

Supraventricular Tachycardia

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THE STORY IN ONE SENTENCE

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“Supraventricular a fast heart rate (tachycardia) (SVT) is when the heart beats too fast—often 150 to 250 times per minute—because electricity spins in a circle above the lower chambers instead of flowing straight down. Try a vagal maneuver (like bearing down or splashing cold water on the face) to break the loop. If that fails, give adenosine (a drug that briefly pauses the electrical system so the loop stops). If the patient is unstable (passing out, chest pain, very low blood pressure), shock the heart right away with cardioversion. If SVT keeps coming back, use catheter ablation (burn the extra pathway so the loop cannot spin anymore).”

Normal anatomy — the body's job

The heart's electrical system starts in the sinoatrial (SA) node (the natural pacemaker at the top of the right atrium), sends a signal to the atrioventricular (AV) node (the gatekeeper that slows the signal so the atria can squeeze first), then travels down the bundle of His and spreads through the ventricles so they pump in one coordinated beat. This cycle happens 60 to 100 times per minute, and the AV node keeps perfect time.

BODY'S JOB

The SA node fires a signal that makes the atria squeeze and push blood into the ventricles. The AV node delays the signal just enough so the ventricles have time to fill. Then the signal shoots down the bundle of His and into the ventricles, making them pump blood to the lungs and body. This keeps oxygen and nutrients flowing to every organ.

1. SA node (natural pacemaker in the right atrium) fires a signal 60 to 100 times per minute.
2. Signal spreads across both atria, making them squeeze and push blood into the ventricles below.

3. Signal reaches the AV node (the gatekeeper between atria and ventricles), which slows it down so the ventricles have time to fill with blood.
4. Signal travels down the bundle of His (a bundle of electrical fibers running down the wall between the ventricles).
5. Bundle of His splits into the left and right bundle branches, sending the signal to the left and right ventricles.
6. Signal spreads through the Purkinje fibers (fine threads that reach every part of the ventricle muscle).
7. Both ventricles squeeze in one strong, coordinated pump, sending blood to the lungs (right ventricle) and the rest of the body (left ventricle).
8. Cycle repeats, keeping oxygen and nutrients flowing steadily to the brain, kidneys, muscles, and every cell.

What breaks

An extra electrical pathway forms, or a loop develops in or near the AV node, so electricity spins in a circle instead of flowing straight down. The heart races 150 to 250 times per minute, and the ventricles do not have time to fill with blood between beats, so each pump is weak and blood pressure drops.

CLINICAL TERM

Supraventricular a fast heart rate (tachycardia) (SVT) due to re-entry circuits: most commonly atrioventricular nodal reentrant a fast heart rate (AVNRT, where electricity loops within the AV node itself using dual pathways), atrioventricular reentrant a fast heart rate (AVRT, where an accessory pathway like the bundle of Kent in Wolff-Parkinson-White syndrome creates a loop between atrium and ventricle), or atrial a fast heart rate (a rapid focus firing from the atrium). The re-entry loop causes sudden-onset a fast heart rate at 150–250 bpm, reducing ventricular diastolic filling time, decreasing stroke volume and cardiac output, and causing palpitations, lightheadedness, trouble breathing (dyspnea), and sometimes fainting (syncope).

How the body compensates

The body tries to keep blood pressure and oxygen delivery steady by releasing adrenaline (epinephrine), squeezing blood vessels, and making you breathe faster. Vital signs can look okay for minutes to hours while the body is working overtime, but if the heart keeps racing, the

compensations run out and blood pressure crashes. The danger is missing the slow slide toward decompensation before it tips into shock or fainting (syncope).

