

Aortic Stenosis

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

The aortic valve opens during systole (when the left ventricle squeezes) to let oxygen-rich blood leave the heart and enter the aorta, then closes during diastole (when the ventricle relaxes) to prevent backflow and maintain pressure in the aorta that feeds the coronary arteries. A normal valve has three thin, flexible leaflets and opens to 3–4 cm² (about the size of a nickel).

BODY'S JOB

Every time your left ventricle squeezes, it needs to push blood through the aortic valve and into the aorta, which carries oxygen and fuel to your entire body. Between beats, the valve slams shut so blood doesn't leak backward, and that closure keeps pressure high in the aorta—high enough to push blood into the coronary arteries that feed the heart muscle itself. Think of the valve as a one-way gate that must open wide and close tight, cycle after cycle, about 100,000 times a day.

1. Left ventricle fills with oxygen-rich blood from the lungs (via the left atrium and mitral valve).
2. Ventricle squeezes (systole), pressure inside rises, and the aortic valve leaflets swing open.
3. Blood rushes through the wide-open valve (3–4 cm²) into the aorta at high speed and low resistance.
4. Ventricle finishes squeezing, pressure drops, and the valve snaps shut to prevent backflow.

5. During relaxation (diastole), the aorta stays pressurized by the closed valve, and that pressure drives blood into the coronary arteries (which fill only when the heart is relaxed).
6. The cycle repeats: fill, squeeze, eject, relax—smooth and efficient when the valve is healthy.

What breaks

The aortic valve opening shrinks because the leaflets thicken, stiffen, and fuse together—like a door hinge that rusts and won't open all the way.

CLINICAL TERM

Senile calcific aortic narrowing (stenosis) (calcium deposits on a normal three-leaflet valve, like plaque on teeth) is the leading cause in adults over 65 in developed countries. Bicuspid aortic valve (a birth defect in which only two leaflets form instead of three) causes faster wear-and-tear and presents one to two decades earlier, often in the 40s or 50s. Rheumatic aortic narrowing (scarring from untreated strep throat that triggers an autoimmune attack on the valve) is now rare in high-resource settings but remains common where acute rheumatic fever is endemic. Regardless of cause, the leaflets thicken and calcify, the valve opening (orifice) shrinks—critical narrowing is less than 1 cm², about the size of a pea—and the left ventricle faces a high-resistance bottleneck every time it tries to push blood out.

How the body compensates

Vital signs stay normal for months to years while the left ventricle thickens to overcome the obstruction, but once symptoms appear—chest pain, fainting, or shortness of breath—the muscle is failing and sudden death or rapid decline is imminent.

1. {'step': 'Left ventricle hypertrophies concentrically (thickens without dilating)', 'why': 'The ventricle responds to chronic pressure overload by growing new muscle and laying down collagen, which increases wall thickness and normalizes wall stress. This compensatory hypertrophy lets the ventricle generate systolic pressures of 200–250 mmHg (far above the normal 120 mmHg) to push blood through the narrowed valve. Stroke volume and resting cardiac output stay near-normal, so the patient feels fine and can perform daily activities without symptoms.'}
2. {'step': 'Compensated phase lasts years—patient is asymptomatic', 'why': 'During this long plateau, exercise tolerance is preserved (or only mildly reduced), blood pressure is normal or even high-normal, and routine exams may show only a harsh systolic murmur. The narrowing (stenosis) slowly worsens (valve area drops about 0.1–0.3 cm² per year on average), but the ventricle keeps up. Many patients have no idea their valve is critically tight until an echocardiogram is done for another reason.'}

