

Chapter 1: Anatomy and Physiology

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After this chapter you should be able to describe the location, size, shape, and surfaces of the heart; identify the pericardium and the three layers of the heart wall; name the chambers and valves and trace the path blood takes through them; describe the coronary arteries and the structures they feed; describe the heart's nerve supply; and explain the cardiac cycle, cardiac output, and blood pressure. **Reading:** Aehlert, *ECGs Made Easy*, 7th ed., Chapter 1 (p. 1 to 20).

Note to students: Every number, range, and definition in this guide follows **Aehlert, 7th edition**, because that is the edition this guide follows. You will find other sources that disagree on some values, and that is normal rather than a sign that someone is wrong. When a website conflicts with this guide, answer from the textbook. **What comes next:** the next chapter takes the structures you learn here and turns them into electricity. Every waveform you will read from here on is a picture of an impulse crossing tissue you are naming right now, so the anatomy is not a warm-up; it is the map the rest of the book is drawn on.

At a Glance: this chapter in one box

Numbers you must know

- The apex sits at the **left fifth intercostal space, midclavicular line**. About **two thirds** of the heart lies left of midline.
- The pericardial space holds about **20 mL** of serous fluid (normal range 10 to 30 mL).
- The left ventricle wall is roughly **three times** as thick as the right ventricle wall.
- SA node intrinsic rate is **60 to 100**. Atrial contraction adds **20 to 30 percent** of ventricular filling.
- About **8 in 10** people are right dominant.
- **CO = HR × SV** and **BP = CO × PVR**. Put both on a card and keep them where you can see them.

The one rule this chapter is built on

- **Pressure explains the anatomy.** Work out which side of a structure carries the higher pressure and everything else follows: which way blood moves, which valve is open, which is shut, and why one ventricle is thick while the other is thin. No valve decides anything. Every valve is a passive door pushed open and pulled closed by the pressure on either side of it.

Top 3 traps

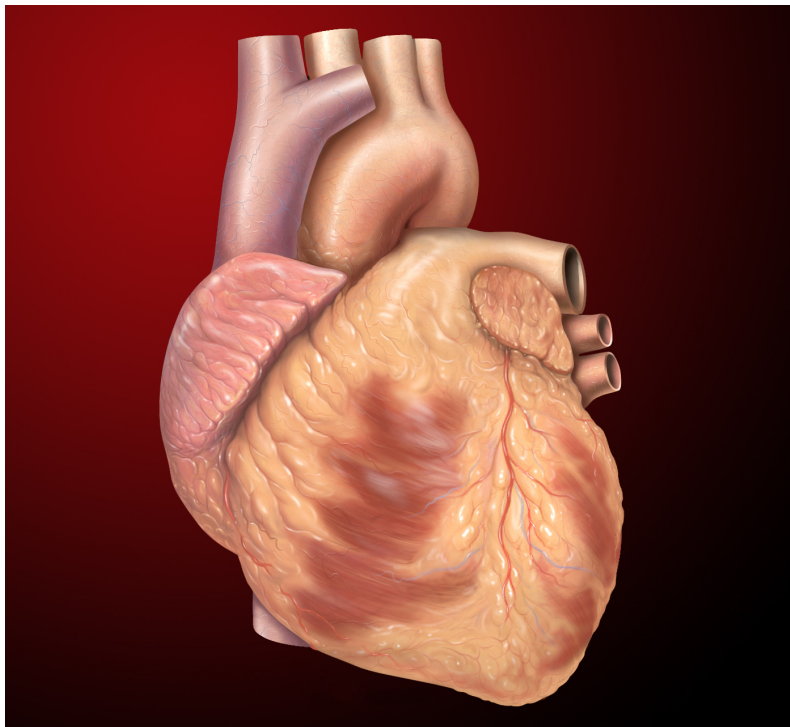
- The **base** of the heart is the top and the **apex** is the bottom. The everyday meaning of the word fights the anatomy.
- **Epicardium and visceral pericardium are one layer under two names**, not two layers stacked on each other.
- The pulmonary artery carries **deoxygenated** blood and the pulmonary veins carry **oxygenated** blood. Artery and vein describe direction relative to the heart, not oxygen content.

Where the Heart Sits, and What Shape It Is

The heart lies in the mediastinum: behind the sternum, between the lungs, above the diaphragm, with about two thirds of it left of midline. It is the size of your own fist, the most useful reference there is because it scales to whichever body you are standing in front of. Make a fist now and hold it against the left of your sternum, knuckles angled down and left. That is the size, the position, and the tilt at once, and you will remember it because your own hand did it.

