Study Guide 4

This guide covers chapters 7 and 8, the physiology that reasons from a few relationships rather than lists. Chapter 7 builds the excitable membrane, the synapse, the reflex arc and the muscle. Chapter 8 makes the heart, lungs and kidneys work as one team. The board rewards students who can run the equations in their head: Nernst, flow equals pressure over resistance, Starling forces, clearance and Henderson Hasselbalch. Every number in the worked cases has been checked. Work one chapter at a time, cover the steps and do each calculation yourself before reading on, then finish with the ten board-style items. The cram sheet at the end puts both chapters on one page.

WHAT THIS GUIDE COVERS

- Chapter 7 · ion gradients, the Nernst and Goldman ideas, the action potential and refractory periods, conduction and myelin, fiber classes, chemical synapses, receptors and G proteins, the neuromuscular junction, transmitters, receptors and adaptation, pain and gate control, spindles, tendon organs and reflexes, UMN and LMN signs, excitation contraction coupling, the cross bridge cycle, length tension, motor units and fiber types, smooth and cardiac muscle, and energy systems.
- Chapter 8 · pacemaker and ventricular action potentials, the ECG, the cardiac cycle and pressure volume loop, heart sounds, cardiac output and Frank Starling, Poiseuille and Starling forces, edema, local flow control, the baroreceptor reflex and RAAS, lung mechanics, volumes and spirometry, V/Q and hypoxemia, oxygen and CO2 transport, control of breathing, GFR and clearance, the nephron, the countercurrent mechanism, ADH and aldosterone, micturition, fluids, electrolytes and acid base.

CHAPTER 7

Neurophysiology and Muscle Physiology

Read it in full: **Chapter 7 in the review book**. Practice it: **the practice exams** (Exam 4 covers this chapter) and **the flashcards**.

What to know

Sodium, chloride and calcium are high outside the cell; potassium is high inside. The **Na+/K+ ATPase** (3 Na+ out, 2 K+ in per ATP) builds and maintains these gradients. Each ion has an equilibrium potential where its electrical and chemical forces balance: E = (61.5/z) log (outside/inside). E(K) is about -90 mV and E(Na) about +60 mV. Because extracellular potassium is low, small changes in it move E(K) a lot: **hyperkalemia depolarizes** cells, hypokalemia hyperpolarizes them.

At rest, potassium leak channels dominate permeability, so the resting potential sits near E(K) (about -70 mV in neurons, about -90 mV in skeletal and ventricular muscle). The rule: whichever ion has the highest permeability drags the membrane toward its own equilibrium potential. Potassium contributes most to the resting potential; the pump maintains the gradients. Changing extracellular potassium changes the resting potential; changing extracellular sodium changes the height of the spike.

The **action potential** fires when the axon initial segment reaches threshold (about -55 mV). Voltage gated Na+ channels open by positive feedback (upstroke toward E(Na), peak about +30 to +40 mV), then inactivation gates close them, and delayed K+ channels repolarize the membrane and cause the afterhyperpolarization. The **absolute refractory period** (sodium channels inactivated) limits firing rate and forces one-way travel; in the relative refractory period a stronger stimulus can fire the cell. Slow depolarization can fail by accommodation.

Conduction speed rises with diameter and myelin. Myelin (oligodendrocytes in the CNS, Schwann cells in the PNS) concentrates Na+ channels at the nodes, so the impulse jumps node to node (**saltatory conduction**). Demyelination (multiple sclerosis, Guillain Barre) slows or blocks conduction, worse with heat. Fiber classes: A alpha (Ia, Ib, alpha motor) 70 to 120 m/s; A beta (II) touch and vibration; A gamma to spindles; A delta (III) sharp pain and cold; B preganglionic autonomic; C (IV) burning pain and warmth, 0.5 to 2 m/s. **Local anesthetics block small fibers first; pressure and ischemia block large fibers first.**

At a chemical synapse the spike opens voltage gated Ca2+ channels, calcium binds synaptotagmin and the **SNARE** proteins (synaptobrevin, syntaxin, SNAP 25) fuse vesicles to release transmitter

in quanta. Botulinum toxin cleaves SNAREs at peripheral cholinergic terminals (descending flaccid paralysis); tetanus toxin travels to the cord and blocks glycine and GABA release from inhibitory interneurons (spastic paralysis, trismus). Lambert Eaton antibodies target presynaptic Ca2+ channels; weakness improves with repetition. Myasthenia gravis antibodies target the nicotinic receptor; weakness worsens with use.

Ionotropic receptors are fast channels: nicotinic, AMPA, NMDA (calcium, blocked by Mg2+ until depolarized, needs glycine), GABA-A and glycine (chloride). Metabotropic receptors act through G proteins: **Gs** raises cAMP (beta 1, beta 2, D1, H2, V2), **Gi** lowers it (alpha 2, M2, D2, GABA-B, mu opioid), **Gq** activates phospholipase C, IP3 and calcium (alpha 1, M1, M3, H1, V1, AT1). Neurons integrate EPSPs and IPSPs by temporal and spatial summation at the initial segment. Glycine is the main spinal inhibitory transmitter (strychnine blocks it); GABA is the main brain inhibitor.

Receptors adapt slowly (Merkel, Ruffini, spindles, tendon organs, nociceptors) or quickly (Meissner, Pacinian). A delta fibers carry first, sharp pain to laminae I and V; C fibers carry second, burning pain to laminae I and II with substance P. Prostaglandins sensitize nociceptors (the basis of NSAID analgesia). Repetitive C fiber input causes wind up and **central sensitization**: hyperalgesia and allodynia. In **gate control**, large A beta fibers excite inhibitory interneurons in the substantia gelatinosa and close the gate; small fibers open it. Descending inhibition runs periaqueductal gray to raphe magnus (serotonin) and locus coeruleus to enkephalin interneurons.

