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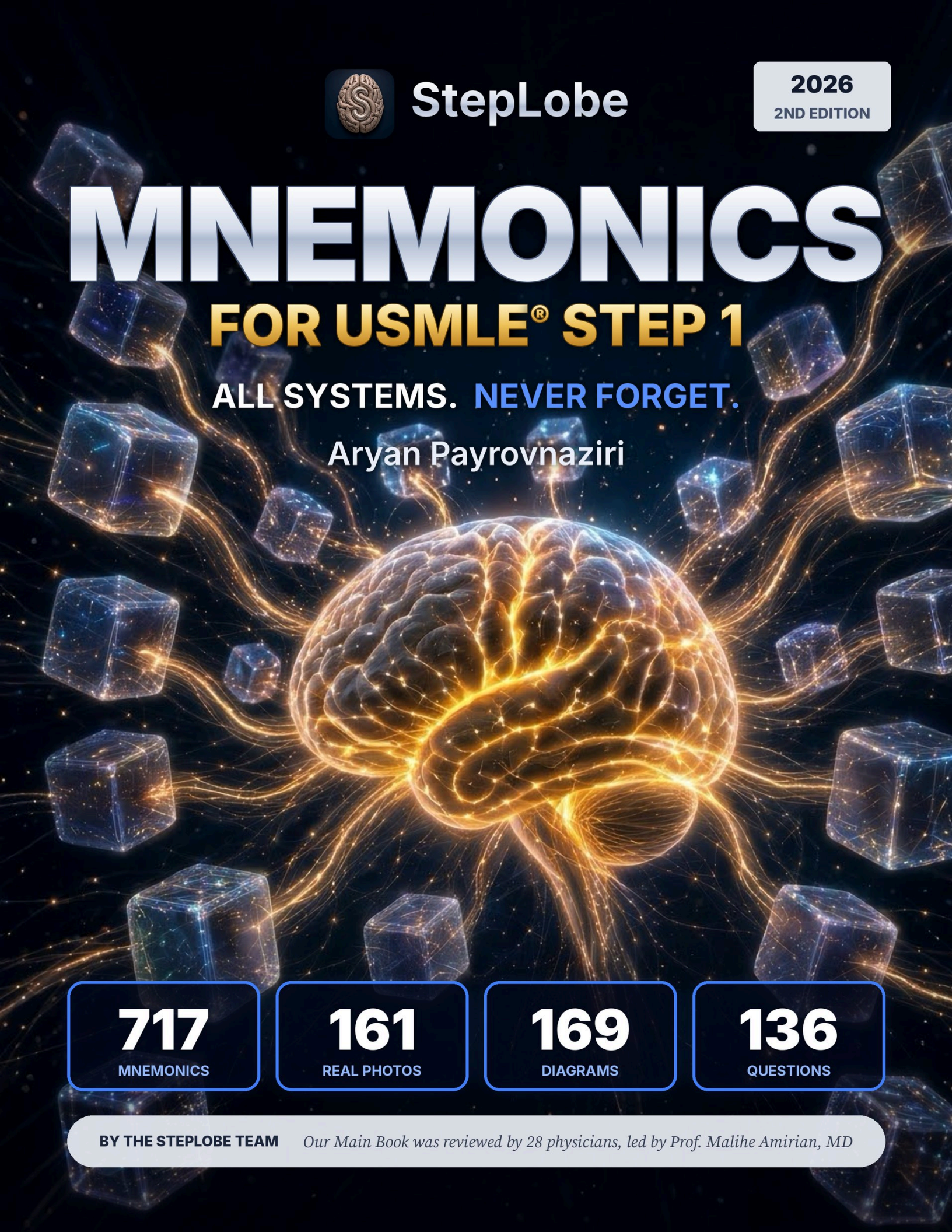
2026
2ND EDITION

MNEMONICS

FOR USMLE® STEP 1

ALL SYSTEMS. NEVER FORGET.

Aryan Payrovnaziri



717	161	169	136
MNEMONICS	REAL PHOTOS	DIAGRAMS	QUESTIONS

BY THE STEPLOBE TEAM

Our Main Book was reviewed by 28 physicians, led by Prof. Malihe Amirian, MD



STEPLOBE

MNEMONICS

for USMLE® Step 1
All systems. Never forget.

SECOND EDITION · 2026

Every mnemonic with what each letter means, why it is true and the exam pattern that triggers it. Real clinical photographs, original diagrams, look-alike tables, exam-style questions and rapid-recall sheets for all 17 subjects.

> From the team behind the StepLobe Main Book, reviewed by 28 physicians led by Prof. Malihe Amirian, MD (Obstetrics and Gynecology, Infertility).

Aryan Payrovnaziri

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A PERSONAL LETTER

I've sat where you're sitting. Let's get you through this, together.



Aryan Payrovnaziri
Founder, StepLobe

Dear future doctor,

If you're holding this book, you've already done something brave. You chose medicine, and you kept going when it got heavy. Be proud of that.

I know that weight, because I carried it too. My road ran from cell biology and biotechnology into pre-clinical medicine and NBME exams. I remember the late nights, the practice scores that wouldn't move, and the moments when one question made me doubt a whole week of work. If you feel that now, you're not behind. You're right on the path.

So we built StepLobe to be the study partner I wished I'd had. Every mnemonic comes with the reason it is true and the way the exam asks about it, so **one card gives you the list, the mechanism and the question**.

Every card in this book was checked against current guidelines, and deep-learning tools helped us check every fact. Our goal is simple: that you walk into that exam room calm, ready and proud of what you've built.

> ***"Struggling doesn't mean you're failing. It means you're learning something hard."***

FIVE THINGS I WISH SOMEONE HAD TOLD ME

1. **Progress beats perfection.** Thirty focused questions today beat a perfect plan you never start.
2. **Every mistake is a gift.** Read the "right when" line. That's where tomorrow's points are hiding.
3. **Protect your sleep.** It's when your brain files away everything you learned today.
4. **Your only competition is who you were yesterday.** Not your classmates, not social media.
5. **On the hard days, remember why you started.** One day a patient will be glad you didn't quit.

I can't sit the exam for you, but I promise you won't study alone. We're with you on every page. And when you pass (because you will), write and tell us. Your story may be the one that keeps the next student going.

With belief in you, *Aryan*

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START HERE

How to use this book

A mnemonic is a retrieval cue, not a substitute for understanding. Every card in this book gives you the cue, what each letter stands for, the reason it is true and the exact way the exam asks about it, so the list comes back when you need it.

M004 **Which side is the murmur on?** ★★★

RILE

- **R** **Right-sided murmurs** · tricuspid and pulmonic
- **I** **Increase on Inspiration** · more blood is pulled into the right heart
- **L** **Left-sided murmurs** · mitral and aortic
- **E** **increase on Expiration** · more blood returns from the lungs to the left heart

WHY IT WORKS Breathing in lowers intrathoracic pressure and fills the right heart, so tricuspid and pulmonic murmurs get louder. Breathing out does the same for the left heart.

EXAM HOOK If you see **a holosystolic murmur at the left lower sternal edge that gets louder with inspiration**, think **tricuspid regurgitation**.

1. **Topic and yield.** Card number, what it covers and how often it is tested.
2. **The mnemonic.** Coloured letters are the cues you will say out loud.
3. **Letter table.** Each cue, what it stands for and the detail the exam wants.
4. **Why it works.** The mechanism, so you can rebuild the list if you forget a letter.
5. **Exam hook.** The stem pattern that should make the mnemonic pop into your head.

