

Slow heartbeat

The heart beats too slowly to pump enough blood for the body's needs.

THE STORY IN ONE SENTENCE

The heart beats too slowly to pump enough blood for the body's needs.

“A slow heart rate is not always a disease — the key question is whether the slow rate causes symptoms like dizziness, tiredness, or fainting.”

Normal anatomy — the body's job

The SA node (the heart's natural pacemaker at the top of the right atrium) fires an electrical signal 60–100 times per minute. The signal travels to the AV node (the relay station between the upper and lower chambers), pauses briefly, then spreads through the bundle of His and Purkinje fibers to make the ventricles (lower pumping chambers) squeeze and push blood out to the body.

BODY'S JOB

Understanding how the electrical signal normally travels through the heart helps you recognize where the system breaks and why symptoms appear.

1. SA node fires
2. Signal spreads across the atria
3. Signal reaches the AV node
4. Signal travels through the bundle of His
5. Purkinje fibers activate the ventricles
6. Cycle repeats

What breaks

Usually one broken part of the heart's electrical system explains all the symptoms together.

CLINICAL TERM

SA node dysfunction (sick sinus syndrome): the SA node fires too slowly or pauses, often from aging, low blood flow (ischemia), or medication. AV node block: first-degree (signal slows but passes), second-degree type I (Wenckebach — signal slows more each beat until one is dropped) or type II (signal suddenly drops without warning), or third-degree (complete heart block — no signal passes, so the ventricles beat on their own backup rhythm at 20–40 bpm). Infranodal block: damage below the AV node in the bundle branches, often from heart attack (myocardial tissue death from blocked blood flow (infarction)) or scar tissue (fibrosis). Medications: beta-blockers, calcium channel blockers (diltiazem, verapamil), digoxin, antiarrhythmics (amiodarone), and opioids all slow the electrical signal. The break upstream (in the electrical wiring) causes low cardiac output (amount of blood the heart pumps per minute) downstream, and every symptom traces back to that.

How the body compensates

The body makes up for a slow heart rate (bradycardia) by squeezing harder with each beat (increasing stroke volume) and tightening blood vessels to keep blood pressure up — so vital signs may look stable even when organs are not getting enough blood. Your job is to recognize early signs that compensation is failing before sudden collapse.

1. {'how': 'Increase stroke volume (amount of blood pushed per beat)', 'why': 'The heart squeezes harder with each beat (increased contractility) to push more blood per beat and offset the slow rate — this keeps cardiac output (total blood pumped per minute) closer to normal.'}
2. {'how': 'Vasoconstriction (blood vessels tighten)', 'why': 'Blood vessels tighten to keep blood pressure up even though cardiac output is falling — this masks the problem because blood pressure may look normal on the monitor.'}
3. {'how': 'Increased sympathetic tone (adrenaline release)', 'why': 'The body releases adrenaline (epinephrine) and norepinephrine to make the heart beat faster and harder — but if the electrical system is blocked, this signal cannot get through and the compensation fails.'}
4. {'how': 'Fluid retention (kidneys hold onto water and salt)', 'why': 'The kidneys sense low blood flow and activate the renin-angiotensin-aldosterone system (RAAS), holding onto water and salt to raise blood volume and pressure — this helps in the short term but can cause swelling (edema) over time.'}

5. {'how': 'Watch for decompensation (compensation failing)', 'why': 'This compensation can mask the problem — patients may have normal blood pressure and look stable while their organs are slowly starving for oxygen. The crash point to watch for: sudden cardiac arrest (asystole — heart stops), dangerously fast rhythm from the ventricles (ventricular a fast heart rate (tachycardia)), or pulseless electrical activity (heart signal shows on the monitor but no pulse).'} }
6. {'how': 'Risk is highest during these triggers', 'why': 'Risk jumps during medication changes, electrolyte imbalance (especially high potassium (hyperkalemia) — potassium too high), or acute starved blood flow (ischemia) (new blockage cutting blood flow to the electrical system).'} }
7. {'how': 'Watch trends, not just one set of numbers', 'why': 'Watch for worsening trends — rising lactate (waste product from oxygen-starved tissues), falling urine output, increasing confusion, or new chest pain — not just a single set of vital signs. A patient with stable vital signs but worsening lactate or mental status is decompensating and needs urgent help.'} }

Domino effect

1. Heart rate drops below 60 bpm (bradycardia) — The SA node fires too slowly, or electrical signals are blocked in the AV node or bundle branches, so fewer beats reach the ventricles each minute.
2. Cardiac output falls (amount of blood pumped per minute decreases) — Cardiac output equals heart rate times stroke volume — if rate drops and stroke volume cannot rise enough to make up for it, total output falls.
3. Blood pressure drops (hypotension) — Lower cardiac output means less blood pushing through the arteries each minute, so pressure falls unless blood vessels tighten enough to hold it up.
4. Brain receives less oxygen (cerebral poor blood flow (hypoperfusion)) — The brain needs constant oxygen delivery — when cardiac output and blood pressure drop, less oxygen-rich blood reaches brain cells, causing confusion, dizziness, or fainting (syncope) (fainting).
5. Muscles and organs become tired and weak — Skeletal muscles and organs like the kidneys and liver do not get enough oxygen and glucose (fuel), so the patient feels weak, tired, and short of breath even at rest.
6. Kidneys reduce urine output (oliguria) — Low blood flow to the kidneys signals them to hold onto water and salt to try to raise blood volume and pressure, so urine output drops.
7. Lactic acid builds up (lactic too much acid in the blood (acidosis)) — Cells switch to anaerobic metabolism (making energy without oxygen) when oxygen delivery is too low, producing lactate as a waste product that piles up in the blood.