1. {'what': 'The sympathetic nervous system (the body's fight-or-flight system) releases adrenaline (epinephrine) and noradrenaline (norepinephrine) to squeeze blood vessels and keep blood pressure from dropping too far.', 'why': 'When cardiac output falls because the ventricles are not filling, blood pressure sensors in the aorta and carotid arteries (baroreceptors) tell the brain to activate the sympathetic nervous system. Adrenaline makes blood vessels tighten (vasoconstriction) so blood pressure stays up even though each heartbeat is weak. This buys time but makes you feel jittery, sweaty, and anxious.'}
2. {'what': 'Breathing rate increases (tachypnea) to pull in more oxygen and blow off carbon dioxide.', 'why': 'The brain and body are not getting enough oxygen because cardiac output is low. Chemoreceptors (sensors for oxygen and carbon dioxide in the blood) signal the brainstem to speed up breathing. This compensates for low oxygen delivery, but it makes you feel short of breath (dyspnea) and sometimes anxious or panicky.'}
3. {'what': 'The kidneys hold onto salt and water (via the renin-angiotensin-aldosterone system, or RAAS) to increase blood volume and keep blood pressure up.', 'why': 'When the kidneys sense low blood flow, they release renin, which triggers a chain that makes aldosterone (a hormone that tells the kidneys to keep salt and water). More volume in the blood vessels keeps pressure from dropping. This works over hours to days, so it is not a factor in acute SVT but matters if episodes are frequent or prolonged.'}
4. {'what': 'Dizziness, lightheadedness, and near-fainting (syncope) (almost passing out) are warning signs that the brain is not getting enough blood or oxygen.', 'why': 'The brain uses 20 percent of the body's oxygen even though it is only 2 percent of body weight. When cardiac output drops, the brain is one of the first organs to complain. Lightheadedness and near-fainting (syncope) mean compensation is failing and you are approaching the crash point.'}
5. {'what': 'Frank fainting (syncope) (passing out) means blood pressure has dropped so low that the brain shuts down non-essential functions to protect itself.', 'why': 'If SVT continues and compensation fails, blood pressure falls below the level the brain needs to stay conscious (usually below 60–70 mmHg systolic). You lose consciousness and fall, which makes you lie flat, so gravity helps blood reach the brain again and you wake up after a few seconds. Fainting (Syncope) during SVT means the patient is hemodynamically unstable and needs immediate cardioversion (electrical shock) to restore normal rhythm.'}
6. {'what': 'Chest pain or pressure (angina) can occur if the heart muscle itself is not getting enough oxygen.', 'why': 'The heart muscle (myocardium) needs oxygen from the coronary arteries. When the heart races at 200+ bpm, it uses more oxygen but has less time to fill the coronary arteries (which fill during diastole, the resting phase). If demand outstrips supply, the heart muscle becomes blood-starved (ischemic) (starved of oxygen), causing chest pain. This is more common in older adults or anyone with coronary artery disease.'}

7. {'what': 'Watch the trend, not just the snapshot: a patient can have normal or near-normal vital signs (blood pressure 100–110 systolic, oxygen saturation 95–98 percent) while compensation is working, but if heart rate stays high and symptoms worsen (more dizzy, more short of breath, new chest pain), the patient is sliding toward decompensation and you must act before the crash.', 'why': 'Compensation masks the true severity. A stable blood pressure does not mean the patient is stable if the body is burning through reserves to maintain it. Once compensation fails, vital signs collapse rapidly—blood pressure drops, oxygen saturation falls, and the patient loses consciousness or develops ventricular arrhythmias. Treat based on the trajectory (getting worse) and symptoms (unstable features like chest pain, severe trouble breathing (dyspnea), altered mental status, low blood pressure (hypotension)), not just one set of numbers.'}

Domino effect

1. A re-entry loop or ectopic atrial focus fires 150 to 250 electrical signals per minute, far faster than the SA node's normal 60 to 100 bpm. — In AVNRT, electricity spins in a circle inside the AV node using two pathways. In AVRT, electricity loops through the AV node and an accessory pathway (like the bundle of Kent in WPW). In atrial a fast heart rate (tachycardia), a rogue cluster of atrial cells fires rapidly on its own. Any of these breaks the normal rhythm and hijacks the heart's pacemaker.
2. The ventricles beat 150 to 250 times per minute but do not have time to fill with blood between beats (shortened diastole), so each contraction pumps only a small amount of blood (reduced stroke volume). — The ventricles fill during diastole, the resting phase between beats. When the heart races, diastole is cut short—there is no time for blood to flow in from the atria. Even though the heart is squeezing often, each squeeze is weak because the chamber is only half full. Total cardiac output (stroke volume × heart rate) may stay near normal early on because rate is high, but as rate climbs above 200 bpm, stroke volume drops so much that cardiac output falls.
3. Cardiac output drops, so blood pressure starts to fall and the brain, kidneys, and muscles receive less oxygen and nutrients. — Cardiac output is the amount of blood the heart pumps per minute. When stroke volume falls, cardiac output falls. Blood pressure depends on cardiac output and how tightly blood vessels are squeezed (vascular resistance). If cardiac output drops, blood pressure drops unless the body compensates by squeezing vessels tighter.