The **muscle spindle** lies in parallel and senses length and its rate of change through Ia (dynamic) and II (static) afferents; gamma motor neurons keep it taut (alpha gamma coactivation). The **Golgi tendon organ** lies in series and senses tension through Ib afferents. The stretch reflex is the only monosynaptic reflex (Ia to the agonist alpha motor neuron, with reciprocal inhibition of the antagonist). The Ib pathway causes autogenic inhibition. Withdrawal and crossed extensor reflexes are polysynaptic. Renshaw cells give recurrent glycinergic inhibition. Jendrassik raises gamma drive.

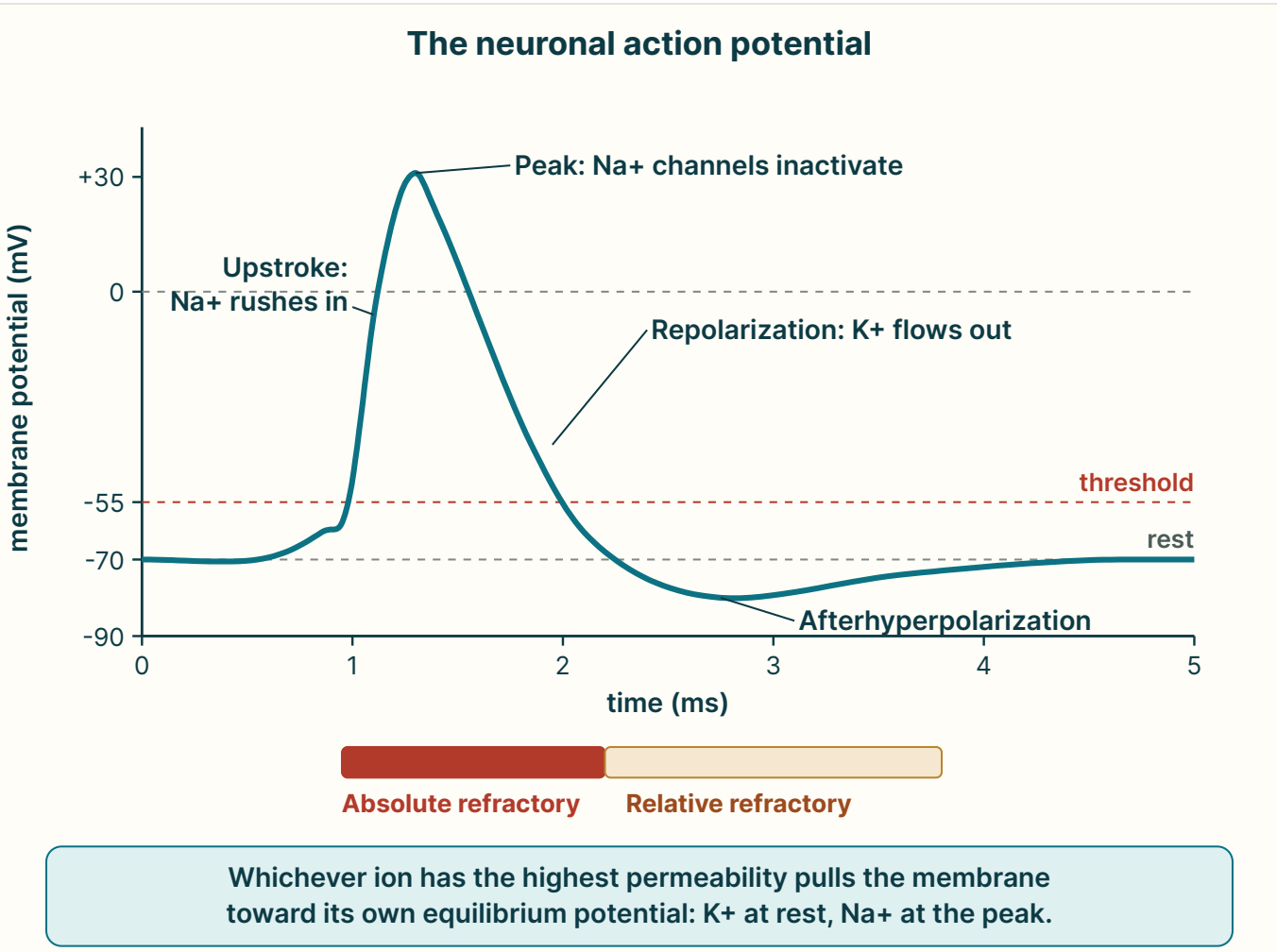
UMN lesions: spasticity, hyperreflexia, clonus, Babinski, lost superficial reflexes, little atrophy. LMN lesions: flaccidity, areflexia, early atrophy, fasciculations. ALS gives both with no sensory

loss. Acute UMN lesions start flaccid (spinal shock). In skeletal muscle, the sarcomere runs Z line to Z line; the **A band never changes length** while the I band and H zone shorten (sliding filaments). Troponin C binds calcium, troponin I inhibits, troponin T binds tropomyosin; titin gives passive tension; absent dystrophin is Duchenne.

Excitation contraction coupling: the action potential runs down T tubules, the **DHPR voltage sensor** mechanically opens **RyR1**, SR calcium binds troponin C and cross bridges cycle. ATP binding detaches myosin, hydrolysis cocks the head, Pi release powers the stroke; no ATP gives rigor. SERCA pumps calcium back (relaxation needs ATP too). Malignant hyperthermia is a RyR1 defect treated with dantrolene. Twitches summate into tetanus. Active tension peaks at optimal overlap (about 2.0 to 2.2 µm); faster shortening gives less force; eccentric force exceeds isometric.

