

AV block

The electrical signal from the top chambers gets delayed or blocked before it reaches the bottom chambers.

THE STORY IN ONE SENTENCE

The electrical signal from the top chambers gets delayed or blocked before it reaches the bottom chambers.

“AV block: say the degree (first, second, third). Say the level (nodal or below). Decide if a pacemaker is needed.”

Normal anatomy — the body's job

The AV node is a cluster of specialized cells between the atria and ventricles that slows down the electrical signal just long enough for the ventricles to fill with blood before they squeeze.

BODY'S JOB

Every heartbeat starts in the sinoatrial (SA) node in the right atrium, which fires a signal that spreads across both atria and makes them contract. That signal reaches the AV node, which delays it for about 0.12 to 0.20 seconds—like a traffic light pausing cars so pedestrians can cross—then passes it down the bundle of His, through the left and right bundle branches, and out to the Purkinje fibers so the ventricles squeeze in a smooth, coordinated wave. Without that brief delay, the atria and ventricles would contract at the same time and the ventricles would not fill properly, so your heart would pump less blood with every beat.

1. SA node fires in the right atrium (the natural pacemaker).
2. Signal spreads across both atria, making them contract and push blood into the ventricles.
3. Signal reaches the AV node (the gateway between atria and ventricles).
4. AV node delays the signal for 0.12 to 0.20 seconds so the ventricles can fill.
5. Signal passes through the bundle of His, then splits into the left and right bundle branches.
6. Purkinje fibers carry the signal to the walls of both ventricles, making them squeeze and pump blood to the lungs and body.

What breaks

AV block happens when the AV node or the wiring below it gets damaged, poisoned, inflamed, or was never built right in the first place.

CLINICAL TERM

AV block is caused by functional or structural disruption of conduction through the AV node or infranodal conduction system (bundle of His, bundle branches).

How the body compensates

When the AV node starts to fail, the heart tries to keep blood flowing by using backup pacemakers lower in the conduction system. These escape rhythms keep you alive but beat slower than normal, so your body squeezes harder and redirects blood to vital organs. If the block is complete and the escape rhythm is too slow or fails, blood pressure drops, you faint, and—without help—the heart can stop.

1. {'what': 'Junctional or ventricular escape rhythm kicks in', 'how': 'If the AV node stops letting signals through, cells in the bundle of His (junctional escape, usually 40 to 60 beats per minute) or in the ventricles themselves (ventricular escape, usually 20 to 40 beats per minute) start firing on their own to keep the heart beating. This is the heart's built-in backup generator.', 'limit': 'Escape rhythms are much slower than the normal 60 to 100 beats per minute, so less blood pumps out every minute. If the rate drops below about 40, the brain and kidneys do not get enough oxygen and you feel dizzy, weak, or faint.'}
2. {'what': 'Stroke volume increases (the heart squeezes harder)', 'how': 'To make up for fewer beats, the ventricles fill with more blood during the longer pauses and then squeeze harder, ejecting a bigger volume per beat (Frank-Starling mechanism). You might feel a strong, pounding heartbeat.', 'limit': 'If the heart is already damaged (from a heart attack, heart failure, or stiff ventricles), it cannot stretch or squeeze harder, so this compensation fails.'}
3. {'what': 'Blood flow is redirected to the brain and heart', 'how': 'Sensors in your arteries detect low blood pressure and tell blood vessels in the skin, gut, and muscles to tighten (vasoconstriction), shunting blood to the most critical organs. Your hands and feet may feel cold and look pale.', 'limit': 'This works only for a short time; if the heart rate stays very low, blood pressure eventually drops and you faint (syncope) because even the brain is not getting enough blood.'}
4. {'what': 'The kidneys hold onto salt and water (renal compensation)', 'how': 'Low blood pressure triggers the kidneys to release renin, which starts a cascade (renin-angiotensin-aldosterone system) that makes you retain fluid and raises blood volume, trying to boost blood pressure.', 'limit': 'If the heart rate does not recover, the extra fluid can back up into the lungs and legs, causing shortness of breath and swelling (congestive heart failure).'} }

5. {'what': 'Watch for sudden loss of escape rhythm (asystole)', 'how': 'If the AV block is complete and the escape pacemaker cells also fail or get suppressed (by low oxygen, high potassium, or more heart damage), the ventricles stop beating entirely—no pulse, no blood flow, and cardiac arrest within seconds.', 'limit': 'Once asystole happens, survival depends on immediate CPR and a pacemaker; there is no natural compensation left.'}

