

Mitral Regurgitation

Each heartbeat sends some blood backward into the left atrium instead of forward into the body.

THE STORY IN ONE SENTENCE

Each heartbeat sends some blood backward into the left atrium instead of forward into the body.

“The mitral valve lets blood leak backward during every squeeze. The left atrium cannot hold it forever.”

Normal anatomy — the body's job

The mitral valve sits between the left atrium (top chamber) and the left ventricle (bottom chamber). It opens to let blood pour down, then closes to keep blood from flowing backward when the ventricle squeezes.

BODY'S JOB

The mitral valve's job is to be a one-way door. When the ventricle relaxes, the valve swings open so blood from the lungs can fill the ventricle. When the ventricle squeezes, the valve snaps shut so all the blood is pushed forward into the aorta and out to the body. This keeps blood moving in the right direction — from lungs to heart to body.

1. Blood returns from the lungs into the left atrium.
2. The mitral valve opens.
3. Blood pours from the atrium down into the left ventricle.
4. The ventricle squeezes.
5. The mitral valve snaps shut.
6. Blood shoots forward through the aortic valve into the aorta.

What breaks

The mitral valve cannot close all the way, so blood leaks backward into the atrium during every heartbeat instead of going forward to the body.

CLINICAL TERM

Mitral regurgitation (MR) is incomplete closure of the mitral valve during ventricular systole, causing backward flow from the left ventricle into the left atrium.

How the body compensates

The body stretches the atrium and ventricle to hold the extra blood and keep vital signs steady for a while. But this buys time at a cost — the heart eventually gets too big and weak, and the window to fix it closes.

1. {'step': 'In acute severe MR, there is no time to compensate.', 'why': 'The leak is sudden. The left atrium is small and stiff, so pressure shoots up immediately, fluid floods the lungs, and the forward flow drops — you see flash pulmonary edema (fluid flooding the lungs) and cardiogenic shock within minutes to hours.'}
2. {'step': 'In chronic MR, the left atrium stretches over weeks to months.', 'why': 'Slow, steady volume overload lets the atrium wall stretch like a balloon. The bigger atrium holds the extra blood at lower pressure, so you do not get lung swelling (edema) right away.'}
3. {'step': 'The left ventricle enlarges and pumps harder.', 'why': 'The ventricle has to pump the normal stroke volume PLUS the blood that leaked back into the atrium. To handle this extra load, the ventricle stretches (eccentric hypertrophy — the chamber gets bigger, and the walls thicken a little). Ejection fraction may look normal or even high at first because the ventricle is ejecting into two easy exits — the aorta and the low-pressure atrium.'}
4. {'step': 'Heart rate may rise slightly.', 'why': 'A faster heart rate tries to keep total cardiac output (amount of blood pumped per minute) steady even though each beat sends less blood forward to the body.'}
5. {'step': 'The ventricle eventually weakens and fails.', 'why': 'Pumping the same blood over and over is exhausting. The ventricle muscle stretches too far (past the optimal point on the Frank-Starling curve where muscle fibers generate the most force), loses strength, and ejection fraction drops. Once the ventricle is damaged, even fixing the valve may not restore full function.'}
6. {'step': 'The atrium stretches so much it triggers atrial fibrillation (an irregular, quivering heartbeat).', 'why': 'The stretched atrium walls disrupt normal electrical signals, and the atrium quivers instead of contracting in a coordinated rhythm. This raises stroke risk and worsens heart failure.'}
7. {'step': 'Pulmonary high blood pressure (hypertension) develops.', 'why': 'Chronic high pressure in the left atrium backs up into the pulmonary veins and capillaries. Over time, the lung blood vessels stiffen and narrow (pulmonary vascular remodeling), raising pressure in the right side of the heart and eventually causing right heart failure.'}

