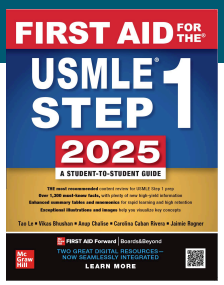
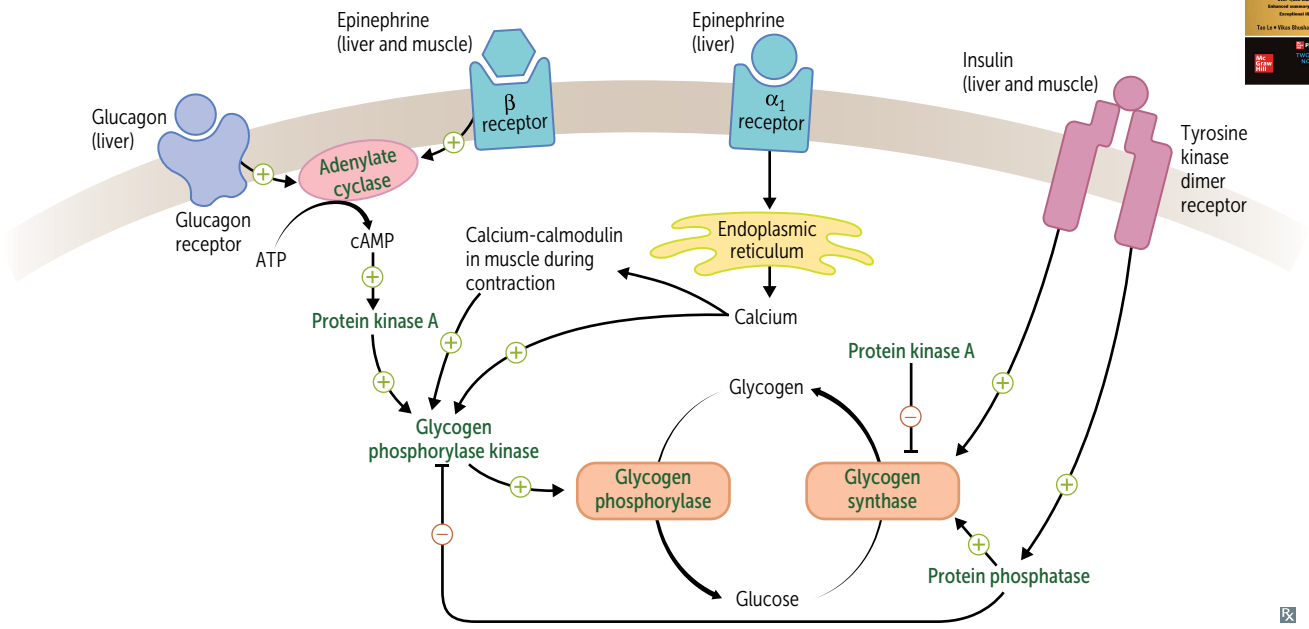
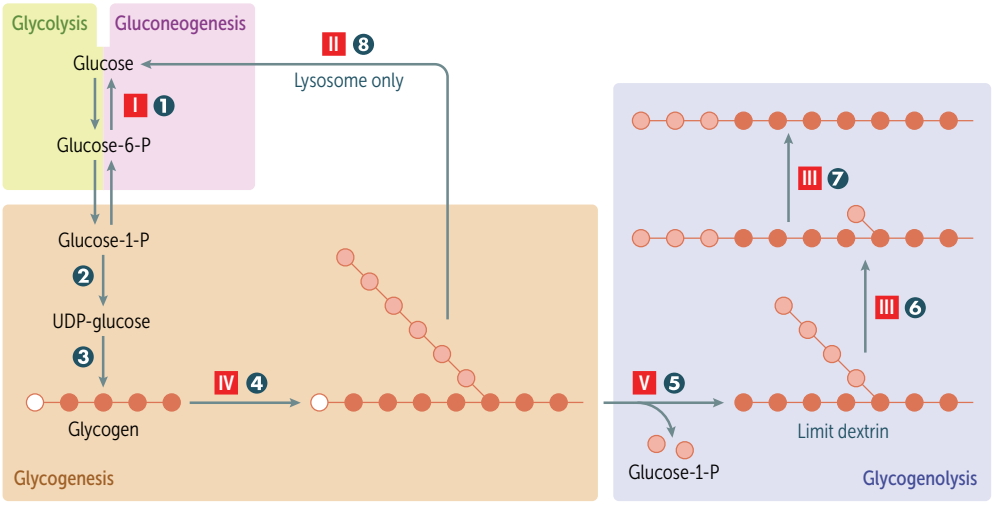


Glycogen regulation by insulin and glucagon/epinephrine



art revised for 2025 2nd pass

Glycogen	Branches have α-(1,6) bonds; linear linkages have α-(1,4) bonds.
Skeletal muscle	Glycogen undergoes glycogenolysis → glucose-1-phosphate → glucose-6-phosphate, which is rapidly metabolized during exercise.
Hepatocytes	Glycogen is stored and undergoes glycogenolysis to maintain blood sugar at appropriate levels. Glycogen phosphorylase ❺ liberates glucose-1-phosphate residues off branched glycogen until 4 glucose units remain on a branch. Then 4-α-D-glucanotransferase (debranching enzyme ❻) moves 3 of the 4 glucose units from the branch to the linear linkage. Then α-1,6-glucosidase (debranching enzyme ❼) cleaves off the last residue, liberating a free glucose. Limit dextrin—2–4 residues remaining on a branch after glycogen phosphorylase has shortened it.



Glycogen storage disease type

- I Von Gierke disease
- II Pompe disease
- III Cori disease
- IV Anderson disease
- V McArdle disease

Glycogen enzymes

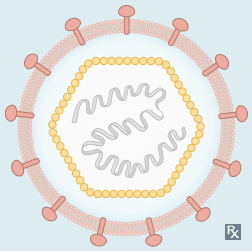
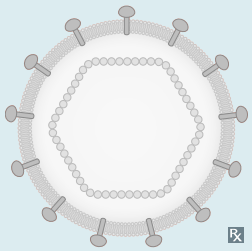
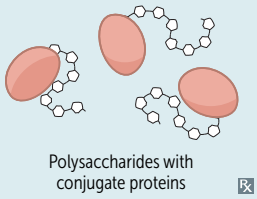
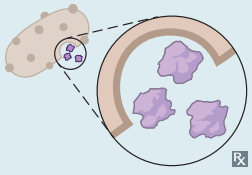
1. Glucose-6-phosphatase
2. UDP-glucose pyrophosphorylase
3. Glycogen synthase
4. Branching enzyme
5. Glycogen phosphorylase
6. Debranching enzyme (4-α-D-glucanotransferase)
7. Debranching enzyme (α-1,6-glucosidase)
8. α-1,4-glucosidase

new image for 2025 3rd pass

Note: A small amount of glycogen is degraded in lysosomes by ❽ α-1,4-glucosidase (acid maltase).

Vaccination

Induces an active immune response (humoral and/or cellular) to specific pathogens.

VACCINE TYPE	DESCRIPTION	PROS/CONS	EXAMPLES
Live attenuated vaccine 	Microorganism rendered nonpathogenic but retains capacity for transient growth within inoculated host. Certain live vaccines (MMR, varicella) may be given to people living with HIV who have a CD4+ cell count ≥ 200 cells/mm^3 in consultation with a specialist in infectious disease or immunology.	Pros: induces cellular and humoral responses. Induces strong, often lifelong immunity. Cons: may revert to virulent form. Contraindicated in pregnancy and patients with immunodeficiency.	Adenovirus (nonattenuated, given to military recruits), typhoid (Ty21a, oral), polio (Sabin), varicella (chickenpox), smallpox, BCG, yellow fever, influenza (intranasal), MMR, rotavirus. "Attention teachers! Please vaccinate small, Beautiful young infants with MMR routinely!"
Killed or inactivated vaccine 	Pathogen is inactivated by heat or chemicals. Maintaining epitope structure on surface antigens is important for immune response. Mainly induces a humoral response.	Pros: safer than live vaccines. Cons: weaker cell-mediated immune response; mainly induces a humoral response. Booster shots usually needed.	Hepatitis A, Typhoid (Vi polysaccharide, intramuscular), Rabies, Influenza (intramuscular), Polio (SalK). A TRIP could Kill you.
Subunit, recombinant, polysaccharide, and conjugate 	All use specific antigens that best stimulate the immune system.	Pros: targets specific epitopes of antigen; lower chance of adverse reactions. Cons: expensive; weaker immune response.	HBV (antigen = HBsAg), HPV, acellular pertussis (aP), *Neisseria meningitidis* (various strains), *Streptococcus pneumoniae* (PPSV23 polysaccharide primarily T-cell–independent response; PCV13, PCV15, and PCV20 polysaccharide produces T-cell–dependent response), Hib, herpes zoster.
Toxoid 	Denatured bacterial toxin with an intact receptor binding site. Stimulates immune system to make antibodies without potential for causing disease.	Pros: protects against the bacterial toxins. Cons: antitoxin levels decrease with time, thus booster shots may be needed.	*Clostridium tetani*, *Corynebacterium diphtheriae*.
mRNA 	A lipid nanoparticle delivers mRNA, causing cells to synthesize foreign protein (eg, spike protein of SARS-CoV-2).	Pros: high efficacy; induces cellular and humoral immunity. Safe in pregnancy. Cons: local and transient systemic (fatigue, headache, myalgia) reactions are common. Rare myocarditis, pericarditis particularly in young males.	SARS-CoV-2.

