

Aortic Regurgitation

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

The aortic valve sits between the left ventricle (the heart's main pumping chamber) and the aorta (the body's biggest artery). It has three thin, flexible leaflets (flaps) that open when the left ventricle squeezes, letting oxygen-rich blood shoot out to the body, and snap shut when the ventricle relaxes, sealing tight so no blood flows backward. That tight seal also keeps pressure high in the aorta during the heart's resting phase, which is the only time the heart muscle's own arteries (the coronary arteries) fill with blood. Keep that picture in mind—open on squeeze, closed on relax—because every clinical finding in aortic regurgitation is a break in this pattern.

BODY'S JOB

If you do not know the normal job first—forward flow when the heart contracts, tight seal when it relaxes—the abnormal signs will look like a random list instead of a logical cascade flowing from one broken seal.

1. Left ventricle contracts → aortic valve leaflets swing open → blood rushes into the aorta and out to the body.
2. Left ventricle relaxes → aortic valve leaflets snap shut → blood cannot flow backward, so pressure in the aorta stays high and fills the coronary arteries that feed the heart muscle itself.

3. Meanwhile, blood from the lungs flows into the left atrium and then into the relaxed left ventricle to prepare for the next beat.
4. This cycle repeats 60 to 100 times per minute, and the aortic valve must close perfectly every single time to keep blood moving forward and pressure in the right place.

What breaks

Usually one structural break explains all the clinical findings together. Either the valve leaflets are damaged so they cannot close, or the ring they sit in (the aortic root) stretches so the leaflets are pulled apart and cannot meet in the middle.

CLINICAL TERM

Aortic regurgitation happens when the aortic valve cannot seal during diastole (the heart's relaxation phase). The break occurs in one of two places: the leaflets themselves or the aortic root. Leaflet damage can come from rheumatic heart disease (scarring from an immune reaction to untreated group A strep throat, common in low-resource settings), infective endocarditis (bacteria or fungi growing on the valve and eating away at the tissue), bicuspid aortic valve (born with two fused leaflets instead of three, so the valve wears out faster), or degenerative calcification (wear and tear with age that stiffens and shrinks the leaflets). Aortic root problems include Marfan syndrome (a genetic defect in fibrillin that weakens connective tissue, causing the root to balloon out), aortic dissection (a tear in the inner wall of the aorta that lets blood track between the layers and distort the root), chronic uncontrolled high blood pressure (hypertension) (high blood pressure stretching the root over time), or tertiary syphilis (rare now, but late-stage infection causes inflammation and aneurysm of the ascending aorta). That structural break does not stay local. The downstream consequence you see at the bedside depends on timing. Acute severe aortic regurgitation (sudden onset, e.g., from endocarditis tearing a leaflet or dissection ripping the root) overwhelms a normal-sized, stiff left ventricle that cannot dilate quickly, so left ventricular end-diastolic pressure spikes, pulmonary edema (fluid flooding the lungs) (fluid in the lungs) and cardiogenic shock follow within hours. Chronic aortic regurgitation (gradual onset over months to years) lets the left ventricle undergo eccentric hypertrophy—the chamber dilates (stretches) and the wall thickens a bit to handle the extra volume—so the body maintains forward blood flow and you see wide pulse pressure (big gap between systolic and diastolic pressure, like 160/40 mmHg) and bounding pulses instead of shock. But eventually, chronic volume overload exhausts the muscle, contractility (squeezing strength) drops, and systolic heart failure (pump failure) sets in. Read all the clinical findings backward from this one broken seal upstream.

How the body compensates

Vital signs can stay near-normal for a long time while the body compensates hard. Your job is to recognize impending decompensation before the crash, because once the left ventricle stretches too far or the ejection fraction drops below 50 percent, recovery after surgery is incomplete.

