

Cardiovascular Anatomy and Physiology

The pump, the circuit, the wiring — and why every cardiac drug and every rhythm makes sense only once you have them

Coronary artery disease is the largest killer of Americans. Most of this chapter is about one organ failing in four ways.
Aligned to Region SOP

One organ, four ways to fail — and the largest killer you will meet

1 PUMP

The myocardium cannot generate adequate stroke volume. Cardiogenic shock, acute heart failure. Fixed by supporting preload, contractility and afterload.

2 PIPE

The coronary arteries cannot deliver blood to the muscle. Angina, acute coronary syndrome, STEMI. Fixed by reopening the artery — which is why destination matters.

3 WIRING

The conduction system fires in the wrong place, at the wrong rate, or not at all. Arrhythmias, blocks, arrest. Fixed by electricity or drugs that change rate and conduction.

4 CONTAINER

The vessels or the pericardial sac fail. Aortic dissection, tamponade, hypertensive emergency. Often fixed only in an operating room.

The number-one cause of death in the US and Canada — and many of those deaths happen before hospital

For most diseases you deliver the patient to definitive care. In this one you are frequently the difference between arriving and not arriving — through early defibrillation, early STEMI recognition, and the transport decision.

Part 1 today — and where it leads

Anatomy

Layers, chambers, valves, flow, coronary and peripheral circulation.

Physiology

The cardiac cycle, stroke volume, cardiac output, autonomic control.

Electrophysiology

Depolarization, the conduction system, intrinsic rates.

→ Part 2

ECG monitoring and arrhythmia analysis — built directly on the wiring.

→ Parts 3-5

Assessment, specific emergencies, and the 12-lead.

Read now. We solve it at the end.

Dispatch

Chest pain, 0640 hours.

On arrival

A 71-year-old man, two hours of crushing substernal pressure radiating to the jaw, nauseated and diaphoretic. He is alert and talking in full sentences.

HR 52, BP 104/62, RR 20, SpO₂ 95% on room air. Lungs clear. His 12-lead shows ST elevation in II, III and aVF.

Aspirin is given. An IV is established. Nitroglycerin 0.4 mg is placed under his tongue for the pain.

Ninety seconds later he is gray and barely responsive. BP 62/40. HR 48. His lungs are still clear.

Hold these questions:

- 1 Which coronary artery is most likely occluded, and which parts of the heart does it feed?
- 2 His lungs are clear. What does that rule out as the cause of the hypotension?
- 3 Which component of stroke volume did the nitroglycerin remove?
- 4 What would you have wanted to know before giving it?

SECTION 1

Anatomy

A muscular pump the size of your fist, with its own blood supply and a one-way circuit through it.

Layers • Chambers and valves • Blood flow • Coronary circulation

The heart in the chest, and its three layers

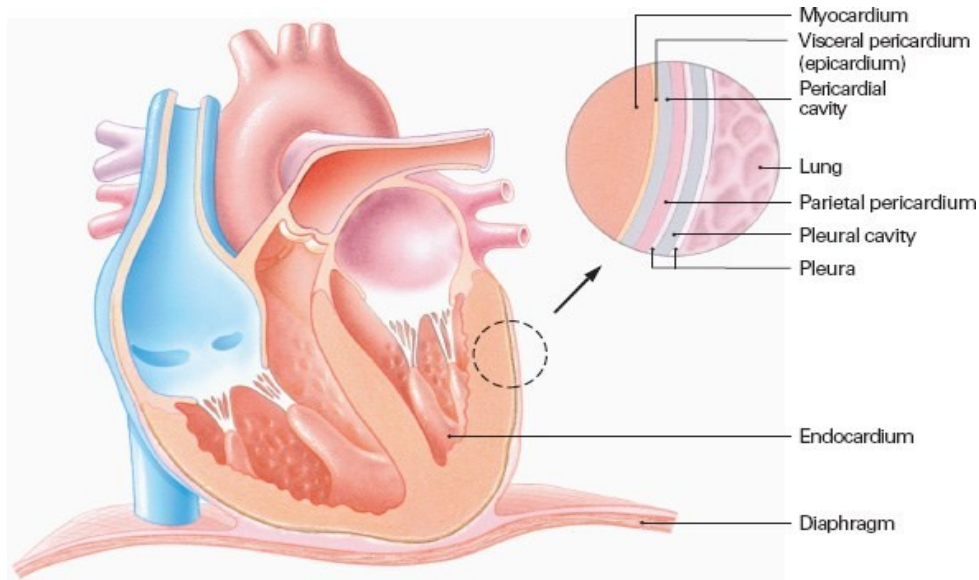


Figure 33-2 Layers of the heart.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

1 Endocardium

The innermost layer, lining the chambers and continuous with the lining of the vessels. Its disease is endocarditis.

2 Myocardium

The thick muscular middle layer — described as skeletal-like muscle with electrical properties. The layer that contracts, and the layer that infarcts.

3 Pericardium

The protective sac. VISCERAL layer against the muscle, PARIETAL layer outside it, with pericardial fluid between to reduce friction.

4 Position and size

About the size of the patient's closed fist. APEX at the bottom, just above the diaphragm. BASE at the top, level with the second rib.

Three layers, three disease families

Endocarditis lives in the lining. Infarction and myocarditis live in the muscle. Pericarditis and tamponade live in the sac — and because the parietal layer is fibrous and will not stretch quickly, even a modest volume in that space stops the ventricles filling. Tamponade is a preload problem created by a container problem.