Spore-forming bacteria

Some gram ⊕ bacteria can form spores when nutrients are limited. Spores lack metabolic activity and are highly resistant to heat and chemicals. Core contains dipicolinic acid (responsible for heat resistance). Must autoclave to kill spores (as is done to surgical equipment) by steaming at 121°C for 15 minutes at a pressure of 15 psi. Hydrogen peroxide and iodine-based agents are also sporicidal.

Examples: *B anthracis* (anthrax), *B cereus* (food poisoning), *Clostridium botulinum* (botulism), *Clostridioides difficile* (pseudomembranous colitis), *Clostridium perfringens* (gas gangrene), *Clostridium tetani* (tetanus).

Autoclave to kill Bacillus and Clostridium (ABC).

Bacterial virulence factors

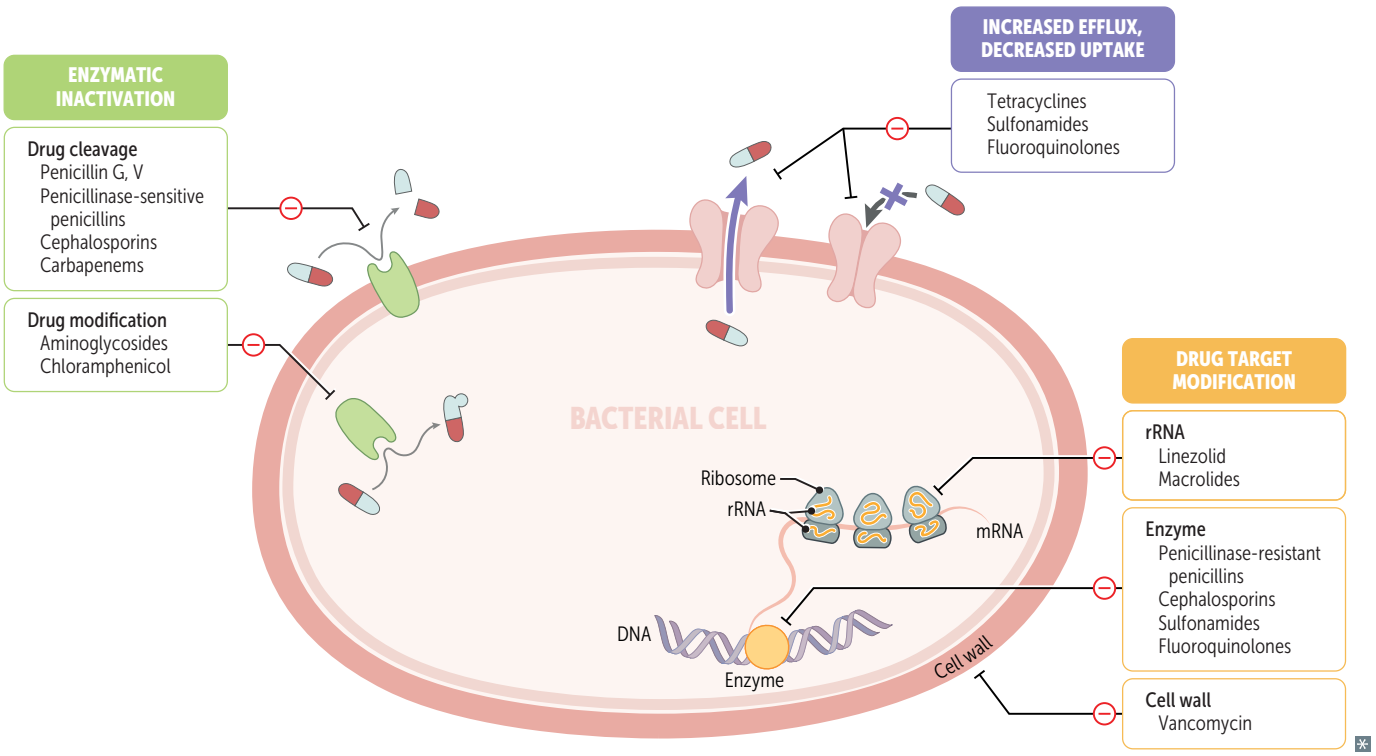
These promote evasion of host immune response.

Capsular polysaccharide	Highly charged, hydrophilic structure. Acts as barrier to phagocytosis and complement-mediated lysis. Major determinant of virulence.
Protein A	Binds Fc region of IgG. Prevents opsonization and phagocytosis. Expressed by S *aureus*.
IgA protease	Enzyme that cleaves IgA, allowing bacteria to adhere to and colonize mucous membranes. Secreted by *S pneumoniae*, *H influenzae* type b, and *Neisseria* (SHiN).
M protein	Helps prevent phagocytosis. Expressed by group A streptococci. Sequence homology with human cardiac myosin (molecular mimicry); possibly underlies the autoimmune response seen in acute rheumatic fever.

New fact for 2025 3rd pass

Antibiotic resistance mechanisms

new image for 2025 3rd pass



Acetylcholine receptors

Nicotinic ACh receptors are ligand-gated channels allowing efflux of K^+ and influx of Na^+ and in some cases Ca^{2+}. Two subtypes: N_N (found in autonomic ganglia, adrenal medulla) and N_M (found in neuromuscular junction of skeletal muscle).

Muscarinic ACh receptors are G-protein–coupled receptors that usually act through 2nd messengers. 5 subtypes: M_{1-5} found in heart, smooth muscle, brain, exocrine glands, and on sweat glands (cholinergic sympathetic).

Pain transmission

New fact for 2025 3rd pass

Nociceptive pain

Pain signals transmitted to the CNS in response to mechanical, thermal, or chemical stimuli. Transient receptor potential vanilloid ligand receptors cause Ca^{2+} influx–induced Na^+ channel activation. Signals transmitted by Aδ fibers (sharp, acute pain) or C fibers (dull, throbbing, chronic pain).

Processes involved in pain transmission:

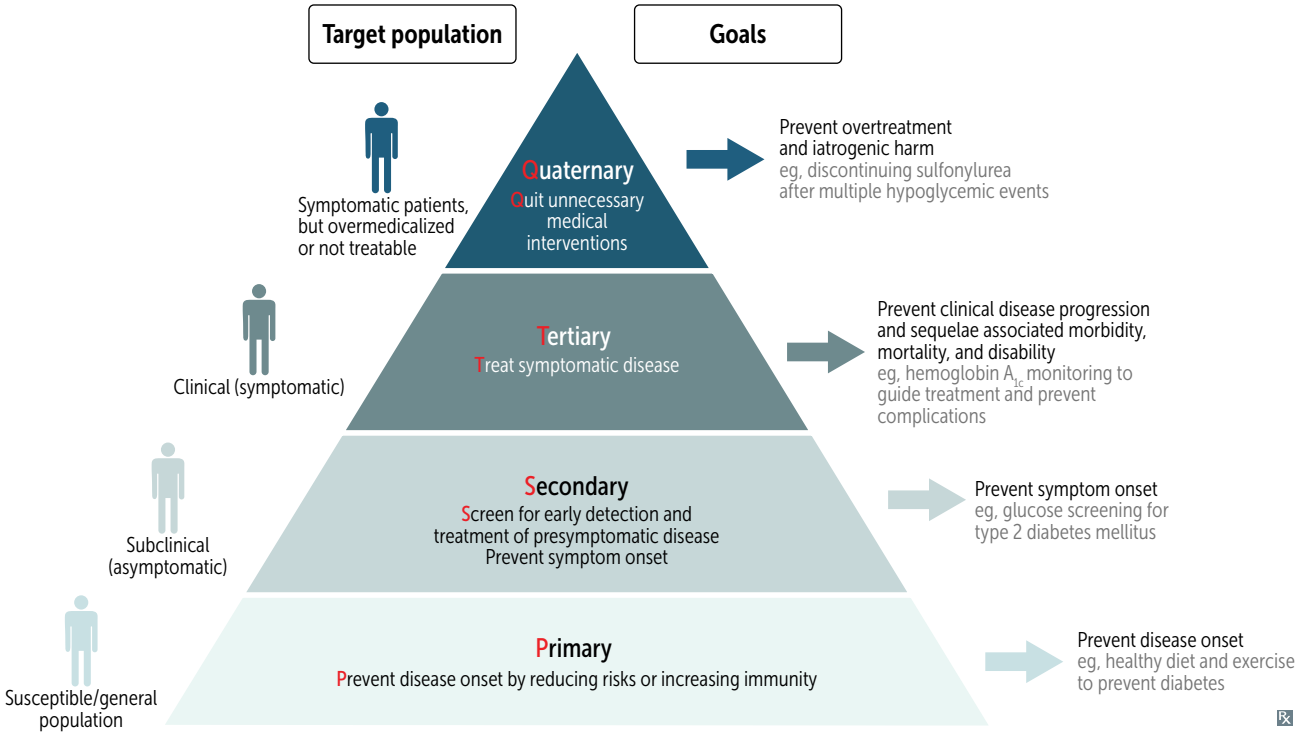
- Transduction—blocked by local anesthetics, α_2-agonists, gabapentinoids, NSAIDs, acetaminophen, glucocorticoids
- Transmission—blocked by local anesthetics, α_2-agonists, opioids
- Modulation—blocked by TCAs, SSRIs, SNRIs, gabapentinoids
- Perception—blocked by α_2-agonists, opioids, TCAs, SSRIs, SNRIs

