

Bradycardia Algorithm

AHA ACLS a slow heart rate (bradycardia) algorithm: give atropine first, then start transcutaneous pacing (electrical shocks through the chest to make the heart beat), then use epinephrine or dopamine drip if the patient stays unstable.

THE ONE-LINE TRUTH

Heart rate below 60 beats per minute with signs that the body is not getting enough blood—like low blood pressure, confusion, chest pain, or pale sweaty skin.

The chain — how it unfolds

1. The SA node fires fewer than 60 sparks per minute, or the AV node blocks some atrial impulses so fewer than 60 ventricular beats occur each minute. — why: Drugs (beta-blockers, calcium-channel blockers, digoxin) directly slow or block the nodes; starved blood flow (ischemia) (MI cutting off blood to nodal tissue) starves the cells of oxygen; electrolyte shifts (hyperkalemia slowing depolarization, low potassium (hypokalemia) destabilizing the rhythm) alter the action potential; high vagal tone (vasovagal reflex from vomiting, pain, carotid massage, or athletic conditioning) floods the nodes with acetylcholine; or intrinsic disease (sick sinus syndrome, fibrosis of the conduction system, second-degree type II or third-degree AV block) means the node cells themselves are failing or the wiring below the AV node is broken—all reduce the rate at which electrical impulses reach the ventricles.
2. Cardiac output—the product of stroke volume (blood pumped per beat) and heart rate (beats per minute)—falls below the threshold needed to maintain mean arterial pressure (MAP) at or above 65 mmHg and perfuse vital organs. — why: Even if stroke volume rises to compensate (because the ventricles fill longer and squeeze harder), a heart rate of 40 beats per minute cannot deliver enough liters per minute to meet the body's oxygen demand, so systemic blood pressure drops and tissues become oxygen-starved (hypoxic) (oxygen-starved).

3. Low blood pressure (Hypotension) (systolic BP below 90 mmHg), altered mental status (confusion, dizziness, near-fainting (syncope), or fainting), chest pain (angina from coronary poor blood flow (hypoperfusion)), and shock signs (pale, cool, drenched in sweat (diaphoretic) or sweaty skin, weak pulses, delayed capillary refill) appear together. — why: Low cardiac output means the brain receives less oxygen (causing confusion, lightheadedness, or fainting because cerebral blood flow (perfusion) pressure drops), the heart muscle itself becomes blood-starved (ischemic) (causing chest pain or pressure because the coronary arteries are not filling well during diastole), and the body shunts blood away from the skin and limbs to vital organs by vasoconstricting peripheral vessels (causing cold, sweaty, pale skin and weak pulses)—these are the body's emergency alarm bells, not separate diseases, all stemming from one upstream electrical slow-down.
4. Prolonged or severe a slow heart rate (bradycardia) drives a vicious cycle: low blood pressure (hypotension) worsens coronary and cerebral blood flow (perfusion), starved blood flow (ischemia) further slows the nodes or triggers lethal arrhythmias, and compensatory mechanisms exhaust. — why: Low blood pressure means even less blood reaches the SA node, AV node, and myocardium (heart muscle), deepening starved blood flow (ischemia) and slowing the heart further; baroreceptors max out vasoconstriction so resistance cannot rise any more; stroke volume cannot increase beyond the physical limits of ventricular filling; and the brain and myocardium become so oxygen-starved (hypoxic) that electrical instability rises, setting the stage for asystole (flatline, no electrical activity), pulseless electrical activity (PEA, organized rhythm on the monitor but no pulse), or slow ventricular escape rhythms that cannot sustain life.
5. Without immediate treatment—atropine to block vagal tone, transcutaneous pacing to electrically capture the ventricles, or epinephrine or dopamine infusion to increase heart rate and contractility—a slow heart rate (bradycardia) can progress to cardiac arrest and death. — why: The body's compensatory reserve is finite: once vasoconstriction is maximal, stroke volume is maximal, and heart rate remains critically low, cardiac output cannot meet even basal metabolic demand; vital organs shut down in sequence (brain, heart, kidneys), and the heart either stops altogether (asystole) or beats in a disorganized, ineffective way (PEA or ventricular escape rhythms too slow to produce a pulse)—all tracing back to that one upstream electrical failure that was never corrected in time.

Spot it — what you see, and why

- Dizziness or lightheadedness: A slow heart rate pumps less blood each minute. When less blood reaches the brain, oxygen levels drop (called low brain blood flow (perfusion)), and you feel dizzy or like you might faint.

- Fainting (Syncope) (fainting or passing out): If the heart beats so slowly that blood pressure falls too low, the brain does not get enough oxygen to stay awake. You briefly lose consciousness. This is a red flag that the heart is not pumping enough blood to keep you safe.
- Confusion or feeling very drowsy: When a slow heart rate (bradycardia) lasts a long time or gets very severe, the brain runs on less oxygen than it needs. This makes thinking foggy and causes tiredness. It is a sign that blood flow to the brain is too low.
- Chest pain or pressure: The heart muscle itself needs oxygen from blood in the coronary arteries (the vessels that feed the heart). A very slow heart rate can lower the pressure in those arteries, so the heart muscle starves for oxygen. That causes chest pain (called angina).
- Shortness of breath: Low heart rate means the heart pumps less blood per minute. The lungs cannot clear carbon dioxide as well, and fluid may back up into the lungs (like a backed-up sink) if the left side of the heart cannot keep up with the blood returning from the body.
- Low blood pressure (systolic less than 90): Blood pressure depends on how much blood the heart pumps each minute. That number equals heart rate times the amount pumped per beat. When the rate drops too low, total blood flow falls and pressure drops with it.