Four chambers, four valves, one direction

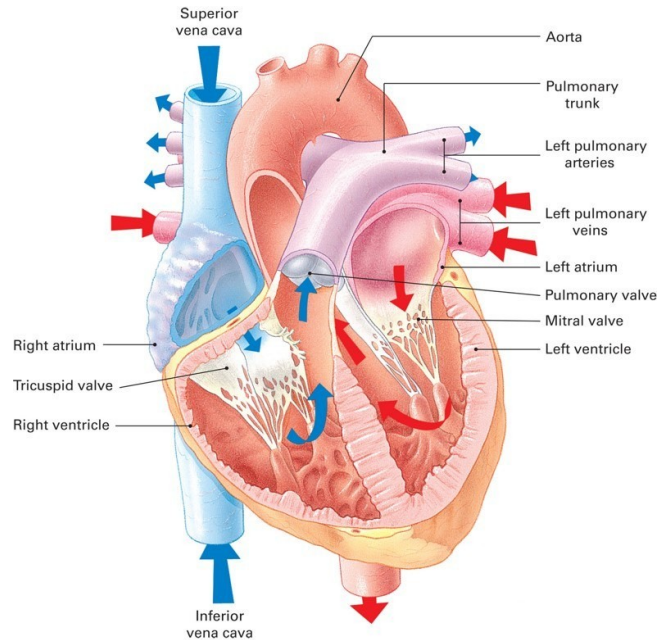


Figure 33-5 Blood flow through the heart.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

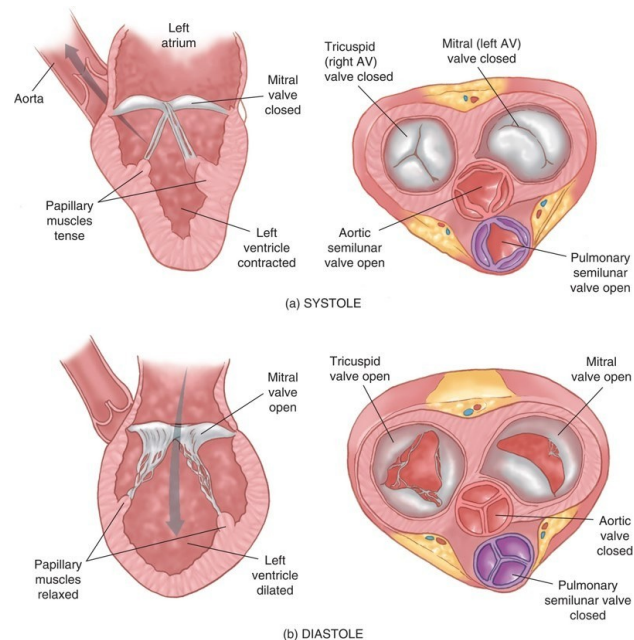


Figure 33-4 Valves of the heart — systole and diastole.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

1 Atria receive, ventricles pump

Atria are the superior receiving chambers. Ventricles are larger and inferior. The left ventricle is thickest because it works against systemic resistance — the right is thin-walled and tolerates a sudden afterload rise badly.

2 AV valves: tricuspid and mitral

Between atria and ventricles, anchored to papillary muscles so they cannot invert. Papillary rupture after an infarct causes acute mitral regurgitation and sudden pulmonary edema.

3 Semilunar valves: pulmonic and aortic

Between the ventricles and the great arteries. Pulmonic to the pulmonary artery on the right; aortic to the aorta on the left.

Recite the path — because backward failure follows it exactly

Vena cavae → right atrium → tricuspid → right ventricle → pulmonic → pulmonary artery → LUNGS → pulmonary veins → left atrium → mitral → left ventricle → aortic → aorta → body.

LEFT ventricular failure backs up into the lungs: pulmonary edema. RIGHT ventricular failure backs up into the body: JVD, peripheral edema, hepatic congestion. Know the circuit and you derive both — no list to memorize.

Two circuits, two pressures — and one pump driving both

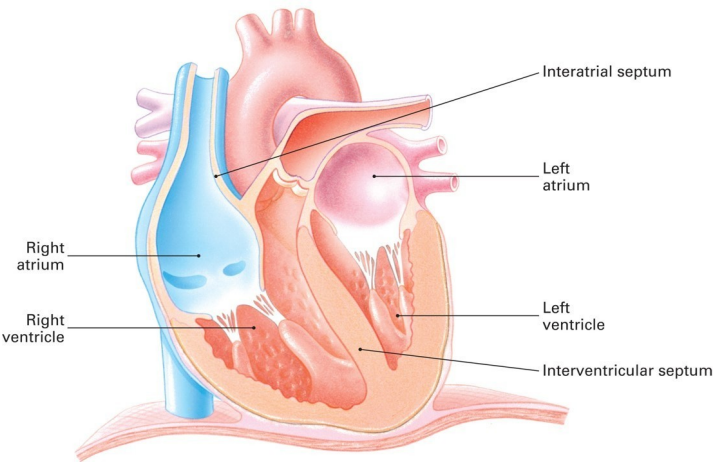


Figure 33-3 Chambers of the heart.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

	PULMONARY circuit	SYSTEMIC circuit
Driven by	Right ventricle	Left ventricle
Path	Pulmonary artery → lungs → pulmonary veins → left atrium	Aorta → body → vena cavae → right atrium
Pressure	LOW — roughly a quarter of systemic	HIGH
Wall thickness	Thin — built for low resistance	About three times thicker
Fails when	Afterload rises suddenly — a large pulmonary embolism	Contractility is lost — infarction
Backs up into	The body — JVD, peripheral edema	The lungs — pulmonary edema

The left ventricle is furthest from its own blood supply

Coronaries perfuse from the outside in, so the subendocardial layer of the thickest, hardest-working chamber is the first tissue to go ischemic and the last to recover — which is why ischemia so often appears first as ST depression in the left-sided leads.

