

Bradycardia

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THE STORY IN ONE SENTENCE

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“A slow heart rate (Bradycardia) means first decide: is the patient feeling symptoms or not? If yes, act fast. Then figure out which part of the heart's electrical system is slow or blocked — the pacemaker, the relay station, or the wires below — because that tells you how to fix it and how urgent it is.”

Normal anatomy — the body's job

The heart beats because of an electrical system. At the top of the heart, a natural pacemaker called the SA node fires a spark 60 to 100 times every minute. That spark travels down to a relay station (the AV node), then races along wires (the bundle of His and bundle branches) to the bottom pumping chambers (the ventricles) so they squeeze and push blood out to the body. Every heartbeat is one cycle: spark, travel, squeeze, pump.

BODY'S JOB

The SA node is the boss — it sets the rhythm. The AV node is the gatekeeper — it checks each signal and passes it down (with a tiny delay so the top chambers can finish squeezing first). The ventricles are the muscle — they do the heavy pumping. Normally the heart beats fast enough to deliver about 5 liters of blood every minute to feed your brain, kidneys, liver, and every other organ. If you don't understand this normal cycle, a slow heart rate will seem like just a number instead of a real problem you can picture and fix.

1. The SA node (a cluster of pacemaker cells in the top-right corner of the heart) fires an electrical spark automatically, about once per second.
2. The spark spreads across the two top chambers (the atria) like a wave, making them squeeze and push blood down into the bottom chambers.
3. The spark reaches the AV node (a relay station sitting between the top and bottom chambers). The AV node pauses the signal for a split second so the top chambers can finish their job.
4. The signal then shoots down a thick wire called the bundle of His, which splits into left and right bundle branches — one for each bottom chamber (ventricle).
5. The ventricles squeeze hard and push blood out: the right ventricle sends blood to the lungs to pick up oxygen, and the left ventricle sends oxygen-rich blood to the whole body.
6. After each beat, the system resets and the SA node fires again. This cycle repeats 60 to 100 times every minute, every day, for your whole life.

What breaks

A slow heart rate (Bradycardia) happens when the heart's electrical system slows down or gets blocked. The break can be at the top (the pacemaker fires too slowly or stops), in the middle (the relay station blocks signals), or below (the wires are damaged so signals never reach the pumping chambers). Often one cause explains all the symptoms together.

CLINICAL TERM

The most common causes are: (1) Medicines — beta-blockers (used for high blood pressure or anxiety), calcium-channel blockers like diltiazem or verapamil, and digoxin (a heart medicine) all slow the heart on purpose, but sometimes too much. (2) Starved blood flow (Ischemia) — a heart attack, especially one on the bottom wall of the heart (inferior MI), damages the SA node or AV node because their blood supply is cut off. (3) High vagal tone — the vagus nerve is like a brake pedal for the heart; things like vomiting, bearing down, or pressure on the eyeball can slam that brake and drop the heart rate suddenly. (4) Sick sinus syndrome — the SA node wears out with age and stops firing reliably. (5) AV block — the relay station starts blocking signals; first-degree is a delay (usually harmless), second-degree means some beats get through and some don't, third-degree (complete heart block) means nothing gets through so the ventricles have to beat on their own, very slowly. (6) Metabolic problems — low thyroid hormone (hypothyroidism) slows everything in the body including the heart; high potassium (hyperkalemia) can stop the heart; low body temperature (hypothermia) slows all chemical reactions. (7) Increased pressure inside the skull (from bleeding, a tumor, or swelling) — the brain's control center gets squeezed and tells the heart to slow down (Cushing reflex). (8) Infections near the heart (like Lyme disease or endocarditis) can damage the conduction system. The broken part stays local, but the effects spread: when the heart beats too slowly, cardiac output (the amount of blood pumped every minute) drops, and that is what causes the symptoms you see at the bedside — fainting, low blood pressure, confusion, chest pain. Every weird finding points back upstream to the same broken thing: not enough beats per minute.