1. In chronic aortic regurgitation, the body keeps cardiac output (liters of blood pumped per minute) near-normal by increasing stroke volume (volume pumped per beat) through ventricular dilation and mild sympathetic activation (adrenaline).
2. The dilated ventricle ejects both the normal forward stroke volume plus the regurgitant volume that leaked back, so total stroke volume can be twice normal even though only half of it reaches the body.
3. Blood pressure may look okay because systolic pressure rises from the large stroke volume, and even though diastolic pressure falls from the leak, mean arterial pressure (the average pressure during the whole cycle) can stay adequate for a while.
4. This is the dangerous middle ground—normal or near-normal blood pressure and heart rate mask severe volume overload and early ventricular dysfunction on imaging.
5. The crash point to watch for is acute severe aortic regurgitation (from endocarditis or dissection) causing flash pulmonary edema (fluid flooding the lungs) and cardiogenic shock within hours, or progressive left ventricular dilatation in chronic AR leading to irreversible systolic dysfunction if surgery is delayed past guideline thresholds (ACC/AHA 2020 and ESC 2021).
6. Assess trends—serial echocardiograms every 6 to 12 months, exercise tolerance, and biomarkers like BNP (B-type natriuretic peptide, released when ventricles stretch)—not just a single snapshot, because a patient with stable symptoms but worsening left ventricular size (end-systolic dimension >50 mm) or falling ejection fraction (<50 percent) is not truly stable; they are pre-decompensation.
7. Once ejection fraction drops below 50 percent or end-systolic dimension exceeds 50 mm, even after valve replacement the heart may never fully recover, so surgery is indicated before these thresholds (Class I recommendation, ACC/AHA).
8. Watch for subtle signs of decompensation: declining exercise capacity, rising BNP, or new atrial fibrillation (an irregular, quivering heartbeat) (irregular heart rhythm from atrial stretch), because symptoms often lag behind structural damage.

Domino effect

1. The aortic valve fails to close completely during diastole (the heart's relaxation phase). — 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. — 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. — 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. — 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. — 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. — 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. — 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. — 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.

Symptoms · what the patient feels, 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.
- Very soft or missing first heart sound (S1) when aortic regurgitation happens suddenly and severely: In sudden severe regurgitation, so much blood leaks back into the left ventricle that pressure inside the chamber rises very high during diastole. This high pressure pushes the mitral valve (the valve between the left atrium and ventricle) almost shut even before the ventricle contracts. Because the valve is already nearly closed, it barely moves when contraction starts, so the closing sound is muffled or silent.
- Low blood pressure, fast heart rate, and crackling sounds (rales) in the lungs when aortic regurgitation happens suddenly and severely: A sudden large leak means half the blood pumped forward immediately leaks back, so the amount actually reaching the body (forward stroke volume) drops sharply. The heart tries to compensate by beating faster, but blood pressure still falls because not enough blood is getting out. Meanwhile, pressure backs up into the lungs so fast that fluid floods the air sacs, producing crackling sounds when you breathe and causing severe shortness of breath.