A motor unit is one alpha motor neuron and its fibers. Force grows by recruitment and rate coding, recruited in size order (**Henneman size principle**: small, slow, fatigue resistant type I units first). Type I fibers are red and oxidative (soleus, multifidus); type IIx are white, glycolytic and fatigable. The motor neuron sets fiber type. Smooth muscle has no troponin: calcium binds **calmodulin**, which activates **MLCK**; MLCP relaxes; Rho kinase sensitizes; cAMP and cGMP relax; the latch state holds tone cheaply. Cardiac muscle uses **calcium induced calcium release** (RyR2) and cannot tetanize because its plateau keeps it refractory until relaxation.

ATP comes from three overlapping systems: **creatine phosphate** (creatine kinase) for about 10 seconds, anaerobic glycolysis (2 ATP per glucose, lactate) for about 10 seconds to 2 minutes, and oxidative phosphorylation (about 30 to 32 ATP per glucose) for anything longer. Creatinine from creatine phosphate reflects muscle mass. Lactate is a fuel, not the cause of fatigue; inorganic phosphate is a major peripheral contributor. Cola colored urine with muscle pain after an unaccustomed workout is rhabdomyolysis: an emergency referral.



Permeability decides the voltage. The spike is a brief switch from potassium to sodium dominance and back; sodium channel inactivation creates the absolute refractory period.

LOCK THESE IN

- Resting potential sits near E(K) because K+ permeability dominates; hyperkalemia depolarizes.
- Upstroke is Na+ in; inactivation plus delayed K+ out ends it; inactivation explains the absolute refractory period.
- Local anesthetics block C fibers first; pressure blocks large myelinated fibers first.
- Botulinum: flaccid (no ACh release). Tetanus: spastic (no glycine or GABA release).
- Gs raises cAMP, Gi lowers it, Gq raises IP3 and calcium.
- Spindle: parallel, length, Ia. Tendon organ: series, tension, Ib. Stretch reflex is monosynaptic.
- Skeletal: DHPR to RyR1 to troponin C. Cardiac: calcium induced calcium release. Smooth: calmodulin and MLCK.
- Phosphagen about 10 s, glycolysis to about 2 min, oxidative beyond.

Nerve fiber classes

Class (sensory)	Speed and myelin	Carries
A alpha (Ia, Ib)	70 to 120 m/s, heavy myelin	Spindle primary, tendon organ, alpha motor
A beta (II)	30 to 70 m/s, myelinated	Touch, pressure, vibration
A gamma	15 to 30 m/s	Gamma motor to spindles
A delta (III)	5 to 30 m/s, thin myelin	Sharp pain, cold
B	3 to 15 m/s, thin myelin	Preganglionic autonomic
C (IV)	0.5 to 2 m/s, unmyelinated	Burning pain, warmth, itch, postganglionic

Skeletal, cardiac and smooth muscle

Feature	Skeletal / Cardiac	Smooth
Striations and troponin	Yes / yes	No; calmodulin
Calcium source	SR via DHPR and RyR1 / trigger entry plus SR (RyR2)	Extracellular plus SR (IP3R)
Needs extracellular calcium	No / yes	Yes, heavily
Gap junctions	No / intercalated discs	Single unit yes
Initiation	Motor neuron / SA node	Myogenic and neural
Tetanus	Yes / no	Tonic contraction, latch
Grading force	Recruitment, rate coding / contractility, preload	Calcium and sensitization

Worked cases

WORKED CASE 1 · POTASSIUM AND THE RESTING POTENTIAL

Greta, 66, with kidney failure, has a serum potassium of 6.8 mEq/L. Take intracellular potassium as 145 mEq/L and a normal extracellular potassium as 5 mEq/L.

STEP 1 · WRITE NERNST FOR K+

E(K) = 61.5 log (outside / inside).

STEP 2 · NORMAL VALUE

With 5 outside and 145 inside: 61.5 × log (5/145) = about -90 mV.

STEP 3 · HYPERKALEMIC VALUE

With 6.8 outside and 145 inside: 61.5 × log (6.8/145) = about -82 mV, about 8 mV less negative.

STEP 4 · APPLY THE PRINCIPLE

The resting potential follows E(K), so cells depolarize toward threshold.

STEP 5 · EXPLAIN THE WEAKNESS

Sustained depolarization keeps sodium inactivation gates shut, so fewer channels are available and action potentials fail: weakness and dangerous cardiac changes. Refer urgently.

WORKED CASE 2 · WHY THE DENTAL BLOCK NUMBS BEFORE IT PARALYZES

During a nerve block, a patient loses pinprick first, then touch, and keeps motor power longest.

STEP 1 · RECALL THE RULE

Local anesthetics block small fibers first.

STEP 2 · ORDER THE FIBERS

C and A delta (pain) are smallest, then A beta (touch), then A alpha (motor and spindle afferents).

STEP 3 · GIVE THE REASON

Thin fibers need only a short blocked length of nerve to stop conduction; large fibers have long internodes and need a longer blocked segment.

CONTRAST

Pressure (sitting on the sciatic nerve) does the opposite: large fibers fail first, so position sense and power go before pain.

WORKED CASE 3 · READING REFLEXES AS UMN OR LMN

Ivan, 59, has neck pain, clumsy hands, brisk knee and ankle reflexes, three beats of ankle clonus, a positive Hoffmann sign and an upgoing toe on the right.

STEP 1 · SORT EACH SIGN

Hyperreflexia, clonus, Hoffmann and Babinski are all upper motor neuron signs.