Neuropathic pain

Caused by neuronal dysfunction of the CNS or PNS. Transmitted via upregulation and persistent activation of voltage-gated Na^+ channels. Example: diabetic peripheral neuropathy.

▶ PUBLIC HEALTH SCIENCES—HEALTHCARE DELIVERY

Disease prevention



Major medical insurance plans

Exclusive provider organization (EPO)	Requires in-network care; out-of-network coverage available for emergencies.
Health maintenance organization (HMO)	Lower premiums, limited to in-network providers, requires primary care referral for specialists, lower out-of-pocket cost.
Point of service (PoS)	Combination of HMO and PPO, allows out-of-network care at higher cost, and requires primary care referrals.
Preferred provider organization (PPO)	Flexibility with in-network and out-of-network care, higher cost for out-of-network services, no referral requirement.
Accountable care organization (ACO)	Group of providers who voluntarily coordinate care for Medicare patients.
High deductible health plan (HDHP)	Low premiums, high out-of-pocket cost, compatible with health savings account (HSA).

Rewritten fact for 2025 2nd pass

Fetal circulation

Fact updated for 2nd pass

3 important shunts:

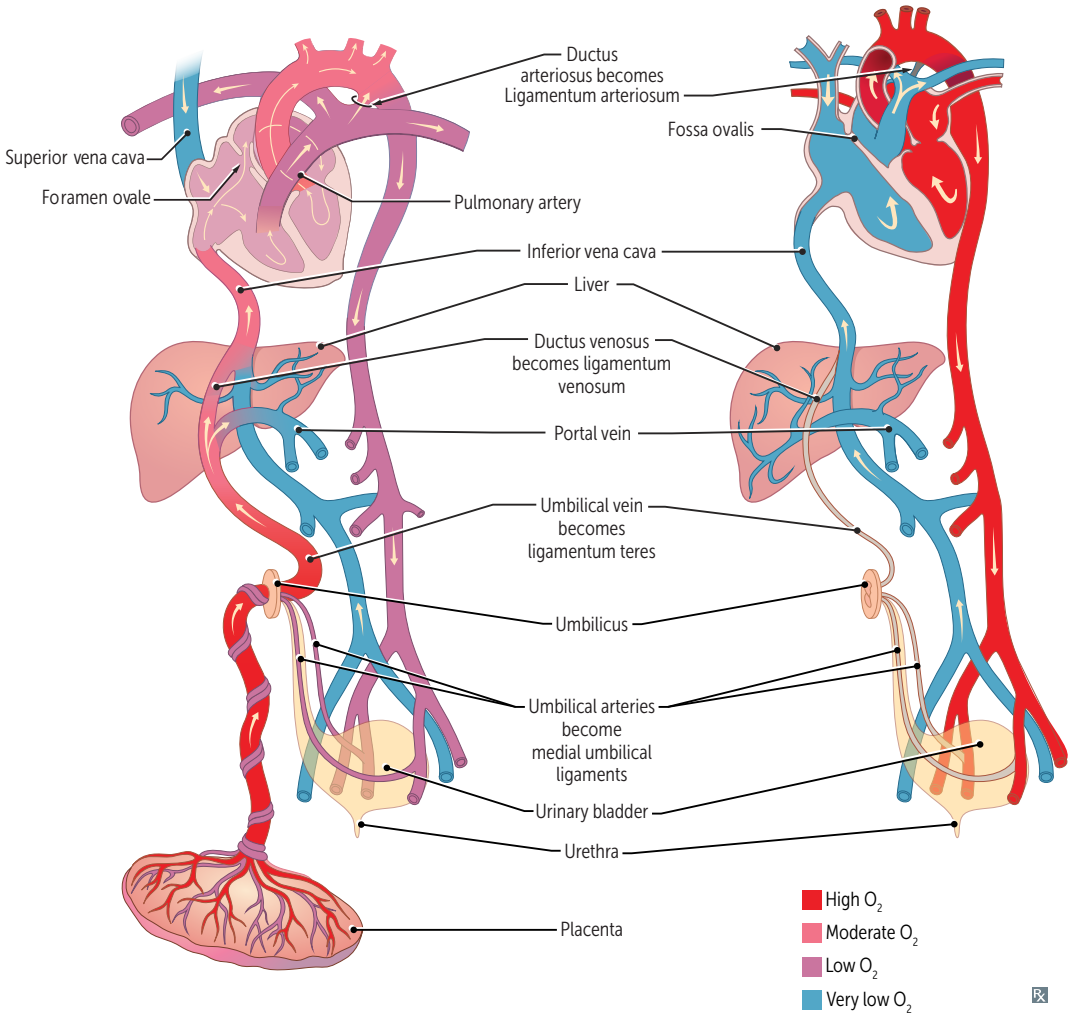
1. Umbilical vein → **ductus venosus** → IVC (bypassing hepatic circulation).
2. Most of the highly oxygenated blood from IVC → **foramen ovale** → LA.
3. Deoxygenated blood from SVC → RA → RV → main pulmonary artery → **ductus arteriosus** → descending aorta; shunt is due to ↑ fetal pulmonary artery resistance.

At birth, infant takes a breath → ↓ resistance in pulmonary vasculature → ↑ LA pressure vs RA pressure → foramen ovale closes (now called fossa ovalis); ↑ in O_2 (from respiration) and ↓ in prostaglandins (from placental separation) → closure of ductus arteriosus.

NSAIDs (eg, indomethacin, ibuprofen) or acetaminophen help close the patent ductus arteriosus (PDA) → ligamentum arteriosum (remnant of ductus arteriosus). "Endomethacin" ends the PDA.

Prostaglandins E_1 and E_2 kEEp PDA open.

Image revised for 2025 4th pass



Myocardial action potential

rev image for 2025 4th pass

new image for 2025 3rd pass

Phase 0 = rapid upstroke and depolarization—voltage-gated Na^+ channels open.

Phase 1 = initial repolarization—inactivation of voltage-gated Na^+ channels. Transient outward voltage-gated K^+ channels begin to open.

Phase 2 = plateau ("platwo")—Ca^{2+} influx through voltage-gated Ca^{2+} channels balances K^+ efflux. Ca^{2+} influx triggers Ca^{2+} release from sarcoplasmic reticulum and myocyte contraction (excitation-contraction coupling).

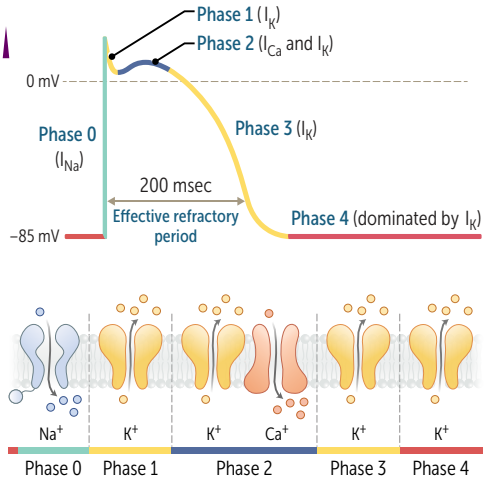
Phase 3 = rapid repolarization—K^+ efflux due to opening of voltage-gated slow delayed-rectifier K^+ channels and closure of voltage-gated Ca^{2+} channels.

Phase 4 = resting potential—high K^+ permeability through K^+ channels.