Focused exam

- High-pitched blowing sound in early diastole (relaxation phase of the heartbeat) at the left sternal border, heard best when the patient leans forward and breathes all the way out: This is the sound of blood leaking backward through an aortic valve that does not close completely. It proves aortic regurgitation (backward leak from the aorta into the heart's main pumping chamber) is happening right now. The leaning-forward position brings the heart closer to the chest wall, making the murmur easier to hear.
- Wide pulse pressure — the top number (systolic) and bottom number (diastolic) of the blood pressure are far apart, often by 60 mmHg or more: The left ventricle (main pumping chamber) stretches to hold extra blood leaking back from the aorta. It then squeezes harder, shooting more blood forward with each beat—this raises the top number. But during diastole, blood leaks backward through the faulty valve instead of staying in the arteries, so the bottom number drops. A very wide gap (like 160/50 mmHg) tells you the leak is moderate to severe or has been going on long enough for the heart to enlarge and compensate.
- Bounding pulses in the neck (carotid arteries) and wrists (radial arteries) — you can see and feel a strong, forceful pulse that rises quickly and falls quickly, like a water hammer: The left ventricle ejects a much larger stroke volume (amount of blood per beat) to make up for the blood that leaks backward. This extra-strong forward surge creates the bounding pulse. The faster it rises and falls, the more severe the regurgitation.
- Soft or absent first heart sound (S1) in acute severe aortic regurgitation: When aortic regurgitation strikes suddenly (like from a valve infection or torn aorta), the left ventricle fills so fast with leaking blood that pressure inside the chamber shoots up. This high pressure pushes the mitral valve (the valve between the left atrium and left ventricle) closed early, before it normally would close. Early closure muffles S1. This is a red flag that the heart is in crisis and cannot adapt.
- Fast breathing (respiratory rate above 20 breaths per minute) and low blood pressure (systolic below 90 mmHg) in a patient with sudden-onset symptoms: Sudden severe aortic regurgitation floods the left ventricle before it has time to stretch and adapt. The ventricle cannot pump enough blood forward to keep organs fed (this is called cardiogenic shock—heart failure causing organ starvation). Fluid backs up into the lungs, causing pulmonary edema (fluid flooding the lungs) (fluid in lung tissue), which makes breathing fast and labored. Low blood pressure means the heart is failing to maintain circulation. Acute causes include infective endocarditis (infection on the valve) or aortic dissection (a tear in the aorta's inner lining). This pattern demands emergency surgery.

- Crackles (wet popping sounds) in the lower lung fields, swollen neck veins (jugular venous distention), or swollen ankles and legs (peripheral swelling (edema)): Chronic aortic regurgitation makes the left ventricle dilate (stretch and enlarge) over months to years. Eventually the muscle weakens and cannot squeeze effectively (systolic dysfunction). Fluid backs up into the lungs (causing crackles) and into the body's veins (causing swelling). These are signs of heart failure and mean the heart is starting to lose the compensation battle.
- Diastolic blood pressure below 60 mmHg, sometimes as low as 40 mmHg: During diastole, blood leaks backward through the incompetent aortic valve instead of staying in the arteries. This drops the baseline pressure in the arteries. The lower the diastolic number, the more blood is leaking backward—diastolic pressure below 60 mmHg signals moderate to severe regurgitation.

Diagnostics · every test answers a question

- Transthoracic echocardiography (ultrasound of the heart through the chest wall) → How much blood is leaking backward? What does the valve look like? How big is the left ventricle? Is the pumping strength normal?
- Electrocardiogram (EKG, tracing of the heart's electrical activity) → Is the left ventricle hypertrophied (thickened walls) or strained from chronic volume overload?
- Chest x-ray (plain radiograph of the chest) → Is the heart enlarged? Is there fluid in the lungs?
- Transesophageal echocardiography (TEE, ultrasound probe inserted into the esophagus) or cardiac CT or cardiac MRI → What caused the aortic regurgitation when chest-wall ultrasound images are poor? Is there aortic dissection (a tear in the aorta's wall)?
- Blood cultures (three sets from different sites before antibiotics) → Is there infective endocarditis (bacterial infection on the valve)?
- Brain natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) → Is there heart failure or left ventricular strain?
- Troponin I or T (cardiac enzyme indicating myocardial injury) → Is there myocardial starved blood flow (ischemia) (heart muscle not getting enough oxygen) or tissue death from blocked blood flow (infarction) (heart attack)?
- Cardiac catheterization (invasive measurement of pressures and blood flow in the heart chambers and great vessels) → Is coronary artery disease present before planned valve surgery, or is there diagnostic uncertainty about regurgitation severity?

Treatment logic

- 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.
- 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.
- 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.
- 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.

