

Aortic Stenosis

Aortic narrowing (stenosis) means the gate to the body is narrowing, and once symptoms start—chest pain, fainting, or heart failure—average survival without a new valve is under three years.

THE ONE-LINE TRUTH

The aortic valve has narrowed, forcing the left ventricle to generate dangerously high pressure to push blood through.

The chain — how it unfolds

1. Valve area shrinks and left ventricular outflow resistance rises — why: Calcium deposits, fibrosis, and fusion of the leaflets reduce the valve opening from a normal 3–4 cm² to less than 1 cm² in critical narrowing (stenosis). Flow through a narrow opening requires much higher pressure upstream, so the left ventricle must generate systolic pressures of 200 mmHg or more (versus a normal 120 mmHg) to push the same amount of blood into the aorta. The pressure gradient across the valve (ventricle minus aorta) jumps from near-zero to 40–50 mmHg or higher.
2. Left ventricle hypertrophies concentrically (wall thickens without chamber dilation) — why: Chronic pressure overload (high afterload) triggers muscle-cell growth and collagen deposition. The ventricle thickens inward to normalize wall stress by Laplace's law: wall stress equals (pressure times radius) divided by (2 times wall thickness). By doubling wall thickness, the ventricle can handle twice the pressure without overstretching the muscle fibers. This concentric hypertrophy maintains stroke volume and systolic blood pressure for months to years, so the patient remains asymptomatic during the compensated phase.
3. Diastolic dysfunction develops (impaired relaxation and filling) — why: A thick, stiff ventricular wall cannot relax quickly or completely during diastole. The chamber does not expand well, so filling pressure inside the ventricle rises (left ventricular end-diastolic pressure climbs from 8–12 mmHg to 20–30 mmHg or higher). Filling becomes highly dependent on atrial contraction—the atrial kick contributes up to 40% of ventricular filling in severe hypertrophy (versus 15–20% normally). Loss of the atrial kick (from atrial fibrillation (an irregular, quivering heartbeat) or even a premature beat) can drop cardiac output acutely and trigger pulmonary edema (fluid flooding the lungs).

4. Coronary blood flow (perfusion) falls, especially during exertion — why: Coronary arteries fill during diastole, when the ventricle is relaxed and aortic pressure is high. In aortic narrowing (stenosis), diastolic pressure inside the thick, stiff ventricle rises, compressing the small vessels in the heart muscle (especially the subendocardium, the inner layer that has the longest, thinnest vessels). Coronary blood flow (perfusion) pressure equals aortic diastolic pressure minus left ventricular diastolic pressure, so as the ventricle stiffens, the driving pressure for coronary flow drops. During exercise, heart rate rises and diastolic time shortens, worsening the supply-demand mismatch. The result is myocardial starved blood flow (ischemia) (the muscle is starving for oxygen) even in patients with no blockages in the epicardial coronary arteries.
5. Cardiac output becomes fixed and cannot rise with demand — why: The narrow valve acts as a bottleneck. Stroke volume is limited by the valve area—the ventricle cannot push more blood per beat through a 1 cm² opening no matter how hard it squeezes. Heart rate can increase during exercise, but there is a ceiling: if heart rate climbs too high, diastolic filling time shrinks and stroke volume drops even further. Systemic vascular resistance normally falls during exercise (blood vessels in the muscles dilate to let more blood in), but in aortic narrowing (stenosis) the body is already maximally vasodilated just to maintain resting tissue blood flow (perfusion). The result is fixed or nearly fixed cardiac output—when metabolic demand rises (exercise, fever, stress), the heart cannot deliver, and blood pressure may drop instead of rising.
6. Symptoms appear: angina, fainting (syncope), trouble breathing (dyspnea) (heart failure) — why: Angina (chest pain) results from myocardial oxygen supply-demand mismatch—the thick muscle mass needs more oxygen, but coronary flow is reduced by high diastolic pressure and shortened diastolic time. Fainting (Syncope) (fainting) results from inability to raise cardiac output and cerebral blood flow (perfusion) during exertion or from arrhythmia (ventricular a fast heart rate (tachycardia), atrial fibrillation (an irregular, quivering heartbeat)) that drops output acutely. Trouble breathing (Dyspnea) (shortness of breath) results from elevated left atrial and pulmonary venous pressures (backward congestion from diastolic dysfunction) and eventual systolic failure when the hypertrophied muscle decompensates (fibrosis, starved blood flow (ischemia), and exhaustion cause ejection fraction to fall).