Whatever the right side pumps, the left side must pump

Beat for beat. Mismatch for more than a few beats and blood accumulates in the circuit ahead of the weaker ventricle — the mechanical definition of congestive failure. It is also why left-sided failure eventually causes right-sided failure: the lungs become a high-resistance circuit, and that is exactly the afterload the right ventricle cannot handle.

Coronary circulation — the anatomy the 12-lead is reading

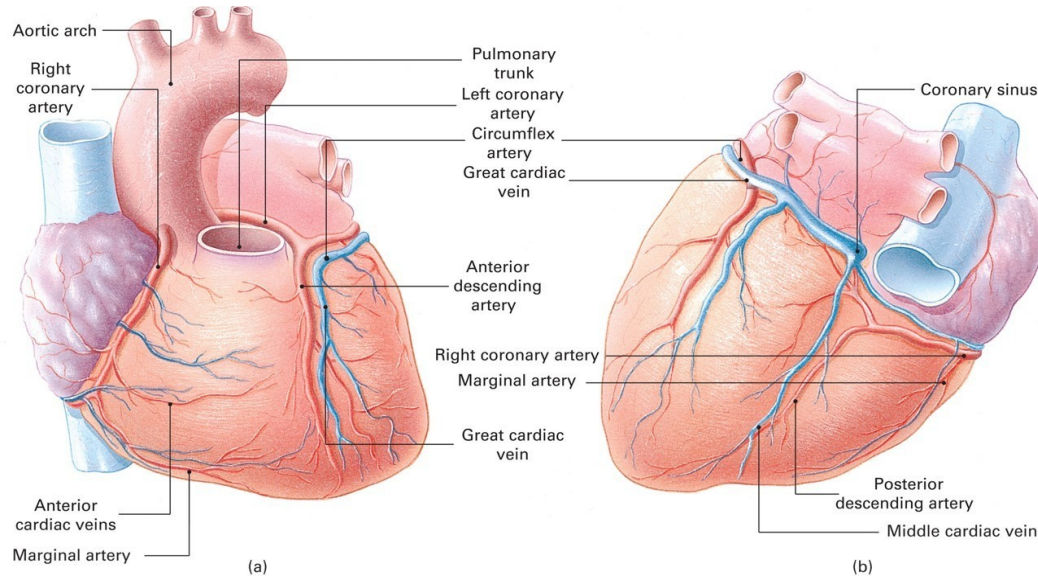


Figure 33-6 Coronary circulation — (a) anterior, (b) posterior.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

The heart perfuses itself during DIASTOLE

The myocardium squeezes its own vessels shut as it contracts, so coronary filling happens between beats. Two consequences: a very fast rate shortens diastole and therefore shortens coronary filling — tachycardia itself can cause ischemia in a narrowed artery. And it is DIASTOLIC pressure that drives coronary flow, which matters in shock and during CPR.

REGION SOP ACS pearl: “If ST Elevation noted in II, III, aVF: contact OLMC as NITROGLYCERIN may be contraindicated. Obtain V4R to evaluate right ventricular involvement. Do NOT transmit V4R.” Coronary anatomy, written as a rule.

1 It starts at the aortic root

Just above the leaflets of the aortic valve — which is why a backward-extending aortic dissection can occlude a coronary and present as an infarct.

2 LAD: anterior wall and septum

From the left main. The largest territory — an LAD occlusion is the classic large anterior MI that becomes pump failure.

3 Circumflex: lateral wall

The other branch of the left main.

4 RCA: inferior wall, right ventricle — and usually the SA and AV nodes

Which is why inferior infarcts arrive with bradycardia and heart block, and why they raise the right ventricular question.

The peripheral circulation — high pressure out, low pressure back

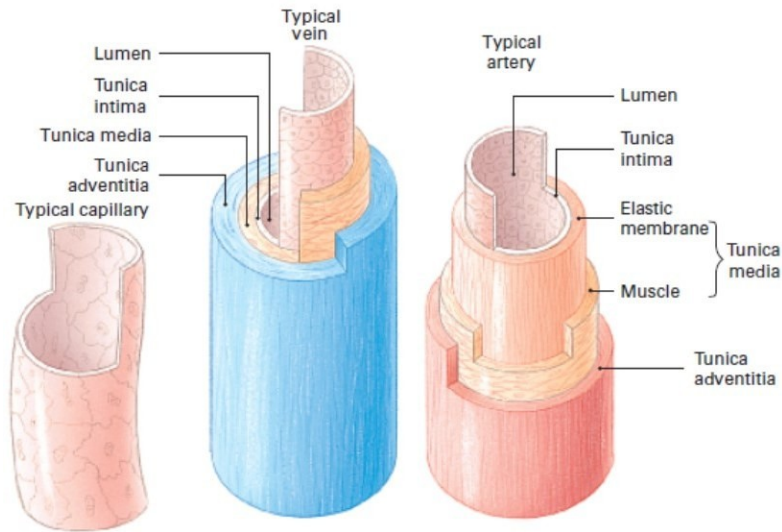


Figure 33-7 Layers of the peripheral vessels.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

1 Arteries: high pressure, resistance

Oxygenated blood under high pressure. Arteries → arterioles → capillaries. The ARTERIOLES set systemic vascular resistance, and therefore afterload — where every vasopressor and vasodilator acts.