- 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.
- Antibiotic prevention (prophylaxis) for dental and invasive procedures after prosthetic valve replacement or valve repair with prosthetic material (per AHA 2021 guidelines) → Prevents bacteremia (bacteria in the bloodstream from dental work or invasive procedures) from seeding the prosthetic valve or valve repair site, which would cause prosthetic valve endocarditis (infection of the artificial valve). Per AHA 2021 guidelines, antibiotic prevention (prophylaxis) (single dose of amoxicillin 2 g orally 30-60 minutes before the procedure, or clindamycin 600 mg if penicillin-allergic) is indicated for high-risk cardiac conditions (prosthetic valve, prior endocarditis, cardiac transplant with valvulopathy, or certain congenital heart disease) before dental procedures involving gingival manipulation or perforation of oral mucosa. Prevention is NOT routinely indicated for native valve aortic regurgitation alone.
- Diuretics (furosemide, bumetanide) for symptomatic volume overload in chronic aortic regurgitation with heart failure → Increases urine output, removing excess sodium and water from the body. This reduces total blood volume, lowers left ventricular filling pressure, and relieves pulmonary congestion (fluid in the lungs causing shortness of breath) and peripheral swelling (edema) (swollen legs). Diuretics improve symptoms but do not slow disease progression.

Medications

Nitroprusside · Intravenous vasodilator (blood vessel relaxer)

Nitroprusside relaxes the muscle layer in the walls of arteries (the tubes that carry blood away from the heart). When arteries relax, they open wider, so it's easier for the left ventricle (main pumping chamber) to push blood forward into the body instead of having it leak backward

through the broken valve. Think of it like opening a second door in a crowded room—people flow out more easily. This drug works only in acute severe aortic regurgitation (AR) as a short-term bridge to emergency surgery; it does NOT fix the valve itself.

1. Nitroprusside breaks down inside artery muscle cells and releases nitric oxide (a gas that signals muscles to relax).
2. Nitric oxide tells the muscle to stop squeezing, so the artery opens wider.
3. With wider arteries, the resistance (how hard it is to push blood forward) drops.
4. The failing left ventricle can now eject more blood forward with each beat instead of just filling up with the blood leaking backward.
5. This buys time until a surgeon can replace or repair the valve.

SAFETY WATCH

Can cause dangerous low blood pressure (hypotension) if the dose is too high—monitor blood pressure every few minutes. · Releases cyanide as it breaks down; if used longer than 24–48 hours or at high doses, cyanide can build up and poison cells (causes confusion, lactic acidosis (acid from oxygen-starved tissues)). Check lactate and thiocyanate levels if infusion runs beyond 48 hours. · Must be given through a central IV line in a monitored setting (ICU or emergency department). · Light breaks it down, so the IV bag and tubing must be wrapped in foil.

Dobutamine · Intravenous inotrope (heart muscle strengthener)

Dobutamine makes the heart muscle squeeze harder and faster, so each beat pumps more blood forward. In acute severe AR, the left ventricle is suddenly flooded with extra blood leaking backward from the aorta, and the muscle cannot keep up—this causes cardiogenic shock (the pump fails and blood pressure drops). Dobutamine whips the tired muscle into working harder, like giving a flagging cyclist a burst of energy. It is a short-term emergency measure to keep blood pressure up until surgery fixes the valve.

1. Dobutamine locks onto beta-1 receptors (docking sites) on heart muscle cells.
2. This triggers a cascade inside the cell that raises calcium levels.
3. More calcium floods the contractile machinery (the sliding filaments that squeeze the heart).
4. The muscle contracts harder (increased inotropy) and slightly faster (mild increase in heart rate).
5. More blood is ejected forward with each beat, raising cardiac output (liters per minute) and blood pressure.

SAFETY WATCH

Can cause dangerous fast heart rhythms (tachycardia, atrial fibrillation (an irregular, quivering heartbeat), ventricular a fast heart rate (tachycardia))—monitor continuous ECG. · May increase oxygen demand by the heart muscle; if coronary arteries are narrowed, this can trigger chest pain or even a heart attack. · Can raise blood pressure too high in some patients, especially if given with vasodilators at the wrong ratio. · Risk of tissue death (necrosis) if the IV leaks into surrounding tissue (extravasation); use a central line when possible.

