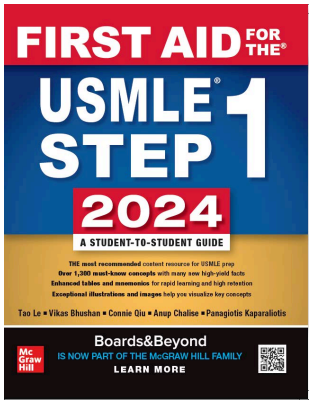


Textbooks and Course Syllabi

Limit your use of textbooks and course syllabi for Step 1 review. Many textbooks are too detailed for high-yield review and include material that is generally not tested on the USMLE Step 1 (eg, drug dosages, complex chemical structures). Syllabi, although familiar, are inconsistent across medical schools and frequently reflect the emphasis of individual faculty, which often does not correspond to that of the USMLE Step 1. Syllabi also tend to be less organized than top-rated books and generally contain fewer diagrams and study questions.

Integration of AI in Medical Education: Transforming USMLE Preparation

The integration of AI into education signals a paradigm shift in the acquisition and application of medical knowledge. AI's increasing ability to process extensive data sets and adapt to various learning styles makes it an attractive tool in medical training and practice.[17] Studies have demonstrated that AI language models are capable of achieving high accuracy rates when answering USMLE-style questions, underscoring its potential in supporting medical education.[18]

Although undeniably powerful, effectively utilizing AI as a study tool requires both practice and individual trial and error. We suggest the following approaches and prompts that might help learners more effectively harness AI for exam preparation:

Tailored Mnemonic Creation: Devise unique mnemonics to aid in memorizing complex medical terms efficiently. AI models can be highly creative in generating new ones, although feedback and iteration will likely be needed to produce mnemonics that are both accurate and memorable.

Example prompt: Create a food-related mnemonic for remembering adverse effects 1, 2, and 3 of Drug A.

Custom Summarization of Medical Texts: Efficiently condense extensive medical literature into concise summaries, facilitating efficient and rapid topic reviews.

Example prompt: Summarize this medical school lecture into bullet points. Decrease length by 80%.

AI-Generated Custom Quizzes: Create focused practice questions.

Example Prompt: Create three vignette-style multiple choice questions testing presentations of lysosomal storage disorders.

Clinical Case Simulations: Utilize AI-powered simulations of realistic clinical scenarios to practice decision-making skills and application of medical knowledge.

Example prompt: Create an exercise to practice analyzing acid-base disorders requiring Winter's formula with step-by-step explanations.

Personalized Learning Schedules: Create customized study schedules, adjusting time allocation based on challenging subjects. Modify schedules daily based on progress.

Example prompt: Prepare a schedule to review this book over 4 weeks.

Though both exciting and promising, pitfalls of using AI models for studying include the potential for outdated information or reliance on data that are not validated, resulting in a potential source of misinformation. AI can become unintentionally trained with human biases, and thus produce results that further reinforce or perpetuate potentially harmful biases. When using AI for personal studying, always validate information and maintain a critical eye when creating prompts.

AI is clearly a rapidly evolving study tool, however, how it can be best integrated with proven study methods remains to be seen. For the most recent updates on effectively leveraging AI in medical education, we encourage you to explore our blog at firstaidteam.com and scan a variety of student-centered discussion forums.

TEST-TAKING STRATEGIES

Practice! Develop your test-taking skills and strategies well before the test date.

Your test performance will be influenced by both your knowledge and your test-taking skills. You can strengthen your performance by considering each of these factors. Test-taking skills and strategies should be developed and perfected well in advance of the test date so that you can concentrate on the test itself. We suggest that you try the following strategies to see if they might work for you.

Pacing

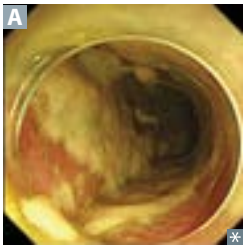
Time management is an important skill for exam success.

You have seven hours to complete up to 280 questions. Note that each one-hour block contains up to 40 questions. This works out to approximately 90 seconds per question. We recommend following the "1 minute rule" to pace yourself. Spend no more than 1 minute on each question. If you are still unsure about the answer after this time, mark the question, make an educated guess, and move on. Following this rule, you should have approximately 20 minutes left after all questions are answered, which you can use to revisit all of your marked questions. Remember that some questions may be experimental and do not count for points (and reassure yourself that these experimental questions are the ones that are stumping you). In the past, pacing errors have been detrimental to the performance of even highly prepared examinees. The bottom line is to keep one eye on the clock at all times!

Bacillus cereus

Gram ⊕ rod. Causes food poisoning. Spores survive cooking rice (reheated rice syndrome). Keeping rice warm results in germination of spores and enterotoxin formation.
Emetic type causes nausea and vomiting within 1–5 hours. Caused by cereulide, a preformed toxin.
Diarrheal type causes watery, nonbloody diarrhea and GI pain within 8–18 hours.
Management: supportive care (antibiotics are ineffective against toxins).

Clostridioides difficile



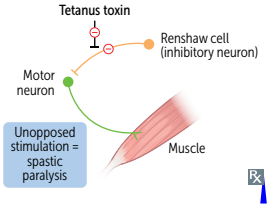
Produces toxins A and B, which damage enterocytes. Both toxins lead to watery diarrhea → pseudomembranous colitis A. Often 2° to antibiotic use, especially clindamycin, ampicillin, cephalosporins, fluoroquinolones; associated with PPIs.
Fulminant infection: toxic megacolon, ileus, shock.

Difficile causes diarrhea.
Diagnosed by PCR or antigen detection of one or both toxins in stool.
Treatment: oral vancomycin or fidaxomicin. For recurrent cases, consider repeating prior regimen or fecal microbiota transplant.

Clostridia

Gram ⊕, spore-forming, obligate anaerobic rods. Tetanus toxin and botulinum toxin are proteases that cleave SNARE proteins involved in neurotransmission.

Clostridium tetani



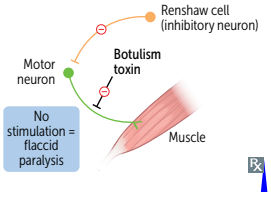
Pathogen is noninvasive and remains localized to wound site. Produces tetanospasmin, an exotoxin causing tetanus. Tetanospasmin spreads by retrograde axonal transport to CNS and blocks release of GABA and glycine from Renshaw cells in spinal cord.
Causes spastic paralysis, trismus (lockjaw), risus sardonicus (raised eyebrows and open grin), opisthotonos (spasms of spinal extensors).

