

MALDEK

PATIENT TEACHING · MALDEK CONDITION CATALOG

Supraventricular Tachycardia

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart has four chambers: two upper rooms called atria and two lower pumps called ventricles. Normally, electricity starts at the top of the right atrium in a spot called the sinoatrial (SA) node—the heart's natural pacemaker. That signal travels down to the atrioventricular (AV) node, a gatekeeper that slows the signal just enough so the atria can squeeze and fill the ventricles with blood before the ventricles pump it out to the body. The signal then shoots down the bundle of His (a highway of electrical fibers) and spreads through the ventricles, making them squeeze in one strong, coordinated beat. In a healthy heart, this cycle happens 60 to 100 times per minute, and the AV node keeps perfect time. In supraventricular a fast heart rate (tachycardia), an extra electrical pathway or a loop forms somewhere above the ventricles—often involving the AV node itself or an accessory pathway like the one in Wolff-Parkinson-White (WPW) syndrome. Electricity starts spinning around that loop instead of flowing straight down, so the atria fire signals 150 to 250 times per minute. The heart races so fast that the ventricles do not have time to fill with blood between beats, so each pump is weak and less blood reaches the brain and body. You feel your heart pounding (palpitations), you get dizzy or lightheaded because your brain is not getting enough oxygen, you feel short of breath because your lungs sense low oxygen, and sometimes you pass out (syncope) if blood pressure drops too much. Your body is not broken everywhere—these symptoms are alarm signals trying to tell you one thing is wrong: an electrical loop is hijacking the heart's rhythm. The job is to stop that loop. A vagal maneuver (bearing down like you are having a bowel movement, holding your breath and pushing, or dunking your face in ice water) can shock the vagus nerve into hitting the brakes on the AV node and breaking the loop. If that does not work, adenosine (a drug that pauses the AV node for a second or two) stops the loop in most cases. If the patient is unstable—passing out, chest pain, very low blood pressure—cardioversion (a controlled electrical shock) resets the heart immediately. If SVT keeps coming back, catheter ablation burns the extra pathway so the loop cannot spin anymore, and most people are cured. One danger to know: if someone has WPW (an extra pathway that conducts very fast) and develops atrial fibrillation (an irregular, quivering heartbeat) (a chaotic rhythm in the atria), never give drugs that block the AV node (like adenosine, beta-blockers, or calcium channel blockers) because the signal will race down the accessory pathway unchecked and can trigger ventricular fibrillation (a deadly rhythm). Always trace the symptoms backward—pounding heart, dizziness, and shortness of breath all point to one upstream break: a re-entry loop spinning too fast for the heart to do its job.

What you might feel

- Heart suddenly racing very fast (150–250 beats per minute)
- Heart beating feels regular (like a drumroll at steady speed)

- Pounding or fluttering feeling in the chest or neck
- Feeling dizzy or lightheaded
- Shortness of breath
- Chest discomfort or pressure
- Feeling anxious or panicky
- Sudden start and sudden stop of the fast heartbeat
- Fainting or nearly fainting
- Severe crushing chest pain

Call the doctor right away if...

- 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)

What the care team will do

- Place patient on continuous cardiac monitor and obtain 12-lead EKG within 2 minutes while assessing blood pressure and mental status
- Establish IV access with normal saline lock in large vein before giving any medicines
- 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
- 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
- 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
- Have second dose of adenosine 12 mg drawn up and ready to give 2 minutes after first dose if rhythm does not convert
- 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
- Document pre-treatment and post-treatment rhythm strips showing at least 6 seconds, heart rate, blood pressure response, and time to cardioversion

Medicines you may be given — and why

Adenosine

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.

Metoprolol

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).

Diltiazem

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.

Procainamide

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.

Verapamil

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.

Amiodarone

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.

Esmolol

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.

Where this information comes from

- 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