1
Read the card
Learn the cue and the reason behind every letter.

2
Cover and recite
Hide the table and say each letter and its meaning aloud.

3
Use the hook
Practise going from the stem pattern to the mnemonic.

4
Compare
"Tell them apart" tables put look-alike conditions side by side.

5
Answer questions
Each section ends with exam-style questions and full explanations.

6
Space it
Use the rapid-recall sheet after 1, 3 and 7 days, then weekly.

★★★ appears on almost every exam form · ★★☆ often tested · ★☆☆ worth knowing

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CONTENTS

Contents

01 **Cardiovascular** 8
Murmurs, congenital heart disease, endocarditis, pericardium, arrhythmias, heart failure, vasculitis and cardiac drugs

02 **Renal & Acid–Base** 42
Acid–base, glomerular disease, diuretics, electrolytes and stones

03 **Pulmonary** 66
Lung anatomy, obstructive and restrictive disease, pneumonia, embolism and lung cancer

04 **Neurology & Cranial Nerves** 91
Cranial nerves, brainstem and stroke syndromes, spinal cord, seizures, dementia and neuro drugs

05 **Gastrointestinal** 153
Embryology, GI bleeding, liver and biliary disease, pancreas, bowel disease and GI drugs

06 **Hematology & Oncology** 193
Anemias, coagulation, leukemias and lymphomas, tumor markers and chemotherapy

07 **Endocrine** 227
Pituitary, thyroid, adrenal, calcium, diabetes and MEN syndromes

08 **Reproductive** 259
Embryology, pregnancy, ovarian and testicular disease, sex chromosome disorders and breast

09 **Musculoskeletal & Anatomy** 281
Brachial plexus, nerves and muscles, joints, bone disease and rheumatology

10 **Dermatology** 317
Skin layers, blistering disease, skin cancer and the classic rashes

11 **Psychiatry** 337
Mood, psychosis, anxiety, personality, substance use and psychiatric drugs

12 **Microbiology** 370
Bacteria, viruses, fungi, parasites and antimicrobials

13 **Immunology** 413
Cells and cytokines, hypersensitivity, immunodeficiency and transplant

14 **Pharmacology** 436
Pharmacokinetics, autonomic drugs, toxicology, antidotes and drug side effects

15 **Biochemistry & Nutrition** 477
Metabolism, enzymes, storage diseases, vitamins and minerals

16 **Genetics** 522
Inheritance patterns, trisomies, trinucleotide repeats and genetic syndromes

17 **Biostatistics & Ethics** 546
Study design, test statistics, bias, ethics and patient safety

- **Image credits** 566
- **Index of mnemonics** 572
- **Index of topics** 577
- **StepLobe Pass Guarantee: full rules** 582

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01

SECTION 1

Cardiovascular

Murmurs, congenital heart disease, endocarditis, pericardium, arrhythmias, heart failure, vasculitis and cardiac drugs

46 mnemonics · 10 clusters · 8 exam-style questions · rapid recall

ROADMAP

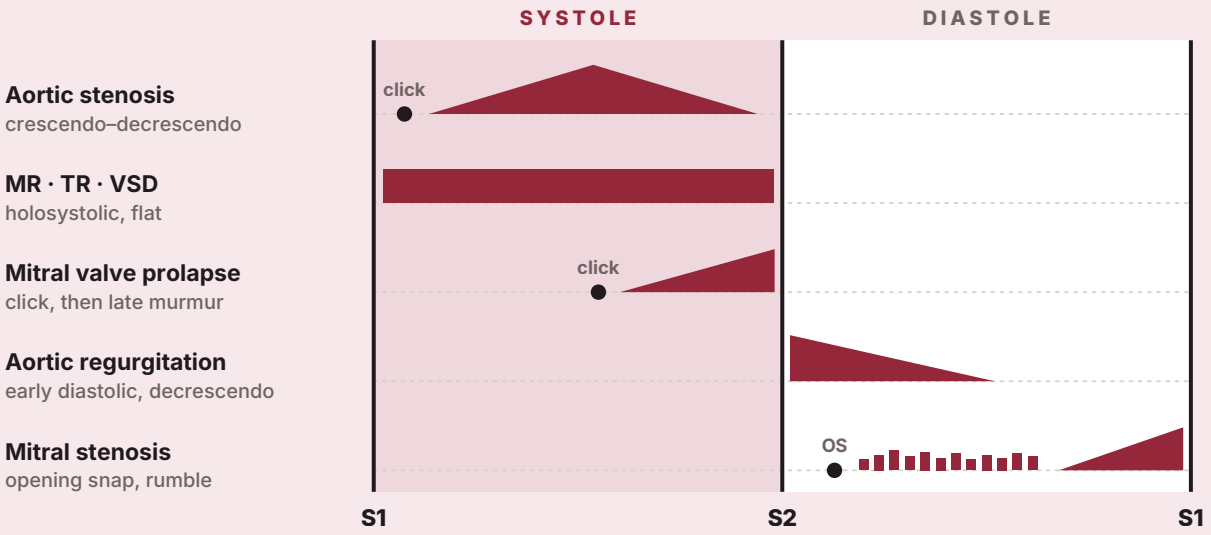
1	**Murmurs & auscultation** All Patients Eagerly Take Medicine, MR ASS, MS ARD, RILE, Less blood = Louder HOCM	★★★	5 cards
2	**Congenital heart disease** The 5 Ts, PROVe, VAD, Late C-C-P, Turner + rib notching + 2 Bs	★★★	5 cards
3	**Rheumatic fever & endocarditis** J♥NES, FROM JANE, Osler = Ouch, IV drug users go TRIcuspid, HACEK	★★★	5 cards
4	**Pericardium** CARDIAC RIND, Beck's 3 Ds	★★★	2 cards
5	**Arrhythmia & arrest** PIRATES, Hs and Ts, CHA_2DS_2-VASc	★★★	3 cards
6	**Ischemia & heart failure** FAILURE, LMNOP, MONA BASH, Days 3–14 = rupture, BAMS	★★★	5 cards
7	**Cardiomyopathy** ABCCCD, Stiff as SHALE after Radiation	★★☆	2 cards
8	**Hypertension** ABCDE	★★☆	1 card
9	**Vasculitis** CRASH and BURN, PAN spares the lungs, ELK, PAAR for the course …	★★★	6 cards
10	**Cardiac pharmacology** Some Block Potassium Channels, Quit Procrastinating, Doc, IB Loves MIs …	★★★	12 cards

1 Murmurs & auscultation

★★★

Stems describe a murmur by where it is loudest, when it happens and what changes it. These five cards turn that description into a diagnosis.

One cardiac cycle: where each murmur sits



Tell them apart: bedside maneuvers

MANEUVER	WHAT IT DOES	LOUDER	SOFTER
Inspiration	↑ venous return to the right heart	Right-sided murmurs (TR, PS)	Left-sided murmurs
Valsalva, standing	↓ preload	HCM; MVP (click earlier, murmur longer)	Most others (AS, MR, VSD)
Squatting, leg raise	↑ preload (squat also ↑ afterload)	AS, MR, VSD, AR	HCM; MVP click later
Handgrip	↑ afterload	MR, VSD, AR	AS, HCM

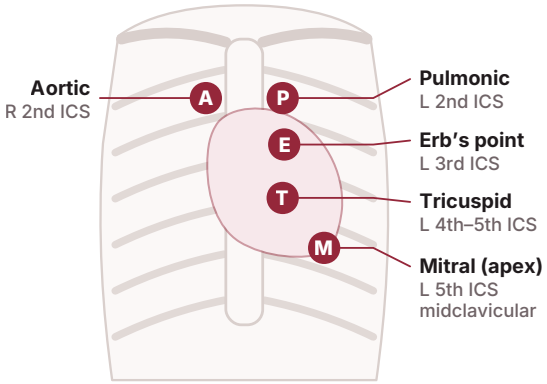
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M001 Heart valve auscultation order ★★★

All Patients Eagerly Take Medicine

- **A** **Aortic area** · right 2nd intercostal space, sternal edge
- **P** **Pulmonic area** · left 2nd intercostal space, sternal edge
- **E** **Erb's point** · left 3rd intercostal space: AR and HCM are often loudest here
- **T** **Tricuspid area** · left 4th–5th intercostal space, lower sternal edge
- **M** **Mitral area (apex)** · left 5th intercostal space, midclavicular line

WHY IT WORKS Questions rarely name the valve. They tell you where the murmur is loudest. Walk the chest from upper right to the apex in this order and every location maps to a valve.