How the body compensates

The body tries hard to keep blood pressure and oxygen delivery steady even when the heart is beating too slowly. It squeezes blood vessels tighter, releases adrenaline, and makes each heartbeat pump more blood. For a while, vital signs can look okay. That is the dangerous middle — the patient seems stable, but they are one step away from a crash. Your job is to see that cliff coming before they fall off it.

1. {'what': "The sympathetic nervous system (the body's fight-or-flight mode) releases adrenaline and norepinephrine to try to speed the heart rate back up and squeeze blood vessels tighter, raising blood pressure.", 'limit': "This only works if the heart's electrical system can still respond. In complete heart block or severe sick sinus syndrome, adrenaline cannot fix the broken wiring, so compensation fails."}

2. {'what': 'The ventricles stretch and fill with more blood between beats (because the pauses are longer), so each squeeze pumps out a bigger volume (increased stroke volume) to make up for fewer beats per minute.', 'limit': 'If the heart rate drops too low (below 40 or so), even a bigger stroke volume cannot make up the difference — total cardiac output still falls and the patient crashes.'}
3. {'what': 'Blood vessels in the arms, legs, and gut constrict (get narrower) to redirect blood flow to the brain, heart, and kidneys — the organs that must stay alive.', 'limit': 'This makes the skin pale, cold, and clammy. It is a sign the body is in crisis mode. It also raises the workload on the heart, which can make starved blood flow (ischemia) worse if the slow rate was caused by a heart attack.'}
4. {'what': 'The kidneys sense low blood flow and start holding onto salt and water to keep blood volume up and blood pressure from dropping further.', 'limit': 'This takes hours to days to help, so it does not protect against sudden a slow heart rate (bradycardia). If the slow heart rate continues, the patient can develop fluid overload and heart failure from too much volume in a weakly pumping heart.'}
5. {'what': 'The patient may feel their heart pounding or skipping (palpitations) as the body tries different rhythms — backup pacemakers firing, extra beats (PVCs), or sudden pauses. This is the heart's electrical system scrambling to find a rhythm that works.', 'limit': 'These backup rhythms are unstable. The patient can suddenly lose consciousness or go into a dangerous rhythm like ventricular a fast heart rate (tachycardia) or asystole (flat line).'}- 6. {'danger': 'Watch for the crash point: if the slow rate is from a block below the AV node (infranodal block, like Mobitz II or complete heart block), giving atropine (a medicine that speeds the heart by blocking the vagus nerve) can make things worse — it speeds up the SA node but the signals still cannot get through, so the ventricles stay slow and the top and bottom of the heart fight each other. The patient can arrest. In that case, you need a pacemaker or external pacing immediately, not atropine.'}
- 7. {'danger': 'If the patient has a long QT interval on the ECG (a sign the ventricles take too long to reset electrically), a slow heart rate can trigger torsades de pointes — a twisting, chaotic rhythm that looks like a tornado on the monitor and can become ventricular fibrillation (the heart just quivers and stops pumping). Treat with magnesium and speed the heart rate up with pacing or isoproterenol, not more medicines that slow it.'}
- 8. {'principle': 'A patient can look stable — talking, awake, normal blood pressure — while sitting on the edge of a cliff. Look at the trend: is the heart rate dropping? Is the blood pressure slowly falling? Are there more and more pauses on the monitor? Are symptoms getting worse? A stable patient on a downhill slope is not really stable. Act before the crash, not after.'}