2 Veins: low pressure, capacitance

Return under low pressure, helped by surrounding muscle and one-way valves. Capillaries → venules → veins → vena cavae → right atrium.

3 Roughly two-thirds of your blood volume is in the veins

They are the reservoir. Venodilation moves blood out of the central circulation into that reservoir and drops preload — which is exactly how nitroglycerin works.

4 Three layers in the wall

Intima, media (the muscular layer that constricts and dilates), adventitia. Thick media in arteries, thin in veins. Capillaries are one cell thick, which is what allows exchange.

Blood pressure = cardiac output × systemic vascular resistance

So a hypotensive patient has a PUMP problem, a VOLUME problem, or a RESISTANCE problem — and the treatment for each is different. Deciding which one you are looking at is most of cardiovascular management.

REGION SOP Cardiogenic Shock: “Verify and check often that lung sounds are clear” before fluid, then NORMAL SALINE in 500 mL increments titrated to MAP ≥ 65.

SECTION 2

Cardiac Physiology

The cycle, the volume it moves, and the nervous system that sets the pace.

The cardiac cycle · Stroke volume · Cardiac output · Autonomic control

The cardiac cycle — filling, then emptying

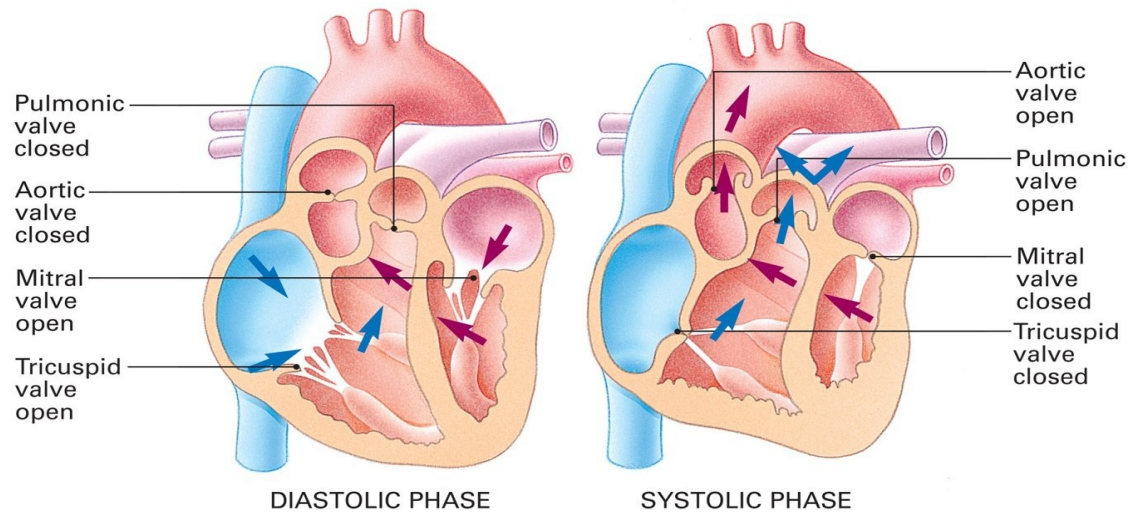


Figure 33-8 Relation of blood flow to cardiac contraction.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

1 DIASTOLE — relaxation and filling

Ventricular filling begins; blood enters the ventricles; the pulmonic and aortic valves are closed. Most filling is passive.

2 The atrial kick finishes the job

The atria contract just before the ventricles, adding the last fifth to a third of filling. Lose organized atrial activity — atrial fibrillation — and you lose it.

3 SYSTOLE — contraction and ejection

Blood leaves through the pulmonic and aortic valves; the mitral and tricuspid close to prevent backflow into the atria.

4 Ejection fraction — about two-thirds

The PROPORTION of ventricular contents ejected, not the amount. A failing ventricle can hold a normal volume and eject a smaller share of it.

Tachycardia steals from diastole — and diastole is when two things happen

The ventricle fills, and the coronary arteries fill. A very fast rate shortens both at once, which is why a sustained tachycardia can produce ischemia in a patient whose arteries were adequate at rest — and why rate control is treatment, not cosmetics.

REGION SOP Rapid Atrial Flutter / Fibrillation is its own protocol — the rhythm matters because the rate steals filling time and the lost atrial kick steals the rest.

Stroke volume — three variables, and what moves each

1 PRELOAD

End-diastolic volume — how full the ventricle is when it starts to squeeze. Set by venous return: volume, venous tone, and the time available to fill.

2 CONTRACTILITY

The force of the squeeze. Starling’s law: the more the muscle is stretched, the greater the force — UP TO A LIMIT.

3 AFTERLOAD

The resistance the ventricle must contract against — systemic vascular resistance for the left, pulmonary vascular resistance for the right.

Variable	Falls with	Rises with	Moved in the field by
PRELOAD	Hypovolemia · venodilation · tamponade · tension pneumothorax · a rate too fast to fill	Fluid · venous constriction · leg elevation	Normal saline · nitroglycerin (reduces it) · position
CONTRACTILITY	Ischemia and infarction · hypoxia · acidosis · electrolyte derangement	Catecholamines, endogenous or given	Push-dose epinephrine · correcting hypoxia and acidosis
AFTERLOAD	Vasodilation · distributive shock	Hypertension · vasoconstriction · aortic stenosis · PE for the right ventricle	Nitroglycerin at higher doses · vasopressors raise it

“Up to a limit” is the whole of heart failure in four words

Stretch the fibers past the optimum and force FALLS rather than rises. That is why a failing heart does not answer more filling the way a healthy one does, why fluid can worsen cardiogenic shock, and why the protocol tells you to check the lungs are clear before you give any.

