

Unstable tachyarrhythmia

A plain-English guide for you and your family

WHAT IS HAPPENING

Your heart has a natural pacemaker called the sinus node that sets the beat based on what your body needs moment to moment. Normally, the heart squeezes, then relaxes and fills with blood, then squeezes again in a steady rhythm. The top chambers (atria) squeeze first and give the bottom chambers (ventricles) a final 20 percent push of blood right before they squeeze. Then the bottom chambers pump that blood out to your whole body. In unstable tachyarrhythmia, something has hijacked that rhythm. Either a different spot in the heart is firing electrical signals faster than the sinus node, or electricity is spinning around in a loop (reentry circuit), or the connection between the top and bottom chambers is letting signals through too fast. Whatever the cause, the heart is now beating so fast—often 150 to 250 times per minute—that it cannot relax long enough to fill with blood. So even though the heart is squeezing hard and fast, almost no blood is getting pumped out with each beat. Blood pressure drops, and the brain, kidneys, and other organs start to starve. Your body tries to fix this by releasing adrenaline (catecholamines) to make the heart pump harder, but that only makes the heart beat even faster and worsens the problem. The heart muscle itself also suffers, because it gets its own oxygen supply only during the relaxation phase—so a heart beating 200 times a minute with almost no rest time is starving itself. This is why people with unstable tachyarrhythmia can have chest pain, confusion, difficulty breathing, and dangerously low blood pressure even if their heart arteries are wide open. The treatment is to stop the broken rhythm—either with electricity (cardioversion) or certain medicines—so the sinus node can take control again and the heart can fill, rest, and pump normally.

What you might feel

- Low blood pressure (systolic below 90)
- Confusion, trouble speaking, or acting strangely
- Chest pain or pressure
- Shortness of breath or rapid breathing
- Very fast pulse (usually over 150 beats per minute at rest)
- Feeling dizzy or about to faint (presyncope)

- Patient may look pale, sweaty, and anxious

Call the doctor right away if...

- Systolic blood pressure below 90 mmHg
- Altered mental status (confused, not responding normally, or unresponsive)
- Chest pain or signs of heart attack (acute heart attack (myocardial infarction))
- Signs of heart failure (fluid in the lungs causing extreme shortness of breath, inability to lie flat)
- Loss of consciousness or about to pass out

What the care team will do

- Place the patient on continuous cardiac monitoring and pulse oximetry, and obtain a 12-lead ECG within 1 minute.
- Establish large-bore IV access in two sites immediately.
- Obtain a manual blood pressure every 2 to 5 minutes and assess mental status continuously.
- Prepare the defibrillator in synchronized mode with pads on the chest and sedation medication drawn up at the bedside.
- Administer high-flow oxygen by non-rebreather mask to keep oxygen saturation above 94 percent.
- Draw blood for troponin, electrolytes including potassium and magnesium, and thyroid studies before giving any medications.
- Attempt vagal maneuvers like bearing down or ice to the face only if the patient is stable with narrow-complex regular a fast heart rate (tachycardia).
- Document the exact time symptoms started, heart rate trends, and any interventions with the patient response every 2 to 5 minutes.

Medicines you may be given — and why

Synchronized cardioversion (electrical procedure, not a drug)

A controlled shock is delivered through pads on the chest, timed to land during the heart's safe moment (the thick part of the heartbeat wave on the monitor, called the R wave). This timing prevents the shock from landing during the heart's vulnerable window (the T wave) when a jolt could flip a fast rhythm into ventricular fibrillation (a chaotic, deadly quiver). The shock briefly stops all electrical activity in the heart, giving the sinoatrial node (the heart's natural pacemaker in the upper-right chamber) a chance to take charge again and restore a normal, organized beat.

Adenosine

Adenosine is a natural chemical that the heart uses to slow down electrical traffic. When given as a medicine, it floods adenosine receptors on cells in the atrioventricular (AV) node (the electrical gate between the upper and lower heart chambers). This makes the gate slam shut for a few seconds, blocking extra signals from getting through. If the fast rhythm is caused by a spinning electrical loop that depends on the AV node, breaking the loop stops the a fast heart rate (tachycardia) instantly. If the rhythm is coming from a different source (like atrial flutter or atrial fibrillation (an irregular, quivering heartbeat)), adenosine will slow the heart just long enough to unmask the underlying pattern on the monitor, which helps the team figure out the diagnosis.