Domino effect

1. The heart's natural pacemaker (SA node) fires too slowly, or the relay station (AV node) blocks signals, or the wires below are damaged — so fewer than 60 electrical sparks per minute reach the ventricles. — Medicines (beta-blockers, calcium-channel blockers, digoxin) put the brakes on the heart's electrical system. A heart attack (especially on the bottom wall) cuts off blood to the SA or AV node, starving the pacemaker cells. The vagus nerve (the body's natural brake) can slam on too hard during vomiting, straining, or even coughing. The SA node can just wear out with age. Or the AV node starts blocking signals — first some, then all. Any of these causes means fewer beats per minute leave the starting gate.
2. Cardiac output (the amount of blood pumped every minute) drops because cardiac output equals heart rate times stroke volume — if the rate is cut in half, output is cut in half, even if each beat pumps a normal amount. — Imagine a factory that ships 70 boxes per minute. If it slows to 35 boxes per minute, it does not matter if each box is bigger — total product delivered still falls. The body needs about 5 liters of blood pumped every minute to keep organs fed. When the heart rate drops below 40 or 50, that delivery system fails.
3. Blood pressure falls because blood pressure depends on cardiac output and how tight the blood vessels are squeezed — when cardiac output drops, pressure drops unless the body can squeeze vessels tight enough to compensate. — At first, adrenaline and vessel constriction keep the pressure up. But if the heart rate stays very slow or keeps dropping, compensation maxes out and blood pressure crashes. You see systolic pressure (the top number) fall below 90, sometimes below 70. The patient feels dizzy and weak.
4. The brain runs out of oxygen and blood flow, so consciousness fades — the patient feels lightheaded, their vision goes gray or black, and they faint (syncope) or collapse. — The brain uses 20% of the body's oxygen even though it is only 2% of body weight. It has almost no backup fuel stored, so it depends on a constant flow of oxygen-rich blood every second. When blood pressure drops and cardiac output falls, the brain is the first organ to fail. Fainting (Syncope) is not a separate disease — it is the brain's alarm bell that the delivery system is broken.

5. Other organs start to suffer: the kidneys make less urine or stop completely (acute kidney injury), the skin turns pale and cold and clammy, the patient becomes confused or unresponsive (altered mental status), and the heart itself does not get enough blood (angina — chest pain — even if the coronary arteries were fine before). — Every organ is downstream from the pump. When cardiac output is low, blood flow is rationed to the brain and heart first; everything else is starved. The kidneys sense low pressure and think the body is dehydrated, so they shut down to save water — but really the problem is the pump is too slow. The heart muscle itself needs oxygen delivered by the coronary arteries; if the heart is beating slowly and blood pressure is low, coronary blood flow drops and the heart starves itself — this can cause more damage and make the a slow heart rate (bradycardia) worse (a vicious cycle).
6. The body releases a flood of adrenaline and squeezes blood vessels tight, trying to force blood pressure back up and speed the heart rate — but if the electrical system is broken (like in complete heart block), adrenaline cannot fix the wiring, so the patient stays slow and unstable. — Compensation is the body buying time, not fixing the root problem. You will see pale, sweaty skin (from blood vessels constricting), anxiety or agitation (from adrenaline), and sometimes extra heartbeats or pauses on the monitor (as backup pacemakers try to take over). This is a red flag: the body is in crisis mode and the system is about to fail.
7. If untreated, the patient can go into cardiac arrest — the slow rhythm stops completely (asystole — a flat line), or it degenerates into a chaotic rhythm like ventricular fibrillation (the heart just quivers and pumps nothing). — A heart that beats 30 times per minute is barely keeping the body alive. The backup pacemaker cells in the ventricles are not designed to run the whole heart — they are fragile and unreliable. Any stress (low oxygen, high potassium, another blocked signal) can make them stop. Once the heart stops, you have minutes to restart it with CPR, pacing, or defibrillation before brain damage or death.
8. Every symptom — the dizziness, the fainting, the confusion, the chest pain, the low blood pressure — is not a separate problem. They are all one chain caused by one broken thing upstream: the heart is not beating fast enough to deliver blood. Fix the heart rate, and the whole chain reverses. — This is why the first question is always: does the patient have symptoms? If yes, the a slow heart rate (bradycardia) is dangerous and you must act — give atropine (if the block is at the AV node level or above), place a temporary pacemaker wire, or use external pacing pads to shock the heart into beating faster from the outside. If the patient has no symptoms and the rate is only mildly slow, you can watch and find the cause without rushing. But never assume slow and stable means safe — check the trend, check the ECG to see where the block is, and be ready to pace before the patient crashes.