Where each valve is heard best. These are listening posts, not the anatomical positions of the valves.

EXAM HOOK If you see **a murmur loudest at the apex that radiates to the left axilla**, think **mitral regurgitation**.

M002 The classic systolic murmurs ★★★

MR ASS

- **MR** **Mitral Regurgitation** · holosystolic, apex, radiates to the axilla
- **AS** **Aortic Stenosis** · crescendo–decrescendo, right 2nd ICS, radiates to the carotids
- **S** **Systolic** · VSD and TR are holosystolic too; MVP gives a mid-systolic click

WHY IT WORKS Systolic murmurs sit between S1 and S2. Learn them by shape: AS is a diamond, MR, TR and VSD are flat plateaus, and MVP is a click followed by a late murmur.

EXAM HOOK If you see **a harsh crescendo–decrescendo murmur radiating to the carotids in an older adult with exertional syncope**, think **aortic stenosis**.

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M003 **The classic diastolic murmurs** ★★★

MS ARD

- **MS** — **Mitral Stenosis** · opening snap, then a low mid-diastolic rumble at the apex
- **AR** — **Aortic Regurgitation** · early diastolic, blowing, decrescendo; wide pulse pressure
- **D** — **Diastolic** · a diastolic murmur is pathologic until proven otherwise

WHY IT WORKS Diastolic murmurs always need an echocardiogram. Mitral stenosis is usually rheumatic; aortic regurgitation comes from a damaged valve or a dilated aortic root.

EXAM HOOK If you see **an opening snap followed by a rumble in a woman who had rheumatic fever as a child**, think **mitral stenosis**.

M004 **Which side is the murmur on?** ★★★

RILE

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EXAM HOOK If you see **a holosystolic murmur at the left lower sternal edge that gets louder with inspiration**, think **tricuspid regurgitation**.

M022 **Hypertrophic cardiomyopathy and bedside maneuvers** ★★★

Less blood = Louder HOCM

- **↓ preload** — **Valsalva, standing up** · smaller ventricle, more outflow obstruction: louder
- **↑ preload** — **squatting, passive leg raise** · bigger ventricle, less obstruction: softer
- **↑ afterload** — **handgrip** · the ventricle empties less: softer
- **MVP** — **moves the same way** · ↓ preload makes the click earlier and the murmur longer



Massive left ventricular hypertrophy at autopsy: the wall is markedly thickened against the scale, as in hypertrophic cardiomyopathy.
Photo: Reza Iuke, CC BY-SA 3.0, via Wikimedia Commons

WHY IT WORKS A thick septum and the anterior mitral leaflet narrow the outflow tract. Less filling brings them closer, so the obstruction and the murmur grow. Aortic stenosis does the opposite and softens with Valsalva.

EXAM HOOK If you see **a young athlete with syncope and a systolic murmur that gets louder when he stands up**, think **hypertrophic cardiomyopathy**.

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2 Congenital heart disease

★★★

Blue at birth means a right-to-left shunt; pink at birth but blue years later means a left-to-right shunt that has reversed.

Early cyanosis (right-to-left shunts): count the 5 Ts

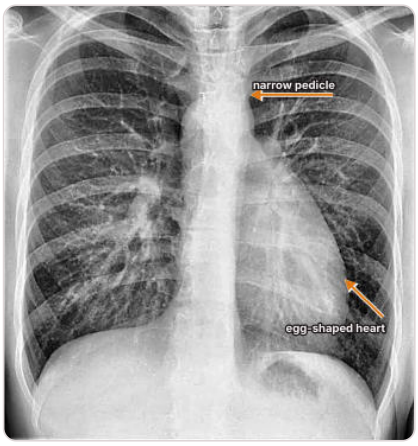
1	2	3	4	5
Truncus arteriosus	**Transposition** of great arteries	**Tricuspid** atresia	**Tetralogy** of Fallot	**TAPVR**
1 vessel	2 vessels switched	tri = 3	4 parts	5 words

M005 Cyanotic congenital heart disease (right-to-left) ★★★

The 5 Ts

- **T1** **Truncus arteriosus** · 1 vessel leaves the heart
- **T2** **Transposition of the great arteries** · 2 vessels switched
- **T3** **Tricuspid atresia** · tri = 3
- **T4** **Tetralogy of Fallot** · 4 parts
- **T5** **TAPVR** · 5 words: total anomalous pulmonary venous return

WHY IT WORKS These lesions send deoxygenated blood to the body from birth. Several are ductus-dependent: prostaglandin E1 keeps the ductus arteriosus open until surgery.



Transposition of the great arteries: an 'egg on a string' heart with a narrow pedicle.
Photo: Madhero88, CC BY-SA 3.0, via Wikimedia Commons

EXAM HOOK If you see **cyanosis in the first hours of life that does not improve with 100% oxygen**, think **a 5 T lesion; transposition is most common in newborns**.

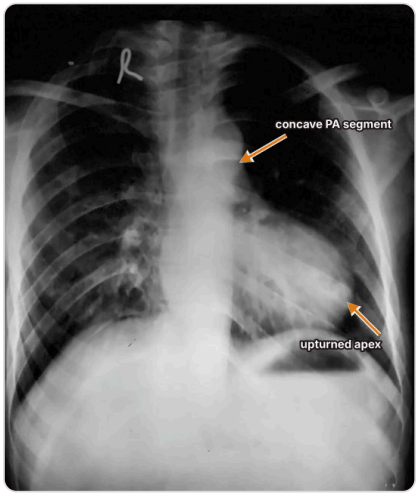
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M006 **Tetralogy of Fallot** ★★★

PROVe

- **P** **Pulmonary infundibular stenosis** · the key lesion: it decides how blue the child is
- **R** **Right ventricular hypertrophy** · from pumping against the stenosis
- **O** **Overriding aorta** · sits over the VSD
- **V** **VSD** · large and unrestrictive

WHY IT WORKS Squatting raises systemic vascular resistance, so less blood shunts right-to-left and more goes through the lungs. That is why children squat during a 'tet spell'.



Tetralogy of Fallot: boot-shaped heart, with an upturned apex and a concave pulmonary artery segment.

Photo: Medicalpal, CC BY-SA 4.0, via Wikimedia Commons

EXAM HOOK If you see **a toddler who squats after running and has a boot-shaped heart on chest X-ray**, think **tetralogy of Fallot**.

M007 **Left-to-right shunts, most to least common** ★★★

VAD

- **V** **VSD** · most common congenital heart defect; harsh holosystolic murmur
- **A** **ASD** · fixed, wide splitting of S2
- **D** **PDA** · continuous 'machine-like' murmur; prematurity, congenital rubella

WHY IT WORKS These children are pink at first. Years of extra flow through the lungs cause pulmonary hypertension, and the shunt can finally reverse.

EXAM HOOK If you see **a continuous machine-like murmur in a baby born very premature**, think **patent ductus arteriosus**.

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M008 **Eisenmenger syndrome: what reversal causes** ★★★

Late C-C-P

- **C** **Clubbing** · chronic hypoxemia
- **C** **Cyanosis** · appears late, once the shunt reverses
- **P** **Polycythemia** · secondary, driven by erythropoietin

WHY IT WORKS Pulmonary pressure rises above systemic pressure and the shunt turns right-to-left. At this stage the defect can no longer be closed.



Digital clubbing: bulbous fingertips with broad, convex nails on every finger.

Photo: Desherinka, CC BY-SA 4.0, via Wikimedia Commons

EXAM HOOK If you see **an adult with a known VSD who becomes cyanotic and develops clubbed fingers**, think **Eisenmenger syndrome**.