Domino effect

1. The AV node delays or blocks the electrical signal from the atria. — Damage, drugs, inflammation, or starved blood flow (ischemia) (not enough blood flow) slows or stops conduction through the AV node or the bundle branches below it, so fewer or no signals reach the ventricles.
2. First-degree AV block: every signal gets through, but slowly (PR interval longer than 0.20 seconds on the ECG). — The AV node is sluggish but still working, so every atrial beat eventually triggers a ventricular beat. You feel nothing because your heart rate and rhythm are normal—this is often found by accident on a routine ECG.
3. Second-degree AV block, Mobitz type I (Wenckebach): the PR interval gets longer with each beat until one signal is completely blocked and a QRS is dropped, then the cycle resets. — The AV node gets more and more tired with each beat (like a failing relay) until it cannot pass the next signal, so one ventricular beat is skipped. Because only occasional beats are dropped and the average heart rate stays near normal, most people have no symptoms or feel only mild lightheadedness or palpitations.
4. Second-degree AV block, Mobitz type II: the PR interval stays constant but suddenly a QRS is dropped without warning. — The block is below the AV node—in the bundle of His or bundle branches—so when conduction fails, it fails completely and unpredictably. Because this can progress to complete block at any moment, people often feel dizzy, weak, or faint, and this type always requires close monitoring or a pacemaker.
5. Third-degree (complete) AV block: no signals get through; the atria and ventricles beat independently (AV dissociation). — The AV node or infranodal conduction system is completely blocked, so the atria fire at their own rate (driven by the SA node, usually 60 to 100 per minute) and the ventricles fire at their own escape rate (40 to 60 if the escape focus is junctional, 20 to 40 if ventricular). The slow ventricular rate drops cardiac output, so you feel very tired, dizzy, short of breath, or faint. If the escape rhythm is too slow or stops, you lose consciousness and can die without emergency treatment.
6. Blood flow to the brain drops, causing dizziness, near-fainting, or fainting (syncope) (Stokes-Adams attacks). — When the heart beats too slowly or pauses for several seconds during the transition to an escape rhythm, blood pressure falls and the brain does not get enough oxygen. You feel lightheaded or faint; in severe cases you black out for a few seconds until the escape rhythm starts or the block resolves.

7. If compensation fails and no escape rhythm rescues the heart, asystole and cardiac arrest occur. — Complete heart block with no backup pacemaker means the ventricles stop beating entirely—no pulse, no blood flow, and death within minutes unless CPR and a temporary pacemaker are started immediately.
8. Finding and fixing the upstream cause determines whether the block is reversible or needs a permanent pacemaker. — If the block is caused by a reversible problem—medicine overdose, Lyme carditis treated with antibiotics, or starved blood flow (ischemia) from a heart attack that resolves—it may go away. If the block is from permanent scarring, degenerative disease, or congenital wiring, a pacemaker is the only way to keep the heart beating at a safe rate and prevent sudden death.

Symptoms · what the patient feels, and why

- Slow heart rate (bradycardia): The electrical signals between the top chambers (atria) and bottom chambers (ventricles) of the heart are blocked or delayed, so the ventricles beat more slowly than normal because they're waiting longer for the signal to arrive.
- Feeling dizzy or lightheaded: When the heart beats too slowly, it pumps less blood to the brain with each minute, so the brain doesn't get enough oxygen and feels foggy or dizzy.
- Feeling very tired or weak: The body's muscles and organs need a steady supply of oxygen-rich blood, and a slow heartbeat means less blood is delivered, leaving you feeling drained like a phone on low battery.
- Shortness of breath: When the heart can't pump enough blood forward, fluid can back up into the lungs, making it harder to breathe, especially during activity or when lying flat.
- Chest discomfort or pressure: The heart muscle itself needs blood and oxygen, and when the heart rate drops too low, the heart may not get enough fuel, causing a squeezing or heavy feeling in the chest.
- Fainting or near-fainting (syncope): If the block is severe, the heart may pause for several seconds, and the brain runs out of oxygen so quickly that you lose consciousness and collapse.
- Confusion or trouble thinking clearly: The brain is extremely sensitive to low oxygen, and even a slight drop in blood flow can make it hard to concentrate, remember things, or think straight.
- Long PR interval, skipped beats, or AV dissociation on electrocardiogram (ECG): The ECG shows the electrical pattern of the heart, and AV block stretches or breaks the normal connection between the atria and ventricles—you see either a longer pause (first-degree), a dropped beat (second-degree), or no connection at all (third-degree).