8. {'step': 'The danger point in acute severe MR is papillary muscle rupture or torn chordae.', 'why': 'No time to stretch. Pressure spikes, lungs flood, blood pressure drops. This is a surgical emergency — the window to save the patient is hours, not days.'}
9. {'step': 'The other danger point is chronic severe MR with falling ejection fraction or rising left ventricle size.', 'why': 'Once ejection fraction drops below 60% or the left ventricle end-systolic diameter grows past 40 mm, permanent heart damage is happening. Surgery before this point can save ventricle function; waiting too long means the heart stays weak even after the valve is fixed. Current guidelines (AHA/ACC 2020) recommend surgery for severe MR before these thresholds are crossed.'}

Domino effect

1. The mitral valve does not close all the way. — The valve flaps are damaged, floppy, torn, or pulled apart, so a gap remains when the ventricle squeezes.
2. Blood leaks backward into the left atrium during each heartbeat. — When the left ventricle contracts, high pressure inside pushes blood through the easiest path. Some goes forward through the aortic valve to the body, and some squirts backward through the leaky mitral valve into the low-pressure atrium.
3. In acute MR, the small stiff atrium cannot stretch fast, so pressure spikes. — The atrium is suddenly overloaded with extra blood it cannot hold. Pressure inside climbs rapidly and backs up into the pulmonary veins.
4. High atrial pressure floods the lungs with fluid (flash pulmonary edema (fluid flooding the lungs)). — Pressure in the pulmonary veins and capillaries rises above the pressure that holds fluid inside blood vessels, so fluid leaks out into the air sacs (alveoli), making it hard to breathe.
5. Forward cardiac output drops, causing low blood pressure and shock. — Blood leaking backward means less blood is pumped forward to the body with each beat. Organs do not get enough oxygen, blood pressure falls, and the kidneys and brain start to fail.
6. In chronic MR, the left atrium slowly stretches to hold the extra blood. — Over weeks and months, the atrium wall stretches like a balloon. A bigger atrium holds more blood at lower pressure, so lung swelling (edema) does not happen right away and you may feel fine.
7. The left ventricle enlarges to pump the extra volume. — The ventricle has to pump the normal amount PLUS the blood that leaked back. To handle this bigger load, the ventricle stretches (dilates) and its walls thicken slightly (eccentric hypertrophy).
8. Eventually the ventricle stretches too far and weakens. — Pumping the same blood over and over is exhausting. The muscle fibers stretch past the point where they can contract forcefully (beyond the optimal length on the Frank-Starling curve), ejection fraction drops, and the heart cannot keep up — this is heart failure.

Symptoms · what the patient feels, and why

- A blowing sound (murmur) during the heart squeeze, loudest at the top of the heart and spreading toward the left armpit: The mitral valve (the door between the upper and lower left chambers of the heart) doesn't close all the way, so blood leaks backward during each squeeze. That backward jet of blood makes a whooshing noise the stethoscope picks up.
- Feeling short of breath, especially when lying flat or during activity: Because blood leaks backward into the left atrium (the lungs' waiting room), pressure builds up and pushes fluid into the tiny air sacs in the lungs, making it harder to pull in oxygen.
- Feeling very tired and weak, even with light tasks: The heart can't pump enough fresh blood forward to the body because some leaks backward with every beat, so muscles and organs don't get the fuel and oxygen they need to work well.
- Swelling in the ankles, feet, or belly: When the heart struggles and pressure backs up through the veins, fluid is squeezed out into the tissues. Gravity pulls it down, so it pools in the lowest parts of the body.
- A fast or fluttering heartbeat (palpitations): The stretched left atrium (the upper left chamber) can develop chaotic electrical signals called atrial fibrillation (an irregular, quivering heartbeat), or the whole heart may speed up to try to keep blood flow normal despite the leak.
- The point where you feel the heart's strongest tap (PMI, or point of maximal impulse) is shifted down and to the left: Years of pumping extra blood to make up for the leak cause the left ventricle (the heart's main pumping chamber) to stretch and grow bigger, pushing the strongest tap farther out on the chest wall.
- An extra low-pitched heart sound (S3 gallop) right after the usual 'lub-dub': When the left ventricle is stretched and floppy from long-term overload, blood rushing in during filling makes the walls vibrate and create a third thump—like a drum that's too loose to sound crisp.
- Coughing, especially at night or when lying down, sometimes with pink frothy spit: Fluid leaks into the lungs' air sacs because of high pressure backing up from the heart. The body tries to cough it out, and the foam can be tinged pink from tiny blood vessels breaking under pressure.
- Crackles (wet popping sounds) heard through the stethoscope at the bottom of the lungs: Fluid that has leaked into the air sacs pops and crackles when air moves through it during breathing, like bubbles popping in a wet sponge.
- Needing to prop up on pillows to sleep (orthopnea) or waking up gasping for air (paroxysmal nocturnal trouble breathing (dyspnea)): When you lie flat, gravity no longer helps drain fluid from the lungs, so pressure rises fast and makes breathing feel impossible until you sit up.

