

Atrial Fibrillation or Flutter with RVR

A plain-English guide for you and your family

WHAT IS HAPPENING

Normally your heart beats in two stages. First, the atria (the two small top chambers) squeeze and push a final splash of blood down into the ventricles (the two big bottom chambers). Then the ventricles squeeze hard and pump that blood out to your lungs and body. A tiny relay station called the AV node sits between the top and bottom and acts like a gatekeeper, letting signals through one at a time so the rhythm stays steady. In atrial fibrillation (an irregular, quivering heartbeat), many spots all over the atria start firing electrical sparks at random—like popcorn popping everywhere at once. The atria quiver instead of squeezing. In atrial flutter, one electrical signal races around and around the atria in a fast circle, like a car on a racetrack. Either way, the atria cannot squeeze, so that final splash of blood never makes it into the ventricles. The AV node tries to block most of the chaos, but hundreds of signals are hitting it every minute and some still get through. The ventricles start beating fast and often irregularly (in AFib) or fast and regularly (in flutter). Because the atria are quivering and not squeezing, the ventricles do not fill up as much as they should, so each heartbeat pumps out less blood. If the ventricles race too fast for too long, the heart muscle gets tired and weak. Blood also sits still and pools in the quivering atria, and when blood sits still it can clot—like a puddle freezing. If a clot breaks off and travels up to the brain, it causes a stroke. Your body tries to keep blood pressure steady by releasing adrenaline, which makes your heart beat even faster and work even harder, but eventually the heart cannot keep up. Every symptom you feel—your heart pounding in your chest, feeling short of breath, being tired all the time, feeling dizzy or faint—traces back to one broken thing: the atria lost their squeeze.

What you might feel

- Palpitations (feeling your heart race or skip)
- Shortness of breath
- Fatigue and getting tired easily during activity
- Chest discomfort or pain
- Lightheadedness or feeling faint
- Fainting (passing out)

- Irregular pulse when checked
- Swelling in legs, neck veins bulging, or crackling sounds in lungs
- Low blood pressure
- Sudden weakness, trouble speaking, or vision loss (stroke symptoms)

Call the doctor right away if...

- Fainting or very low blood pressure (means the heart is barely pumping enough to keep you alive)
- Sudden severe shortness of breath with fluid in the lungs (flash pulmonary edema (fluid flooding the lungs)—heart failure emergency)
- Chest pain with EKG changes showing the heart muscle is starving for oxygen or dying (heart attack)
- Sudden one-sided weakness, slurred speech, or vision loss (stroke from a clot that broke loose)
- Known WPW syndrome (an extra electrical pathway in the heart) with new irregular rhythm (certain drugs can trigger a deadly rhythm called ventricular fibrillation)

What the care team will do

- Place the patient on continuous cardiac monitoring and pulse oximetry, and obtain a 12-lead EKG within 5 minutes.
- Establish large-bore IV access and check blood pressure every 5 to 15 minutes.
- Administer supplemental oxygen to keep oxygen saturation above 90 percent.
- Draw blood for troponin, BNP (a heart-failure hormone), thyroid studies, and electrolytes including magnesium and potassium.
- Prepare for synchronized cardioversion by keeping the patient NPO (nothing by mouth), placing defibrillator pads on the chest, and having sedation medicine ready at the bedside.
- Administer IV rate-control medicine such as metoprolol or diltiazem as ordered, and recheck heart rate and blood pressure 5 minutes after each dose.
- Assess for signs of stroke every 15 to 30 minutes by checking facial symmetry, arm strength, and speech clarity.
- Keep the patient calm and in a semi-upright position, and explain each intervention before performing it.

Medicines you may be given — and why

Atrial Fibrillation (an irregular, quivering heartbeat) and Atrial Flutter Management Algorithm

This is a step-wise approach, not a single drug. (1) If the patient is unstable (hypotension—low blood pressure, chest pain, severe shortness of breath, altered mental status), perform synchronized cardioversion (electrical shock to restore normal rhythm) immediately: 50 joules for atrial flutter or 120–200 joules biphasic for atrial fibrillation (an irregular, quivering heartbeat). (2) If the patient is stable, control the heart rate (ventricular response) with IV diltiazem (a calcium-channel blocker that slows electrical conduction through the AV node—the gate between the atria and ventricles), IV metoprolol (a beta-blocker that blocks adrenaline's effect on the heart's conduction system), or digoxin if the patient has heart failure with reduced ejection fraction (HFrEF—weak pump). Magnesium may be used for refractory cases (not responding to standard therapy). (3) For rhythm control (restoring and maintaining normal sinus rhythm), perform cardioversion if the patient is symptomatic despite rate control, the arrhythmia is new-onset (< 48 hours), or the patient has been anticoagulated for ≥ 3 weeks or has a negative transesophageal echocardiogram ruling out left atrial a blood clot (thrombus) (clot). Pharmacologic options include amiodarone or flecainide. (4) Anticoagulate (prevent stroke from atrial clots) based on CHA₂DS₂-VASc score (tool that estimates stroke risk); direct oral anticoagulants (DOACs—newer blood thinners like apixaban, rivaroxaban, edoxaban, dabigatran) are preferred over warfarin in non-valvular atrial fibrillation per AHA/ACC/HRS guidelines. (5) Address reversible causes and risk factors: excessive alcohol, obstructive sleep pauses in breathing (apnea), obesity, uncontrolled high blood pressure (hypertension), hyperthyroidism, and structural heart disease. Each drug targets a specific step in the pathophysiology: calcium-channel blockers and beta-blockers slow AV nodal conduction; antiarrhythmics (amiodarone, flecainide) prolong refractoriness (the recovery period before a cell can fire again) in atrial tissue and disrupt reentrant circuits (electrical loops); anticoagulants block the clotting cascade to prevent a blood clot formation in the fibrillating or fluttering atrium.

Where this information comes from

- AHA/ACC/HRS 2023 Guideline for the Diagnosis and Management of Atrial Fibrillation
- ESC 2020 Atrial Fibrillation Guidelines
- CHEST 2018 Antithrombotic Therapy for Atrial Fibrillation
- UpToDate

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