Focused exam

- Heart rate and rhythm — count the pulse and feel whether it is regular or irregular: AV block slows or skips the electrical signal from the top chambers (atria) to the bottom chambers (ventricles). A very slow pulse (under 50 beats per minute) or an irregular rhythm with long pauses tells you the signal is being delayed or dropped. This is your first clue that the heart's wiring is broken.
- Blood pressure — measure it now and check if it has been dropping: When AV block is severe, the ventricles beat too slowly to pump enough blood. Low blood pressure (systolic under 90 mmHg) means the brain and organs are not getting enough oxygen. This is a medical emergency.
- Mental status — is the patient alert, confused, or unconscious?: The brain needs steady blood flow to work. If AV block cuts the heart rate so low that blood flow drops, the patient gets lightheaded, confused, or passes out. These symptoms tell you the block is dangerous right now.
- 12-lead electrocardiogram (ECG) — look at the PR interval, dropped beats, and whether the atria and ventricles are beating together or separately: The ECG shows exactly where and how bad the block is. First-degree AV block has a long PR interval (over 0.20 seconds) but every atrial beat still gets through. Mobitz type II (second-degree) block drops some beats without warning. Third-degree (complete) block means the atria and ventricles fire on completely separate schedules — they are not talking to each other at all. The ECG pattern tells you whether the patient needs a pacemaker urgently.
- Neck veins — are they swollen (distended) or flat?: When AV block is severe, the atria keep contracting but the ventricles do not pump blood forward fast enough. Blood backs up into the veins, making the neck veins bulge. This sign tells you the heart is failing to move blood through the body.
- Skin — is it warm and dry, or cold and clammy?: Cold, clammy skin means the body is not getting enough blood flow and is shutting down blood to the skin to save the brain and heart. AV block that drops the heart rate low enough can cause this shock state.

Diagnostics · every test answers a question

- 12-lead electrocardiogram (ECG) → What type of AV block is present, and how severe is it?
- Continuous cardiac monitoring (telemetry) → Is the block getting worse? Are there dangerous pauses or escape rhythms failing?
- Serum electrolytes — potassium, magnesium, calcium → Are electrolyte imbalances slowing AV node conduction?
- Troponin I or T → Is the AV block caused by a heart attack (MI) damaging the AV node or bundle branches?

- Lyme disease serology (ELISA, then Western blot if positive) → Is Lyme carditis (infection spreading to the heart) causing the AV block?
- Medication review — check for beta-blockers, calcium channel blockers (diltiazem, verapamil), digoxin, antiarrhythmics (amiodarone, sotalol) → Is a medication slowing AV node conduction and causing the block?
- Echocardiogram (ultrasound of the heart) → Is there structural heart disease (valve disease, infiltrative disease like sarcoidosis or amyloidosis) causing the AV block?
- Chest X-ray → Is there heart enlargement or lung congestion from heart failure caused by slow heart rate?

Treatment logic

- Atropine 0.5–1 mg IV push (can repeat every 3–5 minutes, max 3 mg total) for symptomatic AV block at or above the AV node level → Atropine blocks the vagus nerve signals that slow the AV node. This speeds up electrical conduction from atria to ventricles and raises the heart rate. It works best for Mobitz type I (Wenckebach) and some cases of first-degree block.
- Transcutaneous pacing (electrical pads on the chest delivering pacing pulses) or transvenous temporary pacemaker wire inserted through a vein into the right ventricle → Pacing sends electrical signals directly to the ventricles, forcing them to contract at a safe rate (usually 60–80 beats per minute). This bypasses the broken AV node and restores blood flow to the brain and body immediately. Use this urgently if atropine fails or the block is Mobitz II or third-degree.
- Permanent pacemaker implantation (a small device placed under the skin with wires threaded into the heart) → A permanent pacemaker continuously monitors the heart rhythm and delivers electrical pulses whenever the natural signal fails. It keeps the heart rate steady for life and prevents fainting (syncope) (fainting), heart failure, and sudden death. Guidelines (AHA/ACC/HRS 2018) recommend permanent pacing for Mobitz II, third-degree AV block, or symptomatic Mobitz I that does not reverse with treatment of the cause.
- Restore blood flow to the right coronary artery (RCA) with percutaneous coronary intervention (PCI) or thrombolytics if AV block is caused by acute inferior MI → The RCA supplies the AV node. Opening the blocked artery brings oxygen back to the AV node cells, allowing them to recover electrical function. Many cases of AV block from inferior MI improve or resolve completely within hours to days after reperfusion.
- Stop or reduce the dose of AV-blocking drugs (beta-blockers like metoprolol, calcium channel blockers like diltiazem or verapamil, digoxin, amiodarone) → Removing the drug that is slowing the AV node lets the natural conduction speed return to normal. Many medication-induced AV blocks resolve within hours to days after stopping the drug.

