eg, oxybutynin)	⊖ M_3 receptor → relaxation of detrusor smooth muscle → ↓ detrusor overactivity	Urgency incontinence
1 **Muscarinic agonists** (eg, bethanechol)	⊕ M_3 receptor → contraction of detrusor smooth muscle → ↑ bladder emptying	Urinary retention
2 **Sympathomimetics** (eg, mirabegron)	⊕ β_3 receptor → relaxation of detrusor smooth muscle → ↑ bladder capacity	Urgency incontinence
3 **α_1-blockers** (eg, tamsulosin)	⊖ α_1-receptor → relaxation of smooth muscle (bladder neck, prostate) → ↓ urinary obstruction	BPH

Phosphodiesterase inhibitors

Phosphodiesterase (PDE) inhibitors inhibit PDE, which catalyzes the hydrolysis of cAMP and/or cGMP, and thereby increase cAMP and/or cGMP. These inhibitors have varying specificity for PDE isoforms and thus have different clinical uses.

TYPE OF INHIBITOR	MECHANISM OF ACTION	CLINICAL USES	ADVERSE EFFECTS
Nonspecific PDE inhibitor Theophylline	↓ cAMP hydrolysis → ↑ cAMP → bronchial smooth muscle relaxation → bronchodilation	COPD/asthma (rarely used)	Cardiotoxicity (eg, tachycardia, arrhythmia), neurotoxicity (eg, headache), abdominal pain
PDE-5 inhibitors Sildena**fil**, varden**afil**, tadal**afil**, avan**afil**	↓ hydrolysis of cGMP → ↑ cGMP → ↑ smooth muscle relaxation by enhancing NO activity → pulmonary vasodilation and ↑ blood flow in corpus cavernosum **fil**ls the penis	Erectile dysfunction Pulmonary hypertension BPH (tadalafil only)	Facial flushing, headache, dyspepsia, hypotension in patients taking nitrates; "**H**ot and sweaty," then **H**eadache, **H**eartburn, **H**ypotension Sildenafil only: cyanopia (blue-tinted vision) via inhibition of PDE-6 in retina
PDE-4 inhibitor Roflumilast	↑ cAMP in neutrophils, granulocytes, and bronchial epithelium	Severe COPD	Abdominal pain, weight loss, mental disorders (eg, depression)
PDE-3 inhibitor Milrinone	In cardiomyocytes: ↑ cAMP → ↑ Ca^{2+} influx → ↑ ionotropy and chronotropy In vascular smooth muscle: ↑ cAMP → MLCK inhibition → vasodilation → ↓ preload and afterload	Acute decompensated HF with cardiogenic shock	Tachycardia, ventricular arrhythmias (thus not for chronic use), hypotension
"Platelet inhibitors" Cilostazol[a] Dipyridamole[b]	In platelets: ↑ cAMP → inhibition of platelet aggregation	Intermittent claudication Stroke or TIA prevention (with aspirin) Cardiac stress testing (dipyridamole only, due to coronary vasodilation) Prevention of coronary stent restenosis	Nausea, headache, facial flushing, hypotension, abdominal pain

[a]Cilostazol is a PDE-3 inhibitor, but due to its indications is categorized as a platelet inhibitor together with dipyridamole.
[b]Dipyridamole is a nonspecific PDE inhibitor, leading to inhibition of platelet aggregation. It also prevents adenosine reuptake by platelets → ↑ extracellular adenosine → ↑ vasodilation.

Meta-analysis

A method of statistical analysis that pools summary data (eg, means, RRs) from multiple studies for a more precise estimate of the size of an effect. Also estimates heterogeneity of effect sizes between studies.

Improves power, strength of evidence, and generalizability of study findings. Limited by quality of individual studies and bias in study selection.

Common statistical tests

t-test	Checks differences between **mean**s of **2** groups.	**T**ea is **mean**t for **2**. Example: comparing the mean blood pressure between men and women.
ANOVA	Checks differences between means of **3** or more groups.	**3** words: **AN**alysis **O**f **VA**riance. Example: comparing the mean blood pressure between members of 3 different ethnic groups.
Chi-square (χ^2)	Checks differences between 2 or more percentages or proportions of **categorical** outcomes (not mean values).	Pronounce **Chi-tegorical**. Example: comparing the percentage of members of 3 different ethnic groups who have essential hypertension.
Fisher's exact test	Checks differences between 2 percentages or proportions of categorical, nominal outcomes. Use instead of chi-square test with small populations.	Example: comparing the percentage of 20 men and 20 women with hypertension.

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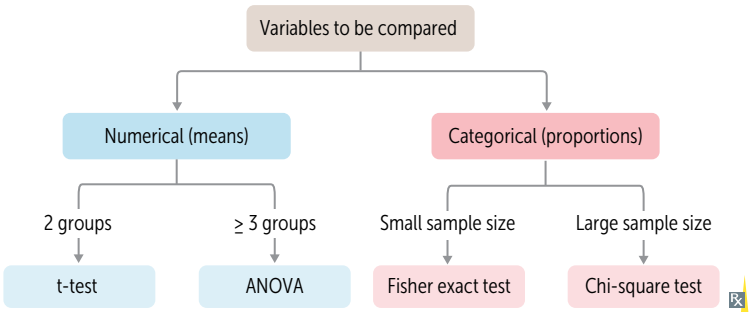


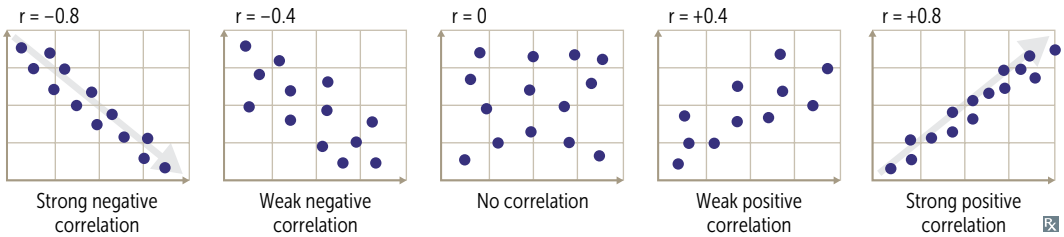
Pearson correlation coefficient

r is always between –1 and +1. The closer the absolute value of *r* is to 1, the stronger the linear correlation between the 2 variables. Variance is how much the measured values differ from the average value in a data set.

Positive *r* value → positive correlation (as one variable ↑, the other variable ↑).

Negative *r* value → negative correlation (as one variable ↑, the other variable ↓).

Coefficient of determination = r^2 (amount of variance in one variable that can be explained by variance in another variable).



Atherosclerosis

Very common. Disease of elastic arteries and large- and medium-sized muscular arteries; a form of arteriosclerosis caused by buildup of cholesterol plaques in intima.

LOCATION

Abdominal aorta > Coronary artery > Popliteal artery > Carotid artery > circle of Willis.
A CoPy Cat named Willis.

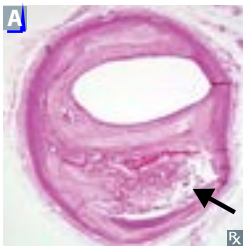
RISK FACTORS

Modifiable: smoking, hypertension, dyslipidemia (↑ LDL, ↓ HDL), diabetes.
Non-modifiable: age, sex (↑ in men and postmenopausal women), family history.

SYMPTOMS

Angina, claudication, but can be asymptomatic.

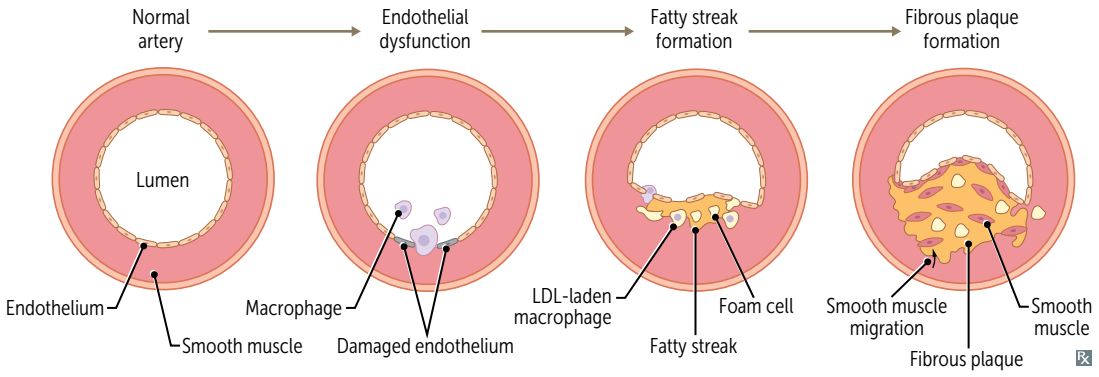
PROGRESSION



Inflammation important in pathogenesis: endothelial cell dysfunction → macrophage and LDL accumulation → foam cell formation → fatty streaks → smooth muscle cell migration (involves PDGF and FGF), proliferation, and extracellular matrix deposition → fibrous plaque → complex atheromas A → calcification (calcium content correlates with risk of complications).

COMPLICATIONS

Aneurysms, ischemia, infarcts, peripheral vascular disease, thrombus, emboli.



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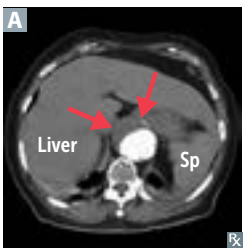
Aortic aneurysm

Localized pathologic dilation of the aorta. May cause abdominal and/or back pain, which is a sign of leaking, dissection, or imminent rupture.

Abdominal aortic aneurysm



Usually associated with atherosclerosis. Risk factors include history of tobacco use, ↑ age, male sex, family history. May present as palpable pulsatile abdominal mass (arrows in A point to outer dilated calcified aortic wall, with partial crescent-shaped non-opacification of aorta due to flap/clot). Most often infrarenal (distal to origin of renal arteries).

Thoracic aortic aneurysm

Associated with cystic medial degeneration. Risk factors include hypertension, bicuspid aortic valve, connective tissue disease (eg, Marfan syndrome). Also associated with 3° syphilis (obliterative endarteritis of the vasa vasorum). Aortic root dilatation may lead to aortic valve regurgitation.