Symptoms · what the patient feels, and why

- **Slow heart rate (below 60 beats per minute at rest):** The heart's electrical system is sending signals more slowly than normal, or signals are being blocked on their way through the heart. This means fewer heartbeats happen each minute.
- **Feeling dizzy or lightheaded:** When the heart beats too slowly, it may not pump enough blood up to the brain. The brain needs a steady stream of oxygen-rich blood to work well, so when it runs low, you feel woozy or unsteady.
- **Extreme tiredness or weakness:** Muscles and organs throughout the body need oxygen from blood to make energy. A slow heart rate means less blood is delivered each minute, so your body feels drained and weak—like trying to run a house on a battery that's running low.
- **Shortness of breath, especially with activity:** When you move, your muscles need more oxygen. A slow heart can't speed up enough to meet that demand, so blood backs up in the lungs and you feel short of breath—like a traffic jam where cars (blood cells) can't move forward fast enough.
- **Chest discomfort or pressure:** The heart muscle itself needs oxygen. If the heart beats too slowly, the blood flow through the heart's own arteries may drop, starving the heart tissue and causing a squeezing or heavy feeling in the chest.
- **Fainting or near-fainting spells:** If the heart rate suddenly drops very low or pauses for a few seconds, blood pressure plummets and the brain shuts down briefly to protect itself. You lose consciousness for a moment and may fall.
- **Confusion or trouble concentrating:** The brain is extremely sensitive to low oxygen. When a slow heart delivers less blood to the brain over time, thinking becomes foggy and memory slips—like a computer running too many programs on low power.
- **Cannon A waves seen in the neck veins (during complete heart block):** In complete heart block, the top chambers (atria) and bottom chambers (ventricles) beat independently. Sometimes the atria squeeze at the exact moment a valve is closed, so blood slams backward into the neck veins and causes a visible bulge or wave—like water hitting a closed door and bouncing back.

Focused exam

- **Heart rate below 60 beats per minute, regular rhythm on the monitor:** A slow heart rate means either the heart's natural pacemaker (the SA node) is firing too slowly, or the electrical signal is being blocked somewhere along the path to the ventricles. A regular rhythm tells you the signal is still organized—the heart is just running slow, not chaotic.

- Blood pressure drops by more than 20 mmHg when the patient sits up or stands: When the heart beats too slowly, it pumps less blood each minute (like a water pump running in slow motion). When the patient stands and gravity pulls blood down to the legs, the slow heart cannot speed up to push blood back up to the brain. This drop in blood pressure means the slow heart rate is already causing problems.
- Patient reports dizziness, feeling lightheaded, or nearly fainting (presyncope): The brain needs a steady stream of oxygen-rich blood to work. A slow heart rate cuts that stream. Dizziness or near-fainting means the brain is not getting enough blood right now—this is a warning sign that the a slow heart rate (bradycardia) is dangerous and could lead to a full blackout.
- Cannon A waves—sudden bulging pulses in the neck veins—visible in complete heart block: In complete heart block, the atria and ventricles beat independently. Sometimes the atrium squeezes against a closed valve, forcing blood backward into the neck veins and causing a sudden visible bulge. This sign tells you the atria and ventricles have lost all communication.
- Slow pulse, cool and clammy skin, confusion or lethargy: Cool, sweaty skin and confusion mean the body is shutting down blood flow to the skin and using less energy to keep the brain and heart alive. This is the body's emergency response to a heart that is not pumping enough blood (cardiogenic shock). The slow heart rate has become life-threatening.
- ECG shows PR interval longer than 200 milliseconds, dropped QRS complexes after some P waves, or P waves and QRS complexes with no relationship at all: The ECG shows the electrical path. A PR interval over 200 ms means the signal is delayed at the AV node (first-degree block). Dropped QRS complexes mean some signals never make it through (second-degree block). P waves and QRS complexes moving independently mean complete heart block—the signal is fully blocked and the ventricles are beating on their own backup pacemaker, which is dangerously slow.