- IV ceftriaxone 2 g daily for 14–21 days if AV block is caused by Lyme carditis (confirmed by positive Lyme serology) → Ceftriaxone kills the Lyme bacteria (*Borrelia burgdorferi*) and reduces inflammation in the AV node. Most patients see their AV block improve or resolve completely within 1–2 weeks. Temporary pacing is often needed as a bridge until antibiotics work.
- Correct electrolyte abnormalities — give IV insulin and glucose or sodium polystyrene sulfonate (Kayexalate) for high potassium (hyperkalemia), IV magnesium sulfate for low magnesium, IV fluids and calcitonin or bisphosphonates for high calcium → Normal electrolyte levels restore the electrical gradients AV node cells need to fire and conduct signals properly. Correcting high potassium (hyperkalemia) (potassium over 5.5 mEq/L) often reverses AV block within minutes to hours.

Medications

Atropine · Anticholinergic (blocks parasympathetic signals)

Atropine blocks the vagus nerve's 'slow down' signals to the AV node. The vagus nerve normally releases a chemical called acetylcholine that tells the AV node to go slower; atropine gets in the way of that chemical so the node can speed up. This only works if the problem is in the AV node itself (nodal block, like Mobitz type I or some cases of first-degree block), because atropine acts at the node. If the block is lower down in the bundles (infranodal, like Mobitz II or third-degree block), atropine won't help and may even make things worse.

1. Atropine enters the bloodstream and reaches the AV node.
2. It sits on acetylcholine receptors and blocks the vagus nerve's 'slow' messages.
3. The AV node is now less inhibited, so electrical signals can travel through faster.
4. Heart rate increases and more atrial beats conduct to the ventricles.

SAFETY WATCH

Can cause fast heart rate (tachycardia) if you give too much. · May worsen block in infranodal AV block by speeding atrial rate without improving conduction below the node. · Can cause dry mouth, blurry vision, confusion, and trouble urinating (classic anticholinergic side effects). · Use cautiously in older adults because confusion and urinary retention are more common.

Temporary transcutaneous or transvenous pacing · Device therapy (electrical pacing, not a drug)

When the heart's own electrical system is blocked and too slow to keep the body fed with blood, an external device steps in to deliver small electrical pulses that make the ventricles

contract. Transcutaneous pacing uses sticky pads on the chest; transvenous pacing threads a wire through a vein into the right ventricle. Either way, the device acts as a stand-in conductor, sending regular beats until the problem is fixed or a permanent pacemaker is placed.

1. The pacing device sends out a timed electrical pulse.
2. The pulse reaches the heart muscle (either directly via a wire or through the chest wall).
3. The ventricle contracts in response to the pulse.
4. Blood is pumped out to the body, maintaining blood pressure and organ blood flow (perfusion).

SAFETY WATCH

Transcutaneous pacing is painful and requires sedation or analgesia if used for more than a few minutes. · Transvenous pacing carries risks of bleeding, infection, pneumothorax (collapsed lung) during insertion, and wire dislodgment. · Requires continuous monitoring; if the wire moves or the pads lose contact, pacing fails. · Must transition to permanent pacemaker if block does not resolve.

Permanent pacemaker implantation · Device therapy (surgical implant, not a drug)

A permanent pacemaker is a small battery-powered computer implanted under the skin, usually below the collarbone, with one or two wires (leads) threaded into the heart. It constantly watches the heart's rhythm and, whenever it detects a pause or a blocked beat, it sends a tiny electrical pulse to make the heart contract. This is the definitive treatment for AV blocks that won't resolve (Mobitz type II, third-degree block, symptomatic a slow heart rate (bradycardia) from any high-grade block). The pacemaker takes over the job of the broken conduction system permanently.

1. The pacemaker continuously senses the heart's own electrical activity.
2. When it detects a missing or blocked beat, it delivers a pacing pulse through the lead.
3. The pulse triggers a contraction in the atrium, ventricle, or both (depending on the type of pacemaker).
4. The heart rate stays steady and the patient's symptoms (dizziness, fatigue, fainting (syncope)) resolve.