In contrast to skeletal muscle, cardiac muscle has the following characteristics:

- Action potential has a plateau due to Ca^{2+} influx and opposing K^+ efflux.
- Contraction requires Ca^{2+} influx from ECF to induce Ca^{2+} release from sarcoplasmic reticulum (Ca^{2+}-induced Ca^{2+} release).
- Myocytes conduct excitation throughout the heart via gap junctions.

Occurs in all cardiac myocytes except for those in the SA and AV nodes.



Pacemaker action potential

rev image for 2025 4th pass

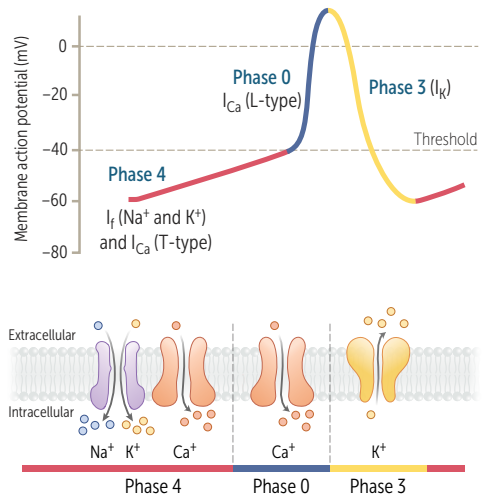
Key differences from the ventricular action potential include:

Phase 4 = slow spontaneous diastolic depolarization due to I_f ("funny current"). HCN channels responsible for a slow, mixed Na^+/K^+ inward current; different from I_{Na} in phase 0 of ventricular action potential. Accounts for automaticity of SA and AV nodes. The slope of phase 4 in the SA node determines HR. ACh/adenosine ↓ the rate of diastolic depolarization and ↓ HR, while catecholamines ↑ depolarization and ↑ HR. Sympathetic stimulation ↑ the chance that I_f channels are open and thus ↑ HR.

Phase 0 = upstroke—opening of voltage-gated Ca^{2+} channels. Fast voltage-gated Na^+ channels are permanently inactivated due to the less negative resting potential of these cells → slow conduction velocity, used by AV node to prolong transmission from atria to ventricles.

Phase 3 = repolarization—inactivation of Ca^{2+} channels and ↑ activation of K^+ channels → ↑ K^+ efflux.

Occurs in the SA and AV nodes. Phases 1 and 2 are absent.



Coronary artery disease

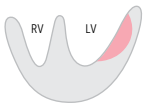
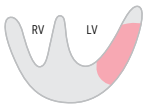
Angina

Chest pain due to ischemic myocardium 2° to coronary artery narrowing or spasm; no necrosis.

- Stable—usually 2° to atherosclerosis (≥ 70% occlusion); exertional chest pain in classic distribution resolving with rest or nitroglycerin.
- Unstable—thrombosis with incomplete coronary artery occlusion; ↑ in frequency or intensity of exertional chest pain or any chest pain at rest. No cardiac biomarker elevation (vs non–ST-segment elevation MI [NSTEMI]).
- Vasospastic (formerly Prinzmetal or variant)—occurs at rest 2° to coronary artery spasm; transient ischemic ST changes on ECG. Tobacco smoking is a major risk factor. Triggers include cocaine, amphetamines, alcohol, triptans. Treat with Ca^{2+} channel blockers, nitrates, and smoking cessation (if applicable).

Myocardial infarction

Most often due to an acute coronary syndrome (ACS): rupture of coronary artery atherosclerotic plaque → acute thrombosis. MI can also occur with prolonged supply-demand mismatch (eg, stable angina → prolonged tachycardia and hypotension from pneumonia → elevated troponin but no acute plaque rupture).

	Stable angina	Unstable angina	NSTEMI	STEMI
PAIN	On exertion	Mild exertion or at rest	At rest	At rest
TROPONIN LEVEL	No elevation	No elevation	Elevated	Elevated
INFARCTION	None	None	 Subendocardial	 Transmural
ECG CHANGES	Possible ST depression and/or T-wave inversion	Possible ST depression and/or T-wave inversion	ST depression and/or T-wave inversion	ST elevation, pathologic Q waves

Ischemic heart disease manifestations

Coronary steal syndrome

Distal to coronary stenosis, vessels are maximally dilated at baseline to compensate for reduced blood flow. Administration of vasodilators (eg, dipyridamole, adenosine, regadenoson) dilates normal vessels → ↓ hydrostatic pressure in normal coronary arteries → blood is shunted toward well-perfused areas → ↓ flow to myocardium perfused by stenosed vessels ("steal") → ischemia of myocardium downstream to pathologically dilated vessels. Vasodilator stress tests rely on differential in flow to detect potential ischemia. Rarely, they can cause coronary steal and true ischemia.

Vasodilation of healthy vessels steals blood from stenosed vessels.

Sudden cardiac death

Unexpected death due to cardiac causes within 1 hour of symptom onset or within 24 hours with no cardiovascular symptoms, most commonly due to lethal ventricular arrhythmia (eg, ventricular fibrillation) impairing blood flow to the brain. Associated with CAD (up to 70% of cases), cardiomyopathy (hypertrophic, dilated), myocarditis, coronary artery anomalies, and hereditary channelopathies (eg, long QT syndrome, Brugada syndrome). Prevent with implantable cardioverter-defibrillator.

Fact replaced for 2025 1st pass

Chronic ischemic heart disease

Progressive exertional symptoms and/or development of HF due to chronic ischemic myocardial damage.

Myocardial hibernation—LV systolic dysfunction in the setting of chronic ischemia. Potentially reversible with myocardial reperfusion. Seen in stable angina, acute MI, or HF. Contrast with myocardial stunning—transient, reversible LV systolic dysfunction after brief, acute ischemia.

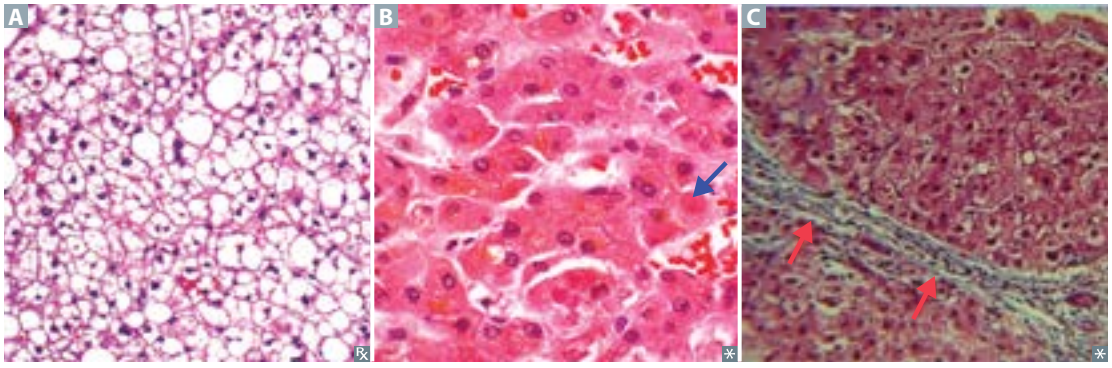
Reye syndrome

Rare, often fatal childhood hepatic encephalopathy.
Associated with viral infection (especially VZV and influenza) that has been treated with aspirin. Aspirin metabolites ↓ β-oxidation by reversible inhibition of mitochondrial enzymes.
Findings: mitochondrial abnormalities, fatty liver (microvesicular fatty changes), hyperammonemia, hypoglycemia, vomiting, hepatomegaly, coma.
↑ ICP ↑ morbidity and mortality. Renal and cardiac failure may also occur.

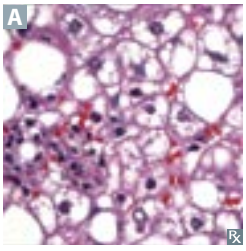
Avoid aspirin (**ASA**) in children, except in Kaw**ASA**ki disease.
Salicylates aren't a ray (**Reye**) of sun**SHINEE** for kids:
Steatosis of liver/hepatocytes
Hypoglycemia/**H**epatomegaly
Infection (VZV, influenza)
Not awake (coma)
Encephalopathy and diffuse cerebral **E**dema

Alcoholic liver disease

Alcoholic liver disease	Excess NADH production → ↓ fatty acid oxidation and ↑ lipogenesis.
Hepatic steatosis	Macrovesicular fatty change A; may be reversible with alcohol cessation.
Alcoholic hepatitis	Requires sustained, long-term consumption. Swollen and necrotic hepatocytes with neutrophilic infiltration. Mallory bodies B (intracytoplasmic eosinophilic inclusions of damaged keratin filaments).
Alcoholic cirrhosis	Final and usually irreversible form. Sclerosis around central vein may be seen in early disease. Regenerative nodules surrounded by fibrous bands (red arrows in C) in response to chronic liver injury → portal hypertension and end-stage liver disease.