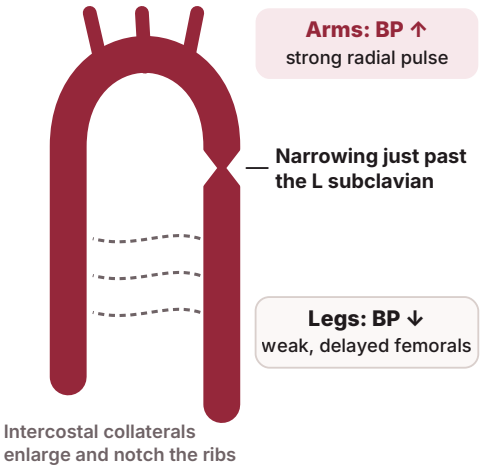
DON'T MISS A reversed PDA gives differential cyanosis: blue, clubbed toes with pink fingers.

M026 **Coarctation of the aorta** ★★★

Turner + rib notching + 2 Bs

- **T** **Turner syndrome (45,X)** · the classic association
- **R** **Rib notching** · enlarged intercostal collaterals erode the ribs
- **B** **Brachial–femoral delay** · arm blood pressure higher than leg blood pressure
- **B** **Bicuspid aortic valve** · found in many patients with coarctation

WHY IT WORKS A narrowing just past the left subclavian artery keeps pressure high in the arms and low in the legs. Collateral arteries grow around it.



Coarctation: high pressure above the narrowing, low pressure below it.

EXAM HOOK If you see **a girl with a webbed neck, high blood pressure in the arms and weak femoral pulses**, think **coarctation of the aorta**.

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3 Rheumatic fever & endocarditis ★★★

Rheumatic fever is an immune reaction weeks after strep throat. Endocarditis is a live infection on a valve. The setting tells you the organism.

Tell them apart: acute vs subacute infective endocarditis

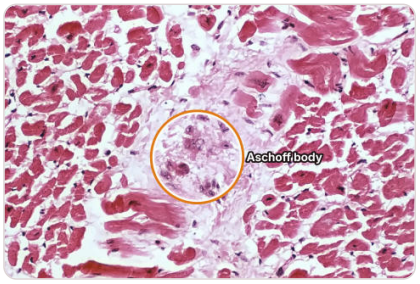
	ACUTE	SUBACUTE
Usual organism	Staphylococcus aureus	Viridans streptococci
Valve	Normal valves; tricuspid in people who inject drugs	Valves already damaged (rheumatic, congenital)
Course	Days; very ill; large, destructive vegetations	Weeks; low-grade fever; small vegetations
Typical trigger	Injection drug use, IV catheters	Dental procedures

M009 **Major Jones criteria for acute rheumatic fever** ★★★

J♥NES

- **J** **Joints** · migratory polyarthritis of large joints
- **♥** **Carditis** · pancarditis; the mitral valve is hit most often
- **N** **Nodules** · painless subcutaneous nodules over bony surfaces
- **E** **Erythema marginatum** · pink rings that spare the face
- **S** **Sydenham chorea** · involuntary, jerky movements

WHY IT WORKS Antibodies against the streptococcal M protein cross-react with heart, joint and brain tissue (molecular mimicry). Diagnosis needs evidence of recent group A strep infection plus 2 major, or 1 major and 2 minor, criteria.



Aschoff body (circled): the granulomatous lesion of rheumatic carditis.
Photo: Ed Uthman, MD, CC BY-SA 2.0, via Wikimedia Commons

EXAM HOOK If you see **migratory arthritis and a new murmur three weeks after an untreated sore throat**, think **acute rheumatic fever**.

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M010 **Signs of infective endocarditis** ★★★

FROM JANE

F	**Fever** · the most common sign
R	**Roth spots** · retinal hemorrhages with pale centers
O	**Osler nodes** · painful nodules on finger and toe pads
M	**Murmur** · new or changed
J	**Janeway lesions** · painless macules on palms and soles
A	**Anemia** · anemia of chronic disease
N	**Nail-bed (splinter) hemorrhages** · thin red-brown lines under the nails
E	**Emboli** · brain, kidney, spleen; lungs if right-sided

WHY IT WORKS Fever plus a new murmur is endocarditis until proven otherwise: take three sets of blood cultures before antibiotics, then get an echocardiogram.



Janeway lesions: flat, painless, embolic macules on the palm.
Photo: Warfieldian, CC BY-SA 4.0, via Wikimedia Commons



Splinter hemorrhages under the nail.
Photo: Splarka, public domain, via Wikimedia Commons

EXAM HOOK If you see **fever, a new murmur and painless red macules on the palms in a person who injects drugs**, think **infective endocarditis**.

M011 **Osler nodes vs Janeway lesions** ★★★

Osler = Ouch

O	**Osler nodes: painful** · immune-complex vasculitis; tender, raised, on finger and toe pads
J	**Janeway lesions: painless** · septic microemboli; flat, on palms and soles

WHY IT WORKS Pain tells you the mechanism. Immune complexes inflame (Osler, 'ouch'); septic emboli simply bleed into the skin (Janeway).

EXAM HOOK If you see **tender purple nodules on the fingertips of a patient with endocarditis**, think **Osler nodes**.

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M012 **Endocarditis: organism by setting** ★★★

IV drug users go TRIcuspid

- **T** — **Tricuspid valve** · the valve most affected in people who inject drugs
- **S** — **Staphylococcus aureus** · the most common cause overall; acute and destructive
- **V** — **Viridans streptococci** · previously damaged native valves; subacute; after dental work
- **E** — **S. epidermidis** · prosthetic valves and devices

WHY IT WORKS The patient's risk factor predicts the organism, and the organism predicts the course and the empiric antibiotics.



Infective endocarditis: surgically excised valve tissue; the arrow marks the red, friable vegetation.
Photo: Mikael Häggström, MD, CC0, via Wikimedia Commons

EXAM HOOK If you see **fever, septic lung emboli and a tricuspid vegetation in a person who injects heroin**, think **Staphylococcus aureus endocarditis**.

DON'T MISS Streptococcus gallolyticus (formerly S. bovis) endocarditis means: look for colon cancer.

M013 **Culture-negative endocarditis** ★★★

HACEK

- **H** — **Haemophilus** · H. parainfluenzae
- **A** — **Aggregatibacter** · formerly Actinobacillus
- **C** — **Cardiobacterium** · C. hominis
- **E** — **Eikenella** · E. corrodens
- **K** — **Kingella** · K. kingae

WHY IT WORKS These slow-growing gram-negative rods live in the mouth and can make routine blood cultures look negative. Also think of Coxiella and Bartonella.

EXAM HOOK If you see **endocarditis with negative routine blood cultures**, think **HACEK organisms (or Coxiella, Bartonella)**.

DON'T MISS The most common reason for negative blood cultures in endocarditis is antibiotics given before the cultures were drawn.

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9 Vasculitis

★★★

Vasculitis questions turn on two clues: the size of the vessel and the patient (age, sex, infection, smoking). Place the vessel size first, then let the patient pick the disease.

Vasculitis by vessel size



LARGE aorta and its branches	MEDIUM main visceral arteries	SMALL arterioles, capillaries, venules
Giant cell arteritis over 50; headache, jaw claudication, ESR ↑	**Polyarteritis nodosa** hepatitis B; spares lungs; ANCA negative	ANCA-ASSOCIATED **GPA** PR3-ANCA; granulomas; sinus, lung, kidney
Takayasu arteritis under 40, female; weak arm pulses	**Kawasaki disease** young child; coronary aneurysms	**Microscopic polyangiitis** MPO-ANCA; no granulomas; lung, kidney
	Buerger disease young smoker; digital gangrene	**EGPA** asthma, eosinophilia, neuropathy
		IMMUNE COMPLEX **IgA vasculitis** child; purpura, joints, gut, kidney

M027 **Kawasaki disease: diagnostic criteria** ★★★

CRASH and BURN

- **C** — **Conjunctivitis** · both eyes, no discharge; spares the rim around the iris
- **R** — **Rash** · polymorphous, mostly on the trunk; not vesicular
- **A** — **Adenopathy** · cervical, usually one-sided; one node at least 1.5 cm
- **S** — **Strawberry tongue** · with red, cracked lips and a red mouth and throat
- **H** — **Hands and feet** · red and swollen early; fingertip peeling in weeks 2–3
- **BURN** — **Fever for at least 5 days** · high and persistent; diagnosis needs fever plus 4 of the 5 signs



Red, cracked lips and a strawberry tongue: together they count as one criterion.