STEP 2 · PLACE THE LESION

UMN signs in the legs and hands with neck pain point to the cervical cord above the levels involved.

STEP 3 · NAME THE LIKELY CAUSE

Cervical spondylotic myelopathy.

STEP 4 · ACT

No high velocity cervical manipulation. Refer for MRI and surgical opinion.

TRAPS PEOPLE FALL INTO

- **Contributes versus maintains.** Potassium permeability contributes most to the resting potential; the pump maintains the gradients.
- **The pump and one spike.** A single action potential does not need the pump; poisoned axons still fire thousands of spikes.
- **Myasthenia versus Lambert Eaton.** Myasthenia worsens with use (postsynaptic); Lambert Eaton improves with repetition (presynaptic).
- **Which band stays the same.** The A band. The I band and H zone shorten.
- **Size principle.** Small motor neurons fire first and supply slow, fatigue-resistant fibers.
- **Lactate.** It is a fuel that heart and type I fibers burn, not the main cause of fatigue.

Chapter 7 self-test · ten board-style items

1. Which change most directly makes a resting neuron less negative inside?

A. A rise in intracellular potassium

B. A rise in extracellular sodium

C. A fall in extracellular chloride

D. A rise in extracellular potassium

D. A rise in extracellular potassium Resting potential follows E(K). Raising outside potassium makes E(K) less negative and depolarizes the cell.

2. What ends the upstroke of a neuronal action potential?

A. Chloride entry through leak channels
B. Na+ channel inactivation gates closing
C. The Na+/K+ ATPase speeding up
D. Calcium entry through L-type channels

B. Na+ channel inactivation gates closing Inactivation (h) gates close sodium channels within about a millisecond; delayed K+ channels then repolarize.

3. Which fiber type is blocked first by a local anesthetic?

A. C fibers
B. A beta
C. A alpha
D. A gamma

A. C fibers Small, unmyelinated or thinly myelinated fibers are most sensitive to local anesthetics, so pain goes first.

4. A child after a deep puncture wound develops trismus and spasms with back arching. Which release is blocked?

A. Norepinephrine from sympathetic nerves
B. Glutamate from primary afferents
C. Glycine and GABA from spinal interneurons
D. Acetylcholine at the motor end plate

C. Glycine and GABA from spinal interneurons Tetanus toxin travels retrograde to the cord and cleaves synaptobrevin in inhibitory interneurons, removing inhibition of motor neurons.

5. Which receptor is coupled to Gq?

A. Alpha 2 adrenergic
B. M2 muscarinic
C. Alpha 1 adrenergic
D. Beta 2 adrenergic

C. Alpha 1 adrenergic Alpha 1, M1, M3, H1, V1 and AT1 use Gq and phospholipase C. Beta 2 is Gs; M2 and alpha 2 are Gi.

6. Rubbing a bumped elbow eases the pain. Which mechanism explains this best?

A. C fiber input releasing more substance P
B. Renshaw cells inhibiting motor neurons
C. Prostaglandins sensitizing nociceptors
D. A beta input closing the dorsal horn gate

D. A beta input closing the dorsal horn gate Large fiber input excites inhibitory interneurons in the substantia gelatinosa, reducing nociceptive transmission to projection cells.

7. Which receptor lies in series with muscle fibers and signals tension?

A. Golgi tendon organ via Ib fibers
B. Muscle spindle via group II fibers
C. Pacinian corpuscle via A beta
D. Muscle spindle via Ia fibers

A. Golgi tendon organ via Ib fibers The tendon organ sits at the musculotendinous junction, in series, and its Ib afferents signal tension.

8. Which sarcomere region keeps the same length during contraction?

A. I band
B. A band
C. Z to Z distance
D. H zone

B. A band The A band equals thick filament length; filaments slide, so only the I band and H zone shorten.

9. Why do calcium channel blockers affect the heart but not the biceps?

A. Cardiac muscle has no SR at all
B. Skeletal muscle lacks troponin C
C. The biceps has no L-type channels
D. Skeletal muscle needs no calcium entry

D. Skeletal muscle needs no calcium entry Skeletal DHPRs act as voltage sensors coupled mechanically to RyR1; cardiac muscle needs trigger calcium entry for calcium induced calcium release.

10. A sprinter's first 8 seconds of maximal effort rely mainly on which system?

A. Creatine phosphate and its kinase
B. Oxidative phosphorylation of fat
C. Beta oxidation of fatty acids
D. Anaerobic glycolysis to lactate

A. Creatine phosphate and its kinase The phosphagen system regenerates ATP fastest and lasts about 10 seconds; glycolysis then dominates up to about 2 minutes.

CHAPTER 8

Cardiovascular, Respiratory and Renal Physiology

Read it in full: **Chapter 8 in the review book**. Practice it: **the practice exams** (Exam 4 covers this chapter) and **the flashcards**.

What to know

SA node cells pace the heart with the **funny current** (If, Na+ through HCN channels), then T-type and L-type calcium. Their upstroke is calcium (slow). Ventricular cells have a fast Na+ upstroke, a calcium plateau (phase 2) and potassium repolarization. Sympathetic beta 1 stimulation steepens phase 4; vagal M2 flattens it. The AV node delays about 0.1 s; Purkinje fibers conduct fastest. ECG: P atrial depolarization, PR 0.12 to 0.20 s, QRS under 0.12 s, T ventricular repolarization, QT the whole ventricular action potential. Heart rate is about 300 divided by large boxes between R waves.