Focused exam

- Holosystolic murmur at the apex that radiates to the axilla: This swooshing sound lasts through the entire squeeze of the heart (systole) and travels toward the left armpit because blood is jetting backward through the mitral valve into the left atrium. The louder and longer the murmur, the more blood is leaking.
- Severe sudden shortness of breath with pink frothy coughed-up mucus (sputum): When the mitral valve tears or ruptures suddenly (acute MR), the left atrium cannot stretch fast enough to handle the extra blood shooting backward. Pressure spikes backward into the lung veins, forcing fluid into the air sacs — this is flash pulmonary edema (fluid flooding the lungs) and can kill quickly.
- Low blood pressure and cool, clammy skin: In acute severe MR, so much blood leaks backward that not enough moves forward into the body. Blood pressure drops and the body shuts down flow to the skin to protect the brain and heart — this is cardiogenic shock and needs immediate help.
- Displaced apical impulse (the heartbeat you feel shifts down and to the left): In chronic MR, the left ventricle pumps extra blood for months or years and slowly stretches. A big left ventricle pushes the spot where you feel the heartbeat lower and farther left on the chest — this tells you the leak has been there a long time.
- Irregular heart rhythm (atrial fibrillation) on pulse check: Chronic backward leak stretches the left atrium, which scrambles the electrical signals that organize the heartbeat. An irregular pulse means the atrium is probably stretched and scarred, and clots can form inside — stroke risk goes up.

Diagnostics · every test answers a question

- Transthoracic echocardiogram (TTE) → Is the mitral valve leaking, how much blood is going backward, and are the heart chambers getting bigger or weaker?
- Transesophageal echocardiogram (TEE) → What exactly is broken on the valve — torn leaflet, ruptured chord, infected vegetation — and can it be repaired?
- Chest X-ray → Is there fluid backing up into the lungs, and is the heart getting bigger?
- Electrocardiogram (ECG) → Is the left atrium or left ventricle enlarged, and is the heart rhythm still organized?
- Brain natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) → Is the heart under strain and stretching?
- Cardiac catheterization with left ventriculography → Is there other heart disease (blocked coronary arteries), and how severe is the leak when measured directly inside the heart?