Photo: Dong Soo Kim, derivative work by Natr, CC BY 2.0, via Wikimedia Commons

WHY IT WORKS Kawasaki disease is a medium-vessel vasculitis with a taste for the coronary arteries. IVIG plus aspirin, given within 10 days of fever onset, sharply cuts the risk of coronary aneurysms, so the diagnosis rests on these bedside signs.

EXAM HOOK If you see **a 3-year-old with 6 days of high fever, red cracked lips, red eyes without discharge and swollen hands**, think **Kawasaki disease; check the coronaries by echocardiography**.

DON'T MISS Kawasaki disease is the classic exception to the rule against giving aspirin to children.

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QUESTION BLOCK

Test yourself: Cardiovascular

Exam-style questions built on this section's mnemonics. Answer all of them before you read the explanations.

1. A 24-year-old man who injects heroin has had fever and rigors for 5 days. Temperature is 103°F (39.4°C). A new holosystolic murmur is heard at the left lower sternal border and grows louder with inspiration. Chest x-ray shows several round peripheral lung opacities, some of them cavitating.

Which organism is the most likely cause?

A Viridans group streptococci
B Staphylococcus aureus
C Staphylococcus epidermidis
D Streptococcus gallolyticus
E Coxiella burnetii

2. A 66-year-old man is admitted with an anterior ST-elevation myocardial infarction 2 days after his chest pain began, too late for reperfusion. On hospital day 5 he suddenly becomes hypotensive. His jugular veins are distended and his heart sounds are muffled.

Which process in the infarcted wall best explains why this complication occurs at this time?

A Neutrophil infiltration
B Dense collagen scar formation
C Macrophage removal of necrotic myocardium
D Autoimmune reaction to myocardial antigens
E Coagulative necrosis with wavy fibers

3. A 17-year-old basketball player collapses during a game and recovers within a minute. His father died suddenly at age 40. A harsh systolic murmur is heard at the left sternal border; it gets louder when he stands up from a squat and softer with sustained handgrip.

Which drug is most likely to worsen his condition?

A Metoprolol
B Verapamil
C Disopyramide
D Nitroglycerin
E Phenylephrine

4. A 4-year-old boy has had a temperature above 102°F (38.9°C) for 6 days despite acetaminophen. He has red eyes without discharge, red cracked lips, a single enlarged lymph node on one side of his neck and swollen, red hands and feet. A rapid streptococcal antigen test is negative.

Which is the most appropriate treatment?

A Oral penicillin V
B Ibuprofen and supportive care
C Oral glucocorticoids alone
D IV ceftriaxone
E IV immune globulin plus aspirin

5. A 64-year-old man with ischemic cardiomyopathy has a left ventricular ejection fraction of 30%. He becomes breathless after walking one block. Furosemide controls his edema. Blood pressure is 118/74 mm Hg, serum potassium is 4.4 mEq/L and eGFR is 58 mL/min/1.73 m^2.

Which combination of additional drugs will most reduce his risk of death?

A Digoxin and hydrochlorothiazide
B Hydralazine alone
C Verapamil and lisinopril
D Metoprolol succinate, sacubitril–valsartan, spironolactone and dapagliflozin
E Amlodipine and hydrochlorothiazide

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6. A 68-year-old man has taken a drug for rhythm control of atrial fibrillation for 3 years. He now has a dry cough, exertional dyspnea, fatigue and weight gain. Sun-exposed skin on his face has a blue-gray tint. TSH is 14 μU/mL, and chest x-ray shows bilateral interstitial opacities.

Which set of tests should have been monitored regularly during his therapy?

A Pulmonary, liver and thyroid function tests
B Serum drug level and potassium
C Creatine kinase and lipid panel
D Complete blood count and antinuclear antibodies
E Serum creatinine and urinalysis

7. A 72-year-old woman with hypertension and type 2 diabetes has a new irregular pulse. ECG shows an irregularly irregular rhythm without P waves. Echocardiography shows normal valves and normal ventricular function. She has never had a stroke or transient ischemic attack.

What is her CHA_2DS_2-VASc score?

A 2
B 3
C 4
D 5
E 6

8. A 2-day-old boy has mild cyanosis and rapid breathing. Echocardiography shows one large arterial trunk leaving the heart above a ventricular septal defect, with both pulmonary arteries branching from this trunk.

Failure of which embryologic structure explains this defect?

A Endocardial cushions
B Truncal ridges and aorticopulmonary septum
C Septum primum
D Ductus arteriosus
E Sinus venosus

ANSWERS AND EXPLANATIONS

1. B Staphylococcus aureus

Injection drug use seeds the tricuspid valve (IV drug users go TRIcuspid), and a right-sided murmur grows louder with inspiration (RILE). Septic lung emboli fit acute, destructive Staphylococcus aureus endocarditis, the most common cause in people who inject drugs.

A Subacute disease of previously damaged valves, often after dental work. · **C** Prosthetic valves and implanted devices. · **D** Linked with colorectal cancer; prompts colonoscopy. · **E** Culture-negative endocarditis after farm animal exposure (Q fever).

2. C Macrophage removal of necrotic myocardium

Beck's 3 Ds (hypotension, distended neck veins, muffled heart sounds) mean tamponade from free wall rupture. Between days 3 and 14, macrophages clear dead myocytes before granulation tissue and collagen restore strength, so the wall is at its weakest (days 3–14 = rupture).

A Days 1–3; linked with fibrinous pericarditis, not rupture. · **B** Weeks 2 to 8; scar strengthens the wall and may bulge into an aneurysm. · **D** Dressler syndrome, weeks later; pericarditis without shock. · **E** First 4–24 hours, when arrhythmias are the main danger.

3. D Nitroglycerin

Less blood = Louder HOCM: lowering preload or afterload shrinks the ventricle and pulls the thick septum and mitral leaflet together. Nitroglycerin dilates veins and arteries, so outflow obstruction and syncope worsen. Dihydropyridines and large diuretic doses do the same.

A Slows the heart and lengthens filling; first-line therapy. · **B** Non-dihydropyridine that improves filling; used if beta blockers fail. · **C** Negative inotrope that lowers the outflow gradient; used in obstructive disease. · **E** Raises afterload and enlarges the ventricle; treats hypotension here.

4. E IV immune globulin plus aspirin

Fever for at least 5 days plus 4 of the 5 CRASH signs (here eyes, lips, node, hands and feet) means Kawasaki disease (CRASH and BURN). IVIG with aspirin, given within 10 days of fever onset, lowers the risk of coronary artery aneurysms; echocardiography is done at diagnosis.

A Treats streptococcal pharyngitis; the antigen test is negative. · **B** Leaves the coronaries unprotected; ibuprofen also blunts aspirin's antiplatelet effect. · **C** Added only for high-risk or IVIG-resistant disease, never alone. · **D** There is no bacterial infection to treat.

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SPACED REVIEW

Rapid recall: Cardiovascular

Cover the right-hand column. For each topic, say the mnemonic and what every letter stands for, then check. Tick the box when you get it right three times. Review after 1, 3 and 7 days, then weekly.