8. Risk of sudden cardiac arrest (asystole or pulseless electrical activity) — If the heart rate drops too low or the electrical system completely fails, the heart may stop pumping altogether (asystole) or show electrical activity on the monitor but no pulse (pulseless electrical activity) — both are life-threatening emergencies.

Symptoms · what the patient feels, and why

- **Feeling tired and weak:** When your heart beats too slowly, it pumps less blood each minute. Your muscles and organs don't get enough oxygen and glucose (the sugar fuel cells burn), so you feel tired even when you haven't done much.
- **Feeling dizzy or lightheaded:** Your brain needs a steady stream of blood. When your heart beats slowly, less blood reaches your brain (like turning down a faucet), so you feel dizzy or like the room is spinning—especially when you stand up quickly.
- **Fainting or feeling like you're about to faint:** If your heart rate drops suddenly or stays very low, not enough blood reaches your brain and you can black out. This is a danger sign—it means your brain isn't getting the oxygen it needs to stay awake.
- **Shortness of breath:** A slow heartbeat can't move blood through your lungs fast enough. Blood backs up in the lungs (like water pooling behind a slow drain), making it harder to breathe. Your body also needs more oxygen than the slow heart can deliver.
- **Chest pain or pressure:** Your heart muscle itself needs oxygen-rich blood. If the heart beats too slowly—or if the slow beat is caused by blocked coronary arteries (the pipes feeding the heart)—the heart muscle doesn't get enough oxygen (called starved blood flow (ischemia)) and you feel pain or tightness in your chest.
- **Confusion or trouble thinking clearly:** Brain cells need a constant supply of oxygen to work. When blood flow slows down, not enough oxygen reaches the cerebral cortex (the thinking part of your brain), so you may become confused, slow to answer, or unable to focus. This is a warning that your brain isn't getting enough fuel.
- **Can't do your usual activities without getting exhausted:** Normally, your heart speeds up when you move around. If it can't speed up (a problem called chronotropic incompetence—the heart's inability to raise its rate when your body needs it), your muscles don't get the extra oxygen they need during activity, so you tire out fast.
- **Low blood pressure:** Blood pressure depends on how fast your heart beats and how much blood it squeezes out with each beat. If the heart beats too slowly and can't pump more blood per beat to make up for it, your blood pressure drops below what your organs need to work properly.

- Heart fluttering or skipping beats: Sometimes a slow heart rhythm is uneven—beats may be skipped or extra beats may pop in. You feel this as a flutter or thump in your chest. It happens when the sinoatrial node (your heart's natural pacemaker in the upper right chamber) or the atrioventricular node (the relay station between the upper and lower chambers) aren't firing electrical signals in a steady pattern.
- Cold, clammy skin and feeling very unwell: When your heart can't pump enough blood, your body tries to save blood for vital organs by narrowing blood vessels in your skin through the sympathetic nervous system (your body's emergency alert system). Your skin feels cold and sweaty, and you may feel a sense of dread or panic—signs that your body is in distress.

Focused exam

- Heart rate below 60 beats per minute, measured by pulse or monitor: A slow heartbeat — under 60 beats per minute, called a slow heart rate (bradycardia) — means the heart's electrical system is not firing or sending signals at a normal speed. This can happen because the heart's natural pacemaker (a small cluster of cells in the right upper chamber called the SA node, which normally fires 60 to 100 times per minute) is firing too slowly, because medicines are putting the brakes on the signals, or because there is a break (called a block) in the wiring between the upper and lower chambers so the signal cannot get through. Even though some athletes have slow resting heart rates and feel fine because their hearts pump a large amount of blood with each beat, a slow heartbeat that causes dizziness, fatigue, or shortness of breath means the heart is not pumping enough blood to meet the body's needs.
- Blood pressure drops or dizziness when standing up: When the heart beats too slowly, it pumps less blood every minute. The total amount of blood pumped per minute is called cardiac output, and it equals heart rate times stroke volume (the amount pumped with each beat). Less blood pumped means less blood reaching the brain. The brain needs steady fresh blood carrying oxygen and sugar to work, so when blood flow drops too low, you feel dizzy or lightheaded, like you might faint. This tells you the slow heartbeat is already cutting blood delivery below the level the body needs, especially when you stand up and gravity pulls blood into your legs.
- Pale or cool skin, or sweating without exercise: These are signs the body has turned on its emergency system. When the heart pumps too little blood, sensors in blood vessels (called baroreceptors, which sit in the neck and chest) detect the drop in pressure and trigger the sympathetic nervous system (the body's fight-or-flight system, which releases adrenaline and norepinephrine). To keep blood pressure from falling too low, blood vessels in the skin and gut squeeze tight (this is called vasoconstriction), which makes skin pale and cool because less warm blood is flowing near the surface. The body may also sweat. These signs mean the slow heartbeat is causing real trouble with blood flow and the body is trying to protect the brain and heart.

