

Supraventricular Tachycardia

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).

THE ONE-LINE TRUTH

An electrical signal spins in a loop above the ventricles, making the heart race uncontrollably.

The chain — how it unfolds

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. — why: 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). — why: 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. — why: 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. — why: 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. — why: 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)). — why: 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. — why: 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. — why: 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.

Spot it — what you see, 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.

Red flags — act now

- 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)

Treat it — in this order

1. 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.
2. 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.
3. 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.

4. 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.
5. 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.

Your top priorities at the bedside

1. Place patient on continuous cardiac monitor and obtain 12-lead EKG within 2 minutes while assessing blood pressure and mental status — why: 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 bod
2. Establish IV access with normal saline lock in large vein before giving any medicines — why: 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 — why: 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