	CARD	TOPIC	MNEMONIC
☐	M001	Heart valve auscultation order	**All Patients Eagerly Take Medicine**
☐	M002	The classic systolic murmurs	**MR ASS**
☐	M003	The classic diastolic murmurs	**MS ARD**
☐	M004	Which side is the murmur on?	**RILE**
☐	M022	Hypertrophic cardiomyopathy and bedside maneuvers	**Less blood = Louder HOCM**
☐	M005	Cyanotic congenital heart disease (right-to-left)	**The 5 Ts**
☐	M006	Tetralogy of Fallot	**PROVe**
☐	M007	Left-to-right shunts, most to least common	**VAD**
☐	M008	Eisenmenger syndrome: what reversal causes	**Late C-C-P**
☐	M026	Coarctation of the aorta	**Turner + rib notching + 2 Bs**
☐	M009	Major Jones criteria for acute rheumatic fever	**J♥NES**
☐	M010	Signs of infective endocarditis	**FROM JANE**
☐	M011	Osler nodes vs Janeway lesions	**Osler = Ouch**
☐	M012	Endocarditis: organism by setting	**IV drug users go TRIcuspid**
☐	M013	Culture-negative endocarditis	**HACEK**
☐	M014	Causes of pericarditis	**CARDIAC RIND**
☐	M015	Cardiac tamponade: Beck triad	**Beck's 3 Ds**
☐	M016	Causes of atrial fibrillation	**PIRATES**
☐	M017	Reversible causes of cardiac arrest	**Hs and Ts**
☐	M046	Stroke risk in atrial fibrillation	**CHA_2DS_2-VASc**
☐	M018	Triggers of heart failure decompensation	**FAILURE**
☐	M019	Acute cardiogenic pulmonary edema	**LMNOP**
☐	M020	Acute myocardial infarction: early treatment	**MONA BASH**
☐	M021	Complications after myocardial infarction	**Days 3–14 = rupture**
☐	M045	Drugs that lower mortality in HFrEF	**BAMS**
☐	M023	Causes of dilated cardiomyopathy	**ABCCCD**
☐	M024	Causes of restrictive cardiomyopathy	**Stiff as SHALE after Radiation**
☐	M025	Causes of secondary hypertension	**ABCDE**
☐	M027	Kawasaki disease: diagnostic criteria	**CRASH and BURN**
☐	M028	Polyarteritis nodosa	**PAN spares the lungs**
☐	M029	Granulomatosis with polyangiitis (formerly Wegener)	**ELK**
☐	M030	IgA vasculitis (Henoch–Schönlein purpura)	**PAAR for the course**
☐	M031	Takayasu arteritis	**Pulseless Young Asian Woman**
☐	M032	Buerger disease (thromboangiitis obliterans)	**STOP smoking**
☐	M033	Antiarrhythmic drug classes (Vaughan Williams)	**Some Block Potassium Channels**
☐	M034	Class IA antiarrhythmics	**Quit Procrastinating, Doc**

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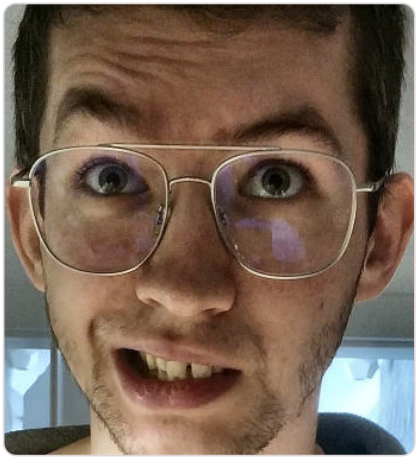
M124 Central vs peripheral facial palsy ★★★

UMN spares the forehead; LMN takes it all

UMN **Upper motor neuron lesion (cortex, internal capsule)** · opposite lower face weak; the forehead still wrinkles

LMN **Lower motor neuron lesion (nucleus or nerve, e.g., Bell palsy)** · whole face on the same side, forehead and eye closure included

WHY IT WORKS Motor neurons for the upper face get input from both hemispheres; those for the lower face mainly from the opposite one. A cortical stroke leaves the forehead working; a nerve lesion cuts the final path to every muscle.



Peripheral (LMN) palsy of the left face: on the weak side the brow stays down, the forehead stays smooth and the mouth does not move.

Photo: Wdobrzanski, CC BY-SA 4.0, via Wikimedia Commons

EXAM HOOK If you see **sudden drooping of the right lower face with normal forehead wrinkling, a weak right arm and trouble finding words**, think **left cortical (UMN) stroke, not Bell palsy**.

DON'T MISS The facial nucleus sits in the pons, so a pontine lesion of the nucleus gives an LMN pattern even though it is inside the brainstem.

M125 Secondary causes of facial nerve palsy ★★★

Lopsided Smile? Hunt Hard To Diagnose

L **Lyme disease** · a leading cause of bilateral palsy; ask about tick bites and rash

S **Sarcoidosis** · Heerfordt syndrome: parotid swelling, uveitis, fever, facial palsy

H **Herpes zoster: Ramsay Hunt syndrome** · ear pain, vesicles in the ear canal, hearing loss or vertigo

H **HIV** · can cause facial palsy, even at seroconversion

T **Tumors** · parotid or cerebellopontine angle; slow onset or no recovery

D **Diabetes** · raises the risk of facial palsy, as does pregnancy

WHY IT WORKS Most sudden one-sided facial palsy is idiopathic Bell palsy, probably from HSV-1 reactivation in the nerve. Look for a cause when the palsy is bilateral, slow, recurrent or comes with a rash, ear vesicles or other deficits.

EXAM HOOK If you see **weakness of both sides of the face in a camper from Connecticut who had an expanding red rash a month ago**, think **Lyme disease (Borrelia burgdorferi)**.

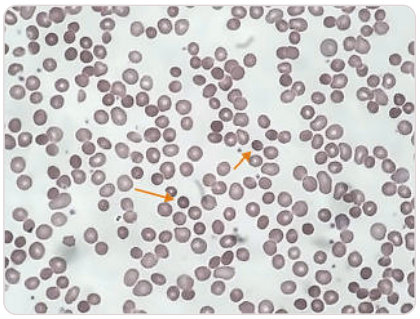
DON'T MISS Treat Bell palsy with oral steroids within 72 hours and protect the cornea; an antiviral alone does not help.

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M264 Warm vs cold autoimmune hemolytic anemia ★★★

IgG Grills, IgM wears Mittens

IgG Grills	**Warm AIHA: IgG binds at body temperature** · splenic macrophages trim coated cells into spherocytes; DAT positive for IgG
Warm causes	**SLE, CLL, drugs (methyldopa)** · most are idiopathic; treat with steroids, then rituximab
IgM Mittens	**Cold agglutinins: IgM binds below body temperature** · IgM fixes complement; red cells clump on the smear; DAT positive for C3
Cold Ms	**Mycoplasma, Mono (EBV), Malignant B cells (lymphoma)** · fingers and ears turn blue in the cold; keep the patient warm



Spherocytes (arrows): small, dense red cells without central pallor. Warm AIHA and hereditary spherocytosis look alike.

Photo: Paulo Henrique Orlandi Mourao, CC BY-SA 3.0, via Wikimedia Commons

WHY IT WORKS IgG binds best at 37 °C and is cleared by Fc receptors on splenic macrophages. IgM binds in cool fingers and ears; as a pentamer it clumps cells and fixes complement, so C3-coated cells are cleared mainly in the liver.

EXAM HOOK If you see **a student with a recent atypical pneumonia who has fatigue, jaundice and blue fingertips in the cold; DAT positive for C3 only**, think **cold agglutinins (IgM) after Mycoplasma**.

DON'T MISS Steroids help warm AIHA but rarely help cold agglutinin disease; there, avoid cold and use rituximab or the C1s inhibitor sutimlimab.

3 Microangiopathy, platelets & coagulation ★★★

Stems give a bleeding pattern and three numbers: platelet count, PT and PTT. Know what each one reads, and the microangiopathies, anticoagulant questions and inherited defects fall into place.