- Shortness of breath or fatigue with light activity: A slow heartbeat means less blood pumped every minute, so the body gets less oxygen delivered each minute. The brain senses this drop in oxygen (through sensors called chemoreceptors) and tells the lungs to breathe faster to try to pull in more oxygen. If you feel short of breath or very tired when your heart is beating slowly — especially with light activity like walking across a room — it means your body is not getting enough oxygen to do normal work. This is a sign that cardiac output is too low.
- Confusion or trouble thinking clearly: The brain uses about 20 percent of the body's oxygen every second even though it is only 2 percent of body weight, and it has no reserve tank — it depends on constant fresh blood flow. When a slow heartbeat cuts blood flow to the brain below the level needed for normal thinking (a mean arterial pressure below about 60 mmHg), confusion or foggy thinking sets in. This is a medical emergency because it means the brain is starving for oxygen right now.
- ECG (heart tracing) shows a long pause between waves, missing beats, or no connection between upper-chamber and lower-chamber signals: The ECG (a paper tracing of the heart's electrical activity) shows exactly where the problem is. The PR interval (the time from the P wave, which is the upper-chamber signal, to the QRS complex, which is the lower-chamber beat) should be 0.12 to 0.20 seconds. A PR interval longer than 0.20 seconds means first-degree AV block — a delay at the relay station called the AV node (a cluster of cells between the upper and lower chambers that controls how fast signals pass through). Missing QRS complexes after some P waves mean second-degree AV block — some signals are blocked completely. No relationship at all between P waves and QRS complexes means third-degree (complete) block — the upper and lower chambers are beating independently because no signal is getting through. The pattern tells you whether the problem is in the natural pacemaker (SA node), the relay station (AV node), or the deeper wiring below the relay station (the bundle of His and bundle branches, which carry the signal down into the lower chambers).

Diagnostics · every test answers a question

- 12-lead electrocardiogram (ECG) → What is the heart rate, and where exactly is the electrical problem — in the natural pacemaker, the relay station, or the lower wiring?
- Serum potassium and magnesium → Are minerals in the blood out of balance and slowing the heart's electrical signals?
- Thyroid-stimulating hormone (TSH) and free T4 → Is an underactive thyroid gland (hypothyroidism) slowing the heart rate?
- Medication review (especially beta-blockers, calcium channel blockers, digoxin, and antiarrhythmic drugs) → Is the patient taking medicines that slow the heart or block electrical signals?

- High-sensitivity troponin I or troponin T → Is a heart attack damaging the heart's electrical system right now?
- Continuous heart monitoring (telemetry) → Is the slow heartbeat constant, or does it come and go? Are there dangerous pauses (times when the heart stops beating for several seconds)?
- Serum creatinine and blood urea nitrogen (BUN) → Are the kidneys failing and causing potassium buildup or drug accumulation that slows the heart?
- Digoxin level (if the patient is taking digoxin) → Is digoxin toxicity slowing the heart?

Treatment logic

- Atropine 1 mg IV push, can repeat every 3 to 5 minutes up to a total of 3 mg → Atropine blocks the body's brake system (the vagus nerve's parasympathetic signals, which release acetylcholine, a chemical messenger that slows the heart). By blocking acetylcholine receptors at the SA node (natural pacemaker) and AV node (relay station), atropine speeds up the heart rate within 1 to 2 minutes. This increases the amount of blood pumped every minute (cardiac output), which raises blood pressure and restores blood flow to the brain and body. ACLS (Advanced Cardiovascular Life Support) guidelines recommend atropine as the first drug for symptomatic a slow heart rate (bradycardia).
- Transcutaneous pacing (TCP) or transvenous temporary pacemaker wire → Transcutaneous pacing uses large sticky electrode pads on the chest and back to deliver electrical shocks (usually 50 to 100 milliamps, about the same power as a strong muscle stimulator) that make the heart's lower chambers (ventricles) squeeze at a set rate, usually 60 to 80 beats per minute. A transvenous pacemaker threads a thin wire through a large vein (usually the internal jugular vein in the neck or subclavian vein below the collarbone) into the right lower chamber to deliver electrical pulses directly to the heart muscle. Both methods force the heart to beat at a safe speed within seconds, immediately increasing cardiac output and restoring blood flow to the brain and body. ACLS says use pacing for symptomatic a slow heart rate (bradycardia) that does not respond to atropine.
- Stop or reduce beta-blockers, calcium channel blockers (diltiazem, verapamil), digoxin, and antiarrhythmic drugs (amiodarone, sotalol) → Removing or lowering the dose of medicines that slow the heart's pacemaker (SA node) or relay station (AV node) allows the heart's own electrical system to speed back up. Heart rate usually rises within hours to days, depending on how long the drug stays in the body. For example, metoprolol's effect wears off in about 12 to 24 hours, but amiodarone can take weeks to months to clear because it dissolves in fat and tissues and is released back into the blood very slowly.