Diagnosis of myocardial infarction

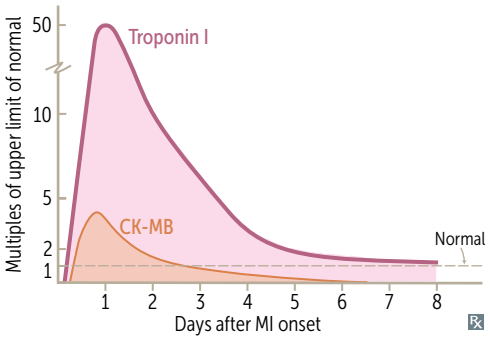
In the first 6 hours, ECG is the gold standard.

Cardiac troponin I rises after 4 hours (peaks at 24 hr) and is ↑ for 7–10 days; more specific than other protein markers.

CK-MB rises after 6–12 hours (peaks at 16–24 hr) and is predominantly found in myocardium but can also be released from skeletal muscle. Useful in diagnosing reinfarction following acute MI because levels return to normal after 48 hours.

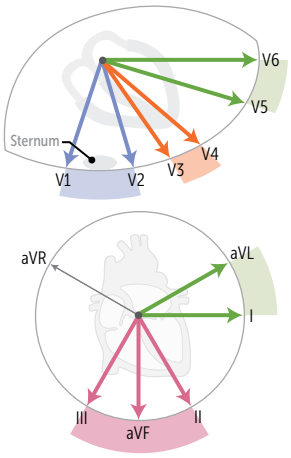
Large MIs lead to greater elevations in troponin I and CK-MB. Exact curves vary with testing procedure.

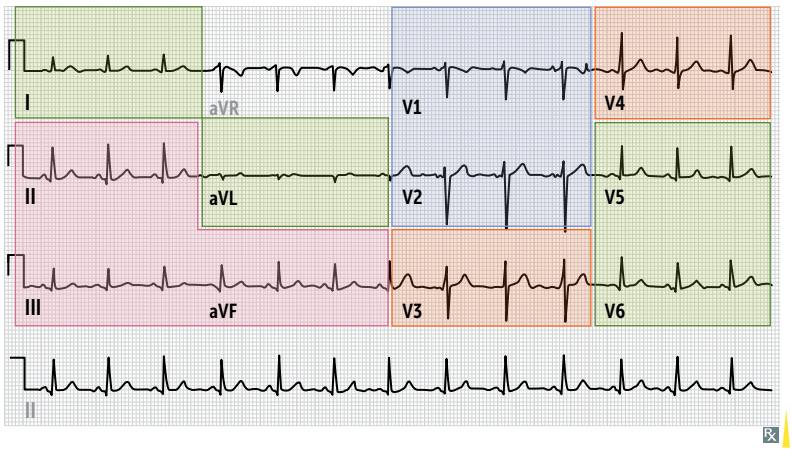
ECG changes can include ST elevation (STEMI, transmural infarct), ST depression (NSTEMI, subendocardial infarct), hyperacute (peaked) T waves, T-wave inversion, new left bundle branch block, and pathologic Q waves or poor R wave progression (evolving or old transmural infarct).



ECG localization of STEMI

INFARCT LOCATION	LEADS WITH ST-SEGMENT ELEVATIONS OR Q WAVES
Anteroseptal (LAD)	V_1–V_2
Anteroapical (distal LAD)	V_3–V_4
Anterolateral (LAD or LCX)	V_5–V_6
Lateral (LCX)	I, aVL
InFerior (RCA)	II, III, aVF
Posterior (PDA)	V_7–V_9, ST depression in V_1–V_3 with tall R waves





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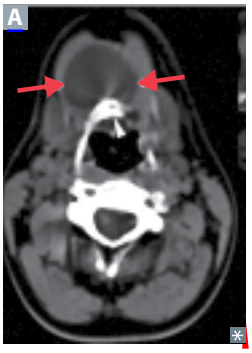
▸ ENDOCRINE—EMBRYOLOGY

Thyroid development

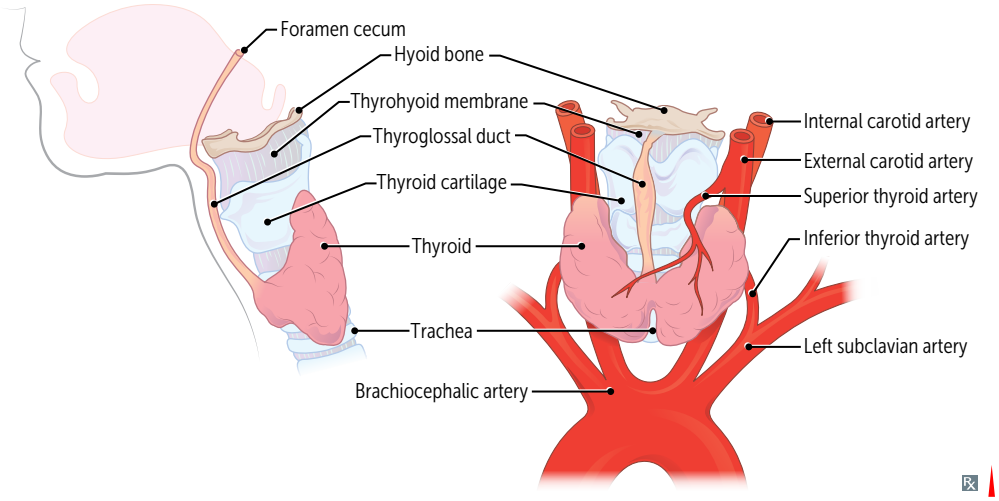


Thyroid diverticulum arises from floor of primitive pharynx and descends into neck. Connected to tongue by thyroglossal duct, which normally disappears but may persist as cysts or the pyramidal lobe of thyroid. Foramen cecum is normal remnant of thyroglossal duct.

Most common ectopic thyroid tissue site is the tongue (lingual thyroid). Removal may result in hypothyroidism if it is the only thyroid tissue present.

Thyroglossal duct cyst A presents as an anterior midline neck mass that moves with swallowing or protrusion of the tongue (vs persistent cervical sinus leading to pharyngeal cleft cyst in lateral neck).

Thyroid follicular cells derived from endoderm.



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Type 1 vs type 2 diabetes mellitus

	Type 1	Type 2
1° DEFECT	Autoimmune T-cell–mediated destruction of β cells (eg, due to presence of glutamic acid decarboxylase antibodies)	↑ resistance to insulin, progressive pancreatic β-cell failure
INSULIN NECESSARY IN TREATMENT	Always	Sometimes
AGE (EXCEPTIONS COMMON)	< 30 yr	> 40 yr
ASSOCIATION WITH OBESITY	No	Yes
GENETIC PREDISPOSITION	Relatively weak (50% concordance in identical twins), polygenic	Relatively strong (90% concordance in identical twins), polygenic
ASSOCIATION WITH HLA SYSTEM	Yes, HLA-DR4 and -DR3 (4 – 3 = type 1)	No
GLUCOSE INTOLERANCE	Severe	Mild to moderate
INSULIN SENSITIVITY	High	Low
KETOACIDOSIS	Common	Rare
β-CELL NUMBERS IN THE ISLETS	↓	Variable (with amyloid deposits)
SERUM INSULIN LEVEL	↓	↑ initially, but ↓ in advanced disease
CLASSIC SYMPTOMS OF POLYURIA, POLYDIPSIA, POLYPHAGIA, WEIGHT LOSS	Common	Sometimes
HISTOLOGY	Islet leukocytic infiltrate	Islet amyloid polypeptide (IAPP) deposits

Diabetic ketoacidosis

Insulin absent, ketones present (→ complications).
Insulin noncompliance or ↑ requirements from ↑ stress (eg, infection) → excess fat breakdown and ↑ ketogenesis from ↑ free fatty acids → ketone bodies (β-hydroxybutyrate > acetoacetate).

SIGNS/SYMPTOMS	DKA is Deadly: Delirium/psychosis, Kussmaul respirations (rapid, deep breathing), Abdominal pain/nausea/vomiting, Dehydration. Fruity breath odor (due to exhaled acetone).
LABS	Hyperglycemia, ↑ H^+, ↓ HCO_3^- (↑ anion gap metabolic acidosis), ↑ urine and blood ketone levels, leukocytosis. Normal/↑ serum K^+, but depleted intracellular K^+ due to transcellular shift from ↓ insulin and acidosis. Osmotic diuresis → ↑ K^+ loss in urine → total body K^+ depletion.
COMPLICATIONS	Life-threatening mucormycosis, cerebral edema, cardiac arrhythmias, HF.
TREATMENT	IV fluids, IV insulin, K^+ (to replete intracellular stores) +/– glucose to prevent hypoglycemia.

Hyperosmolar hyperglycemic state

Insulin present, ketones absent.
Profound hyperglycemia → excessive osmotic diuresis → dehydration and ↑ serum osmolality → HHS. Classically seen in elderly type 2 diabetics with limited ability to drink.

SIGNS/SYMPTOMS	Thirst, polyuria, lethargy, focal neurologic deficits, seizures.
LABS	Hyperglycemia (often >600 mg/dL), ↑ serum osmolality (> 320 mOsm/kg), normal pH (no acidosis), no ketones. Normal/↑ serum K^+, ↓ intracellular K^+.
COMPLICATIONS	Can progress to coma and death if untreated.
TREATMENT	IV fluids, IV insulin, and K^+ (to replete intracellular stores).

