

Aortic Regurgitation

Aortic regurgitation means the door between your heart's main pump and the body's biggest artery does not close tight anymore, so blood flows backward when the heart relaxes—forcing the pump to work overtime.

THE ONE-LINE TRUTH

The aortic valve fails to close all the way when the heart relaxes, so blood that was just pumped out leaks backward into the main pumping chamber with every heartbeat.

The chain — how it unfolds

1. The aortic valve fails to close completely during diastole (the heart's relaxation phase). — why: Leaflet damage from rheumatic scarring, infection, wear-and-tear calcification, or being born with only two leaflets, or aortic root dilation from Marfan syndrome, dissection, high blood pressure, or late-stage syphilis, prevents the three leaflets from meeting and sealing, so blood flows backward from the high-pressure aorta into the left ventricle every time the heart relaxes.
2. In acute severe aortic regurgitation, left ventricular end-diastolic pressure rises sharply within minutes to hours. — why: A normal-sized, non-compliant (stiff) left ventricle cannot stretch quickly to accept the sudden regurgitant volume on top of normal venous return from the lungs, so pressure inside the chamber climbs fast during diastole, like trying to overfill a water balloon that cannot stretch.
3. Pulmonary edema (fluid flooding the lungs) (fluid in the lungs) and cardiogenic shock develop rapidly. — why: High left ventricular diastolic pressure transmits backward through the left atrium into the pulmonary veins and capillaries, forcing plasma (the watery part of blood) across the thin capillary walls into the alveoli (air sacs), impairing oxygen exchange and causing shortness of breath and crackles on exam. At the same time, forward stroke volume drops because much of each ejection leaks back instead of reaching the body, so organs do not get enough blood (cardiogenic shock—shock from pump failure).

4. In chronic aortic regurgitation, the left ventricle undergoes eccentric hypertrophy (dilation with some wall thickening) over months to years. — why: Chronic volume overload triggers the heart muscle cells (myocytes) to add sarcomeres (the tiny contractile units inside each cell) in series, lengthening the cells and enlarging the chamber so it can hold both the normal blood returning from the lungs and the blood that leaked back from the aorta. The wall also thickens a bit to handle the extra work, but not as much as in conditions like aortic narrowing (stenosis) where pressure is the main problem.
5. Pulse pressure widens dramatically, and peripheral pulses become bounding and collapsing. — why: The dilated left ventricle ejects a huge stroke volume (normal forward flow plus the regurgitant volume) into the aorta during systole, raising systolic pressure (the top number, often >160 mmHg). Then blood leaks back rapidly during diastole, dropping diastolic pressure (the bottom number, often <50 mmHg), so the gap (pulse pressure) becomes very wide. You feel this as a strong, bounding pulse that quickly collapses—like a water hammer toy—because pressure falls fast when the valve leaks.
6. Eventually, systolic heart failure (pump failure) occurs as the muscle exhausts. — why: Chronic wall stress from volume overload leads to myocyte apoptosis (programmed cell death), fibrosis (scar tissue replacing muscle), and impaired contractility (weakened squeezing). Ejection fraction (the percent of blood ejected per beat) falls, and the ventricle can no longer compensate for the leak or meet the body's oxygen demand, causing fatigue, shortness of breath, and fluid retention.
7. Coronary blood flow (perfusion) (blood flow to the heart muscle itself) may drop, especially in acute severe aortic regurgitation. — why: The coronary arteries fill during diastole when the aortic valve is closed and aortic pressure is high. In aortic regurgitation, diastolic pressure in the aorta falls because blood leaks back, so less pressure is available to push blood into the coronary arteries. In acute severe AR, this can cause chest pain or even myocardial starved blood flow (ischemia) (not enough oxygen reaching the heart muscle) even without coronary artery blockages.
8. None of these steps is random—each is a direct mechanical consequence of the broken seal. — why: Every clinical finding flows logically from one broken valve forcing the left ventricle to handle a workload it was never designed to sustain long-term. Trace every sign back to the leak, and the whole picture clicks into place.

