

# Atrial Fibrillation or Flutter with RVR

The atria lose their organized squeeze and either fire all at once in chaos (AFib) or race around one fast loop (flutter). You must answer three urgent questions: How do I slow the heart? Do I shock it back to normal rhythm? How do I prevent a stroke?

## THE ONE-LINE TRUTH

The top chambers of the heart (atria) either quiver like a bag of worms (atrial fibrillation) or spin in a very fast circle (atrial flutter), so they cannot squeeze blood down into the bottom chambers anymore.

## The chain — how it unfolds

1. The atria lose organized contraction. — why: In atrial fibrillation (an irregular, quivering heartbeat), multiple ectopic foci (abnormal pacemaker spots) scattered throughout the atrial tissue fire electrical impulses rapidly and randomly—often 400 to 600 times per minute. The SA node is overwhelmed and the atria quiver chaotically instead of squeezing as one unit. In atrial flutter, a single macroreentrant circuit (a looping electrical pathway, usually in the right atrium) spins around and around at a fixed high rate, typically 250 to 350 beats per minute, causing the atria to flutter rapidly but in an organized pattern. Either way, the atrial muscle does not contract effectively.
2. The atrial kick is lost. — why: Normally, atrial contraction at the end of diastole (the filling phase) pushes a final 15 to 30 percent of blood into the ventricles—this is called the atrial kick. When the atria quiver or flutter, they cannot squeeze, so this final splash of blood never makes it into the ventricles. Ventricular preload (the volume of blood in the ventricles before they contract) drops, especially in patients with stiff ventricles (diastolic dysfunction, left ventricular hypertrophy) or mitral narrowing (stenosis) (narrowed mitral valve), who depend heavily on atrial kick for adequate filling.
3. Stroke volume decreases. — why: Because the ventricles are not filled as completely, each ventricular contraction pumps out less blood. Stroke volume (the amount of blood ejected per beat, normally about 70 mL per beat) falls. Cardiac output (stroke volume × heart rate, normally about 5 liters per minute) drops unless the heart rate increases to compensate.

4. The ventricles beat rapidly and irregularly (AFib) or rapidly and regularly (flutter). — why: Hundreds of chaotic atrial impulses bombard the AV node every minute. The AV node filters them and conducts only some impulses to the ventricles. In AFib, the conduction is random, creating an irregularly irregular ventricular rhythm (no pattern at all, beat-to-beat intervals vary). In flutter, the AV node often conducts in a fixed ratio—commonly 2:1 (every other atrial impulse gets through), 3:1, or 4:1—creating a rapid but regular ventricular rhythm. For example, if atrial flutter rate is 300 bpm and AV conduction is 2:1, ventricular rate is 150 bpm. Either way, the fast ventricular rate shortens diastole, giving the ventricles even less time to fill and reducing stroke volume further.
5. Sustained a fast heart rate (tachycardia) weakens the heart (tachycardia-induced cardiomyopathy). — why: When the heart beats too fast for too long—usually sustained ventricular rates above 100 to 120 beats per minute for weeks to months—the heart muscle does not get enough time to rest, relax, and recover between beats. The myocardium (heart muscle) becomes fatigued, the ventricles dilate (stretch out), and ejection fraction (the percentage of blood pumped out of the left ventricle with each beat, normally 55 to 70 percent) falls. This is called a fast heart rate (tachycardia)-induced cardiomyopathy. The good news is it is often reversible if you control the rate or restore normal rhythm in time.
6. Blood stasis in the atria increases clot risk. — why: Blood that sits still tends to clot (this is called Virchow's triad: stasis, endothelial injury, hypercoagulability). In the quivering atria, especially in a small blind pouch called the left atrial appendage (which has low flow even in normal rhythm), blood pools and can form a blood clot (thrombus) (clot). If the clot breaks off (embolizes), it travels through the left ventricle, out the aorta, and up to the brain, where it blocks a cerebral artery and causes an blood-starved (ischemic) stroke (brain tissue dies from lack of oxygen). The annual stroke risk in untreated AFib ranges from about 2 percent (low risk) to over 10 percent (high risk), depending on other factors captured in the CHA<sub>2</sub>DS<sub>2</sub>-VASc score (Congestive heart failure, High blood pressure (Hypertension), Age ≥75 [2 points], Diabetes, prior Stroke/TIA [2 points], Vascular disease, Age 65–74, Sex category [female]).
7. The body compensates by increasing sympathetic tone. — why: Baroreceptors (pressure sensors) in the carotid arteries and aortic arch detect falling cardiac output and send signals to the brainstem. The brainstem activates the sympathetic nervous system, which releases adrenaline (epinephrine and norepinephrine). Adrenaline increases heart rate, contractility (how hard the heart squeezes), and vascular tone (blood vessels tighten), temporarily maintaining blood pressure and organ blood flow (perfusion). But this compensation burns energy and stresses the heart, and it cannot last forever.