Treatment logic

- Vasodilators (sodium nitroprusside or nitroglycerin) plus diuretics (furosemide) in acute severe MR → Vasodilators lower the resistance the left ventricle pumps against (afterload), so more blood moves forward into the aorta and less leaks backward through the broken valve. Diuretics pull fluid out of the lungs by making the kidneys release extra water and salt into the urine, relieving shortness of breath.
- Intra-aortic balloon pump (IABP) in cardiogenic shock from acute severe MR → A balloon in the aorta inflates between heartbeats and deflates just before the heart squeezes. This lowers the pressure the ventricle has to push against, forcing more blood forward into the body and less backward through the leaky valve. Blood pressure and organ blood flow (perfusion) improve.
- Urgent mitral valve repair or replacement surgery → Repairing the leaflets, chords, or annulus (the ring around the valve) restores a tight seal and stops backward leak. If repair is not possible, replacing the valve with a mechanical or tissue prosthesis eliminates the regurgitation. Forward blood flow normalizes, pressure in the left atrium and lungs drops, and symptoms improve rapidly.
- ACE inhibitors or ARBs, beta-blockers, and aldosterone antagonists in chronic secondary MR from heart failure → These medicines shrink an enlarged, failing left ventricle by lowering blood pressure, slowing the heart rate, and blocking hormones (angiotensin, aldosterone) that scar and stretch the muscle. As the ventricle gets smaller, the mitral valve leaflets can meet better in the middle, reducing the leak.
- MitraClip or transcatheter edge-to-edge repair (TEER) in select patients with chronic secondary MR → A catheter threads through a vein to the heart and clips the two mitral leaflets together at their midpoint, creating a smaller opening that leaks less. Backward flow decreases without open-heart surgery. This is an option for patients too sick or frail for traditional surgery.
- Anticoagulation (warfarin or direct oral anticoagulants) if atrial fibrillation (an irregular, quivering heartbeat) is present → Thinning the blood prevents clots from forming in the fibrillating, stretched left atrium. This lowers the risk of stroke, which is very high when blood pools and swirls instead of pumping smoothly.
- Antibiotic prevention (prophylaxis) before dental or invasive procedures after valve surgery → A dose of antibiotics (typically amoxicillin 2 g orally 30–60 minutes before the procedure) prevents bacteria released into the bloodstream from sticking to and infecting the repaired or prosthetic valve.

Medications

Furosemide (Lasix) · Loop diuretic

Furosemide pulls extra water and salt out of the body through urine, which shrinks the blood volume circulating back to the lungs and heart. When the mitral valve leaks backward, blood backs up into the lungs like water pooling behind a dam. By draining off the extra fluid, furosemide lowers the lung pressure and makes breathing easier. It does NOT fix the leaky valve itself.

1. Furosemide blocks a pump in the loop of Henle that normally grabs sodium and chloride and drags them back into the bloodstream.
2. Without that pump working, sodium and chloride stay in the urine tube and pull water along with them by osmosis (water follows salt).
3. The kidneys flush out much more urine, so the total blood volume drops.
4. With less volume, the pressure in the veins feeding the lungs falls, which stops fluid from leaking into the lung air sacs.
5. Breathing gets easier, and leg swelling shrinks because less fluid is backed up in the body.

SAFETY WATCH

Can wash out potassium, magnesium, and calcium — blood tests every few weeks check levels; low potassium can trigger dangerous heart rhythms. · Can make you dizzy or lightheaded when standing up because blood pressure drops. · Kidney function can worsen if the dose is too high or if you get dehydrated — watch creatinine and BUN. · Ringing in the ears or hearing loss can happen at very high IV doses. · Can raise blood sugar and uric acid (gout risk) over time.

Sodium nitroprusside (Nipride) · Arterial and venous vasodilator

Nitroprusside relaxes the smooth muscle wrapped around arteries and veins, which widens the blood vessels and drops blood pressure fast. In acute severe mitral regurgitation (for example, when a heart-attack tears a papillary muscle and the valve suddenly flaps open), the heart is pumping against high resistance and a lot of blood is shooting backward through the leaky valve. By opening up the arteries — especially the big ones leaving the heart — nitroprusside makes it easier for blood to flow forward into the body instead of backward into the lungs. This takes pressure off the lungs within minutes and buys time for emergency surgery.

1. Nitroprusside breaks down inside blood-vessel cells and releases nitric oxide (a gas signal that tells muscle to relax).
2. Nitric oxide turns on an enzyme called guanylate cyclase, which makes a molecule called cyclic GMP.