- Correct high potassium with calcium gluconate or calcium chloride IV, insulin with dextrose IV, sodium bicarbonate IV if acidotic, and sodium polystyrene sulfonate (Kayexalate) or patiomer by mouth, or hemodialysis; replace low magnesium with magnesium sulfate IV; treat hypothyroidism with levothyroxine by mouth → Calcium gluconate (1 to 2 grams IV over 2 to 3 minutes) or calcium chloride (0.5 to 1 gram IV over 2 to 3 minutes) stabilizes heart cell membranes and protects the heart from the dangerous rhythm effects of high potassium within 1 to 3 minutes, though it does not lower potassium itself. Regular insulin (10 units IV) with dextrose (25 grams, or one amp of D50, IV) pushes potassium from the blood into cells by activating the sodium-potassium pump, lowering blood potassium by 0.5 to 1.2 mEq/L within 15 to 30 minutes. Sodium bicarbonate (50 to 100 mEq IV) also shifts potassium into cells if the blood is acidic (pH below 7.35). Kayexalate or patiomer removes potassium through the gut over hours to days by binding it in the intestine so it comes out in the stool. Hemodialysis (filtering the blood through a machine) removes potassium directly from the blood and is the fastest definitive treatment for severe high potassium (hyperkalemia). Magnesium sulfate (1 to 2 grams IV over 15 minutes) replaces missing magnesium and restores normal heart cell electrical function. Levothyroxine (starting dose 1.6 mcg per kg by mouth once daily, usually 100 to 125 mcg in adults) replaces missing thyroid hormone; heart rate usually improves over 2 to 4 weeks as thyroid levels normalize. Fixing these metabolic and hormonal problems removes the causes of a slow heart rate (bradycardia).
- Epinephrine infusion 2 to 10 mcg per minute IV or dopamine infusion 5 to 20 mcg per kg per minute IV if atropine fails and pacing is not immediately available → Epinephrine and dopamine are hormones (chemical messengers released by the adrenal glands) that stimulate beta-1 receptors (the gas pedal) on the heart's pacemaker and relay station, speeding up electrical firing and conduction. This raises heart rate and increases the force of each heartbeat (called inotropy), which increases cardiac output and blood pressure. ACLS guidelines say use these infusions as a temporary bridge when atropine does not work and pacing is delayed or not yet ready.

- Permanent pacemaker implantation for symptomatic sinus node dysfunction (sick sinus syndrome), Mobitz type II second-degree AV block, third-degree (complete) AV block, or symptomatic a slow heart rate (bradycardia) when necessary heart-slowing medicines cannot be stopped → A permanent pacemaker is a small metal device (about the size of a silver dollar) placed under the skin below the collarbone, with one or two thin insulated wires (leads) threaded through a vein into the heart's right upper chamber (atrium) and/or right lower chamber (ventricle). The device constantly watches the heart's own electrical signals. When it detects a heart rate below a programmed threshold (usually 50 to 60 beats per minute) or senses a pause (no heartbeat for a set time, usually 2 to 3 seconds), it delivers a small electrical pulse (about 2 to 5 volts, barely enough to feel) to make the heart beat. This guarantees a reliable heart rate, prevents fainting and sudden death, and allows the person to live a normal active life. ACC/AHA/HRS (Heart Rhythm Society) guidelines give the strongest recommendation (Class I, meaning the benefit is proven and the treatment is definitely recommended) for permanent pacing in symptomatic sinus node dysfunction, any type of second-degree or third-degree AV block that causes symptoms, Mobitz type II block (even without symptoms, because it can suddenly progress to complete block), and third-degree AV block (even without symptoms, because the backup rhythm is unreliable).
- Treat acute heart attack (myocardial infarction) (heart attack) if present: aspirin 162 to 325 mg chewed immediately, anticoagulation with heparin or enoxaparin, and emergency coronary revascularization with percutaneous coronary intervention (PCI, cardiac catheterization with stent) or coronary artery bypass graft (CABG) surgery → If a heart attack is damaging the heart's electrical system — especially the AV node in an inferior (bottom-wall) heart attack caused by blockage of the right coronary artery — restoring blood flow to the heart muscle can stop further damage and sometimes allow the electrical system to recover. Aspirin stops platelets (tiny blood cells that form clots) from clumping and keeps the clot from getting bigger. Heparin or enoxaparin (anticoagulants, also called blood thinners) prevent new clots from forming. Opening the blocked artery with a balloon and stent (PCI, where a thin tube is threaded through an artery in the wrist or groin up to the heart, then a balloon is inflated to squash the blockage and a small metal mesh tube called a stent is left in place to hold the artery open) or surgically bypassing the blockage (CABG, where a vein from the leg or artery from the chest is sewn around the blockage to create a detour) restores oxygen delivery to both the heart muscle and the conduction tissue (SA node, AV node, bundle branches). ACC/AHA guidelines recommend emergency reperfusion (opening the artery) within 90 minutes of first medical contact for ST-elevation heart attack (myocardial infarction) (STEMI, the most severe type of heart attack where a coronary artery is completely blocked).