4. You feel your heart pounding (palpitations), get dizzy or lightheaded, feel short of breath (dyspnea), and may have chest discomfort or anxiety. — Palpitations happen because you can feel the rapid, forceful beats—the heart is working overtime and each beat may be felt in your chest or neck. Dizziness and lightheadedness occur because the brain is not getting enough oxygen-rich blood. Trouble breathing (Dyspnea) happens because low cardiac output triggers chemoreceptors (oxygen sensors) to make you breathe faster. Chest discomfort can occur if the heart muscle itself is not getting enough oxygen (ischemia) because the coronary arteries do not have time to fill. Anxiety is the brain's alarm response to low oxygen and surging adrenaline.
5. The sympathetic nervous system releases adrenaline and noradrenaline to squeeze blood vessels (vasoconstriction) and keep blood pressure from dropping too far, but this makes you sweaty, jittery, and more anxious. — Baroreceptors in the aorta and carotid arteries detect falling blood pressure and signal the brainstem to activate the sympathetic nervous system. Adrenaline constricts blood vessels (raising vascular resistance to defend blood pressure) and increases heart contractility (making each beat stronger). This buys time but adds to the sensation of pounding heart, tremor, and panic.
6. If SVT continues, compensation fails: blood pressure drops below the level needed to keep the brain awake, you lose consciousness (syncope), or the heart develops a dangerous rhythm like ventricular a fast heart rate (tachycardia) or ventricular fibrillation (especially in WPW with atrial fibrillation (an irregular, quivering heartbeat)). — The body can only compensate for so long. Once blood pressure falls below about 60–70 mmHg systolic, the brain does not get enough blood flow (perfusion) pressure and shuts down non-essential functions, causing fainting (syncope). In WPW, if atrial fibrillation (an irregular, quivering heartbeat) develops (a chaotic atrial rhythm with 400–600 signals per minute) and someone gives an AV-blocking drug (adenosine, beta-blocker, calcium channel blocker, or digoxin), the AV node is blocked but the accessory pathway is not—so 300+ signals per minute shoot straight into the ventricles, triggering ventricular fibrillation (a quivering, non-pumping rhythm) and cardiac arrest. This is why you must always check for WPW (short PR interval and delta wave on ECG) before giving AV-blocking drugs.

7. Treatment breaks the re-entry loop or slows the atrial focus: vagal maneuvers, adenosine, or cardioversion stop the a fast heart rate (tachycardia) and restore normal sinus rhythm, so the ventricles have time to fill again, cardiac output recovers, blood pressure normalizes, and symptoms resolve. — Vagal maneuvers (bearing down, breath-holding, ice water on the face, carotid sinus massage in certain patients) activate the vagus nerve, which releases acetylcholine—a neurotransmitter that slows conduction through the AV node and can break the loop. Adenosine briefly blocks the AV node (pausing conduction for 1–2 seconds), which interrupts the re-entry circuit in AVNRT and AVRT and often stops atrial a fast heart rate (tachycardia) by unmasking it. Cardioversion (synchronized electrical shock) depolarizes the entire heart at once, stopping all electrical activity so the SA node can take over again. Once normal rhythm is restored, diastole lengthens, stroke volume increases, cardiac output and blood pressure normalize, and all symptoms resolve. If SVT recurs frequently, catheter ablation (burning the extra pathway or re-entry circuit with radiofrequency energy or cryotherapy) cures the problem in more than 95 percent of cases.
8. Trace every symptom backward: pounding heart, dizziness, shortness of breath, chest pain, and fainting all point to one upstream break—a re-entry loop or ectopic focus making the heart race so fast that the ventricles cannot fill. Fix the loop, fix the problem. — None of the symptoms are separate diseases. They are all downstream effects of one electrical problem. Palpitations = you feel the racing heart. Dizziness = brain is low on oxygen. Trouble breathing (Dyspnea) = body is trying to get more oxygen. Chest pain = heart muscle is blood-starved (ischemic). Fainting (Syncope) = blood pressure too low for brain to stay awake. The job is to stop the abnormal rhythm (break the loop with vagal maneuver, adenosine, or cardioversion) and, if it keeps coming back, eliminate the substrate (ablate the accessory pathway or re-entry circuit). Treat the cause, not just the symptoms.