*Tet*anus is *tet*anic paralysis.
Prevent with tetanus vaccine. Treat with antitoxin +/– vaccine booster, antibiotics, diazepam (for muscle spasms), and wound debridement.

new image for 2024 2nd pass

Revised image for 2024 3rd pass

Clostridium botulinum



Produces a heat-labile toxin that damages SNARE proteins, thus preventing ACh release at the neuromuscular junction, causing botulism. In babies, ingestion of spores (eg, in honey) leads to disease (floppy baby syndrome). In adults, disease is caused by ingestion of preformed toxin (eg, in canned food).
Symptoms of botulism (the 5 D's): diplopia, dysarthria, dysphagia, dyspnea, descending flaccid paralysis. Does not present with sensory deficits.

*Bot*ulinum is from bad bottles of food, juice, and honey.
Treatment: human botulinum immunoglobulin.
Local botulinum toxin A (Botox) injections used to treat focal dystonia, hyperhidrosis, muscle spasms, and cosmetic reduction of facial wrinkles.

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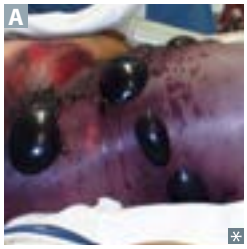
Revised image for 2024 3rd pass

Clostridium perfringens



Produces α-toxin (lecithinase, a phospholipase) that can cause myonecrosis (gas gangrene A; presents as soft tissue crepitus) and hemolysis.
If heavily spore-contaminated food is cooked but left standing too long at < 60°C, spore germinate → vegetative bacteria ingested → enterotoxin → late-onset (10–12 hours) food poisoning symptoms, resolution in 24 hours.

Perfringens perforates a gangrenous leg.
Spontaneous gas gangrene (via hematogenous seeding; associated with colonic malignancy) is most commonly caused by *Clostridium septicum*.

HIV therapy *(continued)*

DRUG	MECHANISM	ADVERSE EFFECTS
Protease inhibitors		
Atazanavir Darunavir Lopinavir Ritonavir	Prevents maturation of new virions. Maturation depends on HIV-1 protease (*pol* gene), which cleaves the polypeptide products of HIV mRNA into their functional parts. All protease inhibitors require boosting with either ritonavir or cobicistat. Navir (never) tease a protease.	Hyperglycemia, GI intolerance (nausea, diarrhea). Rifampin (potent CYP/UGT inducer) ↓ protease inhibitor concentrations; use rifabutin instead. Ritonavir (cytochrome P-450 inhibitor) is only used as a boosting agent.
Entry inhibitors		
Enfuvirtide	Binds gp41, inhibiting viral entry. Enfuvirtide inhibits fusion.	Skin reaction at injection sites.
Maraviroc	Binds CCR-5 on surface of T cells/monocytes, inhibiting interaction with gp120. Maraviroc inhibits docking.	

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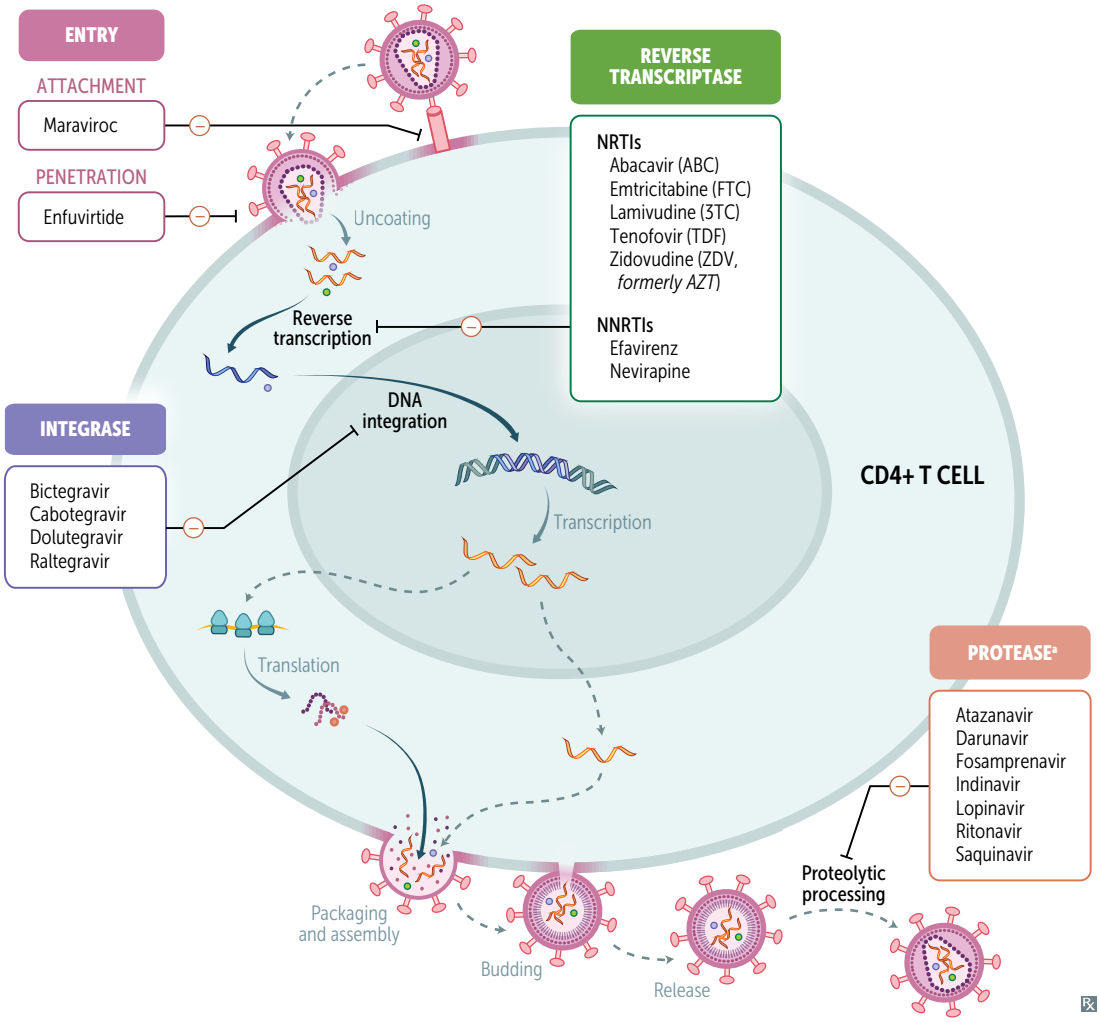


[a]All protease inhibitors require boosting with either ritonavir (protease inhibitor only used as a boosting agent) or cobicistat (cytochrome P450 inhibitor).