SAFETY WATCH

Surgical risks: bleeding, infection at the implant site, collapsed lung (pneumothorax), lead dislodgment. · Device infection (endocarditis on the leads) is serious and may require removal and long-term antibiotics. · Battery will eventually run out and require replacement surgery. · Patients must avoid strong magnets and certain medical equipment (MRI compatibility varies by model; always check). · Regular device checks (every 3–12 months) are required to ensure proper function.

Reversal of causative drugs (e.g., stopping beta-blockers, calcium channel blockers, digoxin, or other AV-nodal blocking agents) · Medication discontinuation or antidote administration (not a single drug)

Many common heart and blood pressure medicines work by slowing conduction through the AV node. If the patient is taking beta-blockers (like metoprolol), non-dihydropyridine calcium channel blockers (like diltiazem or verapamil), or digoxin, these drugs can cause or worsen AV block. Stopping the offending drug—or giving an antidote if needed (e.g., digoxin immune Fab for digoxin toxicity, calcium and glucagon for beta-blocker or calcium-channel-blocker overdose)—removes the brake on the AV node and often allows conduction to recover.

1. Identify any drug the patient is taking that slows AV conduction.
2. Stop the drug or reduce the dose.
3. If overdose or toxicity, give the specific antidote (digoxin immune Fab for digoxin, IV calcium and glucagon for beta-blocker or calcium-channel-blocker toxicity).
4. The drug's effect on the AV node wears off over hours to days (depending on the drug's half-life).
5. Conduction through the node improves and the block may resolve.

SAFETY WATCH

Abruptly stopping beta-blockers or calcium channel blockers can cause rebound high blood pressure or worsening angina; taper when possible. · Digoxin immune Fab is expensive and reserved for true digoxin toxicity; it binds digoxin and clears it but may cause rebound high potassium (hyperkalemia). · Calcium and glucagon for beta-blocker or calcium-channel-blocker overdose must be given IV in a monitored setting; they can cause nausea, vomiting, and high blood sugar (hyperglycemia). · Always check potassium and magnesium levels when reversing these drugs, as electrolyte shifts can worsen arrhythmias.

Electrolyte repletion (potassium, magnesium, calcium correction) · Electrolyte supplementation (not a single drug; includes IV or oral potassium chloride, magnesium sulfate, calcium gluconate)

The heart's electrical system depends on a precise balance of electrolytes—especially potassium, magnesium, and calcium—to conduct signals properly. Low potassium (hypokalemia) and low magnesium (hypomagnesemia) make cells more irritable and can slow or block conduction. High potassium (hyperkalemia) or very low calcium (hypocalcemia) can also impair conduction. Correcting these imbalances restores normal electrical flow through the AV node and bundles and may resolve or improve the block if it was caused or worsened by the electrolyte problem.

1. Measure serum potassium, magnesium, and calcium levels.
2. If low, give replacement: IV or oral potassium chloride for low potassium (hypokalemia), IV magnesium sulfate for low magnesium (hypomagnesemia), IV calcium gluconate for severe low calcium (hypocalcemia).
3. The electrolytes enter the bloodstream and are taken up by heart cells.
4. Normal ion gradients are restored, and the cells can generate and conduct electrical signals properly again.
5. AV conduction improves and the block may lessen or resolve.

SAFETY WATCH

IV potassium must be given slowly (no faster than 10–20 mEq/hour in peripheral IV, faster rates only through central line with monitoring) because rapid infusion can cause dangerous heart rhythms or cardiac arrest. · Magnesium infusion can cause flushing, low blood pressure, and, in large doses, respiratory depression; give slowly and monitor. · Too much calcium (hypercalcemia) from over-replacement can cause heart block or arrhythmias; correct cautiously. · Always recheck levels after replacement and watch for rebound or ongoing losses (e.g., diarrhea, diuretics).

Treatment of underlying cause (e.g., antibiotics for Lyme disease, corticosteroids for sarcoidosis, management of starved blood flow (ischemia)/ tissue death from blocked blood flow (infarction)) · Disease-specific therapy (varies by cause)

AV block can be caused by infection (Lyme carditis is the classic example in endemic areas), inflammation (sarcoidosis, myocarditis), starved blood flow (ischemia) (blocked coronary artery starving the conduction system), or infiltration (amyloidosis, hemochromatosis). Treating the root cause—antibiotics (doxycycline or ceftriaxone) for Lyme, corticosteroids for sarcoidosis, revascularization (stent or bypass) for starved blood flow—can stop or reverse the damage to

the conduction tissue. If caught early, the block may resolve completely; if tissue is already scarred, a pacemaker may still be needed, but treating the cause prevents further damage.