Symptoms · what the patient feels, and why

- Heart suddenly racing very fast (150–250 beats per minute): An extra electrical loop or pathway in the heart starts firing on its own, making the top chambers (atria) beat way faster than normal and dragging the whole heart along.
- Heart beating feels regular (like a drumroll at steady speed): The runaway electrical signal travels the same loop over and over in perfect rhythm, so each heartbeat is evenly spaced even though it is much too fast.
- Pounding or fluttering feeling in the chest or neck: The top chambers of the heart are squeezing so fast that you can feel the rapid pulse thumping against your chest wall and the big veins in your neck.
- Feeling dizzy or lightheaded: When the heart races this fast, it does not have enough time to fill with blood between beats, so less blood (and oxygen) reaches the brain.

- **Shortness of breath:** The heart is pumping so fast that it cannot fill properly, which means less oxygen-rich blood gets out to the body and lungs, making you feel like you cannot catch your breath.
- **Chest discomfort or pressure:** The heart muscle itself needs oxygen, and when it is beating this fast it uses more oxygen than the blood supply can deliver, causing an achy or tight feeling.
- **Feeling anxious or panicky:** The amygdala (the brain's alarm center) senses the sudden pounding heart and low oxygen, which triggers the release of adrenaline and sets off a fear response even if there is no real danger.
- **Sudden start and sudden stop of the fast heartbeat:** SVT flips on when the extra electrical loop locks into place and flips off when something breaks the loop (like a vagal maneuver or medication), so the racing can switch on and off like a light.
- **Fainting or nearly fainting:** If the heart is beating so fast that almost no blood reaches the brain, the brain shuts down non-essential functions to protect itself, and you pass out.
- **Severe crushing chest pain:** Very fast heart rate can starve the heart muscle of oxygen (ischemia), which feels like a heavy weight crushing the chest and may signal the heart is in real danger.

Focused exam

- **Heart rate over 150 beats per minute, perfectly regular rhythm on the monitor:** Supraventricular a fast heart rate (tachycardia) (a runaway fast rhythm that starts above the heart's lower pumping chambers) locks into a steady, rapid beat because electricity is caught in a loop. This regular drumbeat, much faster than normal, is SVT's signature — it does not jump around like atrial fibrillation (an irregular, quivering heartbeat).
- **Narrow QRS complex on EKG (less than 0.12 seconds wide):** A narrow QRS tells you the signal travels down the normal highway from the top chambers through the AV node (the gate between upper and lower chambers) to the bottom chambers. Because the electricity is using the normal fast route, the squeeze happens quickly and the spike on the EKG stays narrow. SVT always starts above the AV node, so the QRS stays narrow.
- **Patient reports sudden start of pounding heartbeat and lightheadedness:** SVT flips on like a light switch — the re-entry loop (electricity spinning in a circle instead of traveling straight) starts instantly, so the heart suddenly races. When the heart beats 170 or more times a minute, it does not have time to fill with blood between beats, so less blood reaches the brain. That causes the dizziness. The sudden start and the dizzy feeling together point to a short circuit above the lower chambers.

- Delta wave visible on baseline EKG (a slurred upstroke at the start of the QRS): A delta wave is a clue that the person has Wolff-Parkinson-White syndrome — an extra wire of muscle that lets electricity skip the AV node and sneak into the lower chambers early. This bypass tract can create a dangerous re-entry loop. Spotting the delta wave tells you which kind of SVT you are dealing with and warns you NOT to use certain drugs like adenosine or diltiazem in atrial fibrillation (an irregular, quivering heartbeat) with WPW because they can speed up the bypass and cause a life-threatening rhythm.
- Blood pressure lower than the patient's normal, skin cool and sweaty: When the heart races too fast, it pumps less blood with each beat because there is no time to fill. Blood pressure drops, and the body shunts blood away from the skin to protect the brain and heart. Cool, clammy skin means the body is in trouble — this is unstable SVT and needs immediate treatment.
- P wave buried inside the QRS or appearing right after it: In most SVT, the top chambers (atria) and bottom chambers (ventricles) fire almost at the same time because electricity is looping back and forth. The P wave (the atrial squeeze signal) gets hidden or shows up just after the QRS. This merged pattern proves the circuit is spinning — it is the EKG footprint of re-entry.