Medications

Atropine · Anticholinergic (blocks acetylcholine, the chemical that slows the heart)

Atropine covers up the landing spots where acetylcholine—the chemical that tells your heart to slow down—normally docks. The vagus nerve (a long nerve running from your brain down to your heart) releases acetylcholine to put the brakes on. When atropine blocks those landing spots (called muscarinic receptors), acetylcholine cannot land, so the brakes lift and the heart's natural pacemaker (the SA node, a small cluster of spark-making cells in the top-right chamber) speeds up. Think of it like unplugging the brake pedal—the engine can now go faster. Atropine works best when the slow heart is caused by too much vagus brake or by drugs that copy what the vagus does.

1. Atropine enters the bloodstream (usually through an IV) and travels quickly to the heart.
2. It binds to (covers up) muscarinic M2 receptors on SA node and AV node cells—these are the docking sites where acetylcholine normally lands.
3. With the receptors blocked, acetylcholine released by the vagus nerve cannot land and send its slow-down signal.
4. The SA node fires faster (makes electrical sparks more often), so heart rate goes up.
5. The AV node lets electrical signals pass through faster, so the signal from atria to ventricles is not delayed as much.

SAFETY WATCH

Never give less than 0.5 mg IV at once—smaller doses can make the heart even slower (paradoxical slowing) because they block receptors in the brain stem that normally help keep heart rate up. · Use carefully during a heart attack (acute coronary syndrome) because speeding up the heart makes the heart muscle demand more oxygen, which can worsen starved blood flow (ischemia) (areas of heart muscle starving for blood). · Can push heart rate too high (tachycardia, over 100 beats per minute), which is dangerous in people with coronary artery disease (clogged heart arteries) because a racing heart may not get enough blood between beats. · May raise pressure inside the eye (worsening glaucoma) and make it hard to empty the bladder (urinary retention), especially in older adults. · If atropine does not raise heart rate after a total of 3 mg or the patient stays unstable (low blood pressure, chest pain, confusion, shortness of breath), immediately start transcutaneous pacing (sticky electrode pads on the chest deliver electrical pulses to pace the heart), prepare for transvenous pacing (a thin pacing wire threaded through a vein into the right side of the heart), or start an IV drip of epinephrine (2 to 10 micrograms per minute) or dopamine (5 to 20 micrograms per kilogram per minute) per ACLS guidelines (American Heart Association Advanced Cardiovascular Life Support emergency protocols). · Always hunt for and treat the root cause: stop or reverse beta-blockers, calcium channel blockers, digoxin, or other drugs slowing the heart; correct low or high potassium and low magnesium; treat low thyroid (an underactive thyroid gland); give IV fluids if blood volume is low. · Arrange a permanent pacemaker (a small battery-powered device implanted under the skin with thin wires threaded into the heart to keep rhythm steady) for symptomatic sinus node dysfunction (SA node cannot keep up and causes dizziness, fainting, or fatigue), Mobitz type II second-degree AV block, third-degree (complete) AV block (no electrical signals get from atria to ventricles), or alternating bundle branch block (the wiring in the ventricles fails on alternating sides) per ACC/AHA/HRS guidelines (American College of Cardiology / American Heart Association / Heart Rhythm Society standards).

Epinephrine infusion · Sympathomimetic (mimics adrenaline, the chemical that revs up the heart and tightens blood vessels)

Epinephrine is synthetic adrenaline. It docks onto beta-1 receptors on heart cells and tells them to beat faster and squeeze harder. At the same time it docks onto alpha-1 receptors on the smooth muscle in blood vessel walls and tells them to tighten (constrict), which raises blood pressure. Think of it like pressing the gas pedal and squeezing a garden hose tighter at the same time—flow speeds up and pressure climbs. Epinephrine is used when atropine has

failed or when the slow heart is causing dangerously low blood pressure, chest pain, confusion, or trouble breathing. It buys time until you can place a pacemaker or fix the root cause.

1. Epinephrine is given as a continuous IV drip (2 to 10 micrograms per minute, adjusted to response) so blood levels stay steady.
2. It binds to beta-1 receptors on heart cells, triggering a cascade inside the cell that releases more calcium (the mineral that makes muscle fibers slide together and contract).
3. The SA node fires faster, raising heart rate.
4. The ventricles squeeze harder (increased contractility), pumping more blood with each beat (increased stroke volume).
5. Epinephrine also binds to alpha-1 receptors on smooth muscle cells in artery walls, making them tighten and raising blood pressure (increased systemic vascular resistance).