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Diabetes mellitus therapy *(continued)*

DRUG CLASS	MECHANISM	ADVERSE EFFECTS
Increase insulin sensitivity		
Biguanides Metformin	Inhibit mGPD → inhibition of hepatic gluconeogenesis and the action of glucagon. ↑ glycolysis, peripheral glucose uptake (↑ insulin sensitivity).	GI upset, lactic acidosis (use with caution in renal insufficiency), vitamin B_{12} deficiency. Weight loss (often desired).
Glitazones/ thiazolidinediones "-gliTs" Pioglitazone, rosiglitazone	Activate PPAR-γ (a nuclear receptor) → ↑ insulin sensitivity and levels of adiponectin → regulation of glucose metabolism and fatty acid storage.	Weight gain, edema, HF, ↑ risk of fractures. Delayed onset of action (several weeks). Rosiglitazone: ↑ risk of MI, cardiovascular death.
Increase insulin secretion		
Sulfonylureas (1st gen) Chlorpropamide, tolbutamide	Close K^+ channels in pancreatic B cell membrane → cell depolarizes → insulin release via ↑ Ca^{2+} influx.	DisulFIRam-like reaction (FIRst-generation only). Rarely used.
Sulfonylureas (2nd gen) Glipizide, glyburide		Hypoglycemia (↑ risk in renal insufficiency), weight gain.
Meglitinides "-gliNs" Nateglinide, repaglinide		
Increase glucose-induced insulin secretion		
GLP-1 analogs Exenatide, liraglutide	↓ glucagon release, ↓ gastric emptying, ↑ glucose-dependent insulin release.	Nausea, vomiting, pancreatitis. Weight loss (often desired). ↑ satiety (often desired).
DPP-4 inhibitors "-gliPs" Linagliptin, saxagliptin, sitagliptin	Inhibit DPP-4 enzyme that deactivates GLP-1 → ↓ glucagon release, ↓ gastric emptying. ↑ glucose-dependent insulin release.	Respiratory and urinary infections, weight neutral. ↑ satiety (often desired).
Decrease glucose absorption		
Sodium-glucose co-transporter 2 (SGLT2) inhibitors "-gliFs" Canagliflozin, dapagliflozin, empagliflozin	Block reabsorption of glucose in proximal convoluted tubule.	Glucosuria (UTIs, vulvovaginal candidiasis), dehydration (orthostatic hypotension), hyperkalemia, weight loss. Use with caution in renal insufficiency (↓ efficacy with ↓ GFR).
α-glucosidase inhibitors Acarbose, miglitol	Inhibit intestinal brush-border α-glucosidases → delayed carbohydrate hydrolysis and glucose absorption → ↓ postprandial hyperglycemia.	GI upset, bloating. Not recommended in renal insufficiency.
Others		
Amylin analogs Pramlintide	↓ glucagon release, ↓ gastric emptying.	Hypoglycemia, nausea. ↑ satiety (often desired).

Digestive tract anatomy

Layers of gut wall A (inside to outside—MSMS):

- Mucosa—epithelium, lamina propria, muscularis mucosa
- Submucosa—includes Submucosal nerve plexus (Meissner), Secretes fluid
- Muscularis externa—includes Myenteric nerve plexus (Auerbach), Motility
- Serosa (when intraperitoneal), adventitia (when retroperitoneal)

Ulcers can extend into submucosa, inner or outer muscular layer. Erosions are in mucosa only.

Frequency of basal electric rhythm (slow waves), which originate in the interstitial cells of Cajal:

- Stomach—3 waves/min
- Duodenum—12 waves/min
- Ileum—8–9 waves/min

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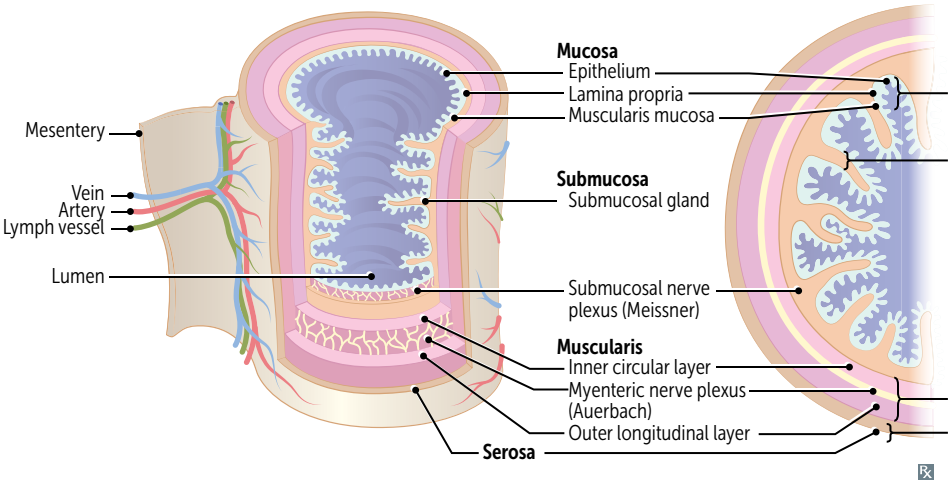

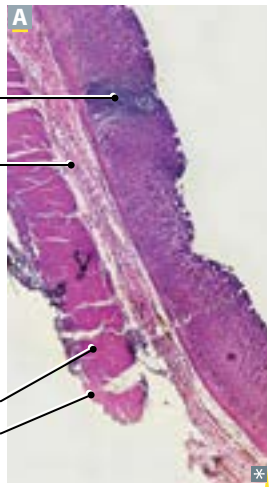

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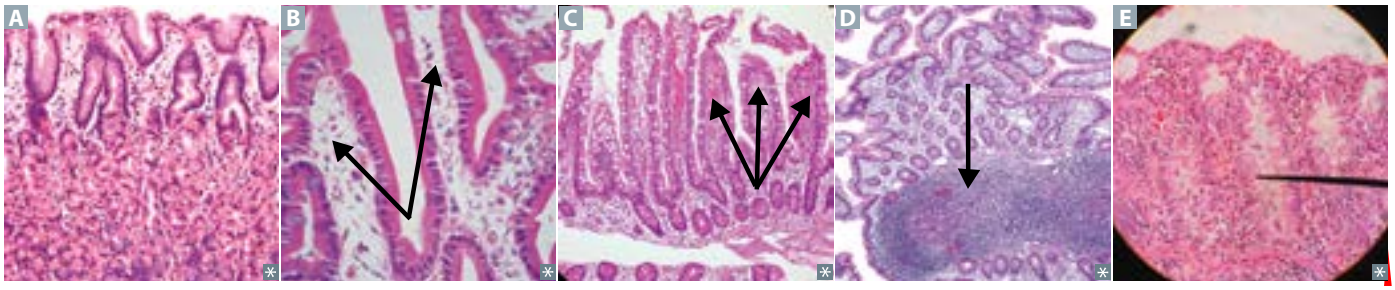
Digestive tract histology

Esophagus	Nonkeratinized stratified squamous epithelium. Upper 1/3, striated muscle; middle and lower 2/3, smooth muscle, with some overlap at the transition.
Stomach	Gastric glands A.
Duodenum	Villi B and microvilli ↑ absorptive surface. Brunner glands (HCO_3^--secreting cells of submucosa) and crypts of Lieberkühn (contain stem cells that replace enterocytes/goblet cells and Paneth cells that secrete defensins, lysozyme, and TNF).
Jejunum	Villi, crypts of Lieberkühn, and plicae circulares (also present in distal duodenum) C.
Ileum	Peyer patches (arrow in D; lymphoid aggregates in lamina propria, submucosa), plicae circulares (proximal ileum), and crypts of Lieberkühn. Largest number of goblet cells in the small intestine.
Colon	Crypts of Lieberkühn with abundant goblet cells, but no villi E.



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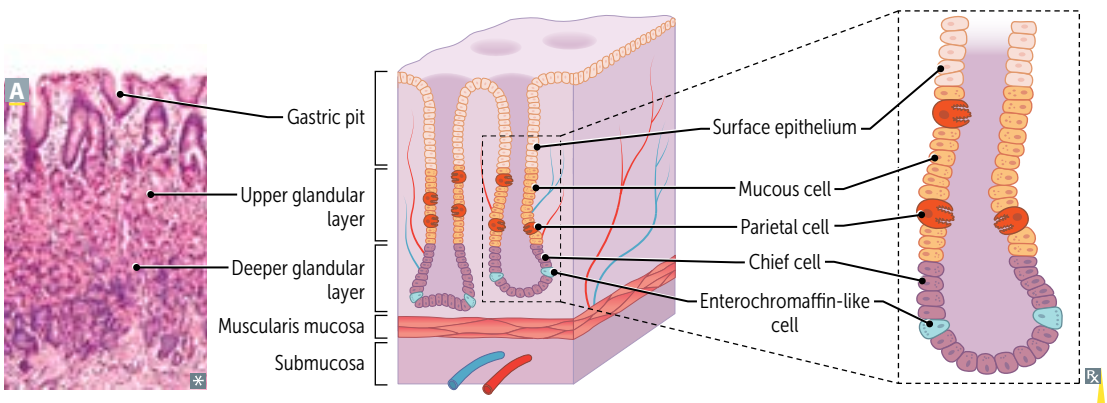
Gastrointestinal secretory products

PRODUCT	SOURCE	ACTION	REGULATION	NOTES
Intrinsic factor	Parietal cells (stomach A)	Vitamin B_{12}–binding protein (required for B_{12} uptake in terminal ileum)		Autoimmune destruction of parietal cells → chronic gastritis and pernicious anemia.
Gastric acid	Parietal cells (stomach)	↓ stomach pH	↑ by histamine, vagal stimulation (ACh), gastrin ↓ by somatostatin, GIP, prostaglandin, secretin	
Pepsin	Chief cells (stomach)	Protein digestion	↑ by vagal stimulation (ACh), local acid	Pepsinogen (inactive) is converted to pepsin (active) in the presence of H^+.
Bicarbonate	Mucosal cells (stomach, duodenum, salivary glands, pancreas) and Brunner glands (duodenum)	Neutralizes acid	↑ by pancreatic and biliary secretion with secretin	Trapped in mucus that covers the gastric epithelium.



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Platelet disorders

All platelet disorders have ↑ bleeding time (BT), mucous membrane bleeding, and microhemorrhages (eg, petechiae, epistaxis). Platelet count (PC) is usually low, but may be normal in qualitative disorders.

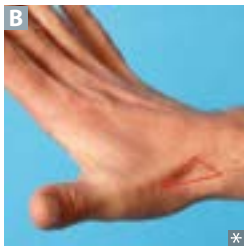
DISORDER	PC	BT	NOTES
Bernard-Soulier syndrome	–/↓	↑	Defect in adhesion. ↓ GpIb → ↓ platelet-to-vWF adhesion. Labs: abnormal ristocetin test, large platelets.
Glanzmann thrombasthenia	–	↑	Defect in aggregation. ↓ GpIIb/IIIa (↓ integrin $\alpha_{IIb}\beta_3$) → ↓ platelet-to-platelet aggregation and defective platelet plug formation. Labs: blood smear shows no platelet clumping.
Immune thrombocytopenia	↓	↑	Destruction of platelets in spleen. Anti-GpIIb/IIIa antibodies → splenic macrophages phagocytose platelets. May be idiopathic or 2° to autoimmune disorders (eg, SLE), viral illness (eg, HIV, HCV), malignancy (eg, CLL), or drug reactions. Labs: ↑ megakaryocytes on bone marrow biopsy, ↓ platelet count. Treatment: steroids, IVIG, rituximab, TPO receptor agonists (eg, eltrombopag, romiplostim), or splenectomy for refractory ITP.