Diagnostics · every test answers a question

- 12-lead EKG during the fast rhythm → Is the QRS narrow or wide, is the rhythm regular, and can I see P waves?
- Baseline (resting) 12-lead EKG when the rhythm is normal → Is there a delta wave or short PR interval that hints at an accessory pathway?
- Serum electrolytes (sodium, potassium, magnesium, calcium) → Are the salts that control heart electricity in the normal range?
- Thyroid-stimulating hormone (TSH) → Is an overactive thyroid revving up the heart?
- Troponin I or T → Did the fast heart rate starve the heart muscle of oxygen and damage it?
- Echocardiogram (ultrasound of the heart) → Is the heart's structure normal, and did repeated SVT weaken the pump?

Treatment logic

- Vagal maneuvers: modified Valsalva (blow into a syringe for 15 seconds, then lie flat with legs raised for 15 seconds) or carotid massage in stable patients → Pressing the vagus nerve (the body's main brake cable) releases acetylcholine (a calming chemical) that slows the AV node for a few seconds. That pause interrupts the re-entry loop — the spinning electricity stops, and the heart resets to normal rhythm. The modified Valsalva boosts success to about 43 percent versus 17 percent for standard strain.

- Adenosine 6 mg rapid IV push, flush immediately; if no response in 1–2 minutes, give 12 mg; may repeat 12 mg once more → Adenosine (a molecule that floods adenosine receptors on AV node cells) blocks the AV node completely for a few seconds, snapping the re-entry circuit like flipping a breaker. The heart resets and normal rhythm returns in about 90 percent of AVNRT and AVRT cases. The block is so brief the patient may feel a scary pause, but it is safe.
- IV diltiazem (0.25 mg/kg over 2 minutes, then infusion) or metoprolol (2.5–5 mg IV every 5 minutes up to 15 mg) for rate control in recurrent stable SVT → Calcium-channel blockers like diltiazem and beta-blockers like metoprolol slow conduction through the AV node by calming the cells that fire the signals. This makes it harder for the re-entry loop to stay spinning and keeps the heart rate under 100 even if SVT tries to start again. Use these for ongoing control after adenosine or when adenosine fails and the patient is still stable.
- Synchronized cardioversion starting at 50–100 joules for unstable patients (low blood pressure, chest pain, shortness of breath, altered mental status) → An electric shock timed to the R wave (the peak of the heartbeat) resets every heart cell at the same instant, wiping out the re-entry loop like rebooting a computer. Normal rhythm comes back immediately in nearly 100 percent of cases. This is the fastest way to restore blood flow when the patient cannot wait for drugs.
- Procainamide 15 mg/kg IV over 30–60 minutes for atrial fibrillation (an irregular, quivering heartbeat) in a patient with known or suspected WPW → Procainamide (a sodium-channel blocker) slows conduction through both the AV node and the bypass tract. It prevents dangerously fast ventricular rates when atrial fibrillation (an irregular, quivering heartbeat) tries to race down the extra pathway. It can also convert atrial fibrillation back to normal rhythm.
- Oral beta-blocker (metoprolol, atenolol) or calcium-channel blocker (diltiazem, verapamil) for daily prevention in patients with frequent symptoms → Taking these pills every day keeps the AV node sluggish, so re-entry circuits cannot get going as easily. Symptoms become less frequent and shorter. This buys time until the patient can have ablation or serves as long-term control if they decline the procedure.
- Catheter ablation (radiofrequency or cryotherapy) for patients with recurrent symptomatic SVT or any WPW → A thin wire threaded into the heart burns or freezes the re-entry pathway or accessory tract, permanently destroying the short circuit. Success rates are 95–98 percent for AVNRT and AVRT. After ablation, the patient is cured — no more SVT, no more daily pills.