Two landmarks matter more than the rest. The **base** is superior and posterior, where the great vessels attach. The **apex** is the inferior tip pointing down and left, at roughly the **left fifth intercostal space, midclavicular line**: where you palpate the point of maximal impulse and where the V4 electrode goes, so learning it once pays twice.

The **surfaces** trip people up because the chamber you see from the front is not the one you expect. The **anterior** surface facing the sternum is mostly **right ventricle**, which is why it is the chamber most often injured in blunt chest trauma. The **inferior** surface rests on the diaphragm and the **posterior** surface is mostly **left atrium**. The left ventricle sits left and posterior, which is why V5 and V6 go toward the armpit.



Anterior exterior view. The right ventricle dominates the front surface. Patrick J. Lynch, CC BY 2.5, via Wikimedia Commons.

The Pericardium and the Three Layers of the Wall

Push your fist into a partly deflated balloon. Your fist is the heart. The balloon wall wrapping snugly against your knuckles is the **visceral pericardium**; the outer wall your fist never touches is the **parietal pericardium**. One membrane folded back on itself, which never makes sense from a diagram and is obvious the moment your hand does it. Between the layers is the **pericardial space**, holding about **20 mL** of serous fluid (normal range 10 to 30 mL) so the surfaces slide instead of grip.

Outside that is the tough **fibrous pericardium**, which anchors the heart and limits acute distension. Because it does not stretch quickly, fluid accumulating fast can compress the heart at a volume that would be tolerated if it collected slowly.

The heart wall itself is three layers, outside in:

Layer	What it is	What it does
Epicardium	Outermost. This is the visceral pericardium.	The coronary arteries run along it
Myocardium	Thick muscular middle layer	All the contracting, and all the depolarizing you see on a strip
Endocardium	Smooth inner lining	Lines chambers and valves; continuous with great-vessel endothelium

Say that first row out loud once: **epicardium and visceral pericardium are the same layer under two names**, depending on whether you are naming the sac or the wall. Students count them as two layers and lose the point.

Chambers, Valves, and the Route Blood Takes

Four chambers: two thin-walled atria that receive blood, two thick-walled ventricles that eject it, divided by the **interatrial** and **interventricular septa**.

Why the left ventricle is thicker. The right ventricle pushes blood across the room, to the lungs next door in a low-pressure circuit. The left pushes blood through the whole house: brain, gut, kidneys, down to your toes. Same blood, wildly different resistance, so the left ventricular wall is roughly **three times** as thick.

The valves. Two atrioventricular valves sit between the atria and ventricles; two semilunar valves guard the exits.

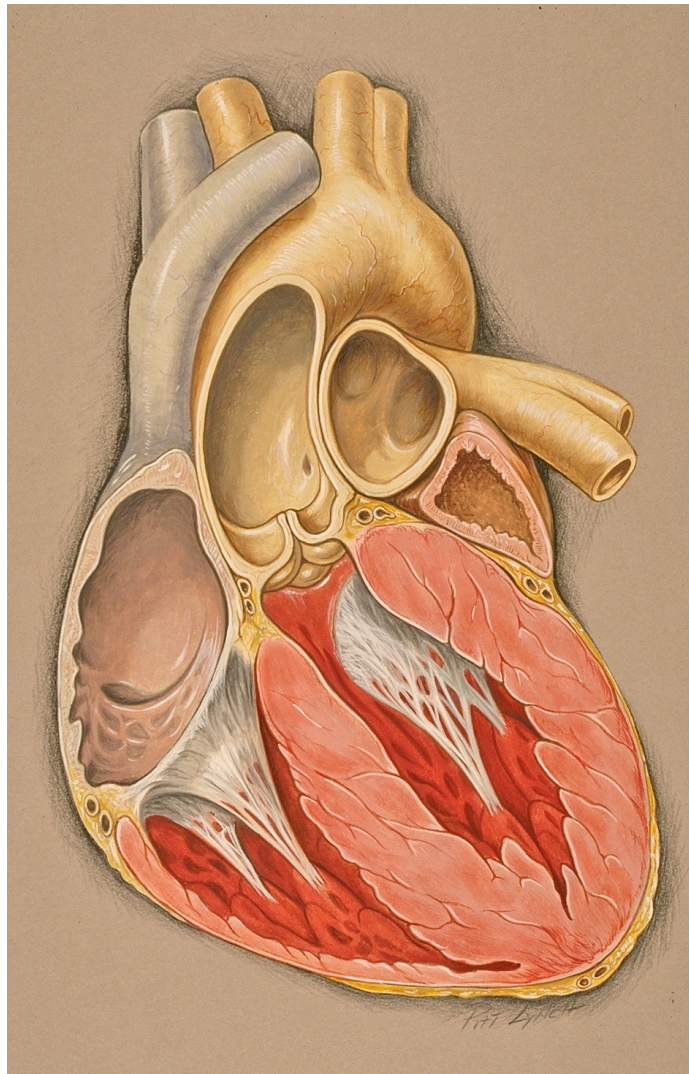
- **Tricuspid valve**, right side, three cusps, and **mitral valve**, left side, two cusps. **Memory hook:** the **TRI**cuspid is on the **R**ight, and you ride a tricycle before you ride a bicycle, so TRI comes before BI.
- **Pulmonic valve**, the exit from the right ventricle, and **aortic valve**, the exit from the left.