3. Cyclic GMP tells the smooth muscle fibers around the vessels to loosen up, so the vessel diameter gets bigger.
4. With wider arteries, the resistance (afterload) the left ventricle pumps against drops sharply.
5. More blood flows forward into the body and less squirts backward through the leaky mitral valve.
6. The lungs decompress and oxygen levels climb.

SAFETY WATCH

Blood pressure can fall too far and too fast — must use in an ICU with a continuous arterial line and minute-by-minute monitoring. · The drug releases cyanide as it breaks down; if used longer than 24–48 hours or at high doses, cyanide and thiocyanate can build up and poison cells — watch for confusion, lactic acidosis (acid from oxygen-starved tissues), and a very high anion gap. · Thiocyanate toxicity shows up as confusion, nausea, and ringing in the ears; check levels if used more than a couple of days. · Can raise pressure inside the skull if given to someone with head trauma or brain swelling. · Needs protection from light — the IV bag and tubing are wrapped in foil or opaque covers.

Enalapril (Vasotec) or other ACE inhibitor · Angiotensin-converting enzyme (ACE) inhibitor

Enalapril blocks an enzyme that normally makes angiotensin II (a hormone that squeezes blood vessels tight and tells the kidneys to hold onto salt and water). By stopping that enzyme, enalapril keeps vessels relaxed and blood pressure lower. In chronic secondary mitral regurgitation — where the valve leaks because the heart chamber is stretched and weak, not because the valve itself is torn — ACE inhibitors are part of standard heart-failure treatment. They lower the resistance the damaged heart pumps against and slow the heart's remodeling (the dangerous enlargement and stiffening over time). They do NOT fix the valve or reverse existing scarring.

1. Enalapril sticks to ACE and blocks it from snipping angiotensin I into angiotensin II.
2. With less angiotensin II, blood vessels stay wider and blood pressure drops.
3. The adrenal glands release less aldosterone, so the kidneys dump more salt and water, which shrinks blood volume.
4. The heart pumps against lower resistance (afterload), so more blood flows forward and less leaks backward through the floppy mitral valve.
5. Over months, lower angiotensin II slows the heart chamber from stretching and scarring (remodeling).

SAFETY WATCH

Can raise potassium because less aldosterone means kidneys hold onto potassium — check a basic metabolic panel in one to two weeks, especially if the patient takes spironolactone or has kidney disease. · Can cause a dry, tickling cough in about 10–15 percent of people because blocking ACE raises bradykinin (a chemical that irritates the throat). · Rarely causes angioedema (sudden swelling of the lips, tongue, or throat that can block breathing) — this is an emergency; stop the drug and never restart it. · Kidney function can worsen, especially if renal-artery narrowing (stenosis) (a narrowing in the kidney artery) is present — watch creatinine. · Absolutely must NOT be used in pregnancy — can cause severe birth defects and fetal death.

Carvedilol (Coreg) or metoprolol succinate (Toprol-XL) · Beta blocker (carvedilol also blocks alpha receptors)

Beta blockers slow the heart rate and lower the force of each heartbeat by blocking adrenaline's landing spots (beta receptors) on heart muscle. In chronic secondary mitral regurgitation with heart failure, the heart is overworked and the nervous system is dumping adrenaline constantly to try to keep up. That adrenaline makes the heart race and squeeze hard, which burns energy and stretches the chamber even more. Beta blockers put the brakes on that vicious cycle, giving the heart time to rest and recover. Over months, the heart may shrink a little and pump more efficiently. They do NOT fix the leaky valve itself.

1. The drug sits on beta-1 receptors and blocks adrenaline (epinephrine) and noradrenaline from binding.
2. With less adrenaline signal, the SA node fires slower, so heart rate drops.
3. Each heartbeat squeezes less hard, which lowers the heart's oxygen demand.
4. Over weeks to months, the heart remodels in reverse — the chamber may shrink slightly and pump more efficiently.
5. Carvedilol also blocks alpha receptors on blood vessels, so resistance drops and the heart pushes against less pressure.