Thrombotic microangiopathies

Disorders overlap significantly in symptomatology.

	Thrombotic thrombocytopenic purpura	Hemolytic-uremic syndrome
EPIDEMIOLOGY	Typically females	Typically children
PATHOPHYSIOLOGY	Inhibition or deficiency of ADAMTS13 (a vWF metalloprotease) → ↓ degradation of vWF multimers → ↑ large vWF multimers → ↑ platelet adhesion and aggregation (microthrombi formation)	Commonly caused by Shiga-like toxin from EHEC (serotype O157:H7) infection
PRESENTATION	Triad of thrombocytopenia (↓ platelets), microangiopathic hemolytic anemia (↓ Hb, schistocytes, ↑ LDH), acute kidney injury (↑ Cr)	
DIFFERENTIATING SYMPTOMS	Triad + fever + neurologic symptoms	Triad + bloody diarrhea
LABS	Normal PT and PTT helps distinguish TTP and HUS (coagulation pathway is not activated) from DIC (coagulation pathway is activated)	
TREATMENT	Plasmapheresis, steroids, rituximab	Supportive care

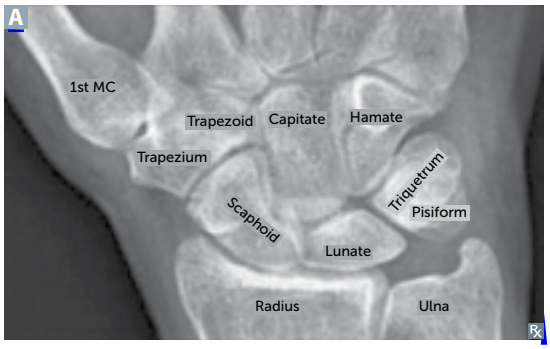
Wrist region



Scaphoid, Lunate, Triquetrum, Pisiform, Hamate, Capitate, Trapezoid, Trapezium A. (So Long To Pinky, Here Comes The Thumb)

Scaphoid (palpable in anatomic snuff box B) is the most commonly fractured carpal bone, typically due to a fall on an outstretched hand. Complications of proximal scaphoid fractures include avascular necrosis and nonunion due to retrograde blood supply from a branch of the radial artery. Fracture not always seen on initial x-ray.

Dislocation of lunate may cause acute carpal tunnel syndrome.



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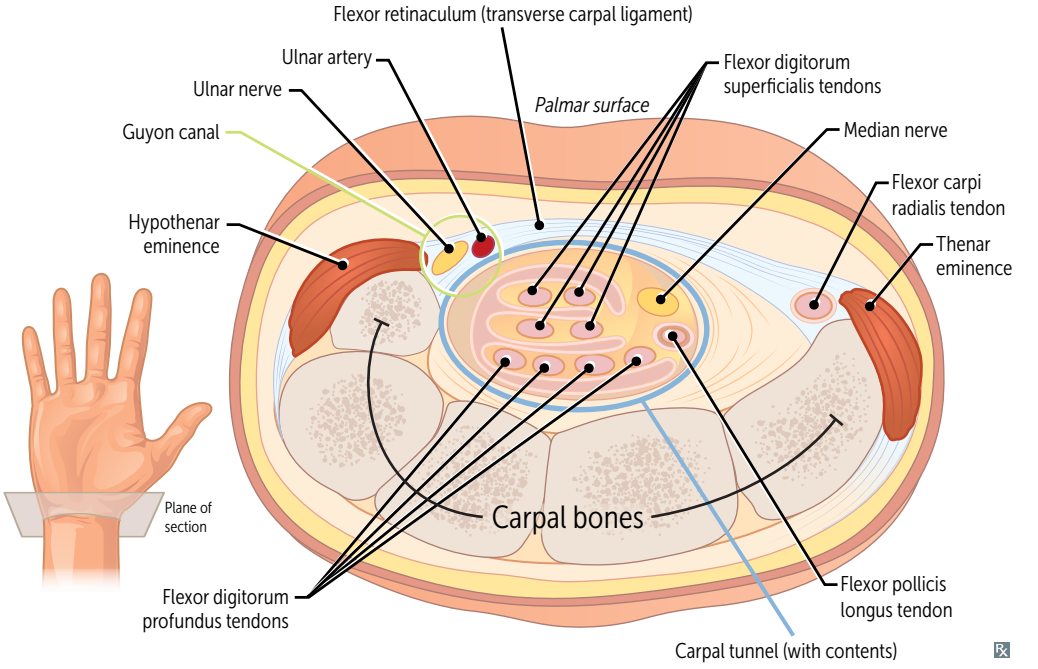

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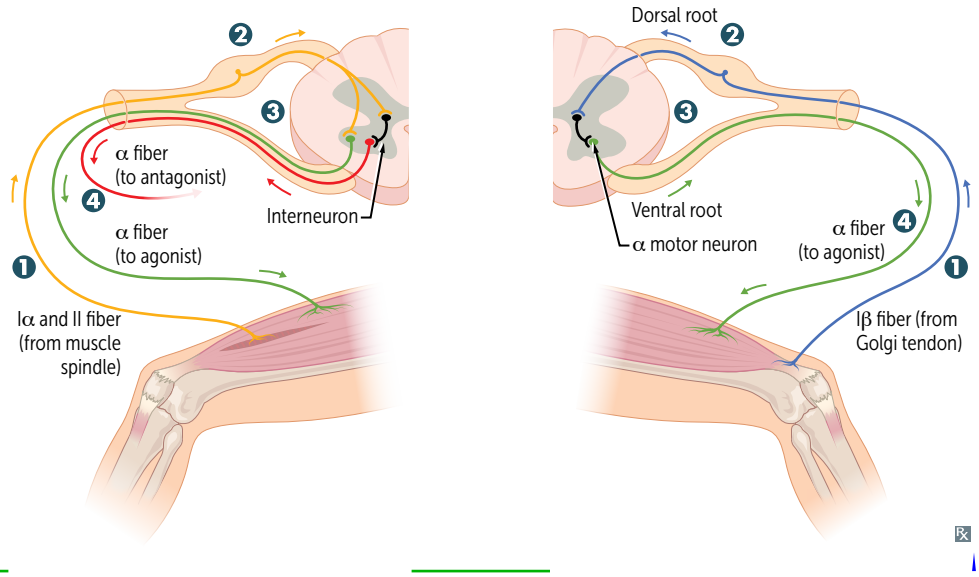
Muscle proprioceptors

Specialized sensory receptors that relay information about muscle dynamics.

	Muscle spindle	Golgi tendon organ
PATHWAY	❶ ↑ length and speed of stretch → ❷ via dorsal root ganglion (DRG) → ❸ activation of inhibitory interneuron and α motor neuron → ❹ simultaneous inhibition of antagonist muscle (prevents overstretching) and activation of agonist muscle (contraction)	❶ ↑ tension → ❷ via DRG → ❸ activation of inhibitory interneuron → ❹ inhibition of agonist muscle (reduced tension within muscle and tendon)
LOCATION	Body of muscle/type Ia and II sensory axons	Tendons/type Ib sensory axons
ACTIVATION BY	↑ muscle stretch	↑ muscle force



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Bone formation

Endochondral ossification	Bones of axial skeleton, appendicular skeleton, and base of skull. Cartilaginous model of bone is first made by chondrocytes. Osteoclasts and osteoblasts later replace with woven bone and then remodel to lamellar bone. In adults, woven bone occurs after fractures and in Paget disease. Defective in achondroplasia.
Membranous ossification	Bones of calvarium, facial bones, and clavicle. Woven bone formed directly without cartilage. Later remodeled to lamellar bone.