Diltiazem

Diltiazem blocks calcium channels on cells in the AV node (the gate between the upper and lower heart chambers) and in the sinoatrial node (the heart's natural pacemaker). Calcium normally rushes into these cells to help them fire electrical signals. When diltiazem blocks the calcium gates, the cells fire more slowly and conduct impulses less eagerly. In atrial fibrillation (an irregular, quivering heartbeat) or atrial flutter with a rapid ventricular response, the AV node is letting too many chaotic signals through from the atria to the ventricles, making the heart race. Diltiazem slows the AV node down so fewer impulses get through, which drops the heart rate and gives the ventricles time to fill with blood, improving circulation and symptoms.

Metoprolol (or other beta-blocker such as esmolol)

Metoprolol blocks beta-1 receptors on cells in the AV node (the electrical gate between the upper and lower chambers) and the sinoatrial node (the heart's natural pacemaker). Normally, adrenaline and noradrenaline (the body's stress hormones) lock onto these receptors and rev up the heart—making it beat faster and conduct signals more eagerly. Metoprolol sits in those receptor parking spots so adrenaline cannot, which slows down the AV node's willingness to pass signals from the atria to the ventricles. In atrial fibrillation (an irregular, quivering heartbeat) or atrial flutter with rapid ventricular response, this means fewer of the chaotic atrial impulses get through, the ventricles slow down, and the heart has time to fill with blood and pump effectively again.

Amiodarone

Amiodarone works on multiple electrical switches in heart muscle cells, making it a broad-spectrum rhythm stabilizer. Its main job is to block potassium channels that help cells reset after each beat. When potassium cannot leave the cell as easily, the recharging phase (repolarization) takes longer. This lengthens the time before the cell can fire again (the refractory period), which breaks up fast spinning loops of electricity (reentry circuits) and quiets cells that are firing on their own. Amiodarone also gently blocks sodium channels

(slowing the upstroke of the electrical signal), calcium channels (calming the AV node), and beta-receptors (reducing adrenaline's push). In stable wide-complex a fast heart rate (tachycardia) that is likely ventricular a fast heart rate, amiodarone slows and stabilizes the chaotic firing in the ventricles, often restoring normal rhythm. It can also be used in atrial fibrillation (an irregular, quivering heartbeat) to help convert the rhythm or prevent it from returning.

Procainamide

Procainamide blocks sodium channels in heart muscle cells, which slows down the speed at which an electrical signal travels through the tissue (conduction velocity). It also blocks potassium channels, which lengthens the time cells need to recharge before they can fire again (the refractory period). Together, these actions help break up reentry circuits (electrical loops spinning too fast) and quiet down cells that are firing extra beats on their own. In stable wide-complex a fast heart rate (tachycardia) that is likely ventricular a fast heart rate, procainamide slows the chaotic electrical activity in the ventricles and often restores normal rhythm. It can also be used in atrial fibrillation (an irregular, quivering heartbeat), especially when there is an accessory pathway (pre-excitation), because it slows conduction through both the normal pathway (AV node) and the extra pathway.

Immediate unsynchronized defibrillation (for pulseless ventricular a fast heart rate (tachycardia) or ventricular fibrillation)

When the heart is in ventricular fibrillation (a chaotic quiver with no organized beat) or pulseless ventricular a fast heart rate (tachycardia) (a very fast rhythm in the lower chambers that produces no blood flow), there is no time to synchronize the shock—the patient has no pulse and is clinically dead. An unsynchronized shock delivers maximum energy immediately, without waiting for any particular part of the heartbeat. The goal is to jolt every heart cell at once, briefly stopping all electrical activity (chaotic and organized alike), so the sinoatrial node (the heart's natural pacemaker) has a chance to take over and restore a normal, life-sustaining rhythm. Defibrillation is the single most important treatment for ventricular fibrillation and pulseless ventricular a fast heart rate; every minute of delay drops survival by about 10%.

Where this information comes from

- AHA/ACC 2023 ACLS guideline
- ESC 2019 guideline for the management of patients with supraventricular tachycardia
- AHA/ACC/HRS 2015 guideline for the management of adult patients with supraventricular tachycardia
- Cochrane review on emergency treatment of tachyarrhythmias

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