Spot it — what you see, and why

- Shortness of breath during activity, getting worse until it happens even at rest in severe cases: Blood leaks backward through the aortic valve into the left ventricle (the heart's main pumping chamber), so the chamber has to pump extra hard to push enough blood forward. Over time, pressure builds up in the chamber and backs up into the lungs, forcing fluid out of tiny blood vessels into the lung tissue and air sacs. This fluid makes it harder for oxygen to reach your blood, so you feel short of breath.
- Shortness of breath when lying flat, or waking up at night gasping for air: When you lie down, gravity pulls blood from your legs back toward your chest and heart. This adds even more volume to an already overloaded left ventricle, pushing more fluid into your lungs. After a few hours lying flat, enough fluid collects in your lungs that you wake up struggling to breathe.
- Feeling your heart pound or thump in your chest, especially when lying on your left side: Each beat pushes a huge burst of blood forward (because the ventricle is overfilled from the leak), so your pulse is extra strong and bounding—like a wave crashing instead of a gentle ripple. When you lie on your left side, your heart is pressed closer to your chest wall, so you feel this powerful thump even more.
- Chest pain or pressure during exercise (angina): The heart muscle itself needs oxygen, which it gets from the coronary arteries during diastole (the resting phase between beats). But in aortic regurgitation, blood leaks back so fast that diastolic pressure in the aorta drops very low—like a leaky hose losing pressure. This means less blood feeds the heart muscle. At the same time, the muscle has grown thicker (hypertrophy) to handle the extra work, so it needs even more oxygen. The supply cannot keep up with the demand, causing chest pain.
- High-pitched blowing sound heard through a stethoscope during diastole, loudest along the left side of the breastbone: During diastole, blood flows backward from the aorta into the left ventricle through the leaky valve. This backward jet is fast and turbulent—like air whistling through a crack—and creates a soft, blowing murmur. Leaning forward and breathing out brings the heart closer to the chest wall, making the murmur easier to hear.
- Very wide gap between the top and bottom blood-pressure numbers, and a pulse that feels like a quick hammer strike then disappears (bounding or water-hammer pulse): Each heartbeat ejects a huge volume of blood into the aorta (because the ventricle is overfilled), shooting the systolic pressure up. But then blood leaks back through the valve and the arteries relax wide open, so pressure drops fast during diastole. This creates a sharp spike and quick collapse in every pulse wave—like a hammer tapping then pulling away.

Red flags — act now

- Sudden severe shortness of breath with low blood pressure and fast heart rate—this means the heart can no longer keep up with a large sudden leak, a surgical emergency requiring immediate valve replacement
- New or changing heart murmur with fever and bacteria growing in blood cultures—this suggests infection on the valve (infective endocarditis) that is destroying the leaflets and needs urgent antibiotics and often surgery
- Sudden loss of consciousness (syncope) or chest pain at rest—this means the heart is not pumping enough blood forward to supply the brain or its own muscle, signaling imminent heart failure
- Pumping strength (ejection fraction) dropping below 50 percent or the left ventricle stretching wider than 50 millimeters at the end of contraction on an ultrasound—these are guideline thresholds that tell doctors the heart is starting to fail permanently and surgery should happen now even if you feel okay
- Sudden tearing chest or back pain with a widened shadow in the center of the chest on X-ray—this suggests the aorta is tearing apart (aortic dissection), which is immediately life-threatening and requires emergency imaging and surgery

Treat it — in this order

1. Emergency aortic valve surgery (valve replacement with mechanical or bioprosthetic valve, or valve repair) → Removes or repairs the incompetent valve leaflets or diseased aortic root, stopping backward blood flow into the left ventricle. This is the only definitive cure for acute severe aortic regurgitation. Per 2020 ACC/AHA guidelines (Class I recommendation), emergency surgery is indicated for acute severe aortic regurgitation with cardiogenic shock (systolic blood pressure <90 mmHg with signs of organ poor blood flow (hypoperfusion)) or pulmonary edema (fluid flooding the lungs) (respiratory failure from fluid in the lungs). Surgery immediately restores forward cardiac output and saves the patient's life.
2. Intravenous vasodilators: sodium nitroprusside or nitroglycerin → Dilates arteries throughout the body, reducing systemic vascular resistance (the resistance arteries create against blood flow, like loosening a clamp on a hose). This makes it easier for the left ventricle to eject blood forward into the aorta instead of backward through the leaky valve. Reducing afterload (the pressure the ventricle must overcome to open the aortic valve and push blood out) decreases left ventricular filling pressure and pulmonary capillary wedge pressure, which relieves pulmonary edema (fluid flooding the lungs) and buys time to stabilize the patient before emergency surgery. Nitroprusside is preferred because it has faster onset and offset than nitroglycerin.