Use the tricycle hook to get started, then drop the word “bicuspid” (see the first trap box) and call the left AV valve the **mitral** valve, its name in every report you will read.

Chordae tendineae and papillary muscles anchor the AV valve leaflets. They are the guy-wires, not the motor: think of parachute strings or umbrella ribs, which open and close nothing and only keep the canopy from blowing inside out. Valves are opened and closed by **pressure**; the chordae only stop the leaflets everting into the atrium during systole.

The route, in order. Venae cavae and coronary sinus into the **right atrium**; through the **tricuspid** into the **right ventricle**; out the **pulmonic** valve to the pulmonary arteries; through the lungs; back through the pulmonary veins into the **left atrium**; through the **mitral** into the **left ventricle**; out the **aortic** valve into the aorta. **Memory hook:** the flow order of the valves is **T**ricuspid, **P**ulmonic, **M**itral, **A**ortic, which spells *Try Pulling My Aorta*. Read the second trap box before trusting that mnemonic for anything else.

One detail trips students up over and over: the **pulmonary artery** is the only artery carrying deoxygenated blood, and the **pulmonary veins** the only veins carrying oxygenated blood. Artery means “away from the heart,” vein means “toward” it; neither says anything about oxygen.



Coronal section: four chambers, four valves, and the great vessels. Note the left ventricular wall thickness. Patrick J. Lynch, CC BY 2.5, via Wikimedia Commons.

Common Trap

The left AV valve has two cusps, so “bicuspid valve” is the right name for it. True or false?

Technically defensible, practically wrong, and on a report actively misleading. In cardiology and sonography the left atrioventricular valve is the **mitral** valve. A clinician who says **bicuspid valve** almost always means a **congenital bicuspid aortic valve**: a different valve in a different position, formed with two cusps instead of three.

Use the tricycle hook to remember that **TRI is on the right**, then retire the word bicuspid. **Tricuspid right, mitral left**; “bicuspid” means the aortic valve with one cusp too few.

Common Trap

“Try Pulling My Aorta” gives you the order the valves close. True or false?

False, and a surprising number of websites label it wrongly. TPMA is the **flow** order, the order one red blood cell meets the valves on a lap. It is not a timing sequence, because the two sides of the heart work *at the same time*.

Closure is paired, not sequential. Mitral and tricuspid close together at the start of ventricular systole and make **S1**, the “lub.” Aortic and pulmonic close together at the end and make **S2**, the “dub.” Separate drawers: **TPMA is a route; S1 and S2 are a clock.**

Common Trap

The left ventricle is thicker because it holds more blood. True or false?