3. {'step': 'Symptom onset marks the crash point', 'why': "Once the ventricle can no longer compensate—either because the valve is so tight that even maximal hypertrophy isn't enough, or because the muscle itself starts to fail (fibrosis, starved blood flow (ischemia), exhaustion)—symptoms appear suddenly or rapidly worsen. The three classic symptoms are angina (chest pain from coronary starved blood flow), fainting (syncope) (fainting from inability to raise cardiac output during exertion), and heart failure (shortness of breath from backward congestion and forward failure)."}
4. {'step': 'Survival without valve replacement drops sharply', 'why': 'Natural history studies (and 2020 ACC/AHA guidelines) show average survival after symptom onset is 2 years for heart failure, 3 years for fainting (syncope), and 5 years for angina. These are averages—many patients die within months. Sudden cardiac death occurs in 15–20% of symptomatic patients, often triggered by exertion, arrhythmia, or a sudden drop in preload (e.g., dehydration, overdiuresis, vasodilator medication).'}
5. {'step': 'Decompensation accelerates once it starts', 'why': 'As the muscle fails, stroke volume drops, cardiac output falls, and blood backs up into the lungs (pulmonary swelling (edema)). Diastolic dysfunction worsens, atrial pressure rises, and atrial fibrillation (an irregular, quivering heartbeat) may develop, which eliminates the atrial kick and drops cardiac output even further. The patient spirals: low output → poor blood flow (perfusion) → more strain on the heart → worsening failure.'}
6. {'step': 'Trending over time reveals the true trajectory', 'why': "A single snapshot can be misleading—valve area, mean gradient, peak velocity, left ventricular ejection fraction, and symptom status must be tracked over months. A patient who looks stable today may be on a steep downhill slope. Serial echocardiograms (every 6–12 months for moderate narrowing (stenosis), every 6 months for severe asymptomatic narrowing) and exercise testing (if the history is unclear) help catch the transition from compensated to decompensated before it's too late."}
7. {'step': 'Refer for intervention as soon as symptoms or severe narrowing (stenosis) with LV dysfunction appear', 'why': 'Do not wait for obvious heart failure or a life-threatening event. Current guidelines (2020 ACC/AHA, 2021 ESC/EACTS) recommend aortic valve replacement for any symptomatic severe narrowing (stenosis) (Class I indication) and for asymptomatic severe narrowing if ejection fraction drops below 50%, if another cardiac surgery is planned, or if exercise testing provokes symptoms or blood-pressure drop. The window for safe surgery narrows rapidly once the muscle decompensates—mortality risk climbs and recovery potential falls.'}

Domino effect

1. Valve area shrinks and left ventricular outflow resistance rises — 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) — 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) — 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 — 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 — 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) — 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 — 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 — 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.

Symptoms · what the patient feels, 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.
- Weak, slow pulse in the neck (pulsus parvus et tardus): The narrow valve blocks and slows the surge of blood leaving the heart, so the wave reaching your neck arteries is smaller and arrives late, like a wave hitting the beach after traveling through thick seaweed.
- Sudden death without warning: The thick heart muscle can develop dangerous rhythm problems or run so low on oxygen that it stops beating. A sudden drop in body fluid (from sweating, diarrhea, or water pills) can also make the heart's output fall to zero in seconds, especially during exercise.

Focused exam

- Harsh systolic ejection murmur at right upper sternal border, radiating to the neck: Blood is forced through a tiny opening in the aortic valve—like water squeezed through a pinched garden hose—creating a loud, rough sound. The tighter the valve, the louder and longer the murmur. This is the single strongest clue on exam that the valve is narrowed.
- Weak, slow carotid pulse (pulsus parvus et tardus): Because the valve is so narrow, the left ventricle can only push blood through slowly. The pulse wave reaches your fingers late and feels weak. If you feel this, the narrowing (stenosis) is severe.
- Narrow pulse pressure (small gap between systolic and diastolic blood pressure): The tight valve limits how much blood shoots out with each heartbeat, so the top number (systolic) cannot climb very high. The bottom number (diastolic) stays normal. A small gap between them—like 110/85 instead of 120/70—tells you the heart is not ejecting much blood per beat.
- Sustained left ventricular heave or lift felt at the apex: The left ventricle must squeeze extra hard to push blood through the narrow valve, so its walls grow thick (concentric hypertrophy—like a bodybuilder's biceps). You feel this powerful, prolonged push under your hand at the bottom tip of the heart.
- Fourth heart sound (S4) or paradoxically split second heart sound (S2): The thickened ventricle becomes stiff and hard to fill, so the atrium (the chamber above it) has to contract forcefully to top it off—that extra squeeze creates the S4 gallop. The ventricle also takes longer to empty through the narrow valve, so the aortic valve closes late, after the pulmonic valve, causing the split S2 to sound backward.
- Murmur gets louder with squatting, quieter with standing or Valsalva: Squatting increases the amount of blood returning to the heart (preload) and the resistance the heart pumps against (afterload), so more blood rushes through the narrow valve and the murmur grows louder. Standing or bearing down (Valsalva) does the opposite—less blood returns, so the murmur softens. This pattern helps you tell aortic narrowing (stenosis) apart from hypertrophic cardiomyopathy, which behaves in reverse.
- Signs of heart failure: wet lung sounds (rales), bulging neck veins (JVD), swollen ankles: When the left ventricle finally cannot keep up with the pressure demand, it starts to fail. Pressure backs up into the lungs, forcing fluid into the air sacs (pulmonary swelling (edema)), which you hear as crackles (rales). Pressure also backs up into the right heart, making neck veins bulge and legs swell. These signs mean the heart is decompensating and the patient needs a new valve urgently.