Gender- and sexuality-inclusive history taking

Avoid making assumptions about sexual orientation, gender identity, gender expression, and behavior (eg, a patient who identifies as heterosexual may engage in same-sex sexual activity).

Use gender-neutral terms when referring to the patient or the patient's family (eg, "partner" rather than "husband" or "wife") upon first meeting the patient until the patient instructs otherwise or uses specific pronouns. A patient's assigned sex at birth and gender identity may differ. Do not bring up gender or sexuality if it is not relevant to the visit (eg, a gender-nonconforming patient seeking care for a hand laceration).

Consider stating what pronouns you use when you introduce yourself (eg, "I'm Dr. Smith, and I use she/her pronouns") and asking patients how they would like to be addressed. Also consider ways of being inclusive (eg, ensuring correct name and pronouns are in the EMR).

Reassure them about the confidentiality of their visits and be sensitive to the fact that patients may not be open about their sexual orientation or gender identity to others in their life.

Remember: trust is built over time, and listening to and learning from patients about how they would like to approach the topics discussed above is key.

Cultural formulation interview

Identify the problem through the patient's perspective. Ask the patient to describe the problem in their own words, or how the patient would describe the problem to their family and friends.

Identify cultural perceptions of factors leading to a problem. Ask the patient to explain why they think they are experiencing their problem.

Identify how the patient's background influences their problem. Ask the patient about what makes their problem better or worse. Investigate roles of family, community, and spirituality.

Identify how culture may impact current and future interventions. Ask the patient if they have any concerns or suggestions about the current plan of treatment. If they do not want to follow medical advice, investigate if there is a way to combine their plans with the standard medical regimen.

Identify possible barriers to care based on culture. Ask the patient if there is anything that would prevent them from seeking care in a standard medical institution. Probe for explanations and what may increase the chance of maintaining a good patient-physician relationship.

Motivational interviewing

Counseling technique to facilitate behavior modification by helping patients resolve ambivalence about change. Useful for many conditions (eg, nicotine dependence, obesity). Helpful when patient has some desire to change, but it does not require that the patient be committed to making the change. May involve asking patients to examine how their behavior interferes with their life or why they might want to change it. Assess barriers (eg, food access, untreated trauma) that may make behavior change difficult.

Assessing a patient's readiness for change is also important for guiding physician-suggested goals. These goals should be **S**pecific, **M**easurable, **A**chievable, **R**elevant, and **T**ime bound (**SMART**).

Trauma-informed care

Patients with history of psychological trauma should receive thorough behavioral health screenings. Regularly assess mood, substance use, social supports, and suicide risk.

Focus assessments on trauma-related symptoms that interfere with social and occupational function. Always be empathetic. Do not ask invasive questions requiring the patient to describe trauma in detail. Ask permission prior to discussion.

Before the physical exam, reassure patients that they may signal to end it immediately if they experience too much physical or emotional discomfort. Offer the presence of additional staff for support. Psychological counseling may be indicated. Follow-up counseling is offered (or advised) as appropriate. Remember 4 R's: **R**ealize, **R**ecognize, **R**espond, **R**esist retraumatization.

Coronary artery disease

new fact for 2024 1st pass

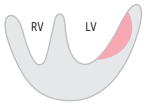
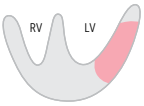
Angina

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 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 rupture of coronary artery atherosclerotic plaque → acute thrombosis. ↑ cardiac biomarkers (CK-MB, troponins) are diagnostic.

	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

updated fact for 2024 1st pass

Coronary steal syndrome

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

Sudden cardiac death

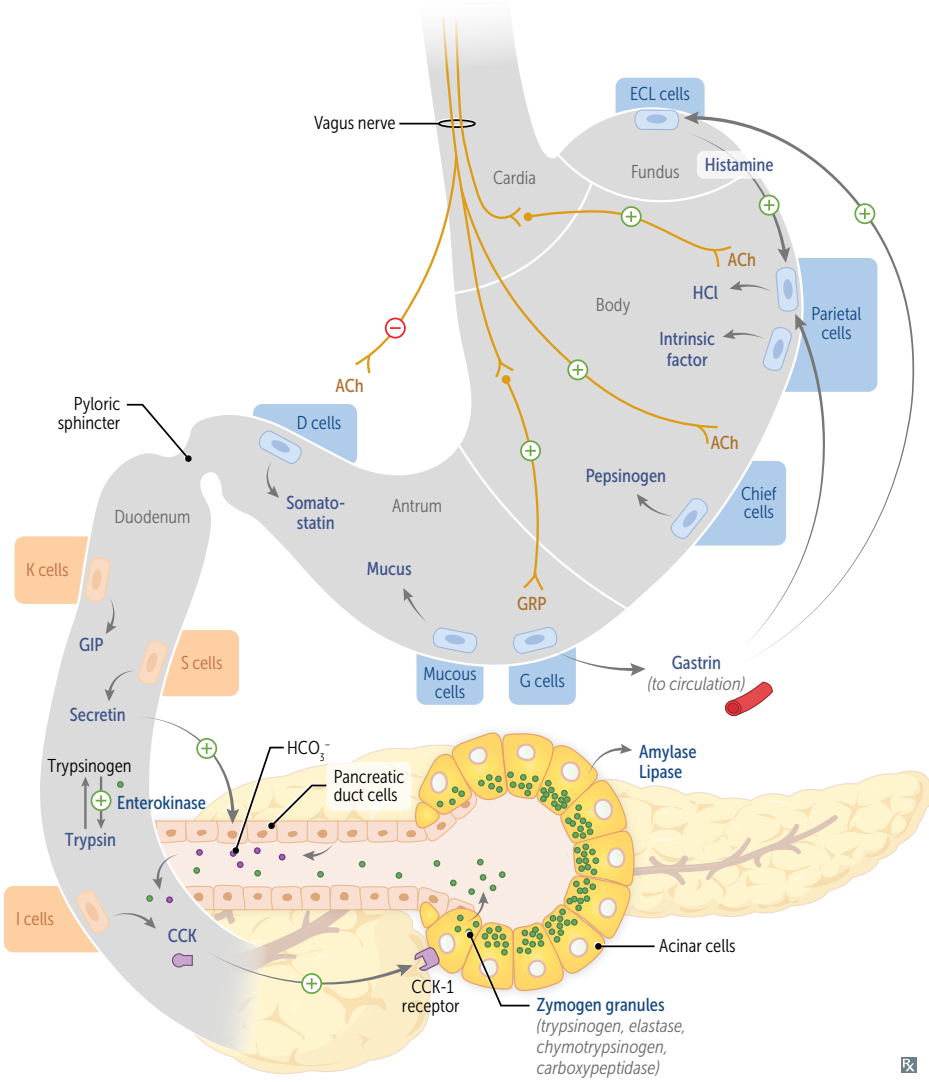
Unexpected death due to cardiac causes within 1 hour of symptom onset, most commonly due to lethal arrhythmia (eg, ventricular fibrillation). Associated with CAD (up to 70% of cases), cardiomyopathy (hypertrophic, dilated), and hereditary channelopathies (eg, long QT syndrome, Brugada syndrome). Prevent with implantable cardioverter-defibrillator.