Diagnostics · every test answers a question

- 12-lead electrocardiogram (ECG) → What is the heart rate, and is there a block in the electrical pathway between the atria and ventricles?
- Serum electrolytes (sodium, potassium, calcium, magnesium) → Are there electrolyte imbalances that can slow the heart's electrical system?
- Thyroid-stimulating hormone (TSH) and free T4 → Is the thyroid gland underactive, slowing the body's metabolism and heart rate?
- Troponin I or T → Is there damage to the heart muscle from a heart attack that could be affecting the electrical system?
- Medication levels: digoxin level, review of beta-blocker or calcium channel blocker dosing → Are medications that slow the heart building up to toxic levels?

- Chest X-ray → Is there heart enlargement or fluid in the lungs that suggests the slow heart rate is causing heart failure?

Treatment logic

- Atropine 1 mg IV push, repeat every 3 to 5 minutes up to a total dose of 3 mg → Atropine blocks the vagus nerve (the nerve that acts like the body's brake pedal on the heart). Blocking the vagus nerve removes the brake and lets the SA node and AV node fire faster. Heart rate increases within 1 to 2 minutes, which immediately increases blood flow to the brain and organs.
- Transcutaneous pacing—large adhesive pads placed on the chest and back deliver electrical pulses that force the heart to beat at a set rate → The pacing pulses bypass the heart's broken electrical system entirely and directly stimulate the ventricles to contract. Heart rate is immediately controlled (usually set to 60 to 80 beats per minute), restoring blood pressure and organ blood flow (perfusion) within seconds.
- Stop the drug causing a slow heart rate (bradycardia)—immediately hold beta-blockers (e.g., metoprolol, atenolol), non-dihydropyridine calcium channel blockers (e.g., diltiazem, verapamil), or digoxin → Removing the drug removes the chemical signal that was slowing the heart. As the drug level in the blood drops over hours to days (depending on kidney and liver function), the heart's natural pacemaker speeds back up on its own. In many cases, this is the only intervention needed if the patient is otherwise stable.
- Glucagon 3 to 5 mg IV bolus followed by infusion of 3 to 5 mg/hour for beta-blocker overdose; calcium chloride 1 g IV (10 mL of 10% solution) or calcium gluconate 3 g IV (30 mL of 10% solution) for calcium channel blocker overdose → Glucagon works through a pathway that does not depend on beta receptors, so it can increase heart rate and force of contraction even when beta-blockers are blocking the usual pathway. Calcium directly competes with calcium channel blockers at the cell membrane, restoring the flow of calcium into heart cells and allowing the heart to speed up and contract more strongly.
- Epinephrine infusion (2 to 10 micrograms per minute) or dopamine infusion (5 to 20 micrograms per kilogram per minute) if atropine and pacing do not restore adequate blood pressure → Epinephrine and dopamine act like adrenaline. They speed up the heart rate by directly stimulating receptors in the SA node and also squeeze blood vessels to raise blood pressure. This buys time by keeping blood flowing to the brain and heart while you figure out and fix the root cause.

- Permanent pacemaker implantation for symptomatic sinus node dysfunction (sick sinus syndrome), Mobitz II second-degree AV block, or complete (third-degree) heart block → A permanent pacemaker is a small device implanted under the skin with wires (leads) threaded into the heart. It monitors the heart's rhythm continuously and delivers tiny electrical pulses whenever the heart rate drops below a safe threshold (usually 60 beats per minute). This provides a reliable backup electrical system for life, preventing fainting (syncope) and sudden death.
- Treat underlying causes: levothyroxine for hypothyroidism, coronary revascularization (PCI or CABG) for starved blood flow (ischemia) causing a slow heart rate (bradycardia), dialysis or insulin-glucose-calcium for severe high potassium (hyperkalemia) → Fixing the root cause removes the trigger for a slow heart rate (bradycardia). Replacing thyroid hormone speeds up metabolism and heart rate. Reopening a blocked artery restores blood flow to the SA or AV node. Lowering potassium restores normal conduction. When the underlying problem is corrected, the heart rate often returns to normal on its own.

Medications

Atropine · Anticholinergic (blocks the vagus nerve's slow-down signal)

Atropine blocks acetylcholine (the chemical the vagus nerve uses to slow your heart). When you block that brake signal, the heart can speed up. It works best when the problem is at the SA node (your heart's natural pacemaker) or the AV node (the gate between the top and bottom chambers).