Medications

Adenosine · Antiarrhythmic (nucleoside)

Adenosine is a natural body chemical that puts a temporary brake on the AV node (the electrical relay station between the top and bottom chambers of the heart). It does this by opening special potassium channels and closing calcium channels in the AV node cells. This creates a brief pause in electrical signals traveling through the node—usually just a few seconds—which breaks the re-entry loop (the circle of electricity spinning around and around) that causes most SVT. Think of it like hitting the reset button on a circuit that's stuck in a loop.

1. Adenosine binds to A1 receptors on AV node cells
2. This opens potassium channels, letting potassium rush out of the cells
3. It also blocks calcium channels, stopping calcium from entering
4. These changes make the AV node cells temporarily unable to pass electrical signals
5. The brief block interrupts the re-entry circuit, stopping the a fast heart rate (tachycardia)
6. Adenosine is broken down by the body in seconds, so the effect lasts only 10–15 seconds

SAFETY WATCH

Common: brief chest tightness, flushing, shortness of breath, or a feeling of impending doom lasting 10–20 seconds as the drug works—warn the patient beforehand so they know it's temporary · Transient asystole (heart pause) for a few seconds is expected and not harmful in most cases · Bronchospasm (airway tightening) in patients with asthma or COPD—use with caution or avoid · Can trigger atrial fibrillation (an irregular, quivering heartbeat) in some patients, especially older adults · Reduce dose to 3 mg initial in patients on dipyridamole or with central line access; increase dose in patients on theophylline or caffeine · Give as rapid IV push (1–2 seconds) followed immediately by saline flush, or it breaks down before reaching the heart

Metoprolol · Beta-blocker (cardioselective)

Metoprolol blocks beta-1 receptors in the heart—the receptors that adrenaline (epinephrine) and norepinephrine normally activate to speed things up. By blocking these receptors, metoprolol slows the heart rate and slows conduction (electrical signal travel) through the AV node. This makes it harder for a re-entry circuit to sustain itself. Think of it like lowering the speed limit on the electrical highway through the heart, so the loop can't keep spinning fast enough to cause a fast heart rate (tachycardia).

1. Metoprolol binds to beta-1 adrenergic receptors in the heart
2. This blocks the stimulating effects of adrenaline and norepinephrine
3. AV node conduction slows, making it harder for rapid electrical loops to complete

4. Heart rate decreases, both during SVT and at rest
5. Over time, this reduces the likelihood of SVT episodes starting or sustaining

SAFETY WATCH

Avoid in decompensated heart failure, severe a slow heart rate (bradycardia) (heart rate under 50), or high-degree AV block without a pacemaker · Can worsen asthma or COPD by blocking beta-2 receptors in the lungs at higher doses · Monitor blood pressure—can cause low blood pressure (hypotension), especially with IV dosing · Fatigue, dizziness, and cold extremities are common · Do NOT stop abruptly in long-term users—can cause rebound a fast heart rate (tachycardia) or chest pain · Use caution in patients with diabetes—can mask low blood sugar (hypoglycemia) warning signs like fast heart rate

Diltiazem · Calcium channel blocker (non-dihydropyridine)

Diltiazem blocks L-type calcium channels in the heart, especially in the AV node. Calcium normally rushes into heart cells to help them fire electrical signals and contract. By blocking calcium entry, diltiazem slows conduction through the AV node and lengthens the refractory period (the time a cell needs to rest before it can fire again). This makes it harder for a re-entry circuit to keep looping. It also slows the heart rate overall.

1. Diltiazem binds to and blocks L-type calcium channels in AV node cells
2. This prevents calcium from entering the cells during each electrical cycle
3. AV node conduction slows, and the refractory period lengthens
4. Re-entry circuits lose the fast pathway they need to sustain a fast heart rate (tachycardia)
5. Heart rate decreases, both during episodes and at baseline

SAFETY WATCH

Avoid in patients with accessory pathways (WPW) who have atrial fibrillation (an irregular, quivering heartbeat) or flutter—can be life-threatening · Avoid in decompensated heart failure, severe a slow heart rate (bradycardia), or high-degree AV block without a pacemaker · Monitor blood pressure—IV diltiazem can cause low blood pressure (hypotension) · Constipation is common with oral use · Dizziness, headache, and ankle swelling can occur · Use caution with beta-blockers—combined use increases risk of severe a slow heart rate (bradycardia) or heart block

Procainamide · Antiarrhythmic (Class Ia sodium channel blocker)

Procainamide blocks sodium channels in heart muscle cells, which slows the upstroke of the action potential (the rapid electrical signal that makes a heart cell fire). It also prolongs repolarization (the recovery phase) by blocking potassium channels. This combination slows

conduction and increases refractoriness in both the atria and the ventricles, AND in accessory pathways like the bundle of Kent in WPW. By slowing conduction in the accessory pathway, it prevents dangerously fast signals from reaching the ventricles when atrial fibrillation (an irregular, quivering heartbeat) or flutter is present.