SAFETY WATCH

Can make heart failure symptoms worse at first — must start at a tiny dose and go up very slowly every two weeks. · Can drop heart rate too low (bradycardia) or cause heart block if the electrical system is already sluggish — check an EKG before starting. · Can make blood sugar drop and hide the warning signs (shakiness, fast heart rate) in people with diabetes. · Can trigger wheezing or trouble breathing in people with asthma or COPD by blocking beta-2 receptors in the lungs. · Never stop suddenly — rebound high blood pressure and chest pain can happen; taper down slowly. · Can cause fatigue, dizziness, and cold hands and feet because circulation slows.

Spironolactone (Aldactone) · Mineralocorticoid receptor antagonist (potassium-sparing diuretic)

Spironolactone blocks aldosterone (a hormone that makes the kidneys grab sodium and water and dump potassium). In heart failure with secondary mitral regurgitation, aldosterone is cranked up high, which traps fluid and scars the heart muscle over time. By blocking aldosterone's receptor in the kidney, spironolactone makes the kidneys let go of some salt and water (which shrinks swelling and lung congestion) while holding onto potassium. It also stops aldosterone from scarring the heart, which slows the heart's dangerous remodeling. It does NOT fix the leaky valve.

1. Spironolactone sits on the mineralocorticoid receptor and blocks aldosterone from binding.
2. Without aldosterone's signal, the kidney tubule stops pulling sodium back into the blood.
3. Sodium and water flow out in urine, which shrinks blood volume and eases lung congestion.
4. Potassium stays in the blood instead of being dumped in urine, so levels stay steady or rise a little.
5. In the heart, blocking aldosterone slows fibrosis (scarring) and stiffening of the muscle, which helps the heart pump better over time.

SAFETY WATCH

Can raise potassium too high (hyperkalemia), especially if used with an ACE inhibitor, ARB, or in someone with kidney disease — check potassium and creatinine in one week, then every few months. · High potassium can cause dangerous heart rhythms with no warning — stop the drug if potassium climbs above 5.5 mEq/L. · Can cause tender, swollen breast tissue (gynecomastia) in men and irregular periods in women because it weakly blocks testosterone and progesterone receptors. · Kidney function can worsen — watch creatinine. · Avoid potassium supplements and salt substitutes (which are potassium chloride) while taking this drug.

Digoxin (Lanoxin) · Cardiac glycoside

Digoxin makes the heart squeeze a little harder and slows certain fast heart rhythms. It blocks a pump on heart cells called the sodium-potassium ATPase, which raises calcium inside the cell, and calcium triggers a stronger contraction. In chronic mitral regurgitation with heart failure and atrial fibrillation (an irregular, quivering heartbeat) (a chaotic, fast rhythm in the top chambers), digoxin can slow the heart rate and improve symptoms. It does NOT fix the leaky valve, and it is NOT a first-choice drug anymore — beta blockers are usually better — but digoxin is still used in some patients who stay symptomatic despite other medicines.

1. Digoxin sticks to the sodium-potassium ATPase pump and shuts it down partially.
2. Sodium builds up inside the cell, which makes the sodium-calcium exchanger run backward.
3. Calcium piles up inside the cell, and calcium is the trigger for muscle contraction.
4. Each heartbeat squeezes harder, which can push a bit more blood forward instead of backward through the leaky valve.
5. Digoxin also boosts vagal tone (the calming brake nerve to the heart), which slows the AV node and lowers heart rate in atrial fibrillation (an irregular, quivering heartbeat).