Cardiac output — and the two ways a heart can fail to deliver

CARDIAC OUTPUT

The volume of blood pumped in one minute. Roughly 5 L/min at rest — about 70 mL at about 70 beats.

= STROKE VOLUME

Set by preload, contractility and afterload — the three variables from the previous slide.

× HEART RATE

Set by the conduction system and the autonomic nervous system.

1 Too SLOW — multiply by a small number

Below roughly 50 the output falls simply because rate is low. The body compensates with stroke volume until it cannot — then you have symptomatic bradycardia.

2 Too FAST — no time to fill

Above roughly 150, diastole becomes too short to fill the ventricle, so STROKE VOLUME collapses even though rate is high. A rate of 200 makes people hypotensive for this reason.

3 Pressure is not perfusion

BP = cardiac output × systemic vascular resistance. A patient can hold a normal pressure with a poor output by clamping down — that is compensated shock. Read mentation and skin, not just the cuff.

Draw the curve: output rises with rate, then falls

Ask the class where atropine moves a patient on that curve, and where cardioversion moves one. Both move the patient toward the same peak — from opposite sides.

REGION SOP Three protocols, one equation: Bradycardia acts on RATE (atropine, pacing). Rapid Atrial Fibrillation acts on RATE. Cardiogenic Shock acts on PRELOAD, then CONTRACTILITY with push-dose epinephrine.

MAP and pulse pressure — reading the numbers you get

1 **MAP — the average across the whole cycle**

Diastole occupies roughly two-thirds of the cycle, so diastolic pressure counts about twice as heavily. Estimate: diastolic + one-third of the pulse pressure.

2 **Why 65 is the number in every protocol**

Below a MAP near 65, autoregulation in the kidneys, brain and coronaries begins to fail. Organs are perfused by the average pressure, not the peak.

3 **Pulse pressure — a rough index of stroke volume**

Normally 30 to 40 mmHg. **NARROWING** means falling stroke volume or rising resistance — the compensating patient, and the classic tamponade finding.

4 **Coronary perfusion is a diastolic number**

Aortic diastolic pressure minus ventricular pressure during diastole. It is why interruptions in compressions cost so much — diastolic pressure falls in seconds and takes many compressions to rebuild.

Blood pressure	Pulse pressure	Estimated MAP	What it tells you
90 / 60	30	≈ 70 — above threshold	Lower systolic, but adequately perfusing
100 / 40	60	≈ 60 — below threshold	Higher systolic, perfusing LESS well. Read both numbers
120 / 100	20	≈ 107	Narrow pulse pressure — clamped down. Look for the reason before the systolic falls

Read both numbers. Reading only the systolic misses the compensating patient — and that patient is the one who still has time.

REGION SOP MAP ≥ 65 is the endpoint written into Acute Coronary Syndrome, Cardiogenic Shock, Bradycardia and Anaphylaxis — fluid and push-dose epinephrine are both titrated to it.

Autonomic control — two opposing systems, four receptors

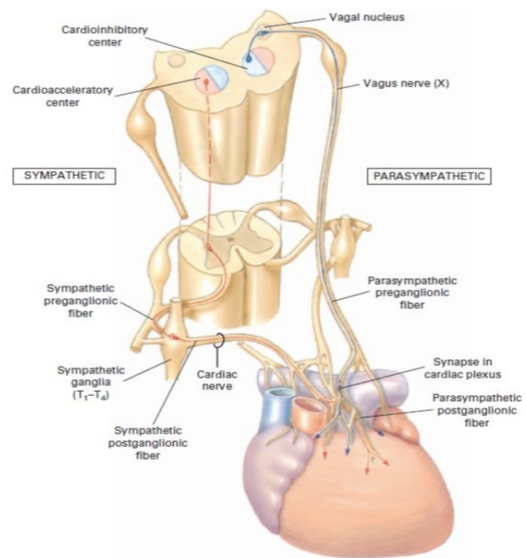


Figure 33-11 Nervous control of the heart.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

REGION SOP Bradycardia pearls: “Patients who have undergone cardiac transplant will not respond to ATROPINE” — a denervated heart has no vagal brake to block. And consider beta-blocker or calcium channel blocker overdose.

Receptor	Where	What it does	Drugs that use it
ALPHA	Peripheral blood vessels	VASOCONSTRICTION	Epinephrine · push-dose epinephrine · norepinephrine
BETA-1	Primarily the HEART	Increases rate and contractility	Epinephrine · dopamine. Blocked by beta-blockers
BETA-2	Lungs and peripheral vessels	BRONCHODILATION and peripheral vasodilation	Albuterol · epinephrine
MUSCARINIC (vagal)	SA and AV nodes	ACETYLCHOLINE slows rate and AV conduction	Blocked by atropine — which takes the brake off

CHRONOTROPY
Rate.

INOTROPY
Strength of contraction.

DROMOTROPY
Speed of impulse conduction.

One heart, two lungs — beta-1, beta-2
Three receptor lines explain most of the drug box. Epinephrine hits all three — pressure, rate and force, and open airways at once. Albuterol is selective for beta-2, so it opens airways and, spilling over, causes tremor and tachycardia. Beta-blockers block beta-1, which is why they slow the rate, weaken the squeeze, and make overdose a cardiac emergency.