Epilepsy therapy *(continued)*

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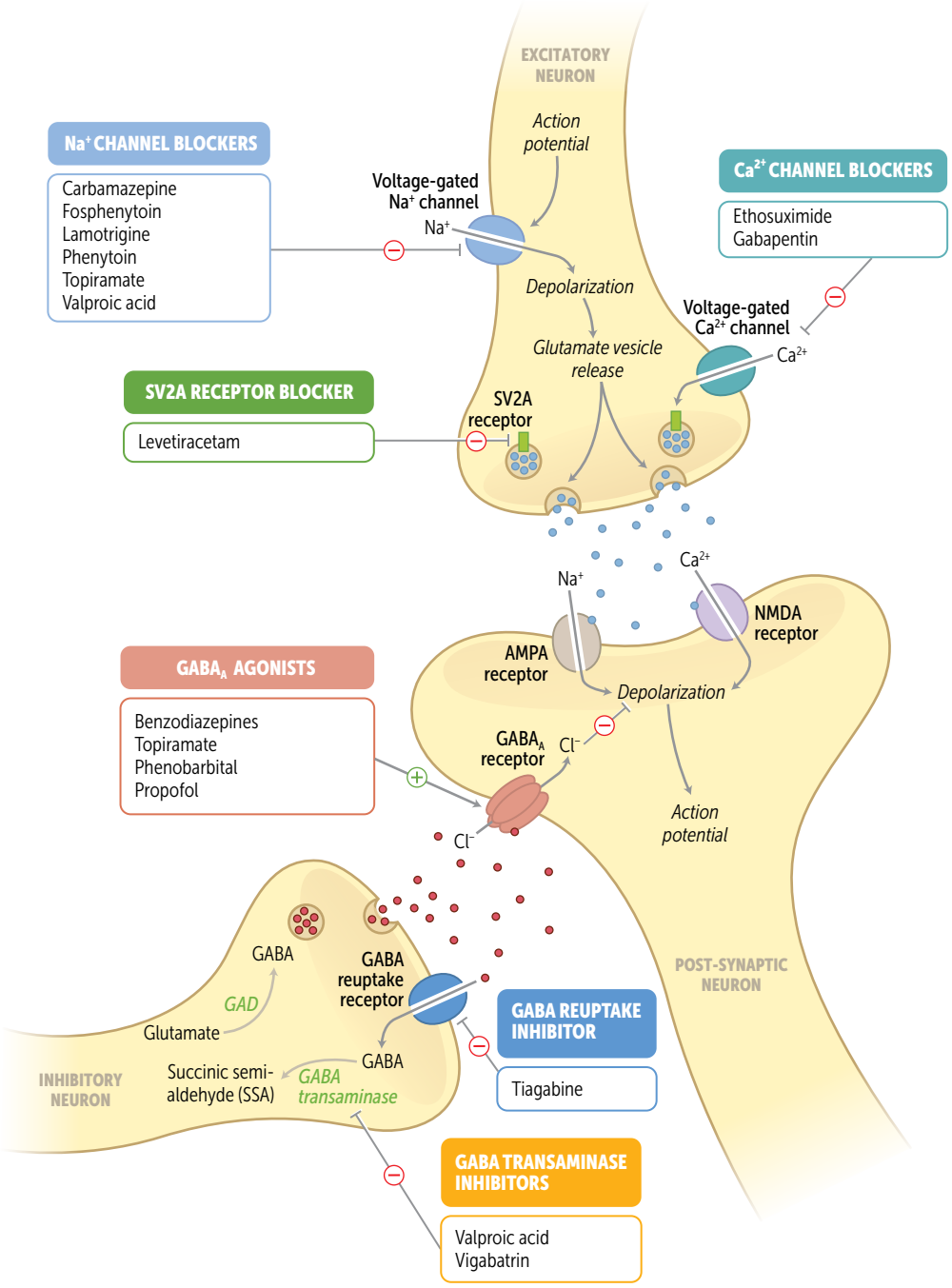
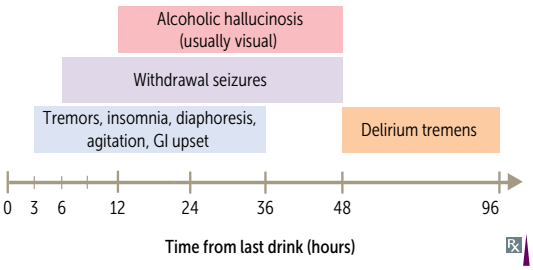


Psychoactive drug intoxication and withdrawal

DRUG	INTOXICATION	WITHDRAWAL
Depressants		
	Nonspecific: mood elevation, ↓ anxiety, sedation, behavioral disinhibition, respiratory depression.	Nonspecific: anxiety, tremor, seizures, insomnia.
Alcohol	Emotional lability, slurred speech, ataxia, coma, blackouts. Serum γ-glutamyltransferase (GGT)—sensitive indicator of alcohol use. AST value is 2× ALT value ("ToAST 2 ALcohol"). Treatment: benzodiazepines.	
Barbiturates	Low safety margin, marked respiratory depression. Treatment: symptom management (eg, assist respiration, ↑ BP).	Delirium, life-threatening cardiovascular collapse.
Benzodiazepines	Greater safety margin. Ataxia, minor respiratory depression. Treatment: flumazenil (benzodiazepine receptor antagonist, but rarely used as it can precipitate seizures).	Sleep disturbance, depression.
Opioids	Euphoria, respiratory and CNS depression, ↓ gag reflex, pupillary constriction (pinpoint pupils), seizures. Most common cause of drug overdose death. Treatment: naloxone.	Sweating, dilated pupils, piloerection ("cold turkey"), rhinorrhea, lacrimation, yawning, nausea, stomach cramps, diarrhea ("flu-like" symptoms). Treatment: symptom management, methadone, buprenorphine.
Inhalants	Disinhibition, euphoria, slurred speech, disturbed gait, disorientation, drowsiness.	Irritability, dysphoria, sleep disturbance, headache.
Stimulants		
	Nonspecific: mood elevation, ↓ appetite, psychomotor agitation, insomnia, cardiac arrhythmias, tachycardia, anxiety.	Nonspecific: post-use "crash," including depression, lethargy, ↑ appetite, sleep disturbance, vivid nightmares.
Amphetamines	Euphoria, grandiosity, pupillary dilation, prolonged wakefulness, hyperalertness, hypertension, paranoia, fever, fractured teeth. Skin excoriations with methamphetamine use. Severe: cardiac arrest, seizures. Treatment: benzodiazepines for agitation and seizures.	
Caffeine	Palpitation, agitation, tremor, insomnia.	Headache, difficulty concentrating, flu-like symptoms.

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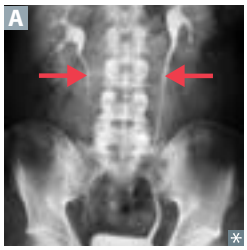
Course of ureters



Course of ureter A: arises from renal pelvis, travels under gonadal arteries → over common iliac artery → under uterine artery/vas deferens (retroperitoneal).

Gynecologic procedures (eg, ligation of uterine or ovarian vessels) may damage ureter → ureteral obstruction or leak.

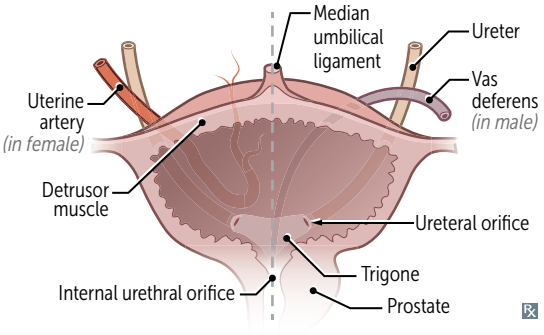
Bladder contraction compresses the intravesical ureter, preventing urine reflux.

Blood supply to ureter:

- Proximal—renal arteries
- Middle—gonadal artery, aorta, common and internal iliac arteries
- Distal—internal iliac and superior vesical arteries

3 common points of ureteral obstruction: ureteropelvic junction, pelvic inlet, ureterovesical junction.

Water (ureters) flows over the iliacs and under the bridge (uterine artery or vas deferens).



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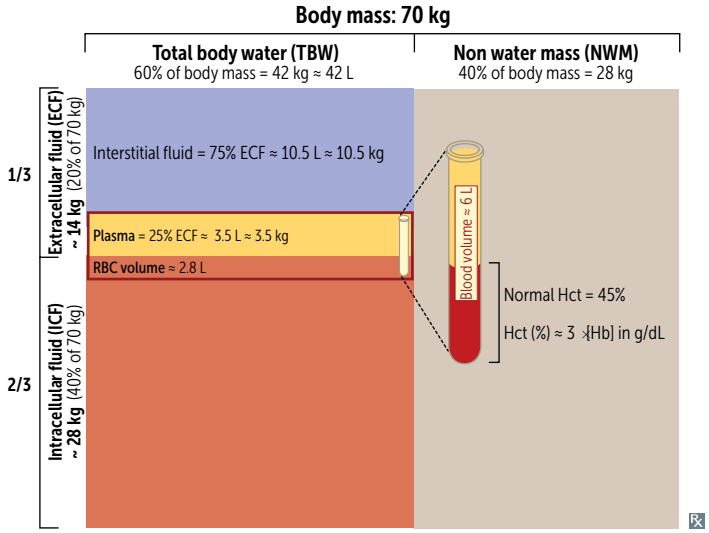
▸ RENAL—PHYSIOLOGY

Fluid compartments



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HIKIN': HIgh K^+ INtracellularly.

60–40–20 rule (% of body weight for average person):

- 60% total body water
- 40% ICF, mainly composed of K^+, Mg^{2+}, organic phosphates (eg, ATP)
- 20% ECF, mainly composed of Na^+, Cl^-, HCO_3^-, albumin

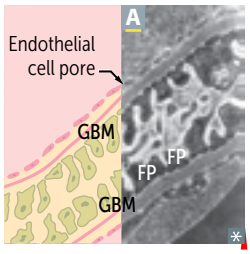
Plasma volume can be measured by radiolabeling albumin.

Extracellular volume can be measured by inulin or mannitol.

Serum osmolality = 285–295 mOsm/kg H_2O.

Plasma volume = TBV × (1 – Hct).

Glomerular filtration barrier



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Responsible for filtration of plasma according to size and charge selectivity.

Composed of:

- Fenestrated capillary endothelium
- Basement membrane with type IV collagen chains and heparan sulfate
- Visceral epithelial layer consisting of podocyte foot processes (FPs) A

Charge barrier—all 3 layers contain ⊖ charged glycoproteins that prevent entry of ⊖ charged molecules (eg, albumin).

Size barrier—fenestrated capillary endothelium (prevents entry of > 100 nm molecules/blood cells); podocyte foot processes interpose with glomerular basement membrane (GBM); slit diaphragm (prevents entry of molecules > 50–60 nm).