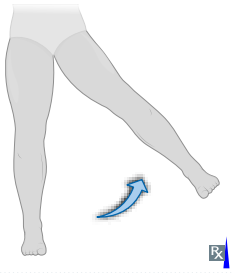
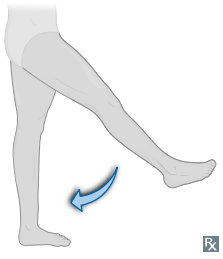
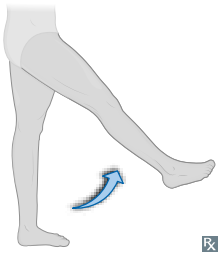
Steatotic liver disease



Steatotic liver disease (SLD) encompasses metabolic dysfunction–associated SLD (MASLD; formerly known as nonalcoholic fatty liver disease), MASLD and increased alcohol intake, alcohol-associated liver disease, specific etiology SLD, and cryptogenic SLD.
MASLD is associated with metabolic syndrome (obesity, insulin resistance, HTN, hypertriglyceridemia, ↓ HDL); obesity → fatty infiltration of hepatocytes A → cellular "ballooning" and eventual necrosis. Steatosis present without evidence of significant inflammation or fibrosis. May persist or even regress over time. Usually asymptomatic.
Metabolic dysfunction–associated steatohepatitis—associated with lobular inflammation and hepatocyte ballooning → fibrosis. May progress to cirrhosis and HCC.

new images for 2025 2nd pass

Actions of hip muscles

ACTION	MUSCLES	PRESENTATION
Abductors	Gluteus medius, gluteus minimus	
Adductors	Adductor magnus, adductor longus, adductor brevis	
Extensors	Gluteus maximus, semitendinosus, semimembranosus, long head of biceps femoris	
Flexors	Iliopsoas (iliacus and psoas), rectus femoris, tensor fascia lata, pectineus, sartorius	
Internal rotation	Gluteus medius, gluteus minimus, tensor fascia latae	
External rotation	Iliopsoas, gluteus maximus, piriformis, obturator internus, obturator externus	

Vasculitides Inflammation and necrosis of blood vessels; either idiopathic or immune mediated (type III hypersensitivity).

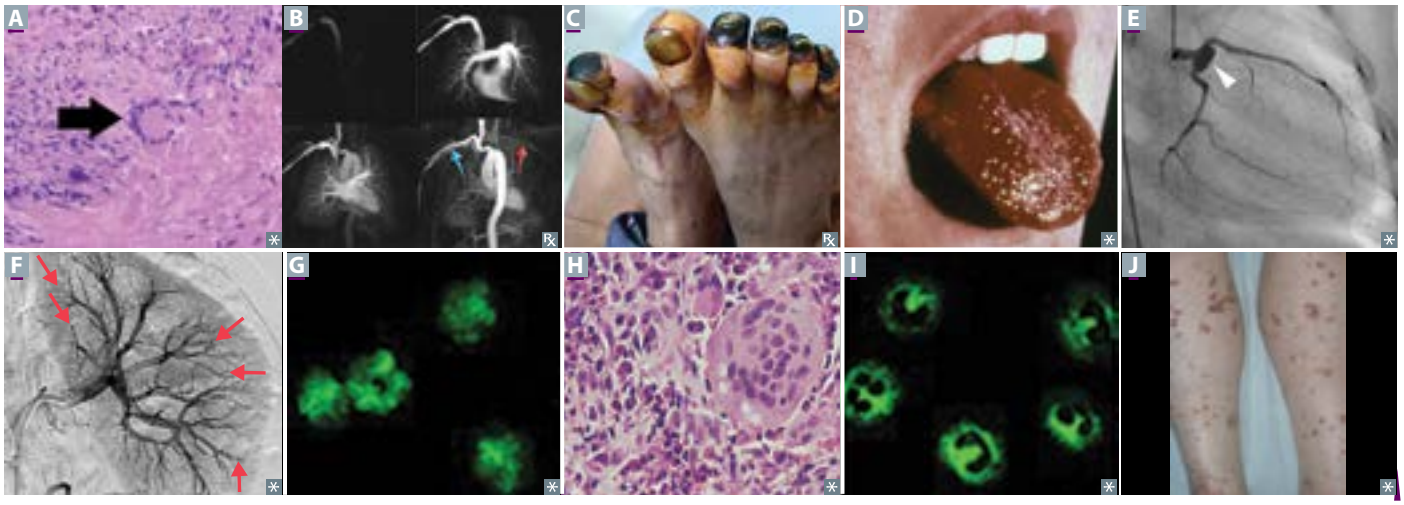
	EPIDEMIOLOGY	PRESENTATION/PATHOPHYSIOLOGY	NOTES
Large-vessel vasculitis			
Giant cell (temporal) arteritis	Females > 50 years old.	Unilateral headache, jaw claudication, temporal artery tenderness, blindness risk (due to anterior ischemic optic neuropathy). Granulomatous inflammation A; affects temporal, vertebral, and ophthalmic arteries.	Associated with polymyalgia rheumatica. ↑↑ ESR/CRP; temporal artery biopsy. Treatment: high-dose glucocorticoids before biopsy.
Takayasu arteritis	Asian females < 40 years old.	"Pulseless disease" (weak upper extremity pulses), fever, night sweats, arthritis, myalgias. Granulomatous thickening and narrowing of aortic arch and proximal great vessels B.	↑ ESR. Treatment: glucocorticoids.
Medium-vessel vasculitis			
Buerger disease (thromboangiitis obliterans)	Heavy tobacco smoking history, males < 40 years old.	Leading to intermittent claudication, risk of gangrene C, Raynaud phenomenon, autoamputation of digits, or superficial nodular phlebitis.	Segmental thrombosing vasculitis with vein and nerve involvement. Treatment: smoking cessation.
Kawasaki disease	Asian children < 4 years old.	Bilateral nonexudative Conjunctivitis, Rash (polymorphous → desquamating), Adenopathy (cervical), Strawberry tongue (oral mucositis) D, Hand-foot changes (edema, erythema), fever (≥ 5 days).	CRASH and burn on a Kawasaki. Complications: coronary artery aneurysms E; thrombosis or rupture can cause death. Treatment: IV immunoglobulin and aspirin.
Polyarteritis nodosa	Middle-aged males; 30% with hepatitis B seropositivity.	Fever, weight loss, abdominal pain, melena, hypertension, neurologic dysfunction, cutaneous eruptions, renal damage. Involves renal and visceral vessels, spares lungs; transmural inflammation with fibrinoid necrosis.	"String of pearls" appearance due to microaneurysms on arteriogram F. Treatment: glucocorticoids, cyclophosphamide. PAN usually affects the SKIN: Skin, Kidneys, Intestines (GI), Nerves.
Small-vessel vasculitis (MPO-ANCA/p-ANCA)			
Microscopic polyangiitis	Typically affects middle-aged adults.	Necrotizing vasculitis involving lungs, kidneys, and skin. Pauci-immune glomerulonephritis (GN) G and palpable purpura.	MPO-ANCA/p-ANCA (anti-myeloperoxidase). Treatment: cyclophosphamide, glucocorticoids.
Eosinophilic granulomatosis with polyangiitis		Combination of asthma, eosinophilia, and systemic vasculitis. Eosinophilic infiltration → inflammation → peripheral neuropathy.	Formerly called Churg-Strauss syndrome. Granulomatous, necrotizing vasculitis with eosinophilia H. ↑ IgE level.

Revised fact for 2025 3rd pass

Vasculitides *(continued)*

	EPIDEMIOLOGY	PRESENTATION/PATHOPHYSIOLOGY	NOTES
Small-vessel vasculitis (c-ANCA)			
Granulomatosis with polyangiitis	Affects males and females equally, typically middle-aged adults.	Triad: lung, vessels, and renal involvement. Upper respiratory (nasal septum perforation, chronic sinusitis, otitis media, mastoiditis), lower respiratory (hemoptysis, dyspnea), renal (pauci-immune rapidly progressive GN).	c-ANCA I (PR3-ANCA). Treatment: glucocorticoids combined with rituximab, cyclophosphamide.
Small-vessel vasculitis (immune complex mediated)			
Hypocomplementemic urticarial vasculitis (anti-C1q vasculitis)	Often associated with SLE.	Presents as urticaria, purpuric rash, arthralgias, stomach pain, lung or ocular manifestations.	Labs show ↓ C1q complement and ↑ anti-C1q antibodies.
Mixed cryoglobulinemia	Often due to viral infections, especially HCV.	Triad of palpable purpura, weakness, arthralgias. May involve peripheral neuropathy and renal disease (eg, GN).	Cryoglobulins precipitate in the cold (mixed IgG and IgM immune complex deposition).
Immunoglobulin A vasculitis	Most common childhood vasculitis often follows URI.	Vasculitis 2° to IgA immune complex deposition. Classic triad: Hinge pain (arthralgias), stomach pain (abdominal pain associated with intussusception), palpable purpura on buttocks/legs J.	Formerly called Henoch-Schönlein purpura. Associated with IgA nephropathy (Berger disease). Treatment: supportive care, glucocorticoids.
Cutaneous small-vessel vasculitis		Palpable purpura, no visceral involvement. Immune complex–mediated leukocytoclastic vasculitis; late involvement indicates systemic.	Occurs 7–10 days after medication use (penicillins, cephalosporins, sulfonamides, phenytoin, allopurinol) or infections (eg, HCV, HIV).
All-vessel vasculitis			
Behçet syndrome	↑ incidence in Turkish, Eastern Mediterranean descent.	Recurrent oral and genital ulcers, uveitis, erythema nodosum. Triggered by HSV or parvovirus. Flares last 1–4 weeks.	Associated with HLA-B51.