The heart as an endocrine organ — and the electrolytes that run it

1 The heart is a pump AND a gland

Left ventricular dysfunction activates the sympathetic nervous system and the renin-aldosterone system, which maintain perfusion and pressure by raising rate and contractile force.

2 Compensation is why failure hides

Sympathetic drive raises rate and force; RAAS retains sodium and water, raising preload. Short term it holds the pressure up. Long term it IS chronic heart failure — a failing heart asked to work harder against more volume.

3 The patient is somewhere on that curve

The tachycardia, the vasoconstriction and the edema are the compensation, not the disease. When they fail, the patient decompensates fast.

4 Stretch also releases natriuretic peptides

Which oppose the retention — and are measured in hospital as BNP to assess heart failure. Recognize the term on a handover.

Electrolyte	What it does in the heart	Why you care
SODIUM	Drives DEPOLARIZATION — the inward rush that creates the action potential	Without it there is no impulse to conduct
POTASSIUM	Governs REPOLARIZATION and resting potential	The one most likely to kill your patient — high: peaked T waves, widening QRS, arrest; low: ectopy and torsades. Watch renal and dialysis patients
CALCIUM	Drives CONTRACTION — couples the electrical event to the mechanical one	Blocked therapeutically by calcium channel blockers; overdose is a cardiac emergency
MAGNESIUM	Stabilizes the membrane	Deficiency is associated with torsades de pointes
CHLORIDE	Follows sodium; contributes to acid-base balance	Part of the acidosis picture that depresses contractility

These are not laboratory trivia. Every one of them is a reason the heart in front of you is behaving badly — and several are correctable.

Myocardial oxygen supply and demand

SUPPLY — coronary blood flow

Arterial patency · diastolic filling TIME · diastolic PRESSURE. Flow happens between beats, not during them.

SUPPLY — oxygen content

Hemoglobin and saturation. Severe anemia, carbon monoxide and profound hypoxia can cause infarction with entirely patent arteries.

DEMAND — heart rate

The largest single determinant. More beats per minute is more work per minute.

DEMAND — contractility

A harder squeeze costs more oxygen.

DEMAND — wall tension

Set by afterload and ventricular size. A dilated ventricle working against high pressure is expensive.

Tachycardia hits BOTH sides of the balance at once

More beats to fuel, and less diastole in which to fuel them. A rate of 180 in a narrowed artery generates ischemia regardless of what caused the rate — which is the whole argument for rate control being treatment rather than cosmetics.

Every treatment for acute coronary syndrome moves one side of this balance

SUPPLY: oxygen raises content when the patient is hypoxic · aspirin now and reperfusion in hospital restore flow through the artery · rate control restores diastolic filling time.

DEMAND: nitroglycerin lowers preload and wall tension · analgesia cuts the catecholamine drive raising rate and contractility · rate control lowers demand at the same time.

SECTION 3

Electrophysiology

Charge, then conduction, then contraction — in that order, every single beat.

Syncytia · Depolarization · The conduction system · Intrinsic rates

Two syncytia, one gate — and the charge that crosses them

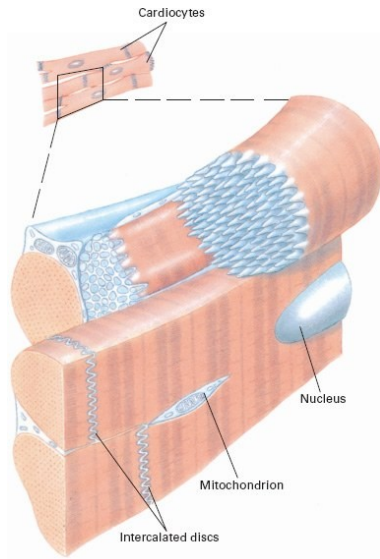


Figure 33-12 Cardiac muscle and intercalated discs.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

1 Intercalated discs make it one unit

They connect the fibers and conduct impulses, so stimulating one cell spreads the impulse through the whole mass.

2 Two syncytia, opposite directions

The ATRIAL syncytium contracts superior to inferior, pushing blood down into the ventricles. The VENTRICULAR syncytium contracts inferior to superior, pushing blood up into the great vessels.

3 A fibrous gate separates them

It PREVENTS conduction between atria and ventricles. The AV bundle is the ONLY electrical route across — which is why AV block disconnects them, and why an accessory pathway is so consequential.

4 Resting, then action, then back

The Na-K pump moves 3 sodium out for 2 potassium in, leaving the inside negative — RESTING potential. Sodium then rushes IN — ACTION potential. Sodium is pumped out again — REPOLARIZATION.

Electrical is not mechanical

Depolarization is an electrical event; contraction is a mechanical event that follows it. They can be separated — and the name for that separation is PULSELESS ELECTRICAL ACTIVITY. Saying this now makes PEA obvious in Part 2 instead of mysterious.

Refractoriness — and why cardioversion is synchronized

A cell cannot be re-excited until it repolarizes, which protects the heart from tetany. The vulnerable window at the end of that period is why a shock landing on the T wave can induce ventricular fibrillation — so the machine waits for the R wave.