Tell them apart: bleeding labs at a glance

DISORDER	PLATELETS	PT	PTT	BLEEDING TIME
Hemophilia A or B	Normal	Normal	↑	Normal
von Willebrand disease	Normal	Normal	Normal or ↑	↑
Vitamin K deficiency, warfarin	Normal	↑	Normal or ↑	Normal
DIC	↓	↑	↑	↑
ITP, TTP, HUS	↓	Normal	Normal	↑
Bernard-Soulier / Glanzmann	↓ (giant) / normal	Normal	Normal	↑

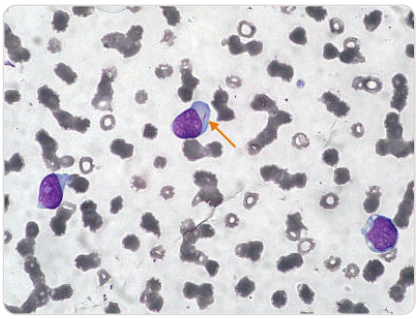
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M279 **Acute promyelocytic leukemia (APL)** ★★★

RARA

- **R** — **RARA fusion, t(15;17)** · PML-RARA stops maturation at the promyelocyte stage
- **A** — **Auer rods** · bundles of them in granular promyelocytes; MPO positive
- **R** — **Retinoic acid (ATRA) + arsenic trioxide** · makes the promyelocytes mature; low-risk APL needs no chemotherapy
- **A** — **Alarm: DIC** · bleeding is the early killer, so start ATRA on suspicion

WHY IT WORKS The PML-RARA fusion protein jams the retinoic acid receptor, so promyelocytes cannot mature. High-dose ATRA and arsenic degrade the fusion protein and release the block. Promyelocyte granules release procoagulant and fibrinolytic factors, so DIC appears early.



A myeloblast with a single Auer rod (arrow); in APL, promyelocytes carry whole bundles of them.

Photo: Paulo Henrique Orlandi Mourao, CC BY-SA 3.0, via Wikimedia Commons

EXAM HOOK If you see **a 35-year-old with gum bleeding, low fibrinogen and a marrow full of granular promyelocytes holding bundles of Auer rods**, think **APL: start ATRA at once**.

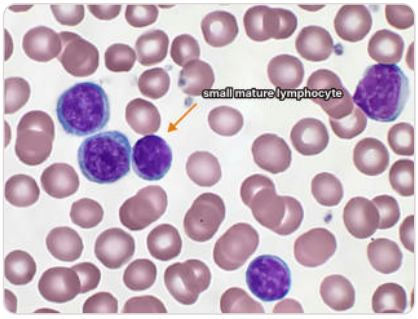
DON'T MISS ATRA and arsenic can cause differentiation syndrome: fever, dyspnea, lung infiltrates and edema. Treat with dexamethasone.

M280 **Chronic lymphocytic leukemia (CLL)** ★★★

SMEAR

- **S** — **Smudge cells** · fragile lymphocytes crushed when the smear is spread
- **M** — **Mature B cells, CD5+** · also CD20+ and CD23+; small cells with clumped chromatin
- **E** — **Elderly** · median age about 70; often found on a routine blood count
- **A** — **Autoimmunity and infections** · warm AIHA and ITP; low IgG brings recurrent infections
- **R** — **Richter transformation** · sudden change to diffuse large B-cell lymphoma

WHY IT WORKS CLL cells are long-lived, fragile B cells that make no useful antibody. They burst when the smear is spread. The disordered immune system attacks the patient's own blood cells, yet fails to fight bacteria.



CLL: small, mature-looking lymphocytes with dense, clumped chromatin and scant cytoplasm.

Photo: Mary Ann Thompson (VashiDonsk at en.wikipedia), CC BY-SA 3.0, via Wikimedia Commons

EXAM HOOK If you see **a 72-year-old with painless lymphadenopathy, lymphocytes 40,000/µL and many crushed cells on the smear**, think **chronic lymphocytic leukemia**.

DON'T MISS CD5+ B cells that are CD23− and cyclin D1+ mean mantle cell lymphoma, not CLL.

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2 Autoimmune blistering diseases ★★★

Three autoimmune diseases split the skin at different levels. The exam gives the age, the mouth and the immunofluorescence pattern, and asks for the target.

Tell them apart: autoimmune blistering diseases

DISEASE	SPLIT	TARGET	IMMUNOFLUORESCENCE	CLUE
Pemphigus vulgaris	Above basal layer	Desmoglein 3 (± 1)	IgG net around keratinocytes	Mouth first; Nikolsky positive
Bullous pemphigoid	Below epidermis	BP180, BP230	Linear IgG and C3 at basement membrane	Elderly; tense; mouth spared
Dermatitis herpetiformis	Below epidermis	Epidermal transglutaminase	Granular IgA in dermal papillae	Itchy; extensors; celiac

M417 Pemphigus vulgaris vs bullous pemphigoid ★★★

Vulgaris is Vulnerable, Bullous is Bulletproof

V **Pemphigus vulgaris: Vulnerable roof** · split above the basal layer; flaccid blisters tear into erosions; Nikolsky positive

Dsg3 **Target: desmosomes (desmoglein 3, often 1)** · IgG net around each keratinocyte; painful mouth erosions often come first

B **Bullous pemphigoid: Bulletproof roof** · split below the epidermis; tense blisters stay intact; Nikolsky negative

BP180 **Target: hemidesmosomes (BP180, BP230)** · linear IgG and C3 at basement membrane; mouth usually spared; adults over 70

WHY IT WORKS Desmosomes bind keratinocytes to each other, so losing them splits the epidermis inside itself and leaves a thin roof that tears. Hemidesmosomes bolt the basal layer to the basement membrane, so losing them lifts the whole epidermis off as a tense blister.



Bullous pemphigoid: tense, dome-shaped bullae that stay intact because the whole epidermis forms the roof.

Photo: Mohammad2018, CC BY-SA 4.0, via Wikimedia Commons

EXAM HOOK If you see **a middle-aged adult with months of painful mouth erosions, then flaccid skin blisters that spread when the edge is pressed**, think **pemphigus vulgaris (anti-desmoglein 3)**.

DON'T MISS DPP-4 inhibitors (gliptins) can trigger bullous pemphigoid; think of the drug in a diabetic patient with new tense blisters.

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M513 Candida vs Aspergillus vs Mucor ★★★

Candida Sausages, Aspergillus V, Mucor T

Sausages **Candida albicans** · budding yeast plus pseudohyphae pinched like sausage links; germ tube positive

V **Aspergillus fumigatus** · thin septate hyphae, acute (45°) branches; invades vessels in neutropenia

T **Mucorales (Rhizopus, Mucor)** · broad, ribbon-like, nearly nonseptate hyphae, right-angle branches; DKA



Candida yeast + pseudohyphae | **Aspergillus** septate, acute angle | **Mucor** nonseptate, right angle

Branching tells the molds apart: Aspergillus forks in a narrow V, Mucor branches at right angles like a T.

WHY IT WORKS All three are opportunists, and the host is the clue. Candida follows antibiotics, catheters or low T-cell immunity; Aspergillus follows neutropenia; Mucorales thrive in acidotic, iron-rich, hyperglycemic tissue. Aspergillus and Mucorales invade vessels and cause infarcts.

EXAM HOOK If you see **facial pain, bloody nasal discharge and a black necrotic palatal eschar in a man with diabetic ketoacidosis**, think **mucormycosis; debride and give amphotericin B**.

DON'T MISS Aspergillus also forms a fungus ball in an old TB cavity and causes allergic bronchopulmonary aspergillosis in asthma or cystic fibrosis.

M514 Cryptococcus neoformans ★★★

Pigeons Ink My Soap

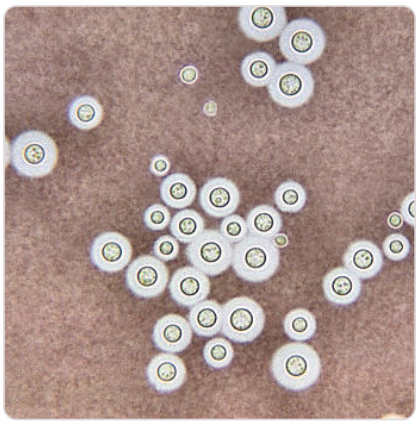
P **Pigeon droppings** · yeast inhaled from soil and bird droppings; lung first, then meninges

I **India ink** · capsule shows as a clear halo; CSF antigen test is more sensitive

M **Mucicarmine** · stains the thick polysaccharide capsule red in tissue

S **Soap-bubble lesions** · gelatinous cysts in the basal ganglia on brain imaging

WHY IT WORKS The polysaccharide capsule blocks phagocytosis and clogs CSF outflow, so opening pressure is high. Meningitis is subacute and strikes when CD4 count is below 100. Treat with amphotericin B plus flucytosine, then fluconazole; repeat lumbar punctures relieve pressure.