Revised fact for 2025 3rd pass



Sleep physiology

Sleep occurs in 4-6 cycles per night, each lasting ~90 mins and consisting of 2 main stages:
- Non-rapid eye movement (NREM) sleep
- Rapid-eye movement (REM) sleep; duration of REM sleep ↑ through the night

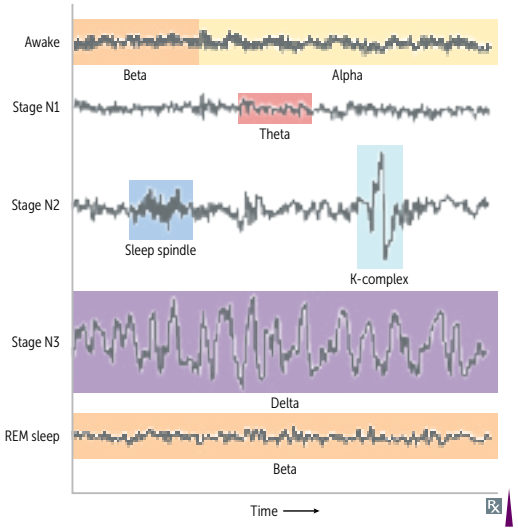
Sleep-wake cycle is regulated by circadian rhythm in the suprachiasmatic nucleus (SCN). ↓ light → ↓ SCN activity → ↑ NE from superior cervical ganglion → ↑ melatonin from pineal gland.

EEG waveforms: Beta, Alpha, Theta, Sleep spindle, Delta, Beta. At night, BATS Drink Blood.

New image for 2025 3rd pass

SLEEP STAGE (% OF TOTAL SLEEP)	DESCRIPTION
Awake	Alert, active mental concentration. Eyes open—beta waves (highest frequency, lowest amplitude). Eyes closed—alpha waves.
NREM sleep	
Stage N1 (5%)	Light sleep; theta waves.
Stage N2 (45%)	Deeper sleep; sleep spindles and K complexes. When bruxism occurs ("twoth" grinding in N2).
Stage N3 (25%)	Deepest NREM sleep (slow-wave sleep); delta waves (lowest frequency, highest amplitude). When bedwetting, sleepwalking, and night terrors occur (wee and flee in N3).
REM sleep (25%)	Loss of muscle tone (atonia) except in diaphragm and extraocular muscles, ↑ brain O_2 use, variable pulse/BP. When dreaming, nightmares, and penile/clitoral tumescence occur (REMember dreams).

EEG WAVEFORM



New fact for 2025 1st pass

REM sleep behavior disorder

Loss of atonia leading to dream enactment (often violent) and vocalization. Most commonly associated with Lewy body dementia and Parkinson disease.

Factors affecting sleep architecture

Alcohol, benzodiazepines, barbiturates: ↓ N3 and REM sleep (benzodiazepines are useful for sleepwalking and night terrors).

Aging: ↓ N3 and REM sleep, ↑ sleep-onset latency, early morning awakening.

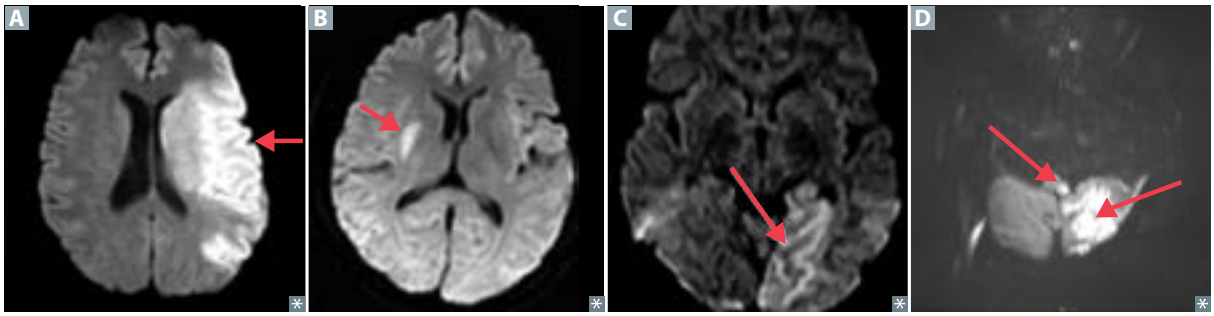
Depression: ↓ N3 sleep, ↑ REM sleep, ↓ REM latency, repeated nighttime awakenings, early morning awakening (terminal insomnia).

Narcolepsy: ↓ REM latency.

Effects of strokes *(continued)*

ARTERY	AREA OF LESION	SYMPTOMS	NOTES
Posterior inferior cerebellar artery	Nucleus ambiguus (CN IX, X).	Dysphagia, hoarseness, ↓ gag reflex, hiccups.	Lateral medullary (Wallenberg) syndrome.
	Vestibular nuclei.	Vomiting, vertigo, nystagmus	Nucleus ambiguus effects are specific to PICA lesions D.
	Lateral spinothalamic tract, spinal trigeminal nucleus.	↓ pain and temperature sensation from contralateral body, ipsilateral face.	"Don't pick a (PICA) lame (lateral medullary syndrome) horse (hoarseness) that can't eat (dysphagia)."
	Sympathetic fibers.	Ipsilateral Horner syndrome.	
	Inferior cerebellar peduncle.	Ipsilateral ataxia, dysmetria.	
Anterior spinal artery	Corticospinal tract.	Contralateral paralysis—upper and lower limbs.	Medial Medullary syndrome—caused by infarct of paramedian branches of ASA and/or vertebral arteries. Ants love M&M's.
	Medial lemniscus.	↓ contralateral proprioception.	
	Caudal medulla—hypoglossal nerve.	Ipsilateral hypoglossal dysfunction (tongue deviates ipsilaterally).	

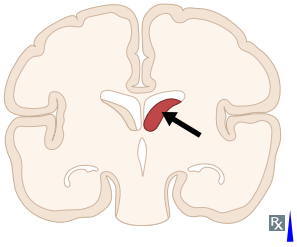
Art revised for 2025 2nd pass



Neonatal intraventricular hemorrhage

Bleeding into ventricles (arrow in illustration shows blood in intraventricular space). ↑ risk in premature and low-birth-weight infants. Originates in germinal matrix, a highly vascularized layer within the subventricular zone. Due to reduced glial fiber support and impaired autoregulation of BP in premature infants. Can present with altered level of consciousness, bulging fontanelle, hypotension, seizures, coma.

New image for 2025 2nd pass



Extracranial injuries

Occur during birth leading to blood accumulation within the scalp and skull. Commonly seen in vacuum-assisted delivery.

DISORDER	PRESENTATION
Caput succedaneum	Self-limited, benign, edematous swelling above periosteum; crosses suture lines. May be caused by prolonged fetal–birth canal engagement. Resolves spontaneously.
Subgaleal hemorrhage	Serious, life-threatening damage of fetal emissary veins → blood accumulation between periosteum and gala aponeurosis. Presents as diffuse, fluctuant scalp swelling extending posteriorly and laterally. May lead to anemia and hypovolemic shock.
Cephalohematoma	Blood accumulation between skull and periosteum; does not cross suture lines. May be caused by forcep delivery. Presents with firm, localized swelling over parietal or occipital lobe. May lead to indirect hyperbilirubinemia.

New fact for 2025 2nd pass

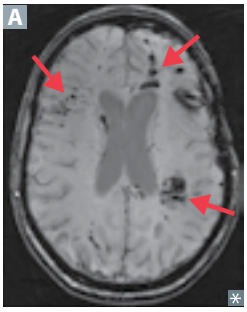
Thalamic pain syndrome

Severe, treatment-resistant neuropathic pain following thalamic lesions; may be due to occlusion of a lenticulostriate artery. Initial paresthesias followed in weeks to months by allodynia (ordinarily painless stimuli cause pain), hyperalgesia (hypersensitivity to pain), and dysesthesia (altered sensation) on the contralateral side.