The action potential in phases — and the windows it creates

Phase	What is happening	Ion movement	Seen on the ECG as
4 Resting	Held near - 90 mV by the sodium-potassium pump	3 Na ⁺ out for 2 K ⁺ in	The baseline
0 Depolarization	Fast sodium channels open; the cell swings positive	Na ⁺ rushes IN	The QRS upstroke
1 Early repolarization	Sodium channels close	Na ⁺ entry stops	The start of the ST segment
2 Plateau	Calcium enters slowly while potassium leaves. The feature that separates cardiac from skeletal muscle — and prevents tetany	Ca ²⁺ in, K ⁺ out	The ST segment
3 Repolarization	Potassium leaves in quantity; the cell returns toward rest	K ⁺ OUT	The T wave

1 Slow-response cells pace themselves

The SA and AV nodes depolarize with CALCIUM, not fast sodium — and their phase 4 drifts upward to threshold instead of sitting flat. That drift IS automaticity.

2 Which is why the autonomics work

Sympathetic stimulation steepens the drift and the rate rises; vagal stimulation flattens it and the rate falls. And calcium channel blockers slow both rate and AV conduction, because those tissues run on calcium.

3 ABSOLUTE refractory

Phases 0 through most of 3. No stimulus of any strength produces another beat — the protection against tetany.

4 RELATIVE refractory — the vulnerable window

End of phase 3, on the upslope of the T wave. A strong enough stimulus fires, and spreads abnormally through partly repolarized tissue.

A stimulus landing in that window — an R-on-T complex, or an unsynchronized shock — can induce ventricular fibrillation. That is why cardioversion waits for the R wave, and why R-on-T ectopy is treated as a warning.

The conduction system, and the four properties that make it work

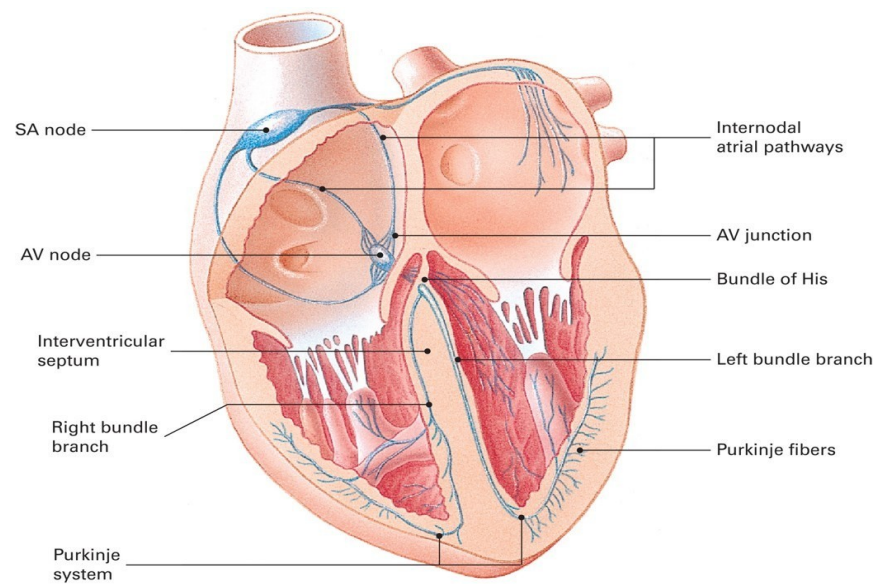


Figure 33-16 Cardiac conductive system.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

The AV node delays on purpose

That delay gives the atria time to finish emptying before the ventricles fire — it is the atrial kick. You will meet it again as the PR interval.

Excitability

Responds to a stimulus.

Conductivity

Passes the impulse on.

Automaticity

Self-generates an impulse.

Contractility

Retains the ability to contract.

Pacemaker	Intrinsic rate	What it means when it is in charge
SA node	60–100	Normal. The fastest pacemaker wins, so it normally sets the rate
AV node / junction	40– 60	The SA node has failed or slowed — a junctional escape rhythm
Purkinje system	15– 40	Both have failed — an idioventricular escape, wide and slow

A hierarchy with a fail-safe — and the rate tells you the level

Every escape rhythm in Part 2 is this hierarchy doing its job. Regular at 45 with no P waves is junctional. Regular at 30 and wide is ventricular. You are reading the rate to find out which pacemaker is driving.

From wiring to waveform — the bridge to Part 2

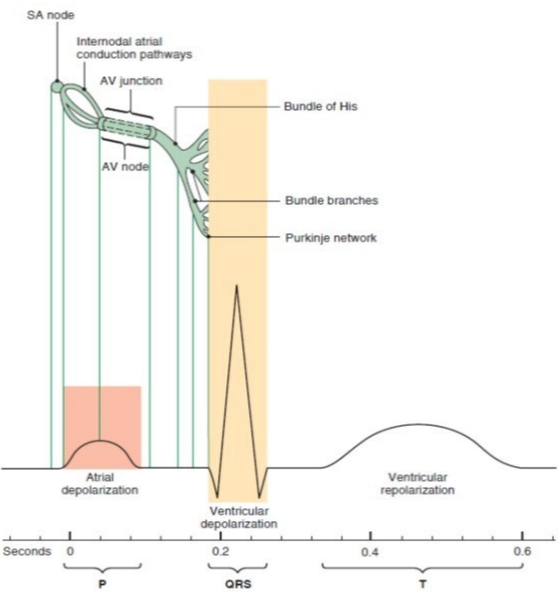


Figure 33-20 The ECG and the heart's electrical activity.

Source: Paramedic Care: Principles & Practice, 6th ed. © Pearson Education.

Treat the patient

A rhythm is an electrical finding. Perfusion is the clinical one.