Red flags — act now

- Fainting or near-fainting (sign the heart is not pumping enough blood; risk of sudden cardiac arrest)
- Chest pain or changes on the ECG suggesting the heart muscle is starving for oxygen
- Confusion or drowsiness (brain is not getting enough blood; collapse may be coming)
- Systolic blood pressure below 90 with signs of shock (pale, cool, sweaty skin; weak pulses)
- Second-degree AV block type II or third-degree (complete) heart block on ECG (high risk the heart will stop; needs pacing right away)

Treat it — in this order

1. Atropine 1 mg IV push, repeat every 3 to 5 minutes up to total dose of 3 mg (2020 AHA ACLS guideline) → Atropine blocks muscarinic receptors, stopping the vagus nerve's parasympathetic (slowing) effect on the SA and AV nodes. This increases heart rate by letting the nodes fire faster. Works best for sinus a slow heart rate (bradycardia) and AV nodal (supranodal) blocks with narrow QRS complexes.

2. Transcutaneous pacing (TCP): place pacing pads on chest, set rate 60 to 80 beats per minute, increase milliamp output until electrical capture (QRS after each pacer spike) is seen → External electrical pulses depolarize the ventricles directly, forcing them to contract at a set rate. This immediately raises heart rate and cardiac output, bypassing the failed SA or AV node. TCP is a bridge to transvenous pacing or to fixing the underlying cause. Provide sedation and pain control (e.g., fentanyl or midazolam) because TCP is uncomfortable.
3. Dopamine infusion 5 to 20 mcg/kg/min IV OR epinephrine infusion 2 to 10 mcg/min IV (2020 AHA ACLS guideline) → Dopamine and epinephrine are catecholamines (sympathetic stimulants) that increase heart rate by activating beta-1 receptors in the SA node and increase blood pressure by causing vasoconstriction (narrowing of blood vessels). These drugs support blood flow (perfusion) while the underlying cause is being treated or while waiting for transvenous pacing.
4. Identify and treat the underlying cause: correct high potassium (hyperkalemia) with IV calcium (calcium chloride 1 g or calcium gluconate 3 g), insulin/glucose (10 units regular insulin with 25 g dextrose), and hemodialysis if needed; hold or reverse bradycardic drugs (beta-blockers with glucagon 3 to 10 mg IV bolus, calcium-channel blockers with IV calcium or high-dose insulin-euglycemia therapy, digoxin with digoxin-specific Fab fragments); emergent reperfusion (PCI or fibrinolytics) if acute MI is present → Correcting electrolytes restores normal SA and AV node function. Reversing drug toxicity removes the pharmacologic suppression of the conduction system. Revascularizing blood-starved (ischemic) tissue (reopening blocked arteries) restores blood flow to the nodes, letting them generate and conduct electrical impulses normally. This is the only way to achieve long-term correction.
5. Transvenous pacing (TVP): insert pacing wire through central vein (internal jugular or femoral) into right ventricle under fluoroscopy or ECG guidance → A transvenous pacemaker delivers reliable electrical impulses directly to the endocardium (inner heart lining), ensuring consistent ventricular depolarization and cardiac output. TVP is more comfortable and reliable than transcutaneous pacing and is used when pacing will be needed for hours to days or when TCP fails to capture or is not tolerated.

Your top priorities at the bedside

1. Place patient on continuous cardiac monitor and pulse oximetry; obtain 12-lead ECG within 5 minutes and assess for heart block or ST-segment changes. — why: Continuous monitoring detects lethal rhythms like complete heart block or ventricular escape rhythms that can deteriorate to asystole (no heartbeat); the 12-lead identifies heart attack (myocardial infarction) and the degree of AV block (first-degree, second-degree type I or II, or third-degree) which determines urgency of pacing and whether atropine will work.

2. Assess and document blood pressure, level of consciousness using AVPU or Glasgow Coma Scale, skin color and temperature, capillary refill, and presence of pulses every 1 to 2 minutes until stable. — why: Low blood pressure (Hypotension) (systolic below 90) and altered mental status confirm the a slow heart rate (bradycardia) is causing inadequate cardiac output (the volume of blood the heart pumps per minute); cool, pale, sweaty skin and delayed capillary refill signal shock, meaning tissues are starving for oxygen and the patient needs immediate atropine or pacing to restore blood flow (perfusion) (blood flow).
3. Establish large-bore IV access (18-gauge or larger) in at least one site and prepare for rapid administration of atropine, dopamine, or epinephrine infusions. — why: Symptomatic a slow heart rate (bradycardia) requires fast IV drug delivery; atropine blocks vagal (vagus nerve) slowing of the heart, and if atropine fails, vasoactive infusions like dopamine or epinephrine increase heart rate and blood pressure by stimulating beta-1 receptors in the heart to speed up electrical conduction and strengthen contractions.