ACE inhibitor (examples: enalapril, lisinopril, ramipril) · Angiotensin-converting enzyme inhibitor (afterload reducer and remodeling blocker)

ACE inhibitors block an enzyme (a protein that speeds up chemical reactions) that makes angiotensin II, a powerful molecule that squeezes arteries tight and tells the kidneys to hold onto salt and water. By stopping angiotensin II, the drug relaxes arteries (lowers afterload, the resistance the heart pumps against) and reduces the extra fluid volume that overloads the heart. In chronic aortic regurgitation, this slows the process by which the left ventricle stretches and weakens (remodeling). Think of it like lowering the weight a tired weightlifter has to lift—over months to years, the heart stays stronger longer.

1. ACE inhibitor blocks the ACE enzyme in the lungs and blood vessels.
2. Angiotensin II levels fall, so arteries relax and open wider.
3. The kidneys release less aldosterone, so you pee out more salt and water; blood volume drops.
4. With lower resistance and lower volume, the left ventricle does not have to work as hard.
5. Over time, the ventricle stretches less, and the muscle cells lay down less scar tissue (collagen), delaying the progression to heart failure.

SAFETY WATCH

Can cause a dry, tickling cough in about 10–15% of patients (due to bradykinin buildup); switch to an ARB if this happens. · Risk of angioedema (sudden swelling of the lips, tongue, or throat that can block breathing)—rare but life-threatening; stop the drug immediately if swelling occurs. · Can raise potassium levels (hyperkalemia), especially in patients with kidney disease or those taking potassium supplements or spironolactone; check potassium within 1–2 weeks of starting. · Can worsen kidney function in patients with severe narrowing of both renal arteries (renal artery narrowing (stenosis)); check creatinine within 1–2 weeks. · Absolutely contraindicated (must not be used) in pregnancy—causes severe birth defects and fetal death.

ARB (angiotensin receptor blocker; examples: losartan, valsartan, candesartan) · Angiotensin II receptor blocker (afterload reducer and remodeling blocker)

ARBs block the receptor (docking site) where angiotensin II would normally land on artery and kidney cells. Even though angiotensin II is still floating in the blood, it cannot squeeze arteries or tell kidneys to hold onto salt and water because the receptor is locked shut. The net effect is the same as an ACE inhibitor—lower resistance, less volume overload, and slower stretching of the left ventricle in chronic AR. ARBs are used when a patient cannot tolerate an ACE inhibitor (usually because of cough).

1. ARB molecule locks onto the AT1 receptor, blocking angiotensin II from binding.
2. Without angiotensin II signaling, arteries relax and widen.
3. Aldosterone release drops, so the kidneys excrete more salt and water.
4. Blood volume and arterial resistance both fall, lowering the workload on the left ventricle.
5. Over months to years, the ventricle remodels more slowly, delaying heart failure.

SAFETY WATCH

Can raise potassium levels (hyperkalemia); monitor potassium and creatinine within 1–2 weeks of starting and periodically thereafter. · Can worsen kidney function in bilateral renal artery narrowing (stenosis) (same as ACE inhibitors). · Risk of angioedema is much lower than with ACE inhibitors but still possible; stop immediately if swelling occurs. · Absolutely contraindicated in pregnancy—causes the same severe birth defects as ACE inhibitors.

Diuretics (loop diuretic example: furosemide; thiazide example: hydrochlorothiazide) · Diuretic (water pill)

Diuretics make your kidneys dump extra salt and water into the urine, shrinking the total volume of blood in your circulation. In chronic AR with volume overload (the left ventricle is stretched by too much blood sloshing backward), lowering volume reduces the preload (how much the chamber fills before it squeezes). Think of it like letting air out of an over-inflated balloon—the walls are less stretched and under less strain. Loop diuretics (like furosemide) are stronger and used when fluid backs up into the lungs (pulmonary swelling (edema)); thiazides are milder and used for mild volume overload or high blood pressure.