Diagnostics · every test answers a question

- Transthoracic echocardiography (TTE) → How tight is the valve opening, how high is the pressure the heart must generate, and is the left ventricle still pumping well?
- CT calcium scoring of the aortic valve and aortic root → How much calcium has hardened the valve leaflets, and what is the shape and size of the aorta for planning valve replacement?
- Brain natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) → Is the left ventricle under strain or starting to fail, even if the patient feels fine?
- Exercise stress test (treadmill or bike, only if the patient has severe AS by echo but reports no symptoms) → Does exertion unmask symptoms, cause blood pressure to drop, or trigger dangerous heart rhythms?
- Cardiac catheterization with direct pressure measurement across the aortic valve → If echo and clinical picture do not match, what is the true pressure gradient and valve area?
- Electrocardiogram (ECG) → Is there evidence of left ventricular hypertrophy, strain, or rhythm problems caused by chronic pressure overload?
- Chest X-ray → Is there cardiomegaly, calcification of the aortic valve, or pulmonary congestion from heart failure?
- Coronary angiography (before surgical aortic valve replacement in adults ≥ 40 years or with cardiac risk factors) → Are the coronary arteries blocked, requiring bypass grafting at the same time as valve replacement?

Treatment logic

- 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.
- 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.

- 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).
- 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—infected endocarditis).
- 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.
- Treat high blood pressure (hypertension) cautiously with beta-blockers or calcium channel blockers (avoid rapid afterload reduction) → Controls blood pressure without causing sudden drops in systemic vascular resistance. Beta-blockers (e.g., metoprolol) slow heart rate and reduce myocardial oxygen demand; non-dihydropyridine calcium channel blockers (e.g., diltiazem) also control rate. Both allow gradual, controlled lowering of blood pressure, which reduces left ventricular wall stress without collapsing preload or afterload.
- Treat atrial fibrillation (an irregular, quivering heartbeat) with rate control and anticoagulation (but avoid aggressive rate control that drops cardiac output) → Controls ventricular rate to optimize diastolic filling time (the period when the ventricle fills with blood). Anticoagulation (warfarin, DOACs) prevents stroke from clot formation in the fibrillating atrium. The stiff, hypertrophied ventricle depends heavily on the atrial kick (the final push of blood from atrial contraction), so losing sinus rhythm worsens symptoms and hemodynamics.

Medications

Valve replacement (SAVR or TAVR) · Surgical or catheter-based intervention (definitive treatment)

Aortic narrowing (stenosis) means the aortic valve—the door between the heart's main pumping chamber and the body—is too stiff and narrow. The heart has to squeeze extra hard to push blood through that tiny opening, like trying to blow up a balloon through a coffee stirrer. Over time the heart muscle gets thick and tired. No pill can widen a calcified (rock-hard) valve.

The only fix is to replace the valve. SAVR (surgical aortic valve replacement) means open-heart surgery: the surgeon cuts through the breastbone, stops the heart, cuts out the old valve, and sews in a new one (either a mechanical metal valve or a tissue valve from a cow or pig). TAVR (transcatheter aortic valve replacement) is a newer way: the doctor threads a collapsed new valve up a blood vessel (usually through a small cut in the groin), parks it inside the old valve, and pops it open—no big incision, heart keeps beating. Both methods give the blood a wide-open door again, so the heart can relax and pump normally.

1. Cardiologist or cardiac surgeon measures the valve: if the opening is less than 1.0 cm^2 (normally $3\text{--}4 \text{ cm}^2$), mean pressure difference across the valve is 40 mmHg or higher, or blood rushes through at 4 meters per second or faster, it is severe narrowing (stenosis).
2. Replace the valve immediately if the patient has any symptoms (chest pain, fainting, shortness of breath) OR if the heart's squeeze strength (ejection fraction) drops below 50 percent OR if another heart surgery is already planned and the valve is at least moderately narrow ($1.0\text{--}1.5 \text{ cm}^2$).
3. For SAVR: surgeon removes the calcified leaflets and sutures a prosthetic valve into the valve ring. For TAVR: interventionalist advances a crimped valve on a catheter, positions it under X-ray and ultrasound guidance, and expands it to push the old leaflets aside.
4. Blood now flows freely; the left ventricle no longer strains; over weeks to months the thickened heart muscle shrinks back toward normal size, and symptoms resolve.