In the cardiac cycle, valves move only with pressure gradients. **S1** is mitral and tricuspid closure at the start of isovolumetric contraction; **S2** is aortic and pulmonic closure at the start of isovolumetric relaxation. The pressure volume loop runs counterclockwise; its width is stroke volume and its area is stroke work. Typical values: EDV 120 to 140 mL, ESV 50 to 70 mL, SV about 70 mL, EF 55 to 70%. S3 in older adults suggests volume overload; S4 is atrial contraction against a stiff ventricle and is absent in atrial fibrillation.

Cardiac output = heart rate × stroke volume, about 5 L/min at rest. Preload (EDV) moves you along a Starling curve; contractility moves you to a new curve; afterload is inversely related to stroke volume. The **Frank Starling** law says more filling stretches sarcomeres toward optimal length and raises stroke volume, matching the two ventricles. Wall stress follows Laplace (pressure × radius / 2 × thickness). EF is preserved in diastolic failure and reduced in systolic failure.

Flow = pressure difference / resistance, so **MAP = CO × TPR** (MAP is about diastolic plus a third of pulse pressure). By Poiseuille, resistance varies with $1/r^4$: halve the radius and resistance rises 16 fold. Arterioles are the resistance vessels; veins are the capacitance vessels holding 60 to 70% of blood. Velocity is slowest in capillaries. Turbulence makes murmurs, bruits and Korotkoff sounds. Hypoxia dilates systemic arterioles but **constricts pulmonary arterioles**.

Capillary exchange follows **Starling forces**: capillary hydrostatic pressure pushes fluid out, plasma oncotic pressure (albumin, about 25 mm Hg) pulls it in; filtration wins at the arterial end, absorption at the venous end, and lymphatics return the small excess. Edema comes from raised

hydrostatic pressure (heart failure, DVT), low albumin (nephrotic syndrome, cirrhosis), leaky capillaries (inflammation, burns) or blocked lymph (nonpitting lymphedema). Autoregulation keeps brain and kidney flow steady from about 60 to 150 mm Hg.

The **baroreceptor reflex** acts in seconds: carotid sinus (CN IX) and aortic arch (CN X) feed the nucleus tractus solitarius; a fall in pressure lowers vagal tone and raises sympathetic outflow. It resets within days, so it does not set long-term pressure. **RAAS** does: renin (low perfusion, low macula densa NaCl, beta 1) leads to angiotensin II (vasoconstriction, efferent arteriole constriction, aldosterone, proximal Na+ reabsorption, ADH, thirst). ACE also breaks down bradykinin (ACE inhibitor cough). ANP and BNP oppose the system; BNP marks heart failure. Orthostatic hypotension is a fall of 20 systolic or 10 diastolic on standing.

Inspiration lowers alveolar pressure below atmospheric. Intrapleural pressure is negative because lung recoil and chest wall spring oppose each other; a pneumothorax collapses the lung. Compliance falls in fibrosis and rises in emphysema. **Surfactant** (type II pneumocytes) lowers surface tension and stabilizes small alveoli. Volumes: TV 500, IRV 3,000, ERV about 1,200, RV about 1,200 mL; FRC = ERV + RV; VC = IRV + TV + ERV; TLC = VC + RV. **Obstructive**: FEV1/FVC below 0.70 with high TLC and RV. **Restrictive**: normal or high ratio with low TLC (fibrosis, kyphoscoliosis, ankylosing spondylitis).

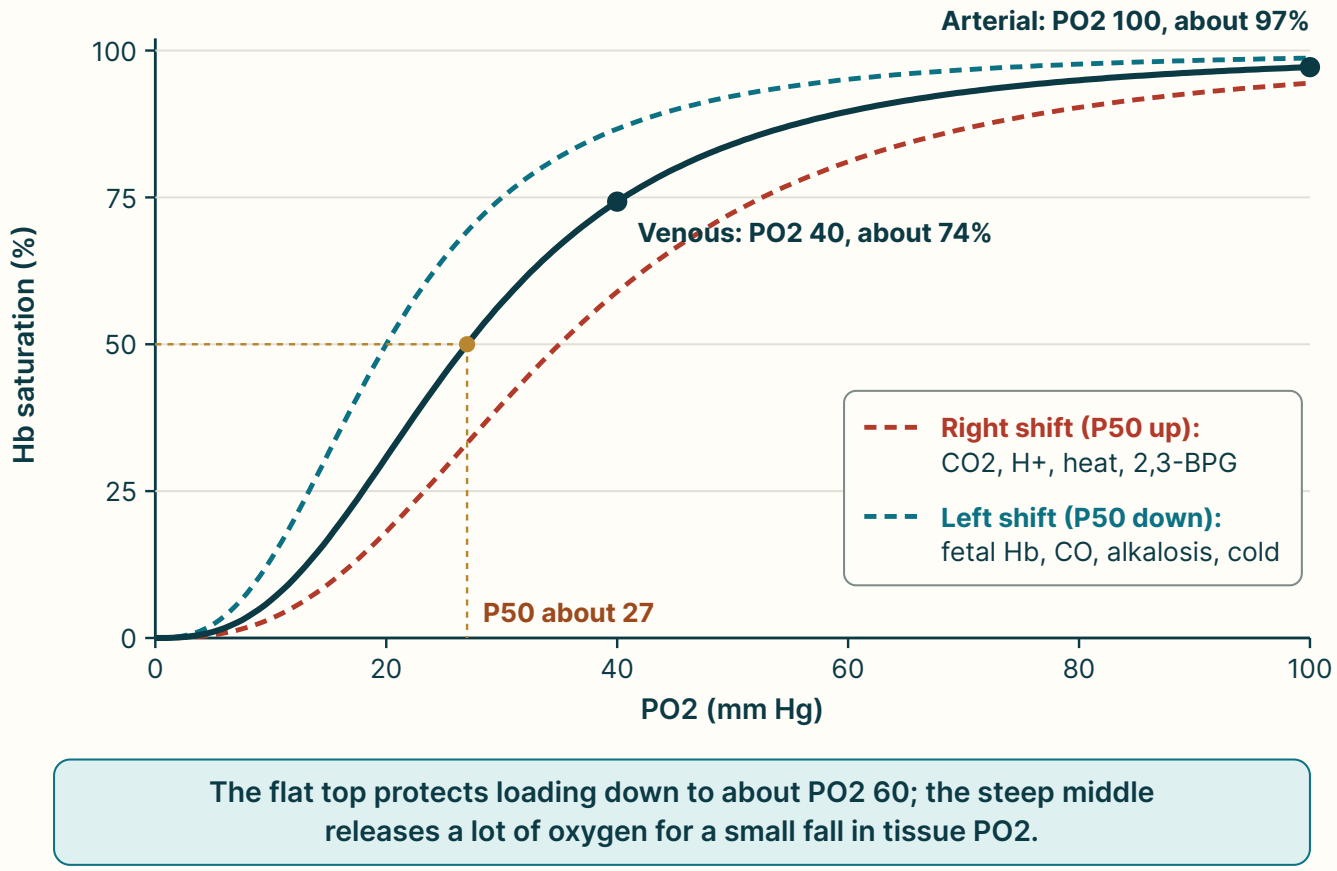
Alveolar ventilation = (TV minus dead space) × rate; PaCO2 varies inversely with it. The apex has high V/Q, the base low V/Q. **Shunt** (V/Q = 0) gives hypoxemia that does not correct with oxygen; dead space (pulmonary embolism) wastes ventilation. Hypoventilation and altitude keep a normal A-a gradient; V/Q mismatch, shunt and diffusion limits widen it. PAO2 = 0.21 (760 - 47) - 40/0.8, about 100 mm Hg. CO2 diffuses about 20 times faster than O2.