1. The vagus nerve releases acetylcholine, which attaches to receptors on SA and AV node cells and slows them down.
2. Atropine blocks those receptors so acetylcholine cannot land and slow the heart.
3. With the brake released, the SA node fires faster and the AV node passes signals more quickly.
4. Heart rate rises, usually within 1–2 minutes after the IV dose.

SAFETY WATCH

Do NOT give doses smaller than 0.5 mg IV—tiny doses can paradoxically slow the heart even more. · Watch for confusion, dry mouth, blurred vision, or trouble urinating (atropine blocks acetylcholine everywhere, not just the heart). · Use carefully in people with glaucoma or urinary-retention problems; blocking acetylcholine can make both worse. · If three doses (total 3 mg) do not work, move to pacing or other drugs—more atropine will not help.

Epinephrine infusion · Catecholamine (directly pushes the heart to beat faster and harder)

Epinephrine mimics adrenaline. It lands on beta-1 receptors in the heart and tells pacemaker cells to fire faster and heart-muscle cells to squeeze harder. It is used when atropine fails or the block is below the AV node.

1. Epinephrine binds to beta-1 receptors on heart cells.
2. This turns on an enzyme (adenylyl cyclase) that raises cyclic AMP inside the cell.
3. More cyclic AMP opens calcium channels and speeds up the pacemaker current (funny current, If).
4. Pacemaker cells fire faster, the heart rate climbs, and each beat is stronger.

SAFETY WATCH

Start at a LOW infusion rate (2–10 micrograms per minute) and titrate carefully; too much causes dangerously fast heart rates, chest pain, or life-threatening arrhythmias. · Monitor blood pressure closely—epinephrine can spike it and cause headache or even stroke if pressure climbs too high. · Watch for tremor, anxiety, and pale or cool skin (signs the dose is too high). · This is a bridge to pacing, not a long-term solution.

Dopamine infusion · Catecholamine (pushes the heart and, at higher doses, squeezes blood vessels)

Dopamine acts on different receptors depending on the dose. At low-to-moderate doses it mainly hits beta-1 receptors in the heart to speed the rate and strengthen contractions. At higher doses it also squeezes blood vessels (alpha effect), raising blood pressure. It is an alternative to epinephrine when atropine fails.

1. At 2–10 micrograms per kilogram per minute, dopamine mostly binds beta-1 receptors in the heart.
2. This raises cyclic AMP, opens calcium channels, and speeds pacemaker firing.
3. Heart rate and contractility both increase.
4. At doses above 10 micrograms per kilogram per minute, alpha receptors in vessel walls are also activated, squeezing arteries and raising blood pressure.

SAFETY WATCH

Titrate carefully; too much causes a fast heart rate (tachycardia) (dangerously fast heart rate), chest pain, or arrhythmias. · At high doses, intense vasoconstriction can harm fingers, toes, kidneys, and gut—watch urine output and extremity color. · Extravasation (leaking into tissue around the IV) causes severe skin damage; use a central line if possible. · This is a temporary bridge to pacing, not a permanent fix.

Glucagon · Hormone (bypasses beta-blocker or calcium-channel-blocker poisoning)

Glucagon works through its own receptor (not the blocked beta receptor) to raise cyclic AMP inside heart cells. This speeds the SA node and strengthens contraction even when beta-blockers or calcium-channel blockers are on board. It is the antidote for overdose of those drugs when they cause severe a slow heart rate (bradycardia).

1. Glucagon binds to its own receptor on heart cells (the glucagon receptor, not the beta receptor).
2. This activates adenylyl cyclase by a different route, raising cyclic AMP.
3. More cyclic AMP opens calcium channels and increases the pacemaker current.
4. Heart rate and contractility rise even though beta receptors are blocked.

SAFETY WATCH

Give 3–5 mg IV bolus, then start an infusion (2–5 mg per hour) because glucagon is cleared quickly. · Common side effects: nausea and vomiting (have suction ready if the patient is drowsy). · Watch blood sugar—glucagon can raise it (it is also used to treat low blood sugar in diabetics). · This is a bridge; arrange pacing and supportive care while the overdose clears.