SAFETY WATCH

Monitor continuously with telemetry (a wireless heart monitor that shows the rhythm on a screen) and check blood pressure every few minutes because epinephrine can cause dangerously fast heart rate (tachycardia over 100 beats per minute) or dangerous rhythms such as ventricular a fast heart rate (tachycardia) (fast chaotic rhythm in the lower chambers) or ventricular fibrillation (the ventricles quiver uselessly instead of pumping). · Use the lowest dose that keeps the patient stable (heart rate above 60, blood pressure acceptable, no chest pain or confusion) and taper down (wean) as soon as the patient is stable or a pacemaker is in place. · Can worsen starved blood flow (ischemia) (starving heart muscle) during a heart attack because faster heart rate and higher blood pressure make the heart demand much more oxygen than narrowed coronary arteries can deliver. · May cause tremor (shaking hands), anxiety, headache, and high blood sugar (hyperglycemia) because epinephrine also tells the liver to release stored glucose. · If the IV line leaks into the tissue around the vein (extravasation), epinephrine can cause tissue death (necrosis) because it constricts the tiny blood vessels feeding that tissue—use a central line (a large IV placed in a big vein near the heart, such as the internal jugular or subclavian vein) when possible. · Prepare for transcutaneous pacing or transvenous pacing—epinephrine is a stopgap measure, not a long-term solution.

Dopamine infusion · Sympathomimetic and inotrope (mimics adrenaline and directly strengthens heart squeeze)

Dopamine is a chemical messenger (neurotransmitter) your body makes naturally; as a drug it acts like adrenaline. At moderate to high doses (5 to 20 micrograms per kilogram per minute), dopamine binds to beta-1 receptors on heart cells to speed up the heart and make it squeeze harder, and it binds to alpha-1 receptors on blood vessel walls to tighten them and raise blood pressure. Think of it as turning up the volume on your heart and squeezing the hose tighter.

Dopamine is used when atropine fails and the patient is unstable (low blood pressure, chest pain, confusion, shortness of breath). It is another temporary fix until a pacemaker is placed or the root cause is treated.

1. Dopamine is given as a continuous IV drip, with the dose titrated (adjusted up or down) based on heart rate and blood pressure response.
2. At 5 to 10 micrograms per kilogram per minute it mainly hits beta-1 receptors on heart cells, raising heart rate and contractility.
3. At 10 to 20 micrograms per kilogram per minute it also hits alpha-1 receptors on blood vessel smooth muscle, constricting (narrowing) vessels and raising blood pressure.
4. The SA node fires faster, increasing heart rate.
5. The ventricles squeeze harder, pumping more blood with each beat (increased stroke volume).
6. Blood pressure climbs as vessels tighten and the heart pumps more forcefully.

SAFETY WATCH

Continuous telemetry and frequent blood pressure checks (every few minutes) are mandatory—dopamine can trigger fast dangerous rhythms such as ventricular a fast heart rate (tachycardia) (fast chaotic rhythm in the lower chambers) or ventricular fibrillation (the ventricles quiver uselessly). · Use the lowest dose that keeps the patient stable (heart rate above 60, blood pressure acceptable, no symptoms); taper down (wean) as soon as pacing is in place or the root cause is fixed. · Can worsen starved blood flow (ischemia) during a heart attack because faster heart rate and higher blood pressure make the heart muscle demand more oxygen than narrowed coronary arteries can deliver. · May cause nausea, headache, tremor (shaking), and high blood sugar (hyperglycemia). · If the IV leaks into the tissue around the vein (extravasation), dopamine can cause tissue death (necrosis) because it constricts tiny blood vessels—use a central line (large IV in a big vein near the heart) when possible. · Prepare for pacing—dopamine is a stopgap, not a long-term answer.

Glucagon · Hormone and inotrope (naturally raises blood sugar and also directly strengthens heart squeeze)

Glucagon is a hormone your pancreas makes to raise blood sugar when it drops too low. It also has a powerful side effect on the heart: it binds to its own glucagon receptors on heart cells and triggers a cascade (through a pathway using cyclic AMP, a chemical messenger inside cells, different from the beta-receptor pathway that adrenaline uses) that floods the cell with calcium, making the heart beat faster and squeeze much harder. Glucagon works even when beta receptors are completely blocked by beta-blocker drugs (like metoprolol, atenolol, propranolol) or when calcium channels are blocked by calcium channel blockers (like

diltiazem, verapamil). Think of it as a back door into the heart that beta-blockers and calcium channel blockers cannot lock. Glucagon is the antidote when these drugs cause dangerous slow heart rate and low blood pressure (overdose or toxicity).

1. Give glucagon 3 to 5 mg IV push (injected quickly into the vein), then start a continuous drip at 3 to 5 mg per hour if the patient responds.
2. Glucagon binds to glucagon receptors on heart cells (different receptors from beta receptors).
3. This activates the enzyme adenylyl cyclase, which makes cyclic AMP (a second messenger inside the cell)—a pathway that is completely independent of beta receptors.
4. Cyclic AMP triggers calcium release inside heart muscle cells, making them contract harder and faster.
5. Heart rate and blood pressure rise even though beta receptors or calcium channels are blocked by the overdose drug.

