

Bradycardia Algorithm

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

The SA node (sinoatrial node, the heart's natural pacemaker sitting in the right atrium or upper chamber) fires electrical impulses 60 to 100 times per minute. Each spark travels to the AV node (atrioventricular node, the relay station between the atria and ventricles), then down special wires called the bundle of His and Purkinje fibers to make the ventricles (the big lower pumping chambers) squeeze. This cycle pumps enough blood every minute—called cardiac output—to deliver oxygen to your brain, kidneys, and every organ.

BODY'S JOB

A steady heart rate between 60 and 100 ensures that the amount of blood pumped per beat (stroke volume) multiplied by the number of beats per minute equals enough total blood flow (cardiac output) to keep blood pressure normal and tissues fed with oxygen.

1. The SA node fires an electrical spark in the right atrium.
2. The spark spreads across both atria, making them squeeze and push blood into the ventricles.
3. The spark reaches the AV node, which delays it just long enough for the ventricles to fill.
4. The spark then shoots down the bundle of His and out through the Purkinje fibers.
5. The ventricles squeeze from bottom to top, pumping blood to the lungs (right ventricle) and the whole body (left ventricle).

6. This cycle repeats 60 to 100 times per minute, delivering 4 to 6 liters of blood every minute in a resting adult.

What breaks

One upstream problem—medication that slows the heart (like beta-blockers, calcium-channel blockers, or digoxin), starved blood flow (ischemia) (not enough blood reaching the SA or AV node to feed it), electrolyte imbalance (potassium too high or too low, calcium too low), high vagal tone (overactive signals from the parasympathetic nerve that acts like a brake), or the node cells themselves wearing out or dying—slows the SA node or blocks the AV node so the heart beats fewer than 60 times per minute and sometimes below 40.

CLINICAL TERM

A slow heart rate (Bradycardia) (heart rate less than 60 beats per minute) becomes symptomatic when cardiac output (heart rate $\times$ stroke volume) falls below the threshold needed to keep mean arterial pressure (MAP) above roughly 65 mmHg and organs perfused. The upstream causes—beta-blockers, calcium-channel blockers (especially diltiazem and verapamil), digoxin toxicity, acute heart attack (myocardial infarction) (heart attack) damaging the SA or AV node, high potassium (hyperkalemia) (potassium above 5.5 mEq/L slowing depolarization), low potassium (hypokalemia) (potassium below 3.5 mEq/L destabilizing the rhythm), hypothyroidism (underactive thyroid slowing metabolism), increased vagal tone (vasovagal reflex from pain, vomiting, suctioning, or carotid sinus hypersensitivity), sick sinus syndrome (SA node failure from fibrosis or starved blood flow (ischemia)), or high-degree AV block (second-degree type II or third-degree complete heart block)—all converge on one shared result: too few ventricular contractions per minute to sustain blood flow (perfusion).

How the body compensates

The body tries to keep blood pressure normal by squeezing peripheral arteries (vasoconstriction, narrowing of blood vessels to boost resistance) and increasing stroke volume (the ventricles fill longer and squeeze harder when rate drops), but once heart rate falls below roughly 40 to 50 beats per minute, cardiac output crashes and organs become oxygen-starved (hypoxic) (oxygen-starved).

1. Early a slow heart rate (bradycardia) (heart rate 50 to 60) triggers baroreceptor reflexes—pressure sensors in the carotid arteries and aorta detect falling pressure and signal the sympathetic nervous system to constrict peripheral vessels, raising total peripheral resistance to keep MAP near normal.

2. Stroke volume rises because the slower rate gives the ventricles more time to fill with blood (increased preload, the volume stretching the heart before it squeezes), and the Frank-Starling mechanism makes the heart squeeze harder when stretched more, partly offsetting the slow rate.
3. This compensated phase can mask the severity—blood pressure may look acceptable (systolic 100 to 110 mmHg) even as heart rate hovers in the high 40s to low 50s, so clinicians must watch for subtle signs like mild confusion, fatigue, or a downward trend in serial heart rates.
4. The crash point arrives when heart rate drops so low (usually below 40 to 50) that even maximal stroke volume cannot sustain cardiac output: low blood pressure (hypotension) (systolic BP below 90 mmHg), altered mental status (confusion, near-fainting (syncope), or fainting from cerebral poor blood flow (hypoperfusion)), chest pain (angina from low coronary artery blood flow (perfusion)), and shock signs (cold, pale, clammy skin, weak or thready pulses, delayed capillary refill) appear together—these are not separate diseases but one shared failure of blood flow.
5. In infranodal blocks (second-degree type II or third-degree block below the AV node, in the His-Purkinje system), atropine can paradoxically worsen conduction by speeding the atrial rate without improving the ventricular escape rhythm, so always prepare transcutaneous pacing and do not rely on atropine alone.
6. Recognize the downward trend—serial heart rates dropping from 55 to 48 to 42 over minutes to hours—not just a single snapshot, because decompensation accelerates once compensatory reserve (maximal vasoconstriction, maximal stroke volume) is exhausted and no further mechanisms remain to maintain cardiac output.