Chronic ischemic heart disease

Progressive onset of HF over many years due to chronic ischemic myocardial damage.
Myocardial hibernation—potentially reversible LV systolic dysfunction in the setting of chronic ischemia. Contrast with myocardial stunning, a transient LV systolic dysfunction after a brief episode of acute ischemia.

Locations of gastrointestinal secretory cells

Gastrin ↑ acid secretion primarily through its effects on enterochromaffin-like (ECL) cells (leading to histamine release) rather than through its direct effect on parietal cells.



new image for 2024 1st pass

Pancreatic secretions

Isotonic fluid; low flow → high Cl^-, high flow → high HCO_3^-.

ENZYME	ROLE	NOTES
α-amylase	Starch digestion	Secreted in active form
Lipases	Lipid digestion	
Proteases	Protein digestion	Includes trypsin, chymotrypsin, elastase, carboxypeptidases Secreted as proenzymes also called zymogens Dipeptides and tripeptides degraded within intestinal mucosa via intracellular process
Trypsinogen	Converted to active enzyme trypsin → activation of other proenzymes and cleaving of additional trypsinogen molecules into active trypsin (positive feedback loop)	Converted to trypsin by enterokinase/ enteropeptidase, a brush-border enzyme on duodenal and jejunal mucosa

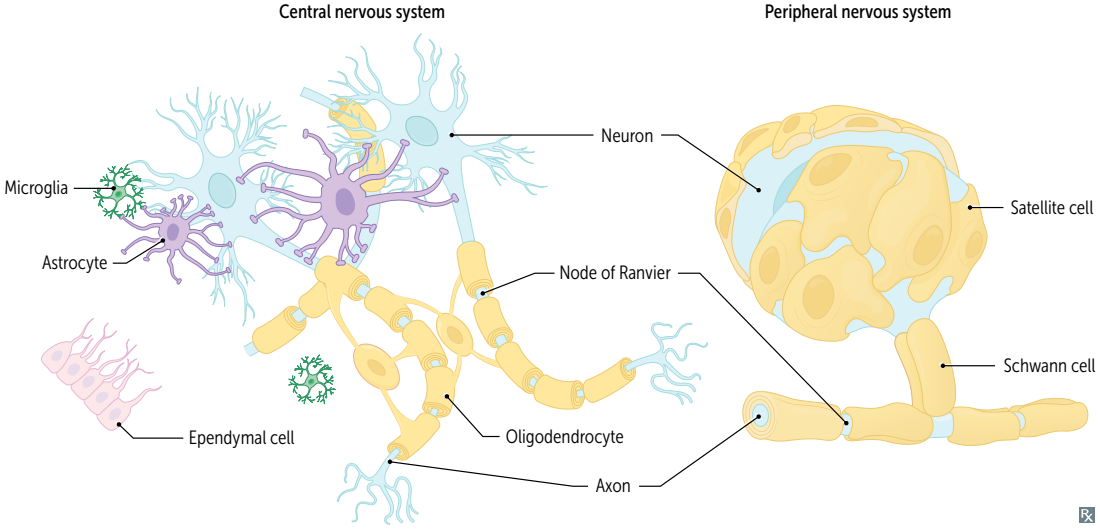
Tongue development fact deleted here for 2024 1st pass

▶ NEUROLOGY—ANATOMY AND PHYSIOLOGY

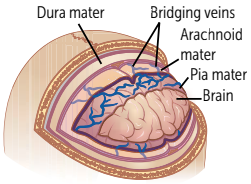
New fact/ 7 deleted facts for 2024 1st pass

Cells of the nervous system	Neurons and nonneuronal (glial) cells. Neurons—permanent, signal-transmitting cells of the nervous system composed of dendrites (receive input), cell bodies, and axons (send output). Dendrites and cell bodies can be seen on Nissl staining (stains RER; not present in axons). Markers: neurofilament protein, synaptophysin.	CNS glial cells—neuroectoderm (except microglia, which derive from mesoderm). PNS glial cells—neural crest ectoderm. Myelin is a multilayer wrapping of electrical insulation formed around axons → ↑ conduction velocity of transmitted signals via saltatory conduction of action potentials at nodes of Ranvier (↑↑ Na^+ channel density).
CNS glial cells		
Astrocytes	Physical support, repair, removal of excess neurotransmitters, component of blood-brain barrier, glycogen fuel reserve buffer. GFAP ⊕.	Largest and most abundant glial cell in CNS. Reactive gliosis in response to neural injury.
Oligodendrocytes	Myelinate axons in CNS (including CN II). "Fried egg" appearance histologically ("oleggodendrocytes").	Each myelinates many axons (~ 30). Predominant type of glial cell in white matter. Injured in multiple sclerosis, leukodystrophies, progressive multifocal leukoencephalopathy.
Ependymal cells	Ciliated simple columnar glial cells lining ventricles and central canal of spinal cord. Apical surfaces are covered with cilia (which circulate CSF) and microvilli (which help with CSF absorption).	Specialized ependymal cells (choroid plexus) produce CSF.
Microglia	Activation in response to tissue damage → release of inflammatory mediators (eg, nitric oxide, glutamate). Not readily discernible by Nissl stain.	Phagocytic scavenger cells of CNS. HIV-infected microglia fuse to form multinucleated giant cells in CNS in HIV-associated dementia.
PNS glial cells		
Satellite cells	Surround neuronal cell bodies in ganglia.	Similar supportive role to astrocytes.
Schwann cells	Myelinate axons in PNS (including CN III-XII). S100 ⊕.	Each myelinates a single axon ("Schwone"). Injured in Guillain-Barré syndrome.

art revised for 2024 2nd pass



Meninges



Rx

Three membranes that surround and protect the brain and spinal cord. Derived from both neural crest and mesoderm:

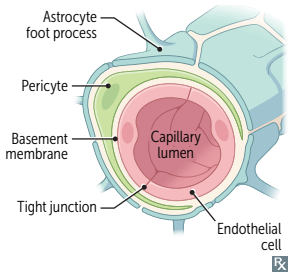
- Dura mater—thick outer layer closest to skull.
- Arachnoid mater—middle layer, contains weblike connections.
- Pia mater—thin, fibrous inner layer that firmly adheres to brain and spinal cord.