SAFETY WATCH

Glucagon often causes nausea and vomiting (because it relaxes the stomach and slows emptying)—have suction equipment ready and protect the airway by placing the patient on their side so they do not choke on vomit. · Large doses deplete the body's glucagon stores quickly (the pancreas and liver run out); the effect may wear off and you will need repeat dosing or a continuous drip to keep levels up. · Raises blood sugar (hyperglycemia, sometimes quite high) because glucagon tells the liver to dump stored glucose into the bloodstream—this is usually not harmful short-term but monitor glucose levels. · May cause low potassium (hypokalemia) because insulin is released in response to high blood sugar and insulin pushes potassium into cells—check potassium levels and replace if low. · If glucagon fails to restore heart rate and blood pressure, start high-dose insulin euglycemic therapy (give a large continuous infusion of insulin along with IV dextrose to keep blood sugar normal; insulin itself is a powerful inotrope that strengthens heart squeeze in beta-blocker and calcium channel blocker overdose), prepare for transcutaneous or transvenous pacing, and consider extracorporeal membrane oxygenation (ECMO, a heart-lung bypass machine that takes over pumping and oxygenating blood) or intra-aortic balloon pump (a balloon placed in the aorta that helps the heart pump) in severe refractory cases per toxicology and critical care guidelines. · Always call poison control (1-800-222-1222 in the United States) for expert guidance, send blood for drug levels if available, and consider hemodialysis (a kidney machine that filters the blood) for drugs that can be removed that way (such as atenolol, sotalol), or lipid emulsion therapy (IV fat emulsion that binds fat-soluble drugs like propranolol) per toxicology protocols.

Isoproterenol infusion · Pure beta-agonist (activates only beta receptors, speeding and strengthening the heart)

Isoproterenol is a synthetic drug that acts only on beta receptors: beta-1 receptors in the heart (which speed up rate and strengthen squeeze) and beta-2 receptors in the lungs and blood vessels (which relax airways and widen blood vessels). It speeds up the SA node, strengthens heart squeeze, and relaxes blood vessel walls (which can drop blood pressure because vessels are wider). Because it is a pure beta drug with no alpha effect (no vessel-tightening), it is used rarely—mainly for refractory a slow heart rate (bradycardia) (slow heart that does not respond to atropine, epinephrine, or dopamine) or as a temporary bridge to pacing in heart transplant patients (who have no vagus nerve, so atropine does not work). Think of it as flooring the gas pedal but loosening the hose clamp at the same time—heart rate goes up but blood pressure may fall.

1. Isoproterenol is given as a continuous IV drip (2 to 10 micrograms per minute), with the dose titrated (adjusted) to heart rate response.
2. It binds to beta-1 receptors on heart cells, triggering the cyclic AMP pathway that releases calcium inside cells and speeds the SA node.
3. Heart rate climbs and contractility (squeeze strength) increases.
4. It also binds to beta-2 receptors on blood vessel smooth muscle, causing relaxation (vasodilation) so vessels widen.
5. The net effect is faster heart rate and stronger squeeze, but blood pressure may stay flat or even drop because vessels are wider (lower resistance).

SAFETY WATCH

Continuous telemetry and blood pressure monitoring (every few minutes) are essential— isoproterenol can cause life-threatening fast rhythms such as ventricular a fast heart rate (tachycardia) (fast chaotic rhythm in the lower chambers) or ventricular fibrillation (the ventricles quiver uselessly). · May worsen starved blood flow (ischemia) during a heart attack because the heart is working much harder and demanding more oxygen than narrowed coronary arteries can deliver. · Can cause tremor (shaking), headache, flushing (red warm skin), and dangerous drop in blood pressure (hypotension) because of vasodilation. · Use only when atropine, epinephrine, and dopamine have failed and pacing is not immediately available, or in special cases such as heart transplant patients (no vagus nerve so atropine does not work), a slow heart rate (bradycardia)-dependent torsades de pointes (a specific dangerous rhythm where speeding the heart stops the arrhythmia), or temporary support after cardiac surgery. · Transition to transcutaneous or transvenous pacing as quickly as possible—isoproterenol is high-risk and not a long-term solution.

Theophylline · Methylxanthine (blocks adenosine, a chemical that slows the heart, and mildly boosts adrenaline-like effects)

Theophylline blocks adenosine receptors in the heart. Adenosine is a natural chemical brake that slows the SA node and AV node; blocking adenosine receptors takes one brake off. Theophylline also mildly inhibits an enzyme called phosphodiesterase, which normally breaks down cyclic AMP (a chemical messenger inside cells), so cyclic AMP levels rise a bit and give a weak adrenaline-like boost. Theophylline is used almost exclusively in heart transplant patients (who have no vagus nerve, so atropine fails) when they develop slow heart rate and pacing is not yet in place. It is rarely used otherwise because safer, faster options (atropine, epinephrine, dopamine, pacing) exist. Think of it as cutting one of the brake cables so the engine can rev a bit higher.