Calcium chloride or calcium gluconate · Electrolyte replacement / antidote (reverses calcium-channel-blocker poisoning)

Calcium-channel blockers work by blocking calcium from entering heart and vessel cells. Giving IV calcium floods the area with so much calcium that some gets through despite the blockade, helping the heart contract and conduct signals again. It is the first antidote for calcium-channel-blocker overdose.

1. Calcium-channel blockers prevent calcium from entering cells through L-type calcium channels.
2. IV calcium raises the calcium level in the blood and interstitial fluid.

3. The huge concentration gradient pushes calcium through even blocked channels, or through other pathways.
4. Contractility, heart rate, and AV conduction improve.

SAFETY WATCH

Give calcium chloride 1 gram IV (10 mL of 10% solution) or calcium gluconate 3 grams IV (30 mL of 10% solution) over 5–10 minutes; calcium chloride delivers more elemental calcium per dose. · Repeat every 10–20 minutes if needed, guided by heart rate and blood pressure. · DO NOT give calcium through a small peripheral IV at full speed—it can burn the vein; use a large IV or central line. · Watch the ECG—too much calcium in a patient on digoxin can trigger dangerous arrhythmias. · Monitor ionized calcium levels if you give many doses.

Transcutaneous pacing · External electrical pacing (not a drug—delivers electrical pulses through skin pads to pace the heart)

Large sticky pads on the chest and back deliver electrical shocks strong enough to pass through skin and chest wall and capture the heart muscle, making it contract. This is the fastest way to pace when atropine fails or the block is infranodal (below the AV node). It buys time while you arrange transvenous pacing.

1. Pads are placed on the chest (usually front and back for best current path).
2. The pacer sends an electrical pulse (usually 50–200 milliamps) through the chest.
3. The pulse depolarizes enough heart muscle to trigger a contraction.
4. Each pulse produces a QRS on the monitor and (ideally) a pulse you can feel.

SAFETY WATCH

Sedate or give pain medicine if the patient is awake—pacing hurts. · Confirm mechanical capture by feeling a pulse; do not rely on the monitor alone. · Watch the skin under the pads for burns if pacing lasts more than 30 minutes. · Move to transvenous pacing as soon as possible; transcutaneous pacing is NOT a long-term solution.

Transvenous pacing · Internal temporary pacing (not a drug—a wire is threaded through a vein into the right ventricle to pace the heart directly)

A pacing wire is inserted through a central vein (internal jugular, subclavian, or femoral) and advanced under fluoroscopy or echo guidance until the tip sits against the inside wall of the right ventricle. Electrical pulses sent down the wire depolarize the ventricle directly, guaranteeing a heartbeat. This is more reliable and comfortable than transcutaneous pacing

and is used when a slow heart rate (bradycardia) persists despite drugs or transcutaneous pacing.

1. A sheath is placed in a central vein, and the pacing wire is threaded through it.
2. The wire tip is positioned in the right ventricle (confirmed by chest X-ray or fluoroscopy).
3. Electrical pulses (usually 2–10 milliamps) are sent down the wire to depolarize the ventricle.
4. Each pulse triggers a ventricular contraction, maintaining heart rate and cardiac output.

SAFETY WATCH

Check wire position on chest X-ray after placement. · Monitor for loss of capture (pacer spikes without QRS complexes) or sensing problems. · Watch for infection at the insertion site (change dressing daily, follow sterile technique). · Watch for pneumothorax (collapsed lung) if a subclavian or internal-jugular approach was used. · Secure the wire carefully—if it dislodges, the patient loses pacing. · Plan for permanent pacemaker placement if the indication is not reversible.

Permanent pacemaker · Implanted device (not a drug—a pulse generator is placed under the skin with leads screwed or wedged into the heart)

A small computer (pulse generator) is implanted under the skin, usually below the collarbone. Insulated wires (leads) run through veins into the right atrium and/or right ventricle. The device senses the heart's own electrical activity and delivers a pacing pulse whenever the natural rate drops below a programmed threshold. This provides a permanent, reliable backup pacemaker.