1. Procainamide binds to sodium channels in heart cells, blocking them
2. This slows the speed of electrical signals traveling through the atria, ventricles, and accessory pathways
3. It also blocks potassium channels, prolonging the refractory period
4. Accessory pathway conduction slows, preventing rapid ventricular rates in atrial fibrillation (an irregular, quivering heartbeat) with WPW
5. Re-entry circuits lose the fast conduction they need, terminating or preventing a fast heart rate (tachycardia)

SAFETY WATCH

QT prolongation (lengthening of the heart's electrical recovery phase)—can trigger torsades de pointes, a dangerous ventricular arrhythmia; monitor EKG during infusion · Low blood pressure (Hypotension), especially with rapid IV infusion—give slowly · Avoid in patients with prolonged QT at baseline, severe heart failure, or myasthenia gravis · Long-term use can cause drug-induced lupus (joint pain, rash, fever)—rare with short-term use · Nausea, dizziness, and a metallic taste during infusion · Monitor drug levels if used for more than a few doses

Verapamil · Calcium channel blocker (non-dihydropyridine)

Verapamil works just like diltiazem—it blocks L-type calcium channels in the AV node, slowing conduction and lengthening the refractory period. This makes it harder for re-entry circuits to sustain themselves. Verapamil also causes some vasodilation (widening of blood vessels), which can lower blood pressure.

1. Verapamil binds to and blocks L-type calcium channels in AV node cells
2. Calcium entry into cells decreases, slowing the speed of electrical signals
3. AV node conduction slows, and the refractory period lengthens
4. Re-entry loops lose the fast pathway needed to sustain a fast heart rate (tachycardia)
5. Heart rate decreases during SVT and at rest

SAFETY WATCH

Avoid in accessory pathway tachycardias (WPW) with atrial fibrillation (an irregular, quivering heartbeat) or flutter—can be lethal · Avoid in decompensated heart failure, severe a slow heart rate (bradycardia), or AV block · Low blood pressure (Hypotension), especially with IV use · Constipation is very common with oral therapy · Dizziness, headache, and peripheral swelling (edema) (ankle swelling) · Do NOT combine with IV beta-blockers—can cause severe a slow heart rate (bradycardia) or asystole

Amiodarone · Antiarrhythmic (Class III, multi-channel blocker)

Amiodarone blocks multiple ion channels—mainly potassium channels (which prolongs repolarization and refractoriness), but also sodium channels, calcium channels, and beta receptors. This broad action slows conduction and increases refractoriness in the atria, ventricles, AV node, and accessory pathways. It stabilizes the heart's electrical system across the board, making re-entry circuits harder to sustain.

1. Amiodarone primarily blocks potassium channels, prolonging the action potential and refractory period
2. It also blocks sodium and calcium channels, slowing conduction
3. It has mild beta-blocking effects, slowing heart rate
4. All of these actions make it harder for re-entry circuits to complete their loop
5. Heart rhythm stabilizes, and recurrence risk decreases

SAFETY WATCH

Long-term use: pulmonary fibrosis (lung scarring), thyroid dysfunction (hyper- or hypothyroidism), liver toxicity, corneal deposits (usually harmless but can blur vision), and blue-gray skin discoloration · Short-term IV use: low blood pressure (hypotension), a slow heart rate (bradycardia), and phlebitis (vein inflammation) at the infusion site · QT prolongation—monitor EKG · Photosensitivity—advise patients to use sunscreen and avoid intense sun · Drug interactions: amiodarone increases levels of warfarin, digoxin, and many other drugs—dose adjustments needed · Reserved for refractory cases or when other agents fail, due to toxicity profile

Esmolol · Beta-blocker (ultra-short-acting, cardioselective)

Esmolol works like metoprolol—it blocks beta-1 receptors in the heart, slowing AV node conduction and heart rate. The key difference is that esmolol has a half-life of only 9 minutes, so its effects wear off quickly if you stop the infusion. This makes it ideal for short-term rate

control in unstable or critically ill patients, where you want tight control without long-lasting effects.