1. Diuretic enters the kidney tubule and blocks sodium transporters (pumps that pull salt back into the blood).
2. With less sodium reabsorbed, water stays in the tubule (water follows salt).
3. More urine is made, carrying away sodium and water.

4. Blood volume drops, so the left ventricle fills with less blood each beat (lower preload).
5. Symptoms of congestion (shortness of breath, leg swelling) improve.

SAFETY WATCH

Can cause low potassium (hypokalemia), especially loop and thiazide diuretics; check potassium regularly and consider a potassium supplement or potassium-sparing diuretic (spironolactone, amiloride). · Can cause dehydration and low blood pressure if dose is too high—monitor weight, blood pressure, and kidney function. · Can raise uric acid and trigger gout attacks in susceptible patients. · Loop diuretics can damage hearing (ototoxicity) at very high doses or when combined with aminoglycoside antibiotics.

Pathogen-directed antibiotics (for infective endocarditis; examples: nafcillin or cefazolin for *Staphylococcus aureus*; penicillin G or ceftriaxone for *Streptococcus viridans*; vancomycin for MRSA; ampicillin + gentamicin for *Enterococcus*) · Antibiotic (bacteria killer)

Infective endocarditis is an infection on the valve, usually caused by bacteria in the bloodstream that stick to damaged valve tissue and form a clump (vegetation) of germs, clotted blood, and inflammatory cells. These vegetations destroy the valve leaflets, causing regurgitation. The antibiotic kills the bacteria by breaking their cell walls (beta-lactams like penicillin, cephalosporins) or blocking their protein-making machinery (aminoglycosides like gentamicin) or punching holes in their membranes (vancomycin). You must pick the right antibiotic based on which bug is growing in blood cultures. Treatment lasts 4–6 weeks intravenously (through a vein) because vegetations are hard for the immune system to reach.

1. Antibiotic enters the bloodstream and travels to the valve vegetation.
2. It penetrates the clump of bacteria, clot, and debris (slowly, which is why treatment is long).
3. Inside the bacteria, the drug blocks a vital process—wall building, protein synthesis, or membrane integrity.
4. Bacteria die and the vegetation shrinks, though dead tissue and scar remain.
5. If the infection is controlled early, valve damage may be limited; if not, surgery is needed to remove the destroyed valve.

SAFETY WATCH

Aminoglycosides (gentamicin) can damage kidneys (nephrotoxicity) and the inner ear (ototoxicity, causing hearing loss or balance problems); monitor blood levels (peaks and troughs) and kidney function. · Beta-lactams can cause allergic reactions ranging from rash to life-threatening anaphylaxis; ask about penicillin allergy history. · Vancomycin can cause 'red man syndrome' (flushing, itching, low blood pressure) if infused too fast—give slowly over at least 60 minutes. Also monitor kidney function. · Long IV therapy risks line infections (catheter-related bloodstream infection); use a PICC line or port and follow sterile technique.

Benzathine penicillin G (for rheumatic heart disease secondary prevention (prophylaxis)) · Long-acting penicillin antibiotic (prevents recurrent strep throat)

Rheumatic heart disease is caused by the immune system attacking the heart valves weeks after a strep throat infection (group A Streptococcus). The attack scars and deforms the valves, leading to regurgitation or narrowing (stenosis) (narrowing). Once someone has had rheumatic fever, they are at high risk for another strep infection triggering another immune attack and more valve damage. Benzathine penicillin G is a slow-release shot given once every 3–4 weeks that keeps a low, steady level of penicillin in the blood, killing any strep bacteria before they cause throat infection. This prevents recurrence, not the valve damage that already happened.