Domino effect

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. — 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. — 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. — 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. — 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. — 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.

Symptoms · what the patient feels, 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.
- **Sweating and pale, cool skin:** The body senses low blood flow and turns on the emergency (sympathetic) nervous system. Blood vessels in the skin tighten to save blood for the brain and heart, making skin pale and cool. Sweat glands also turn on. These are classic signs of shock.
- **Fatigue or weakness:** Muscles and organs do not receive enough oxygen-rich blood, even at rest. Over time, this makes you feel tired and weak because your body is working on a lower fuel supply.

Focused exam

- **Heart rate below 50 beats per minute:** In symptomatic a slow heart rate (bradycardia) (a slow heart rate causing problems), the low rate means the SA node (the heart's natural pacemaker) or AV node (the electrical relay station between the upper and lower chambers) is failing to create enough electrical signals. When a slow rate happens WITH symptoms like low blood pressure or confusion, the patient needs emergency treatment right now, not just watching.

- Low blood pressure (Hypotension) (systolic blood pressure below 90 mmHg): When the heart beats too slowly, cardiac output (the amount of blood pumped each minute) drops because cardiac output equals stroke volume times heart rate. Low blood pressure means tissues are not getting enough oxygen-rich blood. This tells you the slow heart rate is causing poor blood flow (perfusion) (blood flow to organs) right now.
- Altered mental status (confusion, sleepiness, or trouble responding): The brain needs constant oxygen delivery and cannot store energy. When a slow heart rate (bradycardia) reduces cardiac output and blood pressure, cerebral blood flow (perfusion) (blood flow to the brain) drops, causing confusion or altered consciousness. Mental status changes signal that critical organs are being injured by not getting enough oxygen.
- Chest pain or pressure: A slow heart rate (Bradycardia) can reduce coronary blood flow (perfusion) (blood flow to the heart muscle itself), causing myocardial starved blood flow (ischemia) (the heart muscle not getting enough oxygen). Chest pain with a slow heart rate may mean acute coronary syndrome (heart attack or severe blockage) is either causing the slow rate or being made worse by it.
- Signs of shock: cool or mottled skin, weak peripheral pulses, low urine output (oliguria) (urine output below 0.5 mL/kg/hr): Shock happens when tissue blood flow (perfusion) cannot meet the body's needs. In symptomatic a slow heart rate (bradycardia), shock develops because the slow heart rate produces too little cardiac output. Cool skin, weak pulses, and low urine output mean the body is redirecting blood away from the skin and kidneys to protect the brain and heart.
- Narrow QRS complex (less than 0.12 seconds) on ECG: A narrow QRS tells you the electrical block is at or above the AV node (supranodal), meaning atropine (a drug that blocks the vagus nerve's slowing effect) is likely to work. A wide QRS (0.12 seconds or greater) suggests infranodal block (below the AV node), where atropine may make the block worse and pacing should be used first.

Diagnostics · every test answers a question

- 12-lead electrocardiogram (ECG) → What is the rhythm, heart rate, QRS width, and electrical conduction pattern? Does the ECG show evidence of acute heart attack (myocardial infarction) (heart attack) or AV block?
- Serum electrolytes: potassium, magnesium, calcium → Are electrolyte imbalances slowing down the SA or AV node?
- Thyroid-stimulating hormone (TSH) and free T4 → Is hypothyroidism (underactive thyroid) slowing the heart rate?
- Cardiac troponin I or T → Is acute heart attack (myocardial infarction) causing the a slow heart rate (bradycardia) by damaging the conduction system or SA node?

- Toxicology screen and medication review → Are medications or toxins slowing the heart rate?
- Serum digoxin level (if patient takes digoxin) → Is digoxin toxicity causing the slow heart rate?
- Arterial blood gas (ABG) or venous lactate → Is tissue blood flow (perfusion) so poor that cells are switching to anaerobic metabolism (making energy without oxygen)?
- Continuous cardiac monitoring (telemetry) → Is the a slow heart rate (bradycardia) constant or intermittent? Are there pauses (periods when the heart stops beating) or other dangerous rhythms?

Treatment logic

- 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.
- 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.
- 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.