3. Intravenous inotropes: dobutamine or milrinone → Increases myocardial contractility (the force with which heart muscle fibers shorten and squeeze) and improves cardiac output (volume of blood pumped per minute, calculated as stroke volume times heart rate). Dobutamine is a beta-1 adrenergic agonist; milrinone is a phosphodiesterase-3 inhibitor. Both make the left ventricle contract harder and faster, raising forward stroke volume and blood pressure. Used in acute severe aortic regurgitation with cardiogenic shock to maintain organ blood flow (perfusion) (blood flow to brain, kidneys, liver) until surgery can be performed.
4. Avoid beta-blockers (metoprolol, carvedilol, atenolol) and other bradycardic agents (digoxin, diltiazem, verapamil) in acute aortic regurgitation; use cautiously only in chronic stable aortic regurgitation for specific indications → In acute aortic regurgitation, the heart compensates for low forward stroke volume by increasing heart rate (tachycardia). This a fast heart rate (tachycardia) is protective—it maintains total cardiac output (stroke volume times heart rate) despite regurgitant volume. Slowing heart rate with beta-blockers or calcium channel blockers reduces cardiac output, worsens low blood pressure (hypotension), and precipitates cardiogenic shock. In chronic compensated aortic regurgitation, beta-blockers may be used carefully for rate control (if atrial fibrillation (an irregular, quivering heartbeat) develops) or blood pressure management, but only under cardiology guidance and never in decompensated or symptomatic patients.
5. Chronic medical management: afterload reduction with ACE inhibitors (lisinopril, enalapril), ARBs (losartan, valsartan), or dihydropyridine calcium channel blockers (nifedipine, amlodipine); elective aortic valve surgery when ejection fraction <55%, left ventricular end-diastolic dimension >50 mm, left ventricular end-systolic dimension >25 mm, or symptoms develop → Vasodilators reduce afterload (the pressure the left ventricle must pump against), making it easier to eject blood forward. This slows the rate of left ventricular dilation and may delay the onset of irreversible systolic dysfunction. Per 2020 ACC/AHA guidelines, elective valve surgery (Class I recommendation) is indicated when ejection fraction drops below 55% (even without symptoms), when left ventricular end-diastolic diameter exceeds 50 mm, when left ventricular end-systolic dimension exceeds 25 mm, or when symptoms (dyspnea, fatigue, chest pain, fainting (syncope)) occur. Surgery before these thresholds allows better post-operative recovery and prevents permanent ventricular damage. Vasodilators are most helpful in patients with severe regurgitation and high blood pressure (hypertension) who are not yet surgical candidates or who decline surgery.

Your top priorities at the bedside

1. Monitor oxygen saturation continuously and apply supplemental oxygen to keep saturation above 92 percent, positioning the patient upright at 45 to 90 degrees. — why: Blood leaking backward into the left ventricle raises pressure in the left atrium (upper chamber) and pulmonary veins (lung blood vessels), pushing fluid into the air sacs and causing pulmonary edema (fluid flooding the lungs) (fluid in the lungs), so upright positioning uses gravity to reduce lung congestion and oxygen prevents dangerous low oxygen in the blood (hypoxemia) (low blood oxygen).
2. Establish large-bore intravenous access immediately and prepare for urgent transfer to the cardiac catheterization lab or operating room if acute severe aortic regurgitation is suspected. — why: Acute aortic regurgitation causes sudden massive volume overload that the left ventricle cannot handle, leading to cardiogenic shock (heart cannot pump enough blood) within hours, and patients need emergency valve replacement surgery to survive, so intravenous access allows rapid delivery of vasodilators (blood vessel relaxers) and inotropes (heart muscle strengtheners) as a bridge to surgery.
3. Monitor blood pressure, heart rate, and rhythm every 15 minutes in acute cases, watching for low blood pressure (hypotension) (systolic blood pressure below 90), a fast heart rate (tachycardia) (heart rate above 100), or new arrhythmias (irregular heartbeats). — why: The left ventricle responds to volume overload by beating faster to maintain cardiac output (amount of blood pumped per minute), so rising heart rate with falling blood pressure signals decompensation (failing heart) and impending shock, and a slow heart rate (bradycardia) (slow heart rate) worsens the leak by giving more time for blood to flow backward during each longer diastole (relaxation phase).