- 8.** Compensation fails and symptoms appear. — why: When the heart can no longer keep up, cardiac output falls further and organs do not get enough blood. The patient feels fatigued (muscles and brain are underperfused). Shortness of breath (dyspnea) develops because pressure backs up into the lungs, causing pulmonary congestion or pulmonary edema (fluid flooding the lungs) (fluid leaks into the air sacs). The patient may feel palpitations (awareness of the fast or irregular heartbeat). Chest pain can occur if the coronary arteries cannot supply enough oxygen to the racing heart (myocardial starved blood flow (ischemia)). Dizziness, lightheadedness, or fainting (syncope) (fainting) happen when blood pressure drops and the brain does not get enough flow. Some patients have no symptoms and AFib is discovered only on routine exam or EKG—this is especially common in elderly patients or those with chronic AFib.

## Spot it — what you see, and why

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- Palpitations (feeling your heart race or skip): The bottom chambers (ventricles) squeeze too fast—sometimes in a jumbled pattern (fibrillation) or a super-fast regular pattern (flutter)—and you feel the pounding, fluttering, or skipping in your chest because your heart is racing out of control.
- Shortness of breath: The heart pumps less blood forward with each beat, so blood backs up into the lung's blood vessels (like a traffic jam). This makes it harder for oxygen to move from your lungs into your blood, and you feel like you can't catch your breath.
- Fatigue and getting tired easily during activity: When each heartbeat is weak and your heart rate is chaotic, less oxygen-rich blood reaches your muscles and brain. Your body runs low on fuel, so you tire out quickly even with light effort.
- Chest discomfort or pain: The heart is beating so fast that the resting phase (when the heart muscle gets its own blood supply) is too short. If the heart muscle doesn't get enough oxygen (ischemia), it hurts—like a cramp in any muscle that isn't getting the fuel it needs.
- Lightheadedness or feeling faint: When the heart pumps poorly, less blood reaches your brain. Your brain is very sensitive to low oxygen, so you feel dizzy or woozy, especially when you stand up or move around.
- Fainting (passing out): If the heart rate gets dangerously fast or the heart pumps almost no blood, your brain shuts down briefly from lack of oxygen and you lose consciousness—like a computer rebooting when the power dips too low.

## Red flags — act now

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- 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)

## **Treat it — in this order**

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1. Synchronized cardioversion: 50 to 100 joules biphasic shock for atrial flutter, 120 to 200 joules biphasic for atrial fibrillation (an irregular, quivering heartbeat) → An electrical shock is delivered through paddles or pads on the chest, timed to fire during the QRS complex (the moment the ventricles contract) to avoid triggering a dangerous ventricular arrhythmia. The shock depolarizes (resets) all the heart cells at once, stopping the chaotic signals in the atria or the flutter circuit. The heart's natural pacemaker (SA node) can then take over and restore normal sinus rhythm, where the atria contract in sync with the ventricles. Atrial kick returns, filling improves, stroke volume rises, and cardiac output increases immediately. Blood pressure comes up, and symptoms resolve within seconds to minutes.
2. IV metoprolol (2.5 to 5 mg bolus, repeat every 5 minutes up to 15 mg) or IV diltiazem (0.25 mg/kg bolus, then 5 to 15 mg/hour infusion) → These drugs slow conduction through the AV node (the electrical gate between the atria and ventricles). Metoprolol is a beta-blocker—it blocks adrenaline receptors, calming the node. Diltiazem is a calcium channel blocker—it blocks calcium from entering the node's cells, which also slows conduction. Both increase the AV node's refractory period (the time it cannot pass another signal). Fewer signals get through per minute, so the ventricular rate drops from 150 or 160 down to less than 110. Slower rate gives the ventricles more time to fill during diastole, stroke volume improves, and cardiac output stabilizes. Blood pressure comes up, and symptoms improve within minutes.