- 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.
- 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.
- Permanent pacemaker implantation (for irreversible high-grade AV block, symptomatic sinus node dysfunction, or recurrent symptomatic a slow heart rate (bradycardia)) → A permanent pacemaker continuously monitors the heart's rhythm and delivers electrical impulses when the heart rate drops below a set threshold. This prevents recurrent symptomatic a slow heart rate (bradycardia) and improves quality of life and survival in patients with irreversible conduction system disease (2018 ACC/AHA/HRS guideline on evaluation and management of a slow heart rate and cardiac conduction delay).

Medications

Atropine · Anticholinergic (vagolytic agent)

Atropine blocks the vagus nerve's brake pedal on the heart. The vagus nerve normally slows your heart down; atropine stops that signal so your heart can speed up naturally.

1. The vagus nerve releases a chemical called acetylcholine, which slows the heart's electrical firing.
2. Atropine blocks the receptors (docking spots) where acetylcholine would land.
3. Without acetylcholine slowing things down, the SA node fires faster.
4. Electrical signals also travel faster through the AV node to the lower chambers.
5. Heart rate increases, usually within 3–5 minutes.

SAFETY WATCH

Can cause fast heart rate (tachycardia) if you give too much. · May worsen heart function in people having a heart attack because a faster rate makes the heart work harder and need more oxygen. · Can cause confusion, dry mouth, blurry vision, and trouble urinating (especially in older adults). · Use carefully in people with glaucoma (high eye pressure) because it can make it worse. · Maximum safe dose is 3 mg total; higher doses do not help more and increase side effects.

Dopamine infusion · Sympathomimetic (catecholamine)

Dopamine acts like a gas pedal for the heart. It locks onto special docking spots (beta-1 receptors) on heart cells and tells them to beat faster and squeeze harder.

1. Dopamine travels through the bloodstream and binds to beta-1 receptors on heart cells.
2. This binding triggers a cascade inside the cell that increases calcium release.
3. More calcium makes the heart's electrical system fire faster and the muscle squeeze harder.
4. At doses of 5–20 micrograms per kilogram per minute, it mainly speeds the heart rate and boosts how much blood the heart pumps with each beat.
5. At higher doses it also squeezes blood vessels, raising blood pressure.

SAFETY WATCH

Can cause dangerous fast rhythms (like ventricular a fast heart rate (tachycardia)) if the dose is too high or the heart is irritable. · May worsen chest pain or heart attack because the faster rate and stronger squeeze demand more oxygen. · Can cause severe tissue damage if the IV line leaks and dopamine gets into the skin (extravasation). · Raises blood pressure, sometimes too much, which can strain the heart or cause bleeding in fragile blood vessels. · Requires continuous heart monitoring and an IV pump; never stop it suddenly—taper down slowly.

Epinephrine infusion · Sympathomimetic (catecholamine)

Epinephrine (adrenaline) is the body's emergency turbo-charge signal. It tells the heart to beat faster and harder by activating the same fight-or-flight receptors your body uses in danger.

1. Epinephrine binds to beta-1 receptors on pacemaker cells in the SA node.
2. This speeds up the electrical firing rate so the heart beats faster.
3. It also makes heart muscle cells squeeze harder by increasing calcium inside them.
4. At the same time, it can relax some blood vessels (beta-2 effect) and tighten others (alpha effect), which stabilizes blood pressure.
5. At low doses (2–10 micrograms per minute), it mainly increases heart rate and output.

SAFETY WATCH

Can trigger life-threatening fast rhythms (ventricular a fast heart rate (tachycardia) or fibrillation) if the heart is irritable or oxygen-starved. · Increases the heart's workload and oxygen need, which can worsen chest pain or heart attack. · Raises blood pressure sharply, risking stroke or bleeding. · Can cause shaking, anxiety, and pale or cool skin. · Requires close heart monitoring and an IV pump; must never be given as a fast push—only as a controlled drip.

Transcutaneous pacing (TCP) · Non-pharmacologic intervention (external electrical pacing)

Transcutaneous pacing uses sticky electrode pads on the chest and back to send electrical shocks through the skin and force the heart to beat. It acts as a temporary external pacemaker when the heart's own rhythm is too slow or stops.

1. Two large sticky pads are placed on the chest (front and back or front and side).
2. The pacing machine sends a timed electrical current through the skin into the chest.
3. This current reaches the heart muscle and forces it to contract (squeeze) with each pulse.
4. The machine is set to deliver a pulse at a chosen rate (usually 60–80 beats per minute) and strength (measured in milliamps).
5. The heart beats in time with the pacing pulses, even if its own electrical system is failing.