CSF flows in the subarachnoid space, located between arachnoid and pia mater.

Epidural space—potential space between dura mater and skull/vertebral column containing fat and blood vessels. Site of blood collection associated with middle meningeal artery injury.

art revised for 2024 1st pass

Blood-brain barrier



Rx

Prevents circulating blood substances (eg, bacteria, drugs) from reaching the CSF/CNS. Formed by 4 structures:

- Tight junctions between nonfenestrated capillary endothelial cells
- Basement membrane
- Pericytes
- Astrocyte foot processes

Glucose and amino acids cross slowly by carrier-mediated transport mechanisms.

Nonpolar/lipid-soluble substances cross rapidly via diffusion.

Circumventricular organs with fenestrated capillaries and no blood-brain barrier allow molecules in blood to affect brain function (eg, area postrema—vomiting after chemotherapy; OVLT [organum vasculosum lamina terminalis]—osmoreceptors) or neurosecretory products to enter circulation (eg, neurohypophysis—ADH release).

BBB disruption (eg, stroke) → vasogenic edema.

Hyperosmolar agents (eg, mannitol) can disrupt the BBB → ↑ permeability of medications.

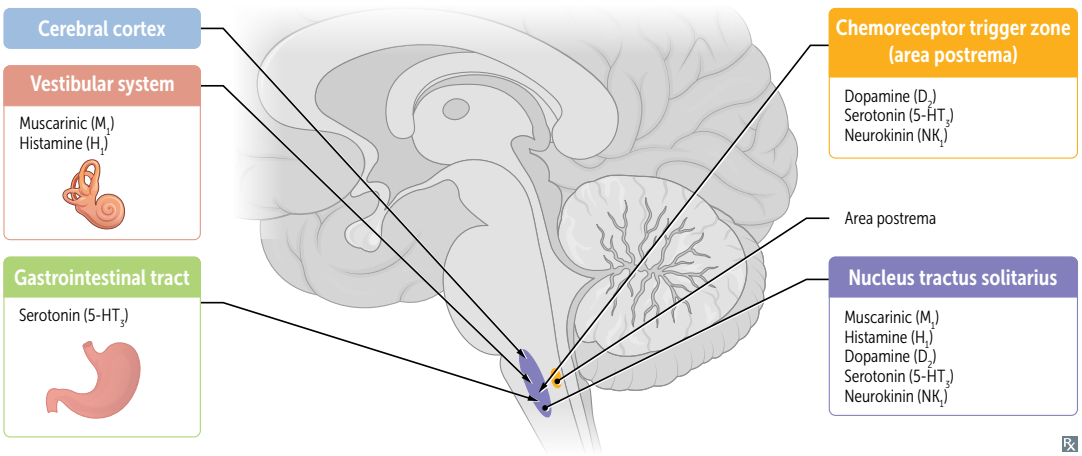
new image for 2024 1st pass

Vomiting center

Coordinated by NTS in the medulla, which receives information from the chemoreceptor trigger zone (CTZ, located within area postrema (pronounce "puke"-strema) in 4th ventricle), GI tract (via vagus nerve), vestibular system, and CNS.

CTZ and adjacent vomiting center nuclei receive input through 5 major receptors: histamine (H_1), muscarinic (M_1), neurokinin (NK-1), dopamine (D_2), and serotonin (5-HT_3).

- 5-HT_3, D_2, and NK-1 antagonists treat chemotherapy-induced vomiting.
- H_1 and M_1 antagonists treat motion sickness; H_1 antagonists treat hyperemesis gravidarum.



Rx

new image for 2024 1st pass

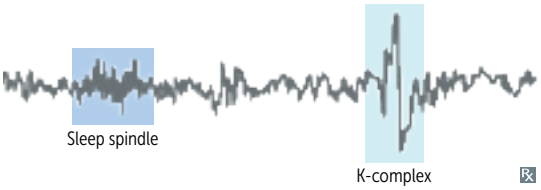
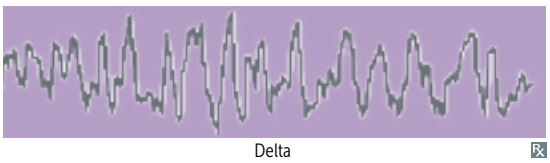
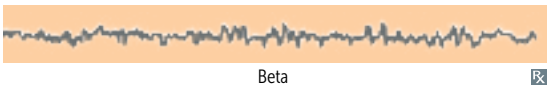
art revised for 2024 2nd pass

Updated fact for 2024 1st 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 (non-REM) sleep
- Rapid-eye movement (REM) sleep; duration of REM sleep ↑ through the night

Sleep-wake cycle is regulated by circadian rhythm, which is driven by suprachiasmatic nucleus (SCN) of hypothalamus. Low light conditions → ↓ SCN activity → ↑ norepinephrine from superior cervical ganglion → ↑ melatonin from pineal gland.

SLEEP STAGE (% OF TOTAL SLEEP)	DESCRIPTION	EEG WAVEFORM
Awake	Alert, active mental concentration. Eyes open—beta waves (highest frequency, lowest amplitude). Eyes closed—alpha waves.	
Non-REM 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 non-REM 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 motor tone, ↑ brain O_2 use, variable pulse/BP. Extraocular movements due to activity of PPRF (paramedian pontine reticular formation/conjugate gaze center). May serve memory processing function. When dreaming, nightmares, and penile/clitoral tumescence occur (remember dreams in REM).	