1. Esmolol binds to beta-1 adrenergic receptors in the heart
2. It blocks adrenaline and norepinephrine from speeding up the heart
3. AV node conduction slows, heart rate decreases
4. Re-entry circuits slow or terminate due to prolonged AV node refractoriness
5. Effects reverse within minutes of stopping the infusion

SAFETY WATCH

Low blood pressure (Hypotension)—especially at higher doses; often given with continuous blood pressure monitoring · A slow heart rate (Bradycardia)—can cause severe slowing or heart block · Avoid in decompensated heart failure, severe a slow heart rate (bradycardia), or high-degree AV block · Can worsen bronchospasm in asthma or COPD · Short half-life means effects reverse quickly if the patient decompensates—just stop the drip

Red flags

- Fainting or nearly fainting during the fast heartbeat
- Crushing chest pain that does not go away
- Severe shortness of breath or gasping for air
- Blood pressure drops very low (shock)
- Signs of heart failure (swelling in legs, fluid in lungs, extreme fatigue)

Nursing priorities

1. Place patient on continuous cardiac monitor and obtain 12-lead EKG within 2 minutes while assessing blood pressure and mental status — The EKG shows if QRS complexes (main heart waves) are narrow confirming SVT versus wide suggesting ventricular a fast heart rate (tachycardia); unstable vital signs mean the heart cannot pump enough oxygen to the brain and body
2. Establish IV access with normal saline lock in large vein before giving any medicines — Adenosine (the first medicine for SVT) must be pushed very fast into a big vein close to the heart to work; delayed IV access wastes time when rhythm is dangerous

3. Prepare crash cart with defibrillator pads on patient and synchronized cardioversion mode ready if patient shows chest pain, systolic blood pressure below 90, altered mental status, or severe shortness of breath — Unstable patients need immediate electrical cardioversion (synchronized shock) at 50 to 100 joules because their heart is failing to pump; having equipment ready prevents delay and brain damage from low oxygen
4. Coach stable patient through modified Valsalva maneuver: blow into 10-mL syringe for 15 seconds while sitting up, then immediately lay flat with legs raised for 15 seconds — The modified Valsalva increases pressure in the chest which stimulates the vagus nerve (a nerve that slows heart rate); the REVERT trial showed this method works in 43 percent of SVT patients versus 17 percent with standard Valsalva
5. Give adenosine 6 mg rapid IV push over 1 to 2 seconds followed immediately by 20-mL normal saline fast flush if vagal maneuvers fail; warn patient they will feel chest tightness and doom for 10 seconds — Adenosine blocks the AV node (the electrical gateway between upper and lower heart chambers) for a few seconds to break the re-entry circuit; the flush pushes medicine to the heart before it breaks down in 10 seconds
6. Have second dose of adenosine 12 mg drawn up and ready to give 2 minutes after first dose if rhythm does not convert — First adenosine dose fails in about half of patients because the medicine breaks down fast or the dose is too low for the patient's body weight; second higher dose succeeds in most remaining cases
7. Monitor for atrial fibrillation (an irregular, quivering heartbeat) (irregular rhythm) with wide QRS after adenosine in any patient; do not give AV nodal blockers like diltiazem if this appears — Wide irregular rhythm after adenosine may reveal WPW with atrial fibrillation (an irregular, quivering heartbeat); AV nodal blocking medicines (drugs that slow the AV node like metoprolol or diltiazem) can cause ventricular fibrillation (deadly chaotic rhythm) in WPW by forcing impulses down the extra pathway
8. Document pre-treatment and post-treatment rhythm strips showing at least 6 seconds, heart rate, blood pressure response, and time to cardioversion — Rhythm strips prove SVT diagnosis for cardiology follow-up and ablation planning; documentation of unstable signs justifies emergency cardioversion; conversion time guides future treatment choices

Sources

- AHA/ACC/HRS Guideline for the Management of Patients with Supraventricular Tachycardia 2015
- American College of Cardiology
- American Heart Association
- Heart Rhythm Society

Last reviewed: 2026-07-07