1. Benzathine penicillin G is injected deep into muscle (usually the buttock).
2. The drug forms a depot (storage pocket) in the muscle and is slowly released over 3–4 weeks.
3. A low, steady level of penicillin circulates in the blood.
4. If group A Streptococcus enters the throat, penicillin kills it before the infection can trigger the immune system.
5. Fewer strep infections mean fewer rheumatic fever recurrences and less progressive valve damage.

SAFETY WATCH

Injection is painful—warn the patient and use proper deep IM technique. · Risk of allergic reaction, including anaphylaxis, especially in patients with penicillin allergy; keep epinephrine nearby for the first dose. · If penicillin allergy is confirmed, use a macrolide (azithromycin or erythromycin) or a first-generation cephalosporin (if the allergy is not severe) instead. · Prevention (Prophylaxis) duration depends on the severity of rheumatic heart disease: at least 10 years after the last episode, or until age 21 (whichever is longer) for mild cases; lifelong for those with significant valve damage.

Red flags

- 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

Nursing priorities

1. Monitor oxygen saturation continuously and apply supplemental oxygen to keep saturation above 92 percent, positioning the patient upright at 45 to 90 degrees. — 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. — 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). — 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).
4. Prepare and administer intravenous vasodilators like sodium nitroprusside or nitroglycerin as ordered, monitoring blood pressure closely and titrating to maintain systolic pressure between 100 and 110. — Vasodilators reduce afterload (resistance the left ventricle pumps against) by relaxing arteries, making it easier for blood to flow forward into the body instead of backward through the leaky valve, improving forward stroke volume (blood pumped per beat) and reducing pulmonary congestion, but dropping blood pressure too low decreases coronary blood flow (perfusion) (blood flow to heart muscle).
5. Assess lung sounds every 2 to 4 hours, listening for crackles (rales) starting at the bases and moving upward, and monitor daily weights and intake and output strictly. — Crackles that start at the lung bases and climb toward the apices (tops) indicate worsening pulmonary edema (fluid flooding the lungs) (fluid in lungs) from rising left atrial pressure, and daily weights catch fluid retention (1 liter of fluid equals 1 kilogram or 2.2 pounds), guiding diuretic therapy (water pills) like furosemide to remove excess fluid.
6. Avoid administering beta-blockers (metoprolol, carvedilol, atenolol), calcium channel blockers (diltiazem, verapamil), or digoxin without clarifying the order with the provider in acute aortic regurgitation. — These medicines slow the heart rate, which lengthens diastole (relaxation time), giving more time for blood to leak backward through the incompetent valve and worsening the regurgitant volume (amount of backward flow), potentially causing sudden cardiovascular collapse (circulatory failure), although they may be used carefully in stable chronic aortic regurgitation.
7. Draw blood cultures before any antibiotics if fever or infection is suspected, then start broad-spectrum intravenous antibiotics as ordered without delay. — Infective endocarditis (valve infection) destroys valve leaflets rapidly, causing acute severe regurgitation, and blood cultures identify the bacteria so antibiotics can be targeted, but antibiotics must start immediately after cultures are drawn because every hour of delay allows more valve destruction and increases mortality (death rate).

8. Educate the patient to report any new chest pain, worsening shortness of breath, dizziness, or feeling faint immediately, and keep the call bell within reach at all times. — These symptoms indicate worsening left ventricular failure (pump cannot keep up), dangerous arrhythmias (abnormal heart rhythms), or cardiogenic shock (circulatory collapse), requiring immediate intervention with medicines or emergency surgery, and early recognition prevents cardiac arrest (heart stops) or sudden death.

Sources

- AHA/ACC 2020 Guideline for the Management of Patients With Valvular Heart Disease
- ESC/EACTS 2021 Guidelines for the Management of Valvular Heart Disease
- American Heart Association
- Cochrane Database of Systematic Reviews

Last reviewed: 2026-07-07