3. IV amiodarone (150 mg bolus over 10 minutes, then 1 mg/min infusion for 6 hours, then 0.5 mg/min) or IV ibutilide (1 mg over 10 minutes, repeat once if needed) for atrial flutter → Amiodarone is a class III antiarrhythmic drug—it blocks potassium channels in the heart cells, prolonging the action potential (the electrical wave inside each cell) and the refractory period (the recovery time before the cell can fire again). This suppresses abnormal firing sites in the atria and interrupts reentry circuits (electrical loops that sustain the arrhythmia). Over several hours, amiodarone can chemically convert the rhythm back to normal sinus, restoring atrial contraction and optimal cardiac output. Ibutilide works similarly but acts faster and is more effective for atrial flutter; it can convert flutter to sinus rhythm in 30 to 60 minutes.
4. Anticoagulation with a direct oral anticoagulant (DOAC: apixaban 5 mg twice daily, rivaroxaban 20 mg daily, edoxaban 60 mg daily, or dabigatran 150 mg twice daily) or warfarin (target INR 2 to 3) if DOAC contraindicated, based on CHA<sub>2</sub>DS<sub>2</sub>-VASc score → In atrial fibrillation (an irregular, quivering heartbeat) or flutter, the atria do not squeeze, so blood pools and can clot in the left atrial appendage (a small pouch off the left atrium). If a clot breaks loose, it travels to the brain and causes a stroke. Anticoagulants block steps in the clotting cascade (the chain reaction that forms clots), preventing clot formation. The CHA<sub>2</sub>DS<sub>2</sub>-VASc score assigns points for stroke risk factors: congestive heart failure (1 point), high blood pressure (hypertension) (1 point), age 65 to 74 (1 point) or 75 or older (2 points), diabetes (1 point), prior stroke or TIA (2 points), vascular disease (1 point), female sex (1 point). A score of 2 or more in men or 3 or more in women means anticoagulation benefit outweighs bleeding risk per 2023 AHA/ACC/HRS guidelines. Starting anticoagulation reduces stroke risk by about 60 percent.
5. IV digoxin (loading dose 0.5 mg, then 0.25 mg every 6 hours for two doses, then 0.125 to 0.25 mg daily) if heart failure with reduced ejection fraction is present → Digoxin inhibits the sodium-potassium ATPase pump on heart muscle cells. This causes sodium to build up inside the cell, which then trades for calcium. More calcium inside the cell makes each contraction stronger (positive inotropy). Digoxin also stimulates the vagus nerve, increasing parasympathetic tone, which slows AV node conduction and reduces ventricular rate. It is useful in atrial fibrillation (an irregular, quivering heartbeat) with heart failure because it improves the squeeze (contractility) while controlling the rate.

## Your top priorities at the bedside

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1. Place the patient on continuous cardiac monitoring and pulse oximetry, and obtain a 12-lead EKG within 5 minutes. — why: The monitor shows the exact heart rhythm and rate every second so we can see if the patient suddenly gets worse or develops a deadly rhythm like ventricular a fast heart rate (tachycardia), and the EKG confirms whether it is atrial fibrillation (an irregular, quivering heartbeat) or flutter and shows if the heart muscle is starving for oxygen.

2. Establish large-bore IV access and check blood pressure every 5 to 15 minutes. — why: IV access lets us give emergency rate-control medicines or start cardioversion sedation immediately without delay, and frequent blood pressure checks tell us if the rapid rate is causing shock (dangerously low pressure) which means the heart cannot keep up and we need to shock it back to normal rhythm right away.
3. Administer supplemental oxygen to keep oxygen saturation above 90 percent. — why: The fast chaotic atria cannot squeeze blood forward efficiently so less oxygen gets to the lungs and body, and giving extra oxygen helps prevent the heart muscle itself from becoming starved for oxygen which would make everything worse.