Glomerular diseases

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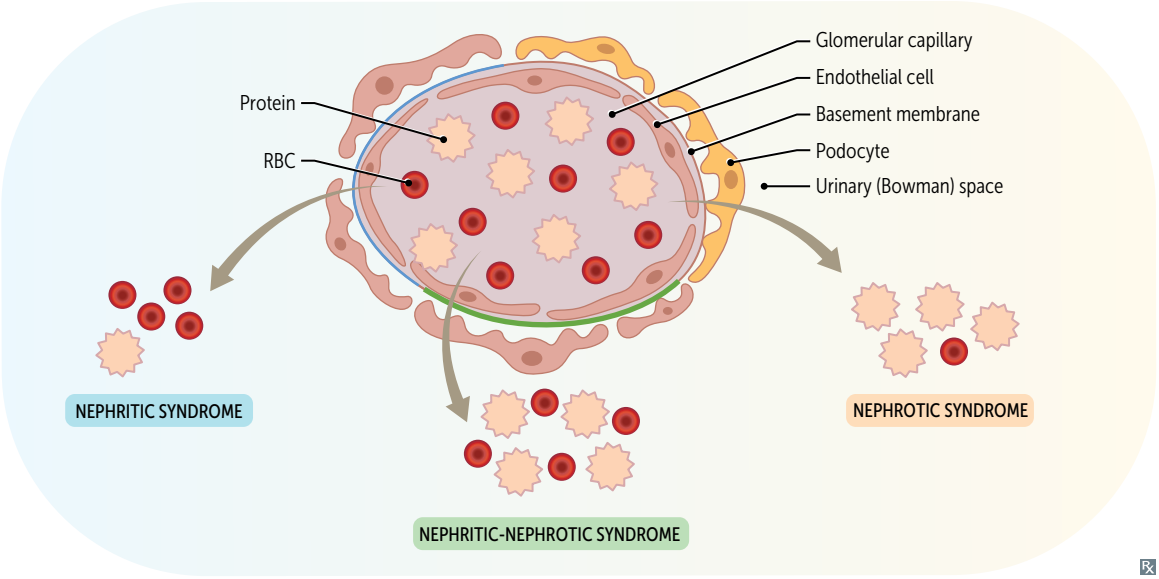

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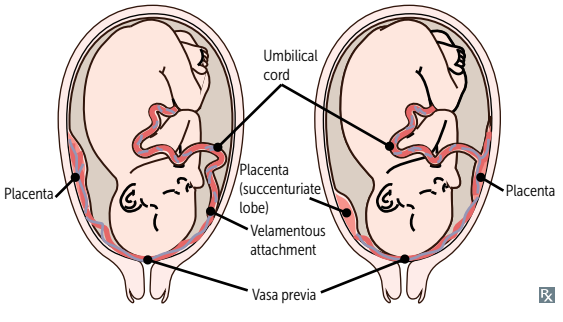
TYPE	ETIOLOGY	CLINICAL PRESENTATION	EXAMPLES
Nephritic syndrome	Glomerular inflammation → GBM damage → loss of RBCs into urine → hematuria	Hematuria, RBC casts in urine ↓ GFR → oliguria, azotemia, ↑ renin release, HTN Proteinuria often in the subnephrotic range (< 3.5 g/day) but in severe cases may be in nephrotic range	▪ Acute poststreptococcal glomerulonephritis ▪ Rapidly progressive glomerulonephritis ▪ IgA nephropathy (Berger disease) ▪ Alport syndrome ▪ Membranoproliferative glomerulonephritis
Nephrotic syndrome	Podocyte damage → impaired charge barrier → proteinuria	Massive proteinuria (> 3.5 g/day) with hypoalbuminemia, edema Frothy urine with fatty casts Associated with hypercoagulable state due to antithrombin III loss in urine and ↑ risk of infection (loss of IgGs in urine and soft tissue compromise by edema)	May be 1° (eg, direct podocyte damage) or 2° (podocyte damage from systemic process): ▪ Focal segmental glomerulosclerosis (1° or 2°) ▪ Minimal change disease (1° or 2°) ▪ Membranous nephropathy (1° or 2°) ▪ Amyloidosis (2°) ▪ Diabetic glomerulonephropathy (2°)
Nephritic-nephrotic syndrome	Severe GBM damage → loss of RBCs into urine + impaired charge barrier → hematuria + proteinuria	Nephrotic-range proteinuria (> 3.5 g/day) and concomitant features of nephrotic syndrome	Can occur with any form of nephritic syndrome, but is most common with: ▪ Diffuse proliferative glomerulonephritis ▪ Membranoproliferative glomerulonephritis

Pregnancy complications *(continued)*

Vasa previa

Fetal vessels run over, or in close proximity to, cervical os. May result in vessel rupture, exsanguination, fetal death. Presents with triad of membrane rupture, painless vaginal bleeding, fetal bradycardia (< 110 beats/min). Emergency C-section usually indicated. Frequently associated with velamentous umbilical cord insertion (cord inserts in chorioamniotic membrane rather than placenta → fetal vessels travel to placenta unprotected by Wharton jelly).



Postpartum hemorrhage

Due to 4 T's: Tone (uterine atony; most common), Trauma (lacerations, incisions, uterine rupture), Thrombin (coagulopathy), Tissue (retained products of conception).

Treatment: uterine massage, oxytocin. If refractory, surgical ligation of uterine or internal iliac artery (will preserve fertility since ovarian arteries provide collateral circulation).

Ectopic pregnancy

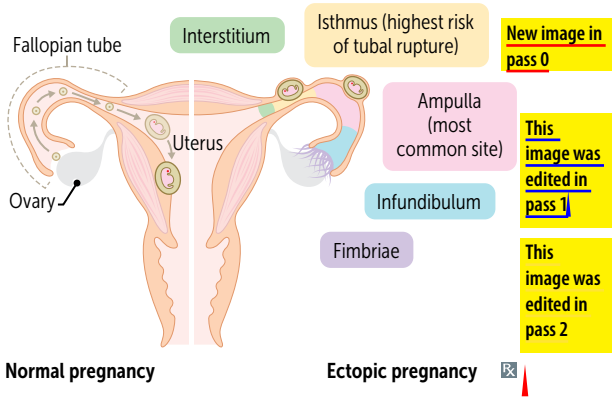
art was revised for 2020

Implantation of fertilized ovum in a site other than the uterus, most often in ampulla of fallopian tube A. Suspect with history of amenorrhea, lower-than-expected rise in hCG based on dates, and sudden lower abdominal pain; confirm with ultrasound, which may show extraovarian adnexal mass. Often clinically mistaken for appendicitis.

Pain +/– bleeding.

Risk factors:

- Prior ectopic pregnancy
- History of infertility
- Salpingitis (PID)
- Ruptured appendix
- Prior tubal surgery
- Smoking
- Advanced maternal age



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Amniotic fluid abnormalities

Polyhydramnios

Too much amniotic fluid. Often idiopathic, but associated with fetal malformations (eg, esophageal/duodenal atresia, anencephaly; both result in inability to swallow amniotic fluid), maternal diabetes, fetal anemia, multiple gestations.

Oligohydramnios

Too little amniotic fluid. Associated with placental insufficiency, bilateral renal agenesis, posterior urethral valves (in males) and resultant inability to excrete urine. Any profound oligohydramnios can cause Potter sequence.

Ovarian cysts

Follicular cyst	Distention of unruptured Graafian follicle. May be associated with hyperestrogenism, endometrial hyperplasia. Most common ovarian mass in young women.
Theca-lutein cyst	Often bilateral/multiple. Due to gonadotropin stimulation. Associated with choriocarcinoma and hydatidiform moles.

Ovarian neoplasms

Most common adnexal mass in women >55 years old. Present with abdominal distention, bowel obstruction, pleural effusion.

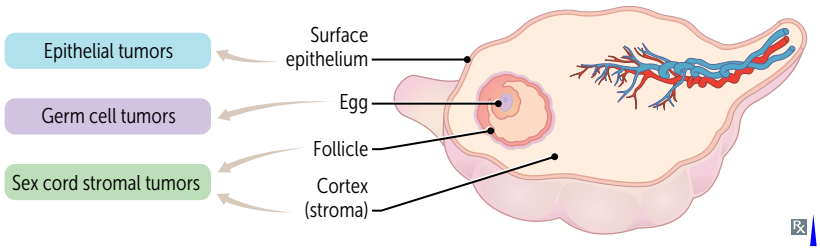
Risk ↑ with advanced age, infertility, endometriosis, PCOS, genetic predisposition (eg, *BRCA1* or *BRCA2* mutations, Lynch syndrome, strong family history).

Risk ↓ with previous pregnancy, history of breastfeeding, OCPs, tubal ligation.

Epithelial tumors are typically serous (lined by serous epithelium natively found in fallopian tubes, and often bilateral) or mucinous (lined by mucinous epithelium natively found in cervix). Monitor response to therapy/relapse by measuring CA 125 levels (not good for screening).

Germ cell tumors can differentiate into somatic structures (eg, teratomas), or extra-embryonic structures (eg, yolk sac tumors), or can remain undifferentiated (eg, dysgerminoma).

Sex cord stromal tumors develop from embryonic sex cord (develops into theca and granulosa cells of follicle, Sertoli and Leydig cells of seminiferous tubules) and stromal (ovarian cortex) derivatives.



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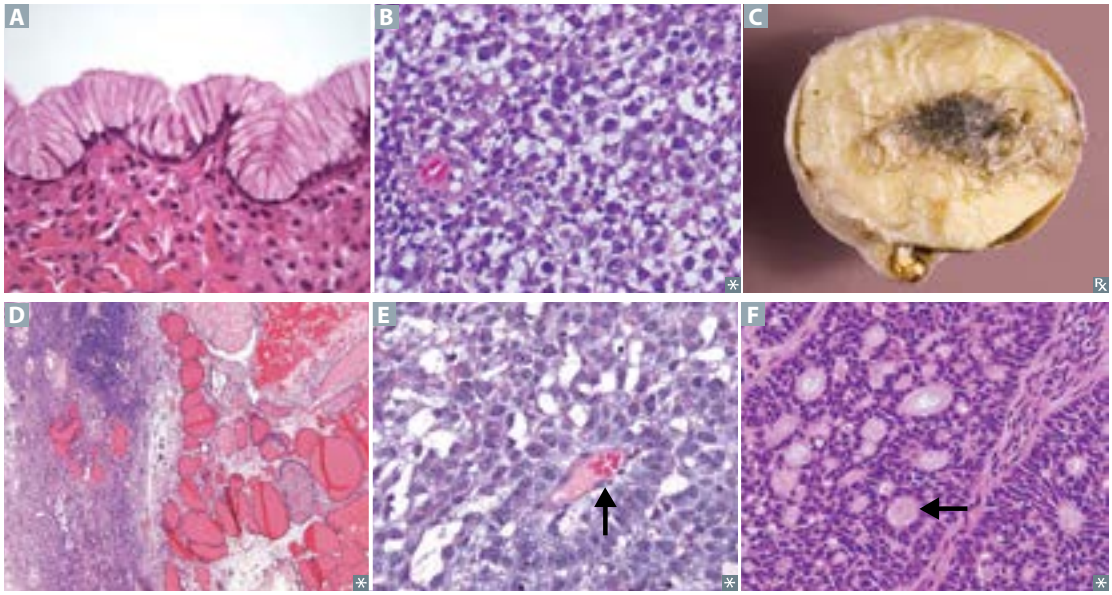
TYPE	MALIGNANT?	CHARACTERISTICS
Epithelial tumors		
Serous cystadenoma	Benign	Most common ovarian neoplasm.
Serous cystadenocarcinoma	Malignant	Most common malignant ovarian neoplasm. Psammoma bodies.
Mucinous cystadenoma	Benign	Multiloculated, large. Lined by mucus-secreting epithelium A.
Mucinous cystadenocarcinoma	Malignant	Rare. May be metastatic from appendiceal or GI tumors. Can result in pseudomyxoma peritonei (intraperitoneal accumulation of mucinous material).
Brenner tumor	Usually benign	Solid, pale yellow-tan tumor that appears encapsulated. "Coffee bean" nuclei on H&E stain.