Oxygen content = 1.34 × Hb × SaO2 + 0.003 × PaO2, about 20 mL/dL. The **dissociation curve** is sigmoidal: about 98% saturated at PO2 100, 75% at 40, P50 about 27. A **right shift** (more CO2, H+, heat, 2,3-BPG, exercise) unloads oxygen; a left shift (fetal Hb, CO, alkalosis, cold) holds it. Carbon monoxide lowers content and shifts the curve left while PaO2 and pulse oximetry can look normal. CO2 travels about 70% as bicarbonate (chloride shift); the Haldane effect releases CO2 in the lungs. Central chemoreceptors respond to CSF pH (PaCO2) and give most resting drive; the carotid bodies respond to hypoxemia below about 60 mm Hg.

The kidneys get 20 to 25% of cardiac output. GFR (about 125 mL/min, 180 L/day) follows Starling forces; filtration fraction is about 20%. Afferent constriction lowers both RPF and GFR; **efferent constriction (angiotensin II) lowers RPF but raises GFR**; ACE inhibitors and ARBs do the reverse. NSAIDs plus ACE inhibitor or ARB plus diuretic is the triple whammy for acute kidney injury. Clearance = **UV/P**: inulin equals GFR, creatinine estimates it (slightly secreted), PAH estimates renal plasma flow. Clearance below GFR means net reabsorption; above means net secretion.

The proximal tubule reabsorbs about two thirds of sodium and water, all glucose (SGLT2) and most bicarbonate. The thick ascending limb (NKCC2, loop diuretics) is impermeable to water and drives the **countercurrent multiplier**; urea recycling and the vasa recta (countercurrent exchanger) finish the medullary gradient up to about 1,200 mOsm/kg. The distal tubule uses NCC (thiazides) and PTH reabsorbs calcium. Principal cells answer to aldosterone (ENaC, K+ secretion) and ADH (aquaporin 2). Glucose appears in urine above about 200 mg/dL. Central DI responds to desmopressin; nephrogenic DI does not. Micturition: S2 to S4 parasympathetic voids, T10 to L2 sympathetic stores, pudendal controls the external sphincter; a cauda equina lesion gives retention with overflow.

Total body water is 60% of weight (ICF 40, ECF 20). Sodium content sets ECF volume; sodium concentration reflects water balance. Potassium shifts into cells with insulin, beta 2 agonists and alkalosis; hyperkalemia peaks T waves and widens QRS. Alkalosis lowers **ionized calcium** (tetany with hyperventilation). PTH raises calcium and lowers phosphate. pH = 6.1 + log (HCO3- / 0.03 PCO2): pH depends on the ratio. Work every gas in order: pH, primary change, anion gap (Na+ minus Cl- plus HCO3-, normal 8 to 12) for metabolic acidosis, then compensation (**Winters: PCO2 = 1.5 × HCO3- + 8 ± 2**). MUDPILES raise the gap; diarrhea and RTA keep it normal.



Shape and shift decide delivery. Right shifts unload oxygen in active tissue; left shifts hold it, which is why carbon monoxide starves tissue even when PaO2 is normal.

LOCK THESE IN

- CO = HR × SV; preload moves along a Starling curve, contractility shifts the curve, afterload lowers SV.
- MAP = CO × TPR; resistance rises 16 fold when the radius halves.
- Edema: high hydrostatic pressure, low albumin, leaky capillaries or blocked lymph.
- Baroreflex is fast (CN IX and X to NTS); RAAS sets long-term volume and pressure.
- Obstructive: low FEV1/FVC, high TLC. Restrictive: normal ratio, low TLC.
- Shunt does not correct with oxygen; right shift unloads oxygen; CO lowers content with normal PaO2.
- Inulin = GFR, creatinine estimates GFR, PAH = renal plasma flow; clearance = UV/P.
- Anion gap = Na+ minus (Cl- + HCO3-); Winters: PCO2 = 1.5 × HCO3- + 8 ± 2.