SAFETY WATCH

Stroke: bits of calcium or air can travel to the brain during the procedure; risk is 1–3 percent. · Bleeding: both SAVR and TAVR require blood thinners during and after; watch for bleeding from any site. · Heart block: the new valve can press on the heart's wiring and slow the heartbeat dangerously; about 5–15 percent of TAVR patients need a permanent pacemaker afterward. · Infection (endocarditis): any prosthetic valve is more prone to infection than a native valve; take preventive antibiotics before dental work or dirty procedures. · Paravalvular leak (TAVR): the new valve may not seal perfectly against the old calcium, letting blood leak backward; severe leaks require a second procedure. · Valve clot formation (thrombosis): blood clots can form on the new valve, especially if you skip your anticoagulant; causes the valve to stick or fail.

Diuretics (e.g. furosemide) · Loop diuretic

If the heart is so stiff and weak from aortic narrowing (stenosis) that fluid backs up into the lungs (causing shortness of breath, coughing up pink foam, swelling in the legs), a diuretic—a 'water pill'—can buy time by telling the kidneys to dump extra salt and water into the urine. Think of it as opening a drain valve: less fluid in the tank means less pressure in the lungs and less work for the struggling heart. BUT diuretics do NOT open the narrow valve; they only

treat the symptom (fluid overload), not the cause. Use them gently and only as a bridge to valve replacement, because taking away too much fluid can drop the blood pressure so low that the brain and kidneys don't get enough blood.

1. Furosemide blocks a transporter protein (the $\text{Na}^+\text{-K}^+\text{-2Cl}^-$ cotransporter) on the cells lining the loop of Henle.
2. Without this pump working, sodium, potassium, and chloride stay in the urine instead of returning to the blood.
3. Water follows salt by osmosis (water always moves toward the saltier side), so more water leaves the body as urine.
4. Blood volume shrinks, pressure in the veins drops, and fluid in the lungs is pulled back into the bloodstream and then out through the kidneys.
5. Breathing gets easier within hours; leg swelling goes down over a day or two.

SAFETY WATCH

Low blood pressure (hypotension): too much diuretic leaves too little blood to fill the stiff left ventricle, and cardiac output crashes; the patient feels dizzy, weak, or faints. · Low potassium (hypokalemia): loop diuretics waste potassium in the urine; low potassium causes dangerous heart rhythms; check potassium often and replace it. · Kidney injury: over-diuresis shrinks blood volume so much that the kidneys don't get enough flow; creatinine rises. · Dehydration and thirst: losing too much water makes blood thick and patients feel parched. · Hearing loss (ototoxicity): high intravenous doses of furosemide can damage the inner ear, causing ringing or deafness; usually temporary but can be permanent.

Beta-blockers (e.g. metoprolol) · Beta-1 selective adrenergic antagonist

Beta-blockers are heart-slowng drugs. Adrenaline and stress hormones normally land on 'beta receptors' on heart cells and say 'beat faster, squeeze harder.' A beta-blocker sits on those receptors like a cork in a bottle, blocking the adrenaline signal. The heart rate drops. In aortic narrowing (stenosis), a very fast heart rate is bad because the heart has less time to fill with blood between beats, and filling time (diastole) is critical for the stiff, thick left ventricle. If a patient has chest pain (angina) because the thickened muscle needs extra oxygen and the heart is racing, slowing it down with a beta-blocker can ease the pain by giving the heart more rest time. BUT use beta-blockers very cautiously and at low doses—the diseased heart already struggles to pump through the narrow valve, and beta-blockers also weaken the squeeze (negative inotropy), which can tip a borderline heart into failure.

1. Adrenaline (epinephrine) and noradrenaline try to bind to beta-1 receptors during stress or exercise.

2. Metoprolol occupies the receptor first, so the adrenaline has nowhere to land.
3. Without adrenaline's signal, less calcium enters heart cells during each beat.
4. The heart beats more slowly (lower heart rate) and with less force (lower contractility).
5. Slower heart rate lengthens diastole (the filling phase), so the coronary arteries—which fill only during diastole—have more time to deliver oxygen to the thick muscle.
6. Angina improves because oxygen supply now matches the muscle's demand.

SAFETY WATCH

Low blood pressure and shock: blocking the heart's compensatory squeeze in a patient with severe narrowing (stenosis) can cause blood pressure to plummet; cardiac output may fall so low that organs fail. · A slow heart rate (Bradycardia) (heart rate too slow): if the rate drops below 50–55 beats per minute, the heart cannot pump enough blood; patient feels tired, dizzy, or passes out. · Heart failure: weakening an already struggling heart can tip it into acute decompensation (fluid backs up, lungs fill, legs swell). · Fatigue and exercise intolerance: patients often feel tired and unable to exert themselves because the heart can't speed up on demand. · Masking low blood sugar (hypoglycemia): beta-blockers blunt the warning signs (shaking, fast heart rate) of low blood sugar in diabetics; blood sugar can drop dangerously low without the patient noticing.