Indents were edited in pass 3

Ovarian neoplasms *(continued)*

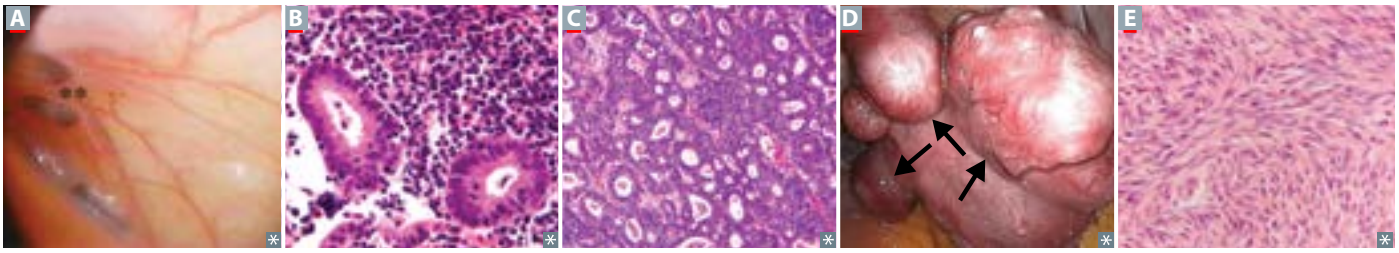
Germ cell tumors		
Dysgerminoma	Malignant	Most common in adolescents. Equivalent to male seminoma but rarer. Sheets of uniform "fried egg" cells B. Tumor markers: ↑ hCG, LDH.
Mature cystic teratoma	Benign	Also called dermoid cyst. Most common ovarian tumor in young females. Cystic mass with elements from all 3 germ layers (eg, teeth, hair, sebum) C. May be painful 2° to ovarian enlargement or torsion. Monodermal form with thyroid tissue (struma ovarii D) may present with hyperthyroidism. Malignant transformation rare (usually to squamous cell carcinoma).
Immature teratoma	Malignant, aggressive	Contains fetal tissue, neuroectoderm. Commonly diagnosed before age 20. Typically represented by immature/embryonic-like neural tissue.
Yolk sac (endodermal sinus) tumor	Malignant, aggressive	Occur in ovaries and sacrococcygeal area in children. Yellow, friable (hemorrhagic) mass. 50% have Schiller-Duval bodies (resemble glomeruli, arrow in E). Tumor marker: ↑ AFP.
Sex cord stromal tumors		
Thecoma	Benign	May produce estrogen. Usually presents as abnormal uterine bleeding in a postmenopausal woman.
Granulosa cell tumor	Malignant	Most common malignant sex cord stromal tumor. Predominantly women in their 50s. Often produces estrogen and/or progesterone and presents with postmenopausal bleeding, endometrial hyperplasia, sexual precocity (in pre-adolescents), breast tenderness. Histology shows Call-Exner bodies (granulosa cells arranged haphazardly around collections of eosinophilic fluid, resembling primordial follicles; arrow in F). "Give Granny a Call!"
Sertoli-Leydig cell tumor	Benign	Small, grey to yellow-brown mass. Resembles testicular histology with tubules/cords lined by pink Sertoli cells. May produce androgens → virilization (eg, hirsutism, male pattern baldness, clitoral enlargement).
Fibromas	Benign	Bundles of spindle-shaped fibroblasts. Meigs syndrome—triad of ovarian fibroma, ascites, pleural effusion. "Pulling" sensation in groin.

B, C, D art was revised for 2020



Uterine conditions

Non-neoplastic uterine conditions	
Adenomyosis	Extension of endometrial tissue (glandular) into uterine myometrium. Caused by hyperplasia of basal layer of endometrium. Presents with dysmenorrhea, AUB/HMB, and uniformly enlarged, soft, globular uterus. Treatment: GnRH agonists, hysterectomy, excision of an organized adenomyoma.
Asherman syndrome	Adhesions and/or fibrosis of the endometrium. Presents with ↓ fertility, recurrent pregnancy loss, AUB, pelvic pain. Often associated with dilation and curettage of intrauterine cavity.
Endometrial hyperplasia	Abnormal endometrial gland proliferation usually stimulated by excess estrogen. ↑ risk for endometrial carcinoma (especially with nuclear atypia). Presents as postmenopausal vaginal bleeding. ↑ risk with anovulatory cycles, hormone replacement therapy, PCOS, granulosa cell tumors.
Endometriosis	Endometrium-like glands/stroma outside endometrial cavity, most commonly in the ovary (frequently bilateral), pelvis, peritoneum (yellow-brown "powder burn" lesions). In ovary, appears as endometrioma (blood-filled "chocolate cysts" [oval structures above and below asterisks in A]). May be due to retrograde flow, metaplastic transformation of multipotent cells, transportation of endometrial tissue via lymphatic system. Characterized by cyclic pelvic pain, bleeding, dysmenorrhea, dyspareunia, dyschezia (pain with defecation), infertility; normal-sized uterus. Treatment: NSAIDs, OCPs, progestins, GnRH agonists, danazol, laparoscopic removal.
Endometritis	Inflammation of endometrium B associated with retained products of conception following delivery, miscarriage, abortion, or with foreign body (eg, IUD). Retained material is nidus for bacteria from vagina or GI tract. Chronic endometritis shows plasma cells on histology. Treatment: gentamicin + clindamycin +/– ampicillin.
Uterine neoplasms	
Endometrial carcinoma	Most common gynecologic malignancy C. Presents with irregular vaginal bleeding. Two types: Endometrioid—most cases caused by unopposed estrogen exposure due to obesity, but also associated with early menarche, late menopause, nulliparity. Histology shows abnormally arranged endometrial glands. Early pathogenic events include loss of PTEN or mismatch repair proteins. Serous—associated with endometrial atrophy in postmenopausal women. Aggressive. Psammoma bodies often seen on histology. Characterized by formation of papillae and tufts.
Leiomyoma (fibroid)	Most common tumor in females. Often presents with multiple discrete tumors D. ↑ incidence in African Americans. Benign smooth muscle tumor; malignant transformation to leiomyosarcoma is rare. Estrogen sensitive; tumor size ↑ with pregnancy and ↓ with menopause. Peak occurrence at 20-40 years of age. May be asymptomatic, cause AUB, or result in miscarriage. Severe bleeding may lead to iron deficiency anemia. Whorled pattern of smooth muscle bundles with well-demarcated borders on histology E.
Leiomyosarcoma	Malignant proliferation of smooth muscle arising from myometrium; arises de novo (not from leiomyomas) usually in postmenopausal women. Exam shows single lesion with areas of necrosis.



Club cells

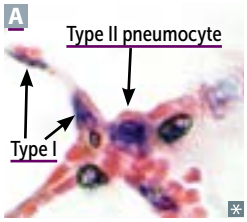
Nonciliated; low columnar/cuboidal with secretory granules. Located in bronchioles. Degrade toxins; secrete component of surfactant; act as reserve cells.

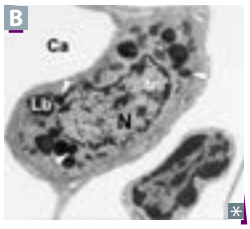
Alveolar cell types

Type I pneumocytes

Squamous. 97% of alveolar surfaces. Thinly line the alveoli (two black arrows in A) for optimal gas exchange.

Type II pneumocytes





These illustrations were switched in pass 3

Cuboidal and clustered A.

2 functions:

1. Serve as stem cell precursors for 2 cell types (type I and type II cells); proliferate during lung damage.
2. Secrete surfactant from lamellar bodies (arrowheads in B)

Surfactant— ↓ alveolar surface tension, ↓ alveolar collapse, ↓ lung recoil, and ↑ compliance.

Composed of multiple lecithins, mainly dipalmitoylphosphatidylcholine (DPPC).

Synthesis begins ~week 20 of gestation and achieves mature levels ~week 35.

Corticosteroids important for fetal surfactant synthesis and lung development.

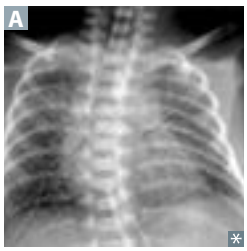
$$\text{Collapsing pressure } (P) = \frac{2\,(\text{surface tension})}{\text{radius}}$$

Law of Laplace—Alveoli have ↑ tendency to collapse on expiration as radius ↓.

Alveolar macrophages

Phagocytose foreign materials; release cytokines and alveolar proteases. Hemosiderin-laden macrophages ("HF cells") may be found in the setting of pulmonary edema or alveolar hemorrhage.

Neonatal respiratory distress syndrome



Surfactant deficiency → ↑ surface tension → alveolar collapse ("ground-glass" appearance of lung fields) A.

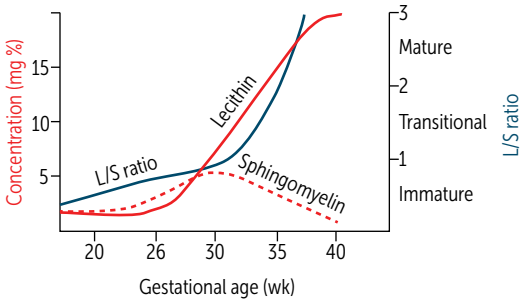
Risk factors: prematurity, maternal diabetes (due to ↑ fetal insulin), C-section delivery (↓ release of fetal glucocorticoids; less stressful than vaginal delivery).

Treatment: maternal steroids before birth; exogenous surfactant for infant.

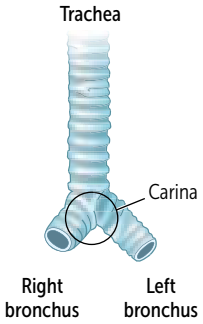
Therapeutic supplemental O_2 can result in Retinopathy of prematurity, Intraventricular hemorrhage, Bronchopulmonary dysplasia (RIB).