1. The device monitors the heart's intrinsic electrical signals through the leads.
2. If no atrial or ventricular signal is detected within the programmed interval, the device sends a small electrical pulse down the lead.
3. The pulse depolarizes the chamber, triggering a contraction.
4. Modern devices adjust pacing rate based on activity (rate-responsive pacing) to mimic normal heart-rate changes with exercise.

SAFETY WATCH

Lifelong follow-up is required (in-person or remote monitoring) to check battery, lead function, and pacing thresholds. · Avoid strong magnets and certain medical equipment (older MRI machines, electrocautery during surgery—modern devices are often MRI-conditional). · Watch the incision site for infection in the first weeks after implant (redness, warmth, drainage). · Patients should carry a pacemaker ID card and inform all doctors and dentists. · If infection occurs around the device, the entire system (generator and leads) usually must be removed, not just treated with antibiotics.

Red flags

- Fainting or blackout spells
- Chest pain or pressure
- Severe shortness of breath at rest
- Sudden confusion or slurred speech
- Heart rate below 40 beats per minute with symptoms

Nursing priorities

1. Place patient on continuous cardiac monitor and pulse oximetry, and obtain a 12-lead ECG immediately. — The ECG shows whether the slow rate comes from the SA node (natural pacemaker), an AV block (traffic jam between upper and lower heart chambers), or another rhythm problem. Each type needs different treatment. Continuous monitoring catches sudden pauses or further slowing.
2. Establish large-bore IV access in at least one arm and draw blood for troponin and electrolytes. — IV access allows immediate delivery of atropine (a drug that speeds up the heart) or other emergency medications. Troponin (a heart enzyme) reveals if a heart attack is causing the a slow heart rate (bradycardia). Low potassium or high potassium can worsen electrical conduction.
3. Assess mental status, skin color, and capillary refill every 5 minutes and document findings. — Confusion, pale or gray skin, and slow capillary refill (longer than 2 seconds) mean the brain and tissues are not getting enough oxygen. These signs tell us the a slow heart rate (bradycardia) is unstable and the patient needs atropine or pacing now.
4. Keep atropine 1 mg syringes at bedside and apply transcutaneous pacing pads to chest and back if heart rate is below 50 with symptoms. — Symptomatic a slow heart rate (bradycardia) can deteriorate to cardiac arrest within minutes. Having atropine ready and pacing pads already attached means we can start life-saving treatment in seconds without delay if the patient worsens.

5. Hold scheduled beta-blockers, calcium channel blockers, and digoxin doses, and notify the provider immediately. — These medications intentionally slow the heart. Giving another dose when the patient already has symptomatic a slow heart rate (bradycardia) can drop the rate even lower and cause cardiac arrest. The provider may need to stop these drugs completely.
6. Position patient flat or in Trendelenburg (head down) if blood pressure is dropping or patient feels faint. — Lowering the head increases venous return (blood flowing back to the heart) and improves cerebral blood flow (perfusion) (blood flow to the brain). This buys time while we prepare atropine or pacing and prevents falls from fainting (syncope) (fainting).
7. Prepare for transvenous pacemaker insertion if transcutaneous pacing is needed for more than 30 minutes. — Transcutaneous pacing (external electrical pacing through skin pads) is uncomfortable and only a bridge treatment. A transvenous pacemaker (temporary wire threaded through a vein into the heart) provides more reliable pacing and comfort while we determine if the patient needs a permanent pacemaker.
8. Reassure patient and explain each intervention in simple terms before performing it. — Anxiety activates the sympathetic nervous system (fight-or-flight response) which can raise heart rate naturally and improve symptoms temporarily. Fear and confusion increase oxygen demand when the heart is already struggling to deliver enough blood.

Sources

- AHA/ACC/HRS 2018 Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay
- American College of Cardiology
- American Heart Association
- European Society of Cardiology 2021 Pacing Guidelines

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