Equations to run in your head

Relationship	Equation	Normal or example
Cardiac output	CO = HR × SV	70 × 70 mL = about 5 L/min
Ejection fraction	EF = SV / EDV	55 to 70%
Fick	CO = VO2 / (a-v O2 difference)	250 / 50 mL per L = 5 L/min
Mean arterial pressure	MAP = DBP + PP/3	120/80 gives about 93 mm Hg
Poiseuille	$R = 8\eta L / \pi r^4$	Half the radius, 16 times the resistance
Alveolar gas	PAO2 = 0.21 (760 - 47) - PaCO2/0.8	About 100 mm Hg
O2 content	1.34 × Hb × SaO2 + 0.003 × PaO2	About 20 mL/dL
Clearance	C = UV / P	Inulin about 125 mL/min
Henderson Hasselbalch	pH = 6.1 + log (HCO3- / 0.03 PCO2)	24 / 1.2 = 20, pH 7.4
Anion gap	Na+ - (Cl- + HCO3-)	8 to 12 mEq/L

Acid base compensation

Disorder	Primary change	Expected compensation
Metabolic acidosis	HCO3- down	PCO2 = 1.5 × HCO3- + 8 ± 2
Metabolic alkalosis	HCO3- up	PCO2 up about 0.7 per 1 mEq/L HCO3-
Respiratory acidosis	PCO2 up	HCO3- up 1 (acute) or 3.5 to 4 (chronic) per 10
Respiratory alkalosis	PCO2 down	HCO3- down 2 (acute) or 4 to 5 (chronic) per 10
High gap acidosis	Added unmeasured acid	MUDPILES: ketoacidosis, lactate, uremia, toxins
Normal gap acidosis	HCO3- lost, Cl- rises	Diarrhea, RTA, acetazolamide

Worked cases

WORKED CASE 1 · CARDIAC OUTPUT FROM A PRESSURE VOLUME LOOP

An echo shows an end diastolic volume of 140 mL and an end systolic volume of 56 mL at a heart rate of 75.

STEP 1 · STROKE VOLUME

SV = EDV - ESV = 140 - 56 = 84 mL.

STEP 2 · EJECTION FRACTION

EF = SV / EDV = 84 / 140 = 0.60, or 60%. Normal.

STEP 3 · CARDIAC OUTPUT

CO = HR × SV = 75 × 84 mL = 6,300 mL, or 6.3 L/min.

STEP 4 · READ THE LOOP

The loop is 84 mL wide. More preload would widen it to the right; more contractility would move its left edge left.

WORKED CASE 2 · READING AN ARTERIAL BLOOD GAS

A patient with poorly controlled diabetes has pH 7.31, PCO2 29 mm Hg, HCO3- 14 mEq/L, Na+ 138 and Cl- 100.

STEP 1 · PH

7.31 is acidemia.

STEP 2 · PRIMARY CHANGE

HCO3- is low (normal 22 to 26), which explains the acidemia: metabolic acidosis. The pH checks out: 6.1 + log (14 / (0.03 × 29)) = 6.1 + log 16.1 = about 7.31.

STEP 3 · ANION GAP

138 - (100 + 14) = 24 mEq/L, high. Think MUDPILES; with diabetes, ketoacidosis leads the list.

STEP 4 · COMPENSATION

Winters: 1.5 × 14 + 8 = 29, range 27 to 31. Measured PCO2 is 29, so compensation is appropriate and no second disorder is present.

WORKED CASE 3 · CLEARANCE AND WHAT IT TELLS YOU

Over a timed collection, urine creatinine is 90 mg/dL, urine flow is 1.2 mL/min and plasma creatinine is 1.0 mg/dL.

STEP 1 · WRITE THE FORMULA

$C = U \times V / P$.

STEP 2 · PLUG IN

$C = 90 \times 1.2 / 1.0 = 108$ mL/min.

STEP 3 · INTERPRET

Creatinine clearance of 108 mL/min is close to a normal GFR of about 125 mL/min; it slightly overestimates GFR because creatinine is also secreted.

STEP 4 · REMEMBER MUSCLE MASS

The same serum creatinine means a lower GFR in a small older woman than in a muscular young man.

TRAPS PEOPLE FALL INTO

- **Preload versus contractility.** Preload moves along one Starling curve; contractility moves you to a different curve.
- **Efferent constriction.** It lowers renal plasma flow but raises GFR and the filtration fraction.
- **Shunt.** Its hypoxemia does not correct with 100% oxygen; V/Q mismatch does.
- **Carbon monoxide.** PaO2 and pulse oximetry can look normal while oxygen content and delivery fall.
- **Hypoxia in the lung.** It constricts pulmonary arterioles, the opposite of systemic arterioles.
- **Compensation.** It never fully returns pH to 7.40 and never overshoots; if it seems to, look for a second disorder.

Chapter 8 self-test · ten board-style items

1. During which phase are all four valves closed while ventricular pressure rises steeply?

A. Rapid ventricular ejection
B. Rapid ventricular filling
C. Isovolumetric relaxation
D. Isovolumetric contraction

D. Isovolumetric contraction After S1 the AV valves are shut and the semilunar valves have not yet opened, so pressure rises at constant volume.