Screening tests for fetal lung maturity: lecithin-sphingomyelin (L/S) ratio in amniotic fluid (≥ 2 is healthy; < 1.5 predictive of NRDS), foam stability index, surfactant-albumin ratio.

Persistently low O_2 tension → risk of PDA.



Lung anatomy



Right lung has 3 lobes; **L**eft has **L**ess **L**obes (2) and **L**ingula (homolog of right middle lobe). Instead of a middle lobe, left lung has a space occupied by the heart **A**.

Relation of the pulmonary artery to the bronchus at each lung hilum is described by **RALS**—**R**ight **A**nterior; **L**eft **S**uperior. Carina is posterior to ascending aorta and anteromedial to descending aorta **B**.

Right lung is a more common site for inhaled foreign bodies because right main stem bronchus is wider, more vertical, and shorter than the left. If you aspirate a peanut:

- While supine—usually enters superior segment of right lower lobe.
- While lying on right side—usually enters right upper lobe.
- While upright—usually enters right lower lobe.

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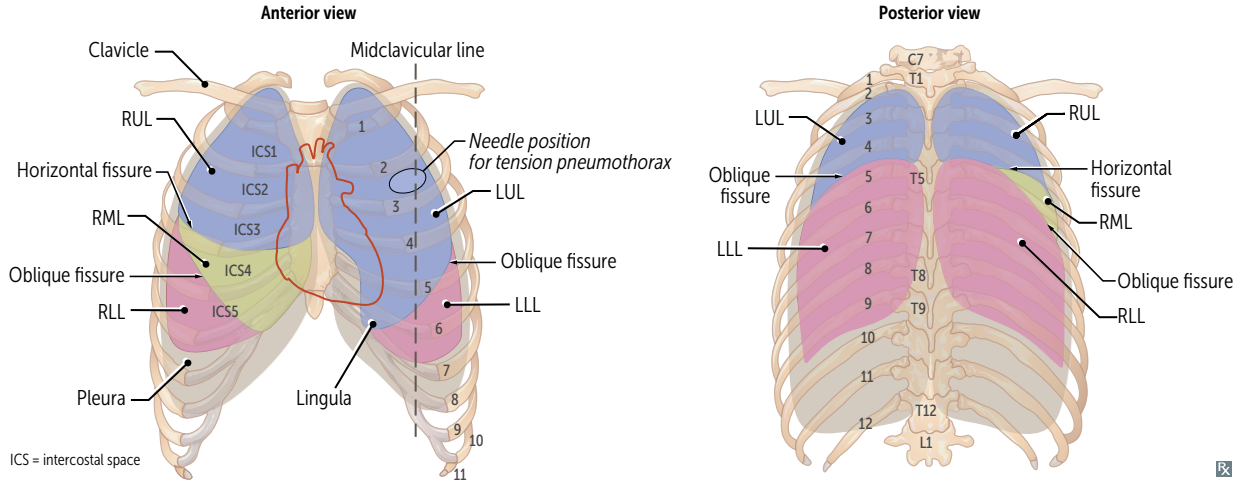
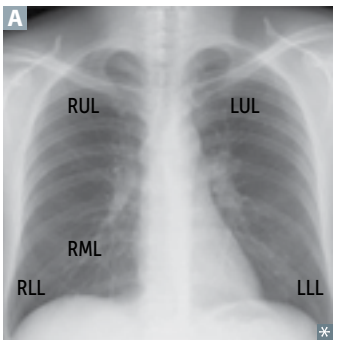
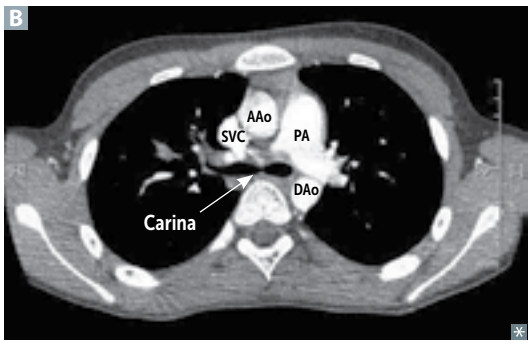
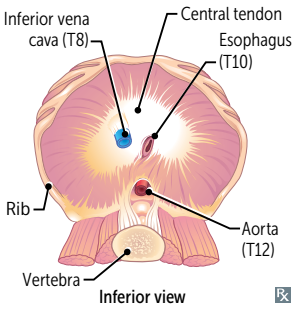






Diaphragm structures



Structures perforating diaphragm:

- At T8: IVC, right phrenic nerve
- At T10: esophagus, vagus (CN 10; 2 trunks)
- At T12: aorta (red), thoracic duct (white), azygos vein (blue) ("At T12 it's the **red**, **white**, and **blue**")

Diaphragm is innervated by C3, 4, and 5 (phrenic nerve). Pain from diaphragm irritation (eg, air, blood, or pus in peritoneal cavity) can be referred to shoulder (C5) and trapezius ridge (C3, 4).

Number of letters = T level:

T8: vena cava (IVC)
T10: (O)esophagus
T12: aortic hiatus

I ate (8) ten eggs at twelve.

C3, 4, 5 keeps the diaphragm alive.

Other bifurcations:

- The common carotid bifourcates at C4.
- The trachea bifourcates at T4.
- The abdominal aorta bifourcates at L4.

Physical findings in select lung diseases

ABNORMALITY	BREATH SOUNDS	PERCUSSION	FREMITUS	TRACHEAL DEVIATION
Pleural effusion	↓	Dull	↓	None if small Away from side of lesion if large
Atelectasis	↓	Dull	↓	Toward side of lesion
Simple pneumothorax	↓	Hyperresonant	↓	None
Tension pneumothorax	↓	Hyperresonant	↓	Away from side of lesion
Consolidation (lobar pneumonia, pulmonary edema)	Bronchial breath sounds; late inspiratory crackles, egophony, whispered pectoriloquy	Dull	↑	None

Atelectasis

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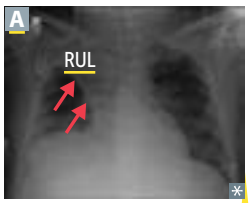
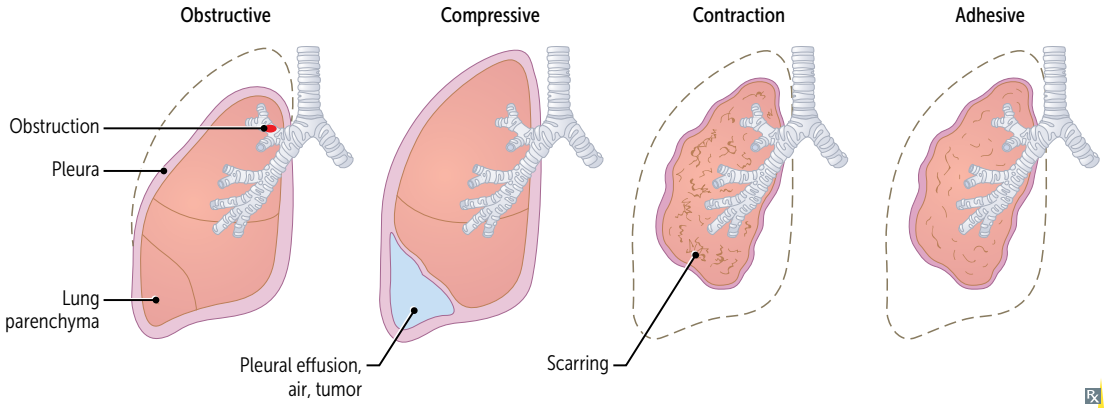
Updated image in pass 3



Alveolar collapse (right upper lobe collapse against mediastinum in A). Multiple causes:

- Obstructive—airway obstruction prevents new air from reaching distal airways, old air is resorbed (eg, foreign body, mucous plug, tumor)
- Compressive—external compression on lung decreases lung volumes (eg, space-occupying lesion, pleural effusion)
- Contraction (cicatrization)—scarring of lung parenchyma that distorts alveoli (eg, sarcoidosis)
- Adhesive—due to lack of surfactant (eg, NRDS in premature babies)



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▸ EASILY CONFUSED MEDICATIONS

DRUG	CLINICAL USE/MECHANISM OF ACTION
Amiloride	K^+-sparing diuretic
Amiodarone	Class III antiarrhythmic
Amlodipine	Dihydropyridine Ca^{2+} channel blocker
Benztropine	Cholinergic antagonist
Bromocriptine	Dopamine agonist
Buspirone	Generalized anxiety disorder (5-HT_{1A}-receptor agonist)
Bupropion	Depression, smoking cessation (NE-DA reuptake inhibitor)
Cimetidine	H_2-receptor antagonist
Cetirizine	2nd-generation antihistamine
Chloramphenicol	Antibiotic (blocks 50S subunit)
Chlordiazepoxide	Long-acting benzodiazepine
Chlorpromazine	Typical antipsychotic
Chlorpropamide	1st-generation sulfonylurea
Chlorpheniramine	1st-generation antihistamine
Chlorthalidone	Thiazide diuretic
Clozapine	5-HT_{2A}-agonist
Clomipramine	Tricyclic antidepressant
Clomiphene	Selective estrogen receptor modulator
Clonidine	α_2-agonist
Doxepin	Tricyclic antidepressant
Doxazosin	α_1-antagonist
Eplerenone	K^+-sparing diuretic
Propafenone	Class IC antiarrhythmic
Fluoxetine	Selective serotonin reuptake inhibitor
Fluphenazine	Typical antipsychotic
Duloxetine	Serotonin-norepinephrine reuptake inhibitor
Guaifenesin	Expectorant (thins respiratory secretions)
Guanfacine	α_2-agonist
Mifepristone	Progesterone receptor antagonist
Misoprostol	PGE_1 synthetic analog
Naloxone	Opioid receptor antagonist (treats toxicity)
Naltrexone	Opioid receptor antagonist (prevents relapse)
Nitroprusside	Hypertensive emergency (↑ cGMP/NO)
Nitroglycerin	Antianginal (↑ cGMP/NO)
Omeprazole	Proton pump inhibitor
Ketoconazole	Antifungal (inhibits fungal sterol synthesis)