Statins (e.g. atorvastatin) · HMG-CoA reductase inhibitor

Statins lower cholesterol by blocking the liver's cholesterol factory. For years doctors hoped statins might also slow the calcium buildup on aortic valves (since calcific AS looks a bit like atherosclerosis—plaque in arteries). Multiple large trials (SALTIRE, SEAS, ASTRONOMER) tested this idea and found statins do NOT stop or slow aortic narrowing (stenosis) progression. However, many AS patients are older and have coronary artery disease (clogged heart arteries) at the same time. Statins DO prevent heart attacks by shrinking cholesterol plaques in those arteries. So give a statin if the patient has high cholesterol or known coronary disease, but do NOT give it to treat the valve itself—only to protect the arteries around the heart.

1. Atorvastatin blocks HMG-CoA reductase, the enzyme that runs the cholesterol assembly line inside liver cells.
2. Without this enzyme, the liver makes much less cholesterol.
3. Liver cells sense the cholesterol shortage and pull more LDL cholesterol (the 'bad' cholesterol) out of the bloodstream to make up for it.
4. Blood LDL levels drop by 30–50 percent.
5. Lower LDL means less cholesterol available to deposit in coronary artery plaques, so plaques stabilize or shrink slightly, and heart-attack risk falls.

SAFETY WATCH

Muscle pain and damage (myopathy): some patients feel aching or weakness; rarely, muscle breaks down (rhabdomyolysis), releasing proteins that clog the kidneys. · Liver enzyme elevation: statins can irritate the liver; check liver tests before starting and if symptoms appear, but routine monitoring is no longer required. · New-onset diabetes: statins slightly raise blood sugar in predisposed patients; the cardiovascular benefit still outweighs this risk. · Memory complaints: some patients report fuzzy thinking; usually reversible when the drug is stopped. · Drug interactions: statins are broken down by liver enzymes (CYP3A4); drugs that block this enzyme (e.g. some antibiotics, antifungals, grapefruit juice) can cause statin levels to spike and increase muscle-damage risk.

Red flags

- 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)

Nursing priorities

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 — 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 — 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 — 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.
4. Give intravenous fluids slowly and monitor lung sounds before and after each fluid bolus, stopping immediately and notifying the provider if crackles appear or worsen — The stiff left ventricle cannot handle sudden increases in volume. Too much fluid too fast will back up into the lungs and cause flash pulmonary edema (fluid flooding the lungs) (sudden severe lung fluid). But too little fluid will drop the preload and blood pressure will crash.
5. Hold or question any order for ACE inhibitors (lisinopril, enalapril), ARBs (losartan, valsartan), nitrates (nitroglycerin), or other vasodilators, and clarify with the provider before giving — Vasodilators drop afterload (the resistance the heart pumps against) and can cause severe low blood pressure (hypotension) (dangerously low blood pressure) because the heart cannot increase flow through the narrow valve to make up for the lower resistance. This can lead to shock, stroke, or cardiac arrest.
6. Teach the patient to stop activity immediately and sit or lie down if they feel chest pain, dizziness, or shortness of breath, and use the call light rather than walking to the bathroom alone — Exertion increases the heart's oxygen demand. When the narrowed valve prevents enough blood flow to meet that demand, the patient can faint, have a heart attack, or suffer sudden cardiac death. Early recognition and rest can prevent these events.
7. Administer preventive (prophylactic) antibiotics exactly as ordered (amoxicillin 2 grams by mouth or ampicillin 2 grams IV) 30 to 60 minutes before any dental procedure or invasive procedure involving mucous membranes — The abnormal valve creates turbulent blood flow that damages the valve surface. Bacteria from dental work or procedures can stick to the damaged valve and cause endocarditis (valve infection), which destroys the valve further and can cause sepsis or embolic stroke.
8. Assess and document the classic triad of symptoms daily—exertional trouble breathing (dyspnea) (shortness of breath with activity), angina (chest pain with activity), and fainting (syncope) (fainting with activity)—and report any new or worsening symptoms to the provider immediately — Once any symptom of the classic triad appears, average survival without valve replacement is 2 to 3 years for trouble breathing (dyspnea), 5 years for angina, and only 1 to 2 years for fainting (syncope). Early detection of symptom onset or progression ensures timely referral for life-saving valve replacement.

Sources

- ACC/AHA 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 Scientific Statements on Valvular Disease
- Cochrane Database Systematic Reviews: Interventions for Aortic Stenosis

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