2. A drug increases contractility without changing preload. What happens on the Starling diagram?

A. The patient moves to a higher curve
B. Stroke volume falls at every preload
C. The patient moves up the same curve
D. The curve flattens and shifts right

A. The patient moves to a higher curve Inotropes shift the Starling curve up and left. Changing preload moves the point along a single curve.

3. An arteriole's radius falls to half. By what factor does its resistance change?

A. It rises 2 fold
B. It rises 16 fold
C. It rises 8 fold
D. It rises 4 fold

B. It rises 16 fold Poiseuille: resistance varies with $1/r^4$, and $(1/2)^4 = 1/16$.

4. A patient with serum albumin of 1.8 g/dL has generalized pitting edema. Which Starling force explains it?

A. Obstructed lymphatic drainage
B. Raised capillary hydrostatic pressure
C. Reduced plasma oncotic pressure
D. Increased capillary permeability

C. Reduced plasma oncotic pressure Low albumin lowers the oncotic pull that holds fluid in capillaries, as in nephrotic syndrome.

5. Spirometry shows FEV1 50% of predicted, FVC 82% of predicted and a raised TLC. Which pattern?

A. Neuromuscular weakness
B. Restrictive
C. Normal
D. Obstructive

D. Obstructive The FEV1/FVC ratio is well below 0.70 and air trapping raises TLC, as in COPD or asthma.

6. Which change shifts the oxyhemoglobin curve to the left?

A. Raised 2,3-BPG
B. High fever
C. Carbon monoxide
D. Mild acidosis

C. Carbon monoxide CO, fetal hemoglobin, alkalosis and cold shift the curve left. The others shift it right and favor unloading.

7. A substance is filtered and then almost completely secreted in one pass. Its clearance estimates

A. renal plasma flow
B. free water
C. filtration fraction
D. filtration rate

A. renal plasma flow That describes PAH. Inulin, neither reabsorbed nor secreted, measures GFR instead.

8. Angiotensin II constricts the efferent arteriole. What happens to GFR and filtration fraction?

A. GFR falls and filtration fraction rises
B. GFR rises and filtration fraction rises
C. GFR falls and filtration fraction falls
D. GFR rises and filtration fraction falls

B. GFR rises and filtration fraction rises Efferent constriction raises glomerular hydrostatic pressure (GFR up) while plasma flow falls, so GFR / RPF rises.

9. After a head injury a patient passes 8 L a day of dilute urine that concentrates after desmopressin. What is the diagnosis?

A. Primary polydipsia alone
B. Central diabetes insipidus
C. Nephrogenic diabetes insipidus
D. SIADH from the head injury

B. Central diabetes insipidus ADH release has failed, but the collecting duct still responds to exogenous ADH, unlike nephrogenic DI.

10. A patient who has been vomiting has pH 7.50, HCO3- 36 mEq/L and PCO2 48 mm Hg. What is the interpretation?

A. Respiratory acidosis with renal compensation
B. Respiratory alkalosis, uncompensated
C. Metabolic alkalosis with respiratory alkalosis
D. Metabolic alkalosis with expected compensation

D. Metabolic alkalosis with expected compensation High pH with high HCO3- is metabolic alkalosis. Expected PCO2 rises about 0.7 per mEq/L: 40 + 0.7 × 12 = about 48, which matches.

ONE PAGE

Cram sheet

Both chapters on one page. Cover the right columns and rebuild each row from memory. If a row will not come back, go back to that chapter.

Neuromuscular physiology

Topic	Rule	Board hook
Gradients	Na+ out, K+ in; pump 3 out, 2 in	Hyperkalemia depolarizes
Action potential	Na+ in, inactivation, K+ out	ARP from Na+ inactivation
Conduction	Diameter and myelin; saltatory at nodes	Local anesthetic: small first
Synapse	Ca2+ in, SNAREs, quanta	Botulinum flaccid, tetanus spastic
G proteins	Gs cAMP up, Gi down, Gq IP3	NMDA needs glutamate, glycine, depolarization
Pain	A delta fast, C slow; gate in lamina II	PAG to raphe magnus descending
Reflexes	Spindle Ia monosynaptic; GTO Ib	Biceps C5 to C6, triceps C7, patellar L4, Achilles S1
UMN versus LMN	Spastic, hyperreflexic, Babinski / flaccid, atrophy, fasciculations	ALS both
Skeletal muscle	DHPR to RyR1; A band constant	Dantrolene for malignant hyperthermia
Smooth and cardiac	Calmodulin and MLCK; CICR	Heart cannot tetanize
Energy	PCr 10 s, glycolysis 2 min, oxidative beyond	Type I first (size principle)

Heart, lungs and kidneys

Topic	Rule	Board hook
ECG	PR 0.12 to 0.20; QRS under 0.12	Rate = 300 / large boxes
Cycle	S1 AV valves close; S2 semilunar close	S4 absent in atrial fibrillation
Output	CO = HR × SV; Frank Starling	Afterload lowers SV
Hemodynamics	MAP = CO × TPR; R ~ $1/r^4$	Arterioles are resistance vessels
Pressure control	Baroreflex seconds; RAAS long term	ACE inhibitor cough from bradykinin
Lung volumes	TLC = VC + RV; FRC = ERV + RV	Obstructive ratio below 0.70
Gas exchange	Shunt no O2 correction; dead space in PE	Hypoxia constricts lung vessels
O2 curve	P50 about 27; right shift unloads	CO lowers content, normal PaO2
Kidney	GFR 125 mL/min; FF 20%	Efferent constriction raises GFR
Nephron	PCT bulk; TAL multiplier; DCT NCC; CD ADH and aldosterone	Glucose threshold about 200 mg/dL
Acid base	Ratio sets pH; gap; Winters	Vomiting alkalosis, diarrhea normal gap

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