Phantom limb pain

Sensation of burning, aching, or electric shock–like pain in a limb that is no longer present. Common after amputation. Associated with reorganization of the 1° somatosensory cortex.

Diffuse axonal injury

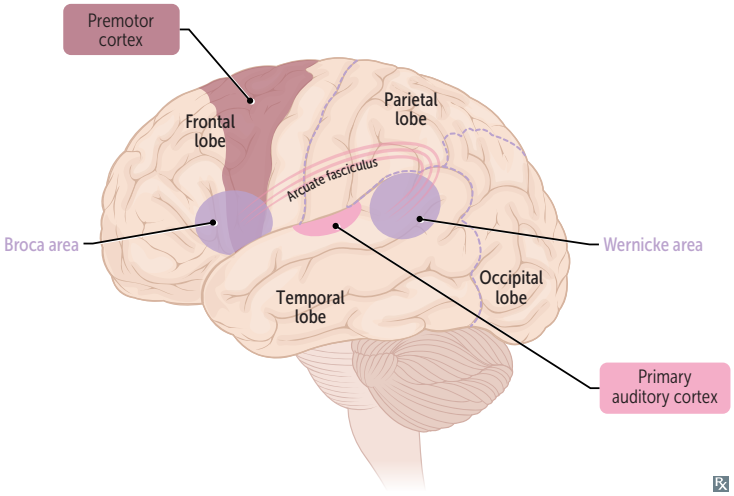


Traumatic shearing of white matter tracts during rapid acceleration and/or deceleration of the brain (eg, motor vehicle accident). Usually results in devastating neurologic injury, often causing coma or persistent vegetative state. MRI shows multiple lesions (punctate hemorrhages) involving white matter tracts A.

Aphasia

Higher-order language deficit (inability to understand/produce/use language appropriately); caused by pathology in dominant cerebral hemisphere (usually left).
Distinguish from Dysarthria—motor inability to produce speech (movement deficit).

TYPE	COMMENTS
Broca (expressive)	Broca area in inferior frontal gyrus of frontal lobe. Nonfluent speech with intact language comprehension. Patients appear frustrated, insight intact. Broca = broken boca (*boca* = mouth in Spanish).
Wernicke (receptive)	Wernicke area in superior temporal gyrus of temporal lobe. Fluent speech with impaired language comprehension. Patients do not have insight. Wernicke is a word salad and makes no sense.
Conduction	Can be caused by damage to arcuate fasciculus. Impaired speech repetition.
Global	Broca and Wernicke areas affected. Nonfluent speech with impaired language comprehension.



Seizures

Characterized by synchronized, high-frequency neuronal firing. Consist of 3 phases:
- Aura—early part of a seizure, may include odd smells or tastes.
- Ictal—time from first symptom to end of seizure activity.
- Postictal—period of gradual recovery back to preseizure baseline level of function/awareness.

Focal seizures

Originate in a single area of the brain, most commonly the medial temporal lobe. Types:
- Focal aware (formerly called simple partial)—consciousness intact; motor, sensory, autonomic, psychic symptoms
- Focal impaired awareness (formerly called complex partial)—impaired consciousness, automatisms

Generalized seizures

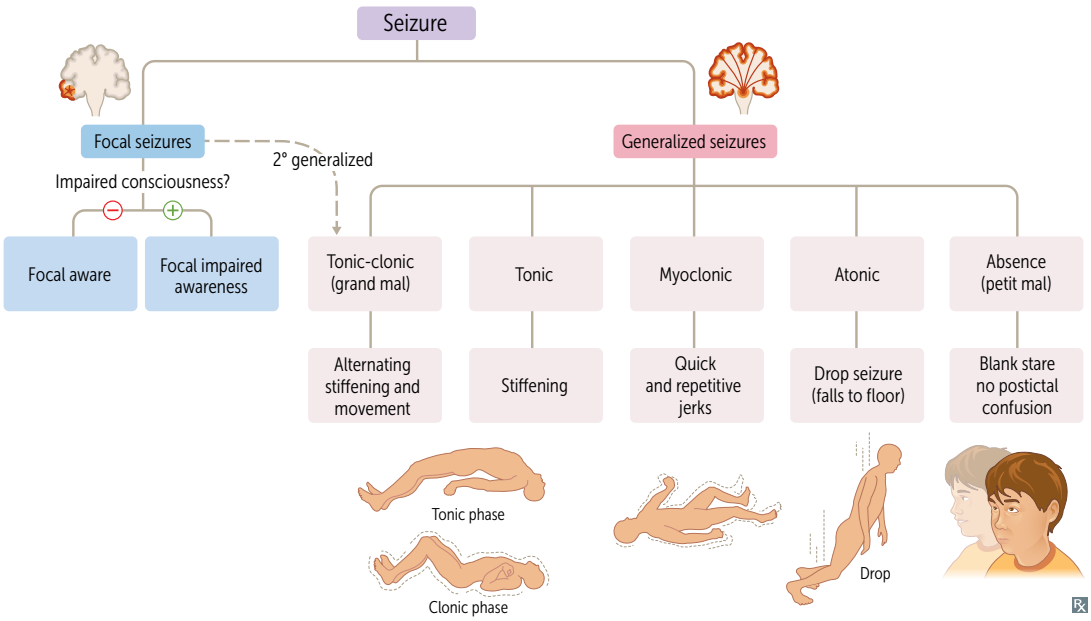
Diffuse. Types:
- Absence (petit mal)—3 Hz spike-and-wave discharges on EEG; short (usually 10 seconds), frequent episodes of blank stare, possible automatisms; no postictal confusion. Can be triggered by hyperventilation
- Myoclonic—quick, repetitive jerks; no loss of consciousness or postictal confusion
- Tonic-clonic (grand mal)—alternating stiffening and movement, postictal confusion, urinary incontinence, tongue biting
- Tonic—stiffening
- Atonic—"drop" seizures (falls to floor); commonly mistaken for fainting

Epilepsy—disorder of recurrent, unprovoked seizures (febrile seizures are not epilepsy).

Convulsive status epilepticus—continuous (≥ 5 min) or recurring seizures without interictal return to baseline consciousness that may result in brain injury.

Causes of seizures by age:
- Children < 18—genetic, infection (febrile), trauma, congenital, metabolic
- Adults 18–65—tumor, trauma, stroke, infection
- Adults > 65—stroke, tumor, trauma, metabolic, infection

Psychogenic nonepileptic events—resemble prolonged (> 1 minute) syncopal or tonic-clonic episodes without postictal phase, autonomic disturbances, or tongue biting. Often witnessed with vocalizations and preceding aura. Female sex predominance. Risk factors: history of psychiatric disorders, substance use. Normal video EEG.

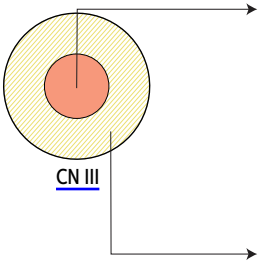


Cranial nerve III, IV, VI palsies

CN III damage

CN III has both motor (central) and parasympathetic (peripheral) components. Common causes include:

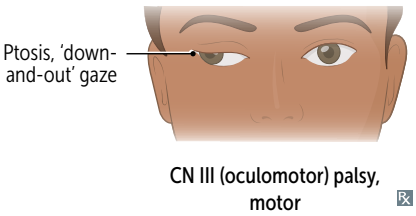
- Ischemia → pupil sparing (motor fibers affected more than parasympathetic fibers)
- Uncal herniation → coma
- PCom aneurysm → sudden-onset headache
- Cavernous sinus thrombosis → proptosis, involvement of CNs IV, V_1/V_2, VI
- Midbrain stroke → contralateral hemiplegia



Motor output to extraocular muscles—affected primarily by vascular disease (eg, diabetes mellitus: glucose → sorbitol) due to ↓ diffusion of oxygen and nutrients to the interior (middle) fibers from compromised vasculature that resides on outside of nerve. Signs: ptosis, "down-and-out" gaze.

Parasympathetic output—fibers on the periphery are first affected by compression (eg, PCom aneurysm, uncal herniation). Signs: diminished or absent pupillary light reflex, "blown pupil" often with "down-and-out" gaze.