7. Without valve replacement, rapid decline and death follow — why: Once symptoms start, the compensatory reserve is exhausted. The ventricle cannot generate enough pressure to maintain flow, stroke volume falls, cardiac output drops, and blood backs up into the lungs (pulmonary swelling (edema)). Forward failure (low output to the body) and backward failure (congestion in the lungs) worsen together. Sudden cardiac death can occur from ventricular arrhythmia (the thick, blood-starved (ischemic) muscle is electrically unstable) or from acute drop in preload (dehydration, vasodilation) that the fixed-output heart cannot tolerate. Average survival is 1–2 years for heart failure, 2–3 years for fainting (syncope), and 3–5 years for angina—but many patients die within months without intervention.
8. Aortic valve replacement reverses the process if done in time — why: Replacing the narrowed (stenotic) valve (surgical AVR or transcatheter TAVR) removes the obstruction, drops left ventricular systolic pressure back to normal, and allows the hypertrophied muscle to remodel (wall thickness decreases, diastolic function improves). If intervention happens before irreversible muscle damage (severe fibrosis, low ejection fraction), symptoms improve, exercise tolerance returns, and survival approaches that of the general population. If intervention is delayed until the muscle has failed, recovery is incomplete and operative risk is much higher—hence the guideline emphasis on timely referral as soon as symptoms or severe narrowing (stenosis) with LV dysfunction appear.

Spot it — what you see, and why

- Shortness of breath with activity (exertional trouble breathing (dyspnea)): The stiff, thick left side of the heart pushes blood backward into the lungs, raising pressure in the tiny lung blood vessels. Fluid leaks out into the lung tissue like water seeping through a sponge, making the lungs heavy and stiff so each breath feels harder.
- Chest pressure or pain during activity (angina): The heart muscle has grown thick to push against the narrow valve, so it needs more oxygen. But high pressure inside the heart squeezes the small blood vessels that feed the muscle, like stepping on a garden hose. Not enough oxygen reaches the tissue, causing pain.
- Fainting or feeling dizzy during exercise (exertional fainting (syncope)): The narrow valve acts like a stuck gate that won't open wide. During exercise, the body needs more blood but the heart can't pump faster or harder through the tiny opening. Blood pressure drops, the brain doesn't get enough fuel, and you pass out.
- Tiring easily and stopping activity sooner than before: Because the valve opening is so small, the heart can't send enough oxygen-rich blood to your muscles when you move. Your muscles run low on fuel fast, like a car on an empty tank, so you feel exhausted and have to stop.

- Trouble breathing when lying flat or waking up gasping at night: When you lie down, gravity lets more blood flow back to the heart. The stiff heart can't handle the extra load, so pressure in the lungs shoots up and fluid floods into the air sacs. You wake up feeling like you're drowning.
- Harsh heart murmur that gets louder then softer, heard at the upper right chest: Blood rushing through the tiny valve opening creates rough, swirling flow, like water spraying through a pinched hose. The vibrations make a harsh sound the doctor hears with a stethoscope, and the sound travels up into the neck arteries.