New fact for 2024 1st pass

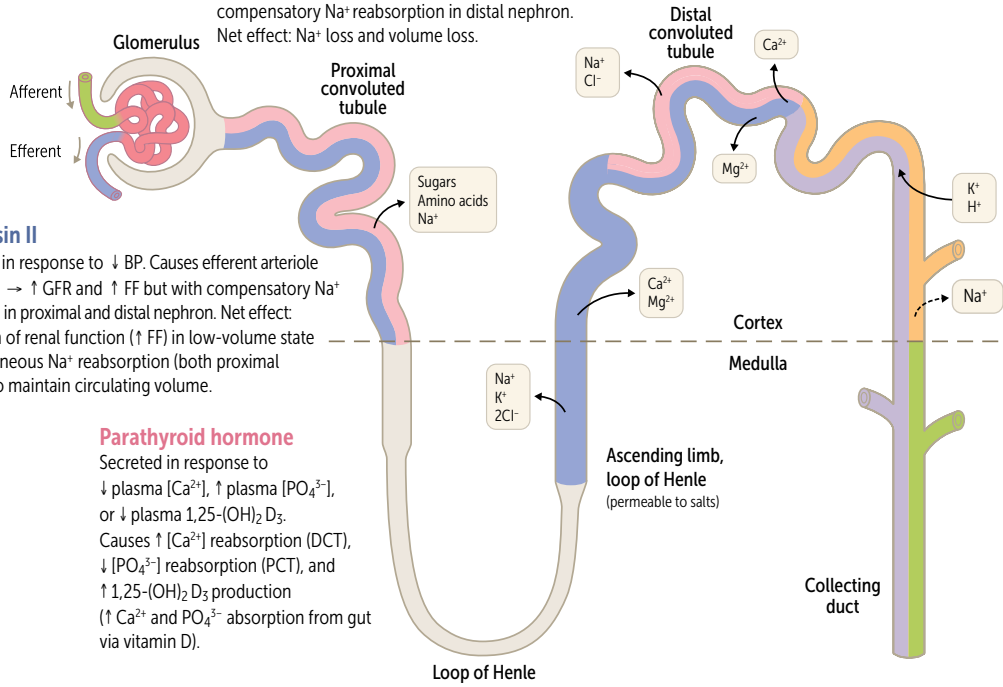
Factors affecting sleep architecture

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.

Hormones acting on kidney

Atrial natriuretic peptide

Secreted in response to ↑ atrial pressure. Causes indirect afferent arteriole dilation (through inhibition of NE). Causes ↑ GFR and ↑ Na^+ filtration with no compensatory Na^+ reabsorption in distal nephron. Net effect: Na^+ loss and volume loss.



Angiotensin II

Synthesized in response to ↓ BP. Causes efferent arteriole constriction → ↑ GFR and ↑ FF but with compensatory Na^+ reabsorption in proximal and distal nephron. Net effect: preservation of renal function (↑ FF) in low-volume state with simultaneous Na^+ reabsorption (both proximal and distal) to maintain circulating volume.

Parathyroid hormone

Secreted in response to ↓ plasma $[Ca^{2+}]$, ↑ plasma $[PO_4^{3-}]$, or ↓ plasma 1,25-$(OH)_2$ D_3. Causes ↑ $[Ca^{2+}]$ reabsorption (DCT), ↓ $[PO_4^{3-}]$ reabsorption (PCT), and ↑ 1,25-$(OH)_2$ D_3 production (↑ Ca^{2+} and PO_4^{3-} absorption from gut via vitamin D).

Aldosterone

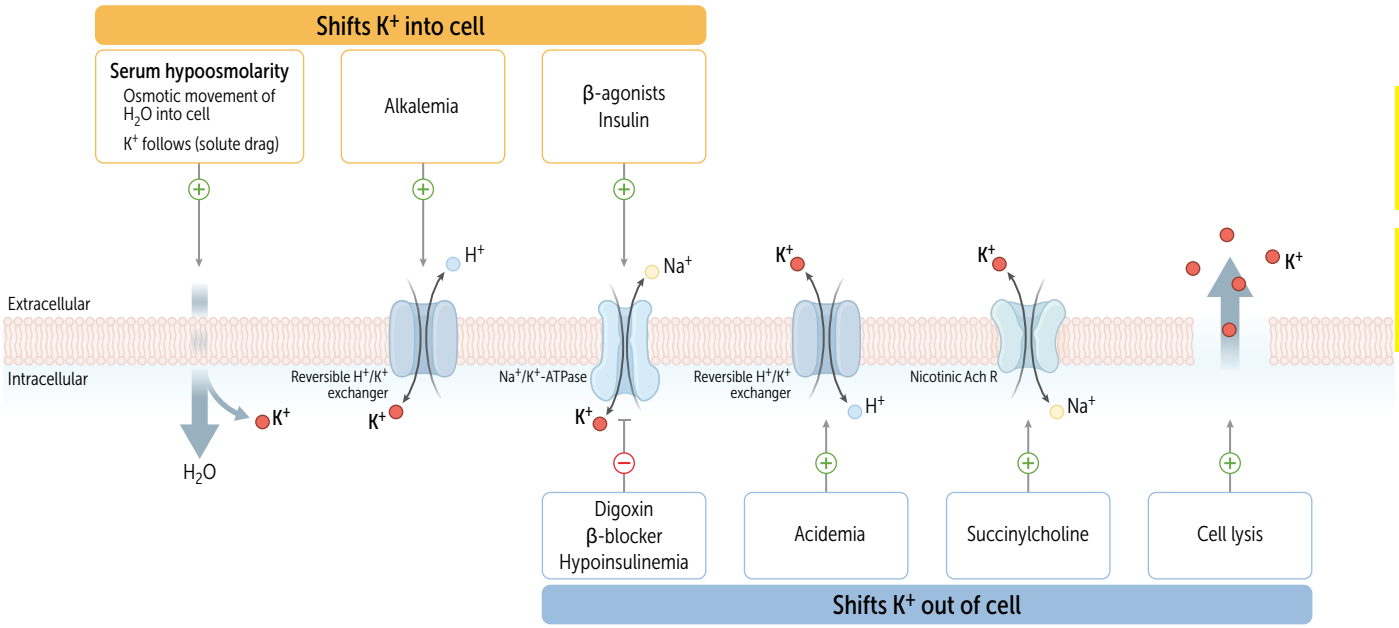
Secreted in response to ↓ blood volume (via AT II) and ↑ plasma $[K^+]$; causes ↑ Na^+ reabsorption, ↑ K^+ secretion, ↑ H^+ secretion.