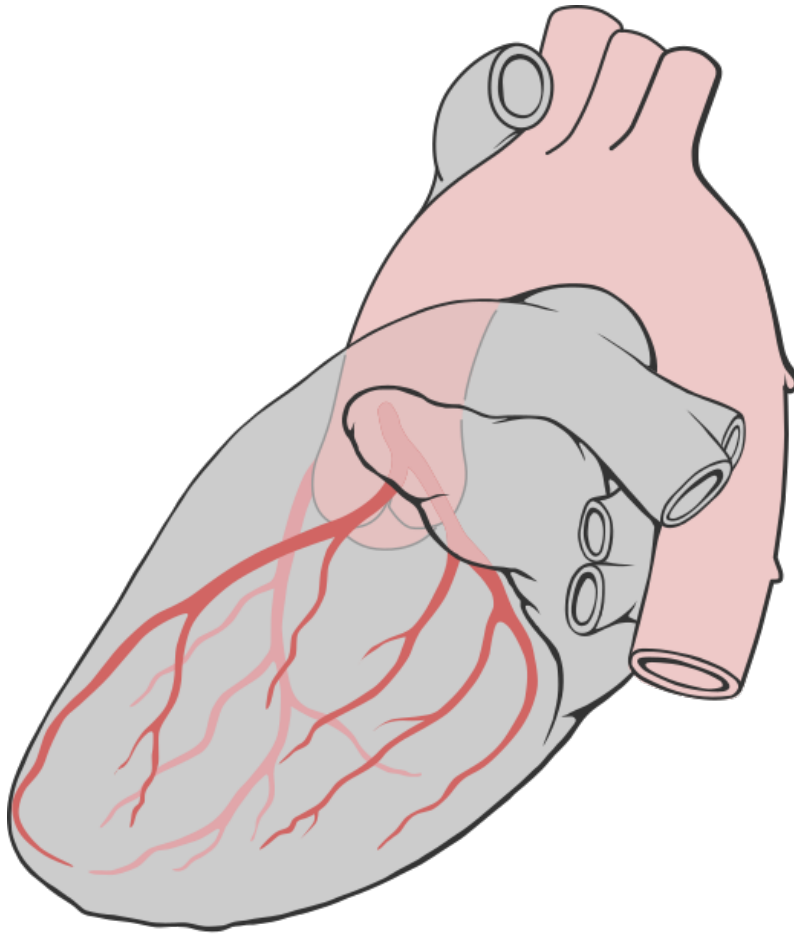
False, and the most common wrong answer on this material. **Both ventricles eject the same stroke volume.** They have to; whatever the right side sends to the lungs comes back for the left side to send onward, so the two outputs must match or blood piles up somewhere.

The difference is **resistance, not volume**: the right ventricle pumps into the low-pressure pulmonary circuit next door, the left into the whole systemic circuit. **Pressure builds muscle.** Which arm gets bigger, the one lifting the box or the one pointing at it?

The Heart's Own Blood Supply, and Why It Decides Your Rhythm

This is the section of the chapter that pays you back the most, so study it hardest. The heart does not feed itself from the blood inside its chambers, but through the **coronary arteries**, which leave the aorta just above the aortic valve and run along the epicardium: the **left main**, dividing into the **left anterior descending (LAD)** and the **circumflex**, and the **right coronary artery (RCA)**.

Artery	Territory it supplies	Why you will care
Left main	Divides at once into LAD and circumflex	One lesion threatens both branches
LAD	Anterior wall, anterior two thirds of the septum, much of the LV	"The widow maker," for how much muscle it feeds
Circumflex	Lateral and posterior LV	Supplies the SA nodal artery in a large minority
RCA	RA, RV, inferior wall , and in most people the SA and AV nodes	Makes an infarct a rhythm problem



Coronary arteries on the heart surface: LAD, circumflex, and RCA branches. Patrick J. Lynch, CC BY 2.5, via Wikimedia Commons.

Dominance names which artery gives rise to the posterior descending artery. **About 8 in 10 people are right dominant**; the rest are left dominant or codominant. Precise percentages are published everywhere and several of the most-cited sets are mutually inconsistent, so learn the shape of it, not a decimal you cannot defend.

Now the payoff. In **most people** the RCA supplies both the SA node and the AV node. Note that phrasing: the SA nodal artery arises from the RCA in roughly **60 percent** of people and from the circumflex in the rest, and the AV nodal artery from the RCA in about **9 in 10**. Strong majority, not a law, so never say “always.”

Now follow it through. The RCA feeds the **inferior wall** and, usually, **both nodes**, so an **inferior MI is not only a wall problem; it is a rate and rhythm problem**. The vessel starving inferior muscle is also starving the tissue that sets the pace and passes the impulse along. **High-grade AV block occurs in about 19 percent of acute inferior MIs**, from ischemia to nodal tissue combined with increased vagal tone, and it is usually **transient**, resolving as the vessel reperfuses.

That one mechanism is why this chapter deserves real study time. When you reach the sinus mechanisms and see sinus bradycardia in a patient with chest pain, reach the atrioventricular blocks and watch Wenckebach appear during an inferior infarct, or reach the infarction patterns and read inferior ST elevation, you are not memorizing three facts; you are recognizing one artery three times.