On the ECG	Electrical event	Mechanical event happening
P WAVE	Atrial depolarization	The atrial kick completes ventricular filling
PR INTERVAL	The deliberate delay at the AV node	The atria finish emptying before the ventricles fire
QRS COMPLEX	Ventricular depolarization	Ventricular systole begins just after. Atrial repolarization hides inside it
ST SEGMENT	Between depolarization and repolarization	The ventricle is contracting. This is the segment that shifts in injury — all of Part 5
T WAVE	Ventricular repolarization	Diastole begins — filling, and coronary perfusion

Systole or diastole? Read it off the strip

Systole runs from the QRS through the ST segment. Diastole begins around the T wave and runs to the next QRS. The electrical event always precedes the mechanical one it causes.

REGION SOP Every cardiac protocol branches on STABLE versus UNSTABLE — altered mental status, MAP under 65 or SBP under 90 — not on the name of the rhythm.

SUMMARY

What Part 1 gives you for the rest of the chapter

1 One organ, four failures

PUMP, PIPE, WIRING, CONTAINER. Most real patients have more than one — a large anterior MI is a pipe failure that becomes a pump failure and often a wiring failure.

2 Trace the path, derive the failure

Left ventricular failure backs up into the lungs. Right ventricular failure backs up into the body. You never have to memorize those signs if you know the circuit.

3 The RCA feeds the inferior wall, the right ventricle, and usually the nodes

Which is why inferior infarcts bring bradycardia, heart block, and the right ventricular question.

4 Stroke volume has three variables

Preload, contractility, afterload. Sort the drug box by which one it moves and the pharmacology stops being a list.

5 Cardiac output = stroke volume $\times$ heart rate

Two terms, two ways to fail. Too slow multiplies by a small number; too fast leaves no time to fill.

6 The pacemaker hierarchy is a fail-safe

SA 60–100, AV 40–60, Purkinje 15–40. The fastest wins — so the rate tells you which one is driving.

7 Two pumps, two circuits, two pressures

Whatever the right side pumps, the left must pump. The thin right ventricle fails against sudden afterload; the thick left ventricle fails when it loses contractility.

8 Ischemia is a balance, not an event

Demand is rate, contractility and wall tension. Supply is coronary flow and oxygen content. Tachycardia worsens both sides at once.

Next: Part 2 — ECG monitoring and arrhythmia analysis

Ninety minutes, the largest single block in the chapter, and built entirely on this deck. Every rhythm you will learn to name is a statement about where an impulse started and how it traveled.

The 71-year-old — physiology, not misfortune

Which artery

ST elevation in II, III and aVF is the INFERIOR wall — usually the RIGHT CORONARY ARTERY, which also feeds the right ventricle and, in most hearts, the SA and AV nodes. That is why he was already bradycardic at 52.

Why clear lungs matter

They rule out acute LEFT ventricular failure as the cause. If the left side had failed, blood would back up along the path you traced and the lungs would be wet. Clear lungs plus hypotension in an inferior MI points RIGHT.

What the nitroglycerin removed

PRELOAD. It is a venodilator, and two-thirds of the volume already sits in the veins. An infarcted right ventricle has lost contractility — and a poorly contracting ventricle depends entirely on filling pressure to generate any stroke volume.

What you would have wanted — and what he needs now

A V4R to look at the right ventricle before giving a venodilator. And now: FLUID. This is the cardiac patient whose hypotension is corrected with volume — after verifying the lungs are clear.

Nothing here required a memorized rule. The anatomy told you which ventricle was hurt, the stroke volume equation told you what it needed, and the SOP pearl is that same reasoning, pre-written.

Check your understanding

- 1 Name the three layers of the heart and the disease family that lives in each.
- 2 Where are the apex and the base, and what landmark is each near?
- 3 Trace a red blood cell from the vena cava to the aorta, naming every chamber and valve.
- 4 Left ventricular failure backs up where? Right ventricular failure backs up where?
- 5 Where do the coronary arteries originate, and during which phase do they fill?
- 6 Which wall does each of the LAD, circumflex and RCA supply — and which also feeds the nodes?
- 7 What allows collateral circulation when a coronary artery narrows?
- 8 Name the three determinants of stroke volume. How does ejection fraction differ from stroke volume?
- 9 State Starling's law — including the three words at the end that explain heart failure.
- 10 Give the cardiac output equation, and explain how a rate of 40 and a rate of 200 both reduce it.
- 11 Locate the alpha, beta-1 and beta-2 receptors and give the effect of each.
- 12 Which neurotransmitter slows the heart, by which nerve, and which drug blocks it?
- 13 Define chronotropy, inotropy and dromotropy.
- 14 Which two systems does left ventricular dysfunction activate, and what is the long-term cost?
- 15 Which electrolyte is most likely to kill your patient, and what does it do at high and low levels?
- 16 Describe the sodium-potassium pump and what changes during depolarization.
- 17 What separates the atrial and ventricular syncytia, and what is the only way across?
- 18 Name the four properties of the conduction system.
- 19 Give the intrinsic rate of the SA node, the AV node and the Purkinje system — and state which one takes charge.
- 20 Match each ECG wave to its electrical event. Which part of the strip is systole?
- 21 Why is the right ventricle so intolerant of a sudden rise in afterload?
- 22 A patient is 100/40. Estimate the MAP and say whether it clears the protocol threshold.
- 23 Name the three determinants of myocardial oxygen demand, and the two of supply.
- 24 Which phase of the action potential prevents tetany — and which creates the vulnerable window?

Answer these before the session and again after — that gap is the measure of what you learned.