India ink: each round yeast sits in a clear halo, the unstained capsule, against the darker ink background.

Photo: CDC/Dr. Leanor Haley, Public domain, via Wikimedia Commons

EXAM HOOK If you see **two weeks of headache and fever in a man with AIDS, CD4 count 40/mm³, and a very high CSF opening pressure**, think **cryptococcal meningitis**.

DON'T MISS Start antiretroviral therapy weeks, not days, after antifungals begin: starting too early raises deaths from immune reconstitution inflammatory syndrome.

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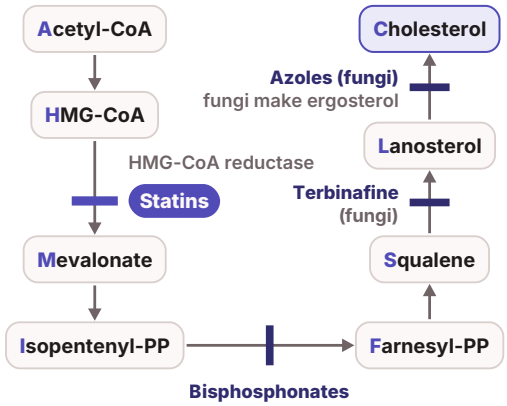
3 Cholesterol synthesis and statins ★★★

Cholesterol is built from acetyl-CoA, and drugs hit the pathway at several steps. Expect questions on the statin target, the antifungal and bisphosphonate targets on the same pathway, and statin muscle injury.

M615 **Cholesterol synthesis and the drugs that block it** ★★★

All Hungry Monkeys Instantly Find Sweet Little Cherries

- **A** **Acetyl-CoA** · leaves mitochondria as citrate; synthesis runs in cytosol and ER
- **H** **HMG-CoA** · HMG-CoA reductase: rate-limiting step, blocked by statins
- **M** **Mevalonate** · product of the committed step
- **I** **Isopentenyl pyrophosphate** · the five-carbon isoprene building block
- **F** **Farnesyl pyrophosphate** · its synthase is blocked by nitrogen-containing bisphosphonates in osteoclasts
- **S** **Squalene** · squalene epoxidase: terbinafine blocks the fungal enzyme
- **L** **Lanosterol** · first sterol ring; azoles block fungal 14α-demethylase
- **C** **Cholesterol** · used for membranes, bile acids, steroid hormones and vitamin D



Cholesterol synthesis: statins block the rate-limiting step; three other drug classes act further down.

WHY IT WORKS The liver controls this pathway at HMG-CoA reductase: insulin activates it, while glucagon and high cell cholesterol shut it down. Statins lower liver cholesterol, so hepatocytes make more LDL receptors and pull LDL out of the blood.

EXAM HOOK If you see **a patient whose LDL falls by half on a drug that blocks hepatic mevalonate production and increases hepatocyte LDL receptors**, think **statin: HMG-CoA reductase inhibitor**.

DON'T MISS Fungi make ergosterol instead of cholesterol. Terbinafine and azoles block fungal enzymes on this pathway, and azoles also inhibit human CYP3A4.

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M677 Chromosome deletion syndromes ★★☆

Cat 5, Elf 7, Catch-22

Cat 5	**Cri-du-chat: 5p deletion** · high-pitched cat-like cry, microcephaly, severe intellectual disability, VSD
Elf 7	**Williams: 7q11.23 (includes elastin)** · elfin face, very sociable, hypercalcemia, supravalvular aortic stenosis
Catch-22	**DiGeorge: 22q11.2** · thymic and parathyroid hypoplasia, conotruncal heart defects, cleft palate

WHY IT WORKS Each syndrome reflects the genes lost. Williams loses the elastin gene, so the aorta narrows just above the valve. DiGeorge disrupts the third and fourth pharyngeal pouches, removing thymus and parathyroids. Microarray finds these deletions; a routine karyotype can miss the small ones.

EXAM HOOK If you see **a very talkative, friendly child with a wide mouth, high calcium and a murmur from narrowing above the aortic valve**, think **Williams syndrome (7q11.23, elastin)**.

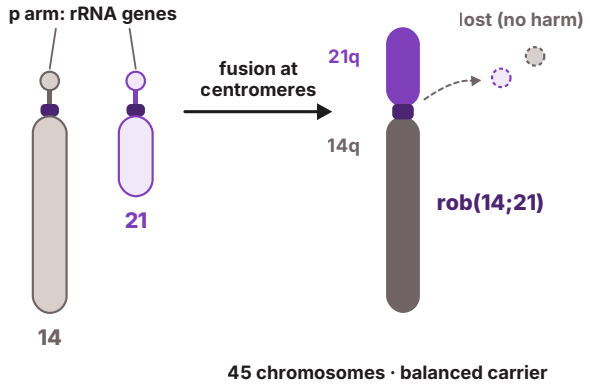
DON'T MISS 22q11.2 deletion is the most common microdeletion syndrome; its CATCH-22 features and psychosis risk are on M252.

M678 Robertsonian translocation ★★☆

Acrobats join hands and drop their hats

Acrobats	**Acrocentric chromosomes** · 13, 14, 15, 21 and 22: centromere near one end
join hands	**Long arms fuse at the centromere** · the carrier has 45 chromosomes yet is healthy (balanced)
drop their hats	**Short arms are lost** · harmless: their rRNA genes have many copies elsewhere
14;21	**Translocation Down syndrome** · familial, not tied to maternal age; recurrence highest when the mother carries it

WHY IT WORKS Two acrocentric chromosomes fuse near their centromeres and the tiny short arms are lost. The carrier keeps all essential genes, but at meiosis the fused chromosome can segregate unevenly, so eggs or sperm carry too much or too little of chromosome 21 or 13.

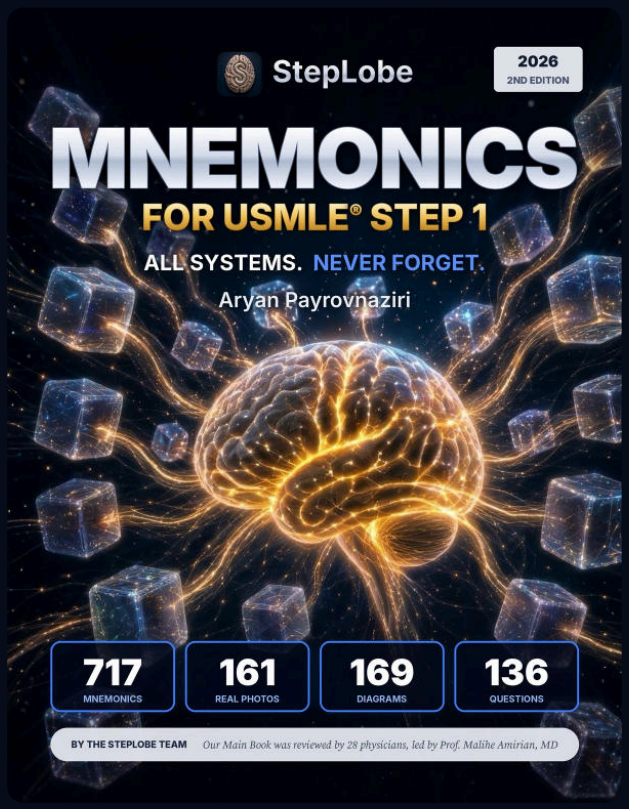


The long arms of 14 and 21 fuse; the lost short arms carry only spare rRNA genes.

EXAM HOOK If you see **a couple with recurrent miscarriages and a child with Down syndrome; the mother's karyotype shows 45 chromosomes**, think **balanced Robertsonian translocation, 14;21**.

DON'T MISS A parent carrying a 21;21 Robertsonian translocation can have only children with Down syndrome or miscarriages.

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