SAFETY WATCH

Painful—each shock feels like a hard thump or muscle cramp; give pain medicine or sedation if the person is awake. · Skin burns can happen if pads are not placed correctly or if used for a long time. · Does not always produce a real heartbeat (mechanical capture)—check for a pulse and blood pressure, not just the monitor. · Can trigger dangerous fast rhythms if the heart is irritable. · Never delay transcutaneous pacing if the person is unstable—it buys time for more definitive treatment like a transvenous pacemaker wire.

Transvenous pacing (TVP) · Non-pharmacologic intervention (internal temporary pacing)

Transvenous pacing threads a thin wire through a vein into the right side of the heart and delivers timed electrical pulses directly to the heart muscle. It is more reliable and comfortable than pacing through the skin because the wire sits right where it is needed.

1. A doctor inserts a thin pacing wire through a large vein (often in the neck, shoulder, or groin) using X-ray or ultrasound guidance.
2. The wire is advanced into the right ventricle and positioned against the inner wall.

3. The wire delivers small electrical pulses directly to the heart muscle.
4. A pacing box outside the body controls the rate (how fast) and output (how strong) of the pulses.
5. The heart beats in sync with the pulses, providing reliable pacing until the underlying problem is fixed or a permanent pacemaker is placed.

SAFETY WATCH

Risk of bleeding, infection, or blood clots at the insertion site. · Wire can perforate (poke through) the heart wall, causing fluid buildup around the heart (tamponade) or bleeding. · Wire can dislodge and stop pacing, so position must be checked regularly with chest X-ray or monitoring. · Can trigger dangerous fast rhythms if the wire irritates the heart muscle. · Requires sterile technique and close monitoring in an intensive care or cardiac unit. · Must be removed or replaced with a permanent pacemaker within days to weeks to avoid infection.

Red flags

- 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)

Nursing priorities

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. — 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. — 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. — 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.
4. If atropine is ineffective or the patient shows signs of shock or high-grade block, immediately apply transcutaneous pacing pads to the chest in anterior-posterior or anterior-lateral position and prepare TCP equipment. — Transcutaneous pacing (TCP) delivers electrical impulses through the skin to directly stimulate the heart when the heart's own electrical system fails or when drugs do not work; high-grade blocks like second-degree type II and third-degree block often do not respond to atropine because the problem is below the AV node where atropine cannot help, so pacing becomes the life-saving bridge until transvenous pacing or permanent pacemaker insertion.
5. Administer atropine 1 mg IV push rapidly; if no response, repeat every 3 to 5 minutes up to maximum total dose of 3 mg while monitoring heart rate and rhythm on the monitor. — Atropine blocks acetylcholine (a chemical messenger) at the SA node (the heart's natural pacemaker) and AV node (the relay station between upper and lower heart chambers), reducing vagal tone and allowing the heart to speed up; giving it quickly and repeating ensures you reach therapeutic levels, but doses above 3 mg total do not add benefit and may cause harm like urinary retention or confusion.
6. Prepare dopamine infusion starting at 5 mcg/kg/min or epinephrine infusion starting at 2 mcg/min if atropine fails or while awaiting pacing; titrate upward to achieve systolic blood pressure above 90 and heart rate above 60. — Dopamine at 5 to 20 mcg/kg/min stimulates beta-1 receptors to increase heart rate and contractility (heart squeeze strength) and alpha receptors to raise blood pressure; epinephrine at 2 to 10 mcg/min does the same but is more potent; these infusions maintain blood flow (perfusion) to vital organs when the heart rhythm cannot be immediately corrected.

7. If transcutaneous pacing is initiated, sedate the patient with small doses of a short-acting benzodiazepine like midazolam or an opioid like fentanyl as ordered, because TCP causes painful muscle contractions. — Electrical current from TCP travels through chest-wall muscle and causes strong, uncomfortable twitching; adequate analgesia (pain relief) and anxiolysis (anxiety reduction) keep the patient calm and cooperative, but you must give the smallest effective dose because oversedation can drop blood pressure further and mask changes in mental status that signal worsening blood flow (perfusion).
8. Notify the physician or rapid-response team immediately and prepare for possible transvenous pacing or transfer to a higher level of care if the patient has second-degree AV block type II, third-degree heart block, or does not respond to atropine and TCP. — High-grade heart blocks and refractory (treatment-resistant) symptomatic a slow heart rate (bradycardia) require a transvenous pacemaker (a wire inserted through a vein into the heart) or permanent pacemaker implantation because the electrical conduction system is severely damaged; early escalation ensures the patient reaches advanced care before complete loss of cardiac output or cardiac arrest.

Sources

- AHA/ACC 2018 Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay
- AHA ACLS Provider Manual 2020 (updated 2023)
- European Society of Cardiology 2021 Guidelines on Cardiac Pacing and Cardiac Resynchronization Therapy
- UpToDate: Sinus Bradycardia (Mangrum & DiMarco, current through 2024)

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