1. Identify the underlying cause through history, exam, labs (Lyme serology, ACE level, cardiac biomarkers), imaging (echo, cardiac MRI), or biopsy.
2. Start targeted therapy: antibiotics for Lyme carditis, corticosteroids ± immunosuppressants for sarcoidosis or autoimmune myocarditis, reperfusion (PCI or thrombolytics) for acute MI, chelation for hemochromatosis.
3. The treatment reduces infection, inflammation, or starved blood flow (ischemia).
4. Swelling and damage to the conduction tissue decrease.
5. Conduction improves; the block may downgrade (e.g., third-degree to first-degree) or resolve entirely.

SAFETY WATCH

Lyme carditis with high-grade block requires hospitalization, telemetry, and often temporary pacing while antibiotics work; IV ceftriaxone is preferred for carditis. · Corticosteroids for sarcoidosis or myocarditis must be tapered slowly to avoid rebound inflammation; side effects include infection risk, high blood sugar (hyperglycemia), osteoporosis. · Revascularization for blood-starved (ischemic) block carries procedural risks (bleeding, stroke, contrast-induced kidney injury). · Monitor closely during treatment; some patients will need a pacemaker even after treating the cause.

Red flags

- Fainting or near-fainting (syncope)
- Sudden severe dizziness or confusion
- Chest pain or pressure that doesn't go away
- Very slow heart rate (under 40 beats per minute) with symptoms
- Shortness of breath at rest or worsening quickly

Nursing priorities

1. Place patient on continuous cardiac monitor (telemetry) and watch for pauses longer than 3 seconds or heart rate dropping below 40 beats per minute — Long pauses mean the ventricles (bottom chambers) are not getting signals to pump, causing low cardiac output (amount of blood the heart pumps) and risk of cardiac arrest (heart stopping)
2. Establish IV access (intravenous line) with at least one large-bore (18-gauge or bigger) catheter — You need immediate IV access to give atropine (a medicine that speeds up the heart) or start a dopamine or epinephrine drip if the heart rate drops dangerously low

3. Keep atropine 1 mg prefilled syringe and transcutaneous pacing pads at bedside, and know how to activate the pacer function on the defibrillator — Symptomatic a slow heart rate (bradycardia) (slow heart rate causing symptoms) can deteriorate to cardiac arrest within minutes, so you must be ready to give atropine or start emergency pacing immediately
4. Hold (do not give) beta-blockers, calcium channel blockers, and digoxin until cleared by the provider — These medicines slow conduction through the AV node (the electrical relay station between top and bottom chambers) and can worsen the block or prevent atropine from working
5. Assess and document level of consciousness, blood pressure, and symptoms (dizziness, chest pain, shortness of breath) every 15 minutes if symptomatic — Worsening mental status or dropping blood pressure means cerebral poor blood flow (hypoperfusion) (not enough blood reaching the brain) from low cardiac output, requiring urgent pacing or pressors (medicines that raise blood pressure)
6. Keep patient on bedrest with call bell within reach and assist with all activity including toileting — Standing or straining increases vagal tone (nerve signals that slow the heart further) and can trigger fainting (syncope) (fainting) and falls when the heart rate is already dangerously slow
7. Obtain 12-lead ECG (electrocardiogram showing electrical activity from 12 views) and compare to any prior ECGs to identify new changes — New ST elevation (a specific ECG pattern) indicates acute heart attack (myocardial infarction) causing the AV block, requiring emergency cardiac catheterization, while old Q waves suggest chronic scarring that may need permanent pacing
8. Draw and monitor troponin (a heart enzyme), electrolytes (especially potassium and magnesium), and digoxin level if patient takes digoxin — Elevated troponin confirms heart muscle damage from heart attack (myocardial infarction); high potassium (hyperkalemia) or digoxin toxicity can cause reversible AV block that resolves when the level is corrected

Sources

- ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay (2018)
- European Society of Cardiology (ESC) Guidelines on Cardiac Pacing and Cardiac Resynchronization Therapy (2021)
- American Heart Association (AHA)
- UpToDate Clinical Reference

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