Red flags — act now

- Fainting or near-fainting during exercise (the heart cannot supply enough blood; sudden death risk is very high)
- New or worse shortness of breath, leg swelling, or trouble lying flat in someone with known aortic narrowing (stenosis) (the heart is failing and the valve must be replaced urgently)
- Chest pain at rest or with only light activity (the heart muscle is starving for oxygen and can die)
- Fast, irregular heartbeat (atrial fibrillation) in a patient with aortic narrowing (stenosis) (losing the normal atrial squeeze can crash blood pressure and cause shock)
- Low blood pressure or shock after taking water pills or blood-pressure medicines (the heart depends on high filling pressure; sudden loss of fluid can stop the pump)

Treat it — in this order

1. Aortic valve replacement (surgical AVR or transcatheter TAVR) → Removes the old, narrowed valve and replaces it with a new one (mechanical, bioprosthetic, or balloon-expandable), restoring a normal opening (valve area 1.5–2.0 cm²) and eliminating the pressure gradient. The left ventricle no longer has to generate extreme pressure to eject blood, so cardiac output increases immediately and the heart begins to shrink (reverse remodeling of hypertrophy) over 6–12 months.
2. Avoid rapid vasodilators (nitrates, ACE inhibitors, ARBs) and maintain adequate preload in severe symptomatic AS before valve replacement → Keeps blood pressure stable by maintaining venous return (preload—the amount of blood filling the heart) and systemic vascular resistance (afterload—the resistance the heart pumps against). The thickened, stiff left ventricle depends on high filling pressure to stretch enough to pump. Avoiding drugs that suddenly drop afterload or reduce preload prevents a dangerous fall in blood pressure (hypotension), which the fixed-output ventricle cannot compensate for, causing fainting (syncope), shock, or cardiac arrest.

3. Diuretics (e.g., furosemide 20–40 mg PO or IV) for pulmonary congestion or volume overload → Reduces total blood volume and the pressure in the pulmonary capillaries (tiny blood vessels in the lungs), pulling fluid back out of the air sacs and relieving shortness of breath (dyspnea) and wet lung sounds (rales). Must be dosed carefully because the stiff left ventricle needs high filling pressure to maintain stroke volume (the amount of blood pumped per beat).
4. Endocarditis prevention (prophylaxis) (amoxicillin 2 g PO or ampicillin 2 g IV 30–60 minutes before dental procedures) ONLY if prosthetic valve is present → Achieves high antibiotic levels in the blood during procedures that cause brief bacteremia (bacteria entering the bloodstream, such as tooth extractions or gum work), preventing bacteria from sticking to the prosthetic valve and forming infected clumps (vegetations—infective endocarditis).
5. Serial echocardiography every 6–12 months for asymptomatic severe AS; every 1–2 years for moderate AS → Tracks how the valve area, pressure gradient, and ejection fraction change over time. Detects the transition to symptoms or left ventricular dysfunction (ejection fraction drops below 50%), which are absolute indications for valve replacement even if the patient reports feeling fine. Early detection prevents sudden death or irreversible heart damage.

Your top priorities at the bedside

1. Monitor blood pressure and heart rate every 4 hours or more often if symptomatic, and call the provider immediately if systolic blood pressure drops below 90 mmHg or patient becomes dizzy or confused — why: The thick left ventricle needs high filling pressure (preload) to pump blood through the narrow valve. Low blood pressure means the brain and other organs are not getting enough blood and the patient can go into shock or have a cardiac arrest.
2. Keep the patient on continuous telemetry (heart monitor) and watch for atrial fibrillation (an irregular, quivering heartbeat), ventricular arrhythmias, or heart block, alerting the provider immediately if any appear — why: Aortic narrowing (stenosis) patients depend on atrial contraction to fill the stiff left ventricle. Atrial fibrillation (an irregular, quivering heartbeat) (irregular heartbeat) causes loss of atrial kick and can drop cardiac output by 25 percent, leading to sudden decompensation. Ventricular arrhythmias can cause sudden death.
3. Position the patient upright at 45 to 90 degrees if they have shortness of breath or crackles in the lungs, and give prescribed diuretics like furosemide on time to remove excess lung fluid — why: Fluid backs up into the lungs when the left ventricle fails. Sitting upright uses gravity to pool blood in the legs and reduce the amount returning to the heart and lungs. Diuretics remove extra fluid and lower the pressure in the lungs so the patient can breathe.