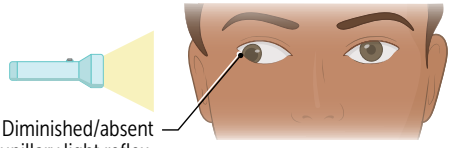
Motor = middle (central)
Parasympathetic = peripheral



CN III (oculomotor) palsy, motor

art revised for 2025 4th pass

new image for 2025 2nd pass

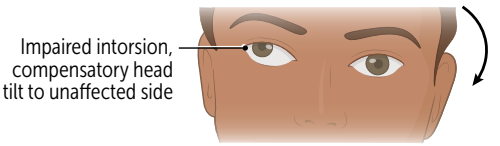


CN III (oculomotor) palsy, parasympathetic

CN IV damage

Pupil is higher in the affected eye. Characteristic head tilt to contralateral/unaffected side to compensate for lack of intorsion in affected eye.

Can't see the floor with CN IV damage (eg, difficulty going down stairs, reading).

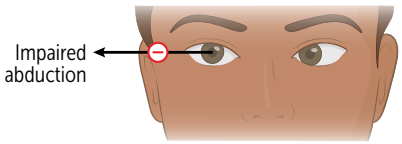


CN IV (trochlear) palsy

new image for 2025 2nd pass

CN VI damage

Affected eye unable to abduct and is displaced medially in primary position of gaze.



CN VI (abducens) palsy

new image for 2025 2nd pass

New fact for 2025 2nd pass

Eyelid disorders

DISORDER	PRESENTATION
Preseptal cellulitis	Anterior soft tissue eyelid infection. Mild presentation with unilateral ocular pain, swelling, and erythema present at rest.
Orbital cellulitis	Posterior eyelid infection affecting orbit contents (fat and muscles). Pain with ocular movement. Infection affecting the orbital contents (fat and muscles), usually secondary to bacterial sinusitis. Pain and double vision (diplopia) with ocular movement. Risk of vision loss, cavernous sinus thrombosis, and intracranial spread. Most commonly caused by *S aureus* and streptococci.
Blepharitis	Eyelid margin and lid inflammation, irritation, and crusting.
Hordeolum (stye)	Acute infection of the sebaceous or sweat glands of the eyelid. Tender, erythematous nodule.
Chalazion	Noninfectious granulomatous inflammation caused by obstruction of a meibomian (modified sebaceous) or Zeis (sebaceous) gland.
Xanthelasma	Yellowish patch on medial eyelid. May be associated with genetic and lifestyle factors, eg, high cholesterol.

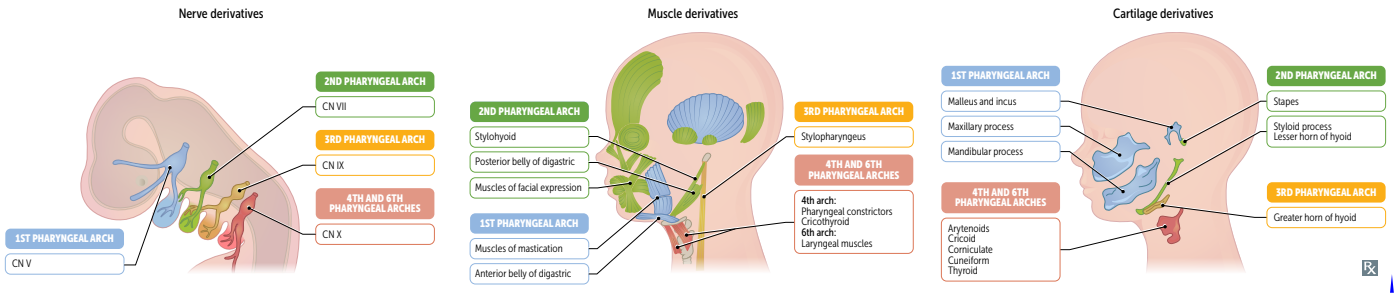
Pharyngeal arch derivatives

Sensory and motor nerves are not pharyngeal arch derivatives. They grow into the arches and are derived from neural crest (sensory) and neuroectoderm (motor). Arches 3 and 4 form posterior 1/3 of tongue. Arch 5 makes no major developmental contributions.

When at the restaurant of the golden **arches**, children tend to first **chew** (1), then **smile** (2), then **swallow stylishly** (3) or **simply swallow** (4), and then **speak** (6).

ARCH	NERVES	MUSCLES	CARTILAGE
1st pharyngeal arch	CN V_3 **chew**	Muscles of **m**astication (**t**emporalis, **m**asseter, lateral and **m**edial pterygoids), **m**ylohyoid, anterior belly of digastric, tensor tympani, anterior 2/3 of tongue, tensor veli palatini	**M**axillary process → **m**axilla, zygo**m**atic bone **M**andibular process → **M**eckel cartilage → **m**andible, **m**alleus and incus, spheno**m**andibular ligament
2nd pharyngeal arch	CN VII (seven)—**smile**	Muscles of facial expression, **s**tapedius, **s**tylohyoid, platy**s**ma, **p**osterior belly of digastric	Reichert cartilage → **s**tapes, **s**tyloid process, le**ss**er horn of hyoid, **s**tylohyoid ligament
3rd pharyngeal arch	CN IX—**swallow stylishly**	**Styl**opharyngeus	Greater horn of hyoid
4th and 6th pharyngeal arches	4th arch: CN X (superior laryngeal branch) **simply swallow** 6th arch: CN X (recurrent laryngeal branch) **speak**	4th arch: most pharyngeal constrictors; cricothyroid, levator veli palatini 6th arch: all intrinsic muscles of larynx except cricothyroid	**A**rytenoids, **C**ricoid, **C**orniculate, **C**uneiform, **T**hyroid cartilage (used to sing and **ACCCT**)

New fact for 2025 2nd pass



new image for 2025 2nd pass

First and second pharyngeal arch syndromes

Pierre Robin sequence	Mandibular hypoplasia (micrognathia) → posteriorly displaced tongue (glossoptosis) → cleft palate, airway compromise. Feeding difficulties are common.
Treacher Collins syndrome	Autosomal dominant neural crest dysfunction → craniofacial abnormalities (eg, zygomatic bone and mandibular hypoplasia), hearing loss, airway compromise.

New fact for 2025 2nd pass

Cyanide vs carbon monoxide poisoning

Both inhibit aerobic metabolism via inhibition of complex IV of ETC (cytochrome c oxidase) → hypoxia that does not fully correct with supplemental O_2 and ↑ anaerobic metabolism.

	Cyanide	Carbon monoxide
EXPOSURE	Synthetic product combustion, amygdalin ingestion (found in apricot seeds), cyanide ingestion (eg, in suicide attempts), fire victims. Risk of cyanide toxicity with use of nitroprusside in hypertensive emergencies.	From tobacco smoke, furnaces, space heaters, fires, motor exhaust (incomplete combustion of carbon-containing compounds). Odorless, tasteless, colorless, non-irritating. Leading worldwide cause of death by poisoning.
Revised fact for 2025 2nd pass PRESENTATION	Headache, dyspnea, drowsiness, seizure, coma. Skin may appear flushed ("cherry red") due to bright red venous blood. Venules in retina appear bright red. Breath may have bitter almond odor.	Headache, vomiting, confusion, visual disturbances, coma. May have cherry-red skin with bullous skin lesions. Multiple victims may be involved (eg, family due to faulty furnace).
LABS	Normal Pao_2. ↑ lactate → anion gap metabolic acidosis.	Normal Pao_2. ↑ carboxyhemoglobin on co-oximetry (cannot be distinguished with pulse oximetry). Classically associated with bilateral globus pallidus lesions on MRI A, although can rarely be seen with cyanide toxicity.
EFFECT ON OXYGEN-HEMOGLOBIN CURVE	Cyanide binds cytochrome a3 in complex IV → ample O_2 but cannot be used due to ineffective oxidative phosphorylation. Curve normal. O_2 saturation may appear normal initially.	Left shift in ODC → ↑ affinity for O_2 → ↓ O_2 unloading in tissues. Binds competitively to Hb with > 200× greater affinity than O_2 to form carboxyhemoglobin → ↓ $\%O_2$ saturation of Hb.
TREATMENT	Decontamination (eg, remove clothing). 100% O_2 is ineffective; instead give treatments to remove and excrete the cyanide: ▪ Hydroxocobalamin (binds cyanide → cyanocobalamin → renal excretion) ▪ Nitrites (oxidize Hb → methemoglobin → binds cyanide → cyanomethemoglobin → ↓ toxicity) ▪ Sodium thiosulfate (↑ cyanide conversion to thiocyanate → renal excretion)	Give 100% O_2 to overcome the increased affinity for CO. Hyperbaric oxygen if severe. CO-Hb half-life is ~300 mins → ↓ to ~80 mins on 100% O_2 → ↓ to ~20 mins in a hyperbaric O_2 chamber. If concurrent CO and cyanide poisoning are suspected (eg, in victims of a fire), give hydroxocobalamin, rather than nitrites or sodium thiosulfate, to avoid increasing methylglobin.