One more piece of mechanics with clinical teeth: **the coronary arteries fill during diastole**, because contracting myocardium squeezes its own vessels shut. The heart feeds itself only while it rests, so a sustained tachycardia shortens diastole and starves the pump of its own supply. Your first example of a rhythm on a monitor being a perfusion problem in a body.

The Nerve Supply

Both arms of the autonomic nervous system reach the heart, but not equally.

- **Sympathetic** fibers (the accelerator) innervate the atria **and** ventricles. Norepinephrine at beta-1 receptors raises rate, conduction speed, and force.
- **Parasympathetic** fibers travel in the **vagus nerve** (the brake), mainly to the SA node, AV node, and atria, with little ventricular reach. Acetylcholine slows the rate and slows AV conduction.

Three terms the book uses constantly: **chronotropy** is **rate** (chrono as in chronometer), **dromotropy** is **conduction velocity** through the AV node, and **inotropy** is **force** of contraction. Positive means faster, quicker to conduct, harder-squeezing.

At rest, **vagal tone dominates**, holding the rate below what the SA node would do alone, which is why a fit adult's resting rate can sit well under the node's intrinsic **60 to 100**. Take the brake away and the rate climbs toward that pace; it is exactly how atropine works. Bearing down, vomiting, or straining pushes vagal tone the other way, so you meet this nerve again in the sinus mechanisms chapter as a cause of transient bradycardia.

The Heart as a Pump: Cycle, Output, and Pressure

The **cardiac cycle** is two phases repeating. **Systole** is contraction and ejection; **diastole** is relaxation and filling. Every chamber has both, so say which chamber you mean.

Picture a squeeze bottle. Squeeze it (systole) and the pressure inside rises until it pops the cap open and liquid shoots out the top. Release it (diastole) and the cap falls shut while the bottle refills from the bottom. The cap decides nothing; the pressure inside opens and closes it. That is exactly how valves behave, and why the right answer to "what closes the mitral valve" is "rising ventricular pressure."

Most ventricular filling is **passive**, happening early in diastole before the atria do anything. The atria then contract and top the ventricle off, adding the last **20 to 30 percent**. That is the **atrial kick**, and losing it is why an already marginal patient tolerates atrial fibrillation badly: the punchline of the atrial rhythms chapter.

Cardiac output is how much blood the heart moves in one minute.

$$\text{CO} = \text{HR} \times \text{SV}$$

You are filling a kiddie pool with buckets. Two ways to fill it faster: carry **more buckets per minute** (heart rate), or carry a **bigger bucket** (stroke volume). That hands you compensation for free: shrink the bucket and the only way to keep filling at the same speed is to carry buckets faster. It is why a patient losing blood, or one whose ventricle is not filling well, shows up **tachycardic**. The rate is not the disease; the rate is the compensation, and reading it that way is much of what makes a good monitor tech.

Stroke volume has three inputs:

- **Preload**, the volume stretching the ventricle at the end of diastole. More filling means a stronger squeeze, up to a point. That is the **Frank-Starling** mechanism, and the honest picture is a person carrying steadily heavier loads up a hill: what they deliver rises, plateaus, then falls off. A curve that rolls over, not a band that snaps.
- **Afterload**, the resistance the ventricle pushes against. Picture watering the garden, then stepping on the hose: same pump, same effort, less water out the end. That is why high blood pressure makes the left ventricle work harder every beat, and why over years it thickens.
- **Contractility**, the strength of the squeeze independent of how full the chamber is. Sympathetic stimulation increases it.

Blood pressure adds one more term, peripheral vascular resistance, which is how tight the vessels are.

$$\text{BP} = \text{CO} \times \text{PVR}$$

Stacked, these two equations explain nearly every "why did that change" question you will meet: the rate on the monitor sits inside cardiac output, and cardiac output sits inside blood pressure. Trace a bad rhythm forward through both and you can predict the patient before you walk in.

Before You Move On

You should now be able to:

- name the chambers, valves and layers of the heart wall, and trace a red cell through the whole circuit
- say what each coronary artery supplies, and explain why the right coronary artery makes an inferior infarct a rhythm problem
- state cardiac output as rate times stroke volume, and explain what preload and afterload each change
- explain why the left ventricle is thicker than the right without saying that it holds more blood

The next chapter turns this anatomy into electricity. The same chambers and the same pathway get described as depolarization and repolarization, and the coronary supply you just learned becomes the reason a blocked artery shows up as a rhythm.

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