ADH (vasopressin)

Secreted in response to ↑ plasma osmolarity and ↓ blood volume. Binds to receptors on principal cells, causing ↑ number of aquaporins and ↑ H_2O reabsorption.
↑ reabsorption of urea in medullary collecting ducts to maximize corticopapillary osmotic gradient.

art revised for 2024 3rd pass

Potassium shifts



art revised for 2024 1st pass

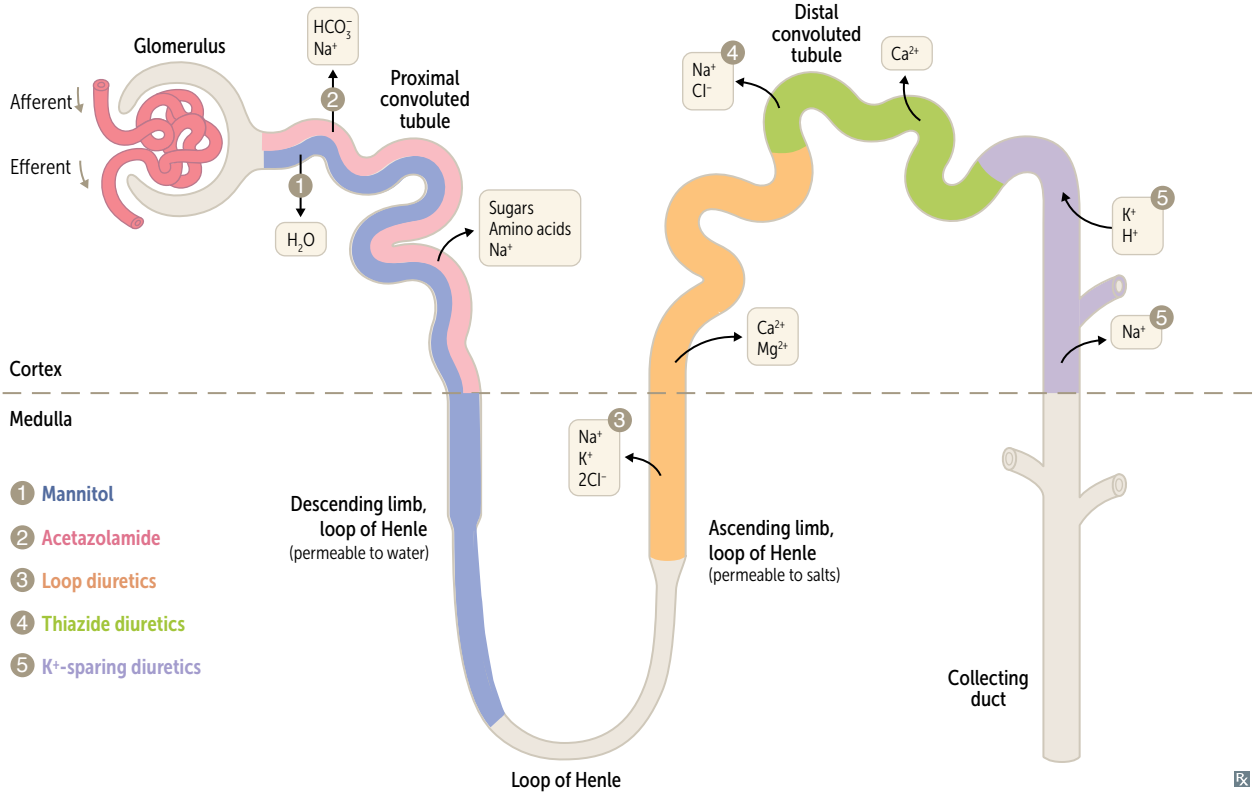
art revised for 2022 2nd pass

art revised for 2024 3rd pass

▸ RENAL—PHARMACOLOGY

art revised for 2024 1st pass

Diuretics: site of action



Diuretics: effects on electrolyte excretion

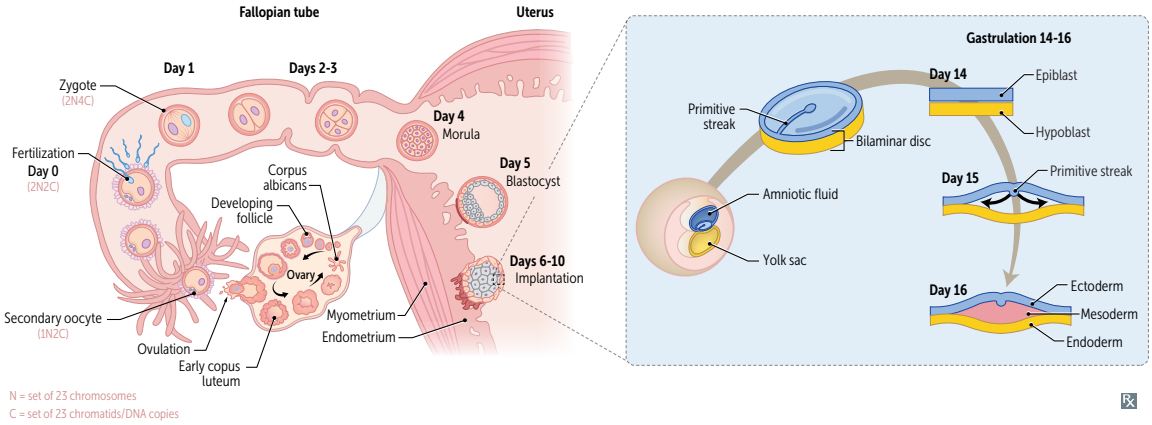
New Facts inserted for 2024 1st pass

	Na^+	HCO_3^-	K^+	Cl^-	Ca^{2+}	Mg^{2+}	H^+
Carbonic anhydrase inhibitors	↑	↑↑	↑↑	–/↑	–	–	↓
Loop diuretics	↑↑	↑	↑↑	↑↑	↑↑	↑↑	↑
Thiazide diuretics	↑	↑	↑↑	↑	↓↓	–/↑	↑
K^+-sparing diuretics	↑	–/↓	↓	↑	↓/–	↓/–	↓
Blood pH	↓ (**acidemia**): carbonic anhydrase inhibitors: ↓ HCO_3^- reabsorption. K^+ sparing: aldosterone blockade prevents K^+ secretion and H^+ secretion. Additionally, hyperkalemia leads to K^+ entering all cells (via H^+/K^+ exchanger) in exchange for H^+ exiting cells. ↑ (**alkalemia**): loop diuretics and thiazides cause alkalemia through several mechanisms: ▪ Volume contraction → ↑ AT II → ↑ Na^+/H^+ exchange in PCT → ↑ HCO_3^- reabsorption ("contraction alkalosis") ▪ K^+ loss leads to K^+ exiting all cells (via H^+/K^+ exchanger) in exchange for H^+ entering cells ▪ In low K^+ state, H^+ (rather than K^+) is exchanged for Na^+ in cortical collecting tubule → alkalosis and "paradoxical aciduria"						