SAFETY WATCH

Very narrow safe window — the toxic dose is close to the therapeutic dose. Check a digoxin level if symptoms appear. · Toxicity causes nausea, vomiting, confusion, yellow-green vision halos, and dangerous heart rhythms (especially ventricular a fast heart rate (tachycardia) and heart block). · Low potassium, low magnesium, and high calcium make toxicity much more likely — check electrolytes regularly. · Kidney disease raises digoxin levels because the drug is cleared by the kidneys — use a lower dose and follow levels. · Many drugs (amiodarone, verapamil, quinidine, spironolactone, antibiotics) raise digoxin levels — always check for interactions. · Elderly patients are more sensitive and need lower doses.

Red flags

- Sudden, severe shortness of breath at rest or waking you from sleep (sign of acute heart failure or flash pulmonary edema (fluid flooding the lungs)—fluid flooding the lungs fast)
- Coughing up pink, frothy spit (sign that fluid and blood are filling the lungs under high pressure)
- New chest pain, dizziness, or fainting (may mean the heart can't keep up with the body's needs or dangerous heart rhythms like ventricular a fast heart rate (tachycardia) are starting)

- Rapid weight gain or swelling that gets worse quickly over days (sign that fluid is building up fast and the heart is decompensating)
- Lips or skin turning blue, especially around the mouth or fingertips (sign the body isn't getting enough oxygen)

Nursing priorities

1. Monitor oxygen saturation continuously and apply supplemental oxygen to keep SpO₂ above 92 percent — Backward blood flow into the left atrium raises pressure in the pulmonary veins (lung blood vessels), pushing fluid into the alveoli (air sacs) and preventing oxygen from reaching the bloodstream
2. Position the patient upright at 45 to 90 degrees with legs dangling if tolerated — Sitting upright uses gravity to pool blood in the lower body and reduce venous return (blood coming back to the heart), lowering pulmonary capillary pressure and decreasing lung fluid
3. Establish IV access and prepare to give loop diuretics like furosemide as ordered — Diuretics reduce total blood volume by making kidneys excrete salt and water, lowering preload (volume stretching the heart) and decreasing the amount of blood regurgitating backward through the leaky valve
4. Monitor blood pressure every 15 to 30 minutes and watch for low blood pressure (hypotension) below 90 systolic — Vasodilators (medications that relax blood vessels) reduce afterload (resistance the heart pumps against) to push more blood forward instead of backward, but can drop pressure too low and reduce organ blood flow (perfusion) (blood flow to tissues)
5. Assess heart rate and rhythm on telemetry and report new atrial fibrillation (an irregular, quivering heartbeat) or sustained a fast heart rate (tachycardia) above 120 — The stretched left atrium from chronic regurgitation triggers atrial fibrillation (an irregular, quivering heartbeat) in 75 percent of severe cases, reducing cardiac output by 20 percent and raising stroke risk from clot formation
6. Auscultate lung sounds every 2 to 4 hours and report new crackles or decreased breath sounds — Crackles (crackling sounds heard with a stethoscope) signal pulmonary edema (fluid flooding the lungs) from rising left atrial pressure pushing plasma into the alveoli, and worsening crackles mean the regurgitation or heart failure is decompensating (getting worse)
7. Weigh the patient daily at the same time with the same clothing and report gains over 2 pounds in one day — Rapid weight gain reflects fluid retention from worsening right heart failure or kidney poor blood flow (hypoperfusion) (reduced kidney blood flow), showing the body cannot compensate for the regurgitant volume anymore

8. Restrict sodium to 2 grams per day and monitor strict intake and output every shift — Sodium holds water in the bloodstream through osmosis (water following salt), so limiting dietary sodium prevents volume expansion that would increase the regurgitant fraction (percentage of blood going backward) and worsen pulmonary congestion

Sources

- AHA/ACC 2020 Guideline for the Management of Patients with Valvular Heart Disease
- American College of Cardiology
- American Heart Association
- European Society of Cardiology 2021 Valvular Heart Disease Guidelines

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