1. Theophylline is usually given by mouth (oral pill or liquid), which takes hours to work, or as aminophylline IV (the water-soluble salt of theophylline, which the body converts to theophylline) if the situation is more urgent—but even IV is slower than other emergency a slow heart rate (bradycardia) drugs.
2. It binds to adenosine A1 receptors on SA node and AV node cells and prevents adenosine from landing.
3. Without adenosine's slow-down signal, the SA node fires a bit faster.
4. Theophylline also weakly inhibits phosphodiesterase (an enzyme that breaks down cyclic AMP), so cyclic AMP levels rise slightly, giving a mild boost to heart rate and contractility.

SAFETY WATCH

Narrow therapeutic window (the safe blood level, 10 to 20 micrograms per milliliter, is close to the toxic level)—too much causes nausea, vomiting, tremor (shaking), seizures, and dangerous fast rhythms such as ventricular a fast heart rate (tachycardia) or supraventricular a fast heart rate. · Blood levels must be monitored (drawn and measured in the lab) and kept in the safe range, especially if giving IV or oral loading doses. · Many drug interactions change theophylline levels: antibiotics (ciprofloxacin, erythromycin), antifungals (fluconazole), anti-seizure drugs (phenytoin, carbamazepine), heart drugs (verapamil, propranolol), and smoking (lowers levels, so dose must be higher in smokers and adjusted if they quit). · Not first-line for a slow heart rate (bradycardia)—use only in special cases such as heart transplant patients when atropine does not work and immediate pacing is not available.

Red flags

- Fainting or nearly fainting—your brain isn't getting enough blood, and you're at risk of dangerous pauses in your heartbeat.

- Chest pain or sudden trouble breathing—may mean a heart attack, heart failure, or fluid backing up into your lungs.
- Confusion or trouble staying awake—means your brain is starved for oxygen and needs emergency care.
- Blood pressure below 90 on the top number, with cold skin, weak pulse, or little urine—your body is going into shock and organs may start failing.
- New complete heart block or advanced second-degree heart block on the ECG (the electrical tracing of your heart)—the electrical signals between your heart's upper and lower chambers are failing, and your heart could stop.

Nursing priorities

1. Put the patient on a continuous heart monitor and watch the heart rate and rhythm every minute. — A slow heartbeat can suddenly stop completely (asystole), and the monitor lets us see when the rhythm gets worse so we can start lifesaving treatment like CPR or pacing in seconds instead of waiting until the patient collapses.
2. Get IV access with at least one large IV line and prepare atropine 1 mg at the bedside, ready to push. — Atropine blocks nerve signals that slow the heart down, so it speeds up the heartbeat quickly—we need it ready because if the heart rate drops more or the patient faints, we have only minutes to give the medicine before the heart stops.
3. Attach transcutaneous pacing (TCP) pads to the chest and turn on the pacing machine, but do not pace yet unless the patient gets worse. — TCP sends electrical shocks through the chest to force the heart to beat faster—having the pads already stuck on means we can start pacing in 10 seconds if the patient faints or the heart slows even more, instead of wasting 2 to 3 minutes setting up.
4. Check blood pressure every 5 to 15 minutes and watch for signs of shock like cold skin, weak pulse, confusion, or little urine. — A slow heartbeat often causes low blood pressure because the heart is not pumping enough blood—low blood pressure means organs like the brain, kidneys, and heart muscle are not getting enough oxygen and will die if we do not speed up the heart or give IV fluids fast.
5. Draw blood for potassium, magnesium, thyroid hormone (TSH), troponin (a heart enzyme), and digoxin level if the patient takes that medicine. — High potassium stops the heart's electrical signals and can make the heart stop—low magnesium also slows the heart—low thyroid hormone slows everything—troponin tells us if a heart attack is causing the slow rhythm—and too much digoxin is a common cause of dangerous slow heartbeat that we fix by stopping the medicine.

6. Hold (do not give) the next dose of beta-blockers, calcium channel blockers, digoxin, or other heart medicines until the doctor reviews them. — These medicines slow the heart down, so giving more will make the slow heartbeat worse and may cause the heart to stop—holding them gives the body time to clear out the medicine already in the blood so the heart can speed up on its own.
7. Keep the patient lying flat or with the head of the bed only slightly up, and help them to the bathroom instead of letting them walk alone. — Standing up makes blood drain away from the brain, and with a slow heartbeat the heart cannot pump hard enough to push blood back up—this causes fainting, falls, and head injuries, so keeping the patient flat keeps more blood in the brain.
8. Prepare for emergency transvenous pacemaker insertion by gathering sterile pacing wire kit, ultrasound machine, and lidocaine (numbing medicine) at the bedside. — If atropine and TCP do not work or the patient keeps getting worse, the doctor will thread a pacing wire through a vein into the heart to control the heartbeat directly—having supplies ready saves 10 to 15 minutes, which may be the difference between life and death when the heart is failing.

Sources

- AHA/ACC/HRS 2018 Guideline on the Evaluation and Management of Patients with Bradycardia and Cardiac Conduction Delay
- European Society of Cardiology 2021 Guidelines on Cardiac Pacing and Cardiac Resynchronization Therapy
- American Heart Association
- UpToDate Clinical Decision Support

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