Fact moved for 2024 1st pass

▸ REPRODUCTIVE—EMBRYOLOGY

Early embryonic development



new image for 2024 1st pass

art revised for 2024 2nd pass

Week 1	hCG secretion begins around the time of blastocyst implantation. Blastocyst "sticks" on day six.
Week 2	Formation of bilaminar embryonic disc; two layers = epiblast, hypoblast.
Week 3	Formation of trilaminar embryonic disc via gastrulation (epiblast cell invagination through primitive streak); three layers = endoderm, mesoderm, ectoderm. Notochord arises from midline mesoderm and induces overlying ectoderm (via SHH) to become neural plate, which gives rise to neural tube via neurulation.
Week 4	Heart begins to beat (four chambers). Cardiac activity visible by transvaginal ultrasound. Upper and lower limb buds begin to form (four limbs).
Week 8	Genitalia have male/female characteristics (pronounce "geneightalia").

Descent of testes and ovaries

	DESCRIPTION	MALE REMNANT	FEMALE REMNANT
Gubernaculum	Band of fibrous tissue	Anchors testes within scrotum	Ovarian ligament + round ligament of uterus
Processus vaginalis	Evagination of peritoneum	Forms tunica vaginalis Persistent patent processus vaginalis → hydrocele	Obliterated

▸ REPRODUCTIVE—ANATOMY

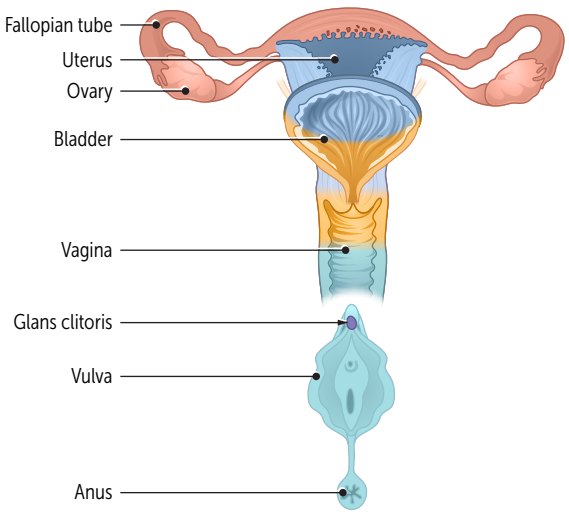
Drainage of reproductive organs

Venous drainage

Right ovary/testis → right gonadal vein → IVC.
Left ovary/testis → left gonadal vein → left renal vein → IVC (takes the longer way).
Left testicular vein enters left renal vein at 90° angle → flow is less laminar on the left than on the right → left venous pressure > right venous pressure → varicocele is more common on the left.

new fact for 2024 1st pass

Lymphatic drainage



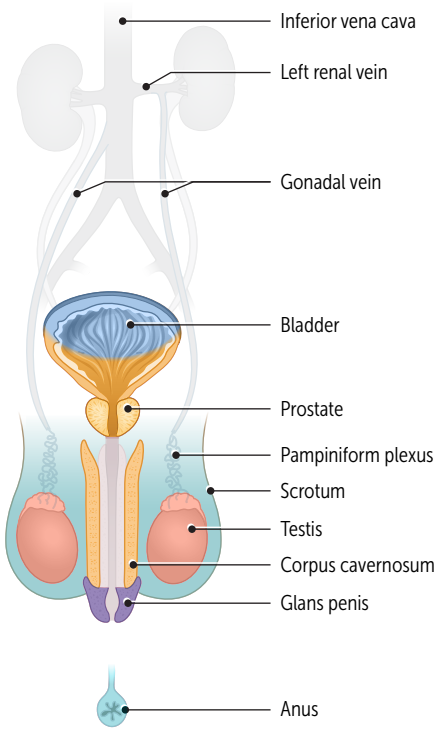
PARA-AORTIC
♀ Ovaries, fallopian tubes, uterine fundus
♂ Testes

EXTERNAL ILIAC
Superior part of bladder
♀ Body of uterus, cervix

INTERNAL ILIAC
Inferior part of bladder
♀ Cervix, proximal vagina
♂ Prostate, corpus cavernosum

SUPERFICIAL INGUINAL
Distal anal canal
♀ Distal vagina, vulva
♂ Scrotum

DEEP INGUINAL
♀ Glans clitoris
♂ Glans penis



new image for 2024 1st pass

art revised for 2024 2nd pass

Progestins	Levonorgestrel, medroxyprogesterone, etonogestrel, norethindrone, megestrol.
MECHANISM	Bind progesterone receptors, ↓ growth and ↑ vascularization of endometrium, thicken cervical mucus.
CLINICAL USE	Contraception (forms include pill, intrauterine device, implant, depot injection), endometrial cancer, abnormal uterine bleeding. Progestin challenge: presence of bleeding upon withdrawal of progestins excludes anatomic defects (eg, Asherman syndrome) and chronic anovulation without estrogen.

Antiprogestins	Mifepristone, ulipristal.
MECHANISM	Competitive inhibitors of progestins at progesterone receptors.
CLINICAL USE	Termination of pregnancy (mifepristone with misoprostol); emergency contraception (ulipristal).

Contraception Birth control.

	METHOD	MECHANISM	NOTES
Hormonal	Estrogen combined with progestins Progestin-only	Prevent ovulation by ↓ GnRH → ↓ LH/FSH → no estrogen surge → no LH surge Progestins also thicken cervical mucus (↓ sperm entry) and thin endometrium (less suitable for implantation)	Forms include pill (OCPs), transdermal patch, vaginal ring
Intrauterine device	Copper IUD (hormone free)	Copper IUD causes local inflammation that is toxic to sperm and ova preventing fertilization and implantation	IUDs ↑ risk for abnormal uterine bleeding; insertion contraindicated in patients with active STI
	Progesterone IUD	Same as progestins	
Surgical	Males—vasectomy Females—tubal ligation	No sperm in ejaculate Sperm cannot reach ova	Irreversible

New-fact for 2024 1st pass

Tocolytics	Medications that relax the uterus; include terbutaline (β_2-agonist action), nifedipine (Ca^{2+} channel blocker), indomethacin (NSAID). Used to ↓ contraction frequency in preterm labor and allow time for administration of glucocorticoids (to promote fetal lung maturity) or transfer to appropriate medical center with obstetrical care.